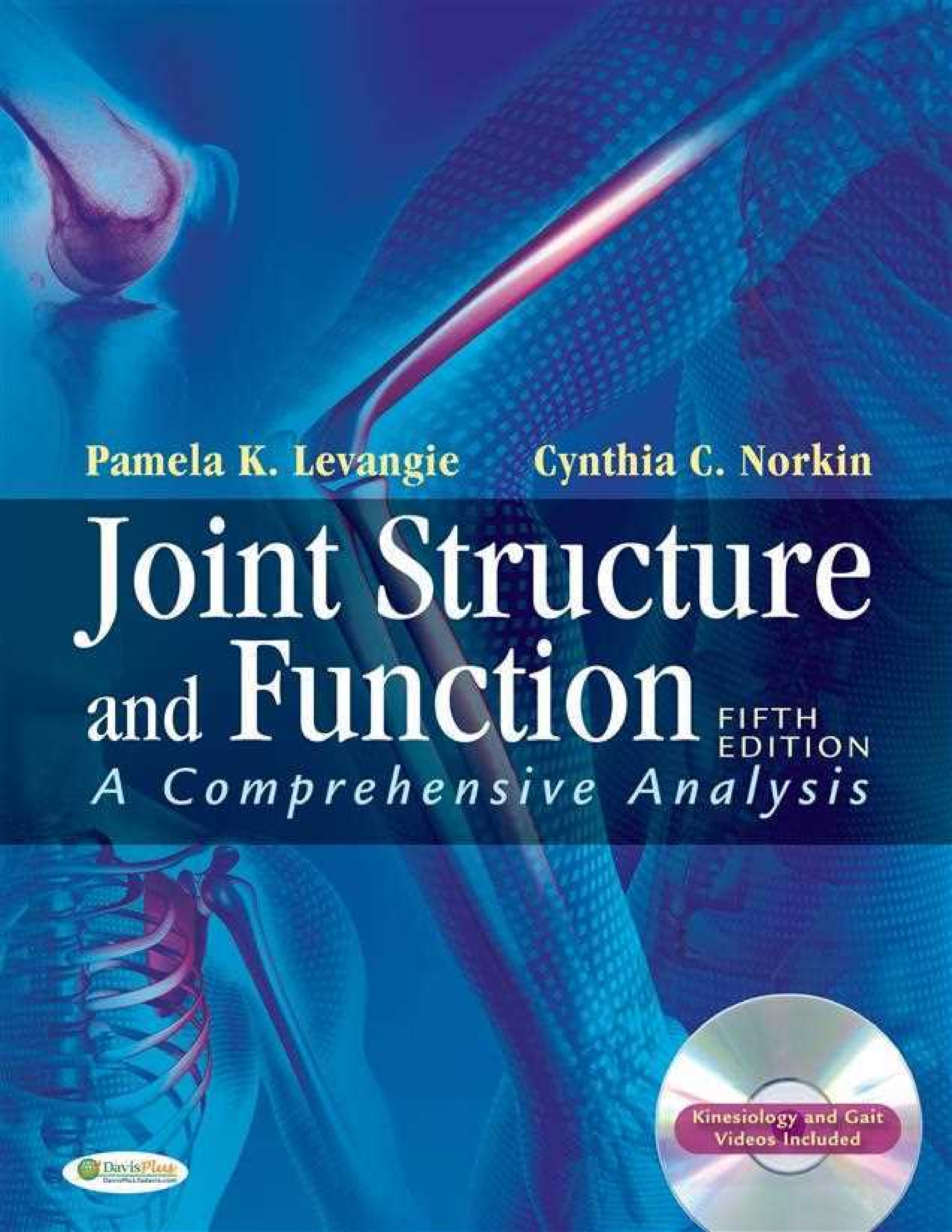

Pamela K. Levangie **Cynthia C. Norkin**

Joint Structure and Function

A Comprehensive Analysis

FIFTH EDITION

Kinesiology and Gait Videos Included

Joint Structure and Function

FIFTH EDITION

A Comprehensive Analysis

Joint Structure and Function

FIFTH EDITION

A Comprehensive Analysis

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PREFACE TO THE FIFTH EDITION

With the fifth edition of *Joint Structure and Function*, we maintain a tradition of excellence in education that began more than 25 years ago. We continue to respond to the dynamic environment of publishing, as well as to changes taking place in media, research technology, and in the education of individuals who assess human function. We include use of two- and four-color line drawings, enhanced instructor's tools, and new videos that all support and enhance the reader's experience.

Our contributors are chosen for their expertise in the areas of research, practice, and teaching—grounding their chapters in best and current evidence and in clinical relevance. Patient cases (in both "Patient Case" and "Patient Application" boxes) facilitate an understanding of the continuum between normal and impaired function, making use of emerging case-based and problem-based learning educational strategies. "Concept Cornerstones" and "Continuing Exploration" boxes provide the reader or the instructor additional flexibility in setting learning objectives.

What remains unchanged in this edition of *Joint Structure and Function* is our commitment to maintaining a text that provides a strong foundation in the principles that underlie an understanding of human structure and function while also being readable and as concise as possible. We hope that our years of experience in contributing to the education of health-care professionals allow us to strike a unique balance. We cannot fail to recognize the increased educational demands placed on many entry-level health-care professionals and hope that the updates to the fifth edition help students meet that demand. However, *Joint Structure and Function*, while growing with its readers, continues to recognize that the new reader requires elementary and interlinked building blocks that lay a strong but flexible foundation to best support continued learning and growth in a complex and changing world.

We very much appreciate our opportunity to contribute to health care by assisting in the professional development of the students and practitioners who are our readers.

Pamela K. Levangie
Cynthia C. Norkin

ACKNOWLEDGMENTS

The fifth edition of *Joint Structure and Function* is made possible only by the continued and combined efforts of many people and groups. We are, first and foremost, grateful for the time, effort, and expertise of our esteemed contributors with whom it has been a pleasure to work. Our thanks, therefore, to Drs. Sam Ward, Sandra Curwin, Gary Chleboun, Diane Dalton, Julie Starr, Pam Ritzline, Paula Ludewig, John Borstad, RobRoy Martin, Lynn Snyder-Mackler, Michael Lewek, Erin Hartigan, Janice Eng, and Sandra Olney, as well as to Ms. Noelle Austin and Mr. Benjamin Kivlan. Additionally, we want to express our appreciation to the individuals who helped develop the ancillary materials that support the fifth edition, including the Instructor's Resources developed by Ms. Christine Conroy and the videos developed by Dr. Lee Marinko and Center City Film & Video. We would also like to acknowledge and thank the individuals who contributed their comments and suggestions as reviewers (listed on page xi), as well as those who passed along their unsolicited suggestions through the years, including our students.

We extend our continuing gratitude to F. A. Davis for their investment in the future of *Joint Structure and Function* and its ancillary materials. Particular thanks go to Margaret Biblis (Publisher), Melissa Duffield (Acquisitions Editor), Karen Carter (Developmental Editor), Yvonne Gillam (Developmental Editor), George Lang (Manager of Content Development), David Orzechowski (Managing Editor), Robert Butler (Production Manager), Carolyn O'Brien (Manager of Art and Design), Katherine Margeson (Illustration Coordinator), and Stephanie Rukowicz (Assistant Developmental Editor) who provided great support. As always we must thank the artists who, through the years, provided the images that are so valuable to the readers. These include artists of past editions, Joe Farnum, Timothy Malone, and Anne Raines. New to the fifth edition is Dartmouth Publishing, Inc., adding both new figures and enhanced color to the text.

Finally, we acknowledge and thank our colleagues and families, without whose support this work could not have been done and to whom we are eternally indebted.

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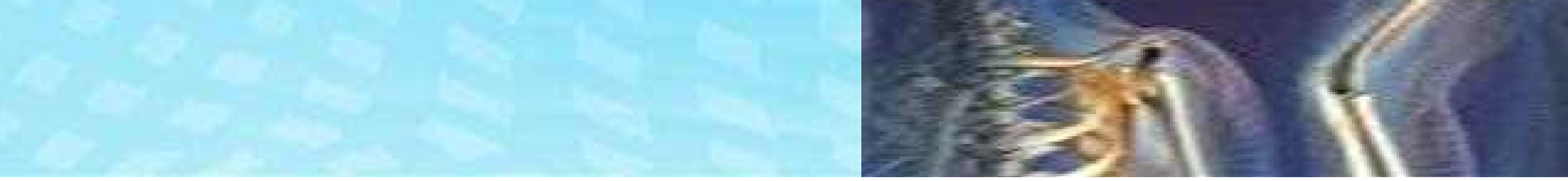

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FIFTH EDITION

A Comprehensive Analysis

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Chapter 1

Biomechanical Applications to Joint Structure and Function

Samuel R. Ward, PT, PhD

"Humans have the capacity to produce a nearly infinite variety of postures and movements that require the tissues of the body to both generate and respond to forces that produce and control movement."

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- Perpendicular and Parallel Force Effects
  - Determining Magnitudes of Component Forces
  - Force Components and the Angle of Application of the Force
- Translatory Effects of Force Components
- Rotary Effects of Force Components
  - Rotation Produced by Perpendicular (Fy) Force Components
  - Rotation Produced by Parallel (Fx) Force Components

Multisegment (Closed-Chain) Force Analysis

INTRODUCTION

Humans have the capacity to produce a nearly infinite variety of postures and movements that require the structures of the human body to both generate and respond to forces that produce and control movement at the body's joints. Although it is impossible to capture all the kinesiologic elements that contribute to human musculoskeletal function at a given point in time, knowledge of at least some of the physical principles that govern the body's response to active and passive stresses is prerequisite to an understanding of both human function and dysfunction.

We will examine some of the complexities related to human musculoskeletal function by examining the roles of the bony segments, joint-related connective tissue structure, and muscles, as well as the external forces applied to those structures. We will develop a conceptual framework that provides a basis for understanding the stresses on the body's major joint complexes and the responses to those stresses. Case examples and clinical scenarios will be used to ground the reader's understanding in relevant applications of the presented principles. The objective is to cover the key biomechanical principles necessary to understand individual joints and their interdependent functions in posture and locomotion. Although we acknowledge the role of the neurological system in motor control, we leave it to others to develop an understanding of the theories that govern the roles of the controller and feedback mechanisms.

This chapter will explore the biomechanical principles that must be considered to examine the internal and external forces that produce or control movement. The focus will be largely on rigid body analysis; the next two chapters explore how forces affect deformable connective tissues (Chapter 2) and how muscles create and are affected by forces (Chapter 3). Subsequent chapters then examine the interactive nature of force, stress, tissue behaviors, and function through a regional exploration of the joint complexes of the body. The final two chapters integrate the function of the joint complexes into the comprehensive tasks of posture (Chapter 13) and gait (Chapter 14).

In order to maintain our focus on clinically relevant applications of the biomechanical principles presented in this chapter, the following case example will provide a framework within which to explore the relevant principles of biomechanics.

1-1 Patient Case

John Alexander is 20 years old, is 5 feet 9 inches (1.75 m) in height, and weighs 165 pounds (~75 kg or 734 N). John is a member of the university's lacrosse team. He sustained an injury when another player fell onto the posterior-lateral aspect of his right knee. Physical examination and magnetic resonance imaging (MRI) resulted in a diagnosis of a tear of the medial collateral ligament, a partial tear of the anterior cruciate ligament (ACL), and a partial tear of the medial meniscus. John agreed with the orthopedist's recommendation that a program of knee muscle strengthening was in order before moving to more aggressive options. The initial focus will be on strengthening the quadriceps muscle. The fitness center at the university has a leg-press machine (Fig. 1–1A) and a free weight boot (see Fig. 1–1B) that John can use.

As we move through this chapter, we will consider the biomechanics of each of these rehabilitative options in relation to John's injury and strengthening goals.

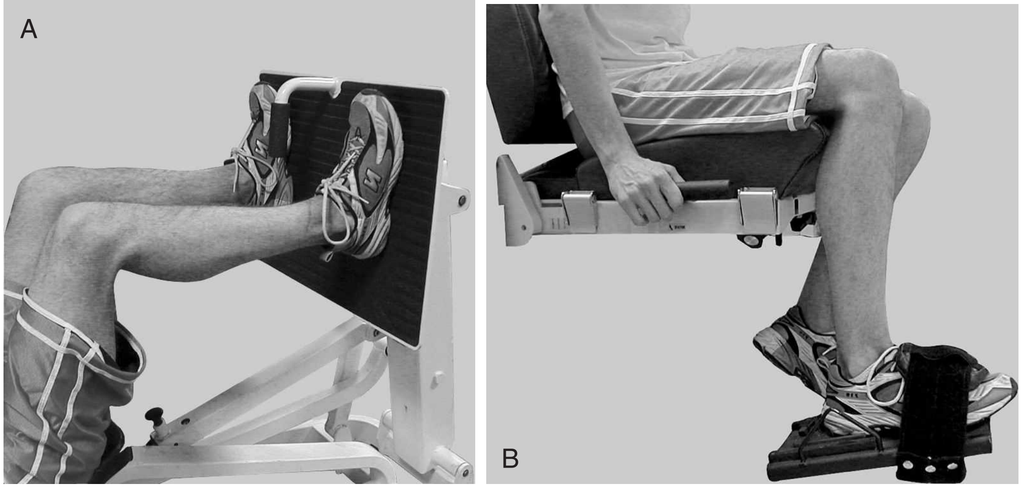

Figure 1–1 **A.** Leg-press exercise apparatus for strengthening hip and knee extensor muscles. **B.** Free weight boot for strengthening knee extensor muscles.

Side-bar: The case in this chapter provides a background for the presentation of biomechanical principles. The values and angles chosen for the forces in the various examples used in this case are representative but are not intended to correspond to values derived from sophisticated instrumentation and mathematical modeling; different experimental conditions, instrumentation, and modeling can provide substantially different and often contradictory findings.

Human motion is inherently complex, involving multiple segments (bony levers) and forces that are most often applied to two or more segments simultaneously. In order to develop a conceptual model that can be understood and applied clinically, the common strategy is to focus on one segment at a time. For the purposes of analyzing John Alexander's issues, the focus will be on the leg-foot segment, treated as if it were one rigid unit acting at the knee joint. Figure 1–2A and 1–2B is a schematic representation of the leg-foot segment in the leg-press and free weight boot situations. The leg-foot segment is the focus of the figure, although the contiguous components (distal femur, footplate of the leg-press machine, and weight boot) are maintained to give context. In some subsequent figures, the femur, footplate, and weight boot are omitted for clarity, although the forces produced by these segments and objects will be shown. This limited visualization of a segment (or a selected few segments) is referred to as a free body diagram or a space diagram. If proportional representation of all forces is maintained as the forces are added to the segment under consideration, it is known as a "free body diagram." If the forces are shown but a simplified understanding rather than graphic accuracy is the goal, then the figure is referred to as a "space diagram."[1] We will use space diagrams in this chapter and text because the forces are generally not drawn in proportion to their magnitudes.

As we begin to examine the leg-foot segment in either the weight boot or leg-press exercise situation, the first step is to describe the motion of the segment that is or will be occurring. This involves the area of biomechanics known as **kinematics.**

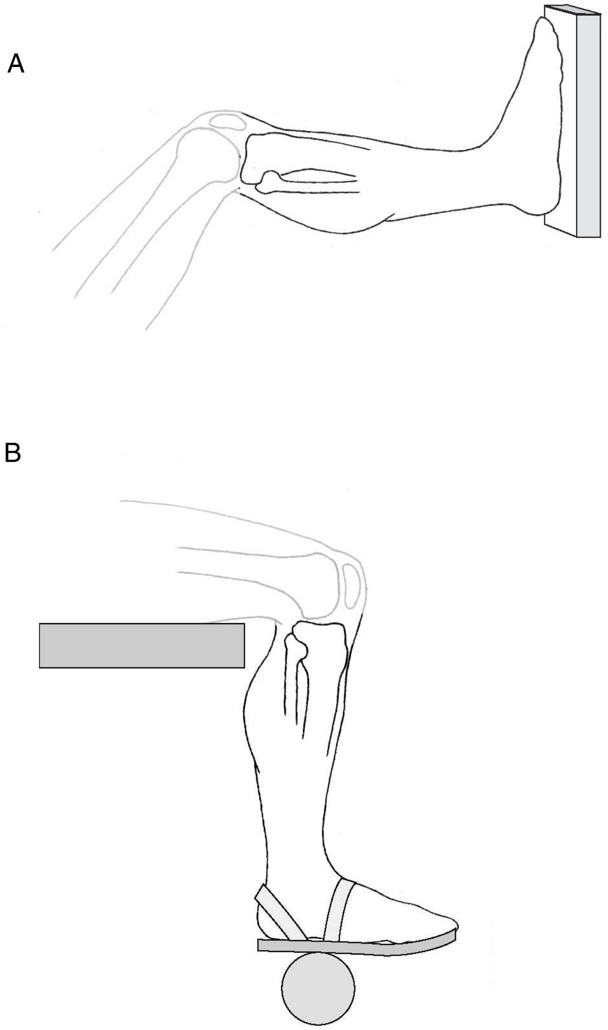

Figure 1–2 **A.** Schematic representation of the leg-foot segment in the leg-press exercise, with the leg-foot segment highlighted for emphasis. **B.** Schematic representation of the leg-foot segment in the weight boot exercise, with the leg-foot segment highlighted for emphasis.

Part 1: Kinematics and Introduction to Kinetics

DESCRIPTIONS OF MOTION

Kinematics includes the set of concepts that allows us to describe the **displacement** (the change in position over time) or motion of a segment without regard to the forces that cause that movement. The human skeleton is, quite literally, a system of segments or levers. Although bones are not truly rigid, we will assume that bones behave as rigid levers. There are five kinematic variables that fully describe the motion, or the displacement, of a segment: (1) the type of displacement (motion), (2) the location in space of the displacement, (3) the direction of the displacement of the segment, (4) the magnitude of the displacement, and (5) the rate of change in displacement (velocity) or the rate of change of velocity (acceleration).

Types of Displacement

Translatory and rotary motions are the two basic types of movement that can be attributed to any rigid segment. General motions are achieved by combining translatory and rotary motions.

Translatory Motion

Translatory motion (linear displacement) is the movement of a segment in a straight line. In true translatory motion, each point on the segment moves through the same distance, at the same time, in parallel paths. In human movement, pure translatory movements are rare. However, a clinical example of attempted translatory motion is joint mobilization, in which a clinician attempts to impose the linear motion of one bony segment on another, allowing joint surfaces to slide past one another. A specific example of such imposed motion is the anterior drawer test for anterior cruciate ligament (ACL) integrity at the knee (Fig. 1–3). This example of translatory motion assumes, however, that the leg segment is free and unconstrained—that is, that the leg segment is not linked to the femur by soft tissues. Although it is best to describe pure translatory motion by using an example of an isolated and unconstrained segment, segments of the body are neither isolated nor unconstrained. Every segment is linked to at least one other segment, and most human motion occurs as movement of more than one segment at a time. The translation of the leg segment in Figure 1–3 is actually produced by the near-linear motion of the proximal tibia. In fact, translation of a body segment rarely occurs in human motion without some concomitant rotation (rotary motion) of that segment (even if the rotation is barely visible).

Rotary Motion

Rotary motion (angular displacement) is movement of a segment around a fixed axis **(center of rotation [CoR])** in a curved path. In true rotary motion, each point on the segment moves through the same angle, at the same time, at a constant distance from the center of rotation. True rotary motion can occur only if the segment is prevented from translating and is forced to rotate about a fixed axis. This does not often happen in human movement. In the example in Figure 1–4, all points on the leg-foot segment appear to move through the same distance at the same time around

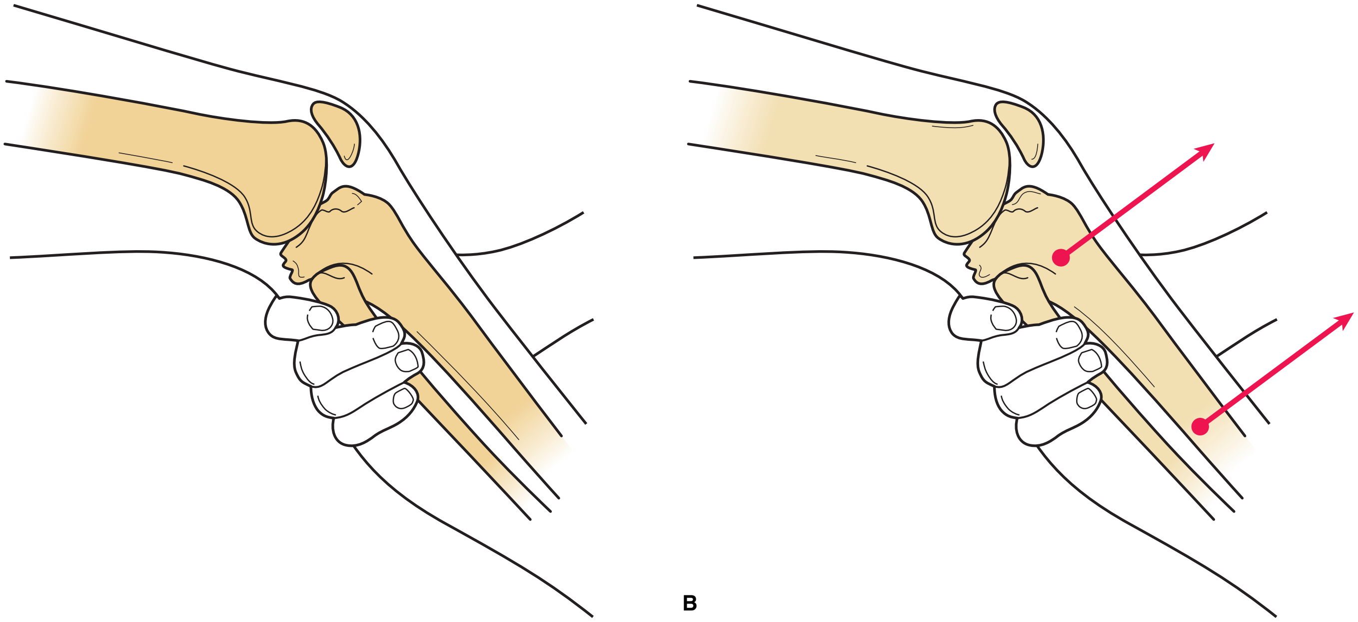

Figure 1–3 An example of translatory motion is the anterior drawer test for ACL integrity. Ideally, the tibial plateau translates anteriorly from the starting position **(A)** to the ending position **(B)** as the examiner exerts a linear load on the proximal tibia. Under ideal conditions, each point on the tibia moves through the same distance, at the same time, in parallel paths.

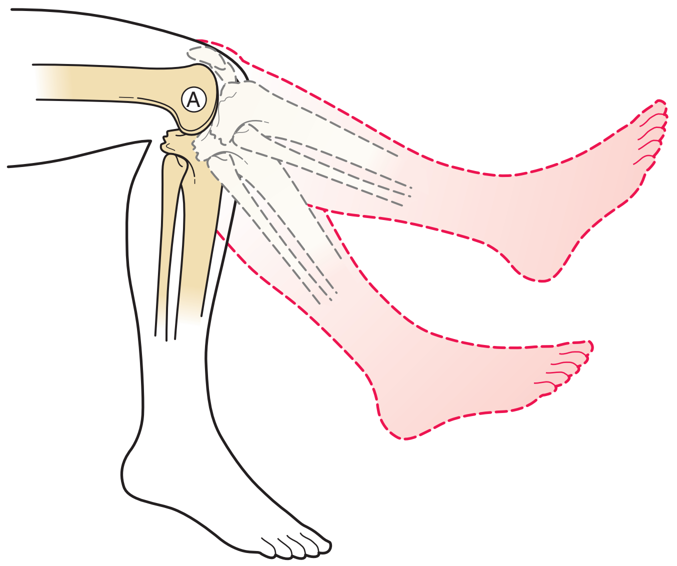

Figure 1–4 Rotary motion. Each point in the tibia segment moves through the same angle, at the same time, at a constant distance from the center of rotation or axis (A).

what appears to be a fixed axis. In actuality, none of the body segments move around truly fixed axes; all joint axes shift at least slightly during motion because segments are not sufficiently constrained to produce pure rotation.

General Motion

When nonsegmented objects are moved, combinations of rotation and translation **(general motion)** are common. If someone were to attempt to push a treatment table with swivel casters across the room by using one hand, it would be difficult to get the table to go straight (translatory motion); it would be more likely to both translate and rotate. When rotary and translatory motions are combined, a number of terms can be used to describe the result.

Curvilinear (**plane** or **planar**) **motion** designates a combination of translation and rotation of a segment in *two dimensions* (parallel to a plane with a maximum of three degrees of freedom).[2–4] When this type of motion occurs, the axis about which the segment moves is not fixed but, rather, shifts in space as the object moves. The axis around which the segment appears to move in any part of its path is referred to as the **instantaneous center of rotation (ICoR),** or **instantaneous axis of rotation (IaR).** An object or segment that travels in a curvilinear path may be considered to be undergoing rotary motion around a fixed but quite distant CoR[3,4]; that is, the curvilinear path can be considered a segment of a much larger circle with a distant axis.

Three-dimensional motion is a general motion in which the segment moves across all three dimensions. Just as curvilinear motion can be considered to occur around a single distant center of rotation, three-dimensional motion can be considered to be occurring around a **helical axis of motion (HaM),** or **screw axis of motion.**[3]

As already noted, motion of a body segment is rarely sufficiently constrained by the ligamentous, muscular, or other bony forces acting on it to produce pure rotary motion. Instead, there is typically at least a small amount of translation (and often a secondary rotation) that accompanies the primary rotary motion of a segment at a joint. Most joint rotations, therefore, take place around a series of instantaneous center of rotations. The “axis” that is generally ascribed to a given joint motion (e.g., knee flexion) is typically a midpoint among these instantaneous centers of rotation rather than the true center of rotation. Because most body segments actually follow a curvilinear path, the true center of rotation is the point around which true rotary motion of the segment would occur and is generally quite distant from the joint.[3,4]

Location of Displacement in Space

The rotary or translatory displacement of a segment is commonly located in space by using the three-dimensional Cartesian coordinate system, borrowed from mathematics, as a useful frame of reference. The origin of the x-axis, y-axis, and z-axis of the coordinate system is traditionally located at the **center of mass (CoM)** of the human body, assuming that the body is in **anatomic position** (standing facing forward, with palms forward) (Fig. 1–5). According to the common system described by Panjabi and White, the x-axis runs side-to-side in the body and is labeled in the body as the **coronal axis**; the y-axis runs up and down in the body and is labeled in the body as the **vertical axis**; the z-axis runs front to back in the body and is labeled in the body as the **anteroposterior (A-P) axis.**[3] Motion of a segment can occur either *around* an axis (rotation) or *along* an axis (translation). An unconstrained segment can either rotate or translate around each of the three axes, which results in six potential options for motion of that segment.

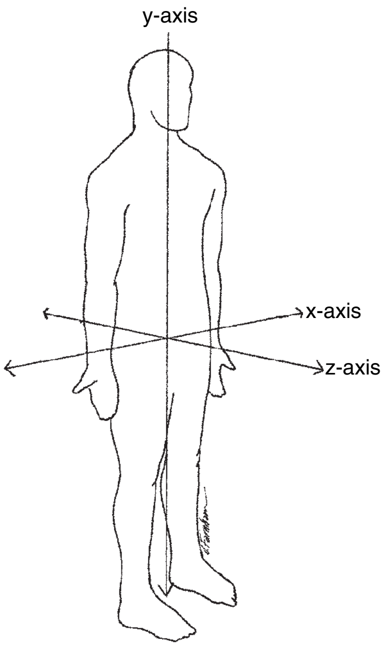

Figure 1–5 Body in anatomic position showing the x-axis, y-axis, and z-axis of the Cartesian coordinate system (the coronal, vertical, and anteroposterior axes, respectively).

The options for movement of a segment are also referred to as **degrees of freedom.** A completely unconstrained segment, therefore, always has six degrees of freedom. Segments of the body, of course, are not unconstrained. A segment may appear to be limited to only one degree of freedom (although, as already pointed out, this rarely is strictly true), or all six degrees of freedom may be available to it.

Rotation of a body segment is described not only as occurring around one of three possible axes but also as moving in or parallel to one of three possible **cardinal planes.** As a segment rotates *around* a particular axis, the segment also moves in a plane that is both perpendicular to that axis of rotation and parallel to another axis. Rotation of a body segment *around* the x-axis or coronal axis occurs in the **sagittal plane** (Fig. 1–6). Sagittal plane motions are most easily visualized as front-to-back motions of a segment (e.g., flexion/extension of the upper extremity at the glenohumeral joint).

Rotation of a body segment *around* the y-axis or vertical axis occurs in the **transverse plane** (Fig. 1–7). Transverse plane motions are most easily visualized as motions of a segment parallel to the ground (e.g., medial/lateral rotation of the lower extremity at the hip joint). Transverse plane motions often occur around axes that pass through the length of long bones that are not truly vertically oriented. Consequently, the term **longitudinal** (or **long**) **axis** is often used instead of "vertical axis." Rotation of a body segment *around* the z-axis or A-P axis occurs in the **frontal plane** (Fig. 1–8). Frontal plane motions are most easily visualized as side-to-side motions of the segment (e.g., abduction/adduction of the upper extremity at the glenohumeral joint).

Rotation and translation of body segments are not limited to motion along or around cardinal axes or within cardinal planes. In fact, cardinal plane motions are the exception rather than the rule and, although useful, are an oversimplification of human motion. If a motion (whether in or around a cardinal axis or plane) is limited to rotation around a single axis *or* translatory motion along a single axis, the motion is considered to have one degree of freedom. Much more commonly, a segment moves in three dimensions with two or more degrees of freedom. The following example demonstrates a way in which rotary and translatory motions along or around one or more axes can combine in human movement to produce two- and three-dimensional segmental motion.

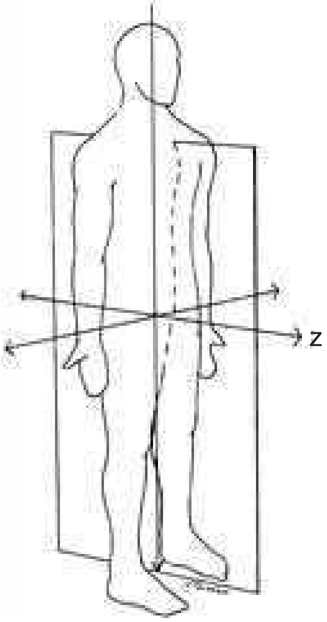

Figure 1–6 The sagittal plane.

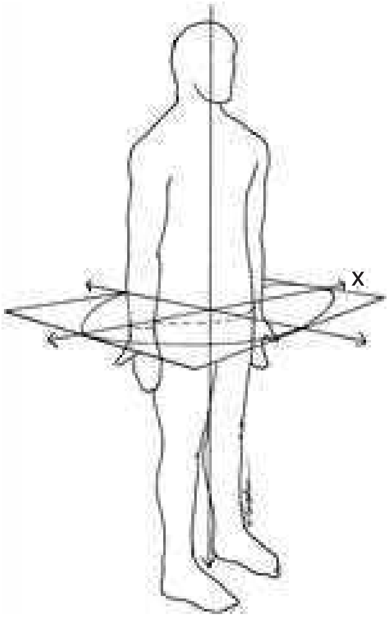

Figure 1–7 The transverse plane.

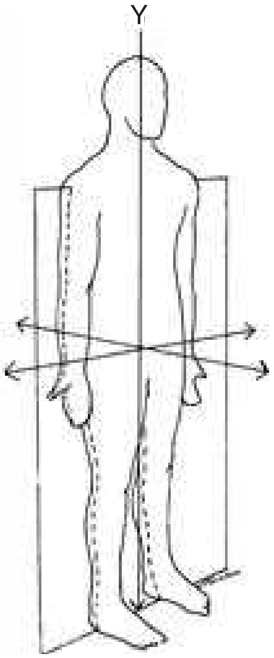

Figure 1–8 The frontal plane.

Example 1-1

When the forearm-hand segment and a glass (all considered as one rigid segment) are brought to the mouth (Fig. 1–9), rotation of the segment around an axis and translation of that segment through space occur simultaneously. As the forearm-hand segment and glass rotate around a coronal axis at the elbow joint (one degree of freedom), the shoulder joint also rotates to translate the forearm-hand segment forward in space along the forearm-hand segment's A-P axis (one degree of freedom). By combining the two degrees of freedom, the elbow joint axis (the instantaneous center of rotation for flexion of the forearm-hand segment) does not remain fixed but moves in space; the glass attached to the forearm-hand segment moves through a curvilinear path.

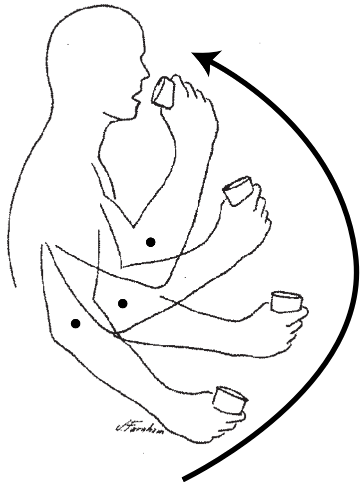

Figure 1–9 The forearm-hand segment rotates around a coronal axis at the elbow joint and along A-P axis (through rotation at the shoulder joint), using two degrees of freedom that result in a moving axis of rotation and produce curvilinear motion of the forearm-hand segment.

Direction of Displacement

Even if displacement of a segment is confined to a single axis, the rotary or translatory motion of a segment around or along that axis can occur in two different directions. For rotary motions, the direction of movement of a segment around an axis can be described as occurring in a clockwise or counterclockwise direction. Clockwise and counterclockwise rotations are generally assigned negative and positive signs, respectively.[5] However, these terms are dependent on the perspective of the viewer (viewed from the left side, flexing the forearm is a clockwise movement; if the subject turns around and faces the opposite direction, the same movement is now seen by the viewer as a counterclockwise movement). Anatomic terms describing human movement are independent of viewer perspective and, therefore, more useful clinically. Because there are two directions of rotation (positive and negative) around each of the three cardinal axes, we can describe three pairs of (or six different) anatomic rotations available to body segments.

Flexion and **extension** are motions of a segment occurring around the same axis and in the same plane (uniaxial or uniplanar) but in opposite directions. Flexion and extension generally occur in the sagittal plane around a coronal axis, although exceptions exist (e.g., carpometacarpal flexion and extension of the thumb). Anatomically, flexion is the direction of segmental rotation that brings ventral surfaces of adjacent segments closer together, whereas extension is the direction of segmental rotation that brings dorsal surfaces closer together.

Side-bar: Defining flexion and extension by ventral and dorsal surfaces makes use of the true embryologic origin of the words *ventral* and *dorsal*, rather than using these terms as synonymous with *anterior* and *posterior*, respectively.

Abduction and **adduction** of a segment occur around the A-P axis and in the frontal plane but in opposite directions (although carpometacarpal abduction and adduction of the thumb again serve as exceptions). Anatomically, abduction brings the segment away from the midline of the body, whereas adduction brings the segment toward the midline of the body. When the moving segment *is* part of the midline of the body (e.g., the trunk or the head), the rotary movement is commonly termed **lateral flexion** (to the right or to the left).

Medial (or **internal**) **rotation** and **lateral** (or **external**) **rotation** are opposite motions of a segment that generally occur around a vertical (or longitudinal) axis in the transverse plane. Anatomically, medial rotation occurs as the segment moves parallel to the ground and toward the midline, whereas lateral rotation occurs opposite to that. When the segment is part of the midline (e.g., the head or trunk), rotation in the transverse plane is simply called rotation to the right or rotation to the left. The exceptions to the general rules for naming motions must be learned on a joint-by-joint basis.

As is true for rotary motions, translatory motions of a segment can occur in one of two directions along any of the three axes. Again by convention, linear displacement of a segment along the x-axis is considered positive when displacement is to the right and negative when it is to the left. Linear displacement of a segment up along the y-axis is considered positive, and such displacement down along the y-axis is negative. Linear displacement of a segment forward (anterior) along the z-axis is positive, and such displacement backward (posterior) is negative.[1]

Magnitude of Displacement

The magnitude of rotary motion (or angular displacement) of a segment can be given either in degrees **(United States [US] units)** or in **radians (International System of Units [SI units]).** If an object rotates through a complete circle, it has moved through 360°, or 6.28 radians. A radian is literally the ratio of an arc to the radius of its circle (Fig. 1–10). One radian is equal to 57.3°; 1° is equal to 0.01745 radian. The magnitude of rotary motion that a body segment moves

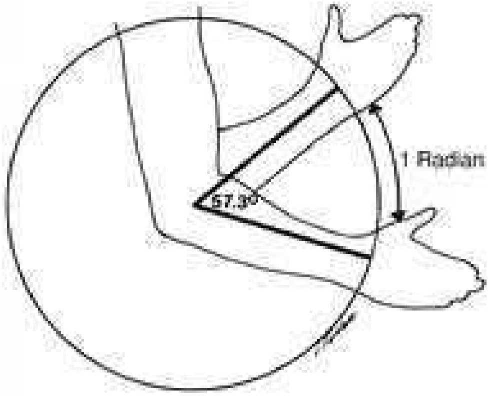

Figure 1–10 An angle of 57.3° describes an arc of 1 radian.

through or can move through is known as its **range of motion (ROM).** The most widely used standardized clinical method of measuring available joint ROM is goniometry, with units given in degrees. Consequently, we typically will use degrees in this text to identify angular displacements (rotary motions). ROM may be measured and stored on computer for analysis by an electrogoniometer or a three-dimensional motion analysis system, but these are available predominantly in research environments. Although we will not be addressing instruments, procedures, technological capabilities, or limitations of these systems, data collected by these sophisticated instrumentation systems are often the basis of research cited through the text.

Translatory motion or displacement of a segment is quantified by the linear distance through which the object or segment is displaced. The units for describing translatory motions are the same as those for length. The SI system's unit is the meter (or millimeter or centimeter); the corresponding unit in the US system is the foot (or inch). This text will use the SI system but includes a US conversion when this appears to facilitate understanding (1 inch = 2.54 cm). Linear displacements of the entire body are often measured clinically. For example, the 6-minute walk[6] (a test of functional status in individuals with cardiorespiratory problems) measures the distance (in feet or meters) someone walks in 6 minutes. Smaller full-body or segment displacements can also be measured by three-dimensional motion analysis systems.

Rate of Displacement

Although the magnitude of displacement is important, the rate of change in position of the segment (the displacement per unit time) is equally important. Displacement per unit time regardless of direction is known as **speed,** whereas displacement per unit time in a given direction is known as **velocity.** If the velocity is changing over time, the change in velocity per unit time is **acceleration.** Linear velocity (velocity of a translating segment) is expressed as meters per second (m/sec) in SI units or feet per second (ft/sec) in US units; the corresponding units for acceleration are meters per second squared (m/sec^2) and feet per second squared (ft/sec^2). Angular velocity (velocity of a rotating segment) is expressed as degrees per second (deg/sec), whereas angular acceleration is given as degrees per second squared (deg/sec^2).

An electrogoniometer or a three-dimensional motion analysis system allows documentation of the changes in displacement over time. The outputs of such systems are increasingly encountered when summaries of displacement information are presented. A computer-generated time-series plot, such as that in Figure 1–11, graphically portrays not only the angle between two bony segments (or the rotation of one segment in space) at each point in time but also the direction of motion. The steepness of the slope of the graphed line represents the angular velocity. Figure 1–12 plots the variation in linear acceleration of a body segment (or a point on the body segment) over time without regard to changes in joint angle.

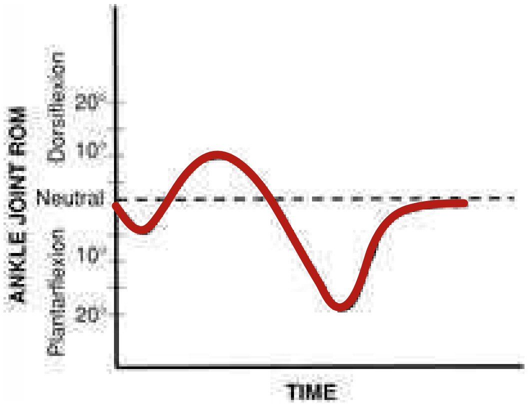

Figure 1–11 When a joint's range of motion is plotted on the y-axis (vertical axis) and time is plotted on the x-axis (horizontal axis), the resulting time-series plot portrays the change in joint position over time. The slope of the plotted line reflects the velocity of the joint change.

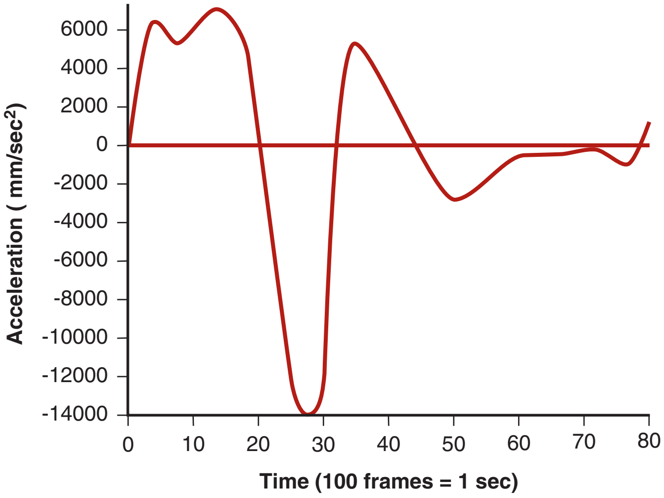

Figure 1–12 Movement of a point on a segment can be displayed by plotting the acceleration of the segment (y-axis) over time (x-axis). The slope and trend of the line represent increases or decreases in magnitude of acceleration as the movement continues. *(Courtesy of Fetters, L: Boston University, 2003.)*

INTRODUCTION TO FORCES

Definition of Forces

Kinematic descriptions of human movement permit us to visualize motion but do not give us an understanding of why the motion is occurring. This requires a study of forces. Whether a body or body segment is in motion or at rest depends on the forces exerted on that body. A force, simplistically speaking, is a push or a pull exerted by one object or substance on another. Any time two objects make contact, they will either push on each other or pull on each other with some magnitude of force (although the magnitude may be small enough to be disregarded). The unit for a force (a push or a pull) in the SI system is the **newton (N)**; the unit in the US system is the **pound (lb).** The concept of a force as a push or pull can readily be used to describe the forces encountered in evaluating human motion.

Continuing Exploration 1-1:

A Force

Although a force is most simply described as a push or a pull, it is also described as a "theoretical concept" because only its effects (acceleration) can be measured.[4] Consequently, a force (F) is described by the acceleration (a) of the object to which the force is applied, with the acceleration being directly proportional to the mass (m) of that object; that is,

$$\text{force} = (\text{mass})(\text{acceleration})$$
$$\text{or } F = (m)(a)$$

Because mass is measured in kilograms (kg) and acceleration in m/sec^2, the unit for force is actually kg-m/sec^2 or, more simply, the newton (N). A newton is the force required to accelerate 1 kg at 1 m/sec^2 (the pound is correspondingly the amount of force required to accelerate a mass of 1 slug [to be described] at 1 ft/sec^2).

External forces are pushes or pulls on the body that arise from sources outside the body. **Gravity (g),** the attraction of the earth's mass to another mass, is an external force that under normal conditions constantly affects all objects. The **weight (W)** of an object is the pull of gravity on the object's mass with an acceleration of 9.8 m/sec^2 (or 32.2 ft/sec^2) in the absence of any resistance:

$$\text{weight} = (\text{mass})(\text{gravity})$$
$$\text{or } W = (m)(g)$$

Because weight is a force, the appropriate unit is the newton (or pound). However, it is not uncommon to see weight given in **kilograms (kg),** although the kilogram is more correctly a unit of mass. In the US system, the pound is commonly used to designate mass when it is appropriately a force unit (1 kg = 2.2 lb). The correct unit for mass in the US system is the infrequently used **slug** (1 slug = 14.59 kg).

Continuing Exploration 1-2:

Force and Mass Unit Terminology

Force and mass units are often used incorrectly in the vernacular. The average person using the metric system expects a produce scale to show weight in kilograms, rather than in newtons. In the United States, the average person appropriately thinks of weight in pounds but also considers the pound to be a unit of mass. Because people commonly tend to think of mass in terms of weight (the force of gravity acting on the mass of an object) and because the slug is an unfamiliar unit to most people, the pound is often used to represent the mass of an object in the US system.

One attempt to maintain common usage while clearly differentiating force units from mass units for scientific purposes is to designate lb and kg as mass units and to designate the corresponding force units as lbf (pound-force) and kgf (kilogram-force).[3,4] When the kilogram is used as a force unit:

$$1 \text{ kgf} = 9.8 \text{ N}$$

When the pound is used as a mass unit:

$$1 \text{ pound} = 0.031 \text{ slugs}$$

These conversions assume an unresisted acceleration of gravity of 9.8 m/sec^2 or 32.2 ft/sec^2, respectively.

The distinction between a measure of mass and a measure of force is important because mass is a scalar quantity (without action line or direction), whereas the newton and pound are measures of force and have vector characteristics. In this text, we will consistently use the terms *newton* and *pound* as force units and the terms *kilogram* and *slug* as the corresponding mass units.

Because gravity is the most consistent of the forces encountered by the body, gravity should be the first force to be considered when the potential forces acting on a body segment are identified. However, gravity is only one of an infinite number of external forces that can affect the body and its segments. Examples of other external forces that may exert a push or pull on the human body or its segments are wind (the push of air on the body), water (the push of water on the body), other people (the push or pull of an examiner on John Alexander's leg), and other objects (the push of floor on the feet, the pull of a weight boot on the leg). A critical point is that the forces on the body or any one segment must come from something that is touching the body or segment. The major exception to this rule is the force of gravity. However, if permitted, the conceit that gravity (the pull of the earth) "contacts" all objects on earth, we can circumvent this exception and make it a standing rule that *all forces on a segment must come from something that is contacting that segment* (including gravity). The obverse also holds true: that *anything that contacts a segment must create a force on that segment*, although the magnitude may be small enough to disregard.

Concept Cornerstone **1-1**

Primary Rules of Forces

- All forces on a segment must come from something that is contacting that segment.
- Anything that contacts a segment *must* create a force on that segment (although the magnitude may be small enough to disregard).
- Gravity can be considered to be "touching" all objects.

Internal forces are forces that act on structures of the body and arise from the body's own structures (i.e., the contact of two structures within the body). A few common examples are the forces produced by the muscles (the pull of the biceps brachii on the radius), the ligaments (the pull of a ligament on a bone), and the bones (the push of one bone on another bone at a joint). Some forces, such as atmospheric pressure (the push of air pressure), work both inside and outside the body, but—in our definition—these are considered external forces because the source is not a body structure.

External forces can either facilitate or restrict movement. Internal forces are most readily recognized as essential for *initiation* of movement. However, it should be apparent that internal forces also control or counteract movement produced by external forces, as well as counteracting other internal forces. Much of the presentation and discussion in subsequent chapters of this text relate to the interactive role of internal forces, not just in causing movement but also in maintaining the integrity of joint structures against the effects of external forces and other internal forces.

Force Vectors

All forces, regardless of the source or the object acted on, are **vector** quantities. A force is represented by an arrow (vector) that (1) has its base on the object being acted on (the point of application), (2) has a shaft and arrowhead in the direction of the force being exerted (direction/orientation), and (3) has a length drawn to represent the amount of force being exerted (magnitude). As we begin to examine force vectors (and at least throughout this chapter), the *point of application* (*base*) of each vector in each figure will be placed on the segment or object to which the force is applied—which is generally also the object under discussion.

Figure 1–13 shows John Alexander's leg-foot segment. The weight boot is shaded-in lightly for context but is not really part of the space diagram. Because the weight boot makes contact with the leg-foot segment, the weight boot must exert a force (in this case, a pull) on the segment. The force, called weightboot-on-legfoot (WbLf), is represented by a vector. The point of application is on the leg (closest to where the weight boot exerts its pull); the action line and direction indicate the direction of the pull and the angle of pull in relation to the leg; and the length is drawn to represent the magnitude of the pull. The force weightboot-on-legfoot is an external force because the weight boot is not part of the body, although it contacts the body. Figure 1–14 shows the force of a muscle (e.g., the brachialis) pulling on the forearm-hand segment. The point of application is at the attachment of the muscle, and the orientation and direction are toward the muscle (pulls are toward the source of the force). The force is called muscle-on-forearmhand (represented by the vector MFh). Although the designation of a force as "external" or "internal" may be useful in some contexts, the rules for drawing (or visualizing) forces are the same for external forces, such as the weight boot, and internal forces, such as the muscle.

The length of a vector is usually drawn proportional to the magnitude of the force according to a given scale. For example, if the scale is specified as 5 mm = 20 N of force, an arrow of 10 mm would represent 40 N of force. The length of a vector, however, does not necessarily need to be drawn to scale (unless a graphic solution is desired) as long as its magnitude is labeled (as is done in Fig. 1–13). Graphically, the action line of any vector can be considered infinitely long; that is, any vector can be extended in either direction (at the base or at the arrowhead) if this is useful in determining the

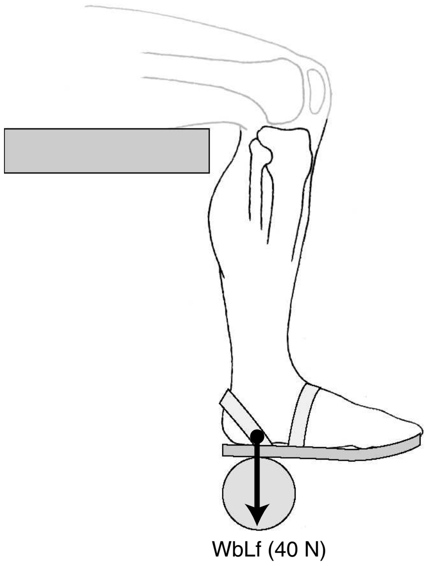

Figure 1–13 Vector representation of the pull of the weight boot on the leg-foot segment (weightboot-on-legfoot [WbLf]), with a magnitude proportional to the mass and equivalent to the weight of the apparatus.

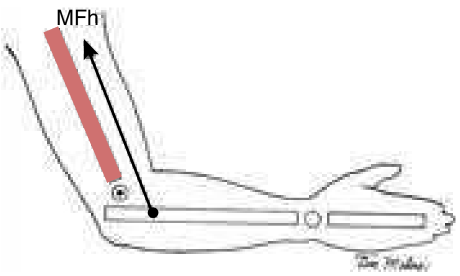

Figure 1–14 Vector MFh represents the pull of a muscle on the forearm-hand segment.

relationship of the vector to other vectors or objects. The length of a vector should not be arbitrarily drawn, however, if a scale has been specified.

Continuing Exploration 1-3:

Pounds and Newtons

Although SI units are commonly used mostly in scientific writing, the SI unit of force—the newton—does not have much of a context for those of us habituated to the US system. It is useful, therefore, to understand that 1 lb = 4.448 N. Vector WbLf in Figure 1–13 is labeled as 40 N. This converts to 8.99 lb. To get a gross idea of the pound equivalent of any figure given in newtons, you can divide the number of newtons by 5, understanding that you will be *underestimating* the actual number of pound equivalents.

Figure 1–15A shows John Alexander's leg-foot segment on the leg-press machine. The footplate is shaded in lightly for context but is not really part of the space diagram. Because the footplate is contacting the leg-foot segment, it must exert—in this case—a *push* on the segment. The force, footplate-on-legfoot (FpLf), is represented by a vector with a point of application on the leg-foot segment and pointing away from the source. The magnitude of this vector will remain unspecified until we have more information. However, the presence of the vector in the space diagram means that the force does, in fact, have some magnitude. Although the force is applied at the point where the footplate makes contact with the foot, the point of application can also be drawn anywhere *along the action of the vector* as long as the point of application (for purposes of visualization) remains on the object under consideration. Just as a vector can be extended to any length, the point of application can appear anywhere along the line of push or pull of the force (as long as it is on the same object) without changing the represented effect of the force (see Fig. 1–15B). In this text, the point of application will be placed as close to the actual point of contact as possible but may be shifted slightly along the action line for clarity when several forces are drawn together.

It is common to see in other physics and biomechanics texts a "push" force represented as shown in Figure 1–15C. However, this chapter will consistently use the convention that the *base* of the vector will be at the point of application, with the "push" directed away from that point of application (see Fig. 1–15A). This convention maintains the focus on the *point of application* on the segment and will enhance visualization later when we begin to resolve a vector into components. When the "push" of "footplate-on-legfoot" is drawn with its base (point of application) on the object (see Fig. 1–15A), the representation is similar in all respects (except name) to the force strap-on-legfoot (SLf), shown in Figure 1–16. This vector, however, is the *pull* of the strap connected to either side of the legfoot segment. It is reasonable for vector FpLf in Figure 1–15A and vector SLf in Figure 1–16 to look the same because the two forces "footplate-on-legfoot" and "strap-on-legfoot" will have an identical effect on the rigid leg-foot segment as long as the point of application, direction/orientation, and

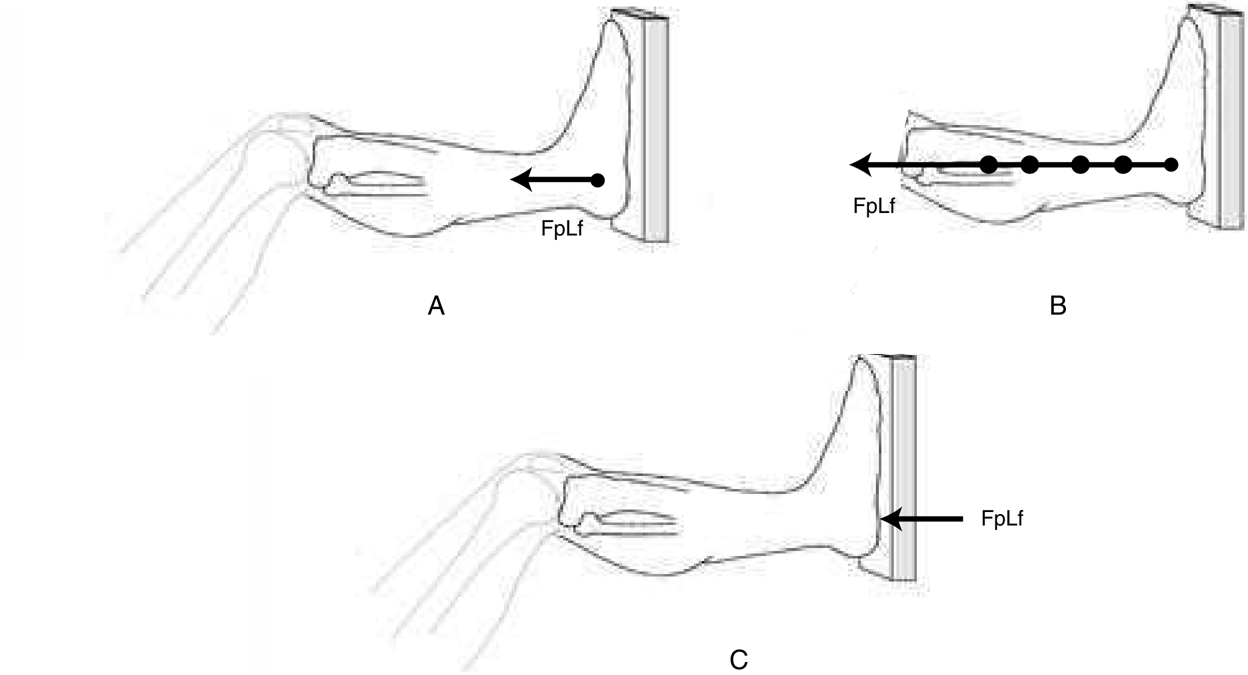

Figure 1–15 **A.** Vector representation of the force of the footplate of the leg-press machine on the leg-foot segment (footplate-on-legfoot [FpLf]). **B.** The vector footplate-on-legfoot (FpLf) may be drawn with any length and with a point of application anywhere along the line of pull of the vector as long as the point of application remains on the leg-foot segment. **C.** The push of the footplate on the leg-foot segment is commonly shown elsewhere by placing the arrowhead of vector FpLf at the point of application.

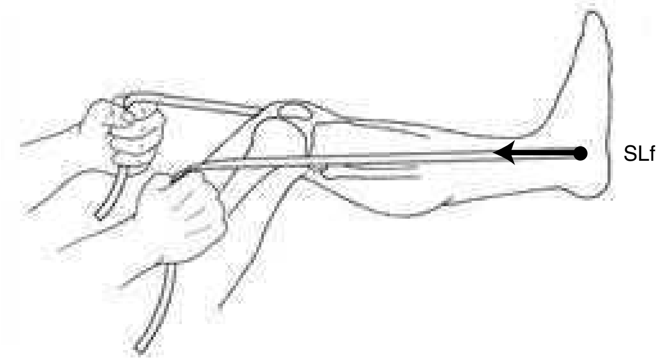

Figure 1–16 The vector representing the pull of a strap connected to each side of the leg-foot segment (strap-on-legfoot [SLf]) will look the same as the push of the footplate on the leg-foot segment (Fig. 1–15A) because both have identical effects on the leg-foot segment as long as the direction and magnitude are the same.

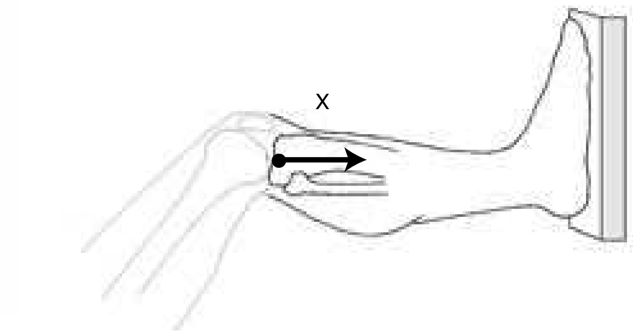

Figure 1–17 An unknown vector (X) can be named by identifying the segment to which it is applied and the source of the force (something that must be touching the segment).

magnitude are similar—as they are here. The magnitude and direction/orientation of a force are what affect the object to which the force is applied, without consideration of whether the force is, in fact, a push or a pull.

Concept Cornerstone **1-2**

Force Vectors Are Characterized By:

- a point of application *on the object acted upon.*
- an action line and direction/orientation indicating a *pull toward* the source object or a *push away from* the source object, at a given angle to the object acted upon.
- length that represents, and may be drawn proportional to, its magnitude (the quantity of push or pull).
- a length that may be extended to assess the relation between two or more vectors or to assess the relation of the vector to adjacent objects or points.

Concept Cornerstone **1-3**

Naming Forces

We have already begun to establish the naming convention of "*something-on-something*" to identify forces and label vectors. The first part of the force name will always identify the *source* of the force; the second part of the force name will always identify the object or segment that is *being acted on.*

Figure 1–17 shows John Alexander's leg-foot segment on the leg-press machine. A new vector is shown in this figure. Because vector X is applied to the leg-foot segment, the vector is named "*blank*-on-legfoot." The name of the vector is completed by identifying the source of the force. The leg-foot segment is being contacted by gravity, by the footplate, and by the femur. We can eliminate gravity as the source because gravity is always in a downward direction. The footplate can only push on the leg-foot segment, and so the vector is in the wrong direction for that to be the source. The femur will also push on the leg-foot segment because a bone cannot pull. Because vector X is directed away from the femur, the femur appears to be the source of vector X in Figure 1–17. Therefore, vector X is named femur-on-legfoot and can be labeled vector FLf.

Force of Gravity

As already noted, gravity is one of the most consistent and influential forces that the human body encounters in posture and movement. For that reason, it is useful to consider gravity first when examining the properties of forces. As a vector quantity, the force of gravity can be fully described by point of application, action line/direction/orientation, and magnitude. Unlike other forces that may act on a point or limited area of contact, gravity acts on each unit of mass that composes an object. For simplicity, however, the force of gravity acting on an object or segment is considered to have its point of application at the center of mass or **center of gravity (CoG)** of that object or segment—the hypothetical point at which all the mass of the object or segment appear to be concentrated. Every object or segment can be considered to have a single center of mass.

In a symmetrical object, the center of mass is located in the geometric center of the object (Fig. 1–18A). In an asymmetrical object, the center of mass will be located toward the heavier end because the mass must be evenly distributed around the center of mass (see Fig. 1–18B). The crutch in Figure 1–18C demonstrates that the center of mass is only a hypothetical point; it need not lie within the object being acted on. Even when the center of mass lies outside the object, it is still the point from which the force of gravity *appears* to act. The actual location of the center of mass of any object can be determined experimentally by a number of methods not within the scope of this text. However, the center of mass of an object can be approximated by thinking of it as the balance point of the object (assuming you could balance the object on one finger), as shown in Figure 1–18A–C.

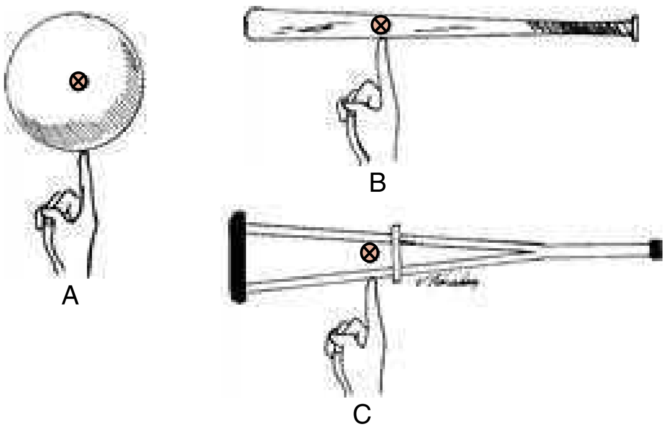

Figure 1–18 **A.** Center of mass of a symmetrical object. **B.** Center of mass of an asymmetrical object. **C.** The center of mass may lie outside the object.

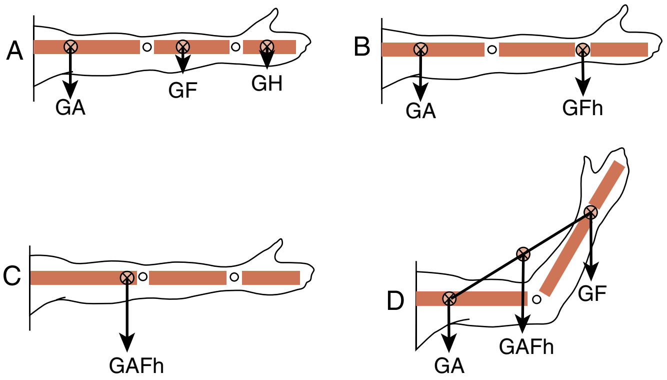

Figure 1–19 **A.** Gravity acting on the arm segment (GA), the forearm segment (GF), and the hand segment (GH). **B.** Gravity acting on the arm (GA) and forearm-hand segments (GFh). **C.** Gravity acting on the arm-forearm-hand segment (GAFh). **D.** The CoM of the arm-forearm-hand segment shifts when segments are rearranged.

Although the direction and orientation of most forces vary with the source of the force, the force of gravity acting on an object is *always* vertically downward toward the center of the earth. The gravitational vector is commonly referred to as the **line of gravity (LoG).** The length of the line of gravity can be drawn to scale (as in a free body diagram, in which the length is determined by its magnitude) or it may be extended (like any vector) when the relationship of the vector to other forces, points, or objects is being explored. The line of gravity can best be visualized as a string with a weight on the end (a plumb line), with the string attached to the center of mass of an object.

Segmental Centers of Mass and Composition of Gravitational Forces

Each segment in the body can be considered to have its own center of mass and line of gravity. Figure 1–19A shows the gravitational vectors acting at the mass centers of the arm, the forearm, and the hand segments (vectors GA, GF, and GH, respectively). The centers of mass in Figure 1–19A approximate those identified in studies done on cadavers and on in vivo body segments that have yielded standardized data on centers of mass and weights of individual and combined body segments.[1,7,8] It is often useful, however, to consider two or more segments as if they were a single segment or object and to treat them as if they are going to move together as a single rigid segment (such as the leg-foot segment in the patient case). When two gravity vectors acting on the same (now larger) rigid object are composed into one gravitational vector, the new common point of application (the new center of mass) is located between and in line with the original two segmental centers of mass. When the linked segments are not equal in mass, the new center of mass will lie closer to the heavier segment. The new vector will have the same effect on the combined forearm-hand segment as the original two vectors and is known as the resultant force. The process of combining two or more forces into a single resultant force is known as composition of forces.

Example 1-2

If we wish to treat two adjacent segments (e.g., the forearm and the hand segments) as if they were one rigid segment, the two gravitational vectors (GH and GF) acting on the new larger segment (forearm-hand) can be combined into a single gravitational vector (GFh) applied at the new center of mass (Fig. 1–19B). This figure shows vector GA on the arm and new vector GFh on the now-combined forearm-hand segment. Vector GFh is applied at the new center of mass for the combined forearm-hand segment (on a line between the original centers of mass), is directed vertically downward (as were both GF and GH), and has a magnitude equal to the sum of the magnitudes of GF and GH. Figure 1–19C shows the force of gravity (GAFh) acting on the rigid arm-forearm-hand segment. Vector GAFh is applied at the new center of mass located between and in line with the centers of mass of vectors GA and GFh; the magnitude of GAFh is equal to the sum of the magnitudes of GA and GFh; the direction of GAFh is vertically downward because it is still the pull of gravity and because that is the direction of the original vectors.

The center of mass for any one object or a rigid series of segments will remain unchanged regardless of the position of that object in space. However, when an object is composed of two or more linked and movable segments, the location of the center of mass of the combined unit will change if the segments are rearranged in relation to each other (Fig. 1–19D). The magnitude of the force of gravity will not change because the mass of the combined segments is unchanged, but the point of application of the resultant force will be different. A more precise method for mathematically composing two gravitational forces into a single resultant force will be addressed later when other attributes of the forces (the torque that each generates) are used to identify the exact position of the new center of mass between the original two.

Center of Mass of the Human Body

When all the segments of the body are combined and considered as a single rigid object in anatomic position, the center of mass of the body lies approximately anterior to the second sacral vertebra (S2). The precise location of the center of mass for a person in the anatomic position depends on the proportions (weight distribution) of that person. If a person really were a rigid object, the center of mass would not change its position in the body, regardless of whether the person was standing up, lying down, or leaning forward. Although the center of mass does not change its location in the rigid body as the body moves in space, the line of gravity changes its *relative* position or alignment within the body. In Figure 1–20, the line of gravity is between the person's feet **(base of support [BoS])** as the person stands in anatomic position; the line of gravity is parallel to the trunk and limbs. If the person is lying down (still in anatomic position), the line of gravity projecting from the center of mass of the body lies perpendicular to the trunk and limbs, rather than parallel as it does in the standing position. In reality, of course, a person is not rigid and does not remain in anatomic position. Rather, a person is constantly rearranging segments in relation to each other as the person moves. With each rearrangement of body segments, the location of the individual's center of mass will potentially change. The amount of change in the location of the center of mass depends on how disproportionately the segments are rearranged.

Example 1-3

If a person is considered to be composed of a rigid upper body (head-arms-trunk [HAT]) and a rigid lower limb segment, each segment will have its own center of mass. If the trunk is inclined forward, the segmental masses remain unchanged but the composite center of mass of the rearranged body segments shifts from its original location within the body at S2. The new center of mass is on a line between the original two centers of mass and is located toward the heavier upper body segment (Fig. 1–21). This new center of mass is physically located outside the body, with the line of gravity correspondingly shifted forward. Figure 1–22 shows a more disproportionate rearrangement of body segments. The centers of mass of the two lower limb segments (segment A and segment B) and the center of mass of the head-arms-trunk segment (segment C) are composed into a new center of mass located at point ABC, the point of application for the gravitational vector for the entire body (line of gravity = GABC).

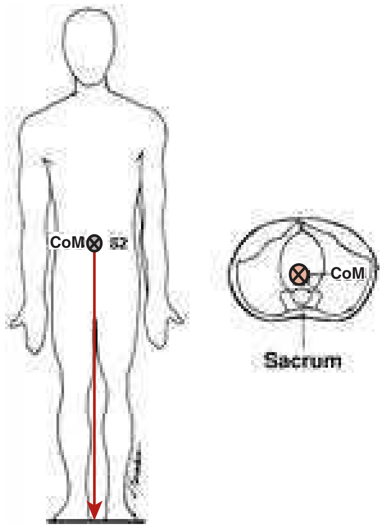

Figure 1–20 The CoM of the human body lies approximately at S2, anterior to the sacrum (inset). The extended LoG lies within the BoS.

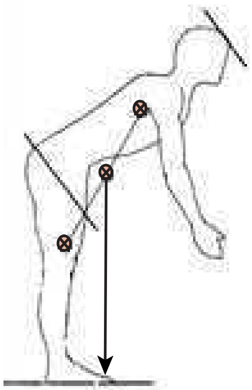

Figure 1–21 Rearrangement of the head, arms, and trunk (HAT) in relation to the lower extremities produces a new combined CoM and a new location for the LoG in relation to the base of support.

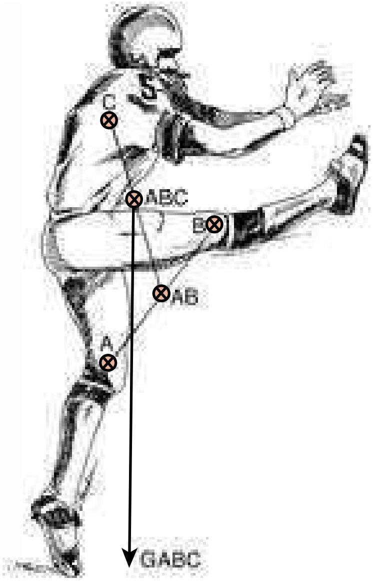

Figure 1–22 CoM of the football player's left leg (A) and the right leg (B) combine to form the CoM for the lower limbs (AB). The CoM (AB) combines with the upper trunk CoM (C) to produce the CoM for the entire body (ABC). The LoG from the combined CoM falls well outside the football player's BoS. He is unstable and cannot maintain this position.

Center of Mass, Line of Gravity, and Stability

In Figure 1–22, the line of gravity (GABC) falls outside the football player's left toes, which serve as his base of support. The line of gravity has been extended (lengthened) to indicate its relationship to the football player's base of support. It must be noted that the extended vector is no longer proportional to the magnitude of the force. However, the point of application, action line, and direction remain accurate. By extending the football player's line of gravity in Figure 1–22, we can see that the line of gravity is anterior to his base of support; it would be impossible for the player to hold this pose. For an object to be stable, *the line of gravity must fall within the base of support.* When the line of gravity is outside the base of support, the object will be unstable. As the football player moved from a starting position of standing on both feet with his arms at his sides to the position in Figure 1–22, two factors changed. He reduced his base of support from the area between and including his two feet to the much smaller area of the toes of one foot. His center of mass, with his rearrangement of segments, also has moved from S2 to above S2. Each of these two factors, combined with a slight forward lean, influenced the shift in his line of gravity and contributed to his instability.

When the base of support of an object is large, the line of gravity is less likely to be displaced outside the base of support, and the object, consequently, is more stable. When a person stands with his or her legs spread apart, the base is large side-to-side, and the trunk can move a good deal in that plane without displacing the line of gravity from the base of support and without the person falling over (Fig. 1–23). Whereas the center of mass remains in approximately the same place within the body as the trunk shifts to each side, the *line* of gravity moves within the wide base of support. Once again, it is useful here to think of the line of gravity as a plumb line. As long as the plumb line does not leave the base of support, the person should not fall over.

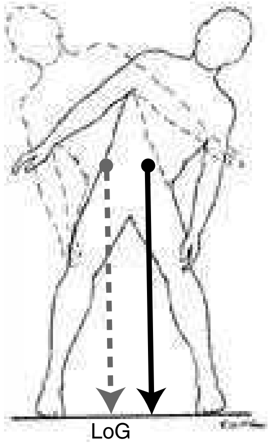

Figure 1–23 A wide base of support permits a wide excursion of the LoG without the LoG falling outside the base of support.

Alterations in Mass of an Object or Segment

The location of the center of mass of an object or the body depends on the distribution of mass of the object. The mass can be redistributed not only by rearranging linked segments in space but also by adding or taking away mass. People certainly gain weight and may gain it disproportionately in the body (thus shifting the center of mass). However, the most common way to redistribute mass in the body is to add external mass. Every time we add an object to the body by wearing it (a backpack), carrying it (a box), or holding it (a power drill), the new center of mass for the combined body and external mass will shift toward the additional weight; the shift will be proportional to the weight added.

Example 1-4

The man in Figure 1–24 has a cast applied to the right lower limb. Assuming the cast is now part of his mass, the new center of mass is located down and to the right of the original center of mass at S2. Because his center of mass with the cast is now lower, he is theoretically more stable. However, if he could not bear weight on his right leg, his base of support would consist of only the left foot. The patient would be stable only if he could lean to the left to swing his line of gravity over his left foot. However, he would still remain relatively unstable because of the very small base of support (it would take very little inadvertent leaning to displace the line of gravity outside the foot, causing the man to fall). To improve his stability, crutches have been added. The crutches and the left foot combine to form a much larger base of support, adding to the patient's stability and avoiding a large compensatory weight shift to the left.

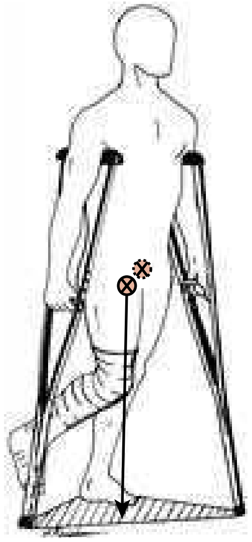

Figure 1–24 The addition of the weight of the cast has shifted the CoM. The addition of crutches enlarges the base of support to the shaded area between the weight-bearing foot and crutches to improve stability.

Concept Cornerstone **1-4**

Stability of an Object or the Human Body

- The larger the base of support of an object, the greater the stability of that object.
- The closer the object's center of mass is to the base of support, the more stable the object is.
- An object cannot be stable unless its line of gravity is located within its base of support.

INTRODUCTION TO STATICS AND DYNAMICS

The primary concern when looking at forces that act on the body or a particular segment is the effect that the forces will have on the body or segment. If all the forces acting on a segment are "balanced" (a state known as **equilibrium**), the segment will remain at rest or in uniform motion. If the forces are not "balanced," the segment will accelerate. **Statics** is the study of the conditions under which objects remain at rest. **Dynamics** is the study of the conditions under which objects move. Isaac Newton's first two laws govern whether an object is static or dynamic.

Newton's Law of Inertia

Newton's first law, the **law of inertia,** identifies the conditions under which an object will be in equilibrium. **Inertia** is the property of an object that resists both the initiation of linear motion and a change in linear motion and is directly proportional to its mass. Similarly, **moment of inertia** is the property of an object that resists rotary motion and changes in rotary motion. The law of inertia states that an object will remain at rest or in uniform (unchanging) motion unless acted on by an unbalanced (net or resultant) force or torque. An object that is acted upon by balanced forces or torques and remains motionless is in **static equilibrium.** However, an object acted upon by balanced forces or torques may also be in uniform motion, moving with a constant speed and direction. Velocity is a vector quantity that describes both speed and direction/orientation. An object in equilibrium can have a velocity of any magnitude, but its velocity remains constant. When the velocity of an object is constant but not zero, the object is in **dynamic equilibrium,** which can be linear (as for translatory motion), angular (as for rotary motion), or a combination of both (as for general motion). With regard to motion at joints of the body, dynamic equilibrium (constant velocity) of segments of the body occurs infrequently. Therefore, within the scope of this text, equilibrium will be simplified to mean an object at rest (in static equilibrium) unless otherwise specified.

Newton's law of inertia (or law of equilibrium) can be restated thus: For an object to be in equilibrium, the sum of all the forces and the sum of all the torques *applied to that object* must be zero.

$$\Sigma F = 0 \text{ and } \Sigma\tau = 0$$

The equilibrium of an object is determined *only by forces or torques applied to (with points of application on) that object.* There is no restriction on the number of forces or torques that can be applied to an object in equilibrium as long as there is more than one force or torque. If one (and only one) force or torque is applied to an object, the sum of the forces or torques cannot be zero. Any time the sum of the forces or torques acting on an object is not zero ($\Sigma F \neq 0$ or $\Sigma\tau \neq 0$), the object cannot be in equilibrium and must be accelerating.

Newton's Law of Acceleration

The magnitude of acceleration of a moving object is defined by Newton's second law, the **law of acceleration.** Newton's second law states that the linear acceleration (a) or angular acceleration (α) of an object is proportional to the net unbalanced forces (F_{unbal}) or torques (τ_{unbal}), respectively, acting on the object. Acceleration is inversely proportional to the mass (m) or moment of inertia (I) of that object:

$$a = \frac{F_{unbal}}{m}$$

$$\alpha = \frac{\tau_{unbal}}{I}$$

Side-bar: Moment of inertia (I) is less intuitive than simple mass. Moment of inertia is defined as $I = mr^2$, where m is the mass of an object and r is the distance that mass lies from the axis of rotation. Therefore, resistance to rotary motion is dependent upon the total mass of an object *and* the spatial distribution of that mass. In other words, a heavy object has more rotary inertia than a lighter object, and an object with mass further from the axis of rotation will have more rotary inertia than an object with mass focused closer to the axis of rotation.

Because an object acted upon by a net unbalanced force or torque *must* be accelerating, it is invariably in motion or in a dynamic state. The acceleration of an object will be in the direction of the net unbalanced force or torque. A net unbalanced force will produce translatory motion; a net unbalanced torque will produce rotary motion; a combination of unbalanced force and torque will produce general motion.

Concept Cornerstone **1-5**

Applying the Law of Acceleration (Inertia)

To put the law of acceleration into simple words: A large unbalanced push or pull (F_{unbal}) applied to an object of a given mass (m) will produce more linear acceleration (a) than a small unbalanced push or pull. Similarly, a given magnitude of unbalanced push or pull on an object of large mass or moment of inertia will produce less acceleration than that same push or pull on an object of smaller mass or moment of inertia. From the law of acceleration, it can be seen that inertia (a body's or object's resistance to change in velocity) is resistance to acceleration and is proportional to the mass of the body or object. The greater the mass or moment of inertia of an object, the greater the magnitude of net unbalanced force or torque needed either to get the object moving or to change its motion. A very large woman in a wheelchair has more inertia than does a small woman in a wheelchair; an aide must exert a greater push on a wheelchair with a large woman in it to get the chair in motion than on the wheelchair with a small woman in it.

TRANSLATORY MOTION IN LINEAR AND CONCURRENT FORCE SYSTEMS

The process of **composition of forces** is used to determine whether a net unbalanced force (or forces) exists on a segment, because this will determine whether the segment is at rest or in motion. Furthermore, the direction/orientation and location of the net unbalanced force or forces determine the type and direction of motion of the segment. The process of composition of forces was oversimplified in Examples 1-2 and 1-3 (see Figs. 1–19 and 1–21). The process of composition depends on the relationship of the forces to each other: that is, whether the forces are in a linear, concurrent, or parallel force system.

Let us return to our case example of John Alexander and the weight boot. In Figure 1–13, we identified the force of weightboot-on-legfoot (WbLf) on John's leg-foot segment. However, Figure 1–13 must be incomplete because this force cannot exist alone; otherwise, the leg-foot segment would accelerate downward. We also have not yet accounted for the force of gravity. Figure 1–25 is the same figure but with the addition of a new vector: gravity-on-legfoot (GLf). This vector is applied at the center of mass of the leg-foot segment, is directed vertically downward, and has a magnitude proportional to the mass of the segment. The leg-foot segment typically has approximately 6.5% of the mass of the body.[1] Because John weighs 734 N (165 lb), his leg-foot segment will weigh about 48 N (10.8 lb). Because these vectors are applied to the same segment, have action lines that lie in the same plane, and act in the same line (co-linear and coplanar), these two vectors are part of a **linear force system.**

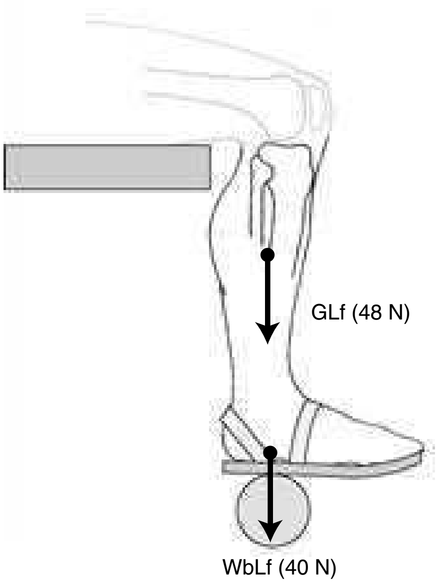

Figure 1–25 The forces of gravity-on-legfoot (GLf) and weightboot-on-legfoot (WbLf) are in the same linear force system when the leg-foot segment is at 90° of knee flexion.

Linear Force Systems

A linear force system exists whenever two or more forces act *on the same segment*, in the same plane, and in the same line (their action lines, if extended, overlap). Forces in a linear force system are assigned positive or negative signs. We will use the same convention previously described for translatory forces. Forces applied up (y-axis), forward or anterior (z-axis), or to the right (x-axis) will be assigned positive signs, whereas forces applied down, back or posterior, or to the left will be assigned negative signs. The magnitudes of vectors in opposite directions should always be assigned opposite signs.

Determining Resultant Forces in a Linear Force System

The net effect, or resultant, of all forces that are part of the same linear force system is determined by finding the arithmetic sum of the magnitudes of each of the forces in that force system (considering the positive or negative value of each). All forces in the *same* linear force system can be composed into a single resultant vector. The resultant vector has an action line in the same line as that of the original composing vectors, with a magnitude and direction equivalent to the arithmetic sum of the composing vectors. Because the vectors in a linear force system are all co-linear and coplanar, the point of application of the resultant vector will lie along the common action line of the composing vectors, and the resultant will have the same orientation in space as the composing vectors.

We previously assigned the weightboot-on legfoot vector a magnitude of 40 N (~8 lb). The weightboot-on-legfoot and gravity-on-legfoot vectors are in the same linear force system. The resultant of the two forces, therefore, can be found by adding their magnitudes. Because both vectors are directed down, they are assigned negative values of –40 N and –48 N, respectively. The sum of these forces is –88 N. The two forces can be represented graphically as a single resultant vector of –88 N. If John is not trying to lift the weight boot yet, there should be no motion of the leg-foot segment. If there is no motion (static equilibrium), the sum of the forces acting on the leg-foot segment must total zero. Instead, there is (in Fig. 1–25) a net unbalance force of –88 N; the leg-foot segment appears to be accelerating downward. In order to "balance" the net downward force to achieve equilibrium, we must identify something touching the leg-foot segment that will be part of the same linear force system and exert an upward force on the leg-foot segment.

Figure 1–25 indicates that the femur is potentially touching the leg-foot segment. However, the contact of the femur would be a *push* on the leg-foot segment and, in the position shown in Figure 1–25, would be directed away from the femur in the *same* direction as the two force vectors that are already shown. Also, the downward forces already on the leg-foot segment would tend to move the leg-foot segment *away* from the femur, minimizing or eliminating the contact of the femur with the leg-foot segment. A net force that moves a bony segment away from its adjacent bony segment is known as a **distraction force.** A distraction force tends to cause a separation between the bones that make up a joint. Consequently, we still need to account for a force of 88 N acting upward on the leg-foot segment to have equilibrium.

In the human body, the two bones of a synovial joint (e.g., the knee joint) are connected by a joint capsule and ligaments made of connective tissue. Until we explore connective tissue behavior in detail in Chapter 2, capsuloligamentous structures are best visualized as strings or cords with some elasticity that can "pull" (not "push") on the bones to which they attach. Figure 1–26A shows a schematic representation of the capsuloligamentous structures that join the femur and the tibia.

Side-bar: In reality, the capsule *surrounds* the adjacent bones, and the ligamentous connections are more complex.

We will nickname the structures "Acapsule" (anterior capsule) and "Pcapsule" (posterior capsule), understanding that these two forces are representing the pull of both the capsule and the capsular ligaments at the knee. Because capsules and ligaments can only pull, the forces that are created by the contact of Acapsule and Pcapsule in Figure 1–26A through C are directed upward toward the capsuloligamentous structures (positive). Under the assumption that the pulls of the capsule anteriorly and posteriorly in this example are likely to be symmetrical, the vectors are given the same length in Figure 1–26A.

The vectors for Acapsule-on-legfoot (AcLf) and Pcapsule-on-legfoot (PcLf) are drawn in Figure 1-26A so that the points of application are at the points on the leg-foot segment where the fibers of the capsular segments converge (or in the center of the area where the fibers converge).

Side-bar: Although the anterior and posterior segments of the capsule also touch the femur, we are considering only the leg-foot segment at this time.

The vector arrows for the pulls of Acapsule-on-legfoot and Pcapsule-on-legfoot must follow the fibers of the capsule *at the point of application* and *continue in a straight line.* A vector, for any given snapshot of time, is always a straight line. The vector for the pull of the capsule does not change direction even if the fibers of the capsule change direction after the fibers emerge from their attachment to the bone.

In a linear force system, vectors must be co-linear and coplanar. These vectors (AcLf and PcLf) are not co-linear or coplanar with the vectors weightboot-on-legfoot and gravity-on-legfoot vectors. Therefore, these vectors cannot all be part of the same linear force system. If vectors Acapsule-on-legfoot and Pcapsule-on-legfoot are extended slightly at their bases, the two vectors will converge (see Fig. 1–26B). When two or more vectors *applied to the same object* are not co-linear but converge (intersect), the vectors are part of a **concurrent force system.**

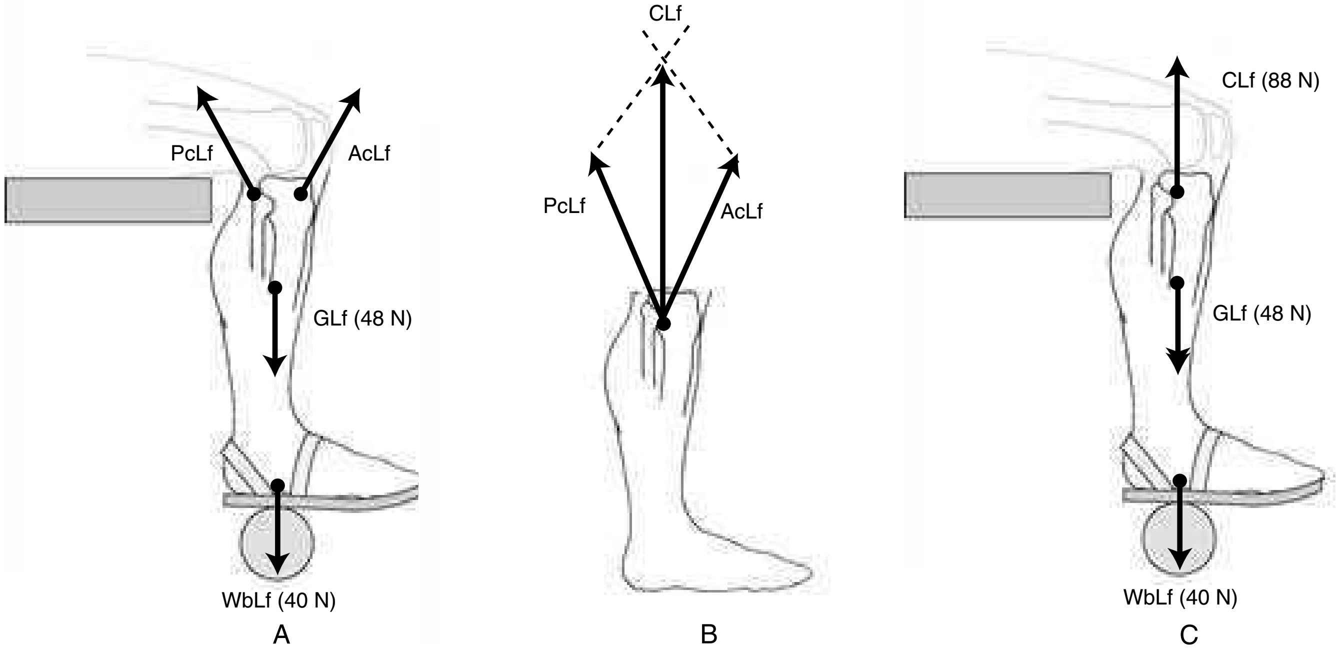

Figure 1–26 **A.** Schematic representation of the pull of the anterior capsule (AcLf) and posterior capsule (PcLf) on the leg-foot segment. **B.** Determination of the direction and relative magnitude of the resultant (capsule-on-legfoot [CLf]) of concurrent forces AcLf and PcLf, through the process of composition by parallelogram. **C.** The resultant force CLf has been added to the leg-foot segment, with a magnitude equivalent to that of GLf + WbLf.

Concurrent Force Systems

It is quite common (and perhaps most common in the human body) for forces applied to an object to have action lines that lie at angles to each other. A common point of application may mean that the forces are literally applied to the same point on the object or that forces applied to the same object have vectors that intersect when extended in length (even if the intersection is outside the actual segment or object, as we saw with the center of mass). The net effect, or resultant, of concurrent forces *appears* to occur at the common point of application (or point of intersection). Any two forces in a concurrent force system can be composed into a single resultant force through a graphic process known as **composition by parallelogram.**

Determining Resultant Forces in a Concurrent Force System

In composition by parallelogram, two vectors are taken at a time. The two vectors and their common point of application or point of intersection form two sides of a parallelogram. The parallelogram is completed by drawing two additional lines at the arrowheads of the original two vectors (with each new line parallel to one of the original two). The resultant has the same point of application as the original vectors and is the diagonal of the parallelogram. If there are more than two vectors in a concurrent force system, a third vector is added to the resultant of the original two through the same process. The sequential use of the resultant and one of the original vectors continues until all the vectors in the original concurrent force system are accounted for.

Example 1-5

In Figure 1–26B, vectors AcLf and PcLf are composed into a single resultant vector (CLf). Vectors AcLf and PcLf are extended to identify the point of application of the new resultant vector that represents the combined action of AcLf and PcLf. A parallelogram is constructed by starting at the arrowhead of one vector (AcLf) and drawing a line of relatively arbitrary length that is *parallel* to the adjacent vector (PcLf). The process is repeated by starting at the arrowhead of PcLf and drawing a line of relatively arbitrary length *parallel* to AcLf. Both the lengths of the two new lines should be long enough that the two new lines intersect. Because the two new lines are drawn parallel to the original two and intersect (thus closing the figure), a parallelogram is created (see Fig. 1–26B). The resultant of AcLf and PcLf is a new vector ("capsule-on-legfoot" [CLf]) that has a shared point of application with the original two vectors and has a magnitude that is equal to the length of the diagonal of the parallelogram. If the vectors were drawn to scale, the length of CLf would represent +88 N.

Capsule-on-legfoot in Figure 1–26C is the resultant of Pcapsule-on-legfoot and Acapsule-on-legfoot in Figure 1–26B. Assuming nothing else is touching the leg-foot segment, capsule-on-legfoot must be equal in magnitude and opposite in direction to the sum of gravity-on-legfoot and weightboot-on-legfoot because these three vectors are colinear, coplanar, and applied to the same object. The arithmetic sum of the three forces must be zero because (1) these vectors are part of the same linear force system, (2) nothing else is touching the leg-foot segment, and (3) the leg-foot segment is not moving.

The magnitude of the resultant of two concurrent forces has a fixed proportional relationship to the original two vectors. The relationship between the two composing vectors and the resultant is dependent on both the magnitudes of the composing vectors *and* the angle between (orientation of) the composing vectors. In composition of forces by parallelogram, the *relative* lengths (the scale) of the concurrent forces being composed must be appropriately represented to obtain the correct relative magnitude of the resultant force. It is always true that the magnitude of the resultant will be less than the sum of the magnitudes of the composing forces.

Trigonometric functions can also be used to determine the magnitude of the resultant of two concurrent forces. The trigonometric solution is presented below. A trigonometric solution requires knowledge of both the absolute magnitudes of the two composing vectors and the angle between them. However, these values are rarely known in a clinical situation.

Continuing Exploration 1-4:

Trigonometric Solution

Let us assume that PcLf and AcLf each have a magnitude of 51 N and that the vectors are at a 60° angle (α) to each other (Fig. 1–27). As was done for the graphic solution, the parallelogram is completed by drawing AcLf′ and PcLf′ parallel to and the same lengths as AcLf and PcLf, respectively. The law of cosines can be used to find the length of the side opposite a known angle (when the triangle is not a right triangle).

The reference triangle (shaded) is that formed by PcLf, AcLf′, and CLf (see Fig. 1–27). To apply the law of cosines, angle β must be known because vector CLf (whose length we are solving for) is the "side opposite" that angle. The known angle (α) in Figure 1-27 is 60°. If PcLf is extended (as shown by the dotted line in Fig. 1-27), angle α is replicated because it is the angle between PcLf and AcLf′ (given AcLf′ is parallel to AcLf). Angle β, then, is the complement of angle α, or:

$$\beta = 180^\circ - 60^\circ = 120^\circ$$

By substituting the variables given in the example, the magnitude of the resultant, CLf, can be solved for using the following equation:

$$\text{CLf} = \sqrt{\text{PcLf}^2 + \text{AcLf}^2 - 2(\text{PcLf})(\text{AcLf})(\cos\beta)}$$

If the value of 51 N is entered into the equation for both PcLf and AcLf and an angle of 120° is used, vector CLf = 88 N. As we shall see, the trigonometric solution is simpler when the triangle has one 90° angle (right triangle).

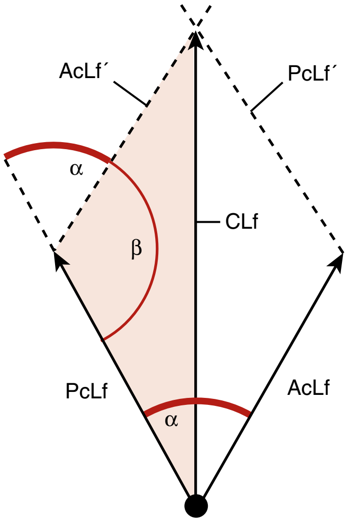

Figure 1–27 The cosine law for triangles is used to compute the magnitude of CLf, given the magnitudes of AcLf and PcLf, as well as the angle of application (α) between them. The relevant angle (β) is the complement of angle α (180° – α).

When there are more than two forces in the concurrent force system, the process is the same whether a graphic or trigonometric solution is used. The first two vectors are composed into a resultant vector, the resultant and a third vector are then composed to create a second resultant vector, and so on until all vectors are accounted for. Regardless of the order in which the vectors are taken, the solution will be the same.

Returning to John Alexander's weight boot, we have established that vectors gravity-on-legfoot and weightboot-on-legfoot have a net force of –88 N and that capsule-on-legfoot has a magnitude of +88 N. In John's case, we must also consider not only the pull of the capsule *on his leg-foot segment* but also the pull of his leg-foot segment *on his capsule*, because John has injured his medial collateral ligament (part of that capsule). We can segue to consideration of this new "object" (the capsule) by examining the principle in **Newton's law of reaction.**

Newton's Law of Reaction

Newton's third law (the law of reaction) is commonly stated as follows: For every action, there is an equal and opposite reaction. In other words, when an object applies a force to a second object, the second object *must* simultaneously apply a force equal in magnitude and opposite in direction to the first object. These two forces that are applied to the two contacting objects are an **interaction pair** and can also be called **action-reaction** (or simply **reaction**) **forces.**

Continuing Exploration 1-5:

Reactions to Leg-Foot Segment Forces

Among the vectors in Figure 1–26C we see the force vector of weightboot-on-legfoot (WbLf). WbLf arises from the contact of the weight boot with the leg-foot segment. If the weight boot contacts the leg-foot segment, then the leg-foot segment must also contact the weight boot. Legfoot-on-weightboot (LfWb) is a reaction force that is equal in magnitude and opposite in direction to WbLf (Fig. 1–28). We did not examine LfWb initially because it is not part of the space diagram under consideration. It is presented here simply as an example of a reaction force.

Side-bar: In Figure 1–28, the points of application and action lines of the reaction forces are shifted slightly so that the two vectors can be seen as distinctly different and as applied to different but touching objects.

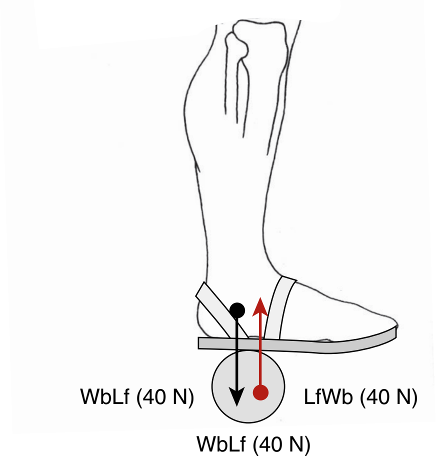

Figure 1–28 Weightboot-on-legfoot (WbLf) and legfoot-on-weightboot (LfWb) are reaction forces or an interaction pair. Both forces exist by virtue of the contact between the two objects. Although separated for clarity, these two vectors will be in line with each other.

Reaction forces are *always* in the same line and applied to the *different* but contacting objects. The directions of reaction forces are always *opposite* to each other because the two touching objects either pull on each other or push on each other. Because the points of application of reaction forces are never on the same object, reaction forces are never part of the same force system and typically are not part of the same space diagram. However, we will see that reaction forces can be an important consideration in human function, because no segment ever exists in isolation (as in a space diagram).

Gravitational and Contact Forces

A different scenario can be used to demonstrate the sometimes subtle but potentially important distinction between a force applied to an object and its reaction. We generally assume when we get on a scale that the scale shows our weight (Fig. 1–29). A person's weight (gravity-on-person [GP]), however, is not applied to the scale and thus cannot act on the scale. What is actually being recorded on the scale is the contact (push) of the "person-on-scale" (PS) and not "gravity-on-person." The distinction between these forces and the relation between these

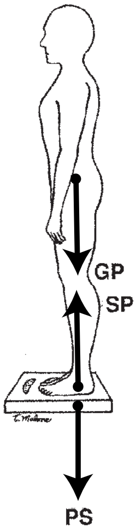

Figure 1–29 Although a scale is commonly thought to measure the weight of the person (gravity-on-person [GP]), it actually measures the contact of the person-on-scale (PS). Vectors GP and PS are equal in magnitude as long as nothing else is touching the person.

two forces can be established by using both Newton's first and third laws.

The person standing on the scale must be in equilibrium ($\Sigma F = 0$). If the gravity-on-person vector is acting down with a magnitude of –734 N (John Alexander's weight), there must also be a force of equal magnitude acting up on the person for the person to remain motionless. The only other object, besides gravity, that appears to be contacting the person in Figure 1–29 is the scale. The scale, therefore, must be exerting an upward push on the person (scale-on-person [SP]) with magnitude equal to that of gravity-on-person (+734 N). The force of scale-on-person, of course, has a reaction force of person-on-scale that is equal in magnitude (734 N) and opposite in direction (down) but applied to the scale. Consequently, in this instance, the magnitude of the person's weight and the magnitude of the person's contact with the scale are equivalent *although applied to different objects.* The vectors person-on-scale and scale-on-person occur as a result of a push by the contacting objects. When reaction forces arise from the *push* of one object on another, they are often referred to as **contact forces (F_C).** When contact forces are perpendicular to the surfaces that produce them, the term **normal force (F_N)** is also used.[5,9] Contact forces, therefore, are a subset of reaction forces.

The distinction between gravity-on-person and the reaction force person-on-scale is not always made, but it can be very important if something else is touching the person or the scale. If the person is holding something while on the scale, the person's weight does not change, but the contact forces between the person and the scale will increase. Similarly, a gentle pressure down on the bathroom countertop as a person stands on the scale will result in an apparent weight reduction. Situations are frequently encountered in which the contact of an object with a supporting surface and its weight are used interchangeably. Care should be taken to assess the situation to determine whether the magnitudes are, in fact, equivalent. The recognition of weight and contact as separate forces permits more flexibility in understanding how to modify these forces if necessary.

Concept Cornerstone 1-6

Action-Reaction Forces

- Whenever two objects or segments touch, the two objects or segments exert a force on each other. Consequently, every force has a reaction or is part of an action-reaction pair.
- The term *contact force* or *contact forces* is commonly used to indicate one or both of a set of reaction forces in which the "touch" is a push rather than a pull.
- Reaction forces are *never part of the same force system* and cannot be composed (cannot either be additive or offset each other) because the two forces are, by definition, applied to different objects.
- The static or dynamic state (equilibrium or motion) of an object cannot be affected by another object that is not touching it or by a force that is not applied to it.
- The reaction to a force should be acknowledged but may be ignored graphically and conceptually *if* the object to which it is applied and the other forces on that object are not of interest.

ADDITIONAL LINEAR FORCE CONSIDERATIONS

The equilibrium established in John Alexander's leg-foot segment as he sits with the dangling weight boot is dependent on the capsule (and ligaments) to pull upward on the leg-foot segment with the same magnitude as that with which gravity and the weight boot pull downward (see Fig. 1–26C). Because the capsuloligamentous structures are injured in John's case, we need to explore the forces *applied to the capsule.* If the capsule pulls on the leg-foot segment with a magnitude of 88 N, the law of reaction stipulates that the leg-foot segment must also be pulling on the capsule with an equivalent force. If the compromised capsule cannot withstand an 88-N pull, then it may not be able to pull on the leg-foot segment with an 88-N force. This relationship requires an understanding of tensile forces and the forces that produce them.

Tensile Forces

Tension in the joint capsule, just like tension in any passive structure (including relatively solid materials such as bone), is created by opposite pulls on the same object. If there are not two opposite pulls on the object (each of which is a tensile force), there cannot be tension in the object. Remembering that the connective tissue capsule and ligaments are best analogized to slightly elasticized cord, we first examine tension in a cord or rope.

If a man pulls on a rope that is not attached to anything, no tension will develop in the rope no matter how hard or lightly he pulls because there is no counterforce. The rope will simply accelerate in the direction of the man's pull (with a magnitude equivalent to the force of pull [F_{unbal}] divided by the mass [m] of the rope). If the rope is tied to an immovable block of cement, there will be two forces applied to the rope. The two forces are created by the only two things contacting the rope: the man's hands and the block (Fig. 1–30). If the hands-on-rope vector (HR) has a magnitude of +110 N (~25 lb), then the block-on-rope vector (BR) must have a magnitude of –110 N because the rope is in equilibrium. If it is assumed that rope has a homogenous composition (unlike most biological tissues), the tension will be the same throughout the rope (as long as there is no friction on the rope), and the tension in the rope will be equivalent to the magnitude of the two tensile forces acting on the rope.[5] In Figure 1–30, both hands-on-rope (+110 N) and block-on-rope (–110 N) can be designated as tensile forces.

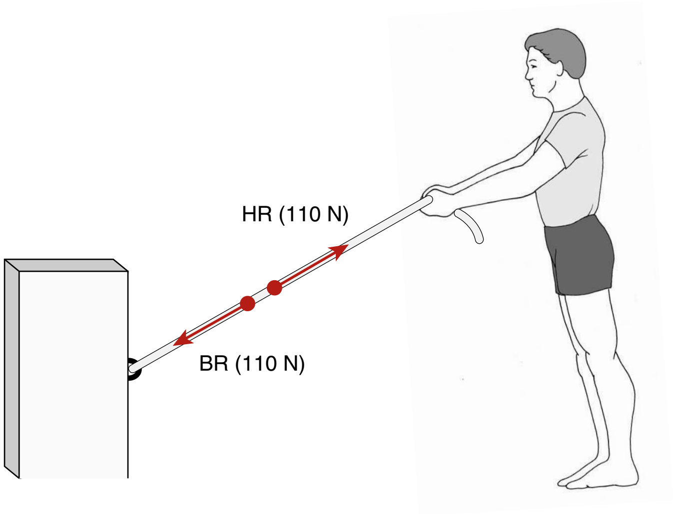

Figure 1–30 The tensile forces of the pull of hand-on-rope (HR) and the pull of the cement block on the rope (BR) produce two forces of equal magnitude (110 N) that result in 110 N of tension within and throughout the rope.

Assume for the moment that the rope is slack before the man begins to pull on the rope. As the man initiates his pull, his hands will accelerate away from the block because the force pulling his hands toward his body (muscles-on-hands [MsH]) will be greater than the pull of the rope on the hands (rope-on-hands [RH]). As the man's hands get farther from the block, the rope will get tighter, and the force of the rope-on-hands will increase. The acceleration of the hands will gradually slow down as the resultant of these two forces approaches equilibrium; when the two forces are equal, there will be zero acceleration. Because rope-on-hands and hands-on-rope are reaction forces (and always equal in magnitude), the tension in the rope (hands-on-rope) will eventually be equivalent to the magnitude of the man's pull (muscles-on-hands) (Fig. 1–31).

Tensile Forces and Their Reaction Forces

The example of the man pulling on the rope and cement block assumed that the rope could withstand whatever tension was required of it. If the rope is damaged, it may be able to withstand *no more than* the required 110 N of tension. If, however, the man in our example pulls on the rope with a magnitude of 200 N (45 lb), the rope will break. Once the rope breaks, there is no longer tension in the rope. The man pulling on the rope with a magnitude of 200 N will have a net unbalanced force that will accelerate his hands (or the man) backward (Fig. 1–32) until his muscles stop pulling (which will, it is hoped, happen before he punches himself in the stomach or falls over!).

Let us go back to John Alexander to determine how the tension example is applied to John's use of the weight boot. The equilibrium of John's leg-foot segment was based on the ability of the capsule to pull on the leg-foot segment with a magnitude equivalent to gravity-on-leg-foot plus weightboot-on-legfoot. If the capsule pulls on the leg-foot segment with a magnitude of +88 N (as we established earlier), the leg-foot segment must pull on the capsule (legfoot-on-capsule [LfC]) with an equivalent

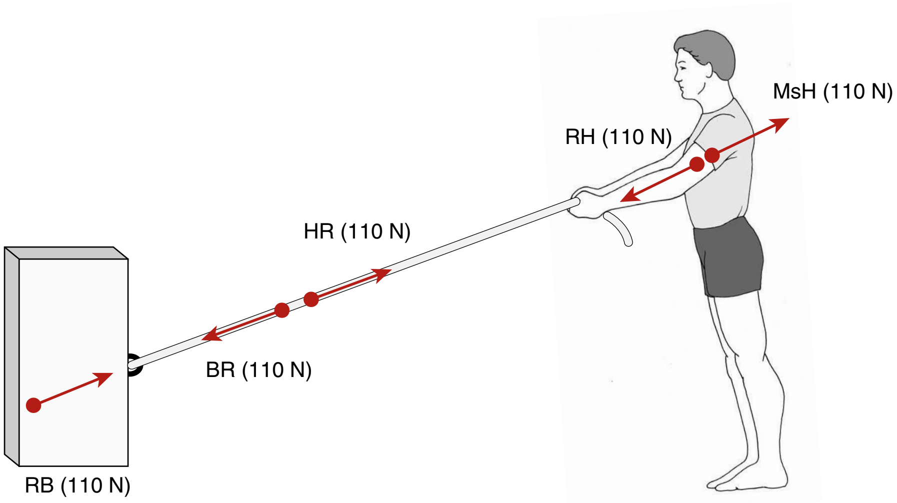

Figure 1–31 Equilibrium of the man will be achieved when the force of the rope-on-hand (RH) reaches the magnitude of muscles-on-hand (MsH). Rope-on-hand will not reach the 110 N magnitude needed to establish equilibrium until the tension in the initially slack rope reaches that magnitude as the man accelerates away from the block.

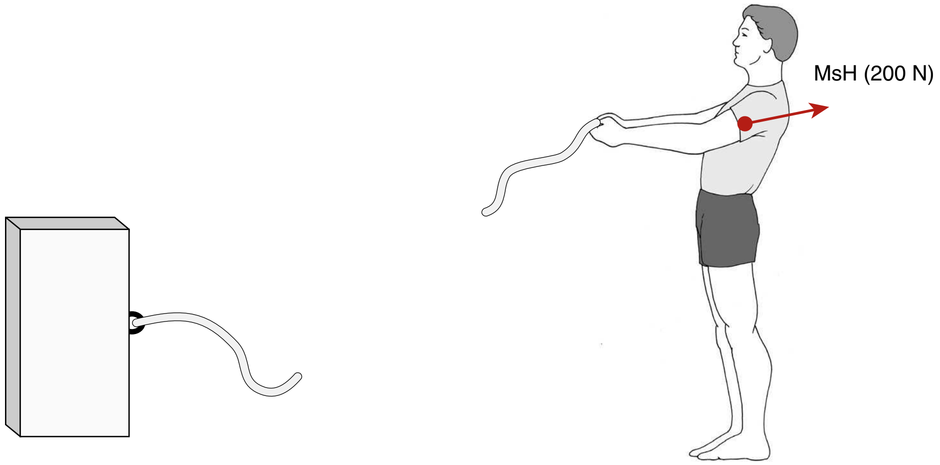

Figure 1–32 If the rope cannot withstand the tensile forces placed on it, it will break. Once the rope breaks, the force of muscles-on-hand (MsH) is unopposed, and the man will accelerate backward.

force of –88 N (Fig. 1–33). Two questions can be raised around the assumption that there is 88 N of tension in the capsule: (1) Does the magnitude of tension reach 88 N in the capsule immediately, and (2) can the injured capsule (and ligaments) withstand 88 N of tension? Case Application 1-1 applies the concepts from the example of tension in the rope to John's joint capsule.

CASE APPLICATION

Tension in the Knee Joint Capsule

case 1–1

The reaction forces of capsule-on-legfoot (CLf) and legfoot-on-capsule (LfC) have a magnitude of 88 N (see Fig. 1–33).

Side-bar: Vectors LfC and CLf should be co-linear in the figure but are separated for clarity.

Legfoot-on-capsule is a tensile vector. Tension can occur in a passive structure only if there are two pulls on the object. Therefore, there must be a second tensile vector (of +88 N) applied to the capsule from something touching the capsule at the other end. The second tensile vector, therefore, must be femur-on-capsule (FC) (Fig. 1–34), where the tensile vectors are co-linear. The magnitudes of capsule-on-legfoot, legfoot-on-capsule, and femur-on-capsule are equivalent because capsule-on-legfoot is part of the same linear force system with weightboot-on-legfoot and gravity-on-legfoot (see Fig. 1–26C) and because vectors legfoot-on-capsule and femur-on-capsule are part of the same linear force system. The sum of the forces in both linear force systems is zero because it is assumed that no movement is occurring.

Concept Cornerstone **1-7**

Tension and Tensile Forces

- Tensile forces (or the resultants of tensile forces) on an object are always equal in magnitude, opposite in direction, and applied parallel to the long axis of the object.
- Tensile forces are co-linear, coplanar, and applied to the same object; therefore, tensile vectors are part of the same linear force system.
- Tensile forces applied to a flexible or rigid structure of homogenous composition create the same tension at all points along the long axis of the structure in the absence of friction; that is, tensile forces are transmitted along the length (long axis) of the object.

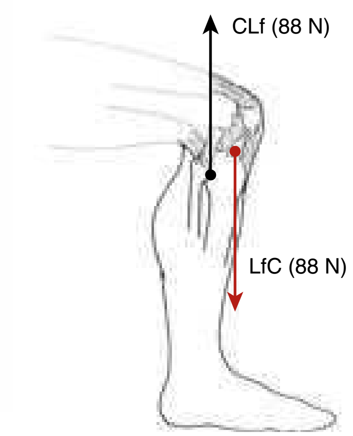

Figure 1–33 The pull of the capsule-on-legfoot (CLf) must have a concomitant reaction force of legfoot-on-capsule (LfC) that is an 88-N tensile force on the joint capsule.

Joint Distraction

Joint capsule and ligaments are not necessarily in a constant state of tension. In fact, if John started out with his leg-foot segment on the treatment table, there would effectively be no tension in his capsule or ligaments because the sum of the forces on the leg-foot segment from the "contacts" of gravity (gravity-on-legfoot) and the treatment table (table-on-legfoot) (Fig. 1–35) would be sufficient for equilibrium ($\Sigma F = 0$). Although both the capsule and the weight boot are still attached to the leg-foot segment, the magnitudes of pull would be negligible (too small to include in the space diagram).

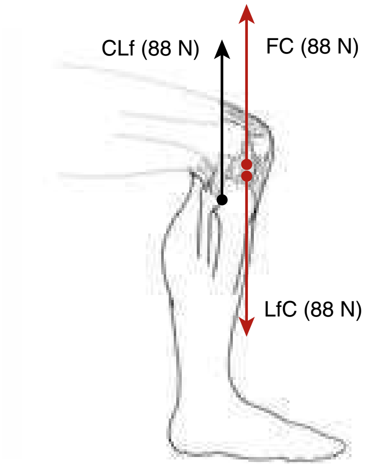

Figure 1–34 The tensile forces of legfoot-on-capsule (LfC) and femur-on-capsule (FC) are shown acting on the capsule.

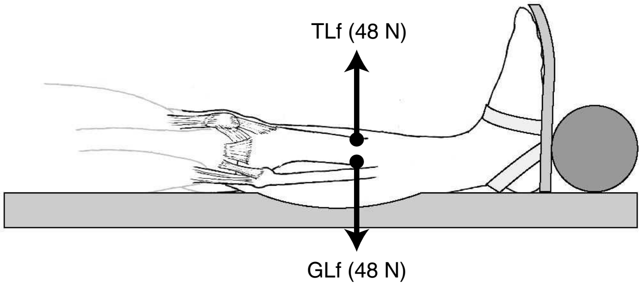

Figure 1–35 The forces of table-on-legfoot (TLf) and gravity-on-legfoot (GLf) in this position are sufficient for equilibrium of the leg-foot segment, with zero (or negligible) tension in the knee joint capsule.

A situation similar to the leg-foot segment on the treatment table would exist if John's foot were supported by someone's hand while his leg-foot segment is in the vertical position. In Figure 1-36, the hand is pushing up (+88 N) on the leg-foot segment (hand-on-legfoot segment [HLf]) with a magnitude equivalent to the pull of gravity and the weight boot (–88 N).

Side-bar: The hand-on-legfoot vector is shown to one side of gravity-on-legfoot and weightboot-on-legfoot for clarity, but assume that the supporting hand is directly below the weight boot.

The magnitude of the pull of the capsule (and ligaments) on the leg-foot segment would be negligible as long as the hand-on-legfoot had a magnitude equal and opposite to that of gravity-on-legfoot and weightboot-on-legfoot. As the upward support of the hand is taken away, however, there would be a net unbalanced force down on the leg-foot segment that would cause the leg-foot segment to accelerate away from the femur. The pull or movement of one bony segment away from another is known as **joint distraction.**[10]

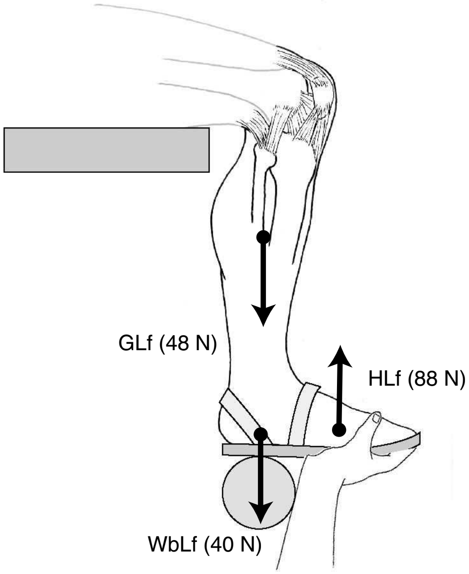

Figure 1–36 As long as the 88-N force on the leg-foot segment from gravity (GLf) and the weight boot (WbLf) are supported by an equal upward force from the hand (HLf), the tension in the capsule and ligaments will be zero (or negligible).

As the upward push of the hand decreases and the leg-foot segment moves away from the femur, the capsule will become increasingly tensed. The magnitude of acceleration of the leg-foot segment will be directly proportional to the unbalanced force and indirectly proportional to the combined mass of the leg-foot segment and weight boot. However, the unbalanced force is difficult to quantify because it is constantly changing. Although the increase in capsular tension occurs concomitantly with the reduction in hand support, the two forces are not equivalent in magnitude because the leg-foot segment must move away from the femur for the capsule to get tighter; that is, there must be a net unbalanced force on the leg-foot segment to create the movement that causes the capsule to get tighter.

The Continuing Exploration box titled "Reactions to Leg-Foot Segment Forces" presented the calculation of acceleration of the leg-foot segment at one point in time (a static rather than dynamic analysis). However, the concepts are more important than the calculations because the magnitudes of the weight of a limb, the support of the hand, and the tension in the capsule are generally unknown in a true clinical situation.

Continuing Exploration 1-6:

Acceleration in Joint Distraction

The leg-foot segment and weight boot together weigh 88 N. To calculate acceleration ($a = F_{unbal} \div m$), however, the mass (not just the weight) of the leg-foot segment and weight boot must be known. Recalling that 1 N is the amount of force needed to accelerate 1 kg at 1 m/sec^2, weight (in newtons or equivalently in kg-m/sec^2) is mass (in kilograms) multiplied by the acceleration of gravity, or:

$$W = (m)(9.8 \text{ m/sec}^2)$$

Solving for mass, a weight of 88 N is equivalent to 88 kg-m/sec^2 ÷ 9.8 m/sec^2 = 8.97 kg. Consequently, the leg-foot segment and weight boot together have a mass of approximately 9 kg. Assigning some arbitrary values, assume that a downward force of –88 N is offset in this static example by an upward push of the supporting hand of +50 N and capsular tensile force of +10 N. The net unbalanced force on the leg-foot segment (–88 + 50 + 10) is –28 N. Therefore:

$$a = \frac{-28 \text{ kg-m/sec}^2}{9 \text{ kg}}$$

$$a = -3.11 \text{ m/sec}^2$$

When the hand in Figure 1–36 is no longer in contact with the leg-foot segment and the tension in the capsule reaches 88 N, the leg-foot segment will stop accelerating away from the femur and will reach equilibrium.

Distraction Forces

The resultant pull of gravity and the weight boot on the leg-foot segment (composed into a single vector) can be referred to as a **distraction force**[3] or **joint distraction force.** A distraction force is directed away from the joint surface to which it is applied, is perpendicular to its joint surface, and leads to the separation of the joint surfaces.

Side-bar: It is important to note that here the term *distraction* refers to separation of rigid non-deformable bones. Distraction across or within a deformable body is more complex and will be considered in Chapter 2.

A joint distraction force cannot exist in isolation; joint surfaces will not separate unless there is a distraction force applied to the adjacent segment in the opposite direction. As the leg-foot segment is pulled away from the femur, any tension in the capsule created by the pull of the leg-foot segment on the capsule results in a second tensile vector in the capsule (femur-on-capsule). If the femur pulls on the capsule (see Fig. 1–34), then the capsule must concomitantly pull on the femur. If there is no opposing force on the femur, the net unbalanced downward force on the leg-foot segment will be transmitted through the capsule to the femur; the femur will also accelerate downward as soon as any appreciable tension is developed in the capsule. If the femur accelerates downward with the same magnitude of acceleration as the leg-foot segment, the joint surfaces will not separate any farther than was required to initiate movement of the femur. Although we did not set the *femur* in equilibrium (did not stabilize the femur) in Case Application 1-1, there must be a force applied to the femur that is opposite in direction to capsule-on-femur for there to be effective joint distraction. Joint distraction can occur only when the acceleration of one segment is less than (or in a direction opposite to) the acceleration of the adjacent segment, resulting in a separation of joint surfaces.

In the human body, the acceleration of one or both segments away from each other in joint distraction (the dynamic phase) is very brief unless the capsule and ligaments (or muscles crossing the joint) fail. John's leg-foot segment will not accelerate away from the femur for very long before the distraction forces applied to the adjacent joint segments (leg-foot and femur) are balanced by the tensile forces in the capsule. Given that John is still relaxed as the weight boot hangs on his leg-foot segment (we have not asked him to do anything yet), the check to joint distraction (the pull of gravity and the weight boot) is the tension in the capsule (and ligaments). John presumably has a ligamentous injury that is likely to cause pain with tension in these pain-sensitive connective tissues. If the distraction force remains, the capsule and ligaments may fail either microscopically or macroscopically (see Chapter 2). For now, we can prevent this problem by putting the supporting hand back under the weight boot. If the upward push of the hand is sufficient, the tensile forces on the ligaments can be completely eliminated.

Continuing Exploration 1-7:

Stabilization of the Femur

Because our interest is primarily in John Alexander's leg-foot segment and secondarily in the injured knee joint capsule, the source of stabilization of the femur (the other joint distraction force) was not a necessary component of our exploration. However, the principles established thus far will allow us to identify that distraction force.

In Figure 1–37, weightboot-on-legfoot and gravity-on-legfoot forces are composed into a single resultant distraction force (GWbLf) of –88 N, with the leg-foot segment once again unsupported. Gravity/weightboot-on-legfoot creates an 88 N tensile force in the capsule that creates a pull of the capsule on the femur (CF) with an equal magnitude of 88 N (see Fig. 1–37). A net distractive force of +88 N applied to the femur is necessary to stabilize the femur and create tension in the capsule.

The femur is contacted by both gravity-on-femur (GF) and treatment-table-on-femur (TF) (see Fig. 1–37). To determine the net force acting on the femur, we must estimate the mass or weight of that segment. John weighs approximately 734 N, and his thigh constitutes approximately 10.7% of his body weight.[1] His thigh, consequently, is estimated to weigh approximately 78 N. With the magnitudes of capsule-on-femur (–88 N) and gravity-on-femur (–78 N) known, it appears that the magnitude of table-on-femur should be the sum of the magnitudes of capsule-on-femur and gravity-on-femur (but opposite in direction). However, these vectors are not in a linear force system because they are not co-linear. Rather, they are parallel forces. Although we will tackle composition (and the effects) of parallel forces in more detail later, we can use the same shorthand system here that we used to compose two gravitational vectors earlier in the chapter.

Because both gravity-on-femur and capsule-on-femur (see Fig. 1–37) are vertically downward, the resultant of these two forces would be a new downward force with the

Continued

combined magnitudes of the original two (88 N + 78 N), with a point of application along a line drawn between the original two points and located slightly toward the vector with the greater magnitude. Because this new resultant vector will lie approximately in line with vector table-on-femur, we now have two forces in a linear force system on an object in equilibrium. Therefore, table-on-femur must have a magnitude of +166 N. It must be the second distraction force because it is applied perpendicular to and away from the joint surface. In Figure 1–37, the two distraction forces (GWbLf and TF) are shown as dashed vectors.

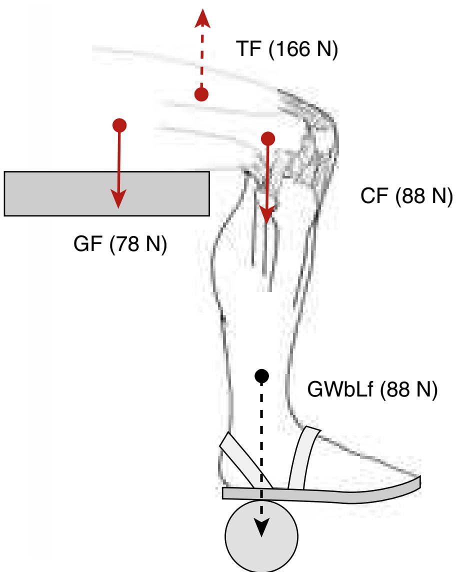

Figure 1–37 Distraction of the joint and tensile forces in the knee joint capsule occurs when there is a net distractive force directed away from the joint surfaces applied to *each* of the adjacent joint segments (dashed vectors). The distractive force on the femur is provided by the force of table-on-femur (TF), whereas the distractive force on the leg-foot segment is provided by GWbLf.

Concept Cornerstone **1-8**

Joint Distraction and Distraction Forces

- Distraction forces create separation of joint surfaces.
- There must be a minimum of one (or one resultant) distraction force on each joint segment, with each distraction force perpendicular to the joint surfaces, opposite in direction to the distraction force on the adjacent segment, and directed away from its joint surface.
- Joint distraction can be dynamic (through unequal or opposite acceleration of segments) or static (when the tensile forces in the tissues that join the segments are balanced by distraction forces of equal or greater magnitude).

Joint Compression and Joint Reaction Forces

Supporting John Alexander's leg-foot segment can minimize or eliminate the tension in his injured capsuloligamentous structures. In Figure 1–38, the supporting upward push of the hand on the leg-foot segment has been increased to +90 N. Given that the magnitude of the gravity and weight boot force is still –88 N, the sum of these forces will result in a net unbalanced force on the leg-foot segment of +2 N. The leg-foot segment will accelerate upward until a new force is encountered. This new force cannot come from the capsule that is now becoming increasingly slack, but it will arise once the leg-foot segment makes contact with the femur. The upward acceleration of the leg-foot segment will stop when the contact force, femur-on-legfoot (FLf), reaches a magnitude of –2 N (see Fig. 1–38), at which point equilibrium of the leg-foot segment is restored.

When the two segments of a joint are pushed together and "touch," as occurs with the upward support of the hand in Figure 1–38 (legfoot-on-femur and femur-on-legfoot), the resulting reaction (contact) forces are also referred to as **joint reaction forces.**[3] Joint reaction forces are contact forces that result whenever two or more forces cause contact between contiguous joint surfaces. Joint reaction forces are dependent on the existence of one force on each of the adjacent joint segments that is perpendicular to and directed *toward* the joint surfaces. The two forces that *cause* joint reactions forces are known as **compression forces.** Compression forces are required to push joint surfaces together to produce joint reaction forces in the same way that distraction forces are required to produce capsuloligamentous or muscular tension across separating (or separated) joint surfaces.

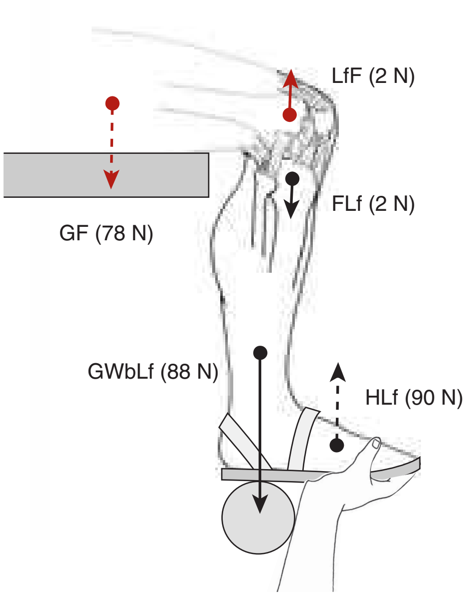

Figure 1–38 Joint compression results in joint reaction forces (FLf and LfF) when there is a net compression force applied to *each* of the adjacent joint segments (dashed vectors) toward the joint surfaces, in this case provided by hand-on-legfoot (HLf) and gravity-on-femur (GF).

Side-bar: It is important to note that here the term *compression* refers to pushing together rigid nondeformable bones to close a joint space. Compression across or within a deformable body is more complex and will be considered in Chapter 2.

In Figure 1–38, one of the forces causing joint compression at the knee joint is hand-on-legfoot because it is applied toward the articulating surface of the leg-foot segment (tibial plateau) and is perpendicular to that surface. If, however, the +2 N push of the leg-foot segment *on the femur* is not offset by a downward force of at least 2 N *on the femur*, the femur will also accelerate upward. If the femur and leg-foot segment were to accelerate upward at the same rate (and in the same direction), the contact between the joint surfaces might be maintained but could not be greater than 2 N.

Side-bar: Although the leg-foot segment is our focus, rather than the femur, it is worth noting that the femur is not likely to move because gravity is acting downward on the femur to stabilize it with a force of 78 N (see Fig. 1–38). Gravity-on-femur is the second joint compression force because it is the only force on the femur that is applied perpendicular to and toward the joint surfaces. In Figure 1–38, the two joint compression forces are shown as dashed vectors.

Whenever there is a net compression of joint surfaces (resulting in joint reaction forces), the capsule and ligaments at the joint are generally not under tension (as long as all forces are perpendicular to contacting surfaces). The pull of the capsule-on-legfoot segment is not shown in Figure 1–38 because the tension in the capsule has effectively been eliminated (or reduced to imperceptible magnitude). Equilibrium between two bony segments with net joint compression and resulting joint reaction forces assumes that the push of one bony segment on another does not result in failure of the bone (i.e., that one bone does not accelerate through the other).

Continuing Exploration 1-8:

Close-Packing of a Joint

Capsuloligamentous structures are typically not under tension when there are *net* compressive forces (with no shear forces) across a joint. There is an important exception, however. With sufficient twisting of the capsuloligamentous structures of a joint, the adjacent articular surfaces are drawn into contact by the pull of the capsule on the bony segments. This is called "close-packing" of the joint. This concept will be elaborated upon in Chapter 2 and in the examination of the individual joint complexes.

Revisiting Newton's Law of Inertia

It would appear that the weight boot is a poor option for John, given the potential tensile forces created in his injured joint capsule (and ligaments), unless we plan to continue supporting his leg-foot segment with a hand (or, perhaps, a bench). However, it has been assumed thus far that John is relaxed. As soon as John initiates a contraction of his quadriceps muscle, the balance of forces will change. Before we add the muscle force to the weight boot exercise, however, let us return to the leg-press exercise to identify what effect, if any, the forces from the leg press will have on the leg-foot segment or John's injured capsuloligamentous structures.

Concept Cornerstone 1-9

Joint Compression and Joint Compression Forces

- Joint compression forces create contact between joint surfaces.
- There must be a minimum of one (or one resultant) compression force on each contiguous joint segment, with each compression force perpendicular to and directed toward the segment's joint surface and opposite in direction to the compression force on the adjacent segment.

In the leg-press exercise (Fig. 1–39), John Alexander's leg-foot segment is contacting the footplate of the leg-press machine, creating the force of footplate-on-legfoot (FpLf). The magnitude of the footplate-on-legfoot vector is not yet known. There are also other forces acting on the leg-foot segment because other things are touching the leg-foot segment. One of these is gravity. Two other possibilities are contacts of femur-on-legfoot and capsule-on-legfoot. Whether the push of the femur on the leg-foot segment or the pull of the capsule on the leg-foot segment is a factor in this space diagram requires further exploration. We will begin with the known force, gravity-on-legfoot.

The magnitude of gravity-on-legfoot (–48 N) remains the same as in the weight boot example, but the orientation to the leg-foot segment differs. Consequently, the orientation of gravity to the leg-foot segment differs (Fig. 1–39). Even if we knew the magnitude of footplate-on-legfoot, gravity-on-legfoot and footplate-on-legfoot cannot be added together to find their resultant effect because the two forces are not in the same linear force system (they are not co-linear). It is theoretically possible to find the resultant of

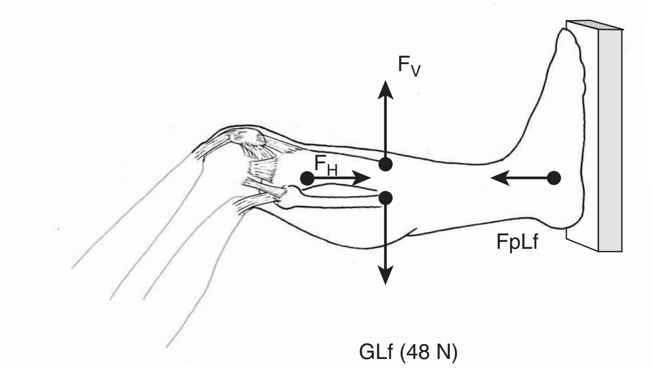

Figure 1–39 The known forces of footplate-on-legfoot (FpLf) and gravity-on-legfoot (GLf) must be balanced by another horizontal (F_H) and vertical (F_V) force, respectively.

these two forces through composition by parallelogram because these two vectors are part of a concurrent force system (the vectors will intersect if they are extended). That solution, however, requires that we know at least the relative magnitudes of these forces. A second option is to consider that these two vectors are each part of different linear force systems; we can then determine the magnitude of the vectors within each linear force system.

Vertical and Horizontal Linear Force Systems

Newton's law of inertia (or law of equilibrium) can be broken down into component parts: The sum of the **vertical forces (F_V)** acting on an object in equilibrium must total zero ($\Sigma F_V = 0$), and, independently, the sum of the **horizontal forces (F_H)** acting on an object in equilibrium must total zero ($\Sigma F_H = 0$). Consequently, there must be at least two additional forces acting on the leg-foot segment that are equal in magnitude and opposite in direction to gravity-on-legfoot and footplate-on-legfoot because the leg-foot segment cannot be at rest unless the sum of forces in both linear forces systems equals zero. Forces F_V and F_H are drawn in Figure 1–39, but the source of each force is not yet established.

We know that the femur and the capsule are both contacting, and potentially creating forces on, the leg-foot segment. Given these possibilities, it appears that vector F_H is likely to be the push of the femur on the leg-foot segment because the pull of capsule-on-legfoot would be in the opposite direction. The magnitude of femur-on-legfoot and footplate-on-legfoot can be estimated to be fairly small if John is relaxed and the footplate is locked in position. Before attempting to determine the magnitude of these forces, we will examine the source and magnitude of F_V because it will be seen that, in this example, F_V and F_H are related although part of different linear force systems.

The source of the F_V is difficult to ascertain because it appears that we have accounted for all objects contacting the leg-foot segment, but none apparently act in the direction of F_V. To identify F_V, we must acknowledge an additional property of all contact forces. Whenever there is contact between two objects (or surfaces of objects), the potential exists for friction forces on both contacting surfaces. The friction forces will have magnitude, however, only if there are concomitant opposing shear forces on the contacting objects.

Shear and Friction Forces

A **shear force (F_S)** is any force (or component of a force) that has an action line parallel to contacting surfaces (or tangential to curved surfaces) and that creates or limits movement between surfaces.

> *Side-bar:* The discussion here is about shear forces *between* two rigid (nondeformable) structures (e.g., bones). Shear within deformable structures will be considered in Chapter 2.

A **friction force (Fr)** *potentially exists* on an object whenever there is a contact force on that object. Friction forces are always parallel to contacting surfaces (or tangential to curved surfaces) and have a direction that is opposite to potential movement. For friction to have magnitude, a net shear force that creates or attempts to create movement between objects must exist. The force of friction can be considered a special case of a shear force because both are forces parallel to contacting surfaces, but friction is a shear force that is *always* in the direction opposite to movement or potential movement.

John Alexander's leg-foot segment is contacting the footplate. Because the footplate-on-legfoot force is a contact (or normal) force, it can also be labeled F_C (Fig. 1–40). The force of gravity-on-legfoot is parallel to the foot and footplate and has the potential to slide the foot down the footplate. Consequently, the gravity-on-legfoot force may also be referred to as a shear force. In the absence of another opposing shear force, there will be potential downward movement of the leg-foot segment and, therefore, a concomitant opposing frictional force (friction-on-legfoot [FrLf]). FrLf will be parallel to the foot and footplate surfaces and in a direction opposite to the potential slide of the foot (see Fig. 1–40). To understand the magnitude of the frictional force, we need to further explore the force of friction.

Static Friction and Kinetic Friction

The magnitude of a friction force on an object is always a function of the magnitude of contact between the objects and the slipperiness or roughness of the contacting surfaces. When two contacting objects with a net shear force applied to each are *not moving*, the magnitude of friction on each object is also proportional to the magnitude of the net shear force. If the two objects are *not* moving (objects are static), the *maximum* magnitude of the **force of static friction (Fr_S)** on each object is the product of a constant value known as the **coefficient of static friction (μ_S)** and the magnitude of the contact force (F_C) on each object; that is,

$$Fr_S \leq \mu_S F_C$$

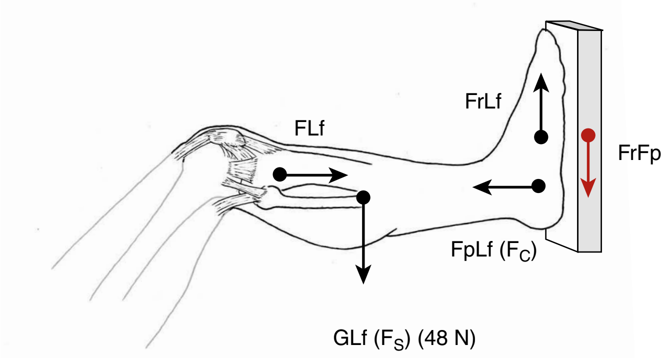

Figure 1–40 Footplate-on-legfoot (FpLf) is a contact force (F_C) that will result in friction-on-legfoot (FrLf) between the foot and footplate, given the shear force (F_S), GLf. Femur-on-legfoot (FLf) is also a contact force, but the low coefficient of friction for articular cartilage makes the value of friction between the femur and leg-foot segment negligible. Shown in a shaded vector that is not part of the space diagram is the reaction force to FrLf, friction-on-footplate (FrFp).

The coefficient of static friction is a constant value for given materials. For example, μ_S for ice on ice is approximately 0.05; the value of μ_S for wood on wood is as little as 0.25.[5] As the contacting surfaces become softer or rougher, μ_S increases. As the magnitude of contact (F_C) between objects increases, so too does the magnitude of potential friction. The greater the contact force on an object and the rougher the contacting surfaces, the greater the maximum potential force of friction. When you use friction to warm your hands, the contact of the hands warms both of them (friction forces exist on both the right and the left hands). If you wish to increase the friction, you press your hands together harder (increase the contact force) as you rub. Pressing your hands together harder increases the contact force between the hands and increases the maximum value of friction (the coefficient of friction remains unchanged because the surface remains skin on skin).

Side-bar: It is commonly thought that the magnitude of friction between two surfaces is related to the amount of surface area in contact. However, the only contributing factors are the magnitude of contact and the coefficient of the contacting surfaces.[2]

In Figure 1–41A, a large box weighing 445 N (~45 kg or 100 lb) is resting on the floor. The floor must push on the box (FB) with a magnitude equal to the weight of the box (GB) because the box is not moving ($\Sigma F_V = 0$). Because nothing is attempting to move the box parallel to the contacting surfaces (bottom of the box and the floor), there will be no friction on either the box or the floor. However, as soon as the man begins to push on the box (see Fig. 1–41B), a shear force (MB) is created, with a concomitant resulting force of friction-on-box (FrB). (Note that Fig. 1–41B is oversimplified because the force of friction-on-box is shown acting in line with the force of the man-on-box, rather than at the bottom of the box as would actually be the case.) Assuming that the man's initial push is not sufficient to move the box, we can begin by calculating the *maximum* possible magnitude of friction-on-box.

The maximum friction force on the box when the box is not moving is a product of the coefficient of static friction of wooden box on wood floor (0.25) and the magnitude (445 N) of the contact of floor-on-box:

$$\text{FrB} \leq (0.25)(445\ \text{N})$$
$$\text{FrB} \leq 111.25\ \text{N}$$

The magnitude of the friction-on-box force will be equal to that of the man-on-box as long as the box is not moving ($\Sigma F_H = 0$). No matter how much the man increases the magnitude of his push on the box (up to a maximum of 111.25 N), the magnitude of friction will increase by the same amount. However, *the magnitude of the force of friction can never exceed the magnitude of the shear force or forces.* Friction can oppose movement of a segment, but it cannot create movement.

If the push of the man on the box exceeds 111.25 N, the box will begin to move because friction cannot be more than 111.25 N and there will be a net unbalanced force to the left. Once an object is *moving*, the magnitude of the **force of kinetic friction (Fr_K)** on the contacting objects is a constant value, equal to the product of the contact force (F_C) and the **coefficient of kinetic friction (μ_K)**:

$$Fr_K = (\mu_K)(F_C)$$

The coefficient of kinetic friction (μ_K) is always smaller in magnitude than the coefficient of static friction (μ_S) for

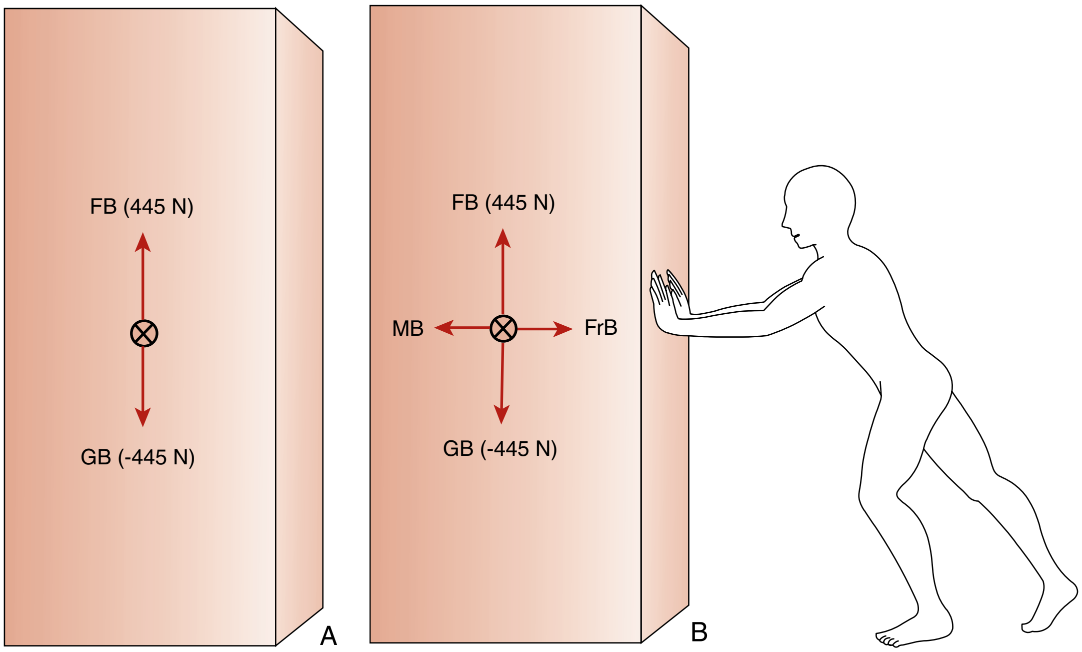

Figure 1–41 **A.** The box is acted on by the forces of gravity (GB) and the contact of floor-on-box (FB). The force of friction has no magnitude (so is not shown) because there is no attempted movement. **B.** The force of the man-on-box (MB) causes an opposing friction force (FrB).

any set of contacting surfaces. Consequently, the magnitude of the force of friction is always greatest immediately before the object is about to move (when the shear force has the same magnitude as the maximum value of static friction). Once the shear force exceeds the maximum value of static friction, the object will move because there will be a net unbalanced force. However, as soon as movement is initiated, the value of friction drops from its maximum static value to its smaller kinetic value, resulting in a sudden increase in the net unbalanced force on the object even if the magnitude of the shear force remains the same. The sudden drop in magnitude of friction results in the classic situation in which the man pushes harder and harder to get the box moving along the floor and then suddenly finds himself and the box accelerating too rapidly.

Concept Cornerstone **1-10**

Friction and Shear Forces

- Shear and friction forces potentially exist whenever two objects touch.
- A shear force is any force (or force component) that lies parallel to the contacting surfaces (or tangential to curved surfaces) of an object and causes or limits movement between the surfaces.
- Friction is a special case of a shear force in which the direction is always opposite to the direction of potential or relative movement of the objects (opposite in direction to the net shear force on that object).
- Friction has magnitude only when there is a *net* shear force applied to an object; that is, friction has magnitude only when two contacting objects move or attempt to move on each other after all potential shear forces are accounted for.
- The magnitude of static friction can change with a change in the net shear force that friction opposes; the magnitude of kinetic friction remains the same regardless of the shear force or forces it opposes or the speed of the moving object.
- The magnitude of friction can never exceed the magnitude of the shear force or forces it opposes.
- Shear and friction forces are always parallel to contacting surfaces, whereas the contact force (or contact force component) that must exist concomitantly is perpendicular (normal) to the contacting surfaces. Consequently, shear and friction forces are perpendicular to a contact force (or, more correctly, the component of a contact force that is “normal” to the contacting surfaces).[3]

Considering Vertical and Horizontal Linear Equilibrium

We can now return to John and the leg-press example and use our understanding of shear and friction forces to calculate the contact between the leg-foot segment and the footplate. Because the leg-foot segment is in equilibrium and there are only two vertical forces, the magnitude of the friction force must be the same as the magnitude of the shear force (GLf at 48 N) (see Fig. 1–40). If we know the magnitude of the frictional force and we estimate the coefficient of static friction between the sole of John’s shoe and the metal footplate at $\mu_S = 0.6$,[5] then we can solve for the contact force of footplate-on-legfoot:

$$48\text{ N} = (0.6)(F_C)$$
$$F_C = 80\text{ N}$$

If the magnitude of the footplate-on-legfoot vector (F_C) (see Fig. 1-40) is –80 N, then the magnitude of the femur-on-legfoot vector must be +80 N, because the sum of the horizontal forces must be zero for the leg-foot to be in equilibrium.

The femur-on-legfoot force (see Fig. 1–40) is also a contact force. Therefore, the potential for a vertical friction force also exists between the femur and the leg-foot segment as gravity attempts to move the leg-foot segment downward on the femur. However, the coefficient of friction between articular cartilage has been determined to be extremely low, with estimates such as 0.016[9] or 0.005.[3] Even if the higher of these two values is used, the magnitude of friction for an 80 N contact force cannot be greater than 1.28 N (~0.28 lb). Given this negligible magnitude, the force of friction on the leg-foot segment arising from the contact of the femur cannot be an important factor contributing to the vertical equilibrium of the leg-foot segment.

Part 2: Kinetics—Considering Rotary and Translatory Forces and Motions

When an object is completely unconstrained (not attached to anything), a single force applied *at or through the center of mass* of the object will produce linear displacement regardless of the angle at which the force is applied (Fig. 1–42A to C). In the previous examples used in this chapter, it has essentially been assumed that linear displacement is occurring. As we begin exploring rotary and general plane (curvilinear) motion, which is more commonly part of human motion, the reader is cautioned that discussion and examples are largely confined to two-dimensional analyses. Although human motion occurs in a three-dimensional environment, it is generally sufficiently challenging for the novice to understand a two-dimensional approach. Subsequent chapters will superimpose the third dimension conceptually, although rarely mathematically. Readers who wish to pursue three-dimensional mathematical analyses are encouraged to access more advanced resources.

TORQUE, OR MOMENT OF FORCE

When the force applied to an unattached object does not pass through the center of mass, a combination of rotation and translation will result (Fig. 1–43). To produce pure rotary motion (angular displacement), a *second force* that is parallel to the original force must be applied to the object or segment. When a second force equal in magnitude, opposite in direction, and parallel to the original force (Fig. 1–44) is applied, the translatory effects of these forces will offset each other and pure rotary motion will occur. If the object is unconstrained, as it is in Figure 1–44, the rotation of the segment will occur around a point midway between the two vectors. If the object is constrained by one of the forces (if the second finger cannot move from its point of contact), the rotation will occur around the point of application of the constrained force (Fig. 1–45). Two forces that are equal in magnitude, opposite in direction, parallel, and applied to the same object at different points are known as a **force couple.** A force couple will always produce pure rotary motion of an object (if there are no other forces on the object). The strength of rotation produced by a force couple is known as **torque (τ),** or **moment of force,** and is a product of the magnitude of one of the forces and the shortest distance (which always will be the perpendicular distance) between the forces:

$$\tau = (F)(d)$$

The perpendicular distance between forces that produce a torque, or moment of force, is also known as the **moment arm** (**MA**). Consequently, we can also say that:

$$\tau = (F)(MA)$$

Because force is measured in newtons and distance in meters, the unit for torque is the **newton-meter (Nm).** In the US system, the torque unit is the **foot-pound (ft-lb).** As already noted under this chapter's section on kinematics, a torque that tends to produce a clockwise rotary motion is generally given a negative sign, whereas a torque that tends to produce counterclockwise motion is given a positive sign. Of course, the direction of potential rotation or torque at a joint segment can also be labeled by using the terms *flexion/extension*, *medial/lateral rotation*, or *abduction/adduction*. The terms *torque* and *moment of force* are synonymous as they are used in this text. Consequently, a torque in the direction of joint flexion, for example, may also be referred to as a **flexion moment** or a flexion torque.

Because the terms *torque* and *moment of force* are often unfamiliar or intimidating to readers, the best simplistic translation we might use here is that torque, or moment of force, is the "strength of rotation" of a segment. Torque is directly proportional to both the magnitude of the applied force and the distance between the force couple. The

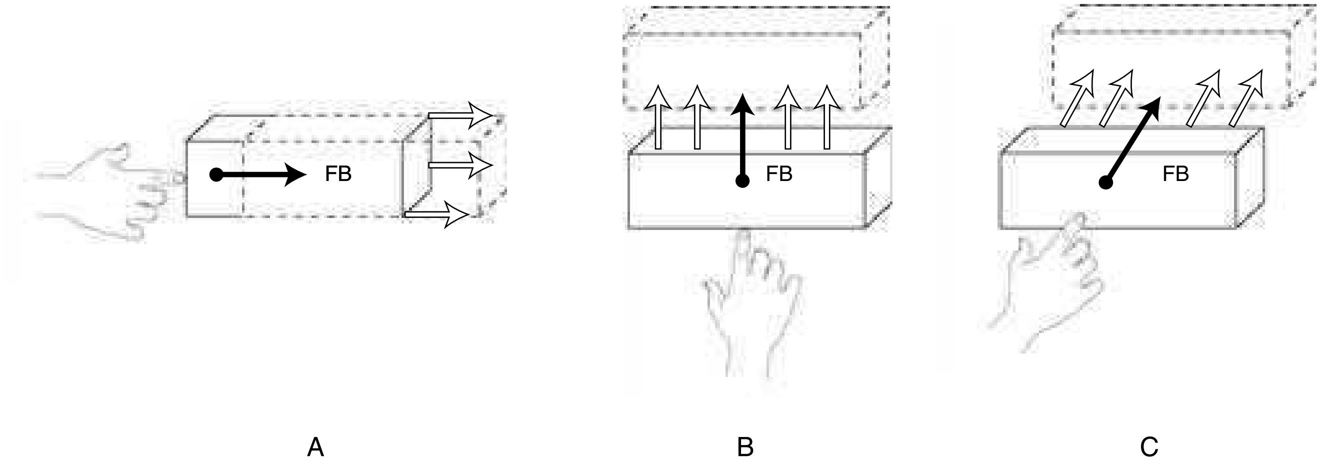

Figure 1–42 An isolated force applied to the block that passes through its CoM will produce linear displacement (translatory motion) of the block in the direction of the unbalanced force.

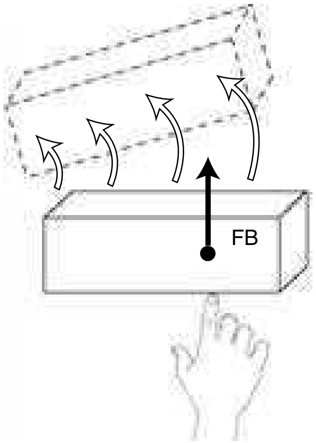

Figure 1–43 When an isolated force that does not pass through its CoM is applied to the block, a combination of rotary and translatory motion of the block will occur (general motion).

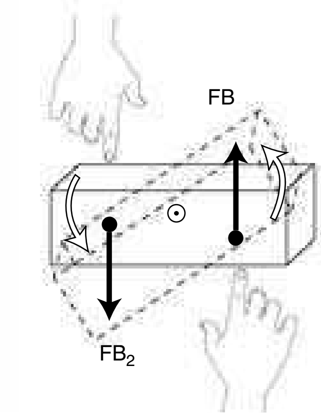

Figure 1–44 Two forces of equal magnitude applied to the block in opposite directions constitute a force couple and will create rotation about a point midway between the forces if both points of application are free to move.

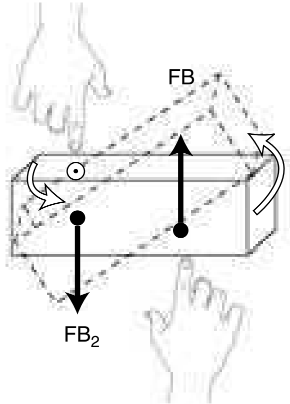

Figure 1–45 Two forces of equal magnitude applied to the block in opposite directions constitute a force couple and will create rotation around the point of application of one of the forces if that point is fixed.

greater the magnitude of the force couple (remember that the forces in a force couple have equivalent magnitudes), the greater the strength of rotation. The farther apart the forces of a force couple (the greater the MA), the greater the strength of rotation.

Angular Acceleration and Angular Equilibrium

If the torque created by the force couple is unopposed (there are no other forces on the segment), the result will be rotary (or angular) acceleration of the segment. Linear (translatory) acceleration (a), as already noted, is a function of net unbalanced force and the mass (m) of the object ($a = F_{unbal} \div m$). Angular acceleration (α) is given in deg/sec^2 and is a function of net unbalanced *torque* and the moment of inertia (I) of the object:

$$\alpha = \tau_{unbal} \div I$$

When the torques on an object are balanced ($\Sigma\tau = 0$), the object must be in angular (rotary) equilibrium (no resultant angular acceleration).

We can now identify three conditions that are *independently* necessary for an object or segment to be completely at rest:

$$\Sigma F_V = 0$$
$$\Sigma F_H = 0$$
$$\Sigma\tau = 0$$

If one or more of the three conditions are not met, the object will be in motion. In Figure 1–42A, the sum of the horizontal forces does not equal zero, which results in a net positive horizontal linear acceleration. In Figure 1–42B, the sum of the vertical also does not equal zero, which results in a net positive vertical linear acceleration. In Figures 1–44 and 1–45, the sum of the torque in each figure is unequal to zero, which results in a net positive (counterclockwise) angular acceleration. In Figure 1–43, there is both a net unbalanced vertical force and a net unbalanced torque, which results in both a positive angular acceleration and a positive vertical linear acceleration. Both together produce general motion. Every time we consider whether a segment is at rest or in motion, *each* of the three conditions for equilibrium of that segment must be considered separately.

The concept of torque is not as intuitive as the concept of magnitude of a force or weight. Nevertheless, we use the principle of maximizing torque on a regular basis.

Example 1-6

The doors of commercial buildings have a mechanism built into the hinges that generates a “closing” torque (or a resistance to opening). If the person is to succeed in opening the door, the “opening” torque must be greater than the “closing” torque. The “opening” torque generated by the force of the person-on-door (PD) (Fig. 1–46) is a product of the magnitude of this force and the distance (moment arm) that it is applied from the axis of rotation (the door hinges). Let us assume that the "closing" torque of the door is set at 5 Nm. The person would have to generate a torque greater than 5 Nm to open the door. If the person pushed at a distance of 0.25 m from the door hinge (vector PD1 in the top view of the door in Fig. 1–46), the person would have to push with a force of more than 20 N (~4.5 lb) to open the door. At 0.5 m from the hinge (vector PD2), the person could open the door with a push of slightly more than 10 N (~2.2 lb). A push at the far edge of the 1-m–wide door would require a push (PD3) of only a little over 5 N (~1+ lb). Because it is easiest to open the door (requires the least force) when the distance from the axis of rotation (the moment arm) is maximized, we have automatically learned to place our hand as far from the axis as possible, thus generating the most torque with the least effort. If a large force is applied far from the axis, it will generate a large amount of unbalanced torque and greater angular acceleration (i.e., the door will open faster!).

Parallel Force Systems

Because the forces in a force couple are parallel to each other, the two forces are part of a **parallel force system.** A parallel force system exists whenever two *or more* forces applied to the same object are parallel to each other. The torque generated by each force is determined by multiplying the magnitude of that force by its distance (MA) either from the point of constraint of the segment or from an arbitrarily chosen point on the segment (as long as the same point is used for all forces). Consequently, the torque generated by a force of constant magnitude may change if the force is moved closer to or farther from the point of constraint. The torque attributed to a force of constant magnitude with a *fixed* point of application on an object can also change if the reference point (axis or point of constraint) is changed.

Determining Resultant Forces in a Parallel Force System

The net or resultant torque produced by forces in the same parallel force system can be determined by adding the torques contributed by each force (with their appropriate signs). Three forces are applied to an unconstrained segment in Figure 1–47. The magnitudes of F1, F2, and F3 are 5 N, 3 N, and 7 N, respectively. The moment arms between F1, F2, and F3 and an arbitrarily chosen point are 0.25 m, 0.12 m, and 0.12 m, respectively. F1 and F2 are applied in a clockwise direction, whereas F3 is applied in a counterclockwise direction (in relation to the chosen point of rotation). The resultant torque (τ) would be:

$$\tau_{unbal} = (0.25\ \text{m})(-5\ \text{N}) + (0.12\ \text{m})(-3\ \text{N}) + (0.12\ \text{m})(+7\ \text{N})$$
$$\tau_{unbal} = -0.77\ \text{Nm}$$

That is, there will be a net rotation of the segment in a clockwise direction with a magnitude of 0.77 Nm.

Because the relatively small net torque in Figure 1–47 is calculated around a point that is *not* at the center of mass of the segment, the segment will not only rotate but will also translate. This is particularly evident if we consider the sum

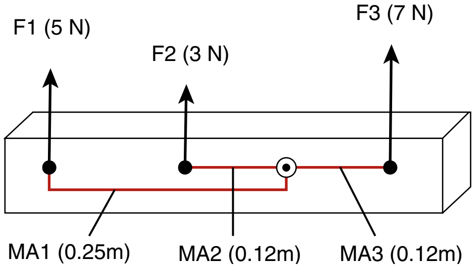

Figure 1–47 The resultant of three parallel forces is found by the sum of the torques produced by the product of each force (F1, F2, and F3) and its MA (distance from the specified point of rotation).

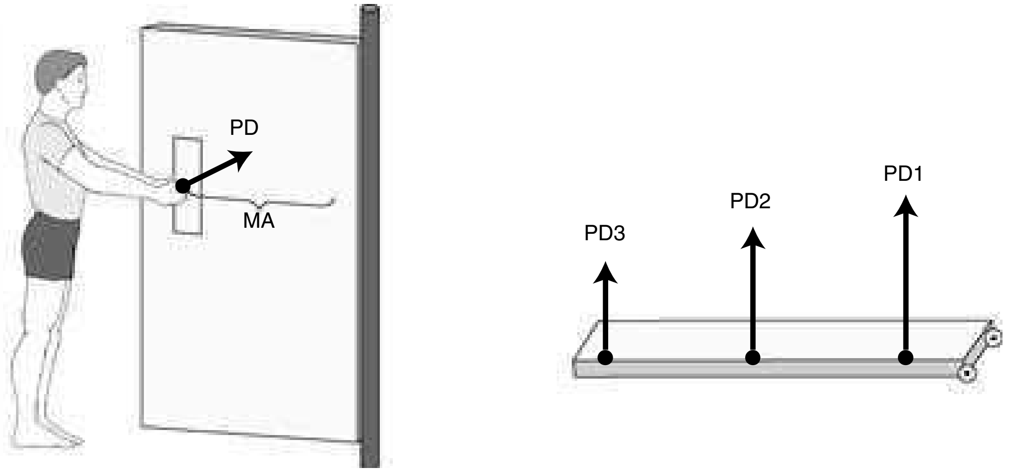

Figure 1–46 The force of person-on-door (PD) creates a torque at the axis (hinges) of the door because the force is applied at a distance (MA) from the hinge. **Inset.** Three different magnitudes of force (PD1, PD2, and PD3) can produce the same "opening" torque only if the magnitudes of force are inversely proportional to their distance from the door hinge.

of the vertical forces as well as the sum of the torques. Because all the vertical forces are upward, there is a concomitant net upward translatory force of 15 N. Consequently, the linear acceleration ($F_{unbal} \div m$) will substantially exceed the angular acceleration ($\tau_{unbal} \div I$), although both angular and linear acceleration will occur.

If the goal in Figure 1–47 is to rotate rather than translate the segment (as is the goal at joints in the human body), then the unwanted translation of the segment must be eliminated by the addition of a new force. However, let us first simplify the figure by composing F1 and F2 into a single resultant force, given that both are producing clockwise rotation of the lever.

Concept Cornerstone **1-11**

Composition of Forces in a Parallel Force System

When two parallel forces create torques in the same direction, the forces may be composed into a single resultant force whereby (1) the resultant force will have the same magnitude as the sum of the original two forces and (2) the resultant force will create the same torque as the sum of the torques of the two composing forces. If F1 (5 N) and F2 (3 N) in Figure 1-47 are composed into a new force, F1-2, the new resultant will have a magnitude of 8 N (3 N + 5 N). The torque of F1-2 would be the sum of the torques of F1 and F2:

$$\tau_{F1\text{-}2} = (0.25\text{ m})(+5\text{ N}) + (0.12\text{ m})(+3\text{ N}) = 1.61\text{ Nm}$$

The point of application of F1-2 can be determined by solving for its moment arm now that we know the torque and the force given that $\tau = (F)(MA)$:

$$MA_{F1\text{-}2} = \frac{1.61\text{ Nm}}{8\text{ N}} = 0.20\text{ m}$$

Consequently, F1 and F2 can be represented by force F1-2, which has a magnitude of 8 N and is located 0.20 m from the point of rotation.

In Figure 1–48, vectors F1 and F2 have been resolved into vector F1-2, and a new vector (F4) has been added.

Side-bar: Vector F4 might be easier in this particular figure to visualize as a push downward, as shown by the dotted gray arrow. However, we will continue to use the convention that the base of the arrow is on the point of application, as shown for the vector labeled F4.

Vector F4 has a magnitude of –15 N. With the introduction of this force on the object, $\Sigma F_V = 0$. Vector F4 is applied at 0 m from the designated point, so the torque produced by this force is zero. Any force applied *through* a reference point or point of rotation will not produce torque. Although the addition of vector F4 results in vertical equilibrium, the net torque is still –0.77 Nm. The effect produced by the addition of F4 is what happens in the human body. A body segment will translate (with or without a concomitant rotation) until an equal and opposite constraint is encountered, at which point the forces on the segment produce pure rotation.

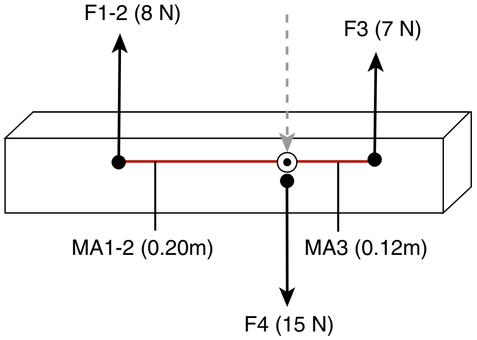

Figure 1–48 Forces F1 and F2 (Fig. 1–47) have been composed into vector F1-2. The addition of F4, applied through the point of rotation, will produce vertical equilibrium without producing any additional torque. The block will now rotate around a fixed axis in the direction of unbalanced torque.

Bending Moments and Torsional Moments

When parallel forces are applied to an unsegmented object (assumed to be rigid) in a way that results in equilibrium (neither rotation nor translation of the segment), the torques, or moments of force, applied to a particular point on the object are considered to be **bending moments.**[3,4] Although a bending moment can also be defined as the torque between two forces that compose a force couple[3] (e.g., vectors F1 and F2 in Fig. 1–49A), the segment will

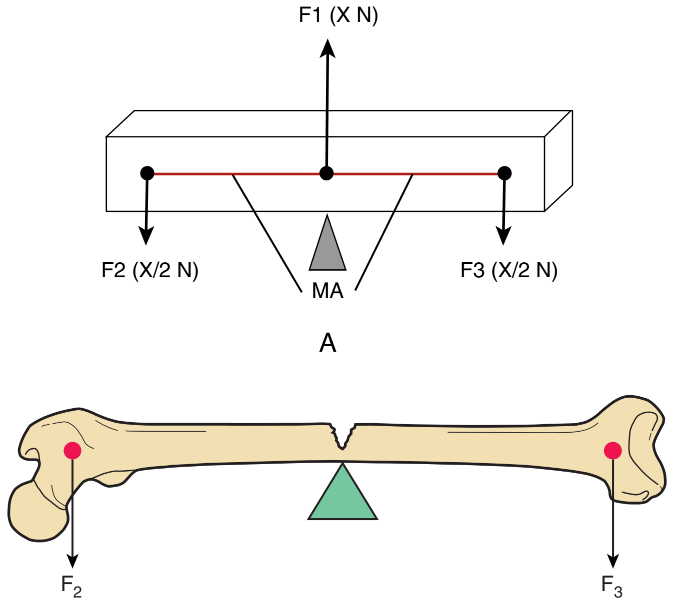

Figure 1–49 **A.** A bending moment is created when a third force is added to a force couple, resulting in rotary and translatory equilibrium but tending to "bend" the object around the center force. **B.** The principle of bending moments (or three-point bending) is often used in biomechanical testing of long bones (e.g., the femur).

rotate (rather than tend to bend) unless a third force is introduced (F3) to prevent this. For this reason, bending moments on a segment that is not rotating are also known as **three-point bending,** because three parallel forces are required. If the segment is in both rotary and translatory equilibrium (Fig. 1–49A), the sum of the vertical forces and the sum of the torques must be zero. Although the segment is neither rotating nor translating, the bending force around the point of application of F1 could result in deformation (bending) of the segment if the segment is *nonrigid.* As long as a body segment is allowed to rotate, three-point bending forces on the segment are minimized. Occasionally, a bony segment is constrained in a way that results in substantial bending forces that cause damage. The obvious clinical example is a bony fracture. Similarly, in research, bone strength is often empirically tested using an apparatus that creates three-point bending as shown in Figure 1–49B.

A **torsional moment** is sometimes considered a special case (or subcategory) of a torque, or moment of force, whereby a so-called torsional force creates (or tends to create) a rotation of a segment *around its long axis* (Fig. 1–50). The magnitude of the moment is still the product of the magnitude of the force and its shortest distance (MA) from the axis of rotation. When a force creates a rotation of a body segment around its longitudinal (or long) joint axis, the resulting torque produces a medial or lateral rotary moment.

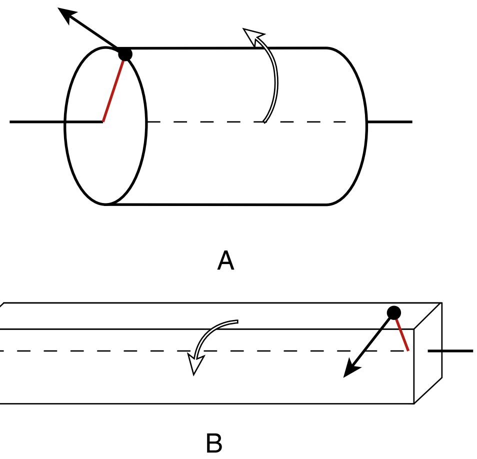

Figure 1–50 A force applied to the periphery of a long segment (through which an axis passes longitudinally) produces a “torsional moment” that is directly proportional to the magnitude of the force and its distance from the longitudinal axis.

Continuing Exploration 1-9:

Torsional Moments Versus Torque

In this text, the terms *torque* and *moment of force* are used whenever there is rotation between two segments around any one of three joint axes. The term *torsion* (or *torsional moment*) is used (and is probably used most often) to describe a torsional force applied to a single object (rather than between two objects); that is, a torsion creates a “twist” within the structure of the segment. Torsional forces certainly exist in rigid structures. However, torsional forces are a consideration primarily when the force has the potential to deform or damage the structure. For example, excessive torsional forces in long bones generate a particularly severe fracture called a spiral fracture. Torsional forces, therefore, will be a more significant consideration when we examine deformable structures such as connective tissue (bone, cartilage, tendon, and ligament) in Chapter 2.

Identifying the Joint Axis About Which Body Segments Rotate

In the human body, the motion of a segment at a joint is ultimately constrained by the articular structures—either by joint reaction forces (bony contact) or by capsuloligamentous forces. Any translatory motion of a segment produced by a force (e.g., gravity) will be checked before too long (we hope!) by the application of a new force (the push of a joint reaction force or the pull of a joint capsule or ligaments). The joint reaction or capsuloligamentous force that constrains further translation of the segment becomes one part of a force couple, resulting in continued movement of the segment as rotation around the point of constraint. The net effect of the translation (to the point of constraint) followed by rotation (around the point of constraint) is a subtle curvilinear motion of the segment with a very small translatory component. However, the implication is that the pivot point, or axis of rotation, for the segment is not fixed. The axis shifts slightly during the motion, with the rotation point for any increment of the motion serving as the instantaneous center of rotation. If the normal articular constraints are inadequate (in pathology, for example), there will be excessive translatory motion and an excessive shift of the joint axis before rotation can occur.

In order to assess the net rotation of a body segment around a joint, a common point needs to be identified from which the torque of each force acting on the segment will be calculated. Although it is acknowledged that the instantaneous center of rotation of a joint shifts, common practice is to identify a fixed point about which the joint rotation *appears* to occur. This is most often assumed to be approximately in the center of the sequential instantaneous centers of rotation and is referred to as the joint axis. It must be acknowledged, however, that this practice oversimplifies the assessment of torques on a body segment.

In Figure 1–51, John’s leg-foot segment is shown in the same position as in the leg-press exercise but without the contact of the footplate. If John is still relaxed in Figure 1–51, his leg-foot segment cannot remain in equilibrium. Initially, the leg-foot segment would translate downward linearly because of the downward force of gravity (vector GLf, 48 N). Gravity in this instance is a shear force because it lies parallel to the contacting surfaces of the femur (femoral condyles) and the leg-foot segment (tibial plateau). A shear force has the potential to generate an opposing friction force (which would be vertically upward in this example). However, the contact

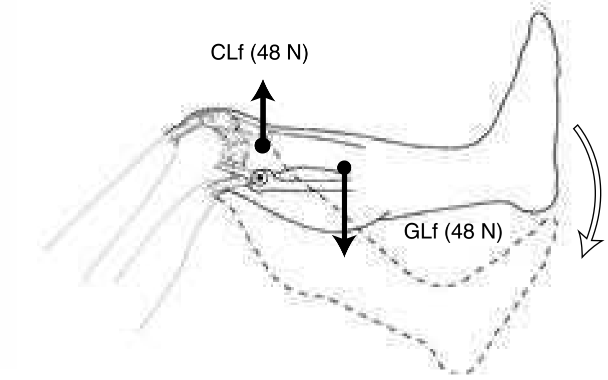

Figure 1–51 The force of gravity (GLf) would translate the leg-foot segment down until tension in the capsule (CLf) reached an equivalent magnitude, at which point GLf and CLf would form a force couple to rotate the leg-foot segment around the point of application of CLf.

between the femur and leg-foot segment would be minimal at the point in time shown in Figure 1–51 and the coefficient of friction would be small, resulting in a negligible upward friction force on the leg-foot segment. The leg-foot segment, therefore, would translate downward until the knee joint capsule became tensed. Once the magnitude of tension in the capsule and the concomitant pull of the capsule-on-legfoot force reached 48 N, it would form a force couple with the gravity-on-legfoot force the motion of the leg-foot segment would continue as a nearly pure rotation around the central point of the attachments of the knee joint capsule.

If, in Figure 1–51, the center of mass of John's leg-foot segment was located 20 cm (0.20 m) from the point of capsular constraint, the torque of the leg-foot segment that weighs 48 N would be 9.6 Nm (or ~7 ft-lb) around the pivot point of the capsule. Note that in Figure 1–51 the axis around which the rotary motion ultimately occurred is shifted down slightly from the capsular force's original point of application because of the downward translation of the segment that preceded the pure rotary motion.

The example presented in Figure 1–51 demonstrates why calculating torques as if there were a fixed joint axis can introduce at least some error in that calculation. Shifts in the point of constraint (or instantaneous center of rotation) during a motion can, by itself, result in slight differences in the moment arm for a given force and, therefore, slight differences in the torque, even if all other factors remained constant. As we will see, however, other factors do not remain constant; there are other, more influential factors that also will cause the torque of a force to vary as the segment moves at the joint. We begin to acknowledge here how complex even a simple joint rotation truly is.

Meeting the Three Conditions for Equilibrium

We have now established that everything that contacts a segment of the body creates a force on that segment and that each force has the potential to create translatory motion (vertical or horizontal), rotary motion (torque), or both. Before we increase the complexity by adding muscle forces to a segment, let us return to a previously oversimplified example to now consider all three conditions needed for equilibrium.

The magnitude of the contact force of the footplate on the leg-foot segment was initially calculated by assuming that the force of gravity and the force of friction were part of a linear force system and were equal in magnitude (see Fig. 1–40). Figure 1–52 shows the same four forces—gravity (GLf), friction (FrLf), footplate (FpLf), and femur (FLf)—all acting on the static leg-foot segment. The vectors femur-on-legfoot and footplate-on-legfoot are part of a horizontal linear force system and, in the absence of any other horizontal forces, must be equal in magnitude. It can now be appreciated that the action lines of femur-on-legfoot and footplate-on-legfoot pass approximately through the knee joint axis; therefore, femur-on-legfoot and footplate-on-legfoot will not create a torque around the knee joint (moment arm ≈ 0). Because these two forces balance each other out linearly ($\Sigma F_H = 0$) and create no other torque, femur-on-legfoot and footplate-on-legfoot have been shaded in Figure 1–52 as a way of removing them from further consideration.

The forces of gravity (a shear force) and friction (a response to that shear force) in Figure 1–52 are not co-linear (as we simplistically considered them in Fig. 1–40) but are both vertical and parallel. *If* these two forces are equal in magnitude (a working assumption only), the sum of the vertical forces would be zero. However, the forces gravity-on-legfoot and friction-on-legfoot also constitute a force couple that would rotate the leg-foot segment counterclockwise. Because friction can resist clockwise rotation but cannot actually move the leg-foot segment in a counterclockwise direction, the force of friction will effectively act as the constraint (its point of application being the point of potential rotation for the force couple). The torque of the force couple, therefore, is equivalent to the magnitude of 48 N (GLf) multiplied by the distance (moment arm) between gravity-on-legfoot and friction-on-legfoot.

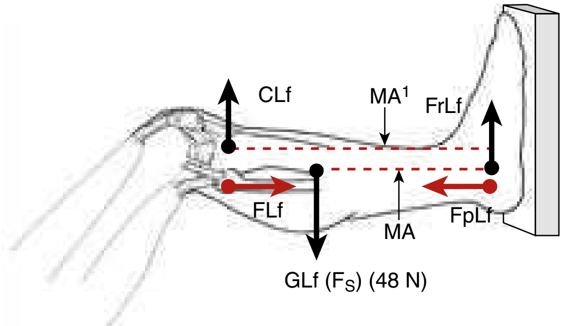

Figure 1–52 Vectors FLf and FpLf are shaded because they produce horizontal equilibrium and do not contribute to torque. The sum of vertical vectors (CLf, GLf, and FrLf) must be zero. Assuming that friction-on-legfoot (FrLf) is fixing the foot to the footplate, the torques are computed as the product of the magnitudes of gravity-on-legfoot (GLf) and capsule-on-legfoot (CLf) and their distances (MA and MA^1, respectively) from the point of application of FrLf. The sum of the torques will be zero.

Given that gravity-on-legfoot is not *actually* rotating the leg-foot segment around the point of application of friction-on-legfoot, there must be at least one other force acting on the leg-foot segment that is creating a clockwise torque with a magnitude equal to that of the torque of gravity-on-legfoot. The only other contact on the leg-foot segment in Figure 1–52 is that of the joint capsule. If the leg-foot segment started to rotate counterclockwise around the point of application of friction-on-legfoot, the proximal articular surface of the leg-foot segment would slide down the femur, as we saw in Figure 1–51. With the downward motion of the leg-foot segment, the capsule would become tight and the force of capsule-on-legfoot (CLf) would be introduced.

To create a torque equivalent to the torque of gravity-on-legfoot, the magnitude of capsule-on-legfoot would have to be only about half the magnitude of gravity-on-legfoot because capsule-on-legfoot lies approximately twice as far (2 × MA) from the point of application of friction-on-legfoot (the presumed point of constraint). If the magnitude of capsule-on-legfoot is 24 N (48 N/2), then the magnitude of friction-on-legfoot must also be 24 N in order for $\Sigma F_V = 0$. We have now met all three conditions for equilibrium of the leg-foot segment:

$$\Sigma F_H = (+FLf) + (-FpLf) = 0$$
$$\Sigma F_V = (+CLf) + (-GLf) + (+FrLf) = 0$$
$$\Sigma t = (+CLf)(2MA)+(-GLf)(MA) + (FrLf)(0) = 0$$

We used the magnitude of friction-on-legfoot to calculate the contact force (footplate-on-legfoot) in Figure 1–40. Because it is now evident that we overestimated the magnitude of friction in that example, we also overestimated the magnitude of the footplate-on-legfoot force in that example. However, the magnitudes of footplate-on-legfoot and femur-on-legfoot will continue to be equivalent (both less than originally estimated) because they are the only horizontal forces on the leg-foot segment.

Thus far, we have considered that John Alexander is relaxed and that the leg-foot segment is in equilibrium in both the weight boot example and in the leg-press example. If the goal is to strengthen John's quadriceps muscle, it is time to introduce a muscle force.

MUSCLE FORCES

Total Muscle Force Vector

The force applied by a muscle to a bony segment is actually the resultant of many individual force vectors (muscle fibers) pulling on a common tendon. Because each muscle fiber can be represented by a vector that has a common point of application (Fig. 1–53), the fibers form a concurrent force system with a resultant that represents the total muscle force vector (Fms). The total muscle force vector can be approximated by putting the point of application at the muscle's attachment and then drawing an action line symmetrically toward the middle of the muscle's fibers. The direction of pull for any muscle is *always* toward the center of the muscle. The length of the resultant vector is proportional to its magnitude. The magnitude assigned to a muscle force is often estimated because the actual magnitude (the pull of a muscle on its attachment) cannot be determined in a living person without invasive measurement tools.

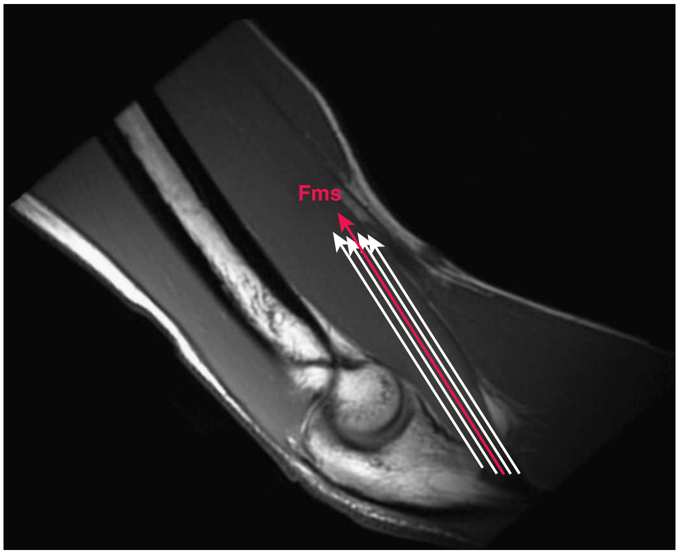

Figure 1–53 Sagittal T1-weighted MRI of the elbow. The brachialis muscle can be seen inserting into the ulna and the total muscle force (Fms) is the resultant of all the individual muscle fiber pulls.

Continuing Exploration 1-10:

Measuring Muscle Force

Electromyography (EMG) can measure the electrical activity of a muscle. The electrical activity is directly proportional to the motor unit activity, which is, in turn, directly proportional to isometric muscle force (see Chapter 3). However, neither electrical activity nor the number of motor units is a measure of absolute force, because muscle is exquisitely sensitive to its mechanical environment (e.g., length, velocity). When the clinical "strength" of a muscle is measured by using weights, force transducers, or isokinetic devices, what is actually being measured directly or indirectly is joint torque. Although the net internal torque on the segment can be estimated if the moment arms of the known external forces (gravity, weights, and so on) can be estimated, the magnitudes of individual internal forces contributing to the net internal torque are difficult, if not impossible, to identify. Even if a single muscle were active (which is almost never the case), it would be difficult to separate out the influence of forces such as joint reaction forces, capsuloligamentous forces, and small friction forces. The result is that the actual magnitude of pull of a single muscle cannot be assessed in a living person (in vivo) without surgically implanting a device directly onto the tendon—which may itself alter the force normally produced by a muscle. It should be noted that individual muscle forces are often approximated using mathematical models of

Continued

varying levels of sophistication. The outputs of these models are simply best estimates of muscle force.

Every muscle pulls on all of its attachments every time the muscle exerts a force. Therefore, every muscle creates a minimum of two force vectors, one on each of the two (or more) segments to which the muscle is attached; each of the two (or more) vectors is directed toward the middle of the muscle. The type and direction of motion that results from an active muscle contraction depends on the net forces and net torques acting on each of its bony attachments. The muscle will move a segment in its direction of pull only when the torque generated by the muscle exceeds opposing torques.

In a space diagram (e.g., John Alexander's leg-foot segment), we will often identify only the pull of the muscle on the segment under consideration. However, it should be recognized that we are purposefully ignoring that same muscle's concomitant pull on one or more *other* segments.

Anatomic Pulleys

Frequently, the fibers of a muscle or a muscle's tendon wrap around a bone or are deflected by a bony prominence. When the direction of pull of a muscle is altered, the bone or bony prominence causing the deflection forms an **anatomic pulley.** Pulleys (if they are frictionless) change the direction without changing the magnitude of the applied force. As we will see, the change in action line produced by an anatomic pulley will have implications for the ability of the muscle to produce torque.

Figure 1–54A shows what the middle deltoid muscle might look like if the muscle were attached to two straight levers. The vector (Fms) would lie parallel to the humerus. Figure 1–54B shows the middle deltoid muscle as it crosses the actual glenohumeral joint, wrapping around the acromion and the rounded head of the humerus. The humeral head and acromion change the direction of the fibers at both ends of the muscle and, therefore, function as anatomic pulleys. The action line and direction of the muscle force vector are significantly different between parts A and B of Figure 1–54, although the point of application and magnitude of the force are the same in each figure part.

Anatomic Pulleys, Action Lines, and Moment Arms

The function of any pulley is to redirect a force to make a task easier. The "task" in human movement is to rotate a body segment. Anatomic pulleys (in the majority of instances) make this task easier by deflecting the action line of the muscle *farther* from the joint axis, thus increasing the moment arm of the muscle force. By increasing the moment arm of a muscle force, a force of the same magnitude will produce greater torque. If the middle deltoid muscle had the action line shown in Figure 1–54A, the moment arm would be quite small. The moment arm is substantially greater when the humeral head and overhanging acromion result in a shift in the action line of the muscle away from the joint axis (see Fig. 1–54B). Consequently, the deltoid muscle will be able to produce an equivalent abduction moment on the humerus with less force in Figure 1–54B than in Figure 1–54A.

There are other sesamoid bones in the human body (although the patella is by far the largest). Each sesamoid bone has the similar effect of changing the direction of a muscle or tendon action line and, as a result, increasing the ability of a muscle to produce torque around one or more joint axes.

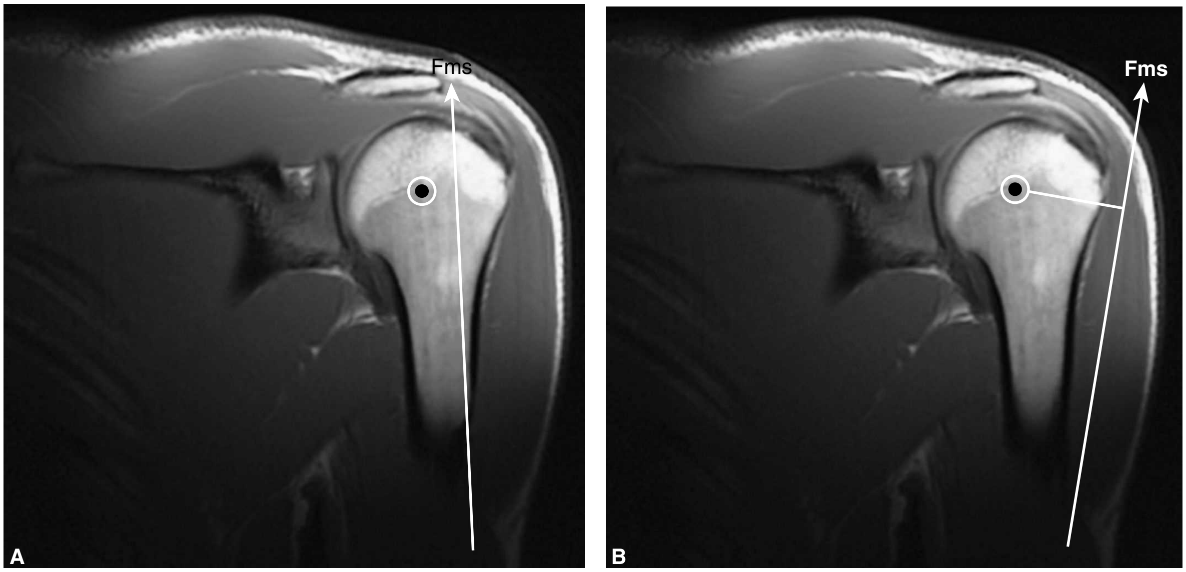

Figure 1–54 Coronal oblique T1-weighted MRIs of the shoulder. **A.** A schematic representation of the muscle force produced by the middle deltoid muscle *if* the muscle acted in a straight line between the origin and insertion. **B.** A more anatomic representation of the middle deltoid muscle, showing its line of pull deflected away from the joint axis by the anatomic pulley of the humeral head.

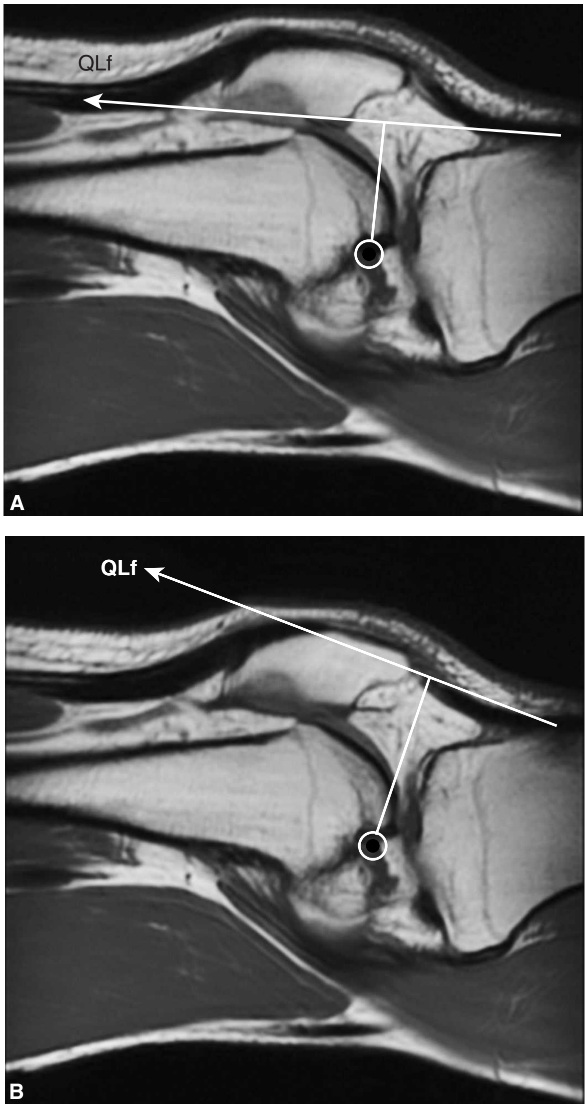

Figure 1–55 Sagittal T1-weighted MRIs of the knee. **A.** The line of pull and moment arm of the quadriceps muscle without the patella. **B.** With the patella's pulley effect, the line of pull of the muscle is deflected away from the joint axis, increasing the moment arm of the muscle force.

Example 1-7

The Patella as an Anatomic Pulley

The classic example of an anatomic pulley is that formed by the patella. The quadriceps muscle belly lies parallel to the femur. The tendon of the muscle passes over the knee joint and attaches to the leg (tibia) via the patellar tendon at the tibial tuberosity. For knee joint extension, the joint axis is considered to be located through the femoral condyles. The moment arm for the quadriceps muscle force (QLf) lies in the space between the vector and the joint axis. Without the patella, the line of pull of the quadriceps muscle on the leg-foot segment would follow the patellar tendon at the tibial tubercle and would lie parallel to the leg-foot segment (Fig. 1–55A). However, the patella is embedded in the quadriceps tendon and pushes the tendon away from the femur, changing the angle that the patellar tendon makes with the leg (tibia) and changing the line of pull of the quadriceps muscle away from the knee joint axis (Fig. 1–55B). The effect of changing the line of pull of the quadriceps muscle on the tibia is to increase the moment arm in Figure 1–55B. Given an increased moment arm, the same magnitude of force would produce greater torque (and a greater angular acceleration) in Figure 1–55B than in Figure 1–55A.

Concept Cornerstone **1-12**

Moment Arm and Lever Arm

The moment arm for any force vector will always be the length of a line that is perpendicular to the force vector and intersects the joint axis (presuming a two-dimensional perspective). In other words, the moment arm will always be the shortest distance between a force vector and the axis of rotation. For a force that is perpendicular to the long axis of a segment (as the forces were for Figs. 1–44 through 1–49), the moment arm will be parallel to and lie along the segment (lever). In this instance, the term **lever arm (LA)** may also be used to describe this distance. Although it is not necessary to employ the term *lever arm* because "moment arm" covers all situations, there are some instances in which the term *lever arm* serves as a convenient reminder that the force is perpendicular to the lever.

Concept Cornerstone **1-13**

Muscle Force Vectors

- Active or passive tension in a muscle creates a force (a pull) on all segments to which the muscle is attached, although one may choose to consider only one segment at a time.
- The point of application of a muscle force vector is located at the point of attachment on a segment.
- The muscle action line is in the direction of the fibers or tendons *at the point of application.*
- Muscle vectors (like all vectors) continue in a straight line from the point of application, regardless of any change in direction of muscle fibers or tendons after the point of application.
- The magnitude of a muscle force is generally a hypothetical or theoretical value because the absolute force of a muscle's pull on its attachments cannot be measured noninvasively.

TORQUE REVISITED

We are now ready to add the quadriceps muscle force to John Alexander's leg-foot segment at the point at which knee extension is to be initiated in the weight boot example. Figure 1–56 shows the pull of the quadriceps

CASE APPLICATION

Composition of GLf and WbLf

case 1–2

The magnitude of the resultant force, gravity/weightboot-on-legfoot, is the sum of the magnitudes of the composing forces (gravity-on-legfoot and weightboot-on-legfoot), and the resultant torque is the sum of the torques contributed by each composing vector. The torques contributed by each of the composing forces can be determined if the moment arm for each force is known. Using segmental lengths from LeVeau,[1] we will estimate (1) that John's center of mass for his leg-foot segment lies 0.25 m from his knee joint axis and (2) that the weight boot (at the end of his leg-foot segment) is 0.5 m from his knee joint axis. Segmental lengths are lever arms—the distances from each point of application of the force to the knee joint axis. These distances are the moment arms of the forces *only* if the forces are perpendicular to the lever (when moment arm = lever arm), and so we will compute the torques for the point at which the leg-foot segment is parallel to the ground (given that the vectors will then be perpendicular to the lever). Using the values for John and recognizing that both forces that are being composed are downward, we find that the torques for the gravity-on-legfoot force and the weightboot-on-legfoot force are:

$$\tau_{GLf} = (48\ \text{N})(0.25\ \text{m}) = -12\ \text{Nm}$$
$$\tau_{WbLf} = (40\ \text{N})(0.5\ \text{m}) = -20\ \text{Nm}$$

If the gravity/weightboot-on-legfoot force has a magnitude of 88 N and a torque of –32 Nm, then the moment arm for the force must be τ ÷ F, or 0.36 m. Because the point of application of the resultant force will not change with the orientation of the leg-foot segment in space, the gravity/weightboot-on-legfoot force will also be applied 0.36 m from the joint axis when the leg-foot segment is as shown in Figure 1–56. However, as we shall see next, the moment arm for the resultant force will change with the change of position of the leg-foot segment in space.

muscle on the leg-foot segment. In the figure, the force of gravity and the force of the weight boot are represented as a single resultant force, GWbLf, as they appeared in Figure 1–37. Case Application 1-2 shows the calculation for the point of application of the resultant vector, gravity/weightboot-on-legfoot.

Changes to Moment Arm of a Force

For some vectors, like the resultant of the forces of gravity and the weight boot on the leg-foot (GWbLf), finding the shortest (or perpendicular) distance between a vector and a joint axis (moment arm) requires that the vector be extended. The effect of a vector is not changed by extending it graphically—nor should it be considered to change the magnitude of the force. When the gravity/weightboot-on-legfoot vector is extended in Figure 1–56 (dashed line), it still has a magnitude of –88 N, but its moment arm is effectively zero because the extended vector passes through the knee joint axis. Therefore, in this position of the knee joint, the gravity/weightboot-on-legfoot force creates negligible torque on the segment. Given that the vector produces no torque in this knee joint position, a relatively small force by the quadriceps muscle applied through its relatively larger moment arm (estimated to be 0.03 m) will yield a net resultant torque in the direction of knee extension. This will not continue to be the case for very long, because as soon as the position of the leg-foot segment changes, so too do the torques created by the forces applied to the leg-foot segment.

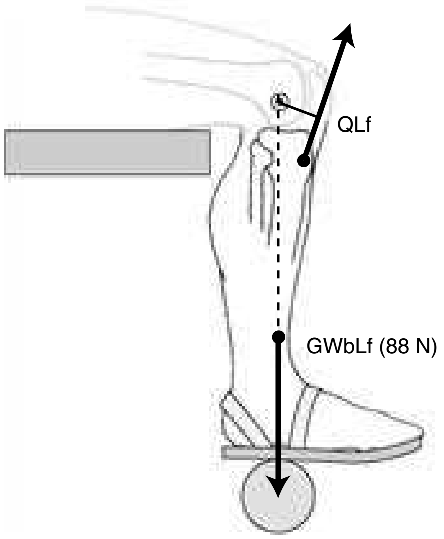

Figure 1–56 The resultant force for gravity and the weight boot (GWbLf) and the force of the quadriceps muscle (QLf) are shown with their respective MAs when the knee is at 90° of flexion. Vector GWbLf must be extended to ascertain its shortest distance to the joint axis.

In Figure 1–57, the leg-foot segment has been brought farther into the knee extension (~45°of knee flexion). As the segment moves in space, the relation between the forces applied to the segment and the segment itself change. The extended gravitational vector (GWbLf) now lies at a substantially greater distance from the knee joint axis. The quadriceps muscle vector also has a larger moment arm (increasing from 0.03 m to 0.05 m), but the increase is minimal in comparison to the increase in the moment of the gravity/weightboot-on-legfoot force, which has increased from 0 m to 0.27 m. The magnitude of the quadriceps force necessary to continue knee extension from this new point in the range of motion can now be estimated.

If the moment arm for the gravity/weightboot-on-legfoot force is 0.27 m in Figure 1–57, then a force of 88 N would create a clockwise (knee flexion) torque of 23.76 Nm. The quadriceps muscle would have to create a counterclockwise (extension) torque of 23.76 Nm to maintain rotary equilibrium. If the moment arm for the quadriceps muscle in Figure 1–57 is 0.05 m, then quadriceps force would have to have a magnitude of 475.2 N (~107 lb) just to maintain the leg-foot segment at 45°. The quadriceps force has to be greater than 475.2 N to create a net unbalanced torque in the direction of knee extension.

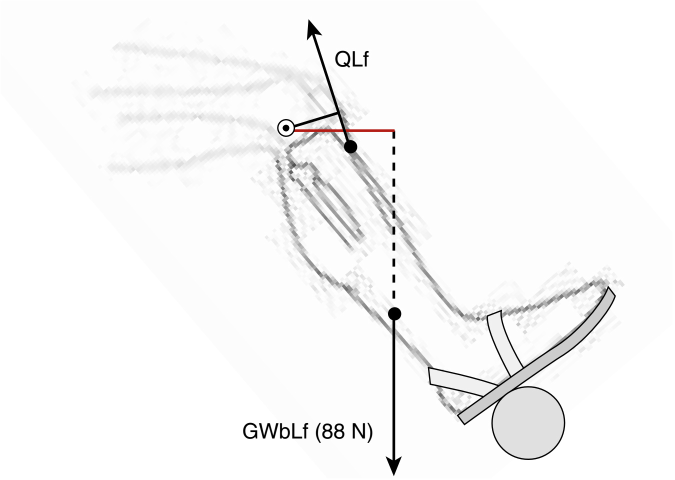

Figure 1–57 As the leg-foot segment moves to 45° of knee flexion, the relative sizes of the MAs for both QLf and GWbLf increase.

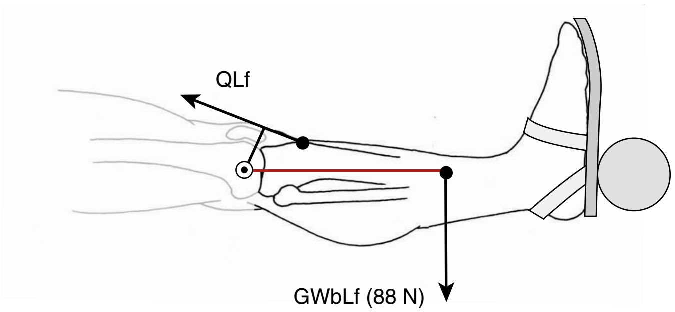

Figure 1–58 At full knee extension, the MA of vector GWbLf is as large as it can be, whereas the MA for QLf is now at its smallest.

Figure 1–58 shows the leg-foot segment at the end of knee extension (0° of knee flexion). The gravity/weight-boot-on-legfoot vector is now perpendicular to the leg-foot segment, and its moment arm has further increased (0.36 m). The moment arm for the quadriceps force in Figure 1–58 is smaller than in the previous figure. The magnitude of the resultant gravitational force remains unchanged at 88 N. However, it now creates a clockwise (flexion) torque of 31.68 Nm. If the moment arm for the quadriceps is now 0.01 m, the muscle will have to generate a force of 3,168 N (712 lb) to maintain this position (rotary equilibrium).

Whenever any force is applied at 90° to the long axis of a segment (as GWbLf is in Fig. 1–58), the length of the moment arm for that force is maximal. The moment arm will lie parallel to the long axis of the segment (the lever) and, under these conditions (as noted in Concept Cornerstone 1-12), can also be referred to as the lever arm of the force. Because the moment arm of a force is greatest when that force is at 90° to the segment, the torque *for a given magnitude of force* will also be greatest at this angle. When lifting the weight boot, John will find the position in Figure 1–58 hardest to maintain. Although John is working against the consistent 88 N weight of the leg-foot segment and weight boot, the flexion torque generated by the combined force is greatest when the leg-foot segment is horizontal (parallel to the ground). At this point, it requires the greatest contraction of the quadriceps muscle to offset the torque generated by the resultant gravitational force (GWbLf).

Concept Cornerstone **1-14**

Moment Arms and Torque

- As the moment arm of a force increases, its potential to produce torque increases.
- The moment arm of a force is maximum when the force is applied at 90° to its segment.
- The moment arm of a force is minimum (0.0) when the action line of the force passes through the center of rotation of the segment to which the force is applied.

Continuing Exploration 1-11:

Work, Energy, and Power

Work, energy, and power are combinations of kinematic and kinetic variables. Although these terms are not the focus of this chapter, they are important concepts in biomechanics are related to muscle force production, and warrant brief discussion. **Work** is defined as force applied over some distance and is expressed in units of joules (Nm).

$$W = F \times D$$

For example, in Figure 1–3A, if the examiner applied a load of 10 N to the proximal tibia and moved the tibia 2 cm, she would have completed 0.2 J (10 N × 0.02 m) of work. **Power** is that rate of work production and is expressed in watts (J/sec).

$$P = W \div \text{time}$$

For example, if the clinician moved the proximal tibia in 2 seconds, she would have generated 0.1 watt of power. Although this concept may seem trivial, power is frequently misused when talking about force. Power is the rate of work production and is much more complicated than simple force production. The final concept is energy. **Energy** is the ability to perform work and is expressed in the same units as work (joules).

Angular Acceleration With Changing Torques

We have approached John's weight-boot example as a series of freeze-frames or single points in time (see Figs. 1–56 through 1–58). We have also established that John will have to contract his quadriceps muscle with a force of approximately 3,168 N to maintain full knee extension against the flexion torque of gravity and the weight boot. If John *initiated* the exercise at 90° of knee flexion (see Fig. 1–56) with a quadriceps muscle contraction of 3,168 N, almost all the torque generated by the muscle contraction would be unopposed, and the unbalanced torque would result in a substantial angular acceleration of the leg-foot segment in the direction of extension with the initiation of the muscle contraction. As the leg-foot segment moves from 90° of flexion toward increased extension, the flexion torque generated by the gravity/weightboot-on-legfoot force gradually increases until it is maximal in full knee extension (see Fig. 1–58). Consequently, the net unbalanced torque and acceleration of the segment must be changing as the knee extends. If knee extension proceeded through the series of freeze-frames from 90° of knee flexion to full knee extension, and *if the force of quadriceps muscle contraction is constant*, the leg-foot segment would generally accelerate less as extension continues until equilibrium is reached in full knee extension (when posterior knee structures would, along with gravity and the weight boot, contribute to checking further knee extension). The decrease in acceleration of the segment, however, is not consistent through the ROM. Whereas the torque produced by the gravity/weightboot-on-legfoot force will continually increase as the force's moment arm increases, the quadriceps torque (even with a constant contraction of 3,168 N) varies with the change in moment arm of the quadriceps muscle. It is a characteristic of the quadriceps muscle that its moment arm is greatest in the middle of the motion and less at either end (being least in full knee extension).

Side-bar: The change in moment arm of a muscle through the range of the joint it crosses is somewhat unique to each muscle and is not predictable.

Moment Arm and Angle of Application of a Force

We have seen that the moment arm of a force can change as a segment rotates around its joint axis and as the segment changes its orientation in space. The length of the moment arm is directly related to the **angle of application of the force** on the segment. The angle of application of a vector is the angle made by the intersection of the force vector and the segment to which it applied, *on the side of the joint axis under consideration.* Any time a vector is applied to a lever (segment), a minimum of two angles are formed. The viewer's eye will automatically tend to find the acute (rather than the obtuse) angle. To identify the angle of application of a force, the angle *on the side of the joint axis* is the angle of interest, regardless of whether that angle is acute (less than 90°) or obtuse (greater than 90°).

Figure 1–59 shows a force vector at three different angles in relation to a segment. We can see in those three depictions how the angle of application affects the size of the moment arm. When the angle of application of a force is small, the moment arm will be small (see Fig. 1–59A). As the angle of application for the force increases, the moment arm increases because the vector lies farther from the joint axis (see Fig. 1–59B). As the angle of application of the force moves beyond 90° (see Fig. 1–59C), the moment arm is measured from the extended tail of the vector; the extended tail swings closer to the joint axis as the angle of application of the force increases beyond 90°. For all forces (internal or external), the moment arm of a force is smallest when the vector is parallel to the segment (whether at 0° or at 180°) and greatest when the vector is perpendicular to the segment.

Figure 1–60 shows the force of gravity (G) with a constant magnitude acting on the forearm-hand segment in four different positions of the elbow joint. Figure 1–61 shows a muscle force (Fms) of constant magnitude acting on the forearm-hand segment at the same elbow-joint angles as shown in Figure 1–60. As the forearm segment rotates

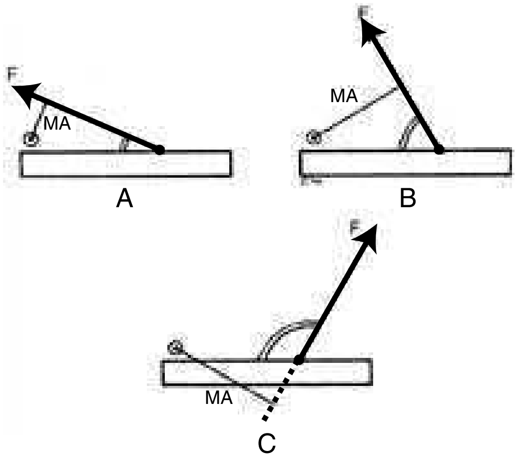

Figure 1–59 Changes in the angle of application of the force (the angle between the force vector and lever *on the side of the axis*) result in changes to the MA of the force, with the MA maximal when the force is at 90° to the lever.

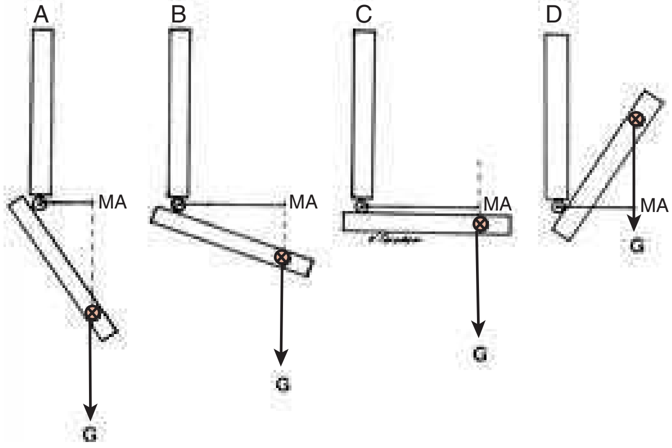

Figure 1–60 Gravity (G) acting on the forearm-hand segment at angles of application of **(A)** 35°, **(B)** 70°, **(C)** 90°, and **(D)** 145° of elbow flexion. The MA of gravity changes with the position of the forearm in space.

around the elbow joint axis, the angles of application for gravity and for Fms change *in relation to the forearm segment.* As the angle of application of the force changes, the moment arm must also change. Although the magnitudes of the gravitational and muscle forces (see Figs. 1–60 and 1–61) do not change from position to position, the *torque* generated by each force changes in direct proportion to the change in length of the moment arm. The moment arms are maximal when the force is applied perpendicular to the segment and at their minimum when the forces lie closest to being parallel to the segment.

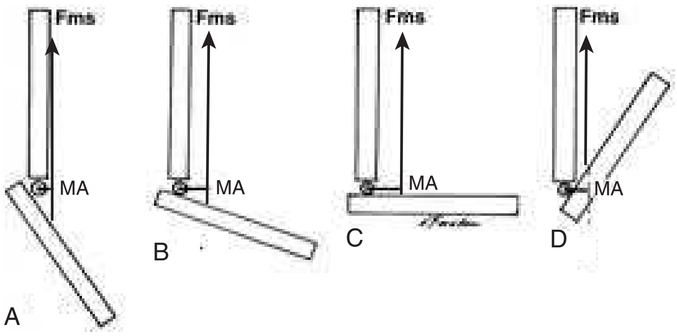

Figure 1–61 A muscle force (Fms) acting on the forearm-hand segment at angles of application of **(A)** 35°, **(B)** 70°, **(C)** 90°, and **(D)** 145°. As the joint crossed by the muscles moves through its range of motion, the MA of the muscle changes.

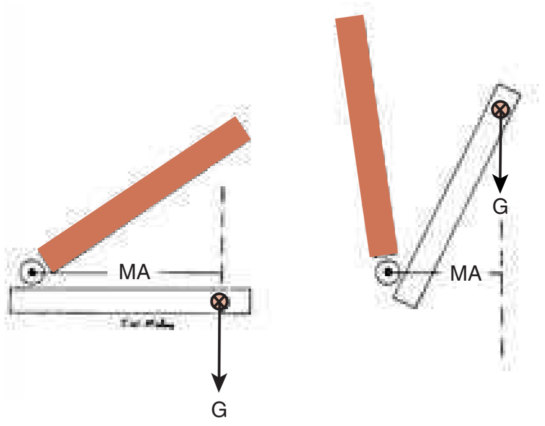

Figure 1–62 The line of gravity changes its angle of application to the forearm-hand segment and its MA when the forearm-hand segment moves in space, although the elbow joint angle remains unchanged.

Although Figures 1–60 and 1–61 show that torques change as a segment moves through a ROM, the basis for the change in angle of application is somewhat different for the gravitational force than for the muscle force. A gravitational force is always vertically downward. Consequently, it is the position of the *segment in space* that causes a change in the angle of application of a gravitational vector, not a change in the joint angle. Figure 1–62 shows the force of gravity (G) applied to the forearm-hand segment at two different orientations in space. Although the elbow joint angle is the same in Figures 1–62A and B, the moment arms are quite different. Because the *magnitude* of the gravitational force does not change as the segment moves through space (as long as its mass is not rearranged), the torque produced by the weight (G) of a segment is directly a function of the position of the segment in space.

Unlike gravity, which has a fixed center of mass in an unsegmented object, other external forces (e.g., a manual resistance, mechanical resistance, or applied load) may be able to change the point of application of the force to different points on the body segment. When the point of application on the segment changes, so too does the moment arm. This was observed in Figure 1–46 as the person moves his hand farther from the door hinge. With the increase in moment arm as the hand moves away from the hinge (the axis), production of the same torque requires less force. The point of application of a gravitational force (center of mass) can be relocated *only* if the segments that compose the object are rearranged. However, such rearrangement of segments often occurs.

Example 1-8

Changing the Gravitation Moment Arm by Changing the Center of Mass

Figure 1–63 shows three graded exercises for the abdominal muscles. The vertebral interspace of L5 to S1 is considered here to be a hypothetical axis about which the head-arms-trunk (HAT) rotates. In each figure, the arms are positioned slightly differently, which results in a rearrangement of mass and a shift in the center of mass. In Figure 1–63A, the arms are raised above the head. As a result, the center of mass of the head-arms-trunk is located closer to the head (cephalad), with a line of gravity that is farther from the axis of rotation (greater moment arm) than when the arms are in the other two positions. The torque generated by gravity is counterclockwise (a trunk extension moment). To maintain this position (rotational equilibrium), the abdominal muscles must generate an equal torque in the opposite direction (an equivalent flexion moment). In Figure 1–63B and C, the centers of mass move caudally as the arms are lowered. The relocation of the center of mass of the head-arms-trunk brings the line of gravity closer to the axis. Because the weight of the upper body does not change when the arms are lowered, the magnitude of torque applied by gravity to the upper body diminishes in proportion to

Continued

the reduction in the moment arm. The decreased gravitational torque requires less opposing torque by the abdominal muscles to maintain equilibrium. Consequently, it is easiest to maintain the position in Figure 1–63C and hardest to maintain the position in Figure 1–63A.

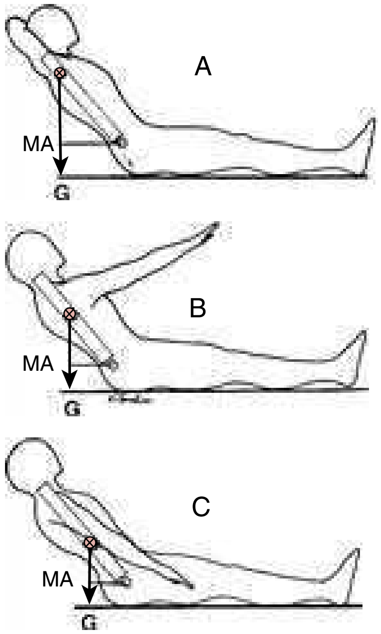

Figure 1–63 Changes in arm position in a sit-up cause the CoM of the upper body segment to move, the MA to change, and the torque of gravity (G) to decrease from **A** to **C.** Although vector G (weight of HAT) varies in length from **A** to **C,** the *magnitude* of G is actually unchanged regardless of arm position.

The angle of application and moment arms of internal forces such as active or passive muscle forces, capsuloligamentous forces, and joint reaction forces—unlike those of external forces such as gravity—are directly affected by the relationship between the two adjacent segments and minimally affected by the position of the segment in space. The angle of application of the muscle force in Figure 1–61 would be the same at each elbow joint angle, regardless of where the adjacent segments are in space (whether you turned the figures right side up, upside down, or sideways). Another distinction to be made between internal forces and most external forces is that the points of application of internal forces typically cannot change but are anatomically fixed. Lastly, the *magnitude* of an internal force (unlike the magnitudes of a gravitational force and many external forces) is rarely consistent as a joint rotates. The magnitude of an internal force is dependent on and responsive to a substantial number of factors (e.g., passive stretch or active contraction) that will be explored further in Chapters 2 and 3.

LEVER SYSTEMS, OR CLASSES OF LEVERS

One perspective used to assess the relative torques of internal and external forces is that of **lever systems,** or **classes of levers.** Although applying the terminology of lever systems to human movement requires some important oversimplifications, the terms (like those of the cardinal planes and axes) provide a useful frame of reference and common language that permit us to break complex kinetics into describable component parts.

A lever is any rigid segment that rotates around a fulcrum. A lever system exists whenever two forces are applied to a lever in a way that produces opposing torques. In order to apply concepts of levers to a bony segment, we must also consider the joint axis of the bony segment to be relatively fixed (a problematic assumption, as we have already discussed). However, it is common to apply the concepts of levers to bony segments when looking at the net rotation produced by (1) a muscle force and (2) a gravitational and/or external force.

In a lever system, the force that is producing the resultant torque (the force acting in the direction of rotation) is called the **effort force (EF).** Because the other force must be creating an opposing torque, it is known as the **resistance force (RF).** Another way to think of effort and resistance forces acting on a lever is that the effort force *is always the winner* in the torque game, and the resistance force *is always the loser* in producing rotation of the segment. The moment arm for the effort force is referred to as the **effort arm (EA),** whereas the moment arm for the resistance force is referred to as the **resistance arm (RA).** Once the effort and resistance forces are identified and labeled, the position of the axis and relative sizes of the effort and resistance arms determine the class of the lever.

A **first-class lever** is a lever system in which the axis lies somewhere between the point of application of the effort force and the point of application of the resistance force (Fig. 1–64). A **second-class lever** is a lever system in which the resistance force has a point of application between the axis and the point of application of the effort force, which always results in the effort arm being larger than the resistance arm (Fig. 1–65). A **third-class lever** is a lever system in which the effort force has a point of application between the axis and the point of application of the resistance force, which always results in the resistance arm being larger than the effort arm (Fig. 1–66).

Muscles in Third-Class Lever Systems

In the human body, a muscle creating joint rotation of its distal segment in the direction of its pull (making the muscle the effort force) is most often part of a third-class lever system. The action of the quadriceps muscle on the leg-foot segment against the resistance of gravity and the weight boot serves as a typical example. Figure 1–67A shows John contracting his quadriceps muscle in the weight boot exercise. If the magnitude of the quadriceps force is at least 3,169 N, the leg-foot

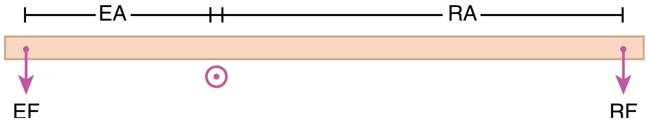

Figure 1–64 In a first-class lever system, the effort arm (EA) may be greater than the resistance arm (RA), smaller than the RA, or equal to the RA, as long as they are on opposite sides of the lever axis.

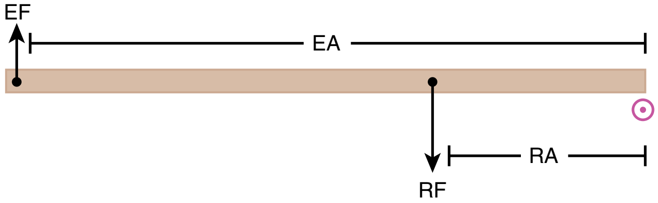

Figure 1–65 A second-class lever system. The effort arm (EA) is always larger than the resistance arm (RA).

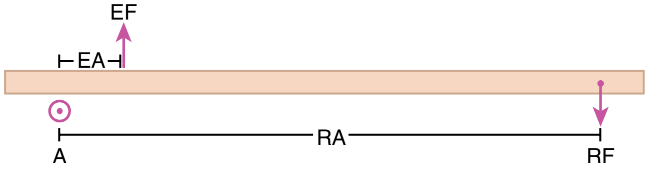

Figure 1–66 A third-class lever system. The effort arm (EA) is always smaller than the resistance arm (RA).

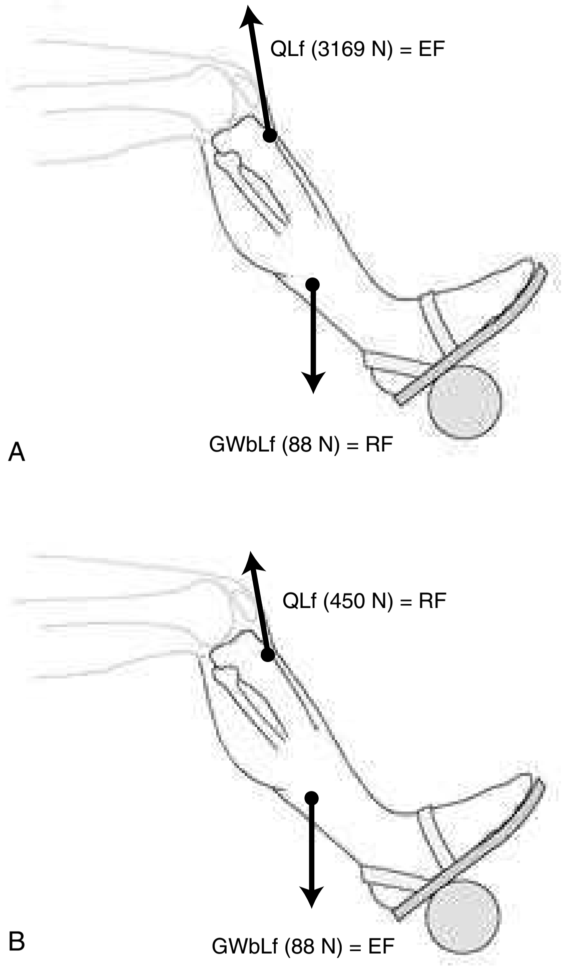

Figure 1–67 **A.** When T_{QLf} is greater than T_{GWbLf}, then QLf is contracting concentrically as the effort force (EF) and GWbLf is the resistance force (RF) in a third-class lever system. **B.** If T_{QLf} is less than T_{GWbLf}, then GWbLf is the effort force (EF) and QLf is contracting eccentrically as the resistance force (RF) in a second-class lever system.

segment will extend (regardless of position) because the quadriceps torque is greater than the gravitational torque. Because the quadriceps force lies closer to the joint axis than does the gravitational force (moment arms are 0.05 m and 0.27 m, respectively), the lever must be third class (EA is less than RA).

The example shown in Figure 1–67A is typical because the point of attachment of a muscle on its distal segment is almost always closer to the joint axis than an external force is likely to be. Consequently, when the muscle is the effort force, the effort arm is likely to be smaller than the resistance arm, with the muscle acting on a third-class lever. It is also important to note that whenever a muscle is the effort force, the muscle must be moving the segment in its direction of pull. This means that the muscle must be performing a shortening contraction, also known as a **concentric contraction.** In fact, any time a muscle is contracting concentrically, the muscle must be the effort force because it is "winning."

Muscles in Second-Class Lever Systems

Muscles most commonly act on second-class levers when gravity or another external force is the effort force ("winner") and the muscle is the resistance force. Figure 1–67B is identical to Figure 1–67A except for the magnitude of the quadriceps force. If the quadriceps exerted a force of 450 N, the net torque would now be in the direction of flexion. If the net torque is in the direction of flexion, gravity/weightboot-on-legfoot would be the effort force and the quadriceps force would become the resistance force. Because the effort arm for the gravity/weightboot-on-legfoot force (0.27 m) is larger than the resistance arm for the quadriceps force (0.05 m), the quadriceps muscle is now working in a second-class lever system. The quadriceps muscle is *still* creating an extension moment, but the net motion is knee flexion, so the muscle must be actively lengthening. Active lengthening of a muscle is referred to as an **eccentric contraction** and always indicates that the muscle is serving as a resistance force. A muscle that is working eccentrically is generally providing *control* (resistance) by minimizing the acceleration produced by the effort force (gravity in this case). In Figure 1–67B, the knee joint is flexing despite the fact that there are no active *knee joint flexors.* In fact, the only active muscle is a knee extensor! Therefore, an understanding of the muscles and other forces involved in any movement can be achieved only

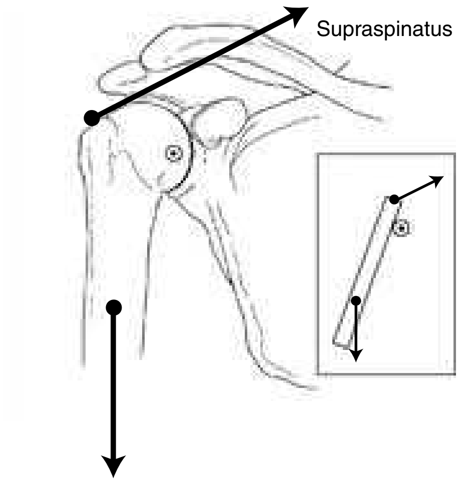

Figure 1–68 The supraspinatus is attached to the humerus on one side of the joint axis, and the point of application of gravity (CoM) lies on the other side of the axis (inset). Regardless of whether the muscle is working concentrically (EF) or eccentrically (RF), the muscle is part of a first-class lever.

through a kinetic analysis of the motion and not simply from a description of the location, direction, or magnitude of the motion.

Muscles in First-Class Lever Systems

There are a limited number of muscles in the human body that work in first-class lever systems because the point of application of the muscle must be on the opposite side of the joint axis from the external force. This is infrequently the case. One example of a muscle working on a first-class lever is the pull of the supraspinatus on the humerus (Fig. 1–68). The attachment of the supraspinatus on the greater tubercle of the humerus is on the opposite side of the composite axis of rotation for the glenohumeral joint from the center of mass of the upper extremity, which is just above the elbow (see inset). Because the muscle and the gravitational force lie on either side of the joint axis, this remains a first-class lever whether the supraspinatus is contracting concentrically (as the effort force) or eccentrically (as the resistance force).

For second- and third-class levers, the classification of a lever is often dependent on whether the muscle is doing a concentric contraction (as the effort force) or an eccentric contraction (as the resistance force). When a muscle is performing an **isometric contraction,** the muscle length is effectively unchanged and the lever must be in rotational equilibrium (the segment is not rotating). When a muscle is acting on a lever in equilibrium, it is common to designate the active muscle as the effort force. The designation of the active muscle as the effort force in an equilibrium situation makes conceptual sense because the muscle contraction uses energy (requires "effort"). However, the effort force and resistance force labels are arbitrary in a lever that is in rotational equilibrium and could just as easily be reversed. It is also true that a muscle uses energy (requires "effort" to resist a force during an eccentric contraction), and so designation of the effort force on the basis of energy consumption alone cannot be done without violating the principles that we have established.

Concept Cornerstone **1-15**

Muscles in Lever Systems

- When a muscle is contracting concentrically (actively shortening), the muscle must be moving the segment to which it is attached in the direction of its pull. Therefore, the muscle will be the effort force.
- When a muscle is contracting eccentrically (actively lengthening), the muscle must be acting in a direction opposite to the motion of the segment; that is, the muscle must be the resistance force. When a muscle is contracting eccentrically, it generally serves to *control* (slow down) the acceleration of the segment produced by the effort force.
- When a lever is in rotational equilibrium, the muscle acting on the lever is contracting isometrically. In such a case, labeling the muscle as the effort or resistance force is arbitrary.

Mechanical Advantage

Mechanical advantage (M Ad) is a measure of the mechanical efficiency of the lever system (the relative effectiveness of the effort force in comparison with the resistance force). Mechanical advantage is related to the classification of a lever and provides an understanding of the relationship between the torque of an external force (which we can roughly estimate) and the torque of a muscular force (which we can estimate only in relation to the external torque). Mechanical advantage of a lever is the ratio of the effort arm (moment arm of the effort force) to the resistance arm (moment arm of the resistance force), or:

$$\text{M Ad} = \frac{\text{EA}}{\text{RA}}$$

When the effort arm is larger than the resistance arm, the mechanical advantage will be greater than one. When the mechanical advantage is greater than one, the magnitude of the effort force working through a larger moment arm can be smaller than the magnitude of the resistance force, yet still create greater torque to "win." The "advantage" is that a small force can defeat a larger force. In the example shown in Figure 1–67B, the effort force is GWbLf with an effort arm of 0.27 m. The resistance force is the quadriceps force with a resistance arm of 0.05 m. Therefore, for the freeze-frame in Figure 1–67B:

$$\text{M Ad} = 0.27\text{ m} \div 0.05\text{ m} = 5.4$$

In this second-class lever system, the effort force is mechanically efficient because the 88 N force creates more torque than the 450 N force of the quadriceps resistance.

Continuing Exploration 1-12:

Mechanical Advantage and the Effort Force

The mechanical advantage of a lever is determined by the lengths of the moment arms and not by the *magnitudes* of the effort and resistance forces. An effort force with a magnitude smaller than the resistance force *must* be working through a larger moment arm; otherwise, it cannot produce a larger torque (cannot be the "winner"). However, the effort force is *not necessarily* smaller than the resistance force. If we added 400 N to John's weight boot in Figure 1–67B, the net gravitational force (now 488 N) would still be the effort force (although larger in magnitude than the 450 N quadriceps force) and the mechanical advantage would still be 5.4. However, the angular acceleration of the flexing leg-foot segment would be extremely large because there would be a large increase in the net unbalanced torque. The "advantage" remains with the effort force.

In third-class levers, the mechanical advantage will always be less than one because the effort arm is always smaller than the resistance arm (the effort force lies closer to the axis than the resistance force). A third-class lever is "mechanically inefficient" or is working at a "disadvantage" because the magnitude of the effort force *must* always be *greater* than the magnitude of the resistance force in order for the torque of the effort force to exceed the torque of the resistance force (as it must for the force to "win"). Similarly, because the distal attachment of a muscle tends to be closer to the joint axis than is the point of application of an external force even when muscles are working on first-class levers, muscles working in first-class lever systems (like those in third-class systems) tend to be at a mechanical *disadvantage.*

Trade-Offs of Mechanical Advantage

It has already been observed that the majority of the muscles in the human body, when contracting concentrically with the distal lever as the moving segment, work with a shorter moment arm than does the external force. To rotate a lever, a muscle typically must exert a proportionally larger force to produce a "winning" torque. It appears, then, that the human body is structured inefficiently. In fact, the muscles of the body are structured to take on the burden of "mechanical disadvantage" to achieve the goal of rotating the segment through space.

Figure 1–69A shows the forearm-hand segment being flexed (rotated counterclockwise) through space by a concentrically contracting muscle (Fms) against the resistance of gravity (G). Consequently, this is a third-class lever because the effort arm (muscle moment arm) is smaller than the resistance arm (gravitational moment arm). The magnitude of the muscle force must be much larger than the magnitude of gravity for the muscle to "win," and so the lever is, indeed, mechanically inefficient. However, as the muscle pulls its point of application (on the proximal forearm-hand segment) through a very small arc, the distal portion of the segment is displaced through a much greater arc. Although the magnitude of force needed to create the rotation is large in comparison with the magnitude of the resistance force, the result is that linear muscle displacement and velocity are small compared to the linear displacement and velocity of the segment's more distal components. Because one of the goals of human function is to maximize angular displacement of a distal segment through space while minimizing muscle length and muscle velocity changes, third-class lever systems are very common.

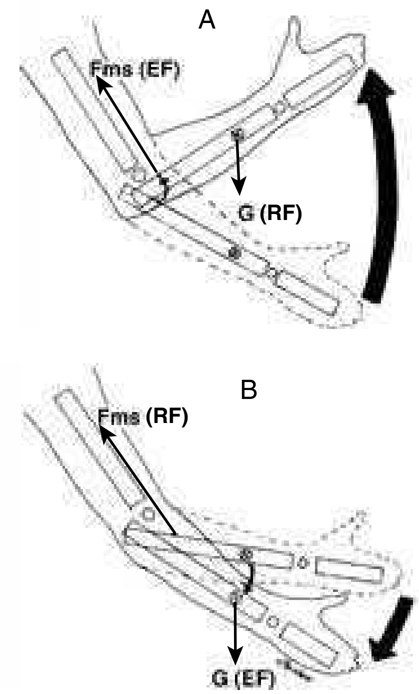

Figure 1–69 **A.** In a mechanically inefficient third-class lever, movement of the point of application of EF (Fms) through a small arc produces a large arc of movement of the lever distally. **B.** In a mechanically efficient second-class lever, movement of the point of application of EF (G) through a small arc produces little increase in the arc distally.

In second-class levers in the human body, the effort force is usually (but not always) the external force. Although the effort force in a second-class lever can be smaller in magnitude than the resistance force (and still "win"), less is gained in angular displacement and velocity at the distal end of the segment (per unit displacement of the effort force). In Figure 1–69B, a small arc of movement at the point of application of the effort force (G) results in only a small increase in angular displacement of the more distal segment. In any second-class lever (and in a first-class lever in which the effort arm is greater than the resistance arm), the lever is mechanically efficient in terms of the ratio of force output to torque production, but relatively less is gained in terms of angular displacement of the distal end of the segment through space.

Concept Cornerstone **1-16**

Mechanical Advantage and Classes of Levers

- In all second-class levers, the mechanical advantage (M Ad) of the lever will always be greater than one. The magnitude of the effort force can be (but is not necessarily) less than the magnitude of the resistance.
- In all third-class levers, the mechanical advantage of the lever will always be less than one. The magnitude of the effort force *must* be *greater* than the magnitude of the resistance for the effort to produce greater torque.
- The mechanical advantage of a first-class lever can be greater than, less than, or equal to one. However, it is often true of first-class levers in the body that the moment arm of the muscle will be shorter than the moment arm of the external force.
- When the muscle is the effort force in a lever with a mechanical advantage of less than one, the necessary expenditure of energy to produce sufficient muscle force to "win" is offset by the need for minimal linear displacement and velocity of the muscle to produce proportionally greater angular displacement and angular velocity of the distal portions of the segment.
- When an external force is the effort force in a lever with a mechanical advantage greater than one (e.g., a second-class lever), the magnitude of the effort force can be small in comparison with the resistance force, but less is gained in angular displacement and velocity.

We have identified that most muscles have a moment arm that is smaller than that of the external force against which they are working because most muscles lie closer to joint axes than do external forces. Consequently, the muscle must be able to create large forces not only to "win" (work concentrically as the effort force) but also to control external forces eccentrically as the resistance force. As shall be shown in Chapter 3, the muscle is structured to optimize production of the large forces required to produce large torques as either effort or resistance forces.

Limitations of Analysis of Forces by Lever Systems

Although the conceptual framework of lever systems described here provides useful terms and some additional insights into rotation of segments and muscle function, there are distinct limitations to this approach. Our discussion of lever systems ignored the established fact that the rotation of a lever requires at least one force couple. An effort force and a resistance force are *not* a force couple because effort and resistance forces produce rotation in the opposite (rather than the same) direction. Consequently, in an analysis of a simple two-force lever system that includes an effort force and a resistance force, at least one force that forms the second part of the force couple for the effort force is missing. The second force in the "couple" is generally an articular constraint (a joint reaction force or capsuloligamentous force) that may serve as the pivot point for the rotation (see Figs. 1–45 and 1–51). Consequently, a simple lever-system approach to analyzing human motion requires oversimplification that fails to take into consideration key elements that affect function and structural integrity. Torques on human segments are not simply produced by muscles and external forces; they result from both additional internal forces *and* the vertical and horizontal forces produced by muscles and external forces that are often ignored in a simple lever approach.

FORCE COMPONENTS

In the analysis of John's leg-foot segment in Figure 1–56, only the *torques* produced by the forces applied to the segment were considered. The substantial vertical gravity/weightboot-on-legfoot force was ignored in that analysis because the force produced very little torque. In identifying the quadriceps force as the force that created the rotation of the leg-foot segment, we did not identify the other force (the second part of the force couple) required to produce an extension torque. We also did not identify the mechanism by which translatory equilibrium ($\Sigma F_V = 0$ and $\Sigma F_H = 0$) of the leg-foot segment was established—a necessary condition for rotation of a joint around a relatively fixed axis. To examine the degree to which these conditions are met (net extension torque and translatory equilibrium), we need to understand how to resolve forces into their component parts.

A force applied to a lever produces its greatest torque when the force is applied at 90° to the lever (presuming there is a second part to the force couple). If the *same* magnitude of force produces *less* torque when the angle of application is not 90°, some of that force must be doing something other than producing rotation. Torque, in fact, is typically considered to be produced only by that *portion* of the force that is directed toward rotation. When the force is applied to a lever at some other angle (greater than 90° or less than 90°), the component of force that *is* applied at 90° to the lever will contribute to rotation. Consequently, the portion of a force that is applied at 90° to the segment is known as the **perpendicular** (**rotary** or **y**) **component** of the force **(Fy).** The rotary component is the "y" component because the long axis of the body segment is usually the reference line or essentially the x-axis. Consequently, the component that is perpendicular to the segment is the y-axis.

The magnitude of Fy can be found graphically by the process of **resolution of forces.** In resolution of forces, the original (or **total**) force **(F_{TOT})** is broken down into two components. Just as two concurrent forces can be composed into a single resultant vector, a single vector (F_{TOT}) can be resolved into two concurrent components. In this instance, the components will be specifically constructed so that one component (Fy) lies perpendicular to the segment. The second component is the so-called **parallel** (**translatory,** or **x**) **component** and is drawn parallel to the lever. The abbreviation **Fx** is used because, again, the body segment is always the reference or x-axis, and a line drawn parallel to the segment will be along or parallel to that x-axis. The process of resolution of forces is essentially the reverse of composition of forces by parallelogram. With resolution of forces, the parallelogram will always be a rectangle because the sides (Fx and Fy) are perpendicular to each other (parallel and perpendicular to the bony segment, respectively).

Resolving Forces Into Perpendicular and Parallel Components

In Figure 1–70, three steps are shown to resolve the quadriceps force into its perpendicular (Fy_{QLf}) and parallel (Fx_{QLf}) components:

Step 1 (see Fig. 1–70A). A line with the *same point of application* as the original force is drawn *perpendicular* to the long axis of the lever in the direction of the original force (this is the draft line of component Fy). A second line with the *same point of application* as the original force is drawn *parallel* to the long axis of the lever in the direction of the original force (this is the draft line of component Fx). The draft Fx and Fy component lines should reach or go past the arrowhead of the original vector.

Step 2 (see Fig. 1–70B). The rectangle is completed (closed) by drawing (from the arrowhead of the F_{TOT} vector) lines that are parallel to each of the draft Fx and Fy components.

Step 3 (see Fig. 1–70C). All the lines are "trimmed," leaving only the completed rectangle. Components Fy (the rotary component) and Fx (the translatory component) each form one side of the rectangle, have a common point of application, and have arrowheads at the corners of the rectangle; force QLf (F_{TOT}) is now the diagonal of the rectangle. It is important that the lengths of vectors Fy and Fx are "trimmed" to the confines of the rectangle to maintain the proportional relation among Fx, Fy, and F_{TOT}. This proportional relation will permit determination of the predominant and relative effects of F_{TOT}.

Perpendicular and Parallel Force Effects

Once the quadriceps force is resolved into its components, it will be more evident that components Fy_{QLf} and Fx_{QLf} (see Fig. 1–70C) have the potential to create three different motions of the leg-foot segment: vertical

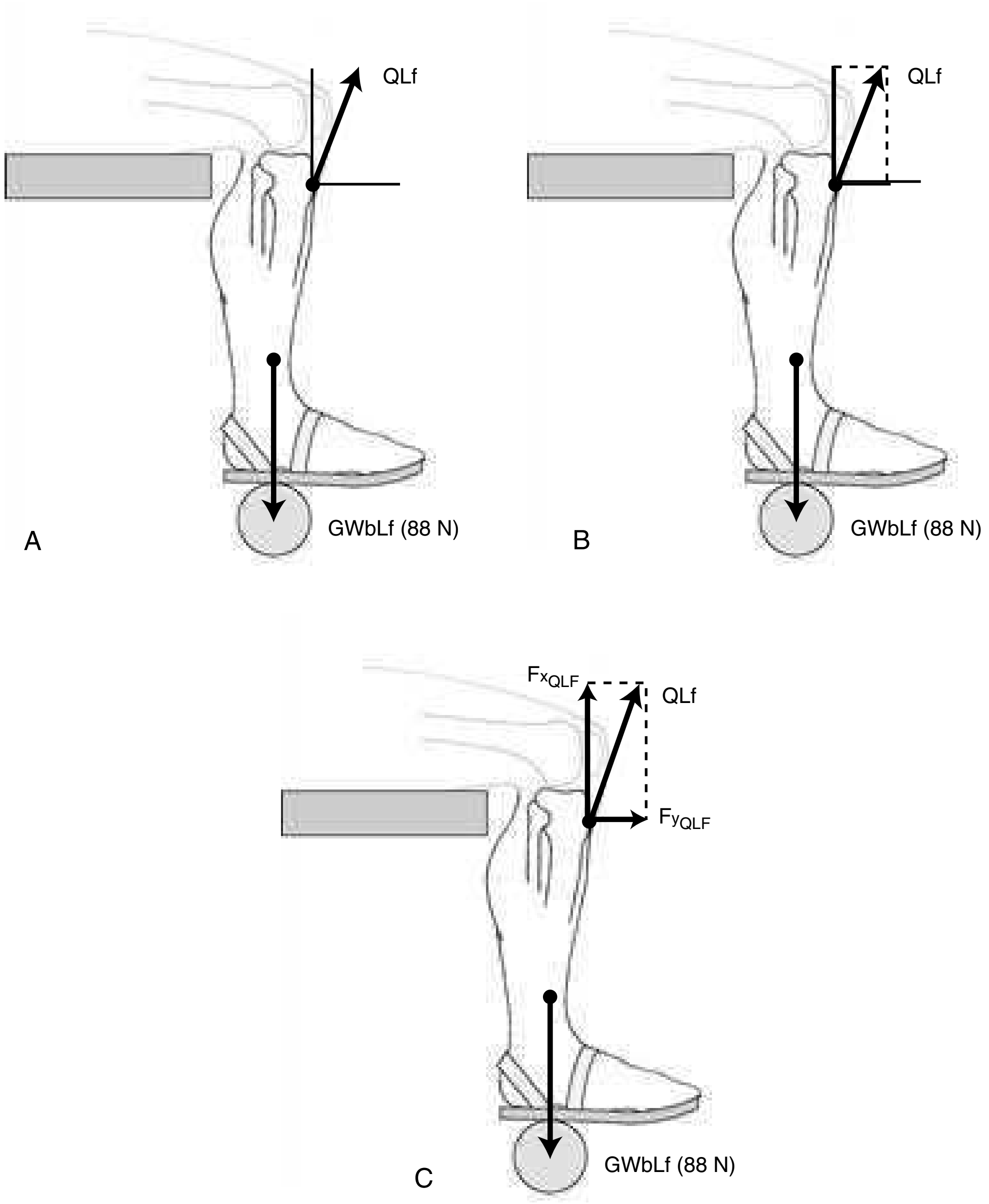

Figure 1–70 When a force is resolved into its components, **(A)** two lines that are perpendicular and parallel to the long axis of the bone are drawn from the point of application of the original force; **(B)** a rectangle is completed by drawing parallel lines from the arrowhead of the original force; and **(C)** the perpendicular (Fy) and parallel (Fx) component vectors are the adjacent sides of the rectangle that have a common point of application with the original force.

motion, horizontal motion, and rotary motion. Component Fx will tend to create vertical translatory motion in Figure 1–70C. However, component Fy will both create rotation and tend to create horizontal translation. Because a force component (e.g., Fy) may create both rotation and translation, labeling components as "rotary" and "translatory" can be confusing. We will, therefore, proceed in the remainder of this chapter to refer to Fy exclusively as the perpendicular component and to Fx exclusively as the parallel component, with their effects (rotation or translation) determined by the situation.

We have already established that determining the state of motion of a segment requires assessment of ΣF_V, ΣF_H, and $\Sigma\tau$. We have also established that the Fx and Fy components of a force will tend to create both translatory (F_V and F_H) and rotary (τ) motion. In order to reduce the number of terms, consider that a force that is both perpendicular to the segment and horizontally oriented in space (e.g., Fy_{QLf} in Figure 1–70C) may become vertically oriented in space, although still remaining perpendicular to the segment, if the segment moves in space. The same shift in spatial orientation may happen with an Fx component. Consequently, we will assess translatory equilibrium of a limb by considering the Fx and the Fy components (that have a fixed relation to the limb) rather than labeling them F_V or F_H components (which vary with the orientation of the limb in space). The rotary equilibrium of the body segment will be assessed by determining the sums of the torques contributed by the forces (or force components) that are perpendicular to the long axis of the segment ($\Sigma\tau$).

Determining Magnitudes of Component Forces

In a figure drawn to scale, the relative vector lengths can be used to ascertain the net unbalanced forces. Because the vectors are not drawn to scale in Figure 1–70C, the relative magnitudes of Fy and Fx must be determined by using trigonometric functions of sines (sin) and cosines (cos) that are based on fixed relationships for right (90°) triangles.

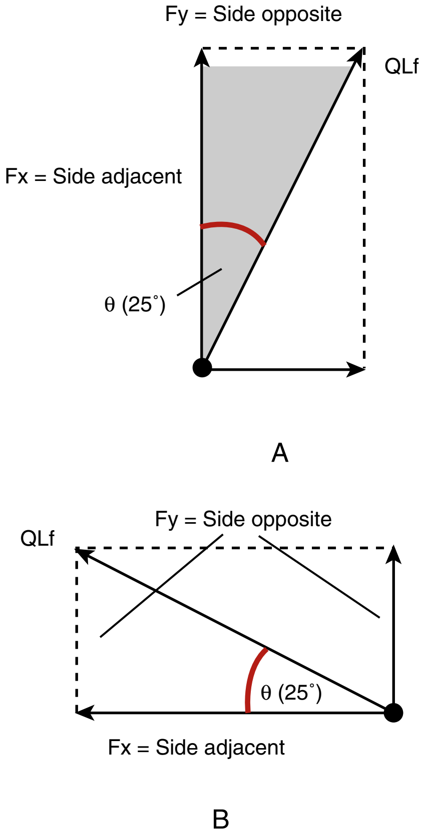

Figure 1–71 **A.** Vector QLf and its Fx and Fy components are replicated (although enlarged) in the same orientation shown in Figure 1–70C. The reference triangle (shaded) shows that QLf is the hypotenuse, Fx is the side adjacent to angle θ, and Fy (or its equivalent length) is the side opposite to angle θ. **B.** The same figure is reoriented in space to provide an alternative visualization.

It must first be recognized that Fx and Fy are always part of a right triangle in relation to F_{TOT}. In Figure 1–71A, the quadriceps vector (QLf) and its components have been pulled out and enlarged. As the diagonal of a rectangle, QLf (F_{TOT}) divides the rectangle into two right triangles. The shaded half of the rectangle is the triangle of interest because the angle of application (as previously defined) is the angle between the vector and the segment on the side of the knee joint (θ in Fig. 1–71A). In the shaded triangle, vector QLf is the hypotenuse (side opposite the 90° angle) and is now assigned a magnitude of 1,000 N. The angle of application θ is, in this example, presumed to be 25°. Vector Fx is the side adjacent to angle θ, and vector Fy is the side opposite angle θ. Because the orientation of the triangle in Figure 1–71A is visually a little different from what the reader might be used to (given the position of the leg-foot segment in Fig. 1–70), the force components have been replicated in a more "typical" orientation in Figure 1–71B. It should be noted that the "side opposite" the angle θ is not *literally* the perpendicular component (Fy) as we initially labeled it but is identical in magnitude (see Fig. 1–71B) because these are opposite sides of a rectangle and, therefore, are equivalent in length.

Simple trigonometry is now used to solve for the magnitudes of Fx and Fy, where the angle of application θ is given as 25° and QLf (F_{TOT}) is assigned a value of 1,000 N. The magnitudes of Fx_{QLf} and Fy_{QLf} can now be computed (see Continuing Exploration: Trigonometric Resolution):

$$Fx_{QLf} = (\cos 25°)(1{,}000\ N) = (0.91)(1{,}000\ N) = 910\ N$$
$$Fy_{QLf} = (\sin 25°)(1{,}000\ N) = (0.42)(1{,}000\ N) = 420\ N$$

It should be noted that the sum of the magnitudes of the Fx and Fy components will always be greater than the magnitude of the resultant force (F_{TOT}). As with composition of forces, the resultant is more "efficient." However, analysis of the translatory or rotary effects of any set of forces will produce mathematically equivalent results whether the total force or the force components are used in appropriate analyses.

Continuing Exploration 1-13:

Trigonometric Resolution of Forces

The relation between the lengths of the three sides in a right (90°) triangle is given in the Pythagorean theorem: $A^2 + B^2 = C^2$, where C is the length of the hypotenuse of the triangle (the side opposite the 90° angle) and A and B are, respectively, the lengths of the sides adjacent to and opposite the angle θ (Fig. 1–72A). This equation is useful when the lengths (magnitudes) of two of the three sides are known. However, in resolution of forces, we often know the length of the hypotenuse and one angle. In these cases, the sine and cosine rules allow the unknown lengths in the triangle to be computed:

$$\sin \theta = \frac{\text{side opposite}}{\text{hypotenuse}}$$

$$\cos \theta = \frac{\text{side adjacent}}{\text{hypotenuse}}$$

These formulas may be used to solve for the length of the adjacent side (Fx) or opposite side (Fy) when angle θ (the angle of application of the force) and the hypotenuse (the magnitude of the force) are known:

$$\text{side opposite} = (\sin \theta)(\text{hypotenuse})$$
$$\text{side adjacent} = (\cos \theta)(\text{hypotenuse})$$

The values of the sine and cosine functions in trigonometry are not arbitrary numbers. The sine and cosine values represent that fact that there is a fixed relationship between the lengths of the sides for a given angle. In Figure 1–72B, a right triangle with an angle θ of 25° is drawn. The triangle is divided into three triangles of different sizes; the hypotenuses are assigned scaled values of 10 cm, 15 cm, and 20 cm for the smallest to largest triangles, respectively. On the same scale, the values of Fy (side opposite) will be 4.2 cm, 6.3 cm, and 8.5 cm, respectively; the values of Fx (side adjacent) will be 9.1 cm, 13.6 cm, and 18.1 cm, respectively. The ratio of any two sides of one of these 25° triangles will be the same, regardless of size, and *that ratio will be the value of the trigonometric function for that angle*.

1. For the (side opposite/hypotenuse) for each of the three triangles: (4.2/10 = 0.42); (6.3/15 = 0.42); and (8.5/20 = 0.42). The value of sin 25° is 0.42.
2. For the (side adjacent/hypotenuse) for each of the three triangles: (9.1/10 = 0.91); (13.6/15 = 0.91); and (18.1/20 = 0.91). The value of cos 25° is 0.91.
3. For the (side opposite/side adjacent) for each of the three right triangles: (4.2/9.1 = 0.46); (6.3/13.6 = 0.46); and (8.5/18.1 = 0.46). The value of the tangent (tan, or sin/cos) 25° is 0.46.

This is the "proof" that, for a given angle of application of F_{TOT}, Fx and Fy have a fixed proportional relationship to F_{TOT} *and* a fixed relationship to each other, regardless of the magnitude of F_{TOT} (and regardless of the orientation of the segment in space or whether the force is internal or external).

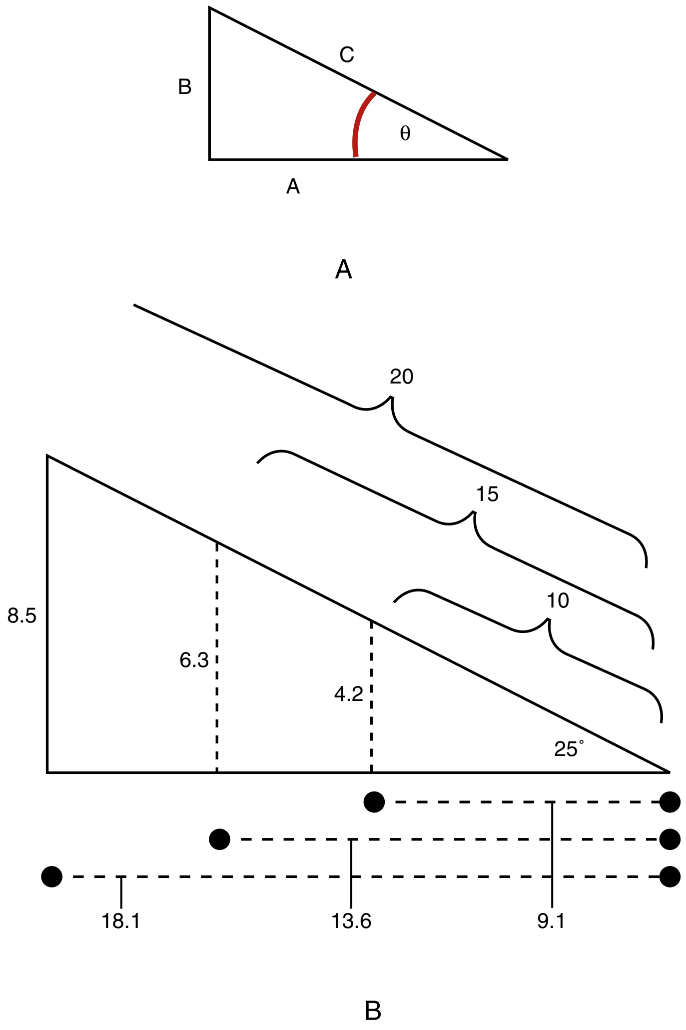

Figure 1–72 **A.** The Pythagorean theorem states that $A^2 + B^2 = C^2$, where A, B, and C are the lengths of the sides of a right triangle. **B.** For a given angle θ (e.g., 25°), the lengths of the side opposite and side adjacent to the angle will have a fixed relationship to each other and to the original force, regardless of the size of the triangle.

Concept Cornerstone **1-17**

Angles of Application Greater Than 90°

We have identified that the magnitudes of Fx and Fy are calculated by using the angle of application of the F_{TOT}. However, there is a variation of this rule. When the angle of application of F_{TOT} is greater than 90° (an obtuse angle), as in the schematic in Figure 1–73, the complement of θ ($\theta^1 = 180° - \theta$) must be used. This makes sense both in looking at Figure 1–73 and because none of the three angles in a right triangle can be greater than 90°. Note in Figure 1–73, however, that Fx remains the side adjacent to θ^1 and that the magnitude of Fy remains the side opposite to θ^1. Figure 1-74 shows gravity (G) acting on the leg-foot segment at 45° (see Fig. 1–74A) and at 135° (see Fig. 1–74B). The magnitudes of Fx and Fy are identical in both figures because both the sides of the rectangle (in this case, a square) are based on a 45° angle. The angle used to compute the components for the force applied at 135° is the complement of the angle (180° – 135°, or 45°).

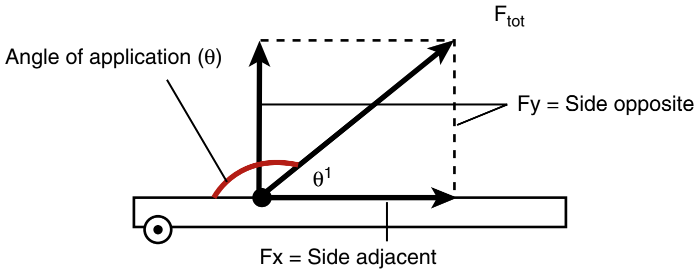

Figure 1–73 When the angle of application (θ) is greater than 90° (an obtuse angle), the angle (θ^1) used to compute Fx and Fy will be the complement (180° – θ) of the angle of application.

It may seem like the definition of angle of application of a vector should be changed to define the *acute angle* between the segment and the vector, rather than the *angle on the side of the joint axis*. However, allowing the angle of application (θ) to be any size from 0° to 180° permits a useful general statement to be made: Whenever the angle of application of a force is less than 90°, the Fx component will be directed toward the joint axis and will be a compressive force (see Fig. 1–74A). Whenever the angle of application is greater than 90°, the Fx component will be a distractive force (see Fig. 1–74B).

Force Components and the Angle of Application of the Force

When a force of constant magnitude is applied to a segment as the segment rotates around its joint axis (see Figs. 1–60 and 1–61), there is a change in the moment arm and, therefore, in the torque produced by the force at different joint angles. The change in the moment arm is a function of the change in angle of application of the force to the segment. Because the perpendicular component (Fy) of a force is effectively the portion of the total force that produces torque, a change in torque produced by a force of constant magnitude must mean that the magnitude of Fy is changing. This is quite logical because it has just been established that the magnitude of Fy is a function of the angle of application of the force [Fy = (sin θ) (hypotenuse)].

Figure 1–75 shows a muscle force (Fms) of constant magnitude acting on the forearm-hand segment at the same four positions of the elbow (and the same angles of application for the force) that were shown in Figure 1–61. In Figure 1–75, the changes in the magnitudes of the Fx and Fy components of the force are shown, rather than the changes in the moment arms. Given that the changes in torque produced by Fms were a function of the changes in moment arm in Figure 1–61 and a function of the changes in angle of application or Fy in Figure 1–75, moment arm and Fy must be directly proportional to each other. We have established that when a force is applied at 90° to the segment (e.g., Fig. 1–75C), the moment arm is as large as it can be for this force. When a force is applied at 90° to the segment, Fy is equivalent in magnitude to the total force (F_{TOT}) and is, therefore, as large as it can be. Consequently, both moment arm and Fy have their greatest magnitude

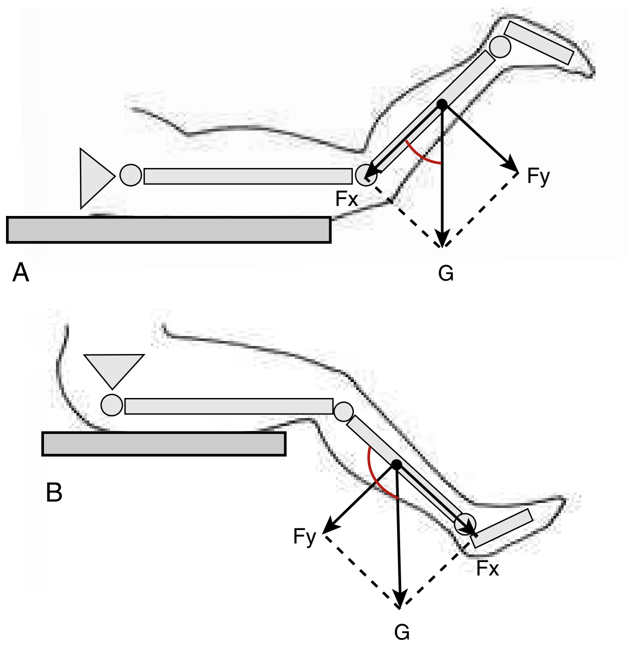

Figure 1–74 Vector G is applied to the leg-foot segment at **(A)** 45° and at **(B)** 135°. The magnitudes of Fx and Fy are the same in both **A** and **B** because the angle used to compute the components for a force applied at 135° is the complement of the angle (180° – 135°) or 45°. However, Fx is compressive in **A** and distractive in **B.**

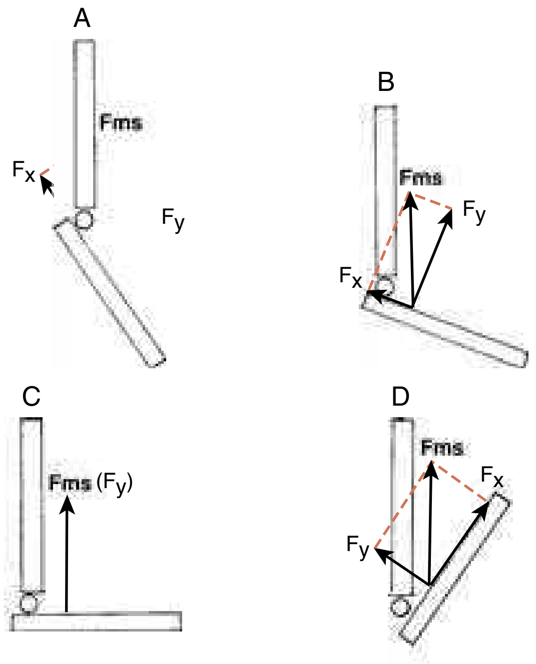

Figure 1–75 Resolution of the muscle force (Fms) into perpendicular (Fy) and parallel (Fx) components at angles of application of **(A)** 35°, **(B)** 70°, **(C)** 90°, and **(D)** 145° of elbow flexion produces changes in the magnitudes of the components and in the direction of Fx.

when a force is applied at 90° to a segment. Importantly, there will also be a proportional decrease in Fx as Fy increases because Fx is inversely proportional to Fy.

The change of the parallel (Fx) component from compression to distraction shown in Figure 1–75D is unusual for a muscle force. In fact, the majority of muscles lie nearly parallel to the segment, have relatively small angles of pull (Fx is greater than Fy) regardless of the position in the joint ROM, and almost always pull in the direction of the joint axis. The effect of this arrangement of muscles is that a muscle force generally has a relatively small Fy component contributing to rotation, with a larger Fx component that is nearly always compressive. Therefore, most of the force generated by a muscle contributes to joint *compression*, rather than joint *rotation*! This arrangement enhances joint stability but means that a muscle must generate a large total force to produce the sufficient torque to move the lever through space.

Concept Cornerstone **1-18**

Components of Muscle Forces

- The angle of pull of the majority of muscles is small, with an action line more parallel to the bony lever than perpendicular to the bony lever.
- The parallel (Fx) component of a muscle force most often is larger than the perpendicular (Fy) component.
- The parallel component of most muscle forces contributes to joint compression, making muscles important joint stabilizers.

The constraints that exist on muscle forces in the body (and, therefore, the generalizations identified in Concept Cornerstone 1-18) do not apply to external forces. We have already seen examples in which gravity is compressive in one instance or distractive in another, depending on the location of the segment in space (see Fig. 1–74). Gravity is constrained in its direction (always vertically downward) and, therefore, has some predictability (the torque of gravity is always greatest with the limb segment is parallel to the ground). Other external forces (e.g., a manual resistance) have few if any constraints and, therefore, most often do not have predictable effects on a segment. However, the *principles* of forces in relation to angle of application and the consequential magnitudes of the moment arm, Fy components, and Fx components apply to any and all forces, including those commonly encountered in clinical situations.

Example 1-9

Manipulating External Forces to Maximize Torque

In Figure 1–76, a manual external force (hand-on-legfoot [HLf]) of the same magnitude is applied to a leg-foot segment in two different positions. If the goal is to obtain a maximum *isometric* quadriceps muscle contraction ($\Sigma\tau = 0$) with minimum effort on the part of the person applying the resistance, the position of the manual force in Figure 1–76B provides a distinct advantage. The force hand-on-legfoot in Figure 1–76A has a substantially smaller moment arm than that in Figure 1–76B because the resisting hand is placed more proximally on the leg-foot segment. Hand-on-legfoot is also applied at an angle to the segment in Figure 1–76A that leads to a potentially undesirable parallel component (Fx). By moving the resisting hand down the leg-foot segment and changing the angle of application of the force to 90° to the segment as in Figure 1–76B, the moment arm is maximized and all the force from the hand contributes to rotation (HLf = Fy). In Figure 1–76B, the muscle must offset the greater torque produced by the resisting hand. Because the joint angle and angle of pull of the muscle has not changed, the muscle can change its torque only by changing its force of contraction. An understanding of the ability to manipulate both moment arm and angle of application by the person providing the resistance will allow that person to either increase or decrease the challenge to the quadriceps muscle, depending on the goal of the exercise.

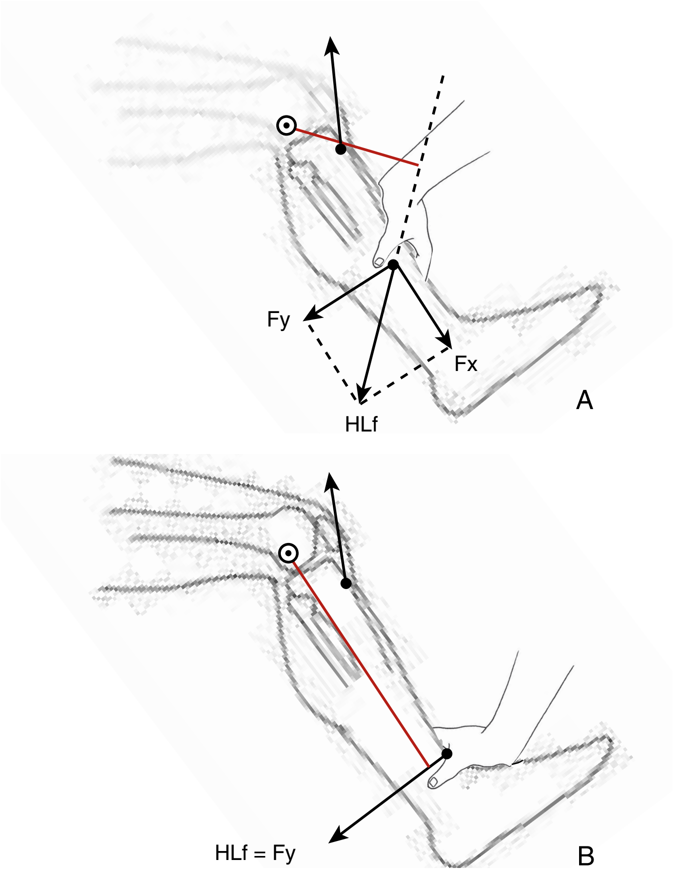

Figure 1–76 **A.** A manual force is applied at an angle to the leg-foot segment. **B.** A manual resistance of the same magnitude produces substantially greater torque because all the force is directed toward rotation (Fy) and the point of application is farther from the joint axis.

Concept Cornerstone **1-19**

Manipulating External Forces to Maximize Torque Production

- The torque of an external force can be increased by increasing the magnitude of the applied force.
- The torque of an external force can be increased by applying the force perpendicular to (or closer to perpendicular to) the lever.
- The torque of an external force can be increased by increasing the distance of the point of application of the force from the joint axis.

Translatory Effects of Force Components

Let us return once again to John and the weight boot exercise. The goal as John attempts to lift the weight boot (and the goal in all purposeful joint motions) is to have the segment in translatory equilibrium ($\Sigma Fx = 0$; $\Sigma Fy = 0$) while having a net torque in the direction of desired motion. Before the torque can rotate the segment, however, we must identify the translatory effects of the forces already applied to the leg-foot segment, as well as determine what additional forces, if any, are necessary to create translatory equilibrium.

In Figure 1–77, the force gravity/weightboot-on-legfoot (GWbLf) (–88 N) and the Fx component of the quadriceps (Fx_{QLf}) (+910 N) are both parallel to the leg-foot segment and in opposite directions. Gravity/weightboot-on-legfoot is a joint distraction force because it is away from the joint, whereas vector Fx_{QLf} is a joint compression force because it is toward the joint. Given the magnitudes of GWbLf and Fx_{QLf}, there appears to be a net unbalanced force parallel to the leg-foot segment, or a net compression force of +822 N. As is true whenever there is a net compression force, the segment will translate until it contacts the adjacent segment (femur), at which point a new force (femur-on-legfoot [FLf]) is introduced. When femur-on-legfoot reaches a magnitude of 822 N, the leg-foot segment will reach translatory equilibrium parallel to its long axis.

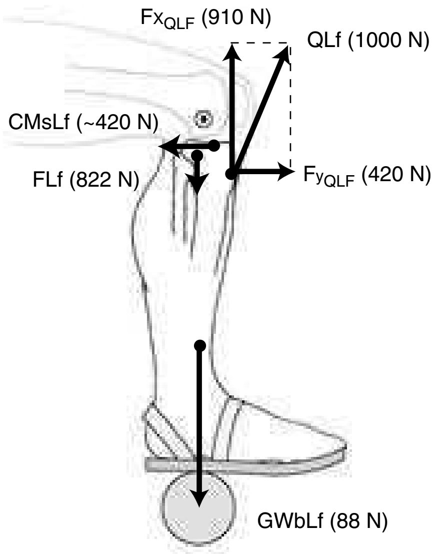

Figure 1–77 The leg-foot segment in the weight boot exercise with all forces and force components identified for 90° of knee flexion as knee extension is initiated.

The Fy component of the quadriceps (Fy_{QLf}) has a magnitude of 420 N to the right (see Fig. 1–77). There must be another force (or forces) of 420 N to the left before Fy_{QLf} can cause torque rather than translation. The force will not come from bony contact in this example because of the shape of the articular surfaces; rather, it would presumably come from one or more internal forces because there is nothing else "touching" the leg-foot segment that would produce an external force. The most obvious source of the internal force is capsuloligamentous tension. It is possible that muscles such as the hamstrings may also be contributing. Although it is unlikely that posterior knee joint muscles such as the hamstrings are *actively contracting* in this activity, muscles have connective tissue elements that can generate *passive* tension (see Chapter 3), but such tension at the posterior knee should be minimal with the knee at 90° of flexion. Because vector Fy_{QLf} is parallel to the articular surfaces (tibial plateau and tangent of femoral condyles), Fy_{QLf} would create an anterior shear between articular surfaces. The shear force would generate a corresponding friction force. The resulting friction force (friction-on-legfoot, or FrLf) is unlikely to be of sufficient magnitude to check the anterior shear, given the low coefficient of friction for articular cartilage.

Side-bar: Given Fr_S is less than or equal to $\mu_S\ F_C$ (and given $\mu_S \approx 0.016$), then Fr_S is less than or equal to (0.016)(822 N).

The most likely explanation in Figure 1–77 is that the Fy_{QLf} will translate the leg-foot segment to the right (anteriorly) until a new force from the joint capsule and passive muscles (CMsLf) reaches the necessary estimated force (tension) of approximately 420 N to the left (posteriorly).

Continuing Exploration 1-14:

Tendon Friction

Vector QLf in Figure 1–77 has been assigned a value of 1,000 N. It seems reasonable to presume that the quadriceps femoris muscle is contracting with a force of 1,000 N, but this may not be the case. *If* the quadriceps tendon (above the patella) and the patellar tendon (below the patella) were a continuous structure that passed over a frictionless pulley, the tension above and below the patella would be equivalent (1,000 N each). However, it appears that this is not the case. As is true for the majority of tendons that pass around bony prominences (such as the femoral condyles) or over sesamoid bones (such as the patella), some friction exists even if accompanying bursae or tendon sheaths minimize that friction. Figure 1–78 shows the schematic relation between a flexible structure and a rigid "pulley" that is similar to what the quadriceps muscle would look like passing over the patella and/or femoral condyles (because the femoral condyles also deflect the pull of the quadriceps muscle when the knee is flexed, independent of the patella). The maximum tension is generated by the pull of the quadriceps muscle. As the tendon passes over the patella and femoral condyles, however, the tension on the patellar tendon is reduced. Although the mathematical analysis is beyond the scope of the text, the reduction in tension is a function of the angle of contact between the surfaces (θ in Fig. 1–78) and a function of μ_S or μ_K.[1] As the angle of contact increases, there will be a greater differential between the pull of the muscle on the tendon and the tension in the tendon at its bony attachment. As the coefficient of friction is reduced (as it will be with an interposed bursa or tendon sheath), the differential in tension proximal and distal to the pulley is reduced. Given the structure of the quadriceps muscle and its associated elements, one study found an 8:5 ratio between quadriceps tendon tension above the patella and patellar tendon tension below the patella.[11]

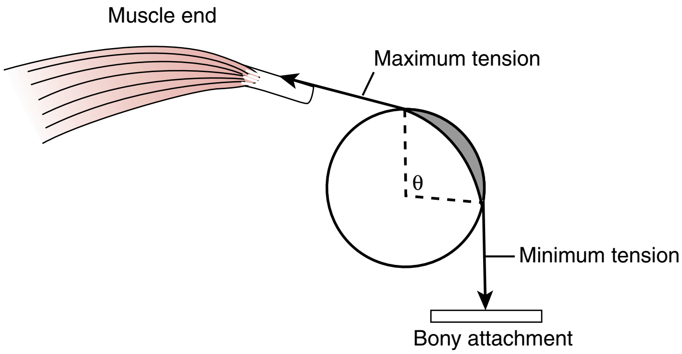

Figure 1-78 If there is friction between a tendon passing over an anatomic pulley and the anatomic pulley, the tension above and below the pulley will be different. The loss of tension in the tendon distal to the pulley is a function of the angle (θ) of contact and the coefficient of friction.

Rotary Effects of Force Components

Rotation Produced by Perpendicular (Fy) Force Components

In Figure 1–77, the capsule/muscles-on-legfoot force (CMsLf) and the Fy component of the quadriceps (Fy_{QLf}) are perpendicular to the leg-foot segment. We know that these two forces (like any forces perpendicular to a segment) will contribute to rotation of the segment if translation is prevented. These two force vectors have been isolated in Figure 1–79, and it can now be seen that capsule/muscles-on-legfoot and Fy_{QLf} are a force couple. These two forces are applied to the same segment, in opposite directions, with equal magnitudes—both producing counterclockwise rotation of the leg-foot segment (extension) if the segment were not constrained in any way. Remember, however, that the force of the capsule and passive muscles does not reach 420 N until the leg-foot segment has already translated anteriorly (to the right) by a small amount. Consequently, rotation will be initiated around the pivot point of the capsular attachment at the particular freeze-frame shown in Figure 1–79 (similar to what was seen in Figs. 1–45 and 1–51). Overall, however, it is more correct to think of the motion of the leg-foot segment as a combination of rotation and translation (curvilinear motion of the segment) around a point that is the center of some larger circle. This is why the joint axis in Figure 1–79 appears to be at a slight distance from, although close to, the point of application of capsule/muscles-on-legfoot.

The torque produced by a force couple is calculated as either (1) the product of the magnitude of one force (given that the magnitudes are the same for both forces) and the distance between the forces or (2) the product of the magnitude of each force and its distance from a common point between the forces. Although in the particular freeze-frame shown in Figure 1–79 the distance between vectors capsule/muscles-on-legfoot and Fy_{QLf} can be used to calculate their resulting torque, it is accepted practice to use the joint axis (center of rotation) as the common reference point for the moment arm of both forces because this is the point about which *composite* rotation (rotation throughout the knee extension ROM) appears to occur.

Thus far in the text, torque has been computed as the product of F_{TOT} and moment arm. When a force is *not* applied at 90° to the segment (as is true for the quadriceps force), the torque can alternatively be computed as the product of the perpendicular component (Fy) of that force

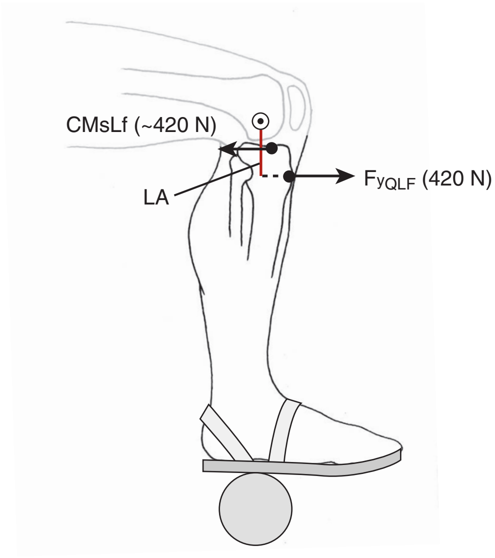

Figure 1–79 The forces (or force components) applied perpendicular to the leg-foot segment in Figure 1–77 have been isolated in this figure to facilitate assessment of their effect.

and *its* lever arm (see Fig. 1–79). Recall that the lever arm is simply a special case of the moment arm when the force is at 90° to the segment. The distinction is made between moment arm and lever arm because the moment arm for vector QLf will be different (and shorter) than the lever arm for the perpendicular component of QLf (Fy_{QLf}); that is, the shortest distance between the joint axis and QLf is different from the shortest distance between the joint axis and Fy_{QLf}. Consequently, the torque of QLf can also be computed as:

$$\tau_{FyQLf} = (Fy_{QLf})(LA)$$

If the magnitude of Fy_{QLf} is 420 N (see Fig. 1–79) and if the lever arm for Fy_{QLf} is given as 0.06 m, the torque (τ_{FyQLf}) would be 25.2 Nm. We will assume (as is most often done) that capsule/muscles-on-legfoot (the force closest to the joint axis) is sufficiently close to the joint axis that its torque is inconsequential (that the attachment of the capsule and the joint axis nearly coincide). This theoretically means that the torque produced by the force couple can be quantified by knowing the torque of Fy_{QLf} alone.

Rotation Produced by Parallel (Fx) Force Components

Most often, only the total forces (F_{TOT}) or components of the total forces that are perpendicular to the segment (Fy) are considered to produce torques. However, any force that lies at some distance from an axis will produce rotation around that axis, regardless of its orientation to the segment. Because the contribution of an Fx component to torque (as opposed to translation) for any given force is generally small (or smaller than that of Fy), estimating torque based on Fy alone is generally considered to be a reasonable (albeit conservative) estimate of torque produced by the total force.

Concept Cornerstone **1-20**

Force Components and Joint Motion

- Rotation around a joint axis requires that $\Sigma Fx = 0$. If $\Sigma Fx \neq 0$ initially, translatory motion of the segment will continue (alone or in combination with rotation) until checked either by a capsuloligamentous force or by a joint reaction force.
- Rotation around a joint axis requires that the $\Sigma Fy = 0$. If $\Sigma Fy \neq 0$ initially, translatory motion of the segment will continue (alone or in combination with rotation) until checked either by a capsuloligamentous force or by a joint reaction force (depending on the articular configuration), if the effects of external and muscular forces have already been accounted for.
- The majority of torque on a segment will be produced by forces or force components (Fy) that are applied at 90° to the segment and at some distance from the joint axis.
- Whenever the goal is the rotation of a joint, a net unbalanced torque in the direction of desired movement ($\Sigma\tau \neq 0$) is necessary to reach the goal.
- The greater the net unbalanced torque, the greater the angular acceleration of the segment.

Before we conclude the chapter, let us return to our original question of whether John Alexander should strengthen his quadriceps muscle by using the weight boot exercise or the leg-press exercise.

CASE APPLICATION

Summary of Weight Boot Exercise at 90° of Knee Flexion

case 1–3

We analyzed the weight boot at 90° of knee flexion (where the exercise begins) and found that there is a potentially problematic net distraction force at the knee joint before the initiation of the quadriceps muscle contraction (see Fig. 1–26C). With the initiation of an active quadriceps muscle contraction, the joint capsule is required to offset the anterior shear of the Fy component of the quadriceps force (see Fig. 1–77), although the magnitude of capsuloligamentous tension at 90° is likely to be minimal.[12] Just as the moment arms and magnitudes of the quadriceps force changed as the leg-foot segment moved through the knee joint ROM in Figures 1–56 through 1–58, there will be corresponding changes in the other forces with a change in position of the leg-foot segment.

We now need to examine the forces produced by the leg-press exercise at the equivalent 90° knee flexion angle (and with an equivalent small weight or resistance of 40 N) to determine the comparative effects of this exercise on John's knee joint structures, as well as to introduce a new level of complexity to a segmental force analysis.

MULTISEGMENT (CLOSED-CHAIN) FORCE ANALYSIS

The primary difference between the weight boot and leg-press exercise is that the leg-foot segment is "fixed," or weight-bearing at both ends, when using the leg press. The distal end of the leg-foot segment is constrained by its contact with the footplate and is not free to move in space; the proximal end is connected to or contacting the femur and also is not free to move in space. Whenever one end of a segment or set of segments is free to move in space, this is referred to as an **open chain.** When *both* ends of a segment or set of segments are constrained in some way (and not free to move in space), this is referred to as a **closed chain.**

Continuing Exploration 1-15:

Open and Closed Chains

The adjectives "kinetic" or "kinematic" are often used to modify the terms "open chain" and "closed chain." Although either might be justified, there is no consensus about which is preferred. At this juncture, the terms "open chain" and "closed chain" are now in such common use that the modifiers no longer seem necessary. What is necessary, however, is to avoid *misuse* of the term "closed chain" as equivalent to "weight-bearing" (unfortunately, a commonly used but inappropriate synonym). Although a segment *may* be fixed at both ends by proximal joint attachments and distal weight-bearing, a segment or set of segments can be weight-bearing *without* being fixed at both ends and without being subjected to the constraints of a closed chain. The effects of open and closed chains on segments will be encountered in subsequent chapters.

With the leg-foot segment in a closed chain leg-press exercise, the analysis of forces becomes substantially more complex. In a closed chain, motion of one segment of a joint can be produced by forces applied to the adjacent segment. Figure 1–80 is an oversimplified representation of the leg-press exercise, showing *only* the force of the gluteus maximus muscle extending the hip. The femur (like the leg-foot segment) is in a closed chain because it is fixed to both the pelvis and to the leg-foot segment. Consequently, the force of the gluteus maximus on the femur has the potential to produce motion of the femur that will result in extension *at both the hip joint and the knee joint as the extending femur pushes on the tibial plateau of the leg-foot segment.*

In Figure 1–80, the very substantial force that can be generated by the large gluteus maximus on the femur (although substantially underestimated by the length of the vectors in the figure) creates an extension torque at the knee joint. The muscle also acts through a large moment arm at the knee joint (again underestimated because the actual point of application of the gluteus maximus on the proximal femur is beyond the limits of the figure). The clockwise torque on the femur will cause the femur to contact the leg-foot segment and potentially push the leg-foot segment to the right.

Side-bar: At first glance, it might appear that the leg-foot segment is being acted on by a force (the gluteus maximus) that does not contact the segment, thus violating a basic tenet that we have set. However, the extension (clockwise) torque on the femur initiates a sequence of forces that *are* applied to the leg-foot segment, including femur-on-legfoot.

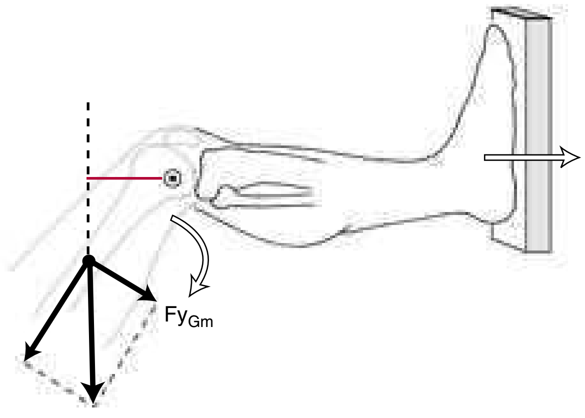

Figure 1–80 The force of the gluteus maximus muscle (Glut max) is applied to the femur in a closed chain. The resulting extension of the hip from Fy_{Gm} will push the leg-foot segment to the right against the footplate and create an extension torque at the knee joint.

Because the leg-press exercise involves forces on the femur as well as forces on the leg-foot segment, a complete analysis would involve (at a minimum) identification of the sources, magnitudes, and effects (ΣFy, Σx, $\Sigma \tau$) of all forces on both the leg-foot and femur segments—a task beyond the scope or intent of this chapter. An oversimplified example of the complex interrelationships is that the magnitude of torque generated by the quadriceps muscle is likely to be indirectly proportional to the torque generated by the gluteus maximus because both are extending the knee; more torque generation by one muscle would mean that less would be needed by the other.

Side-bar: This is an important concept in exercise prescription. Although weight-bearing exercises are more "functional," meaning they more closely approximate activities of daily living, the combinations of forces that may be used to achieve these exercises is nearly infinite. The downside of such exercises, therefore, is that patients may avoid the use of an injured joint or muscle simply by shifting the load to another muscle or joint.

The superior mechanical advantage (larger moment arm) and large force-generating capability of the gluteus maximus would make the gluteus maximus more likely to be the primary "effort force" used to extend the knee. Consequently, the forces acting on the leg-foot segment in the leg-press exercise are presented with the understanding that principles rather than actual quantitative analyses are being presented.

Figure 1–81 shows the knee at approximately 90° in the leg-press exercise. Gravity (the first force to consider because it is consistently present) is shown at the center of mass of the leg-foot segment (more proximally located than when forces of gravity and the weight boot were combined) with the previously identified magnitude of 48 N (the weight of the leg-foot segment has not changed). The quadriceps muscle (QLf) is shown generating the same 1,000 N contraction given for our weight boot exercise analysis (to keep that variable constant and comparable) and has the same force components because the angle of application of quadriceps will be the same whenever the knee joint is at 90° (regardless of the position of the segment in space). The footplate is contacting and creating a force (footplate-on-legfoot, or FpLf) on the leg-foot segment. A 40 N weight has been placed on the machine (the same weight that was used in the weight boot exercise). The footplate cannot push back on the leg-foot

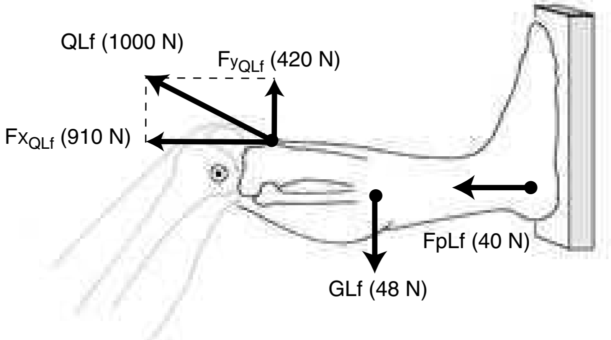

Figure 1–81 The forces of quadriceps muscle (QLf), gravity (GLf), and the footplate (FpLf) on the leg-foot segment in the leg-press exercise at 90° of knee flexion.

segment with more than a 40 N force, so the footplate-on-legfoot force has been assigned that value. We will identify the remaining forces by looking at the "need" generated by the forces already in place.

With the forces in place in Figure 1–81, there is a net compressive force (ΣFx) of 950 N ($Fx_{QLf} = –910$ N and FpLf = –40 N). There is a net upward shear (ΣFy) of 372 N (Fy_{QLf} = 420 N, and GLf = –48 N). The quadriceps muscle is generating the same extension torque of 25.2 Nm [(Fy_{QLf})(LA), or (420 N)(0.06)] that it did in the weight boot exercise, but the footplate now creates a flexion torque of 10 Nm [(40 N)(0.25 m)]. Therefore, the net torque in Figure 1–81 is 15.2 Nm in the direction of extension. The effect of the gluteus maximus must now be added.

The gluteus maximus force (shaded as a vector applied to the femur) has been added to Figure 1–82, along with its concomitant effects on the leg-foot segment. The gluteus maximus creates a push of the femur on the leg-foot segment (FLf) that is at an angle to the tibial plateau (and, more importantly, at an angle to the long axis of the leg-foot segment) because the femur is being rotating clockwise by the muscle. Femur-on-legfoot has been resolved into its parallel (Fx) and perpendicular (Fy) components. The magnitude of the Fx component of the force femur-on-legfoot (Fx_{FLf}) will have to exceed +950 N in order to push the footplate to the right, given the resultant –950 N force of the Fx component of the quadriceps (Fx_{QLf}) and footplate-on-legfoot. If the Fx component of femur-on-legfoot is assigned a value of 960 N, it appears that the Fy component of femur-on-legfoot (Fy_{FLf}) has the same magnitude because the components are approximately equivalent in size (at least in Fig. 1–82).

CASE APPLICATION

Calculation of the Magnitude of Femur-on-Legfoot

case 1–4

If the Fx and Fy components (see Fig. 1–82) of femur-on-legfoot have equal magnitudes, the angle of application of F_{TOT} (or femur-on-legfoot in this instance) must be either 45° or 135° (180° – 45°). Here we can see that femur-on-legfoot is applied at a 45° angle to the long axis of the leg-foot segment. The estimated magnitude of the Fx component of femur-on-legfoot can be used to calculate the magnitude of femur-on-legfoot. The formula cos θ = side adjacent ÷ hypotenuse can be used to solve for the hypotenuse, where hypotenuse = side adjacent ÷ cos θ. The "side adjacent" is Fx_{FLf} (960 N), and the cosine of 45° is 0.707 (according to a scientific or math calculator). The hypotenuse (femur-on-legfoot) has a calculated value of approximately 1,359 N. Although the magnitude of femur-on-legfoot is not the same as the force of the gluteus maximus, it should be proportional to the perpendicular (Fy) component of the gluteus maximus. Because the Fy component for most muscles is substantially smaller than the Fx component, we can assume that the resultant gluteus maximus force on the femur is substantially greater than 1,359 N (and substantially greater than the 1,000 N quadriceps force that has already been factored into this analysis).

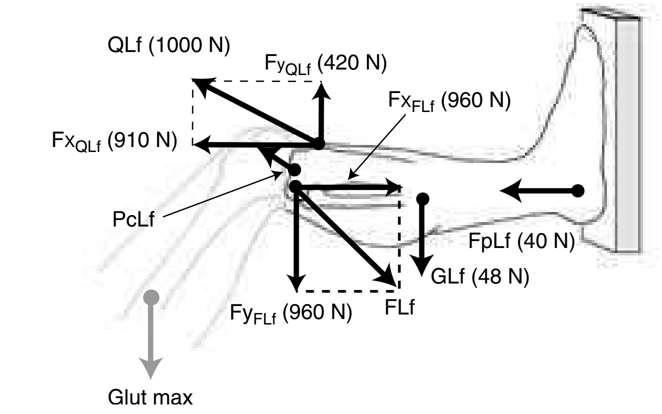

Figure 1–82 The contact of the femur with the leg-foot segment (FLf) and its components have been added to the force analysis, along with the force of the posterior cruciate ligament on the leg-foot segment (PcLf). The force of the gluteus maximus (Glut max) on the femur is shaded and remains only as a reminder of one of the potential sources of FLf.

If the Fy component of femur-on-legfoot has a magnitude of 960 N, there will be a net downward (posterior) shear force of 588 N ($Fy_{FLf} + Fy_{QLf}$ + GLf). Assuming again that friction, although present, will be a minimal restraint, we must now identify a structure that will prevent this posterior displacement. The likely source is a capsuloligamentous force—more specifically, tension in the anterior capsule or in the posterior cruciate ligament (PCL). Instrumented measurements and mathematical modeling indicate that a closed-chain leg press at 90° of flexion results in peak PCL tension.[12,13] Consequently, the force of posteriorcruciate-on-legfoot (PcLf) has been added to the figure. Although no attempt has been made to resolve PcLf into its components, its Fy component (anterior displacement) should be approximately 588 N. There will also be an Fx component of PcLf, with a magnitude that will depend on the angle of pull but will contribute further to the 950-N joint compression force already created by Fx_{QLf} and FpLf. The joint compression in the leg-press exercise, therefore, will exceed the 822-N compression estimated for the weight boot exercise at the same knee joint position and same magnitude of quadriceps muscle contraction.

Our oversimplified analysis of the leg-press exercise demonstrates the increased complexity of a closed-chain analysis over open-chain analysis. It should also demonstrate, however, that the strategy for understanding and analyzing the forces remains similar. In spite of very rough estimates of angles of application of forces (and concomitant Fy components and moment arms), our findings with regard to net effects for both the weight boot and leg press are fairly in line with findings from at least one group of researchers (although we used a minimum resistance in both the weight boot and leg-press exercises, in comparison with their substantially heavier loads).[12,13] This group of researchers also demonstrated the imprecise nature of biomechanical analyses (even using sophisticated instrumentation) because they found differing results (e.g., magnitudes of joint compression and joint shear forces), using the same data from the same subjects, on the basis of different mathematical modeling variables. Their results also contradicted some of the findings in at least one previous study.[14]

CASE APPLICATION

Case Summary

case 1–5

For those of you who do not wish to leave John Alexander without an "answer," we must first grant that our analyses of the weight boot and leg-press exercises are subject to the many limitations that have been acknowledged throughout the chapter. The most important of these—even using our simplistic approach—is that we compared the exercises in detail at only one knee joint angle (90°), but it must be understood that the relative merits change through the ROM. From what we have done, however, we can draw a couple of tentative conclusions. One is that John should not be permitted to let the weight boot hang freely (if the weight boot is used) because the direct tensile stresses placed on the capsuloligamentous structures of the knee may further damage his injured ligaments. The other conclusion is that the final choice of exercise may be dependent on further information about his injury. If John's PCL is injured, the leg press has a distinct disadvantage, especially in the more flexed knee positions.[12] Regarding an ACL injury, the evidence shows increased stress on this ligament in the weight boot exercise, but only as the knee approaches full extension.[12] Because the ACL is the more commonly injured of these ligaments, we can recommend the leg-press exercise, assuming the PCL is uninjured. If joint compressive forces need to be avoided for some reason (e.g., joint cartilage damage), the weight boot might be a better choice while avoiding completing the extension ROM that might stress the ACL.

SUMMARY

The goal of this chapter was to use John Alexander to present the biomechanical principles necessary to establish a conceptual framework for looking at the forces and the effects of those forces on joints at various points in a joint ROM (using a predominantly simplistic two-dimensional approach that was based on sequential static rather than dynamic analyses). We identified many (but not all) of the limitations of this conceptual framework when attempting to understand or explain the extremely complex phenomena of human function and dysfunction. We paid particular attention to the interdependence of muscular forces, gravitational forces (or other external forces), and articular constraints. The need for articular constraint (joint reaction forces or capsuloligamentous forces) to accomplish joint rotation is too often underappreciated, assessed only when there are problems with these constraints and ignored when they are effective in their function. To avoid this omission, we will next explore the structural composition and properties of the articular constraints (bone, cartilage, capsule/ligament/fascia), as well as those of the muscles and their tendons (Chapters 2 and 3). The composition and behavior of various tissues are key to understanding the stresses (force per unit area) that these tissues may create on other tissues or to which the tissues must characteristically respond. With that information in hand, the reader is then prepared to understand the basis of both normal and abnormal function at each of the presented joint complexes.

STUDY QUESTIONS

Part 1: Kinematics

1. Name three types of motion, and provide examples of each.
2. In what plane and around what axis does rotation of the head occur?
3. Is naming the plane of motion considered part of kinetics or part of kinematics? Why?
4. How does a center of rotation differ from a fixed axis? How does the distinction apply to motion at human joints?
5. What is the definition of a force, what are the units of measure for force, and how are forces applied to a segment?
6. What characteristics apply to all force vectors? What characteristics apply to all gravitational vectors?
7. What generalizations can be made about the line of gravity (gravity vector) of all stable objects?
8. What happens to the center of mass of a rigid object when the object is moved around in space?
9. What happens to the center of mass of the body when the body segments are rearranged? What happens to the body's center of mass if the right upper extremity is amputated? Provide a simple drawing to support your answer.
10. A student is carrying all of her books for the fall semester courses in her right arm. What does the additional weight do to her center of mass? To her line of gravity? How will her body most likely respond to this change?
11. Why did your Superman punching bag always pop up again?
12. Describe the typical gait of a child just learning to walk. Why does the child walk this way?
13. Give the name, point of application, magnitude, and direction of the *contact* force applied to a man weighing 90 kg as he lies on a bed.
14. Are the two forces of an action-reaction pair (reaction forces) part of the same linear force system? Defend your answer.

Continued

STUDY QUESTIONS—cont'd

15. What conditions must exist for friction on an object to have magnitude? When is the magnitude of the force of friction always greatest?

16. How do a contact force, shear force, and friction force differ?

17. A man who weights 90 kg (882 N, or ~198 lb) is lying on a bed in cervical traction with a weight of 5 kg (49 N, or ~12 lb) suspended from a horizontal rope. Assuming that the entire body is a single unsegmented object and that the body is in equilibrium, identify all the forces acting on the body (assuming that μ_S for skin on bed is 0.25).

18. The man in Question 17 is no longer in cervical traction. A nurse standing at the foot of his bed has grasped his right foot and is pulling him down toward the foot of the bed. Again treating the man as a single unsegmented object, determine the minimum force that the nurse must have applied to the man to initiate the movement.

19. Assuming now that the leg-foot segment of the man in Question 18 is joined to the rest of his unsegmented body by capsuloligamentous structures at his knee, what must be the magnitude of tension in the knee joint capsuloligamentous structures just as the man begins to be pulled toward the foot of the bed? What are the names of the forces causing tension in the capsuloligamentous structures? What are the names of the forces causing distraction of the knee joint segments?

20. How do you graphically determine the net (resultant) effect of two forces applied to the same segment that intersect at an angle to each other? What is this process called?

Part 2: Kinetics

21. What is the *minimum* requirement to produce rotation of a segment? What are the terms used to refer to the *strength* of rotation? How is the *strength* of rotation computed when the minimal conditions for rotation of a segment exist?

22. Define *moment arm*. How does moment arm affect the ability of a force to rotate a segment?

23. If two forces exist on a body segment on the same side of the joint axis but in opposite directions, how does one determine which is the effort force and which is the resistance force?

24. How do anatomic pulleys affect the magnitude and direction of a muscle force (Fms)?

25. *Muscle strength* and *muscle power* are often used interchangeably. What is the difference between these two terms?

26. What factors can cause the net torque on a segment to change?

27. A 2-year-old has difficulty pushing open the door to McDonald's. What advice will you give him on how to perform the task independently? What is the rationale for your advice?

28. What kind of contraction is a muscle performing when it is the effort force on a rotating segment? What kind of contraction is a muscle performing when it is the resistance force on a rotating segment?

29. What are the benefits of a mechanical advantage greater than one? Does this pose potential problems for muscles?

30. Using the values below, identify the class of the lever, its mechanical advantage, what kind of contraction the muscle is performing, and the point of application of the resultant force of gravity-on-forearm and ball-on-forearm (the hand will be considered part of the forearm). Here Fms = muscle force, LA = lever arm, G = gravity-on-forearm, and B = ball-on-forearm (assume that all forces are applied perpendicular to the forearm lever):

Fms = 500 N (counterclockwise), LA = 2 cm
G = 32 N (clockwise), LA = 18 cm
B = 20 N (clockwise), LA = 28 cm

31. Describe how the perpendicular component of a force and the moment arm of that force are related. When is the moment arm potentially greatest?

32. Describe how you would position a knee in space so that gravity exerts the least torque on the joint. How would you position the knee so that gravity exerts the greatest torque?

33. A muscle (Fms) is rotating a segment around a joint against the resistance of gravity (G). Identify a name for at least one force other than Fms and G that *must* be applied to the segment in order for rotation to occur.

34. If not all of a muscle's force is contributing to rotation, what happens to the "wasted" force?

35. What effects at a joint may a perpendicular force have on a segment other than rotation?

36. When a force is applied at 135° to a segment, what proportion of the force will rotate the segment? What proportion of the force will translate the segment, and will the direction of that force be compressive or distractive?

37. The quadriceps muscle is acting on the free leg-foot segment with a force of 500 N at an angle of 45° (unrealistic, but simplified). Identify the names (not the magnitudes or locations) of the other forces that would be required for the leg-foot segment to successfully extend at the knee joint.

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Chapter 2

Joint Structure and Function

Sandra Curwin, PT, PhD

“ Human joints must serve many functions; they are more complex than most man-made designs.”

Introduction
- Joint Design
  - Form Follows Function
  - Basic Principles

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  - Cartilage Response to Exercise
  - Tendon Response to Exercise
  - Ligament Response to Exercise
- Overuse

INTRODUCTION

Joint Design

Form Follows Function

The joints of the human body, like those used in the construction of buildings, furniture, and machines, connect different segments together and often allow movement between those segments. The design of the joint reflects these demands. The dictum *form follows function*, coined by the American architect Louis Sullivan and promoted by the Bauhaus school of design of post–World War I Germany,[1] suggests that the appearance (form) of an object or building should allow an observer to determine its function. The form of a chair, for example, with a seat, arms, and a back at appropriate heights and angles, tells us that its function is to support a sitting person. The function of the joint between most tabletops and table legs is support; therefore, the joints form a stable union. The joint between a leg and the top of a folding table, however, has a different function (mobility and stability), and therefore requires a different design, or form. We might design a folding table joint, for example, to have a metal brace fitted with a locking device. The table leg would be free to move when the brace is unlocked; when the brace is locked, the joint would be stable (Fig. 2–1).

The relationship between form and function means we can often determine the function of joints in the human body by examining their structure; we do this by studying the anatomy (structure) of joints.

Unlike man-made structures, in which form is static, the relationship between form and function in living structures is a two-way street—functional demands often help to determine the form of the structure. For example, the structural elements of the hip joint develop before birth, but the mature shape of the head of the femur and the acetabulum is determined by the functional interaction between these two structures. Decreased contact between the acetabulum and femoral head leads to developmental dysplasia of the hip (Fig. 2–2).[2]

Form includes an object's composition as well as its structure, and a change in either structure or materials (or both) can affect function. One striking example of the dependence of form on composition is sickle cell anemia. A mutation in the gene that encodes for the protein hemoglobin, which changes just one amino acid among thousands in the hemoglobin molecule, leads to changes in red blood cell shape and function. The health consequences that result from this change in function, which in turn result from a change in shape (form), can be severe. The sickle cells' decreased flexibility blocks blood flow in smaller vessels, which can cause tissue damage, and they also rupture easily, leading to anemia.[3]

The prosthetic foot shown in Figure 2–3 also reflects the relationships among physical form, composition, and function. The design of the curved foot allows the structure to flex as body weight is applied, while the heel extension provides stable support. Flexing the material stretches it, creating an elastic recoil that contributes to the subsequent movement as the person with a prosthetic foot moves forward. Alterations in the material (usually a carbon fiber composite) affect the amount of elastic recoil. A person with a lower extremity amputation can order limbs made of different materials, depending on their functional demands. Springier materials, which are harder to stretch and which recoil more, are used for activities with large loads and high speeds (e.g., running, jumping), whereas materials with less elastic recoil are used for walking. A track athlete with lower extremity amputations can order feet that are less stable but have greater recoil.[4]

The composition of human joint structures is determined by a number of factors, including genetic expression, cellular interaction during development, and functional demands. Human connective tissues and joints, in fact, depend on function to assume and maintain their final forms (Fig. 2–4).

All human joint structures—bones, cartilage, ligaments, muscles, and tendons—adapt their appearance and composition to match functional demands. These demands are usually mechanical and can change with immobilization, inactivity, or training. Knowing the functional demands and the tissues' responses, therapists can manipulate the functional demands on joint structures during rehabilitation to optimize tissue structure and function.

2-1 Patient Case

George Chen is a 40-year-old male electrician and business owner who suffered a supination-lateral rotation fracture of his right ankle 2 weeks ago while playing hockey. This type of fracture usually includes (1) a deltoid ligament sprain or medial malleolus avulsion fracture, (2) a spiral or oblique fracture of the distal fibula, and (3) a sprain of the anterior talofibular ligament, and may also involve a fracture of the posterior distal tibia (creating the so-called trimalleolar fracture) and/or separation of the ankle syndesmosis.[5] George had a bimalleolar fracture that was treated with open reduction and internal fixation (ORIF) and he is now non-weightbearing (NWB)

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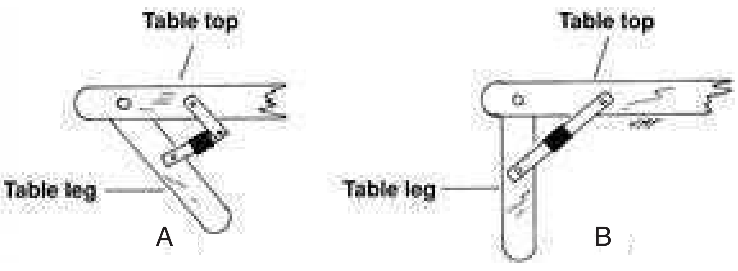


Figure 2–1 Folding table joint. **A.** The table leg is free to move, and the joint provides mobility when the brace is unlocked. **B.** The table leg is prevented from moving, and the joint provides stability when the brace is locked.

on crutches, with his leg and foot immobilized in a cast. A plate and screws were placed along the distal fibula; additional screws were placed to secure the medial malleolus and the syndesmosis (Fig. 2–5).

George, naturally, has many questions that he may expect you to answer. Does his ankle really need to be in a cast? Why does it still swell and hurt so much? Does the cast have to stay on for 8 weeks? Is his foot in the right position? Is there anything he can do to shorten his recovery time? Is it really necessary to be NWB for 8 weeks? When can he drive?

You, as a therapist, also are likely to have many additional questions. What structures are likely to have been damaged with this type of injury? What is the degree and nature of the injuries? Which structures are not injured? What changes will take place as the structures heal, and how will these changes affect lower extremity function? What are the ideal stimuli (functional demands) to preserve cartilage, bone, muscle, and tendon and ligament structure and function? Does the treatment scenario for this injury create negative consequences for uninjured structures? Are there ways to offset possible changes? What types of exercise should be used in the rehabilitation process, and what are their effects on the tissues that are not injured, as well as those that are?

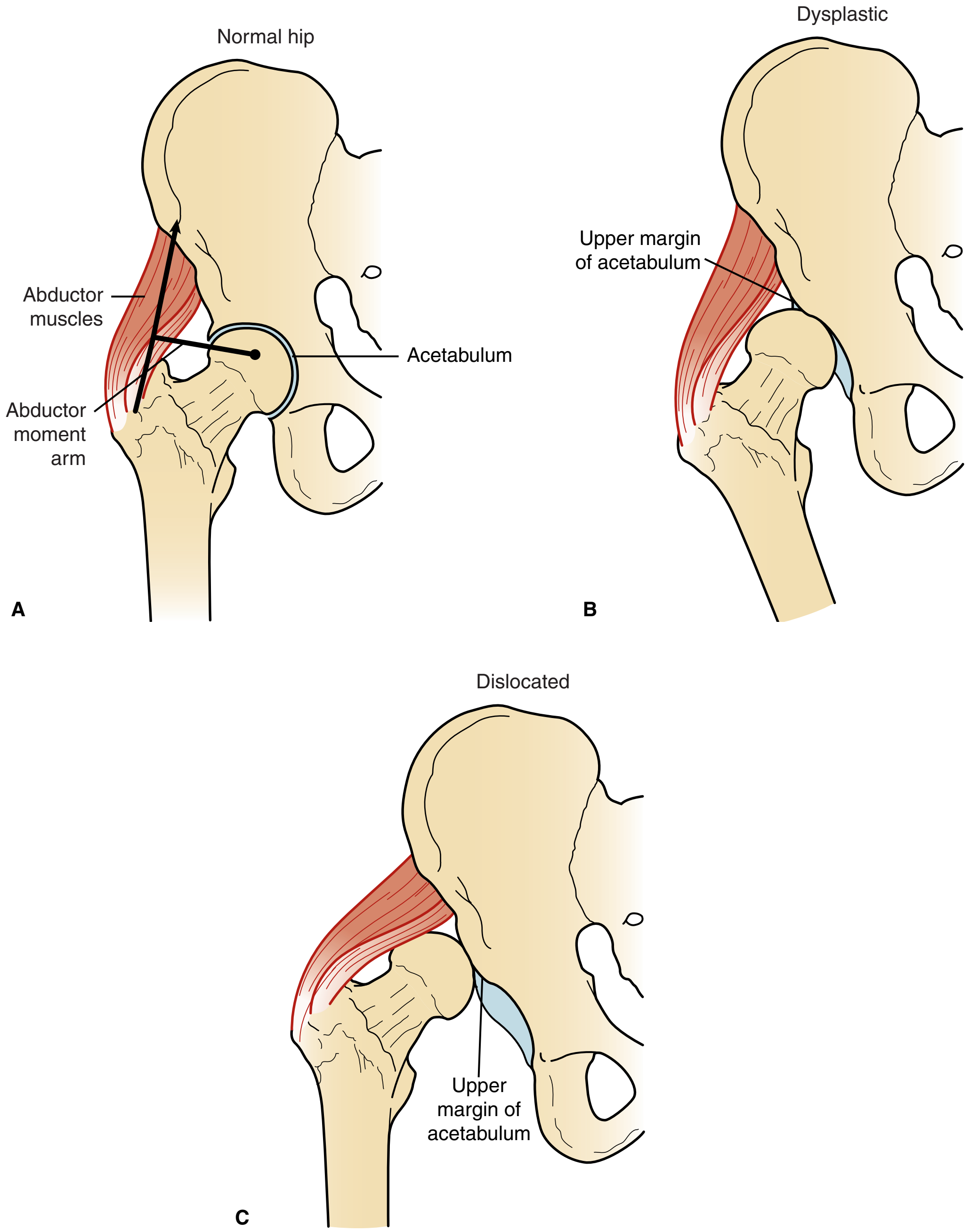


Figure 2–2 In the normal hip **(A)** the interaction between the head of the femur and the acetabulum causes the formation of the femoral head's round shape and the acetabulum's cuplike structure. The muscle lever arm enables the abductors to successfully create adequate torque during stance and gait. In the dysplastic hip **(B)**, the femur is not seated in the acetabulum in infancy, and the lack of interaction prevents normal development of the hip joint. The shallow acetabulum may allow dislocation of the femoral head **(C)**. The muscle lever (moment) arm is reduced, and the abductors are less effective at creating torque at the hip.

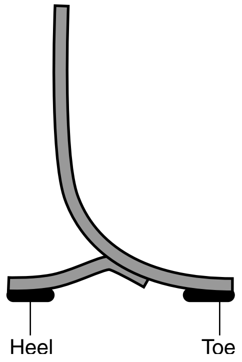


Figure 2–3 The “flex foot” facilitates gait through its design and composition. The curved blade bends during loading and then assists with propulsion. A change in material affects how much the structure bends and how much energy it provides to subsequent forward movement.

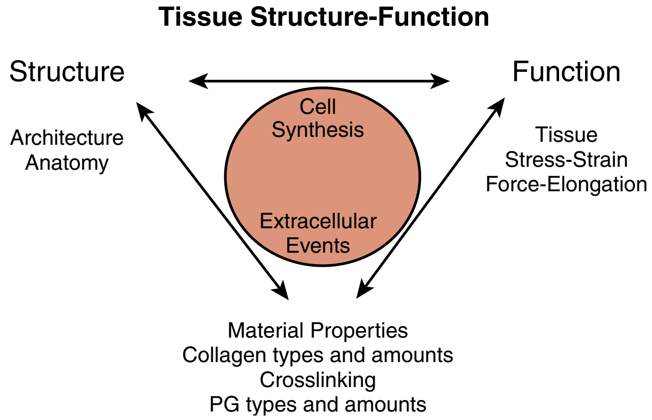


Figure 2–4 Form determines the overall structure of connective tissues, but the characteristics of the tissue are affected by functional use. Collagen type, cross-links, and PG type and amount all can be affected by the type and amount of stress applied to the tissue. Alternatively, the tissue may adapt to altered function by becoming larger, longer, or shorter. The size of the tissue and its composition will determine the types of loads the tissue can bear; these loads will likewise signal the cells to synthesize the appropriate type and amount of tissue and either dictate or facilitate extracellular events (e.g., cross-linking) that enhance tissue function.

Basic Principles

A joint (articulation) connects two or more components of a structure. The design of a joint and the materials used in its construction depend partly on the function of the joint and partly on the nature of the components. Joints that provide stability have a different design than those that provide mobility. The complexity of the design and composition matches the range of functional demands—the more varied the demands, the more complex the design. Human joints serve many functions, so they are usually more complex than most man-made designs.

The composition of materials used in joint construction also may influence design, and vice versa. Table legs made of particleboard would need to be larger than legs made of steel. The reverse also may be true: design requirements may dictate material composition. A car tire that is designed to last for more miles than is typical will require a change in material composition, rather than appearance. Changes occur in joint structures in order to allow them to meet functional demands. Julius Wolff described the adaptation of bone to changes in demand (Wolff’s law); similar changes can occur in tendons and ligaments.[6]

Concept Cornerstone **2-1**

Relationships Among Function, Structure, and Composition

Joint function both depends on and affects:
- Structure (design)
- Composition (materials)

MATERIALS FOUND IN HUMAN JOINTS

Human joints comprise living tissues that change their structure in response to changing environmental or functional demands. Tissues require nourishment to survive and are subject to disease, injury, and aging; they can adapt to imposed demands or become injured if the adaptation fails or demands are too great. To understand joint structure and function, we need to examine the forces acting at the joint and the composition of the tissues. The nervous, circulatory, and muscular systems also are integrally involved in overall joint function; however, this chapter focuses on the tissues that make up the actual joint structures: the connective tissues.

CASE APPLICATION

Materials (Living Tissues) Affected by the Fracture

case 2–1

The materials (tissues) likely to have been affected in George Chen’s case include bone, ligaments, blood vessels, nerves, and the joint capsule. Cartilage also may be involved, since 60 to 90% of these fractures involve an injury to the surface of the talus. Muscles and tendons, even if not directly injured, will be affected by immobilization. Thus, a number of structures will undergo a change in size and/or composition. We will be most concerned initially with structures that have been injured (bone, ligament, cartilage), as incorrect loading may interfere with their healing.

Structure of Connective Tissue

Joint structures include many connective tissues—bones, bursae, capsules, cartilage, discs, fat pads, labra, menisci, plates, ligaments, and tendons. The gross anatomic

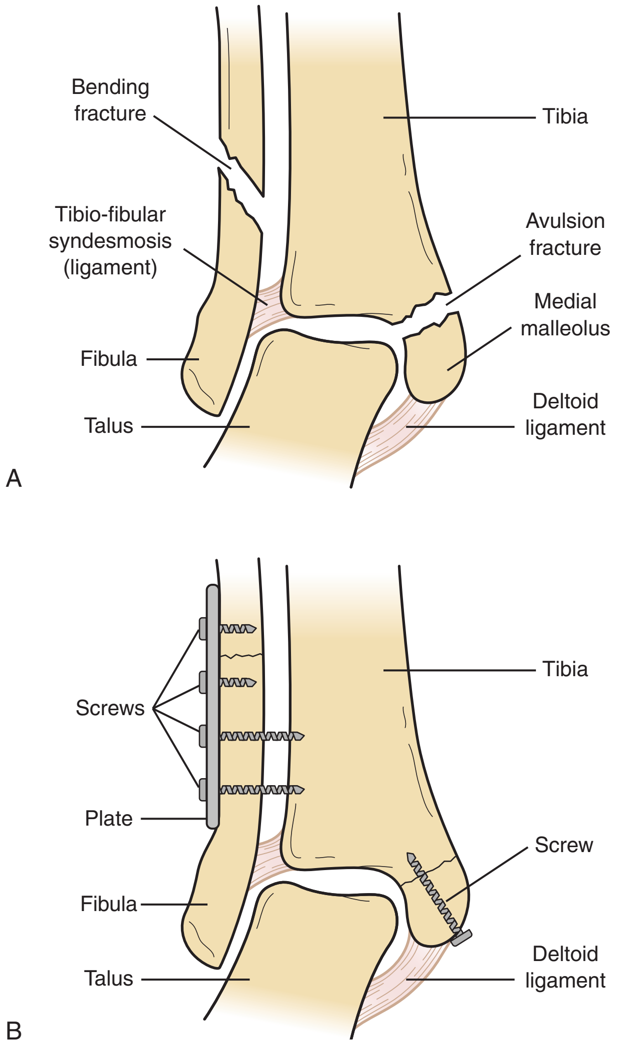


Figure 2–5 **A.** The foot (and talus) moves laterally and rotates outward, spraining the anterior talofibular ligament and fracturing the fibula. If the movement of the foot continues, the deltoid ligament may avulse the tip of the medial malleolus and separate the syndesmosis between the tibia and fibula. **B.** Stability must be restored and maintained via internal fixation in order for the fractures and ligament injuries to heal. A plate and screws have been applied to the fibula, while the tip of the medial malleolus has been reattached with a screw.

structure and microarchitecture of these connective tissue structures are extremely varied, and the biomechanical behaviors and composition of capsules, cartilage, specific ligaments, menisci, and tendons are still being investigated.[7–31] There are four classes of connective tissues (Fig. 2–6). Connective tissue is characterized by widely dispersed cells and a large volume of **extracellular matrix.** At the microscopic level, the extracellular matrix has both **interfibrillar** (previously known as the ground substance) and **fibrillar** (fibrous) **components.** The function of most tissues, such as nerve and muscle, depends on cell structure and function. Connective tissue function, by contrast, is primarily determined by its extracellular components (Table 2–1).

Cells

The cells of all connective tissues derive from mesenchymal precursor cells that differentiate into different connective tissue cells, either fixed in tissues or transient within the circulatory system (see Table 2–1). The **fibroblast** is the basic cell of most connective tissues; it produces the extracellular matrix. Depending on its mechanical and physiological environment, the fibroblast produces different types of connective tissue and receives a new name. Fibroblasts may specialize to become **chondroblasts** (cartilage), **tenoblasts** (tendon), or **osteoblasts** (bone); these cells are called **fibrocytes, chondrocytes,** and **osteocytes** when they mature and become less metabolically active. The distinction between a "blast" and a "cyte" is based primarily on appearance, which reflects cell synthetic activity; the same cell can go through several cycles as a fibroblast/fibrocyte, depending on the need to produce new connective tissue matrix. Connective tissue cells can "de-differentiate" and change the type of extracellular matrix they produce, given the appropriate environment and/or stimuli. For example, tendon cells can produce cartilage-like tissue when subjected to compressive forces.[37–39] Such findings suggest that connective tissue structure can be modified by changes in loading conditions and that we may be able to manipulate the mechanical environment to cause connective tissues to synthesize materials that will enhance their function.

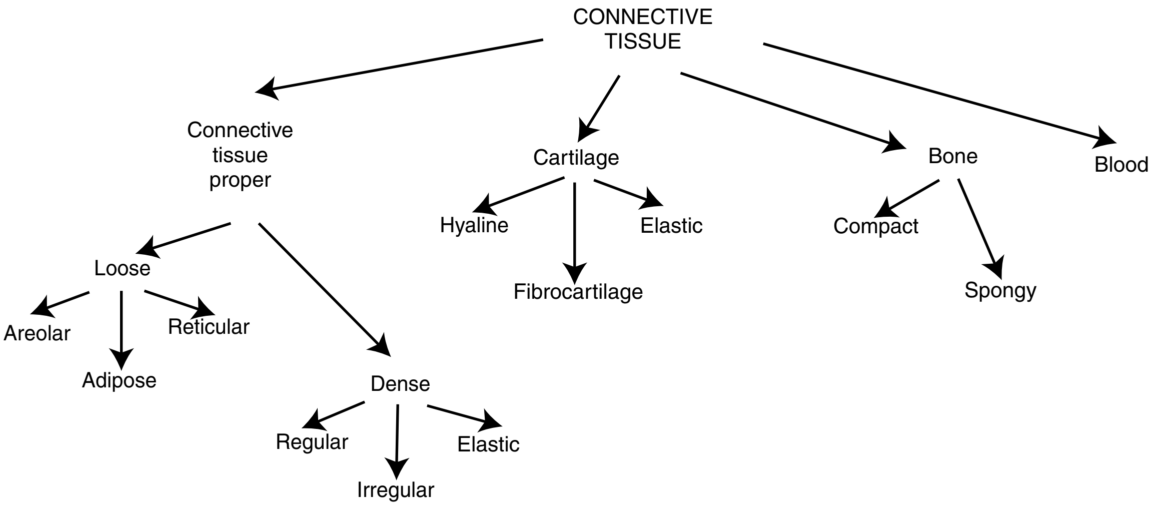


Figure 2–6 Classes of connective tissue. Tendons and ligaments are considered to be dense regular connective tissues. Bone is considered to be a highly specialized, mineralized form of connective tissue.

Table 2–1 Connective Tissue Cell Types

TYPE	NAME	LOCATION AND FUNCTION
Fixed	Fibroblast	Found in tendon, ligament, skin, bone, etc. Creates mostly type I collagen
	Chondroblast	Differentiated fibroblast found in cartilage Produces mostly type II collagen
	Osteoblast	Differentiated fibroblast found in bone Produces type I collagen and hydroxyapatite
	Osteoclast	Monocyte-derived, found in bone Responsible for bone resorption
	Mast cells	Found in various connective tissues Inflammatory mediators
	Adipose cells	Found in adipose tissue Produce and store fat
	Mesenchyme cells	Undifferentiated cells found primarily in embryos and in bone marrow Can differentiate into any connective tissue cell
Transient	Lymphocytes	White blood cells that have surface proteins specific for antigens
	Neutrophils	White blood cells involved in fighting infection
	Macrophages	Derived from monocytes, move into specific tissues, involved in immune response
	Plasma cells	B lymphocytes producing antibodies

Extracellular Matrix

The extracellular matrix is the part of connective tissues outside the cells. It comprises almost the entire volume of the tissue and determines the tissue's function. The extracellular matrix contains mainly proteins and water and is organized into fibrillar components and a surrounding matrix.

Fibrillar Component

The fibrillar, or fibrous, component of the extracellular matrix contains two major classes of structural proteins: **collagen** and **elastin.**[7] Collagen, the main substance of most connective tissues, is found in all multicellular organisms. The most abundant protein in the human body, it accounts for 25% to 30% of all protein in mammals.[41] Collagen has a tensile strength similar to steel and is responsible for the functional integrity of connective tissue structures and their resistance to tensile forces.[41–49]

Many types of collagen have been identified, but the functions of many are not yet well understood.[41–45] Some of the types of collagen and their distribution in joint structures are presented in Table 2–2. The Roman numerals that name each type of collagen—for example, type I, type II—reflect the order in which each type of collagen was discovered.[42,43] The fibril-forming collagens (types I, II, III, V, and XI) are the most common. Type I collagen, which accounts for 90% of the total collagen in the body, is found in most connective tissues, including tendons, ligaments, menisci, fibrocartilage, joint capsules, synovium, bones, labra, and skin.[41–47] Type I collagen appears to be responsible for the tensile strength of tissues. Type II collagen is found mainly in cartilage and intervertebral discs.[42,43] Type III collagen is found in skin, joint capsules, muscle and tendon sheaths, and in healing tissues.[43,49]

The basic building block of collagen is a triple helix of three polypeptide chains called the **tropocollagen molecule.** The repeating Gly-X-Y amino acid pattern of each chain causes the triple helix formation. Collagen peptide chains are synthesized in the rough endoplasmic reticulum inside the fibroblasts and then move through the cell toward the cell membrane. The tropocollagen molecules aggregate in groups of five to form microfibrils as they leave the cell; outside the cell, the microfibrils in turn combine to form **fibrils (fibers)** of varying size.[49] Intramolecular (between peptide chains within a collagen molecule) and intermolecular **cross-links** (between molecules of adjacent fibrils) stabilize and strengthen the enlarging fibrils.[50] Fibrils collect to form a **fascicle** that is surrounded by an endotendon sheath. Fibrils enlarge as more collagen is added to them and more cross-linking occurs among neighboring molecules; thus, older fibrils are larger and contain more cross-links, making them stronger. Collagen fibers may be arranged in many different ways and vary in size and length. In most relaxed tissues, the fibers have a wavy configuration called a **crimp.** When collagen fibers are stretched, the crimp disappears.

Elastin also is found in many connective tissues, but, unlike collagen, the molecule consists of single alpha-like strands without a triple helix.[7] The alpha-like strands are cross-linked to each other to form rubber-like, elastic fibers. Each elastin molecule uncoils into a more extended formation when the fiber is stretched and recoils spontaneously when the stretching force is removed. Elastin fibers branch freely and are found in all joint structures, as well as in skin, the tracheobronchial tree, and the walls of arteries. Elastin makes up a much smaller portion of the fibrous component in the extracellular matrix than collagen. As one might expect, tissue that requires "give" contains more elastin. The aorta contains approximately 30% elastin and 20% collagen (percentage of the tissue dry weight), the

Table 2–2 **Collagen Types**

CLASSIFICATION	TYPE	COMMON LOCATIONS
Fibrillar	I	Tendons, bone, ligaments, skin, anulus fibrosis, menisci, fibrocartilage, joint capsules, cornea Accounts for 90% of body collagen
	II	Hyaline articular cartilage, nucleus pulposus, vitreous humor
	III	Skin, blood vessels, tendons, ligaments
	V	Cartilage, tendons
	XI	Cartilage, other tissues (associated with type V)
	IX	Cartilage, cornea (found with type II)
Fibril-associated	XII	Tendons, ligaments (found with type I)
	XIV	Fetal skin and tendons
	IV	Basement membrane
Network-forming	X	Hypertrophic cartilage
	VIII	Unknown
Filamentous	VI	Blood vessels, skin
Anchoring	VII	Anchoring filaments

ligamentum nuchae has 75% elastin and 15% collagen, while the Achilles tendon contains only 4.4% elastin and 86% collagen.

Interfibrillar Component

The interfibrillar component of connective tissue contains water and proteins, primarily glycoproteins and proteoglycans (PGs).[32,33] A glycoprotein is a protein with a carbohydrate (sugar-type molecule) attached. There are thousands of glycoproteins in the body. The term *glycoprotein* technically includes PGs, but PGs were previously considered a separate class of compounds because their carbohydrates differ from the carbohydrates found in other glycoproteins, and because of their unique distribution: while glycoproteins are found in all tissues, PGs are found mainly in connective tissues. In the past, PGs were also called **mucopolysaccharides,** and the interfibrillar matrix was referred to as the **ground substance.** The "ground substance" is really a mixture of PGs and water. An overview of some of the PGs found in connective tissues is shown in Table 2–3.

The carbohydrate portion of PGs consists of long chains of repeating disaccharide units called **glycosaminoglycans (GAGs).**[30] The GAGs are all very similar to glucose in structure and are distinguished by the number and location of attached amine and sulfate groups (Table 2–4). The major types of sulfated GAGs are **chondroitin 4** and **chondroitin 6 sulfate, keratan sulfate, heparin, heparan sulfate,** and **dermatan sulfate.** Most GAGs attach to proteins to form PGs, except hyaluronic acid, which exists on its own. A PG can contain one or more (up to about 100) GAGs, which stick out from the protein core to form a shape like a bottle brush.[34,35] Once a GAG has been attached to the protein core of the PG by a specific trisaccharide molecule, more GAGs can added to the chain. Glucosamine (part of a GAG) and chondroitin sulfate are frequently used as supplements to treat osteoarthritis, though their efficacy remains unproven.[51]

Hyaluronan differs from the other GAGs because it is not sulfated and does not attach to a protein core. Hyaluronan exists as either a free GAG chain of variable length (e.g., in a tendon or ligament) or a core molecule to which large numbers of PGs are attached (e.g., in cartilage). Hyaluronic acid is sometimes injected into osteoarthritic joints to relieve symptoms.

One large cartilage PG is called **aggrecan.** The protein portion of aggrecan, with GAGs bristling out from its length, attaches at one end, via a separate link protein, to a hyaluronan chain.[34,35] Aggrecan molecules in the extracellular matrix do not exist in isolation but as proteoglycan aggregates.[34] Each aggregate is composed of a central filament of hyaluronan with up to 100 aggrecan molecules radiating from it, with each interaction stabilized by a link protein (Fig. 2–7).[35] These large aggregates, with their attached chondroitin sulfate and keratan sulfate chains of GAGs, are largely responsible for the water-binding that characterizes the extracellular matrix of cartilage and gives it the ability to withstand compression.

The PGs in the extracellular matrix of a structure (bone, cartilage, tendon, or ligament) affect its hydration through their attached GAGs.[34] The GAG chains attract water into the interfibrillar matrix, creating a tensile stress on the surrounding collagen network. The collagen fibers resist and contain the swelling, thus increasing the rigidity of the extracellular matrix and its ability to resist compressive forces as well as supporting the cells. The PGs also form a reservoir for nutrients and growth factors that attach to the PG molecules, and they may play a role in directing or limiting the size of collagen fibrils. The amount and type of PG in a tissue is another example of the form-function interaction of connective tissues. Tissues that are subjected to high compressive forces (like cartilage) have more PG, with different GAGs, than tissues that resist tensile forces.[38,39] The type of GAG in the

Table 2–3 **Proteoglycans**

CLASSIFICATION	NAME	LOCATION, COMPOSITION, AND FUNCTION
Large extracellular aggregating	Versican	Found in smooth muscle cells, fibroblasts; function unknown
	Aggrecan	Found in numerous chains of KS and CS Binds to hyaluronan Creates osmotic swelling pressure in cartilage by attracting water
	Brevican	Found in nervous system; cell adhesion and migration
	Neurocan	Found in nervous system; cell adhesion and migration
Small leucine-rich proteoglycans (SLRPs)	Decorin	One or two CS or DS chains Binds and regulates growth factors, modulates cell functions, regulates collagen fibrillogenesis, interacts with collagen types I, II, III, V, VI, XII, XIV
	Biglycan	Two GAG chains containing CS or DS Directs type VI collagen network assembly Binds to complement and transforming growth factor beta (TGF-β,)
	Fibromodulin	One KS GAG chain Interacts with type I and II collagen, binds to growth factors
	Lumican	Similar to fibromodulin, found in cornea, muscle, intestine, cartilage
	Epiphycan	Found in epiphyseal cartilage
Cell-associated PGs	Serglycins	Protein core of heparin: PGs regulate enzyme activities in secretory granules
	Syndecans	Cell transmembrane PG containing HS acts as a receptor for heparin-binding factors
	Betaglycan	Contains HS and CS Binds TGF-β,
	CD44 family	Cell surface receptor for hyaluronan
	Thrombomodulin	Binds to thrombin
Basement membrane PGs	Perlecan	Found in all tissues; function uncertain
	HS and CS PGs	Found in all tissues; function uncertain
	Bamacan	Found in various tissues; function uncertain
Nervous tissue PGs	Phosphacan	Nervous tissue cell adhesion
	Agrin	Aggregates acetylcholine receptors
	NG2 PG	Found in developing cells

CS, chondroitin sulfate; DS, dermatan sulfate; GAG, glycosaminoglycan; HS, heparan sulfate; KS, keratan sulfate; PG, proteoglycan.

Table 2–4 **Glycosaminoglycans (GAGs)**

GAG	LOCALIZATION	COMMENTS	COMPOSITON
Hyaluronan	Synovial fluid, vitreous humor, loose CT, healing CT, cartilage	Forms large PG aggregates	Glucuronate uronic acid Glucosamine
Chondroitin sulfate	Cartilage, bone, heart valves, tendons, ligaments	Most abundant GAG, increased with compression	Glucuronate Galactosamine with 4-sulfate or 6-sulfate
Heparan sulfate	Basement membranes, cell surfaces	Interacts with numerous proteins	Glucuronate Glucosamine Variable sulfation
Heparin	Intracellular granules in mast cells lining arteries	Key structural unit is 3-glucosamine + 2-glucuronate	Glucuronate, iduronate Glucosamine Variable sulfation
Dermatan sulfate	Skin, blood vessels, tendons, ligaments	Increased with tensile stress	Iduronate Galactosamine
Keratan sulfate	Cornea, bone, cartilage	Forms part of large PG aggregates in cartilage	Galactose Glucosamine

CT, connective tissue; PG, proteoglycan.

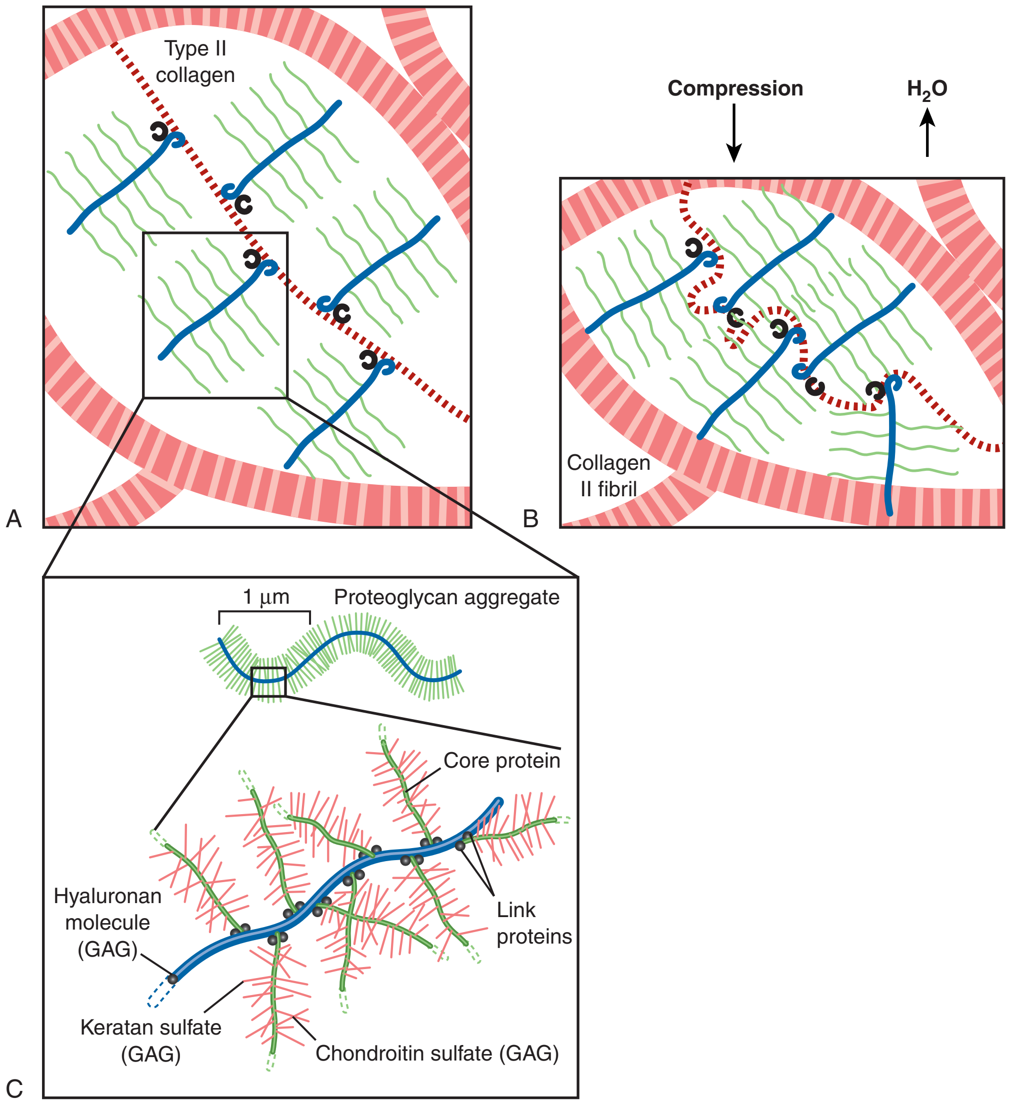


Figure 2–7 The extracellular matrix (ECM) of articular cartilage contains numerous proteins, GAGs, and proteoglycans. The type II collagen fibers, with collagen XI embedded within them, form a meshwork that contains the proteoglycan (PG) aggregates, with water attached, preventing their escape into the joint **(A).** When compression is applied to the cartilage, water is squeezed out but the PG aggregates remain trapped **(B).** The proteoglycans **(C)** contain chondroitin sulfate and keratan sulfate GAGs attached to a protein core, which in turn is attached, via link proteins, to a hyaluronic acid GAG chain. These proteoglycan aggregates form long bottle brush structures; aggrecan is one of the largest. *(Adapted from Adams M, Roughley P: What Is Intervertebral Disc Degeneration, and What Causes It? Spine 31(18):2151, 2006.)*

PG also may change, depending on whether the tissue is subjected to tensile or compressive forces.[39] Tissues subjected to compression (like discs) have larger amounts of chondroitin sulfate and keratan sulfate, whereas tissues subjected to tension (like tendon) contain more dermatan sulfate.[39]

Concept Cornerstone **2-2**

Proteoglycan Characteristics

Proteoglycans:

- are distinguished by their protein core and by their attached GAGs;
- attract water through their attached GAGs;
- regulate collagen fibril size;
- may attach to hyaluronate (another GAG) to form large aggregating structures; and
- are increased in tissues subjected to alternating cycles of compression.

Glycoproteins such as **fibronectin, laminin, chondronectin, osteonectin, tenascin, tenomodulin, chondromodulin,** and **entactin** play an important role in fastening the various components of the extracellular matrix together, in the adhesion between collagen and integrin molecules in cell membranes, and as inhibitors of angiogenesis (Table 2–5).[52]

Concept Cornerstone **2-3**

Extracellular Matrix Function and Structure

The extracellular matrix of connective tissue determines its function, and vice versa. The type and proportions of the components create the different tissues:

- Interfibrillar component: PGs (protein + GAGs), glycoproteins
- Fibrillar component: collagen (mainly type I or II), elastin

CASE APPLICATION

Materials Involved in the Fracture Healing Process

case 2–2

The early callus of fracture healing (at 2 weeks) usually consists largely of fibrocartilage material (extracellular matrix) containing a high proportion of PGs, GAGs, other glycoproteins, and collagen. Undifferentiated mesenchymal cells migrate to the fracture site from the nearby periosteum or bone marrow; these will form cartilage, bone, or fibrous tissue as they mature. The fracture hematoma becomes organized, fibroblasts and chondroblasts appear between the bone ends, and cartilage is formed (type II collagen). Fibroblasts begin to form type I collagen that becomes mineralized to form bone. The amount of callus formed is inversely proportional to the amount of immobilization of the fracture. In fractures that are fixed surgically, there can be primary bone healing with little or no visible callus formation. These fractures will tend to heal a bit more slowly, because primary bone formation takes longer. This explains why our patient must be non-weightbearing for 6 to 8 weeks, even though the fracture fragments have been rejoined with screws and plates. At 2 weeks, there is probably very little bone at the fracture site.

Specific Connective Tissue Structures

Ligaments

Ligaments connect one bone to another, usually at or near a joint. Ligaments may blend with the joint capsules and appear as thickenings in the capsule (e.g., the anterior band of the inferior glenohumeral ligament). Other ligaments are distinct, easily recognizable structures that often appear as distinct dense white bands or cords of connective tissue (e.g., the anterior cruciate ligament).

Ligaments are heterogeneous structures containing a small amount of cells (about 10% to 20% of the tissue volume, mainly fibroblasts) and a large amount of extracellular matrix (about 80% to 90% of tissue volume). PGs constitute only about 0.2% of the tissue dry weight and contain primarily dermatan sulfate GAG. The fibrillar component of the extracellular matrix in most ligaments is composed of mainly type I collagen, with lesser amounts of type III, type IV, and type V collagen and varying amounts of elastin. One notable exception is the ligamentum flavum, which has a distinctly yellowish color and contains a large amount of elastin (75% of the tissue dry weight).[7] The type I collagen fibrils in ligaments are densely packed, with fiber bundles arranged in line with the applied tensile forces. The arrangement of the collagen fibers and the collagen/elastin fiber ratio in various ligaments determines

Table 2–5 Glycoproteins

CLASSIFICATION	NAME	COMMENTS
Cartilage	Asporin	Related to decorin and biglycan, found in cartilage Increases in osteoarthritis
	Chondronectin	Attaches chondrocytes to type II collagen
	Chondroadherin (CHAD)	Found in cartilage Binds to cells via integrin Function unknown
Bone	Osteoadherin	Found in bone trabeculae Binds to cells via integrin
	Osteonectin	Binds to hydroxyapatite, collagens, growth factors, osteoadherin; inhibits cell spreading
	Osteopontin	Binds to osteoclast via integrin Assists osteoclast function
	Osteocalcin (BGP)	Thought to be involved in bone formation
Basement membrane	Laminin	Binds type IV collagen, HS, integrin (cell membrane)
	Entactin	Interacts with laminin
Multiple sites	Collagen	Structural component
	Fibronectin	Interacts with cell-surface receptors, blood-clotting components, denatured collagen, cytoskeleton, GAGs
	Tenascin	Function unclear; increases in developing or healing tissue
Synovial fluid	Lubricin	Adheres to articular surface to provide boundary lubrication

GAG, glycosaminoglycan; HS, heparan sulfate.

the relative abilities of these structures to provide stability and allow mobility for a particular joint.[53] Ligaments are subjected to varying directions of tensile force, depending on the joint angle, so the collagen fibers in ligaments run in many directions, enabling the ligament to resist forces in more than one direction. For example, the posterior fibers of the medial collateral ligament of the knee may be stressed by extension, while the middle fibers are under tension when a varus stress is applied. The collagen fibers line up parallel to the tensile forces applied to the ligament in order to resist them. The tensile forces on tendons are created by muscle force and tend to be applied in primarily one direction, so the collagen fibers in tendons are straight and parallel.

The appearance and composition of ligaments and tendons change near their **entheses,** the attachments to bone. Tendons and ligaments may insert directly into bone via fibrocartilage or indirectly via fibrous attachments.[56] In fibrous attachments, the collagen fibers blend into the **periosteum** of the bone, which is in turn attached to the underlying cortical bone via **Sharpey's fibers.** These fibrous attachments have been likened to trees that are rooted in the ground, with Sharpey's fibers serving as the "roots" for the tendon or ligament. The stiffening of the ligament-bone interface at direct fibrocartilage insertions decreases the likelihood that the ligament will give way at the enthesis; however, it is a common site for degenerative change, usually in the underlying bone.[53–57]

Ligaments are often named descriptively according to their location, shape, bony attachments, and relationship to one another.[6] The anterior longitudinal ligament of the spine derives its name from both location (anterior) and shape (longitudinal). The medial and lateral collateral ligaments of the elbow and knee joints are examples of ligaments named for location. Ligaments such as the coracohumeral ligament, which connects the coracoid process of the scapula to the humerus at the shoulder, and the radioulnar ligaments, which connect the radius to the ulna at the distal radioulnar joint, are named for their bony attachments. The deltoid ligament at the ankle joint is named for its shape. Occasionally, ligaments are named for the individuals who first identified them. The Y ligament of Bigelow at the hip joint is named both for its inverted Y shape and for an individual.

Tendons

Tendons connect muscle to bone and transmit forces developed by the muscles to their bony attachments. Each muscle has tendinous material interspersed between it and bone, although the attachments may vary widely in configuration[55,56] and one tendon may be much more prominent than another. Prominent tendons are usually named for the muscle to which they are attached: for example, the biceps tendon is named for the biceps brachii and the triceps tendon for the triceps brachii. Occasionally, tendons are named in a different way; for example, the Achilles tendon is named after the famous warrior of Greek mythology whose heel was the only part of his body vulnerable to injury.

Tendons and ligaments have a similar composition and basic structure, though the proportions and organization of the components of their extracellular matrix differ.[48,53–57] Tendons contain mostly type I collagen (more than 95%), with type III in the tendon sheaths and types IV and V associated with the basal lamina of the fibroblasts.[45,57] Tendons contain slightly more type I collagen and slightly less type III collagen than ligaments (Table 2–6). The increased type I collagen in tendons (compared to ligaments) is thought to be an adaptation to larger tensile forces, since type I collagen is considered stronger than type III collagen.[45] The interfibrillar component of the extracellular matrix in tendons contains water, PGs that contain mostly dermatan sulfate, and other glycoproteins. Dermatan sulfate GAG appears to correlate with tensile loading.

The collagen fibrils of tendon form successively larger subunits, primary bundles known as fibers.[48] The diameter of these fibers increases with age and with increased tensile loads.[54,58] Groups of fibers, enclosed by a loose connective tissue sheath called the **endotendon,** form a secondary bundle called a *fascicle* (Fig. 2–8). The endotendon, containing mostly type III collagen fibrils, also encloses the nerves, lymphatic vessels, and blood vessels supplying the tendon.[45] Each fascicle is associated with a discrete group of muscle fibers or motor units at the muscle-tendon junction.[59,60] Several fascicles may form a larger group (tertiary bundle) that also is enclosed in the endotendon. The sheath that encloses the entire tendon is called the **epitenon.** The **paratenon** is a double-layered sheath of areolar tissue that is loosely attached to the outer surface of the epitenon. The epitenon and paratenon together are sometimes called the **peritendon.** The peritendon may become a synovium-filled sheath called the **tenosynovium** (or **tendon sheath**) in tendons that are subjected to high levels of friction. The paratenon protects the tendon from friction, allowing it to slide past adjacent structures, and also provides a source of replacement cells if the tendon is injured. The collagen

CASE APPLICATION

Mechanism of Injury

case 2–3

In the case of George Chen, the mechanism of injury involved a lateral rotation twist of the ankle, with the foot probably in a supinated position.[5] Tensile stress was created in the deltoid ligament. The relative strengths of the ligament, enthesis, and bone will determine where (and whether) tissue failure will occur. In George's case, an avulsion of the tip of the medial malleolus occurred. The fibula was fractured by the talus, forcing it outward, and the interosseous attachments between the tibia and fibula were disrupted. It is likely that the deltoid ligament, anterior talofibular ligament, and ankle joint capsule also were damaged. An osteochondral defect in the talar joint surface also may be present.

Table 2–6 Composition of Dense Connective Tissues

TISSUE	WATER CONTENT*	COLLAGEN†	PG/GAGS†	COMMENT
Bone	25%	25%–30% Mainly type I	Mainly CS	65%–70% dry weight is inorganic
Cartilage	60%–85%	10%–30% >90% type II	8%–10% aggregating PGs	Cells are 10% of total weight
Ligament	70%	75% 90% type I 10% type III	< 1% Mainly DS	20% dry weight unknown
Tendon	60%–75%	80% 95% type I <5% type III	0.2%–1% Mainly DS	More linear collagen fibrils than ligament
Capsule	70%	90% Mainly type I	CS, DS	Some elastin
Menisci	70%–78%	60%–90% Mainly type I	<10%	Fibrocartilage
Anulus	65%–70%	50%–60% Types I and II	20% CS, KS	
Nucleus	65%–90%	20%–30% Mainly type II	65% aggregating PGs	

*Water content is percentage of total tissue weight.
†Collagen and PG content is percentage of tissue dry weight after water has been removed.
CS, chondroitin sulfate; DS, dermatan sulfate; GAG, glycosaminoglycan; KS, keratan sulfate; PG, proteoglycan.

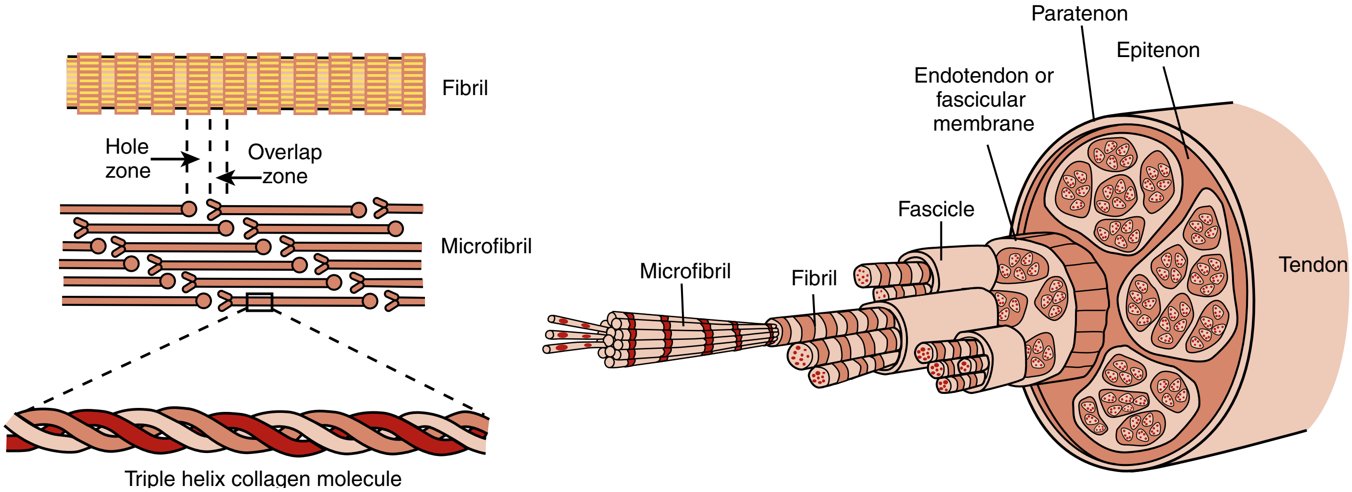


Figure 2–8 Dense connective tissues such as tendon have a hierarchical structure from the molecule to the entire tissue.

fibers in tendons and ligaments line up with the loads applied to the tissue.[46,48] The relaxed collagen fibers appear crimped due to PG interaction with the collagen fibrils.

Like ligaments, tendons have two types of bone attachments: **fibrocartilaginous** and **fibrous.**[56] The fibrocartilaginous attachment of tendon to bone (enthesis) involves gradual changes in the tendon structure over a length of about 1 mm, divided into four zones based on histological observation (Fig. 2–9). The first zone consists of tendon proper and is similar to the tendon midsubstance. The second zone contains fibrocartilage and marks the beginning of the transition from tendon to bone. The third zone contains mineralized fibrocartilage, and the fourth zone consists of bone. The changes in tissue are gradual and continuous, which is presumed to aid in effective load transfer between two very different materials. A tidemark frequently appears between the calcified and uncalcified parts of the enthesis, representing the boundary between hard and soft tissues; however, the tidemark is not a barrier, and collagen fibers pass through it to the mineralized fibrocartilage. Dramatic changes in gene expression and tissue composition occur in the different zones. Collagen types II, IX, and X and aggrecan are localized to the bony insertion, whereas decorin and biglycan are localized to the tendon side of the insertion.[55,56] Collagen types I, III,

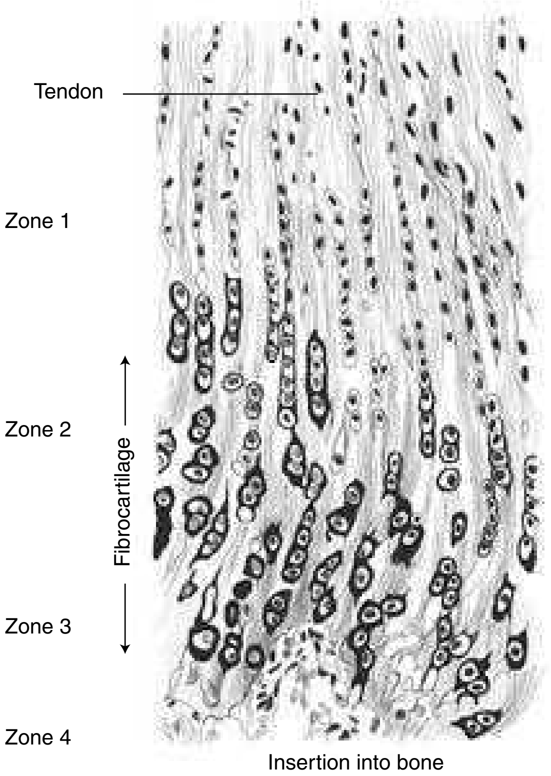


Figure 2–9 The bone-tendon (or ligament) junction. There are four zones, from pure tendon (zone 1) to bone (zone 4). In between, the material gradually transitions from fibrocartilage (zone 2) to mineralized fibrocartilage (zone 3).

and XII have been found at both locations. These findings suggest that the tendon insertion is subjected to both compressive and tensile forces.

The fibrous entheses may be divided into two categories: **periosteal** and **bony.**[55,56] At the former, the tendon fibers attach to the periosteum, which indirectly attaches the tendon to the bone. With bony entheses, the tendon attaches directly to the bone. There can be a mixture of the two types of attachment, and periosteal attachments sometimes convert to bony attachments with age. Examples of these mixed attachments include the attachments of the deltoid muscle to the humerus, scapula, and clavicle. The new enthesis formed after the surgical reattachment of a tendon to bone is initially fibrous, although fibrocartilage may be re-formed with time.[56]

The attachment of tendon to muscle at the **myotendinous junction (MTJ)** comprises interdigitation between collagen fibers and muscle cells.[59,60] Surface friction and direct connections among collagen and PGs, the basal lamina, and integrins in the muscle cell membrane create a strong interaction (Fig. 2–10). The interdigitating configuration of the MTJ is essential for normal function of the muscle-tendon unit, which is very sensitive to mechanical conditions and tends to become flatter and less infolded when loads are decreased. This change weakens the junction and may make it more susceptible to injury. Thus, when muscle-tendon loading begins after a period of immobilization, loads should begin at a lower level and progress gradually.

Concept Cornerstone 2-4

Gradual Transition Between Tissues Serves to Diffuse Load

Attachments of tendon and ligament to bone reflect a gradual transition between tissue subjected primarily to tensile force and tissue subjected to compressive and tensile forces. This transition “diffuses” the load at the junction, perhaps helping prevent injury.[56] The differences in composition suggest that tension increases collagen type I concentration whereas compression increases PG concentration. Tendon attachment to muscle depends on interaction between collagen fibers in a tendon and the basal lamina and cell membrane of muscle fibers. Attachment sites are highly sensitive to changes in loading, especially decreases.

Bursae

Bursae are flat sacs of synovial membrane in which the inner sides of the sacs are separated by a fluid film. Bursae are found where moving structures are in tight approximation: that is, between tendon and bone, bone and skin, muscle and bone, or ligament and bone. Bursae located between the skin and bone, such as those found between the patella and the skin and between the olecranon process of the ulna and the skin, are called **subcutaneous bursae.**[6] **Subtendinous bursae** lie between tendon and bone, and **submuscular bursae** lie between muscle and bone.

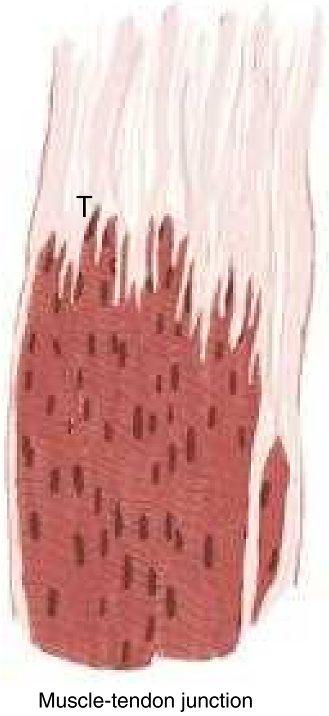


Figure 2–10 The muscle-tendon junction. The muscle cells interdigitate with the tendon (T). There are direct connections between the muscle cell membrane and fibroblasts, PGs, and collagen. The endotendon blends into the endomysium, and the epitenon blends into the epimysium, which forms a meshwork of connective tissue around the muscle fibers.

Cartilage

Cartilage is usually divided into the following types: **fibrocartilage (white), elastic cartilage (yellow),** and **hyaline (articular) cartilage.**[11] Cartilage contains mainly type II collagen and large amounts of aggregating PGs. Fibrocartilage forms the bonding cement in joints where little motion occurs, such as, the intervertebral discs, the glenoid and acetabular labra, and one articular surface of the temporomandibular and sacroiliac joints. Unlike hyaline cartilage, which contains almost exclusively type II collagen, white fibrocartilage also contains type I collagen. Yellow elastic fibrocartilage is found in the ears and epiglottis and has more elastin than white fibrocartilage, which consists primarily of collagen fibers.[11]

Hyaline (articular) cartilage, from the Greek word *hyalos*, meaning "glass," forms a relatively thin (1 to 7 mm) covering on the ends of the bones in **synovial joints.** It provides a smooth, resilient, low-friction surface that is capable of bearing and distributing weight over a person's lifetime. Once injured, articular cartilage has limited and imperfect mechanisms for repair.[11,13,61,62] Articular cartilage has the same general structure as other connective tissues—that is, a small cellular component and a large extracellular matrix—however, its extracellular matrix contains much more interfibrillar material than that of tendons and ligaments.[63] The cells in articular cartilage are chondrocytes and chondroblasts. Chondroblasts produce collagen, GAGs, PGs (mainly aggrecan), link protein, and hyaluronan that are all extruded into the extracellular matrix and aggregate spontaneously. Chondrocytes are the less synthetically active cells observed in nongrowing tissues. The fibrillar component of the articular cartilage extracellular matrix includes some elastin and other types of collagen, but type II collagen accounts for about 90% to 95% of the collagen content.[63] Type XI collagen regulates the fibril size, and type IX facilitates fibril interaction with PG molecules. Collagen is dispersed throughout the interfibrillar component of the extracellular matrix[32] and also forms a hooplike meshwork at the joint surface that attaches to bone at the articular cartilage margins.[32,63] The meshwork surrounds and compresses the PGs and their attached water molecules.

Articular cartilage contains much more PG than other joint structures. The major PG is aggrecan, which contains keratan sulfate and chondroitin sulfate GAG chains. Numerous aggrecan PGs attach to hyaluronic acid, each via a special link protein, to form large aggregating structures.[32] Aggrecan contains the GAGs chondroitin sulfate and keratan sulfate.[35] The ratio of chondroitin sulfate to keratan sulfate varies among individuals, with age, and at different locations within the joint. The higher the chondroitin sulfate concentration is, the better the tissue can resist compressive forces. Keratan sulfate concentration increases with age and in joints with arthritic changes and decreases with immobilization.[61] If the amount of keratan sulfate exceeds the amount of chondroitin sulfate, the load-bearing ability of the cartilage appears to be compromised. **Chondronectin,** a cartilage glycoprotein, plays an important role in the adhesion of chondroblasts to type II collagen fibers in the presence of chondroitin sulfate.[61,62]

Three distinct layers (zones) of articular cartilage are found on the ends of the bony components of synovial joints[11] (Fig. 2–11). In the outermost layer (zone 1), radially oriented type II collagen fibers are arranged parallel to the surface (this is the top of the hoop of the meshwork). This smooth outer layer helps reduce friction between opposing joint surfaces and distributes forces over the joint surfaces. In the second and third zones, type II collagen fibers form an open meshwork. The collagen network keeps the PGs and water contained, giving the cartilage its ability to absorb compressive forces. In the third layer (radiate stratum), perpendicular collagen fibers cross the interface between uncalcified and calcified cartilage to find a secure hold in the calcified cartilage.[6,32] The calcified layer of cartilage, sometimes referred to as the fourth zone, lies adjacent to subchondral bone and anchors the cartilage securely to the bone.[63] The interface between the calcified and uncalcified cartilage is called the **tidemark.**[11,63] The tidemark area of the cartilage is important because of its relation to growth, aging,[64] injury,[61] and healing.[62]

Little is known about how articular cartilage is maintained in mature joints. Cartilage is thought to have virtually no cellular turnover, based on the facts that the tissue (1) is hypocellular and avascular, (2) relies on diffusion for its nutrient supply, and (3) contains only terminally differentiated cells. Normally, replacement of the calcified layer of articular cartilage with bone occurs by **endochondral ossification.** The calcification front advances toward the noncalcified area of cartilage slowly, in equilibrium with the rates of absorption of calcified cartilage and, perhaps, new extracellular matrix formation by cells.[64,65] In aging, the balance changes as deeper layers of cartilage are gradually replaced with bone, resulting in cartilage thinning superficially. In injuries that involve microfractures to the subchondral bone, a secondary ossification center in the bone may be activated to produce new bone growth, which expands into the calcified layer of cartilage, advancing the tidemark and thinning the noncalcified layer.[66]

Figure 2–11 Structure of hyaline cartilage.

The design of articular cartilage is a remarkable example of the interaction between form and function. Aggregating PGs attract a large volume of water, creating an osmotic swelling pressure in the cartilage.[67] As the interfibrillar matrix expands, tension is created in the hoops of the collagen network, creating an opposing force and keeping the PGs and water contained. Equilibrium is reached between the swelling pressure and the load on the joint, and deformation stops.[68] Cartilage resistance to compressive force thus depends on two features: (1) a large volume of aggregating PGs and (2) an intact collagen network. The first step in cartilage degeneration is loss of the superficial collagen fibers, which allows more water to enter, swelling the cartilage. Without the collagen meshwork, PGs and GAGs later begin to escape into the joint, and the cartilage narrows and loses its ability to resist compression. Eventually, the cartilage is worn away completely.

Concept Cornerstone **2-5**

Proteoglycan Type Related to Types of Loading in Tendon, Ligament, and Cartilage

The types of PGs found in tendon, ligament, and cartilage suggest that:

- PGs containing dermatan sulfate are associated with tensile loading (e.g., tendon).
- PGs containing chondroitin sulfate are associated with compressive loading (e.g., cartilage).
- Aggregating PG structures are associated with compressive loading.
- PGs increase with compressive loading.

During joint motion, or when the cartilage is compressed, some of the fluid content of the cartilage exudes into the joint space through pores in the outermost collagen layer.[67–69] The fluid then flows back into the cartilage after the load is removed, carrying nutrients from **synovial fluid** that supply the chondrocytes. Because hyaline cartilage is devoid of blood vessels, its nourishment is derived solely from this flow of fluid; thus, the free flow of fluid is essential for the survival of hyaline cartilage.

Fluid flow is affected by the magnitude and duration of the applied force.[69] If the force is increased and sustained over a long period, the equilibrium between swelling pressure in the cartilage and external load reduces fluid flow.[67] Diminished flow also occurs with decreased loading. The absence of compressive forces on the cartilage surface reduces the movement of fluid, which remains immobilized in the extracellular matrix. Hyaline cartilage can thus undergo degenerative changes after either prolonged loading or unloading, due to inadequate nutrition as fluid flow is diminished.[70] Cartilage health thus appears to depend on alternating cycles of moderate compressive forces.[71]

Damage to the superficial collagen layer, through excessive frictional forces or trauma, removes the meshwork that resists the swelling pressure of the PGs. Initially, the articular cartilage swells and become thicker as the PGs attract more water without the opposing force of the now-absent superficial collagen network.[72] Eventually, fluid movement slows, reducing cell nutrition and synthetic ability. Without the containment of the superficial collagen meshwork, PGs begin to escape into the synovial fluid, gradually eroding and thinning the cartilage. This is the sequence that occurs during the development of osteoarthritis.[72]

CASE APPLICATION

Effects of Immobilization on Our Patient's Cartilage and Protection of Cartilage After Immobilization

case 2–4

While George's ankle and foot are in a cast and he is non-weightbearing, the immobilized joints encounter decreased loading forces. The cartilage will swell, straining the superficial collagen network, reducing nutrient diffusion into the cartilage, and making the superficial collagen layer more susceptible to damage. The early period after immobilization ends is thus a precarious one for articular cartilage, and loading should be resumed gradually. This is even more important if George, like more than 60% of patients with this mechanism of injury, has an osteochondral injury. Some cartilage loading can be produced during immobilization via isometric muscle contraction (which compresses joint surfaces). A better treatment would avoid immobilizing the joint while still reducing loads. Joint motion alternately compresses and unloads the joint surfaces, maintaining cartilage nutrition and helping to maintain capsule mobility. A removable brace might be preferable to a cast so that the patient can remove it for range-of-motion exercise, if the patient will adhere to weight-bearing restrictions. The postoperative cast is often changed for a cast-brace approximately 3 to 4 weeks after surgery, when some new bone has started to form at the fracture sites.

Bone

Bone is the hardest of all the connective tissues. The organic fibrillar extracellular matrix, with mainly type I collagen, is also impregnated with inorganic materials, primarily **hydroxyapatite.**[6,7] The organic material gives bone its flexibility and tensile strength, while the inorganic material gives bone its compressive strength.[73] Bone cells include fibroblasts, osteoblasts, osteocytes, osteoclasts, and progenitor cells that can differentiate into osteoblasts. The fibroblasts produce type I collagen and other extracellular matrix components. The osteoblasts are the primary bone-forming cells that are responsible not only for the synthesis of bone but also for its deposition and mineralization. Osteoblasts also secrete **procollagen** (the precursor of type I collagen) into the surrounding matrix. **Osteoclasts,** large monocyte-derived polymorphous cells with multiple nuclei, are responsible for bone resorption. Homeostasis between synthesis and deposition is fine-tuned by nutrition, hormonal status, and mechanical loading.[73]

Bone's complex architecture has been described in numerous ways. Bone has two layers, a dense outer layer and a spongier inner layer.[6] The inner layer is called **cancellous** (also **trabecular** or **spongy**) **bone;** the outer layer is called **compact,** or **cortical, bone.** In cancellous bone, the calcified tissue forms thin plates called **trabeculae** that are laid down in-line with stresses placed on the bone. It has been suggested that the loading history of the trabeculae, including loading from multiple directions, influences the distribution of bone density and trabecular orientation.[74] Increases in bone density in some areas and decreases in density in other areas occur in response to the loads placed on the bone. The cancellous bone is covered by a thin layer of dense, compact cortical bone, which is laid down in concentric layers.

The periosteum is a fibrous layer that covers the entire surface of each bone except the articular surface. Collagen fibers from ligaments and tendons blend into the periosteum, and Sharpey's fibers pass from the periosteum to deeper layers of bone. The periosteum houses cells that are precursors to osteoblasts and osteoclasts and includes many capillaries that provide nourishment. The periosteum thus acts as a reservoir for cells that are needed for growth and repair. If the periosteum and underlying bone are damaged as a result of trauma or surgery, the healing capacity of the bone will be decreased.

At the microscopic level, both cortical and cancellous bone show two distinct types of bone architecture: **woven bone** and **lamellar bone.** In woven (primary) bone, collagen fibers are irregularly arranged to form a pattern of alternating coarse and fine fibers that resembles woven material. Woven bone is young bone, formed rapidly without an underlying framework. It is often found in newborns, fracture callus, and metaphyseal regions of long bones. Lamellar bone requires an extracellular matrix framework to form, is organized into parallel layers, and is the older bone that constitutes most of the adult skeleton.

Bone is dynamic and remodels throughout life as it responds to changes in forces, such as an increased pull of tendons with strengthening or the weight of the body during new activities.[74] The change in bone shape (form) to match function is known as *Wolff's law*. The application of new forces causes osteoblast activity to increase, and as a result, bone mass increases. With reduction of usual forces, osteoclast activity predominates and bone mass decreases. Internal influences, such as aging and nutritional, metabolic, and disease processes, also may affect bone remodeling, since bone serves physiological as well as mechanical functions.[73]

An imbalance between bone synthesis and resorption, in which osteoclasts break down or absorb the bone at a faster rate than the osteoblasts can remodel or rebuild the bone, results in a condition called **osteoporosis.**[75] In osteoporosis, bones have decreased mineral density (mass per unit volume) compared to normal bone and thus are weaker and more susceptible to fracture. Bone mineralization may also be decreased while cells continue to synthesize other elements of the extracellular matrix; this is called **osteopenia.** Bones that have been immobilized are frequently osteopenic.

The preceding paragraphs provided a brief overview of the composition of the various connective tissue structures that are associated with the joints. The composition of bones, capsules, cartilage, intervertebral discs, menisci, ligaments, and tendons were summarized in Table 2–6.

Concept Cornerstone **2-6**

Dense Connective Tissue Function

The function of dense connective tissues is characterized by:

- Cell type
- Collagen: type, amount, and arrangement
- Interfibrillar matrix: PG type, amount, and arrangement

CASE APPLICATION

Status of Fracture Healing, Articular Cartilage, and Bone: Types of Appropriate Exercise and Activity

case 2–5

The surgery used to stabilize George's fractures, while necessary to restore anatomical structure, also damaged the periosteum of the distal tibia and fibula. Thus, an early increase in stability comes at the cost of an increased healing time. The early woven bone must be replaced with stronger lamellar bone. The unloaded bones will have tipped toward the resorption end of the scale and will need to be gradually reloaded to resume their original strength. George may be allowed to begin weight-bearing at a reduced level after 6 weeks, with a gradual increase to full weight-bearing by 12 weeks. Meanwhile, articular cartilage has swelled and softened and will need to be gradually reloaded after cast removal.

Safe exercises at the 6-week point would include active range-of-motion (ROM) exercises (excellent for cartilage nutrition) and low-level physiological loading, such as walking, at a reduced load level—say, 50% of body weight. Joint mobilizations can be used to increase joint ROM (if required)—this will facilitate normal motion during walking and also will increase the articular cartilage contact area during movement. Impact-type loading (e.g., running, jumping) and high-load resistance exercises (including isometric exercises) should be avoided

Continued

soon after cast removal. Although the time period for cartilage recovery is unknown, loading should probably be increased gradually over a period of weeks or months. If George had his cast replaced with a brace at 2 weeks, the next 4 to 6 weeks should consist mainly of ROM exercises and isometric muscle exercises. These exercises will help maintain cartilage nutrition while avoiding overloading the healing fracture sites. Once George begins weight-bearing, the next 6 weeks should be devoted to gradually increasing weight-bearing loads and working on resistance exercises through the weight-bearing and non-weight-bearing ROM.

Continuing Exploration 2-1:

Connective Tissue Research With Fibroblasts

Connective tissues are now being grown under laboratory conditions for later implantation to replace damaged tissues. For example, new chondrocytes can be grown and later injected into areas of cartilage degeneration.[76] The starting point is fibroblasts, usually seeded in some sort of mesh or scaffold. What types of loads do you think should be applied to this raw material to produce the following tissues?

- Bone
- Cartilage
- Ligament
- Tendon

GENERAL PROPERTIES OF CONNECTIVE TISSUE

Homogeneous materials, such as steel, display the same mechanical behavior no matter the direction in which forces are applied and are called **isotropic** materials. In contrast, heterogeneous connective tissues behave differently depending on the size and direction of applied forces and are called **anisotropic.** Connective tissues are called *heterogeneous* because they are composed of a mixture of solid and semisolid components. The function of the structure as a whole depends on a combination of the properties of the different components, the varying proportions of each component in the structure, and the interactions among these components.[18,29,30]

Example 2-1

The compressive strength of compact bone is about 250 MPa, greater than that of concrete (approximately 4 MPa) or wood (100 MPa) but less than that of steel (400 to 1500 MPa). Bone is about as strong as cast iron (140 to 300 MPa) but weighs much less. Bone is not as strong in tension as in compression, and its strength depends on density (mineralization) as well as the orientation of the trabeculae in relation to the load. If the load is applied along the long axis of the bone, it is much stronger.

Connective tissues change their structure and/or composition (and thus their function) in response to applied forces; that is, they adapt.[53,77–80] The nature and extent of these adaptations is an area of active research.[81,82] Tendon responds to changes in applied compression forces by increasing the amount of GAGs and PGs it contains and by changing the type of GAG (from dermatan sulfate to chondroitin sulfate).[38,39] Increases in tensile forces cause increases in type I collagen in ligaments and tendons. This adaptive behavior illustrates the dynamic nature of connective tissue and the strong relationships among structure, composition, and function. The remarkable ability of connective tissues to respond to load alterations is often referred to as the **SAID principle (*s*pecific *a*daptation to *i*mposed *d*emand).**[79] The principles of tissue adaptation have been used to create a "physical stress theory" that can be used to guide intervention during rehabilitation.[80]

Mechanical Behavior

Human joint components experience changing forces during ordinary daily activities, and they must have the ability to withstand these forces in order to provide support and protection. To understand how different materials and structures are able to provide support (in other words, the mechanical behavior of these structures), it will help to be familiar with the concepts and terminology used to describe their behavior, such as **stress, strain, failure,** and **stiffness,** among others. The types of tests used to determine the mechanical behavior of human tissues are similar to those used for building materials.

Load, Force, and Elongation

The term **load** refers to a force or forces applied to a structure. Many examples of loads are given in Chapter 1, including the forces exerted by the weight boot, the leg-press footplate, and gravity on John Alexander's leg-foot segment. The magnitude, direction, and rate of force application, as well as the size and composition of the tissue, will all affect the tissue's response to load.

When a force acts on an object, it produces a **deformation.** A tensile load produces **elongation**; a compressive force produces **compression.** The **load-deformation curve** is the result of plotting the applied load (force) against the deformation, providing information about the strength properties of a particular material or structure.[16,17,21,53] The load-deformation curve (Fig. 2–12) shows the **elasticity, plasticity, ultimate strength,** and **stiffness** of the material, as well as the amount of energy that the material can absorb before it fails. The portion of the curve between point A and point B is the **elastic region.** If the response to loading is confined to the elastic region, the deformation of the material will not be permanent; the structure will return to its original dimensions immediately after the load is removed. After point B, the **yield point** at the end of the elastic region, the material will no longer immediately return to its original state when the load is removed, although it may recover in time. The portion of the curve between B and C is the **plastic region.** Although the structure will appear to be intact, after the load is removed the material will not recover its original length—the deformation is permanent.

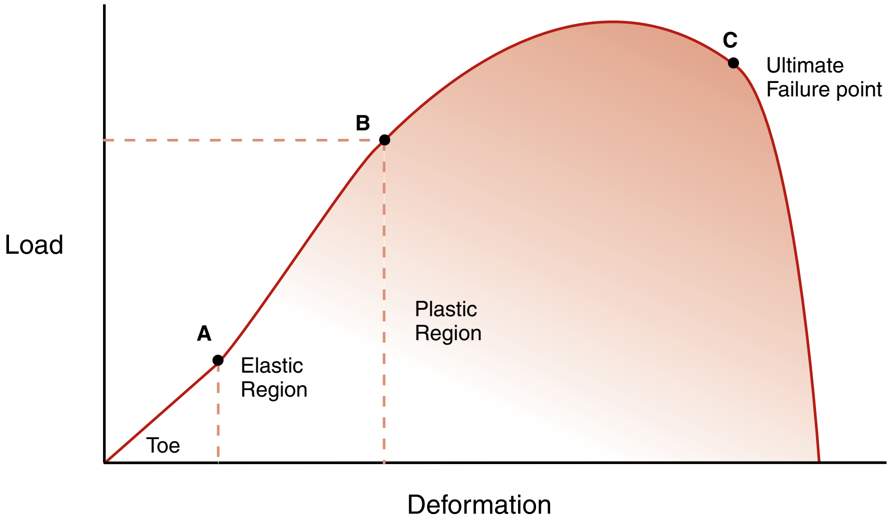


Figure 2–12 Load-deformation curve for a connective tissue tested in tension. Initially, the crimp straightens with little force (toe region). Then, collagen fibers are stretched as the elastic region begins at A. After the elastic region ends (B), further force application causes a residual change in tissue structure (plastic region). Continuation of load may cause the tissue to rupture at its ultimate failure point (C). *(From Butler DL, Grood ES, Noyes FR, et al: Biomechanics of ligaments and tendons. Exerc Sport Sci Rev 6:144, 1978, with permission from Lippincott William & Wilkins.)*

Recovery of the original structure probably could only occur through the synthesis and reorganization of new tissue components (this may apply to some ligament sprains). If loading continues through the plastic region, the material will continue to deform until it reaches the **ultimate failure point,** C. The load being applied when this point is reached is the **failure load.**

Force values on the load-deformation curve depend on both the size of the structure and its composition[53]. A structure with a greater cross-sectional area can withstand more force with less deformation than a structure of the same original length with less cross-sectional area (Fig. 2–13A). A longer structure deforms more when a force is applied than does a shorter structure of similar cross-section (Fig. 2–13B). If two tissues are composed of the same material, the tissue with greater cross-sectional area will have greater tensile strength (stiffness), and the longer tissue will be less stiff. Because the forces and deformations measured during testing are so dependent on the structural features of the tissue, the load-deformation curve is said to reflect the **structural properties** of the structure being tested.[53] Tensile force is measured in newtons (N), compressive force (pressure) is measured in pascals (Pa), and elongation or compression is measured in units of length.

Stress and Strain

When loads (forces) are applied to a structure or material, forces within the material are produced to oppose the applied forces. These forces within the material depend on the composition of the material. If we account for the mechanical effect of the tissue structure (cross-sectional area and length), testing can tell us something about the tissue material. We account for tissue cross-sectional area by converting the applied force into a number independent of tissue size. When the applied force is tensile, we calculate the stress on the tissue. Stress, the force per cross-sectional unit of material, can be expressed mathematically with the following formula, where S = stress, F = applied force, and A = area:

$$S = F/A$$

Stress is expressed in units of pascals (P = N/m^2) or megapascals (MPa = N/mm^2). These are the same units used to measure **pressure,** which is also a force per unit area. However, pressure is produced by a compressive force applied perpendicular to the surface of the material.

The percentage change in the length or cross-section of a structure or material is called **strain.**[19,20] Like stress, it cannot be measured directly but is calculated mathematically with the following formula, where L1 = original length and L2 = final length:

$$\text{Strain} = (L2 - L1) \div L1$$

Strain is expressed as a percentage and thus has no units.

The types of stress and strain that develop in human tissues depend on the material, the type of load applied, the point at which the load is applied, the direction and magnitude of the load, and the rate and duration of loading.[53,83,84] When a structure can no longer support a load (i.e., force drops to zero), the structure is said to have failed. **Ultimate stress** is the stress just before the material fails; **ultimate strain** is the strain at the same point.

If two applied forces act along the same line but in opposite directions, they create a distractive or **tensile load** and cause **tensile stress** and **tensile strain** in the structure or material (Fig. 2–14A).[30,31] If two applied forces act in a line toward each other, they constitute **compressive loading** and **compressive stress** and, as a result, **compressive strain** will develop in the structure (see Fig. 2–14B). If two applied forces are parallel and are applied in opposite directions but are not in-line with one another, they constitute **shear loading** (see Fig. 2–14C). Forces applied perpendicular to the long axis of a structure constitute **torsional loading.** When combined or **bending forces** are applied to a structure, both tensile and compressive stresses and strains are created. For

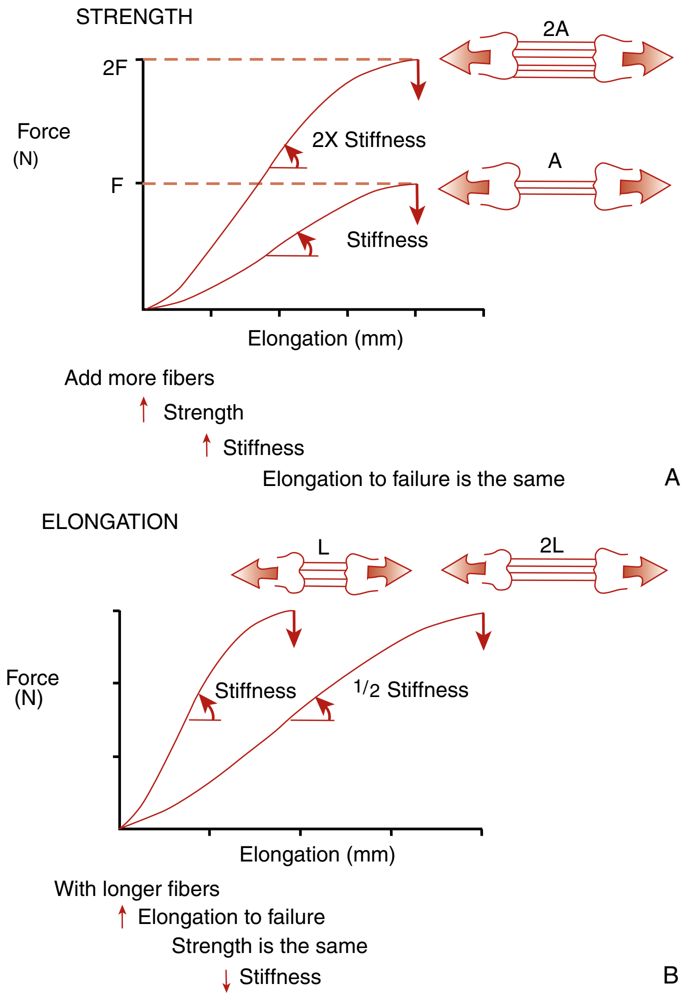


Figure 2–13 The size of a tissue (cross-sectional area and length) will affect its overall response to load. **A.** Increasing cross-sectional area means the tissues can withstand more force at any given length (i.e., it's more stiff). **B.** Increasing tissue length means it can elongate further under the same loading conditions (i.e., it's less stiff). *(From Butler DL, Grood ES, Noyes FR, et al: Biomechanics of ligaments and tendons. Exerc Sport Sci Rev 6:144, 1978, with permission from Lippincott Williams & Wilkins.)*

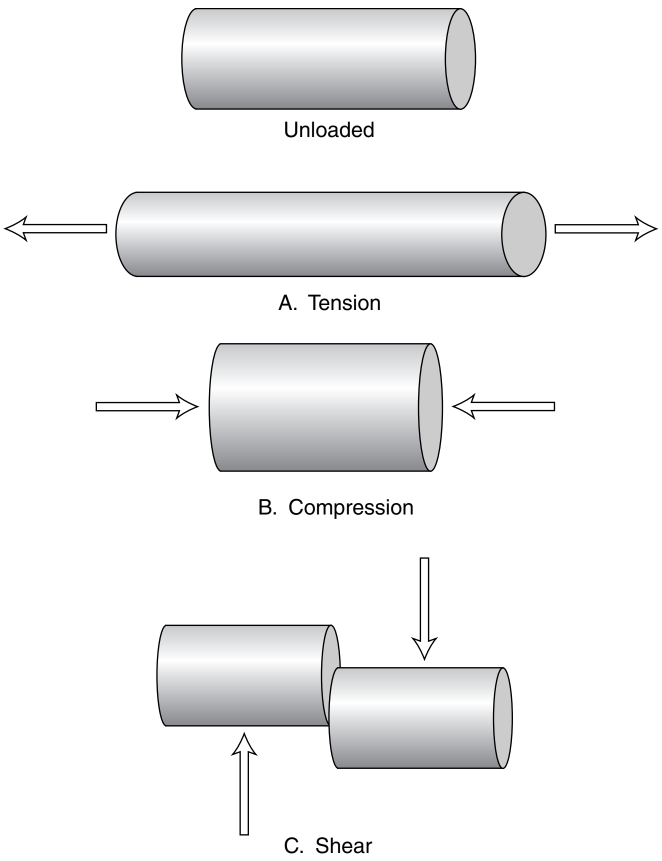


Figure 2–14 The various types of loads to which connective tissues can be subjected. **A.** Tensile loading. **B.** Compressive loading. **C.** Shear loading.

example, when a longitudinal force is applied to a long bone, tensile stress and strain develop on the convex side of the long axis of the bone and compressive stress and strain develop on the concave side.[83]

Because stress and strain are independent of size of the material, the stress-strain curve is said to reflect the **material properties** of the tissue.[53] With size accounted for, only changes in the material constituting the tissue will alter the stress-strain curve. The reason for calculating stresses and strains is to compare these material properties. The stress-strain curve can be used to compare the strength properties of one material with that of another material or to compare the same tissue under different conditions (e.g., ligaments before and after immobilization). The stress-strain curve contains the same defining points (A, B, and C) as the load-deformation curve, but the shape of the curve and the amount of stress and strain will vary with the composition of the material (Fig. 2–15). The curve will be flatter in more elastic materials and steeper in stiffer materials. Weaker materials won't resist as much stress but will elongate further (they are less stiff); thus, the values on the x-axis will be lower, and the values on the y-axis higher, than those shown in Figure 2–15. For stronger materials, the reverse would be true.

Young's Modulus

The **Young's modulus (E),** or **modulus of elasticity,** of a material under compressive or tensile loading is represented by the slope of the linear portion of the curve between point A and point B in Figure 2–15. The modulus of elasticity is a measure of the material's stiffness (its resistance to external loads).[84] A value for stiffness can be found by dividing the change in (Δ) stress by the change in (Δ) strain for any two consecutive sets of points in the elastic range of the curve. The inverse of stiffness is **compliance.** If the slope of the curve is steep and the modulus of elasticity is high, the material exhibits high stiffness and low compliance. If the slope of the curve is gradual and the modulus of elasticity is low, the material exhibits low stiffness and a high compliance.

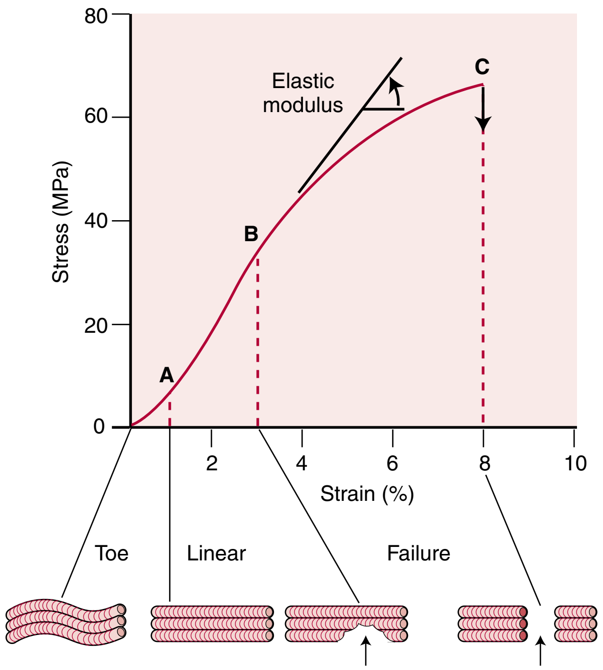


Figure 2–15 An example of a stress-strain curve for collagenous materials. The results are independent of tissue dimensions and thus reflect the material of which the tissue is made. A–B is the elastic region, and B–C is the plastic region. Failure usually occurs at about 8% to 10% strain. *(From Butler DL, Grood ES, Noyes FR, et al: Biomechanics of ligaments and tendons. Exercise and Sport Science Reviews 6:125, 1978, with permission from Lippincott Williams and Wilkins.)*

Example 2-2

Cortical bone has a high modulus of elasticity, high stiffness, and low compliance, whereas subcutaneous fat has a low modulus of elasticity, low stiffness, and high compliance. The stiffness (elastic modulus) of cortical bone is between 17 and 25 GPa (gigaPascals), similar to that of wood.

Load Deformation and Stress-Strain Curves

Each material has its own unique stress-strain curve. A typical curve for tendons and extremity ligaments is presented in Figure 2–15.[53] The first region of the curve (0 to A) is called the **toe region.** Very little force is required to deform the tissue as the wavy crimp pattern is straightened out and PGs and GAGs allow interfibrillar sliding. In this region, a minimal amount of force produces a relatively large amount of deformation (elongation); stress is low, and the strain is typically in the 1% to 2% range. Clinical examples involving the toe region include tests for ligament integrity (for noninjured ligaments) and the slack taken up in a tendon by its attached muscle before the force is transmitted to a bone.

The second portion of the curve A to B is the elastic region, in which elongation (strain) has a linear relationship with stress. Each additional unit of applied force creates an equal stress and strain in the tissue. In this region of the curve, collagen fibrils are being stretched and are resisting the applied force. Thus, the linear region of the stress-strain curve reflects the type of collagen, the fibril size, and the cross-linking among collagen molecules. When the load is removed, the ligament or tendon will return to its prestressed dimensions, although this return will take some time. This level of loading includes the stresses and strains that occur with normal activities and typically extends to about 4% strain.[53,85,86]

In the third region (B to C, the plastic region), the failure of collagen fibers **(microfailure)** begins, and the ligament or tendon is no longer capable of returning to its original length after the force is removed. Clinical examples include grade I and II ligament sprains and tendon strains. Recovery after this level of loading requires considerable time because it involves aspects of healing such as synthesis of new tissue and cross-linking of collagen molecules.

If force continues to be applied beyond the plastic region, the remaining collagen fibrils experience increased stress and rapidly rupture sequentially, creating overt failure **(macrofailure)** of the tissue. In the case of a ligament or tendon, if the failure occurs in the middle of the structure through a disruption of the connective tissue fibers, it is called a **rupture.** If the failure occurs at the bony attachment of the ligament or tendon, it is called an **avulsion.** When failure occurs within bony tissue, it is called a **fracture.** Slow loading rates tend to create avulsions or fractures, whereas fast loading rates create midsubstance tears.

Each type of connective tissue is able to withstand a different percentage of strain before failure. In general, ligaments and tendons are able to deform more than cartilage, and cartilage is able to deform more than bone. However, the total deformation also depends on the size (length, width, or depth) of the structure.

CASE APPLICATION

Immobilization Alters Patient's Mechanical Properties

case 2–6

After immobilization, all George Chen's connective tissues will have altered mechanical properties. The tissues may easily be re-injured, even under previously normal loading conditions. This means loading should be started at low levels and increased gradually, with adequate time between sessions to allow the tissue mechanical properties to recover, in case tissues have been loaded into the elastic region.

Continuing Exploration 2-2:

Can You Find the Correct Location on the Stress-Strain Curve?

Where on the stress-strain curve do you think a ligament may have been loaded to create the following injuries?

- Grade I sprain: injury to a few fibers of the ligament
- Grade II sprain: injury to a variable amount of fibers, a partial tear
- Grade III sprain: complete rupture of the ligament

Viscoelasticity

All connective tissues are **viscoelastic** materials: they combine the properties of elasticity and **viscosity,** making their behavior time-, rate-, and history-dependent.[83,84] Elasticity refers to the material's ability to return to its original length or shape after the removal of a deforming load. The term *elasticity* implies that length changes or deformations are directly proportional to the applied forces or loads. The elastic qualities in connective tissues primarily depend on collagen and elastin content and organization.

When a material is stretched, work is done (work = force × distance) and energy in the stretched material increases. An elastic material stores this energy and readily returns it as work so that the stretched elastic material can recoil immediately to its original dimensions. For example, during many functional activities (e.g., walking, running, jumping), a lengthening (eccentric) muscle contraction stretches the attached tendon, and this elastic energy is returned during the subsequent shortening (concentric) contraction of the muscle-tendon unit.[85–87]

Viscosity refers to a material's resistance to flow. It is a fluid property, and depends on the PG and water composition of the tissue. A tissue with high viscosity will exhibit high resistance to deformation, whereas a less viscous fluid will deform more readily. When forces are applied to viscous materials, the tissues exhibit time-dependent and rate-dependent properties. Viscosity diminishes as temperature rises or loads are slowly applied and increases as pressure increases or loads are rapidly applied.

Example 2-3

Motor oils with different viscosities are used when more or less resistance to deformation is required. When temperatures are high, the oil's resistance will be lower and a more viscous type of oil (10-W-30) may be used. During winter, a less viscous oil (5-W-30) will allow the oil to deform more readily and coat the engine surfaces.

Time-Dependent and Rate-Dependent Properties

Viscoelastic materials are capable of undergoing deformation under either tensile or compressive forces and returning to their original state after removal of the force. However, their viscous qualities make the deformation and return time-dependent. A viscoelastic material possesses characteristics of **creep, stress-relaxation, strain-rate sensitivity,** and **hysteresis.**[84]

Creep

If a force is applied to a tissue and maintained at the same level while the deformation produced by this force is measured, the deformation will gradually increase. Force remains constant while length changes. For example, if you hang a weight on the end of an elastic band, you will get an immediate elastic deformation. However, it will also gradually elongate further over time. Connective tissues also will gradually elongate (creep) after an initial elastic response to a constant tensile load and then gradually return to their original length (recovery) after the load is removed. In a clinical setting, this might apply to stretching shortened tissue: The clinician applies a constant force and the tissue gradually elongates. For cartilage and bone, compressive loading is used to test creep, and so the depth of indentation represents creep and recovery (Fig. 2–16A).

Stress-Relaxation

If a tissue is stretched to a fixed length while the force required to maintain this length is measured, the force needed will decrease over time. Length remains constant while force decreases (Fig. 2–15B). In a clinical setting, a therapist may perceive this as a reduced resistance to stretch (less force is required to maintain tissue length).

Hysteresis

When the force and length of the tissues are measured as force is applied (loaded) and removed (unloaded), the resulting load-deformation curves do not follow the same path. Not all of the energy gained as a result of the lengthening work (force × distance) is recovered during the exchange from energy to shortening work. Some energy is lost, usually as heat (Fig. 2–16C).[85]

Strain-Rate Sensitivity

Most tissues behave differently if loaded rapidly or slowly.[53,62] When a load is applied rapidly, the tissue is stiffer, and a larger peak force can be applied to the tissue than if the load was applied slowly. The subsequent stress-relaxation also will be larger than if the load was applied slowly. Creep will take longer to occur under conditions of rapid loading (Fig. 2–16D).

Example 2-4

To increase the length of a connective tissue structure with minimal risk of injury, a load should be applied slowly to the maximum tolerable level and then maintained while creep occurs. If stress-relaxation occurs (i.e., the applied force decreases), the force can be returned to the original level and maintained as further creep occurs. To avoid injury, the total length change should probably be no more than 2% to 6% strain.

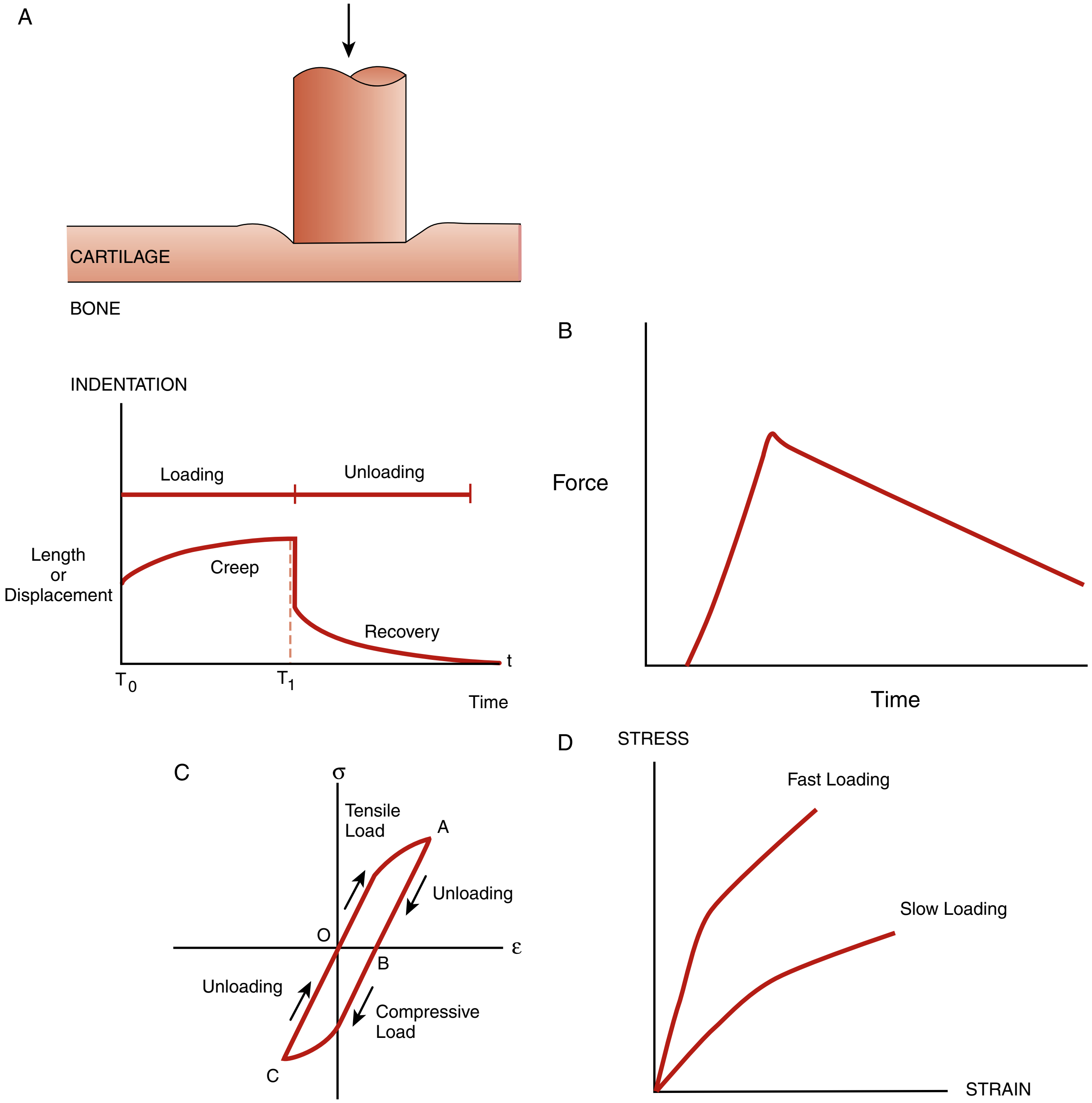


Figure 2–16 Time- and rate-dependent properties of dense connective tissues. **A.** Creep: When the tissue is loaded to a fixed force level, and length is measured, the latter increases with time (T_0 to T_1) and the tissue recovers its original length in a nonlinear manner (T_1 to T_0). **B.** Force or stress-relaxation: If the tissue is stretched to a fixed length and held there, the force needed to maintain this length will decrease with time. **C.** Hysteresis: As the tissue is loaded and unloaded, some energy is dissipated through tissue elongation and heat release. **D.** If the tissue is loaded rapidly, more energy (force or stress) is required to deform the tissue. *(From Oskaya N, Nordin M: Fundamentals of Biomechanics, Equilibrium Motion and Deformation [ed. 2]. New York, Springer-Verlag, 1999, with permission from the publisher as well as the author, Margarita Nordin.)*

Properties of Specific Tissues

Bone

Cortical bone is stiffer than cancellous (trabecular) bone; that is, cortical bone can withstand greater stress, while deforming less, than cancellous bone. Cancellous bone can sustain strains of 75% before failing in vivo, but cortical bone will fail if strain exceeds 2%.[83] When cortical bone is loaded in compression, longitudinal sections of the bone show the greatest strength. In tensile testing of the femur, longitudinal sections display twice the modulus of elasticity of transverse sections. Cortical bone can withstand much higher compressive stresses before failing than tensile stresses. The compressive strength of trabecular bone is also greater than the tensile strength. In other words, bone can withstand greater stress, and will undergo less strain, in compression than in tension.[83]

Large loads applied over short periods of time will produce high stresses and low strains (strain-rate sensitivity), whereas loads applied over longer periods of time will produce lower stresses and higher strains (creep and stress-relaxation). The physiologic response of trabecular bone to increased loading is hypertrophy. If loading is decreased or absent, trabeculae become smaller and weaker. The rate, frequency, duration, magnitude, and type of loading all affect bone. Repeated loadings, either high numbers of low loads (stress fracture) or one application of a high load (fracture), can cause permanent strain and bone failure. Bone loses stiffness and strength with repetitive loading as a result of **creep strain.** Creep strain occurs when a tissue is loaded again (and again) while the material is undergoing creep.

Tendons

Tendons exhibit creep when subjected to tensile loading, most often when stress is created via muscle contraction. If the muscle to which the tendon is attached generates just enough force to straighten out the crimp in the tendon, the tendon will be loaded in the toe region of the stress-strain curve. In this region, there is little stress and less than 2% strain.[53] Increases in force that stretch the now-straightened collagen fibers bring the tendon into the linear (elastic) region of the curve, where there is a linear relationship between the applied force and resulting tissue deformation. In this region, the collagen fibrils are loaded directly and may be damaged. If loading increases higher into the linear region, the first tissue damage is slippage between collagen molecules within fibrils, then interfibrillar slippage between fibrils, and, finally, gross disruption of collagen fibers. The fibers are not perfectly parallel, so they are not equally straightened as the load is increased. The fibers that become straightened first may be the first to fail, or the smaller, weaker fibrils may rupture. Most normal activities load tendons in the toe region and in the first part of the linear region.[53]

The cross-sectional area, the material, and the length of the tendon determine the amount of force that a tendon can resist and the amount of elongation that it can undergo.[43,58] Under normal conditions, tendons with greater cross-sectional areas should be able to withstand larger forces than tendons with smaller cross-sectional areas, unless they are composed of weaker material; thus, the Achilles tendon can probably be assumed to be stronger than the palmaris longus tendon.[88] Under unusual conditions, this relationship may not be true. For example, a healing tendon may be much larger in diameter than an uninjured tendon, but because the injured tendon contains less collagen, smaller fibrils, and fewer cross-links, it may actually be weaker than its smaller counterpart.[58]

The physiological response of tendons to intermittent tension (application and release of a tensile force) is a moderate increase in thickness and strength.[88] Differences in stress-strain curves among different tendons reflect differences in the proportion of type I and type III collagen, differences in cross-linking, maturity of collagen fibers, organization of fibrils, variations in ground substance concentration, and level of hydration. Because of the change in composition as the tendon inserts into the bone, such that stresses are not uniformly distributed, the enthesis is a common site of degenerative changes and injury.[55,56] The myotendinous junction (MTJ) appears to be stronger, so that although this a common site for muscle strains and pulls, the injury is typically on the muscle side of the MTJ.[89] However, because the MTJ depends on interdigitation of muscle and tendon for its strength, any injury that distorts the form of the MTJ may decrease its tensile strength and predispose it to further injury.

Under normal conditions, the tendon is most vulnerable at either end rather than in its midsubstance. Healthy tendons rarely rupture under normal conditions and are able to withstand large tensile forces without injury.[90] However, if tendons are weakened, they are more likely to be injured. Tendons subject to immobilization show atrophy at the MTJ, with a loss of infolding, and a decrease in collagen concentration and cross-linking.[89–91] Exposure to corticosteroids, nutritional deficiencies, hormonal imbalance, dialysis, chronic loading into the high linear region of the stress-strain curve with inadequate time for recovery, and sudden large loads may predispose the tendon to injury at previously physiological levels of loading. In other words, the same load now produces more deformation in the tendon.

Tendons adapt readily to changes in the magnitude and direction of loading. Tendons subject to continual compressive forces will alter their composition to resemble cartilage, and their tensile strength may decline.[92] Conversely, tendons subjected to tensile loads, especially physiological loads over long periods of time, will increase in size, collagen concentration, and collagen cross-linking.[93–95] Progressive loading programs are successfully used to treat tendon dysfunction, presumably through inducing changes in tendon composition.[58,78,97–99]

Ligaments

Tendons are difficult to test mechanically (it is hard to get a solid grip on the ends), so most of our knowledge of connective tissue response to tensile loading comes from ligament testing.[100] Tendons and ligaments are very similar mechanically. Ligaments must withstand forces in all directions, so they have a more varied orientation of collagen fibrils that makes them slightly less resistant to tensile stress but better able to function within a range of load directions without being damaged. This is another example of form (in this case, collagen fibril orientation) following function.

The physiological response of ligaments to intermittent tension (application and release of a tensile force) is an increase in thickness and strength. Immobilized ligaments weaken rapidly and can take more than 12 months to recover their mechanical properties.[16,17]

Cartilage

Three forces interact in cartilage to resist applied load: (1) stress developed in the fibrillar portion of extracellular matrix (type II collagen), (2) swelling pressures developed in the fluid phase (PGs and water), and (3) frictional drag resulting from fluid flow through the extracellular matrix.[32] Compression of cartilage reduces the volume of the cartilage and increases the pressure, causing outward flow of interstitial fluid. Fluid flow through the extracellular matrix creates frictional resistance to the flow within the tissues (frictional drag). Exudation of fluid occurs rapidly at first, causing a concomitant rapid rate of deformation. Subsequently, fluid flow and deformation gradually diminish and cease when compressive stress in cartilage balances the applied load.[62,66,67] Magnetic resonance imaging (MRI) has made it possible to study changes in cartilage volume and thickness in the joints of living subjects. In an MRI study of the knee joints of eight volunteers, Eckstein and colleagues[101] found that up to 13% of the fluid was displaced from the patellar cartilage 3 to 7 minutes after exercise (50 knee bends).

Tensile stresses called **hoop stresses** are created in the superficial collagen network as the compressed PGs and water push against the collagen fibers.[63] The tensile behavior of cartilage collagen is similar to that of ligaments and tendons but is caused in different ways. The nonlinear behavior in the toe region of the curve is thought to be caused by the drag force between the collagen meshwork and the PGs; in ligaments and tendons, the nonlinear behavior in the toe region is attributed to the straightening of collagen fibers. As in ligaments and tendons, cartilage collagen fibers become taut in the linear region of the curve and are stretched. Cartilage specimens from the different zones of cartilage (zones I, II, and III) (see Fig. 2–11) show differences in tensile behavior, presumably due to differences in the orientation of the collagen fibers among the zones.[63] Cartilage resistance to shear depends on the amount of collagen present because PGs provide little resistance to shear. Shear stresses are apt to develop at the interface between the calcified cartilage layer and the subchondral bone.

Concept Cornerstone **2-7**

Resistance of Tissues to Compression and Tension

Resistance of connective tissues to compression and tension depends on an intact collagen network that can resist tensile stress. In tendons and ligaments, the tensile stress is directly caused by the applied load. In cartilage, the tensile stress is created by the fluid pressure of the water and PGs in the interfibrillar part of the extracellular matrix pushing against the collagen meshwork, which must be firmly anchored in bone at either end. Bone depends on both organic and inorganic components to resist tension and compression.

This description of the properties of connective tissue structures is aimed at providing an introduction to the nature of the joint components and should facilitate an understanding of basic joint structure and function. The following two sections, Complexity of Human Joint Design and Joint Function, include the traditional classification system for human joints, as well as a detailed description of synovial joint structure and function.

COMPLEXITY OF HUMAN JOINT DESIGN

An appreciation of the complexities of human joint design may be gained by considering the nature of the bony components and the functions that the joints must serve. The human skeleton has about 200 bones that are connected by joints. Bones vary in size from the pea-sized distal phalanx of the little toe to the long femur of the thigh. The shape of the bones varies from round to flat, and the contours of the ends of the bones vary from convex to concave. The task of designing a series of joints to connect these varied bony components to form stable structures would be difficult. The task of designing joints that are capable of working together to provide both mobility and stability for the total structure represents an engineering problem of considerable magnitude.

Joint designs in the human body vary from simple to complex. Simple human joints usually have stability as a primary function; the more complex joints usually have mobility as a primary function. However, most joints in the human body serve a dual mobility-stability function and must also provide dynamic stability. The human stability joints are similar in design to table joints in that the ends of the bones may be contoured either to fit into each other or to lie flat against each other. The bracing of human joints is accomplished through the use of joint capsules, ligaments, and tendons. These are components of synovial joints, which are designed primarily for mobility. Synovial joints are constructed so that the ends of the bony components are covered by hyaline cartilage and enclosed in a synovial sheath and fibrous layer that together constitute the joint capsule. The capsules, ligaments, and tendons located around mobility (synovial) joints not only help to provide stability for the joint but also guide, limit, and permit motion. Wedges of cartilage, called **menisci, discs, plates,** and **labra,** in synovial joints help increase stability, provide shock absorption, and facilitate motion. A lubricant, synovial fluid, is secreted in all synovial joints to help reduce friction between the articulating surfaces.[102]

In the traditional method of joint classification, the joints (**arthroses** or **articulations**) of the human body are divided into two broad categories on the basis of the type of materials and the methods of uniting the bony components. Subdivisions of joint categories are based on materials used, the shape and contours of the articulating surfaces, and the type of motion allowed. The two broad categories of arthroses are **synarthroses** (nonsynovial joints) and **diarthroses** (synovial joints).[6,29]

Synarthroses

The material connecting the bony components in synarthrodial joints is interosseous connective tissue (fibrous and/or cartilaginous). Synarthroses are grouped into two divisions according to the type of connective tissue in the union of bone to bone: **fibrous joints** and **cartilaginous joints.** The connective tissue directly unites one bone to another, creating a solid connective tissue-bone interface.

Fibrous Joints

In fibrous joints, the fibrous tissue directly connects bone to bone. Three different types of fibrous joints are found in the human body: **sutures, gomphoses,** and **syndesmoses.** A suture joint is one in which two bony components are united by a collagenous sutural ligament or membrane. The ends of the bony components are shaped so that the edges interlock or overlap one another. This type of joint is found only in the skull and, early in life, allows a small amount of movement. Fusion of the two opposing bones in suture joints occurs later in life and leads to the formation of a bony union called a **synostosis.**

Example 2-5

Coronal Suture

The serrated edges of the parietal and frontal bones of the skull are connected by a thin fibrous membrane (the sutural ligament) to form the coronal suture (Fig. 2–17). At birth, the fibrous membrane allows some motion for ease of passage through the birth canal. Also, during infancy, slight motion allows for growth of the brain and skull. In adulthood, the bones grow together to form a synostosis and little or no motion is possible.

A **gomphosis joint** is a joint in which the surfaces of bony components are adapted to each other like a peg in a hole. In this type of joint, the component parts are connected by fibrous tissue. The only gomphosis joint that exists in the human body is the joint between a tooth and either the mandible or maxilla.

Example 2-6

The conical process of a tooth is inserted into the bony socket of the mandible or maxilla. In the adult, the loss of teeth is, for the most part, caused by disease processes affecting the connective tissue that cements or holds the teeth in approximation to the bone. Under normal conditions in the adult, these joints do not permit motion between the components.

A **syndesmosis** is a type of fibrous joint in which two bony components are joined directly by an interosseous ligament, a fibrous cord, or an aponeurotic membrane. These joints usually allow a small amount of motion.

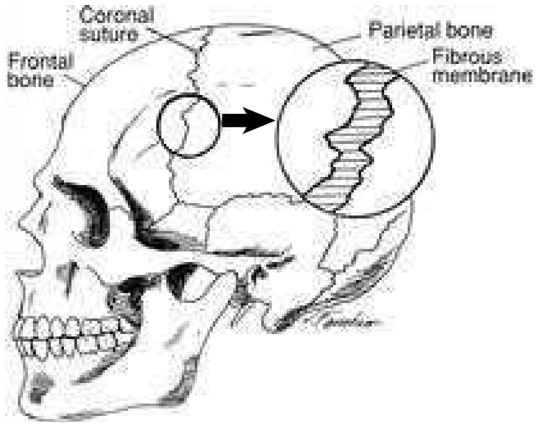


Figure 2–17 The coronal suture. The frontal and parietal bones of the skull are joined directly by fibrous tissue to form a synarthrodial suture joint.

Example 2-7

The shaft of the tibia is joined directly to the shaft of the fibula by an interosseous membrane (Fig. 2–18). A slight amount of motion at this joint accompanies movement at the ankle joint.

CASE APPLICATION

Disruption of the Interosseous Membrane

case 2–7

In George's ankle fracture, the interosseous membrane was disrupted, allowing some separation of the tibia and fibula. Restoration of normal talocrural anatomy is crucial after such injuries, which is one of the major reasons open reduction and internal fixation is used to treat these fractures. The surgeon must be careful that the screw connecting the tibia and fibula is not too tight, in order to leave enough space to accommodate the talus in full dorsiflexion. The "give" of the joint may take some time to recover, making it difficult for patients like George to jog or run comfortably for some months.

Cartilaginous Joints

The materials connecting the bony components in cartilaginous joints are fibrocartilage and/or hyaline cartilage. These materials directly unite one bony surface to another, creating a bone-cartilage-bone interface. The two types of cartilaginous joints are **symphyses** and **synchondroses.**

In a **symphysis joint (secondary cartilaginous joint),** the two bony components are covered with a thin lamina of hyaline cartilage and directly joined by fibrocartilage in the form of discs or pads. Examples of symphysis joints include the intervertebral joints between the bodies of the vertebrae, the joint between the manubrium and the sternal body, and the symphysis pubis in the pelvis.

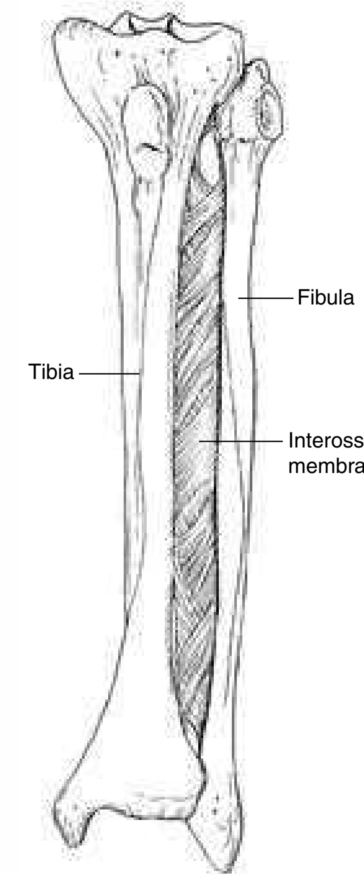


Figure 2–18 The shafts of the fibula and tibia are joined directly by a membrane to form a synarthrodial syndesmosis.

Example 2-8

The Symphysis Pubis

The two pubic bones of the pelvis are joined by fibrocartilage. This joint serves as a weight-bearing joint and is responsible for withstanding and transmitting forces; therefore, under normal conditions, very little motion is permissible or desirable. During pregnancy, when the connective tissues are softened, some slight separation of the joint surfaces occurs to ease the passage of the baby through the birth canal. However, the symphysis pubis is considered to be primarily a stability joint with the thick fibrocartilage disc forming a stable union between the two bony components (Fig. 2–19A).

A **synchondrosis (primary cartilaginous joint)** is a type of joint in which the material used to connect the two components is hyaline cartilage. The cartilage forms a bond between two ossifying centers of bone. The function of this type of joint is to permit bone growth while also providing stability and allowing a small amount of mobility. Some of these joints are found in the skull and in other areas of the body at sites of bone growth. When bone growth is complete, some of these joints ossify and convert to bony unions (synostoses).

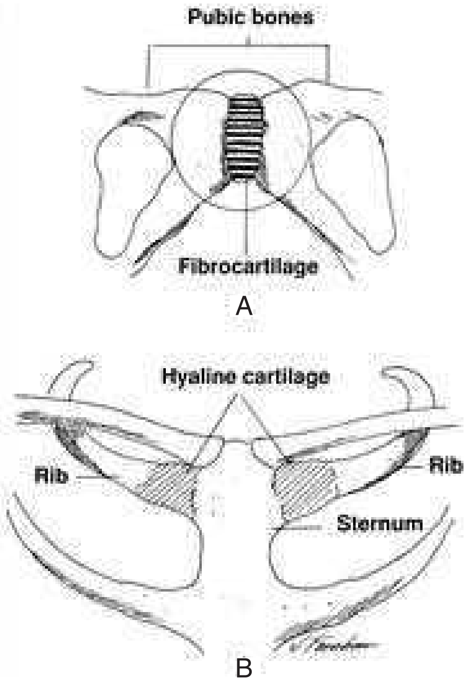


Figure 2–19 Cartilaginous joints. **A.** The two pubic bones of the pelvis are joined directly by fibrocartilage to form a symphysis joint called the symphysis pubis. **B.** The first rib and the sternum are connected directly by hyaline cartilage to form a synchondrosis joint called the first chondrosternal joint.

Example 2-9

The First Chondrosternal Joint

The adjacent surfaces of the first rib and sternum are connected directly by articular cartilage (Fig. 2–19B).

Diarthroses

The joint construction in diarthrodial (synovial) joints differs from that found in synarthrodial joints. In synovial joints, the ends of the bony components are free to move in relation to one another because no connective tissue directly connects adjacent bony surfaces. The bony components are *indirectly connected to one another by means of a joint capsule that encloses the joint.* All synovial joints are constructed in a similar manner and have the following features: (1) a joint capsule that is composed of two layers[8]; (2) a joint cavity that is enclosed by the joint capsule; (3) synovial tissue that lines the inner surface of the capsule; (4) synovial fluid that forms a film over the joint surfaces; and (5) hyaline cartilage that covers the surfaces of the enclosed contiguous bones[13] (Fig. 2–20). Synovial joints may also include accessory structures, such as fibrocartilaginous discs, plates, or menisci; labra; fat pads; and ligaments. Articular discs, menisci, and the synovial fluid help to prevent excessive compression of opposing joint surfaces by spreading applied forces over larger areas. Articular discs and menisci often occur between articular surfaces where congruity is low, thus increasing surface

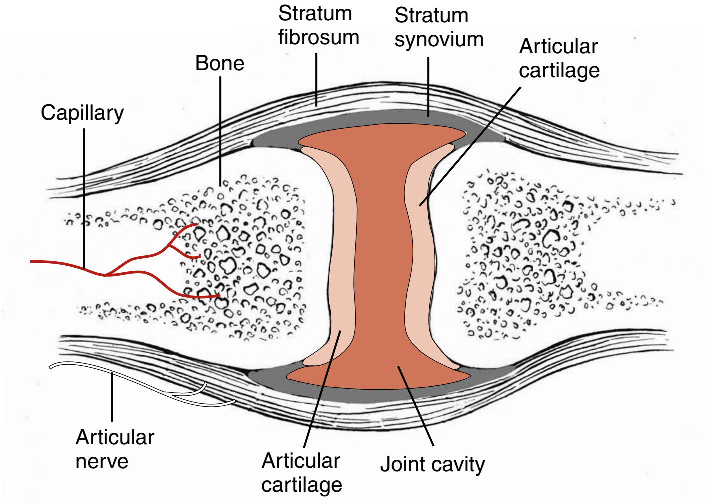


Figure 2–20 A typical diarthrodial joint.

contact area. In some cases, articular discs extend all the way across a joint and actually divide it into two separate cavities, such as the articular disc at the distal radioulnar joint. Menisci usually do not divide a joint but provide lubrication and increase congruity. Ligaments (and the tendons associated with muscles crossing the joint) play an important role in keeping joint surfaces aligned and in guiding motion. Excessive separation or translation of joint surfaces is limited by passive tension in ligaments, the fibrous joint capsule, and tendons (passive stability).[9] Active tension in muscles (dynamic stability) also limits the separation or translation of joint surfaces.

CASE APPLICATION

Joints Affected by Our Patient's Injury

case 2–8

Which joints and joint structures are directly and indirectly affected in the case of George Chen? Consider the talocrural, subtalar, transverse tarsal (talonavicular and calcaneocuboid), and first metatarsophalangeal (MTP) joints.

Joint Capsule

Joint capsules vary considerably in both thickness and composition. The capsule enclosing the shoulder joint is thin, loose, and redundant, sacrificing stability for mobility. Other capsules, such as the hip joint capsule, are thick and dense and thus favor stability over mobility. The thickness, fiber orientation, and composition of the capsule depend on the stresses placed on the joint, illustrating the dynamic nature of the joint capsule. For example, in portions of the capsule that are subjected to compression forces, the capsule may become fibrocartilaginous.[7,8] In patients with shoulder instability in which the joint capsule is subjected to repeated tensile deformation, collagen fibrils and elastin fibers are larger and denser compared to normal capsules, adaptations that may increase capsular strength and resistance to stretching deformation.[9]

The fibrous capsule may be reinforced by and, in some instances, incorporate ligaments or tendons as part of the capsule. For example, the capsule of the proximal interphalangeal joint of the fingers is reinforced by collateral ligaments superficially and a central slip of the extensor tendon superficially and posteriorly.[10]

The joint capsule is composed of two layers: an outer layer called the **stratum fibrosum** and an inner layer called the **stratum synovium** (see Fig. 2–20). The stratum fibrosum, sometimes referred to as the **fibrous capsule,** is composed of dense fibrous tissue. Water accounts for about 70% of tissue weight; collagen and elastin account for about 90% of the tissue dry weight.[8,10] The predominant type of collagen is type I, which is arranged in parallel bundles. As the capsule nears its insertion to bone, the tissue changes to fibrocartilage and then mineralized fibrocartilage and bone, similar to the fibrocartilaginous (direct) type of ligament or tendon enthesis. The stratum fibrosum is poorly vascularized but richly innervated by joint receptors located in and around the capsule (see Table 2–7).[12]

The inner layer (stratum synovium) is the lining tissue of the capsule. It also consists of two layers: the **intima** and the **subsynovial tissue.** The intima is the layer of cells that lines the joint space. It is composed of a layer of specialized fibroblasts known as **synoviocytes** that are arranged one to three cells deep and set in a fiber-free intercellular matrix.[11] Two types of synoviocytes are generally recognized: type A and type B.[2] **Type A synoviocytes** are macrophage-like cells with prominent Golgi apparatus but sparse granular endoplasmic reticulum. Type A cells are primarily responsible for the removal of debris from the joint cavity. During phagocytosis, type A cells synthesize and release lytic enzymes that have the potential to damage joint tissues.

Type B synoviocytes have abundant granular endoplasmic reticulum and are twice as numerous as type A cells in normal synovium.[13] Type B cells produce substances that inhibit the lytic enzymes, and are responsible for initiating immune responses through the secretion of antigens. Both types of cells synthesize the hyaluronic acid component of the synovial fluid, as well as the matrix in which the cells are embedded. Type A and B cells also secrete a wide range of cytokines, including multiple growth factors. The interplay of the cytokines acting as stimulators or inhibitors of synoviocytes allows structural repair of synovium, response to foreign or autologous antigens, and tissue destruction.[14]

The subsynovial tissue lying outside the intima is a loose network of highly vascularized fibrous connective tissue. It attaches to the margins of the articular cartilage through a transitional zone of fibrocartilage and blends with the periosteum covering the intracapsular portions of the bones. Its cells differ from the intima cells in that they are more spindle-shaped, are more widely dispersed among collagen fibrils, and produce matrix collagen.[103] The subsynovial tissue supports the intima and merges on its external surface

Table 2–7 **Joint Receptors**

TYPE	NAME	SENSITIVITY	LOCATION
I	Ruffini	Stretch—usually at extremes of extension	Fibrous layer of joint capsules on flexion side of joints, periosteum, ligaments, and tendons[10]
II	Pacini or pacini-form	Compression or changes in hydrostatic pressure and joint movement[1]	Located throughout joint capsule, particularly in deeper layers and in fat pads
III	Golgi, Golgi-Mazzoni	Pressure and forceful joint motion into extremes of motion	Inner layer (synovium) of joint capsules, ligaments, and tendons[10]
IV	Unmyelinated free nerve endings	Non-noxious and noxious mechanical stress or biomechanical stress	Located around blood vessels in synovial layer of capsule and in adjacent fat pads and collateral ligaments, tendons, and the periosteum

with the fibrous capsule. The intima is richly endowed with capillary vessels, lymphatic vessels, and nerve fibers. The blood vessels in the subsynovial tissue transport oxygen, nutrients, and immunologic cells to the joint.

Branches of adjacent sensory nerves and branches of nerves from muscles near the joint penetrate the fibrous joint capsule.[12] Large-diameter sensory efferent nerves and thinly myelinated nerves are present in the fibrous capsule; nonmyelinated C-type fibers are found in the synovium. The joint receptors found in the fibrous joint capsule are sensitive to stretching or compression of the capsule, as well as to any increase in internal pressure due to increased production of synovial fluid (joint swelling).

Most of the joint receptors in the knee are located in the subsynovial layer of the capsule close to the insertions of the anterior cruciate ligament (ACL). Mechanoreceptors (predominantly Ruffini receptors) in the subsynovial capsule and ACL respond primarily to the stretch involved in terminal knee extension. Pacini receptors are reported less frequently and are thought to be activated by compression. Free nerve endings are more numerous than mechanoreceptors and function as nociceptors that react to inflammation and pain stimuli. Afferent free nerve endings in joints not only transmit information but also serve a local effector role by releasing neuropeptides.[12,15]

Synovial Fluid

The thin film of synovial fluid that covers the surfaces of the inner layer of the joint capsule and articular cartilage helps to keep the joint surfaces lubricated and reduces friction.[102,103] The fluid also provides nourishment for the hyaline cartilage covering the articular surfaces, as fluid moves in and out of the cartilage as compression is applied, then released. The composition of synovial fluid is similar to that of blood plasma, except that synovial fluid also contains hyaluronate (hyaluronic acid) and a glycoprotein called **lubricin.**[26] The hyaluronate component of synovial fluid is responsible for the viscosity of the fluid and is essential for joint lubrication. Hyaluronate reduces the friction between the synovial folds of the capsule and the articular surfaces.[26] Lubricin is the component of synovial fluid thought to be responsible for cartilage-on-cartilage lubrication; it also is thought to provide synovial fluid with the ability to dissipate energy.[11,102,104,105] Changes in the concentration of hyaluronate or lubricin in the synovial fluid will affect the overall lubrication and the amount of friction that is present. Many experiments have confirmed that articular coefficients of friction in synovial joints are far lower than those created with manufactured lubricants.[103] The lower the coefficient of friction, the lower the resistance to movement. Injections of hyaluronate and lubricin have been used successfully to alleviate symptoms of osteoarthritis in both hip and knee joints.

Normal synovial fluid appears as a clear, pale yellow, viscous fluid present in small amounts at all synovial joints.[105] There is a direct exchange between the vasculature of the stratum synovium and the intracapsular space, where nutrients can be supplied and waste products can be taken away from the joint by diffusion.[26] Usually, less than 0.5 mL of synovial fluid can be removed from large joints such as the knee.[6] However, when a joint is injured or diseased, the volume of the fluid may increase.[26] The synovial fluid, like all viscous substances, resists shear loads.[102] The viscosity of the fluid *varies inversely* with the joint velocity or rate of shear; that is, it becomes less viscous at high rates. Thus, synovial fluid is referred to as **thixotropic.** When the bony components of a joint are moving rapidly, the viscosity of the fluid decreases and provides less resistance to motion.[102] When the bony components of a joint are moving slowly, the viscosity increases and provides more resistance to motion. Viscosity also is sensitive to changes in temperature. High temperatures decrease the viscosity, whereas low temperatures increase the viscosity.[102]

Joint Lubrication

The minimal wear shown by normal cartilage, despite varied loads, is the result of the structure of the cartilage matrix and the presence of lubricating fluid.[103–105] A number of models have been proposed to explain how diarthrodial joints are lubricated under varying loading conditions. The general consensus is that no single model is adequate to explain human joint lubrication and that human joints are lubricated by two or more types of lubrication; the two basic types are **boundary lubrication** and **fluid-film lubrication.**[28]

Boundary lubrication occurs when each load-bearing surface is coated with a thin layer of large molecules that forms a gel that keeps the opposing surfaces from touching each other[102] (Fig. 2–21). The layers slide over each other more readily than they are sheared off the underlying surface. In human diarthrodial joints, these layers contain the lubricin that adheres to the articular surfaces.[28] This type of lubrication is considered to be most effective at low loads.[11]

Fluid-film lubrication involves a thin fluid film that provides separation of the joint surfaces. Surfaces lubricated by a fluid film typically have a lower coefficient of friction than do boundary-lubricated surfaces, and the fact that the coefficient of friction is very low in synovial joints suggests that some sort of fluid-film lubrication exists. Several models of fluid-film lubrication exist, including **hydrostatic (weeping) lubrication; hydrodynamic, squeeze-film lubrication;** and **elastohydrodynamic** (a combination of hydrodynamic and squeeze-film) and **boosted lubrication.**[102]

Hydrostatic or weeping lubrication is a form of fluid lubrication in which the load-bearing surfaces are held apart by a film of lubricant that is maintained under pressure (see Fig. 2–21). In engineering, the pressure is usually supplied by an external pump. In the human body, the pump action can be supplied by contractions of muscles around the joint or by compression from weight-bearing. Compression of articular cartilage causes the cartilage to deform and to “weep” fluid, which forms a fluid film over the articular surfaces. The fluid can only move into the joint, because the impervious layer of calcified cartilage keeps it from being forced into the subchondral bone.[65] When the load is removed, the fluid flows back into the articular cartilage through osmotic pressure created by the PGs and GAGs in the cartilage. This type of lubrication is most effective under conditions of high loading, but it works under most conditions.[69]

Hydrodynamic lubrication is a form of fluid lubrication in which a wedge of fluid is created when nonparallel opposing surfaces slide on each other. The resulting lifting pressure generated in the wedge of fluid and by the fluid’s viscosity keeps the joint surfaces apart. In squeeze-film lubrication, pressure is created in the fluid film by the movement of articular surfaces that are perpendicular to one another.[28] As the opposing articular surfaces move closer together, they squeeze the fluid film out of the area of impending contact. The resulting pressure created by the fluid’s viscosity keeps the surfaces separated. This type of lubrication is suitable for high loads maintained for a short duration.

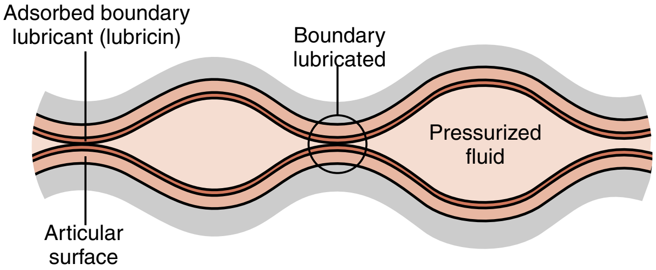


Figure 2–21 Joint lubrication models. Lubricin molecules coat the joint surfaces in boundary lubrication. The fluid film keeps joint surfaces apart in hydrostatic lubrication.

In the elastohydrodynamic model, the protective fluid film is maintained at an appropriate thickness by the elastic deformation of the articular surfaces. In other words, the elastic cartilage deforms slightly to maintain an adequate layer of fluid between the opposing joint surfaces. The boosted lubrication model suggests that pools of concentrated hyaluronate molecules are filtered out of the synovial fluid and are trapped in the natural undulations and areas of elastic deformation on the articular surfaces.[28]

These joint lubrication models provide a number of possible ways to explain how diarthrodial joints are lubricated. The variety of conditions under which human joints function makes it likely that more than one of the lubrication models is operating. Until a unified model of joint lubrication is proposed, proved, and accepted, the exact mechanisms involved in human joint lubrication will be subject to speculation.[102]

Concept Cornerstone 2-8

Essentials of Joint Lubrication

Lubrication depends on

- light irregularities in the joint surface that “trap” hyaluronate;
- lubricin molecules to create a fluid film over the cartilage surfaces;
- elastic deformation of the cartilage to maintain a layer of fluid between opposing cartilage surfaces;
- fluid being squeezed out of cartilage into the joint space as loading increases; and
- alternate application and removal of compressive forces to create fluid flow.

Diarthrodial Subclassifications

Traditionally, synovial joints have been divided mechanically into three main categories based on the number of axes about which “gross visible” motion occurs.[29–31] A further subdivision of the joints is made on the basis of the shape and configuration of the ends of the bony components. The three main traditional categories are **uniaxial, biaxial,** and **triaxial.** A uniaxial joint involves visible motion of the bony components in one plane around a single axis. The axis of motion usually is located near or in the center of the joint or in one of its bony components. Because uniaxial joints permit visible motion in only one plane or around only one axis, they are described as having one degree of freedom of motion. The two types of uniaxial diarthrodial joints found in the human body are **hinge joints** and **pivot (trochoid)** joints. A hinge joint is a type of joint that resembles a door hinge.

Example 2-10

Interphalangeal Joints of the Fingers

These hinge joints are formed between the distal end of one phalanx and proximal end of another phalanx (Fig. 2–22A). The joint surfaces are contoured so that motion can occur only in the sagittal plane (flexion and extension) around a coronal axis (see Fig. 2–22B).

A pivot (trochoid) joint is a type of joint constructed so that one component is shaped like a ring and the other component is shaped so that it can rotate within the ring.

Example 2-11

The Median Atlantoaxial Joint

The ring portion of the joint is formed by the atlas and the transverse ligament (Fig. 2–23). The odontoid process (dens) of the axis, which is enclosed in the ring, rotates within the osteoligamentous ring. Motion occurs in the transverse plane around a longitudinal (vertical) axis.

Biaxial diarthrodial joints are joints in which the bony components are free to move in two planes around two axes; these joints have two degrees of freedom. There are two types of biaxial joints in the body: **condyloid** and **saddle.** The joint surfaces in a condyloid joint are shaped so that the concave surface of one bony component slides over the convex surface of another component in two directions.

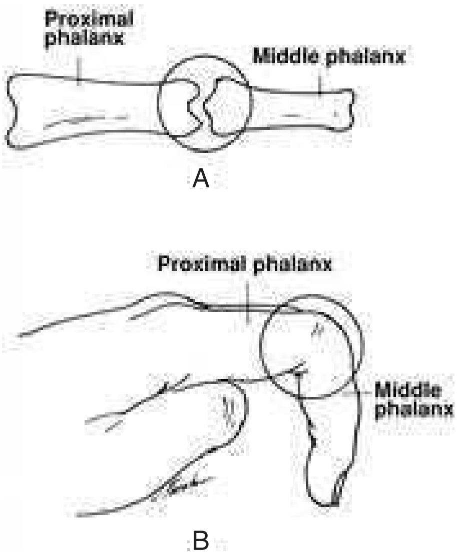


Figure 2–22 A uniaxial hinge joint. **A.** The interphalangeal joints of the fingers are examples of simple hinge joints. The joint capsule and accessory joint structures have been removed to show the bony components in the superior view of the joint. **B.** Motion occurs in one plane around one axis.

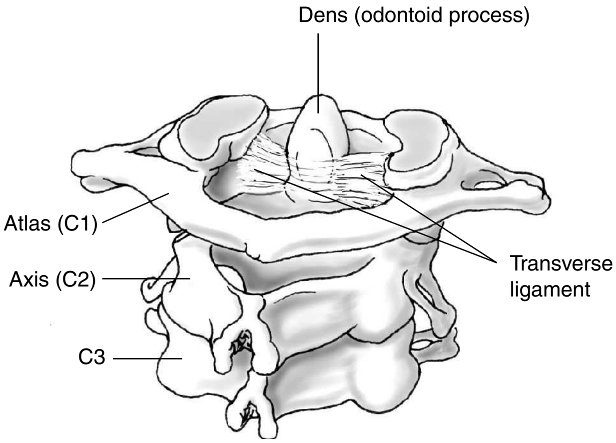


Figure 2–23 A pivot joint. The joint between the atlas, transverse ligament, and the dens of the axis is a uniaxial diarthrodial pivot joint called the median atlantoaxial joint. Rotation occurs in the transverse plane around a vertical axis.

A saddle joint is a joint in which each joint surface is convex in one plane and concave in the other, and these surfaces fit together like a rider on a saddle.

Example 2-12

Metacarpophalangeal Joint

The metacarpophalangeal joint is formed by the convex distal end of a metacarpal bone and the concave proximal end of the proximal phalanx (Fig. 2–24A). Flexion and extension at this joint occur in the sagittal plane around a coronal axis (Fig. 2–24B). Abduction is movement away from the middle finger, whereas adduction is movement toward the middle finger. Adduction and abduction occur in the frontal plane around an anteroposterior (A-P) axis (Fig. 2–24C).

Example 2-13

Carpometacarpal Joint of the Thumb

The carpometacarpal joint of the thumb is formed by the distal end of the trapezium and the proximal end of the first metacarpal. The motions available are flexion/extension and abduction/adduction.

Triaxial or **multiaxial** diarthrodial joints are joints in which the bony components are free to move in three planes around three axes. These joints have three degrees of freedom. Motion at these joints may also occur in oblique planes. The two types of joints in this category are **plane joints** and **ball-and-socket joints.** Plane joints have a variety of surface configurations and permit gliding between two or more bones.

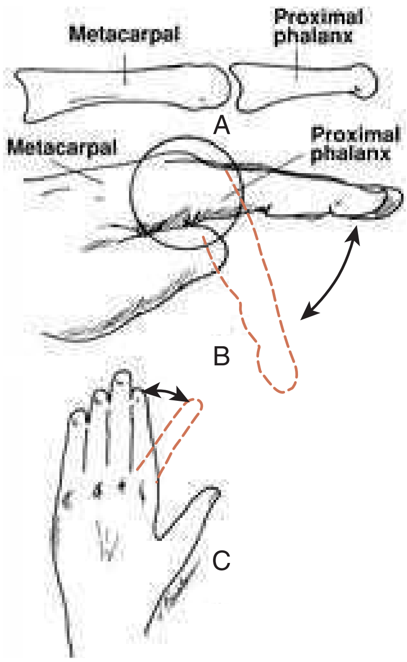


Figure 2–24 A condyloid joint. **A.** The metacarpophalangeal joints of the fingers are biaxial condyloid joints. The joint capsule and accessory structures have been removed to show the bony components. Motion at these joints occurs in two planes around two axes. **B.** Flexion and extension occur in the sagittal plane around a coronal axis. **C.** Abduction and adduction occur in the frontal plane around an A-P axis.

Example 2-14

Carpal Joints

Plane joints are found between the adjacent surfaces of the carpal bones. The adjacent surfaces may glide on one another or rotate with regard to one another in any plane.

Ball-and-socket joints are formed by a ball-like convex surface being fitted into a concave socket. The motions permitted are flexion/extension, abduction/adduction, rotation, and combinations of these movements.

Example 2-15

Hip Joint

The hip joint is formed by the head of the femur and a socket called the acetabulum (Fig. 2–25A). The motions of the flexion/extension occur in the sagittal plane around a coronal axis (Fig. 2–25B). Abduction/adduction occurs in the frontal plane around an anteroposterior axis (Fig. 2–25C), whereas rotation of the femur occurs in the transverse plane around a longitudinal axis (Fig. 2–25D).

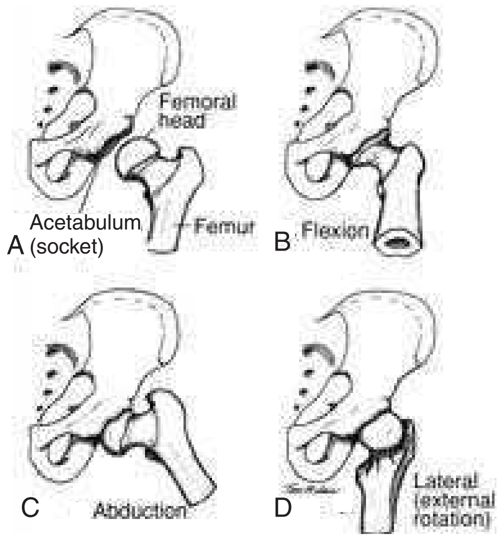


Figure 2–25 A ball-and-socket joint. **A.** The joint between the femoral head and the acetabulum is a triaxial diarthrodial joint. Motion may occur in three planes around three axes. **B.** Flexion/extension occurs in the sagittal plane around a coronal axis. **C.** Abduction/adduction occurs in the frontal plane around an A-P axis. **D.** Rotation occurs in the transverse plane around a longitudinal axis.

CASE APPLICATION

Classification of Affected Joints

case 2–9

Consider the classification of the joints affected after George Chen's fracture:

- Uniaxial: talocrural and subtalar joints
- Biaxial: first metatarsophalangeal, calcaneocuboid, and talonavicular joints

JOINT FUNCTION

The structure of the joints of the human body reflects their functions. Synarthrodial joints are relatively simple in design and function primarily as stability joints, although some motion does occur. Diarthrodial joints are more complex and primarily provide mobility, though these joints also provide some measure of stability. Effective human functioning depends on the integrated action of many joints, some providing stability and some providing mobility. In general, the ability to stabilize one or more body segments is essential if predictable joint motion and normal function are to occur.

Kinematic Chains

Kinematic chains, in the engineering sense, are composed of a series of rigid links that are interconnected by a series of pin-centered joints. The kinematic chain can be open or closed. In an **open kinematic chain,** the distal end of the chain is free to move, and one joint can move independently of others in the chain. When both the proximal and the distal ends of the chain remain fixed, it creates a closed system or **closed kinematic chain.** Under these conditions, movement at one joint automatically creates movement in one or more other joints in the chain.[29]

The term *closed chain* has often been applied to human movements that take place under weight-bearing conditions, when the distal segment of a limb is not free to move. Under weight-bearing conditions, motion at one joint generally is accompanied by motion at one or more other joints for the body to remain stable. For instance, when a person in the erect standing position bends both knees, simultaneous motion must occur at the ankle and hip joints if the head is to remain upright (Fig. 2–26A). The motions of hip flexion and ankle dorsiflexion predictably accompany knee flexion. Under open-chain (non-weightbearing) conditions, the foot is not fixed, and knee flexion can occur independent of motion at continguous joints (Fig. 2–26B). In the human system of joints and links, the joints of the lower limbs and the pelvis function as a closed kinematic chain when a person is in the erect weight-bearing position, because the feet are fixed on the ground and the head usually remains aligned over the sacrum. Most functional activities involving the lower extremities involve closed-chain motion.

The ends of human limbs, especially the upper extremities, frequently are not fixed but are free to move. In these open kinematic chain motions, joint motion is much more varied. In an open kinematic chain, motion does not occur in a predictable manner because joints may function either independently or in unison.

Example 2-16

A person may wave the whole upper limb by moving the arm at the glenohumeral joint at the shoulder or may move only at the wrist. In the first instance, all of the degrees of freedom of all of the joints from the shoulder to the wrist are available to the distal segment (hand). If the person is waving from the wrist, only the degrees of freedom at the wrist are available to the hand, and motion of the hand in space is more limited than in the first situation.

The concept of open and closed chains, which is useful for analyzing human motion, therapeutic exercise, and the effects of injury and disease on the joints of the body, will be used throughout this text. Although the joints in the human body do not always behave in an entirely predictable manner in either a closed or an open chain, the joints are interdependent. A change in the function or structure of one joint in the system will usually cause a change in the function of a joint elsewhere in the kinematic chain. For example, if the range of motion (ROM) at the knee is limited, the hip and/or ankle joints have to compensate so that the foot can clear the floor when the person is walking, so that he or she can avoid stumbling.

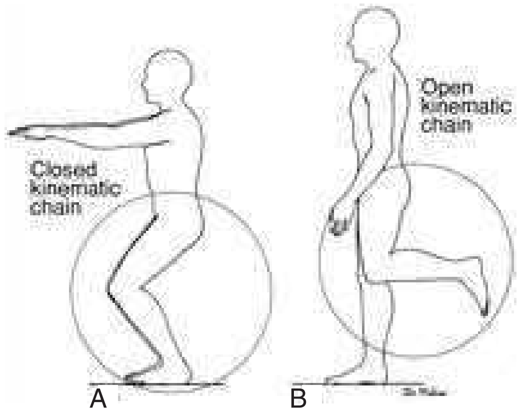


Figure 2–26 Closed and open kinematic chains. **A.** In a closed kinematic chain, knee flexion is accompanied by hip flexion and ankle dorsiflexion. **B.** Knee motion in an open kinematic chain may occur with or without motion at the hip and ankle. In the diagram, knee flexion is shown without simultaneous motion at the hip and the ankle.

Joint Motion

Range of Motion

The normal ROM of a joint is sometimes called the anatomic or physiologic ROM, referring to the amount of motion available to a joint within the anatomic limits of the joint structure.[29] The extent of the anatomic range is determined by a number of factors, including the shape of the joint surfaces, the joint capsule, ligaments, muscle bulk, and surrounding musculotendinous and bony structures. In some joints there are no bony limitations to motion and the ROM is limited only by soft tissue structures. Other joints have definite bony restrictions to motion in addition to soft tissue limitations. The humeroulnar joint at the elbow is limited in extension by bony contact of the ulna on the olecranon fossa of the humerus, while the knee has no bony limits. The sensation experienced by the examiner performing passive physiologic movements at each joint is referred to as the **end-feel.**

A ROM is considered to be pathological when motion at a joint either exceeds or fails to reach the normal anatomic limits of motion. When a ROM exceeds the normal limits, the joint is **hypermobile.** When the ROM is less than what would normally be permitted by the structure, the joint is **hypomobile.** Hypermobility may be caused by a failure to limit motion through bony elements or soft tissues, including weak musculature, leading to instability. Hypomobility may be caused by bony or cartilaginous blocks to motion or by the inability of the capsule, ligaments, or muscles to elongate sufficiently to allow a normal ROM. A **contracture,** which is the shortening of soft tissue structures around a joint, is one cause of hypomobility. Either hypermobility or hypomobility of a joint may have undesirable effects, not only at the affected joint but also on adjacent joint structures.

Example 2-17

Limitation of hip extension as a consequence of osteoarthritis may lead to excessive lumbar spine movement in order to achieve adequate movement of the lower extremity during gait.

Osteokinematics

Osteokinematics refers to the rotary movement of the bones in space during physiological joint motion.[29] These are the observable movements of the bony levers in the sagittal, frontal, and transverse planes that occur at joints. The movements are typically described by the plane in which they occur, the axis about which they occur, and the direction of movement.

Example 2-18

Osteokinematic movements at the knee joint include flexion or extension of the tibia on the femur (or the femur on the tibia) in the sagittal plane about a coronal axis. Note that movements are always described as if they are occurring from the anatomical position. Sometimes the direction of the movement is described by the direction of the bone in space: that is, posterior movement of the distal tibial on the femur (flexion) or anterior movement of the proximal femur on the tibia (extension).

Arthrokinematics

Physiological joint motion involves rotation of bony segments (osteokinematics) as well as motion of the joint surfaces in relation to another.[29,31] Movements between adjacent joint surfaces accompany voluntary osteokinematic movement but cannot be voluntarily isolated under normal conditions. The term **arthrokinematics,** or accessory motion, is used to refer to these movements of joint surfaces relative to one another. Often, one of the joint surfaces is relatively stable and serves as a base for the motion, whereas the other surface moves on this relatively fixed base. The terms **roll, slide,** and **spin** (Fig. 2–27) are used to describe the type of motion that the moving part performs.[6,29–31,103] A roll refers to the rolling of one joint surface on another, as in a tire rolling on the road. In the knee, the femoral condyles roll on the fixed tibial surface during the initial stages of knee flexion or extension in standing. The direction of rolling is described by the direction of movement of the bone; thus, the femur rolls forward during knee extension in standing. During a pure rolling motion, a progression of points of contact between the surfaces occurs (see Fig. 2–27A).

Sliding, which is a pure translatory motion, refers to the gliding of one component over another, as when a braked wheel skids. The same point on the sliding surface moves over changing points of contact in the fixed component (see Fig. 2–27B). In the hand, the proximal phalanx slides over the fixed end of the metacarpal during flexion and extension. Spin refers to a rotation of the movable component, as when a top spins. Spin is a pure rotary motion. The point of contact changes for the moving component but not for the fixed (see Fig. 2–27C). At the elbow, the head of the radius spins on the capitulum of the humerus during supination and pronation of the forearm.

During human joint motion, combinations of rolling and sliding usually occur in order to maintain joint integrity. The types of arthrokinematic motion that occur at a particular joint depend on the shape of the articulating surfaces. When a concave articulating surface is moving on a stable convex surface, sliding typically is considered to occur in the *same direction* as motion of the bony lever (Fig. 2–28). Because the motion of the bony lever is the direction of the roll of the bone, the roll and slide are in the same direction. This allows the joint surfaces to stay in optimum contact with each other. When a convex joint surface moves on a concave surface, the bone typically rolls in one direction and glides in the *opposite direction* in order to maintain optimum contact. This is known as the **convex-concave rule.**

Most joints fit into either an **ovoid** or a **sellar** category. In an ovoid joint, one surface is convex and the other surface

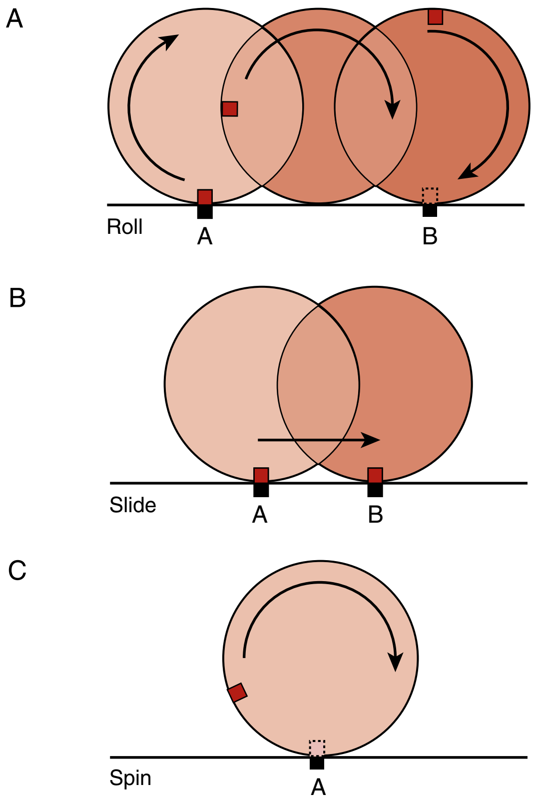


Figure 2–27 Arthrokinematic joint motions include rolling **(A),** sliding **(B),** and spinning **(C).**

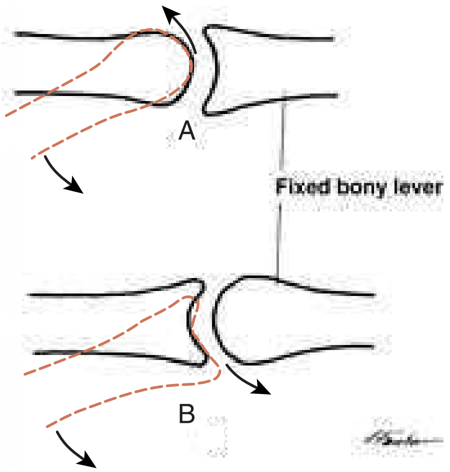


Figure 2–28 Motion at ovoid joints. **A.** When a convex surface is moving on a fixed concave surface, the convex articulating surface moves in a direction opposite to the direction traveled by the shaft of the bony lever. **B.** When a concave surface is moving on a fixed convex surface, the concave articulating surface moves in the same direction as the remaining portion of the bony lever (proximal phalanx moving on fixed metacarpal).

Concept Cornerstone **2-9**

Convex-Concave Joint Surface Motion

Convex-concave rule: Convex joint surfaces generally roll and glide in *opposite* directions, whereas concave joint surfaces generally roll and slide in the *same* direction.

is concave (Fig. 2–29A). In a sellar joint, *each* joint surface is *both* convex and concave (Fig. 2–29B). The arthrokinematic motion of the moving segment is described in relation to the nonmoving segment. Thus, knowledge of the structure of the moving segment and the movement that is occurring allows prediction of the arthrokinematics that accompany the movement. The movement that occurs between articular surfaces is an essential component of joint motion and must occur for normal functioning of the joint. If the articular end of the bone is not free to move in the appropriate direction, then when the distal end of the bone moves, abnormal forces will be created in the joint.

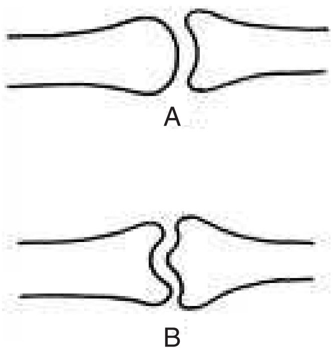


Figure 2–29 Ovoid and sellar joints. **A.** In an ovoid joint, one articulating surface is convex and the other articulating surface is concave. **B.** In a sellar joint, each articulating surface is concave and convex.

Example 2-19

Abduction of the humerus at the glenohumeral joint must be accompanied by downward sliding (inferior movement) of the proximal convex head of the humerus on the concave surface of the glenoid fossa to avoid damaging joint structures (Fig. 2–30A). If downward gliding is restricted, abduction of the humerus may cause impingement of superiorly located soft tissue structures between the humerus and the acromion. Superior gliding of the humeral head on the glenoid must occur as the distal end of the humerus is brought back downward into adduction (Fig. 2–30B).

For articular surfaces to be free to move in the appropriate direction (arthrokinematics) as the bony lever rotates (osteokinematics), the joint must have a certain amount of **"joint play."** This freedom of movement of one articular surface on another can be tested by an examiner when the joint is in a **loose-packed** position. The joint should have a sufficient amount of play to allow normal motion at the joint's articulating surfaces. If the supporting joint structures are excessively lax, the joint may have too much play and become unstable. If the joint structures are excessively tight, the joint will have too little movement between the articular surfaces, and movement of the bony lever will be restricted because the appropriate intra-articular movement will not accompany the physiological movement.

CASE APPLICATION

Effects of Limited Range of Motion

case 2–10

When the cast is removed from George's leg and foot, he has 10° dorsiflexion and 20° plantarflexion, and his subtalar motion is restricted. This will affect the entire lower extremity kinematic chain during squatting, walking, and other activities. His ability to adapt to uneven surfaces will be compromised by ankle discomfort and by restriction of subtalar joint motion. The latter usually can be readily restored through joint mobilization and will result in much more comfortable walking and standing.

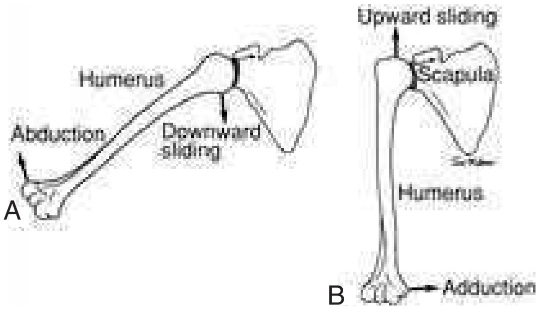


Figure 2–30 Sliding of joint surfaces. **A.** Abduction of the humerus must be accompanied by inferior sliding of the head of the humerus in the glenoid fossa. **B.** Adduction of the humerus is accompanied by superior sliding of the head of the humerus.

Joint motions commonly include a combination of sliding, spinning, and rolling. Although we typically use anatomical landmarks to represent the axis of rotation for various joints, the combination of sliding and spinning or rolling produces curvilinear motion and a moving axis of motion. An axis that moves during rolling or sliding motions forms a series of successive points (or axis locations). The axis of rotation at any particular point in the joint motion is called the **instantaneous axis of rotation (IAR).** Moving axes of rotation occur most notably when opposing articular surfaces are of unequal size. In some joints, such as the shoulder, the articulating surface of the moving bone (humerus) is larger than the surface of the stabilized component (glenoid fossa of the scapula). In other joints, such as the metacarpophalangeal and interphalangeal joints of the fingers, the articulating surface of the moving bone is smaller than the surface of the stabilized component. When the articulating surface of a moving component is larger than the stabilized component, a pure motion such as rolling would result in the larger moving component's rolling off the smaller articulating surface before the motion is completed. Therefore, combination motions, in which a moving component rolls in one direction and slides in the opposite direction, help to increase the ROM available to the joint and keep opposing joint surfaces in contact with each other. The rolling and sliding arthrokinematic movements of the articular surfaces are not usually visible and thus have not been described in the traditional classification system of joint movement. However, these arthrokinematics motions are considered in the six degrees of freedom model described by White and Panjabi.[27] These authors have suggested that motion at the intervertebral symphysis joints between the bodies of the vertebrae in the vertebral column occurs in six planes, around three axes. The implication is that motion at the joints of the body might be more thoroughly described by using a six-degrees-of-freedom model, as is done to describe available motions in Chapter 1 and at many of the joints covered in this text.

All synovial joints have a **close-packed** position in which the joint surfaces are maximally congruent and the ligaments and capsule are maximally taut. The close-packed position is usually at the extreme end of a ROM. In the close-packed position, a joint possesses its greatest stability and is resistant to tensile forces that tend to cause distraction (separation) of the joint surfaces. Little or no joint play is possible. The position of full extension is the close-packed position for the humeroulnar, knee, and interphalangeal joints.[6,29] In the loose-packed position of a joint, the articular surfaces are relatively free to move in relation to one another. The loose-packed position of a joint is any position other than the close-packed position, although the term is most commonly used to refer to the position at which the joint structures are most lax and the joint cavity has a greater volume than in other positions. In the loose-packed position, the joint has a maximum amount of joint play (accessory movements). An externally applied force, such as that applied by a therapist or physician, can produce movement of one articular surface on another and enable the examiner to assess the amount of joint play that is present. Movement in and out of the close-packed position is likely to have a beneficial effect on joint nutrition because of the squeezing out of fluid during each compression and imbibing of fluid when the compression is removed.[32]

GENERAL CHANGES WITH DISEASE, INJURY, IMMOBILIZATION, EXERCISE, AND OVERUSE

Each part of a joint has one or more specific functions that are essential for the overall performance of the joint. Therefore, anything that affects one part of a joint will disrupt the total function of the joint. Likewise, anything that affects joint motion will affect all the structures that constitute that joint. This relationship between form and function is essential for therapists to remember during rehabilitation after injury. For example, when a bone is broken, the fracture may be the main injury that dictates subsequent treatment, but lack of motion and decreased loading also will affect cartilage, ligaments, joint capsule, tendons, and muscles. The ideal rehabilitation protocol considers the behavior of all the affected structures and includes interventions tailored to induce adaptations in each structure. This means understanding the time course and nature of the adaptation of each tissue to altered loading conditions.

Complex joints are more likely to be affected by injury, disease, or aging than are simple joints. Complex joints have more parts and are subject to more wear and tear than are stability joints. The function of the complex joints depends on a number of interrelated factors. For example, the capsule must produce synovial fluid, which must be of the appropriate composition and of sufficient quantity to lubricate and nourish the joint. The hyaline cartilage must be smooth enough that the joint surfaces can move easily, yet permeable enough to receive nourishment from the joint fluid. The cartilage must undergo periodic compressive loading and unloading to facilitate movement of the fluid, and the collagen network must be intact to contain the fluid attracted to the PGs. The ligaments and capsules must be strong enough to provide sufficient support for stability and yet be flexible enough to permit normal joint motion. Tendons must be able to withstand the forces generated by muscles as they produce movement.

Disease

The general effects of disease, injury, immobilization, and overuse may be illustrated by using the normal function of a joint structure as a basis for analysis. For example, when the synovial membrane of a joint is affected by a disease like rheumatoid arthritis, the production, and perhaps the composition, of the synovial fluid changes. Lubrication of the joint is also affected. The disease process and the changes in joint structure that occur in rheumatoid arthritis involve far more than just synovial fluid alteration, but the disease does change the composition and the quantity of the synovial fluid. In another type of arthritis, osteoarthritis, which may be genetic and/or mechanical in origin, the cartilage is the focus of the disease process. Erosion and splitting of the cartilage occur. As a result, friction is increased between the joint surfaces, thus further increasing the erosion process.

Injury

Joint support is decreased after injury to one or more of its components. If a table has an unstable joint between a leg and the table top, damage and disruption of function may occur as a result of instability. If a heavy load is placed on the damaged table joint, the joint surfaces will separate under the compressive load and the leg may be angled. The once-stable joint now allows mobility, and the leg may wobble back and forth. This motion may cause screws to loosen or nails to bend and ultimately to be torn out of one of the wooden components.

Complete failure of the table joint may result in splintering of the wooden components, especially if the already weakened joint is subjected to excessive, sudden, or prolonged loads. The effects of decreased support in a human joint are similar to those in the table joint. Separation of the bony surfaces occurs and may result in wobbling or a deviation from the normal alignment of the bony components. Changes in alignment create abnormal joint opening on the side where a ligament is torn. Other ligaments, tendons, and the joint capsule may be subjected to increased loading, leading them to become excessively stretched and unable to provide protection. The intact side of the joint may be subjected to abnormal compression during weight-bearing or motion. In canine experiments in which an unstable knee joint is produced as a result of transection of the anterior cruciate ligament of the knee, morphologic, biochemical, biomechanical, and metabolic changes occur in the articular cartilage shortly after the transection.[106] Later, articular cartilage becomes thicker and shows fibrillation, and osteophytes develop. The cartilage shows much higher water content than in the opposite knee, and the synovial fluid content of the knee is increased. In addition, a sharp increase in bone turnover occurs, as does a thickening of the subchondral bone.[106] According to Van Osch and colleagues, joint instability is a well-known cause of secondary osteoarthritis involving the knee joint.[107] The recognition that joint injuries, especially ligament injuries, lead to osteoarthritis suggests greater efforts are required to prevent and treat sport injuries in young people.

Immobilization (Stress Deprivation)

Any process or event that disturbs the normal function of a specific joint structure will start a chain of events that eventually affects every part of a joint and its surrounding structures. Immobilization is particularly detrimental to joint structure and function. Immobilization may be externally imposed by a cast, bedrest, weightlessness, or denervation or may be self-imposed as a reaction to pain and inflammation. An injured joint subjected to inflammation and swelling will assume a loose-packed position to accommodate the increased volume of fluid within the joint space. This position may be referred to as the position of comfort because pain is decreased in this position. Each joint has a position of minimum pressure and maximum volume. For the knee and hip joints, the position of comfort is between 30° and 45° of flexion; for the ankle joint, the position is at 15° of plantar flexion.[29] If the joint is immobilized for a few weeks in the position of comfort, the joint capsule will adapt (shorten), and contractures[108] will develop in the surrounding soft tissues. Consequently, resumption of a normal range of joint motion will be difficult.

Effects on Ligament and Tendon

Ligaments and tendons adapt to decreased load by decreasing their collagen content and reducing cross-linking among collagen molecules, although their overall size may remain the same. The tissue thus weakens, and the resumption of previously normal loading may cause increased stress and strain.[16,17] The musculotendinous junction of tendons loses its interdigitating structure when not loaded, which makes it weaker.[89] The time course of these adaptations is fairly rapid. Ligaments and tendons show a 50% decrease in tensile strength and stiffness after 8 weeks of immobilization.[16,17] It is assumed that ligaments and tendons eventually recover their mechanical properties, but the time course of this recovery appears to be slow, and the total time for recovery is unknown. In general, the time course for the loss of mechanical properties occurs over weeks, whereas recovery can take 12 to 18 months or more.[21] Gradual reloading is necessary to restore tendon and ligament strength.

Effects on Articular Surfaces and Bone

The effects of immobilization are not confined to the surrounding soft tissues but may also affect the articular surfaces of the joint and the underlying bone. Biochemical and morphological changes may include: proliferation of fibrofatty connective tissue within the joint space, adhesions between the folds of the synovium, atrophy of cartilage, regional osteoporosis, weakening of ligaments at their insertion sites as a result of osteoclastic resorption of bone and Sharpey's fibers, a decrease in the PG content, and increase in the water content of articular cartilage.[32,109,110] Thinning and softening of the articular cartilage occur, and deformation under compressive test load increases up to 42%. As a result of changes in joint structures brought about by immobilization, decreases may be evident in the ROM available to the joint, the time between loading and failure, and the energy-absorbing capacity of the bone-ligament complex. Swelling or immobilization of a joint also inhibits and weakens the muscles surrounding the joint.[111–114] Therefore, the joint is unable to function normally and is at high risk of additional injury.[115] A summary of the possible effects of prolonged immobilization is presented in Table 2–8.

CASE APPLICATION

Deleterious Effects of Immobilization *case 2–11*

Mr. Chen's joints and surrounding structures will undergo striking changes during immobilization:

- Bone: weakened, decreased collagen and mineral content (osteopenia)
- Capsule: shrinking, increased resistance to movement (stiffer)
- Ligament: decreased cross-links, decreased tensile strength (weaker)
- Tendon: decreased cross-links, disorganization of collagen fibrils, decreased tensile strength (weaker)
- Muscle: loss of sarcomeres in series, decreased contractile proteins (shorter, less force production)
- Cartilage: swelling, decreased PG concentration (softer, weaker)

These changes occur within 8 weeks, but recovery may take 18 months or longer.

Recognition of the adverse effects of immobilization has led to the development of several strategies to help minimize the consequences of immobilization: (1) use of continuous passive motion (CPM) devices after joint surgery, (2) reduction in the duration of casting periods after fractures and sprains, (3) development of dynamic splinting devices to allow joint motion while preventing unwanted motion that may damage healing structures, (4) use of graded loading after immobilization, and (5) extension of the recovery period to months rather than days or weeks. The continuous passive motion device can move joints passively and repeatedly through a specified portion of the physiological ROM. The speed of the movement and the ROM can be controlled. Continuous passive motion devices produce joint motion under low loading conditions, which in turn produces medium-frequency alternating compression, which may stimulate cartilage formation. It is easier to control loading with these devices than with active movements and therefore easier to avoid the potentially deleterious compressive-tensile stresses and strains produced by active muscle contractions. Continuous passive motion was shown to prevent some of the tendon weakening that occurs during immobilization, though not enough to maintain normal tissue strength.[116]

Concept Cornerstone 2-10

Effects of Altered Loading on Connective Tissue

- Connective tissues become weaker and lose their normal structure if they are not loaded.
- Changes with decreased load occur rapidly.
- Recovery of normal structure and function requires gradual progressive loading.
- Loads should be tailored to the connective tissue.

Table 2–8 Effects of Increased and Decreased Load on Connective Tissues

TISSUE	DECREASED LOAD	INCREASED LOAD
Tendon and ligament	Decreased collagen concentration Decreased cross-linking Decreased tensile strength	Increased cross-sectional area Increased collagen concentration Increased cross-linking Increased tensile strength Increased stiffness
Menisci	Decreased PGs	Increased PGs
Bone	Decreased collagen synthesis Decreased bone formation Increased bone resorption	Denser bone Increased synthesis of collagen and bone
Cartilage	Thinning of cartilage Advancing of subchondral bone Decreased PG synthesis Fewer PG aggregates	Increased PG synthesis Increased volume?
Joint capsule	Disordered collagen fibrils Abnormal cross-linking	Not specifically examined
Synovium	Adhesion formation Fibrofatty tissue proliferation into joint space	Not specifically examined

PG, proteoglycan.

Exercise

All tissues appear to respond favorably to gradual progressive loading by adapting to meet increased mechanical demands. Exercise influences cell shape and physiological functions and can have a direct effect on matrix alignment. The response to exercise varies among tissues and depends on the nature of the stimulus, including the amount, type, and frequency of loading. The mechanism of connective tissue response to exercise appears to involve cells' detecting tissue strain and then modifying the type and amount of tissue they produce. The amount, type, and frequency of deformation are important. Low-frequency compressive loading will increase cartilage formation, whereas higher frequencies can enhance bone synthesis. Higher magnitude or sustained loading will induce fibrocartilage formation, whereas tensile loads induce tissue formation resembling that found in tendon or ligament. According to Mueller and Maluf's Physical Stress Theory,[80] maintaining the normal mechanical state of connective tissues appears to require repetitive loading beyond a threshold level. Below this threshold, the immobilization changes previously described rapidly occur.

Bone Response to Exercise

Bone deposition is increased with weight-bearing exercise and in areas of bone subjected to increased muscle force.[117,118] This response of bone form to function, Wolff's law, has been known for over 100 years, and exercise is now used as a therapeutic intervention to prevent bone loss.[119] A systematic review showed that in 11 of 16 studies, postmenopausal women showed improvements in bone density with either exercise or exercise plus calcium or estrogen.[118] The use of interventions to prevent bone loss and resulting osteoporosis during space flight is an area of active research. Bone formation appears very sensitive to strains as well as (or perhaps instead of) the magnitude of the applied load. Very low magnitude high-frequency vibration has been shown to increase trabecular bone formation by 34%.[120] This suggests that even very low loads, well below the threshold for physical damage, may increase bone density. Rubin et al. proposed that these far smaller high-frequency (10- to 30-Hz) mechanical signals that continually barrage the skeleton during longer term activities, such as standing and walking, are what regulate skeletal architecture.[120–122] Even short durations of loading are effective, and just 10 minutes of low-load, high-frequency stimulation has been shown to prevent bone loss induced by disuse.[120] Lanyon suggested that the asymmetrical strains during normal load-bearing activities create an ever-changing strain distribution, that it is the novelty of the strain that induces bone adaptation, and that the osteogenic response saturates rapidly.[123] He further suggested that exercise regimens designed to control bone architecture can usefully capitalize on this feature of the adaptive (re-)modeling response. Each exercise session should include as many novel strain distributions as possible, preferably involving high peak strains and strain rates.

Cartilage Response to Exercise

The response of cartilage to immobilization has been described,[124] but its response to increased physiological loading is largely unknown. The health of articular cartilage depends on the application and removal of compressive loads. Chondrocytes are directly connected to their microenvironment through attachments between cell membrane proteins (integrins) and collagen fibrils, and mechanical forces are transduced into intracellular synthetic activity. The mechanisms of this transduction and the magnitude and frequency of the loading that will optimize cartilage structure are not yet known. This is an area of active research, as cartilage injuries heal very poorly, and the use of transplanted material to repair cartilage defects is being explored. Since Salter's work,[125] it is well known that motion enhances tissue formation in cartilage defects, but the tissue formed is fibrocartilage, not hyaline articular cartilage. Unlike fibroblasts in bone, ligament, and tendon, chondrocytes do not readily migrate and repair areas that have been injured. Defects that extend to subchondral bone are thought to have better healing potential because of the presence of pluripotential mesenchymal cells (from bone marrow) that can differentiate into chondroblasts under the right loading conditions—hence the use of drilling to treat osteochondral defects.[124] There are no quantitative data available about changes in human articular cartilage after immobilization or exercise, although MRI shows promise in this regard. It appears that cyclic low-magnitude, low-frequency (less than 1-Hz) compressive loads may be best for inducing or maintaining cartilage structure.

Tendon Response to Exercise

Tendons respond to increased tensile loads by increasing their collagen concentration, collagen cross-linking, tensile strength, and stiffness. Woo et al. showed that after 12 months of physical training, the extensor digitorum tendons of swine increased their weight, strength, collagen content, and stiffness to match the normally stronger flexor digitorum longus, which did not change in response to the same program.[95] Biochemical changes occurred in chicken Achilles tendons after strenuous intermittent running, with increased collagen synthesis, cross-linking, tensile strength, and stiffness, although tendon size and weight did not change.[126,127] Chronic increased loading causes tendon hypertrophy and increased cross-linking.[127–129] In other words, both structural and material changes can take place. Interestingly, exercise also appears to offset some of the changes that occur in connective tissue with age.[130] Progressive tensile loading has been used successfully to treat chronic tendon disorders, under the assumption that the tendon adapts to the increased loads.[77,78,97–99]

Ligament Response to Exercise

The effects of exercise in preventing negative changes in healing ligaments and the positive effects of activity on ligament healing have been well demonstrated, but the effects of exercise on normal ligament are less clear.[131–133] Recovery of normal ligament structure and mechanics after immobilization, under normal loading conditions, is a slow process that can take months. It appears that exercise may speed this process, but the volume of loading and the time course of the adaptations are not known (Fig. 2–31).[134]

Concept Cornerstone **2-11**

Connective Tissue Adaptation

All connective tissues will adapt to increased load through changes in structural and/or material properties (form follows function).

The load must be gradual and progressive; as the tissue adapts to the new loading conditions, the load must change to induce further adaptation.

The type of connective tissue formed will match the type and volume of the load:

- Compression: cartilage or bone
- Tension: ligament or tendon

Optimization of load volume and frequency, and the nature of human tissue adaptation, remain largely unknown as yet.

CASE APPLICATION

Facilitation of Tissue Recovery

case 2–12

The types of exercise that may facilitate tissue recovery in George's case might include:

- Bone: walking, standing with weight shifts, bouncing, on/off pressure
- Cartilage: moderate loads through available ROM, walking, isometrics
- Ligament: gentle, progressive tensile stress, change directions
- Tendon: progressive tensile loading, strength training
- Muscle: progressive tensile loading at various speeds (recruit different motor units) and lower load exercise to fatigue (induce metabolic adaptation)
- Capsule: repeated exercise throughout physiological ROM

In general, loads should be kept light for the first 2 to 3 weeks, with the emphasis on repeated motion through the ROM and passive joint mobilization to nontraumatized joints (such as the subtalar joint). When the talocrural joint has adequate dorsiflexion, closed chain exercises can begin. Although isometric exercises (no joint motion) can be used at any point, it is recommended that large loads be avoided while the joint is immobilized.

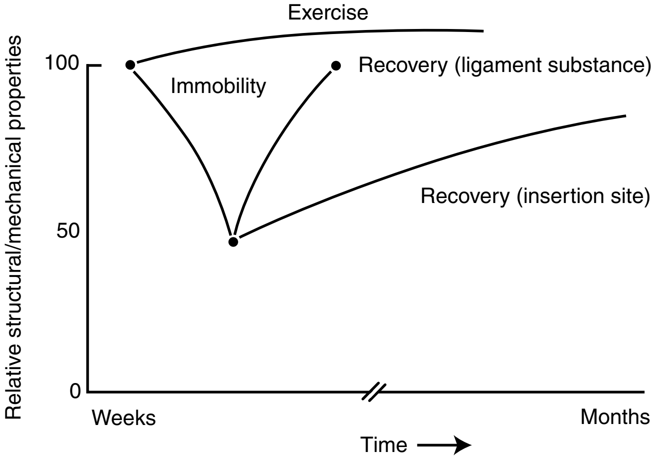


Figure 2–31 Effects of load alteration of normal and immobilized tissues. Adaptation to a decreased load is rapid, whereas recovery is slower. The response of normal tissues to increased load remains uncertain.

Overuse

Although immobilization is detrimental, and exercise beyond a threshold level is necessary to maintain connective tissue structure and function, both sustained and repetitive loading of articular structures may have adverse effects. Damage can occur in one of two ways: (1) sudden application of large loads and (2) repeated or sustained application of low loads. The former are nonphysiological loads that create large stresses and strains, thus creating a rupture of the tissue on a microscopic or macroscopic scale (e.g., tendon ruptures, bone fractures). The latter are physiological loads that are sustained or repeated while creep is occurring. Recovery of normal tissue structure takes an as yet unknown time after the load is removed. When a structure is subjected to new loading of already deformed tissue, the tissues may enter the plastic range and undergo microfailure. This may account for some cases of chronic back pain or tendon injuries. Ligaments, or capsule, subjected to constant tensile loading may undergo permanent changes in length. For example, after loss of the ACL, increased load on the posterior knee joint capsule may cause plastic deformation, leading to increasing knee hypermobility. Cartilage subjected to constant compressive loading will creep, becoming thinner, and may show evidence of permanent deformation. Cell death may occur with rigid sustained pressure at focal points on the cartilage, and permeability and fluid flow will be decreased.[11]

Joints and their supporting structures subjected to repetitive loading thus may be injured because they do not have enough time to recover before they are subjected to another loading cycle, even though the load magnitude may be within the normal loading range. An injury resulting from this type of repetitive strain loading may be

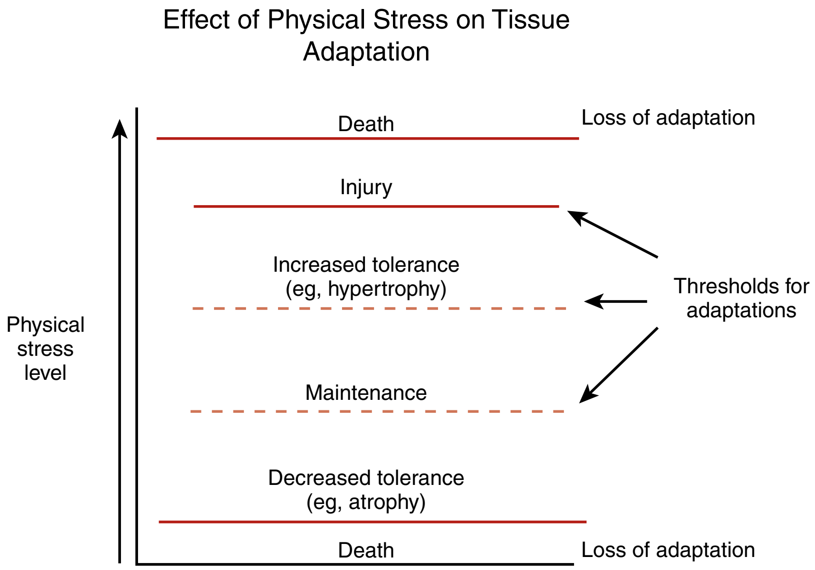


Figure 2–32 The range of adaptations possible in joint structures in response to different levels of loading. *(From Mueller MJ, Maluf KS: Tissue adaptation for physical stress: A proposed "physical stress theory" to guide physical therapist practice, education and research. Phys Ther 82:383, 2002, with permission of the American Physical Therapy Association.)*

called **overuse injury** or **syndrome, repetitive motion disorder,** or **repetitive strain injury.**[13] These disorders have been identified in athletes, dancers, farmers, musicians, and factory and office workers. Such injuries appear to affect a greater proportion of women than men, for unknown reasons.[25] Hart and associates hypothesized that intrinsic gender differences may exist in the regulation of connective tissue structures, perhaps related to hormone levels, which fluctuate in women during pregnancy and menstrual cycles.[25] Biopsy material from tendons from human subjects undergoing surgery for repetitive motion disorders shows an inflammatory process in some tendons and a degenerative process in others.[134] In view of the findings to date, it appears that simple tissue fatigue is not a sufficient explanation for the cause of repetitive motion disorders and that additional research is needed to determine all of the factors involved in the cause, effect, prevention, and treatment of repetitive motion disorders. The threshold between loading-induced adaptation and overuse may be a fine one (Fig. 2–32).

Concept Cornerstone 2-12

Chronic Overuse Injuries

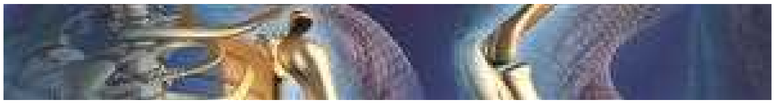
- Chronic overuse injuries may involve repeated or sustained loads while the tissue is still in a deformed state. Recovery time, not load size, may be the critical variable.
- The role of systemic influences (e.g., hormones, nutrition) and neurophysiologic influences (referred pain, focal dystonia) in repetitive injuries remains to be explored.

SUMMARY

This chapter has presented the elementary principles of joint design, a classification system for human joints, and an introduction to the materials found in human joints and their properties, as well as the effects of disease, immobilization, and overuse on joint structures. The health, strength, and function of joint structures depend on a threshold amount of stress and strain; this threshold can move up or down, depending on the state of the tissue. Cartilage and bone nutrition and growth depend on joint movement and muscle contraction. Cartilage nutrition depends on joint movement through a full ROM to ensure that the entire articular cartilage receives the nutrients necessary for survival. Ligaments and tendons depend on stress and strain to maintain or increase their strength. Controlled loading and motion applied early in the rehabilitation process stimulate collagen synthesis and help align collagen fibrils. Bone density and strength increase following the stress and strain created by muscle activity and weight-bearing. In contrast, bone density and strength decrease when stress and strain are absent. Micromotion and compression facilitate fracture healing; the rigid immobilization of internal devices actually slows healing, although their use may be necessary. Controlled mobilization, rather than complete immobilization, is preferred whenever possible. There is still a great deal left to learn about adaptation in connective tissues, especially in humans. It appears that tissues have a movable threshold, below which they atrophy and above which they become injured (see Fig. 2–32). Progressive loading involves gradually moving this threshold so that the tissue can withstand the forces accompanying functional activities.

The inadequacy of cartilage repair mechanisms and the slow recovery of bones, ligaments, and tendons suggest that the prevention of injury to joint structures through the avoidance of excessive loading is crucial. Gradual, progressive loading is the ideal. The therapist must skillfully load the tissues with the appropriate direction, magnitude, and frequency of loading to prevent weakening or to induce adaptation. In subsequent chapters, the specific structure and function of each of the major joints in the body will be explored. Knowledge of the basic elements of normal joint structure and function and understanding the changes that function can induce in structure, and vice versa, are helpful for recognizing abnormal joint function; analyzing the effects of injury, disease, or aging on joint structure and function; and appreciating the complex nature of human joints.

STUDY QUESTIONS



1. Describe the structure of a typical diarthrodial joint.
2. Describe the type of motion that is available at a pivot joint, and give at least two examples of pivot joints.
3. Describe the composition of the interfibrillar component of the extracellular matrix in connective tissue.
4. Describe how diarthrodial joints are lubricated.
5. Describe the movements of the bony lever during motion at an ovoid joint.
6. Describe what is meant by the term *toe region*.
7. Explain creep and how it affects joint structure and function.
8. Explain how immobilization affects joint structures.
9. Explain what happens to a material when hysteresis occurs.
10. Explain how an overuse injury may occur.
11. Compare the structure and function of synarthroses with that of diarthroses.
12. Compare a closed chain with an open chain and give examples of each.
13. Compare the composition, properties, and function of ligaments with those of tendons, cartilage, and bone.
14. Compare stress and strain. Give at least one example, using a load-deformation curve.
15. Design a rehabilitation program for an ankle ligament sprain that will ensure joint protection and prevent re-injury and the subsequent development of osteoarthritis.

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Chapter 3

Muscle Structure and Function

Gary Chleboun, PT, PhD

Introduction

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INTRODUCTION

The skeletal muscles, like the joints, are designed to contribute to the body's needs for mobility and stability. Muscles serve a mobility function by producing or controlling the movement of a bony lever around a joint axis; they serve a stability function by resisting extraneous movement of joint surfaces and through approximation of joint surfaces. The body is incapable of either supporting itself against gravity or producing motion without muscle function.

Human movement is a complex interaction of muscle function and joint lever systems under the control of the nervous system. Daily, clinicians evaluate the muscle function of patients in order to determine the extent of the loss of muscle function and to formulate appropriate interventions to help the patient regain muscle function. Understanding muscle function begins with a clear picture of the structure of a muscle from the contractile proteins within each muscle fiber to the organization of the fibers in the whole muscle. From the muscle structure, we examine the basic mechanical properties of muscle fibers, whole muscles, and groups of muscles. To complete our understanding of muscle function, we analyze the function of muscles working across joints to produce the intricate movements we use for daily activities, work, sport, and play. Unfortunately, some of the movements used in these activities may cause injury to the muscles and tendons. The following case identifies a common muscle injury. Throughout this chapter, you will see how the structure and function of the muscles can be applied to this clinical situation.

3-1 Patient Case

case

Vik Patel, a 50-year-old man, was playing softball one summer evening. He was trying to catch a fly ball when he stepped back with his right foot and slipped slightly. As his foot slipped, the motion at the ankle was dorsiflexion, and the ankle plantar flexor muscles were contracting as he tried to push off so that he could run forward. At the moment he tried to push off, he felt a twinge of pain in his right calf muscle. Vik states that he has pain in the calf muscle and along the Achilles tendon when he tries to stand on his toes and when he does calf-stretching exercises. After an evaluation of Vik, it appears that he may have strained the calf muscle or caused some tendinitis.

ELEMENTS OF MUSCLE STRUCTURE

Skeletal muscles are composed of muscle tissue (contractile) and connective tissue (noncontractile). The muscle tissue has the ability to develop tension in response to chemical, electrical, or mechanical stimuli. The connective tissue, on the other hand, develops tension in response to passive loading.[1] The properties of these tissues and the ways in which they are interrelated give muscles their unique characteristics.

Composition of a Muscle Fiber

Contractile Proteins

A skeletal muscle is composed of many thousands of muscle fibers. A single muscle contains many **fascicles,** which are made up of groups of muscle fibers (cells) surrounded by connective tissue (Fig. 3–1A). The arrangement, number, size, and type of these fibers may vary from muscle to muscle,[2,3] but each fiber is a single muscle cell that is enclosed in a cell membrane called the **sarcolemma** (Fig. 3–1B). Like other cells in the body, the muscle fiber is composed of cytoplasm, which in a muscle is called **sarcoplasm.** The sarcoplasm contains **myofibrils** (Fig. 3–1C), which are the contractile structures of a muscle fiber, and nonmyofibrillar structures such as ribosomes, glycogen, and mitochondria, which are required for cell metabolism.

The myofibril is composed of thick **myofilaments** of the protein **myosin** and thin myofilaments of the protein **actin** (Fig. 3–1D). The interaction of these two myofilaments is essential for a muscle contraction to occur. The thin myofilaments are formed of two chainlike strings of actin molecules wound around each other. Molecules of the globular protein **troponin** are found in notches between the two actin strings, and the protein **tropomyosin** is attached to each troponin molecule (Fig. 3–2A). The troponin and tropomyosin molecules control the binding of actin and myosin myofilaments.

Each of the myosin molecules has globular enlargements called **head groups** (Fig. 3–2B).[4] The head groups, which are able to swivel and are the binding sites for

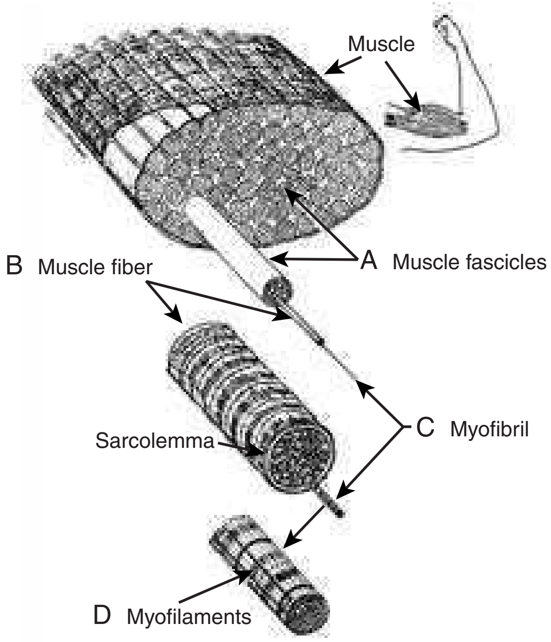


Figure 3–1 Composition of a muscle fiber. **A.** Groups of muscle fibers form bundles called **fascicles. B.** The muscle fiber is enclosed in a cell membrane called the **sarcolemma. C.** The muscle fiber contains myofibrillar structures called **myofibrils. D.** The myofibril is composed of thick myosin and thin actin **myofilaments.**

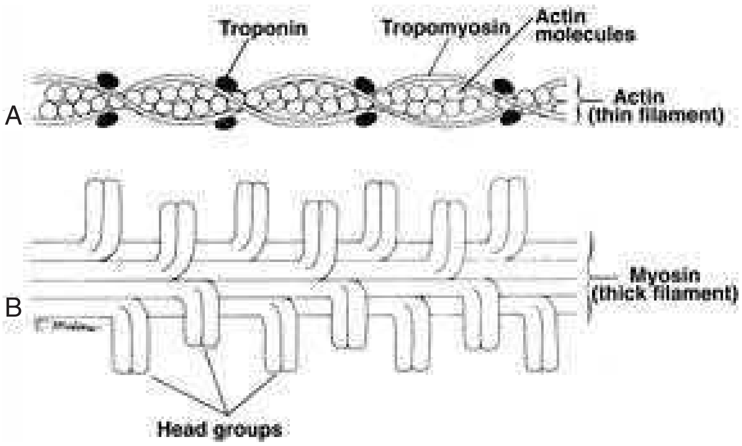


Figure 3–2 Myofilaments. **A.** The actin molecules are shown as circles. The troponin molecules are globular and are shown located in notches between the two strands of actin molecules. The tropomyosin molecules are thin and are shown lying along grooves in the actin strands. **B.** A myosin myofilament showing head groups or globular enlargements.

attachment to the actin, play a critical role in muscle contraction and relaxation. When the entire myofibril is viewed through a microscope, the alternation of thick (myosin) and thin (actin) myofilaments forms a distinctive striped pattern, as seen in Figure 3–1D. Therefore, skeletal muscle is often called **striated muscle.** A schematic representation of the ordering of the myofilaments in a myofibril is presented in Figure 3–3.

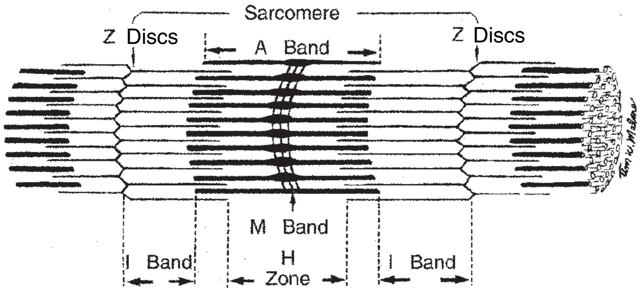


Figure 3–3 Ordering of myofibrils in a muscle at rest. The sarcomere is the portion of the myofibril that is located between the Z discs. The A band portion of the sarcomere contains an overlap of the myosin and actin filaments. The portion of the A band that contains only myosin filaments without overlap is called the H zone. The M band, located in the central portion of the H zone, contains transversely oriented myosin filaments that connect one myosin filament with another. The I band portion contains only actin fibers.

Structural Proteins

The muscle fiber also consists of several structural proteins (see Patel and Lieber[5] for a review of these proteins). Some of these proteins **(intermediate filaments)** provide a structural scaffold for the muscle fiber, whereas others (e.g., **desmin**) may be involved in the transmission of force along the fiber and to adjoining fibers. One protein, **titin,** has a particularly important role in maintaining the position of the thick filament during a muscle contraction and in the development of passive tension.[6,7] Titin is a large protein that is attached along the thick filament and spans the gap from the thick filament to the Z discs (Fig. 3–4). More will be said about titin in the discussion on the passive length-tension relationship.

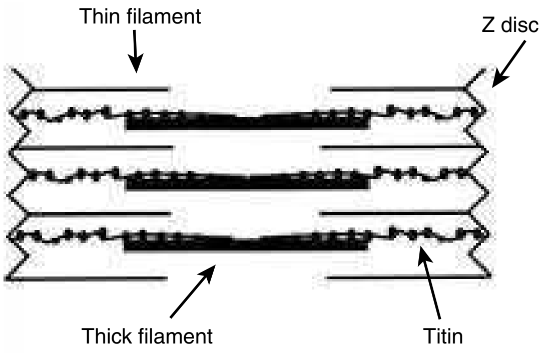


Figure 3–4 Sarcomere depicting the relationship between titin and the thick and thin filaments.

The Contractile Unit

Organization of the Contractile Unit

The portion of the myofibril that is located between two Z discs is called the **sarcomere** (see Fig. 3–3). The Z discs, which are located at regular intervals throughout the myofibril, not only serve as boundaries for the sarcomere but also link the thin filaments together. Areas of the sarcomere called **bands** or **zones** help to identify the arrangement of the thick and thin filaments. The portion of the sarcomere that extends over both the length of the thick filaments and a small portion of the thin filaments is called the **anisotropic** or **A band.** Areas that include only actin filaments are called **isotropic** or **I bands.**[4] The terms *anisotropic* and *isotropic* refer to the behavior of these portions of the fibers when light shines on them. The central portion of the thick filament **(A band area),** in which there is no overlap with the thin filaments, is called the **H zone.** The central portion of the H zone, which consists of the wide middle portion of the thick filament, is called the **M band.**

Cross-Bridge Interaction

Interaction between the thick and thin filaments of the sarcomere leading to muscle contraction is initiated by the arrival of a nerve impulse at the motor end plate, which evokes an electric impulse, or **action potential,** that travels along the muscle fiber.[8] The action potential initiates the release of calcium ions,[9] and the calcium ions cause **troponin** to reposition the tropomyosin molecules so that receptor sites on the **actin** are free and the head groups of the **myosin** can bind with actin. This bonding of filaments is called a **cross-bridge.** Tension is generated with the hydrolysis of **adenosine triphosphate (ATP)** and the release of **adenosine diphosphate (ADP)** from the myosin head[1,9,10] (Fig. 3–5).

Types of Muscle Contraction

The sliding of the thin filaments toward and past the thick filaments, accompanied by the formation and re-formation of cross-bridges in each sarcomere, results in the shortening of the muscle fiber and the generation of tension. The muscle fiber shortens (contracts) if a sufficient number of sarcomeres actively shorten and if either or both ends of the muscle fiber are free to move. The active shortening of a muscle is called a **concentric contraction,** or **shortening contraction** (Fig. 3–6). In contrast to a shortening contraction, in which the thin filaments are pulled toward the thick filaments, the muscle may undergo an **eccentric contraction,** or **lengthening contraction.** In a lengthening contraction, the thin filaments are pulled away from the thick filaments, and cross-bridges are broken and re-formed as the muscle lengthens. Tension is generated by the muscle as cross-bridges are re-formed. Eccentric contractions occur whenever a muscle actively resists motion created by an external force (such as gravity). The muscle fiber will not change length if the force created by the cross-bridge cycling is matched by the external force. The contraction of a muscle fiber without changing length is called an **isometric contraction.**

Continuing Exploration 3-1:

Terminology: Muscle Action Versus Muscle Contraction

A potentially more accurate way to describe the types of muscle "contraction" is to use the term *action*. Because the word *contract* means to draw together or shorten, it is an oxymoron to refer to an "eccentric contraction" or "lengthening contraction." However, during the eccentric contraction, the contractile units (cross-bridges) are attempting to contract and pull the thin filaments toward the thick filaments, but the external forces are greater than the internal forces, which results in lengthening. Therefore, the term *contraction* implies the attempt to shorten and the term *eccentric* describes the lengthening. We have chosen to use the more common term *contraction* in this text, but we realize that muscle *action* is a synonymous term.

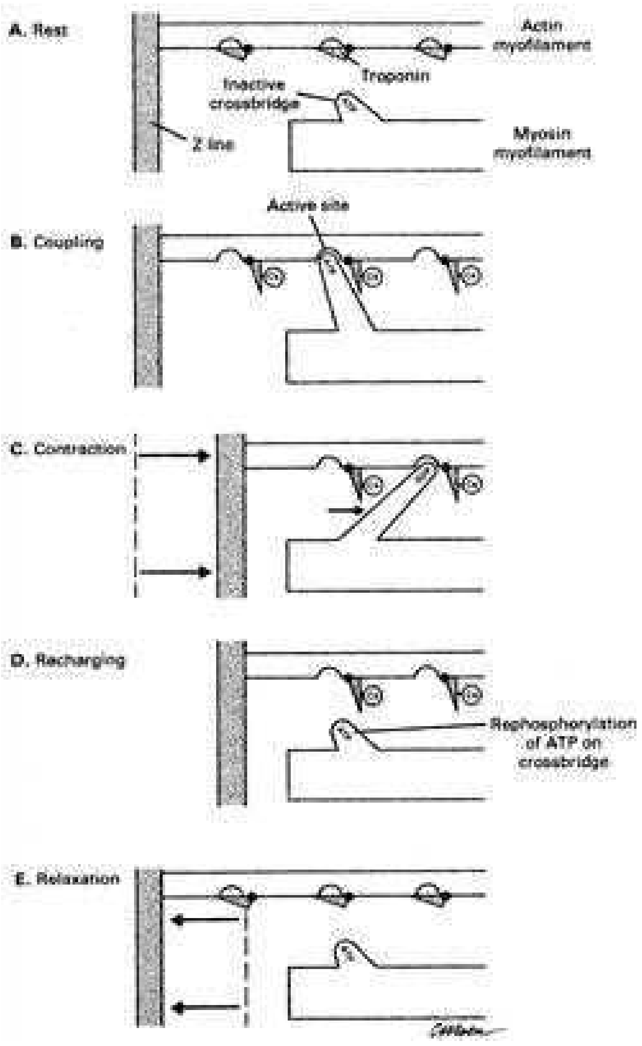


Figure 3–5 Cross-bridge cycle. **A.** Rest. Cross-bridges project from a myosin myofilament but are not coupled with an actin myofilament. Adenosine triphosphate (ATP) is attached near the head of the cross-bridge; troponin covers the active sites on the actin myofilament; and calcium ions are stored in the sarcoplasmic reticulum. **B.** Coupling. Arrival of the muscle action potential depolarizes the sarcolemma and T tubules; calcium ions are released and react with troponin; and the change in the shape of the troponin-calcium complex uncovers active sites on actin; a cross-bridge couples with an adjacent active site, thereby linking myosin and actin myofilaments. **C.** Contraction. Linkage of a cross-bridge and an active site triggers adenosine triphospatase (ATPase) activity of myosin; ATP splits into adenosine diphosphate (ADP) + PO_4 + energy; the reaction produces a transient flexion of the cross-bridge; the actin myofilament is pulled a short distance past the myosin myofilament; and Z discs are moved closer together. **D.** Recharging. The cross-bridge uncouples from the active site and retracts; ATP is replaced on the cross-bridge. The recoupling, flexion, uncoupling, retraction, and recharging processes are repeated hundreds of times per second. **E.** Relaxation. Cessation of excitation occurs; calcium ions are removed from the vicinity of the actin myofilament and are returned to storage sites in the sarcoplasmic reticulum; troponin returns to its original shape, covering active sites on the actin myofilament; and actin and myosin myofilaments return to the rest state. *(From Smith LK, Weiss EL, Lemkuhl LD (eds): Brunnstrom's Clinical Kinesiology (ed. 5). Philadelphia, FA Davis, 1996, p 83, with permission.)*

CASE APPLICATION

Possible Mechanism of Injury

case 3–1

In the case study at the beginning of the chapter, our patient, Vik, stepped back with his right foot, causing the foot to go into dorsiflexion. The external force was greater than the muscle force; therefore, the muscle was lengthened as it tried to resist the external force (an example of an eccentric contraction). This situation is a common mechanism of injury to the muscle and/or tendon that will result in pain localized to the muscle or tendon.

Concept Cornerstone **3-1**

Muscle Contraction Facts

The following is a summary of the important facts about muscle contraction at the sarcomere level:

- Tension is generated whenever cross-bridges are formed.
- Calcium influx initiates the muscle contraction.
- ATP hydrolysis fuels the cross-bridge cycle.
- In a concentric contraction, the thin myofilaments are pulled toward the thick myofilaments, and cross-bridges are formed, broken, and re-formed.
- In an eccentric contraction, the thin myofilaments are pulled away from the thick myofilaments, and cross-bridges are broken, re-formed, and broken again.
- In an isometric contraction, the length of the sarcomere is constant.

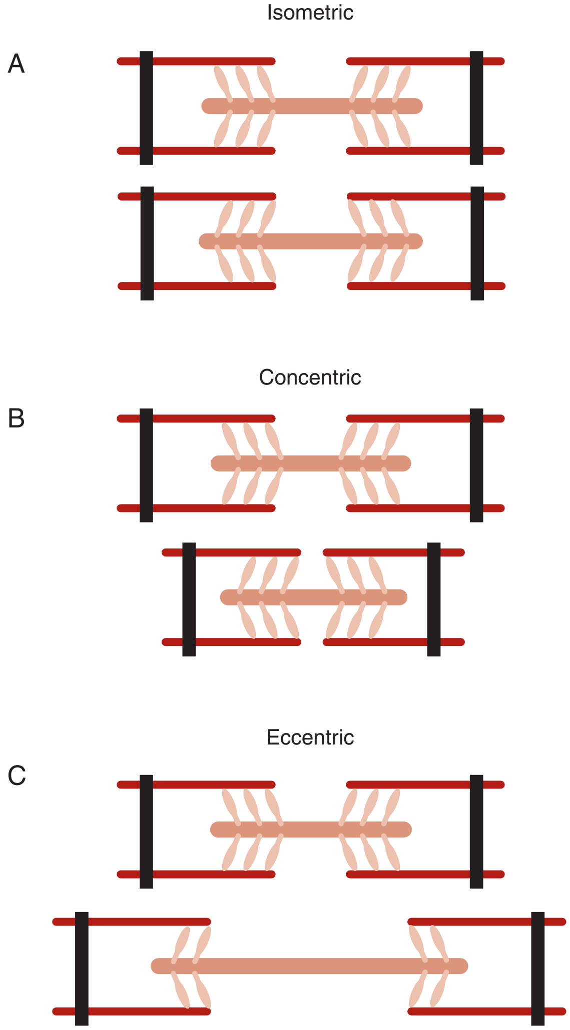


Figure 3–6 Types of muscle contraction from the perspective of change (or lack of change) in sarcomere length during the contraction. **A.** Isometric contraction with no change in length. **B.** Concentric or shortening muscle contraction. **C.** Eccentric or lengthening muscle contraction. The top illustration in each type of contraction represents the beginning of the contraction, and the bottom illustration represents the end of the contraction.

The Motor Unit

Organization of the Motor Unit

Although the sarcomere is the basic unit of tension in a muscle, it is actually part of a larger complex called the **motor unit.** The motor unit consists of the **alpha motor neuron** and all of the muscle fibers it innervates. The stimulus that the muscle fiber receives initiating the contractile process is transmitted through an alpha motor neuron (Fig. 3–7). The cell body of the neuron is located in the ventral horn of the spinal cord. The nerve cell **axon** extends from the cell body to the muscle, where it divides into either a few or as many as thousands of smaller branches. Each of the smaller branches terminates in a motor end plate that lies in close approximation to the sarcolemma of a single muscle fiber. All of the muscle fibers on which a branch of the axon terminates are part of one motor unit, along with the cell body and the axon.

The contraction of the entire muscle is the result of many motor units firing *asynchronously* and *repeatedly.*[1] The magnitude of the contraction of the entire muscle may be altered by changing the number of motor units that are activated or the frequency at which they are activated. The number of motor units in a muscle, as well as the structure of these units, varies from muscle to muscle.

Motor units vary according to the size of the neuron cell body, diameter of the axon, number of muscle fibers, and type of muscle fibers.[1] Each of these variations in structure affects the function of the motor unit. Some motor units have small cell bodies, and others have large cell bodies. Units that have small cell bodies have small-diameter axons (see Fig. 3–7). Nerve impulses take longer to travel through small-diameter axons than they do through large-diameter axons. Therefore, in the small-diameter units, a stimulus will take longer to reach the muscle fibers than it will in a unit with a large-diameter axon.

The size of the motor unit is determined by the number of muscle fibers that it contains and the size of the motor nerve axon (see Fig. 3–7). The number of fibers may vary from two or three to a few thousand. Muscles that either control fine movements or are used to make

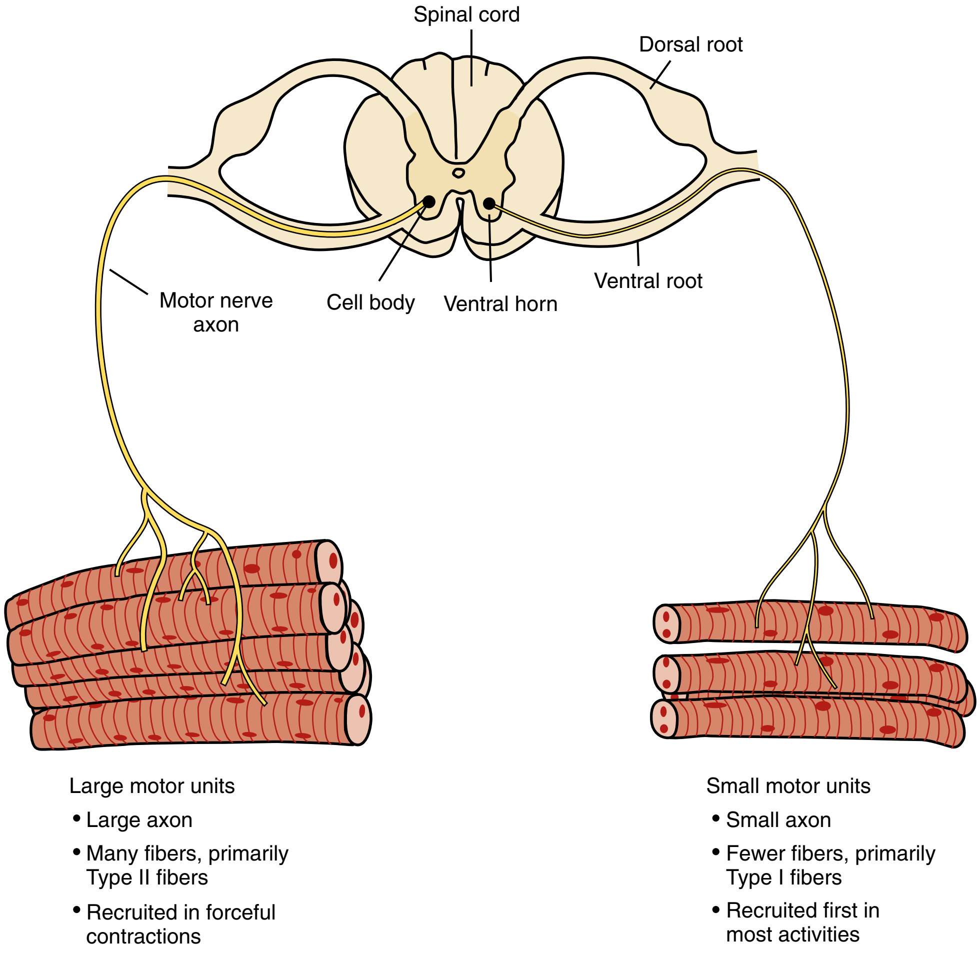


Figure 3–7 An alpha motor neuron. The cell body is in the ventral horn with the motor axon leaving the ventral root. As can be seen in the diagram, the muscle fibers innervated by a single axon are not necessarily located next to one another. The size of the motor unit is determined by the number of muscle fibers that it contains and by the size of the motor nerve axon. Large units may contain as many as a few thousand muscle fibers, whereas small units may contain as few as three.

small adjustments have small motor units. Such motor units generally have small cell bodies and small-diameter axons. Muscles that are used to produce large increments of force and large movements usually have a predominance of large motor units, large cell bodies, and large-diameter axons. The motor units of the small muscles that control eye motions may contain as few as nine muscle fibers, whereas the gastrocnemius muscles have motor units that contain about 2,000 muscle fibers.[1] Muscles with a predominantly large number of fibers per motor unit usually have a relatively small total number of motor units than do muscles that have few fibers per motor unit. The platysma muscle in the neck has relatively small motor units consisting of approximately 25 muscle fibers each, but the muscle has a total of 1,000 of these motor units. The gastrocnemius, on the other hand, has relatively large motor units consisting of about 2,000 muscle fibers per unit, but the muscle has a relatively small number (600) of such units. In most instances, a muscle has at least some mix of small and large motor units.

Recruitment of Motor Units

Usually, when an isometric muscle action is desired, the motor units with the small cell bodies and few motor fibers are recruited first by the nervous system and then, as force is increased, larger motor units are recruited.[11,12] This recruitment strategy is referred to as the **size principle of motor unit recruitment.**[11] Small motor units generate less tension than do large motor units and require less energy expenditure, and therefore this recruitment strategy is based ultimately on the force required for the motion. In this sense, it is thought to be energy conserving. If a few small motor units are capable of accomplishing the task, the recruitment of large motor units is unnecessary. If the task demands are such that the small motor units are unable to complete the task, larger motor units can be recruited. However, the recruitment strategy may be based not only on energy conservation but also on the nature of the task[13] (multitask muscles such as the biceps brachii are recruited differently for flexion than they are for supination) and the type of muscle action[12,14] (concentric, eccentric, or isometric) may be different for each motion. The recruitment strategy may also vary among the individual muscles of a synergistic group, such as the quadriceps femoris, despite all having a common functional task.[15] The frequency of a motor unit's firing also affects the force modulation. The contribution of recruitment or firing frequency to the development of muscle force may be different, depending on the muscle. Small, distal muscles tend to rely more on the increased frequency of firing, and larger, proximal muscles may rely more on the recruitment of additional motor units.[16] Finally, a more rapid recruitment of motor units may occur during fatiguing contractions in which a certain *position* is maintained than when a constant *force* is maintained.[17] It should be obvious from this brief discussion that motor unit recruitment is a complicated and multifaceted topic.

CASE APPLICATION

Possible Role of High-Velocity Eccentric Contraction in Injury

case 3–2

Vik was most likely performing a high-velocity eccentric contraction of the plantar flexor muscles, and he may have been selectively recruiting fast motor units rather than relying on the sequential recruitment of slow motor units and then fast motor units. This may have contributed to the high forces he experienced and increased his chance of injury.

Concept Cornerstone **3-2**

Summary of Factors Affecting Active Muscle Tension

To review, tension of the whole muscle may be affected by:

- the number of muscle fibers (which affects the magnitude of the response to a stimulus).
- the diameter of the axon (which determines the conduction velocity of the impulse).
- the number of motor units that are firing at any one time (which affects the total response of the muscle).
- the frequency of motor unit firing (which affects the total response of the muscle).

In addition, the type of muscle fibers contained within a motor unit will affect the response of a muscle. All of the muscle fibers contained in a single motor unit are of one type, but the type of muscle fibers within a muscle may vary from one motor unit to another motor unit.

Muscle Structure

Fiber Types

Three principal types of muscle fibers are found in varying proportions in human skeletal muscles. These fiber types may be distinguished from one another histochemically, metabolically, morphologically, and mechanically. Although there are different systems of nomenclature used in different texts, in this text the three primary muscle fiber types will be referred to as **type I (slow), type IIA (intermediate),** and **type IIX (fast)** (Table 3–1).[1,3,4] In this classification system, which is the most common system for human skeletal muscle fiber typing, the myofibrillar ATPase activity under varying acidic and alkaline conditions is used to delineate fiber types. In fact, several intermediate fiber types have been identified through this scheme.

Continuing Exploration 3-2:

Another Muscle Fiber Classification Scheme

Another classification scheme uses the response of the muscle to metabolic enzymes. This scheme identifies three main fiber types: fast glycolytic (FG), fast oxidative glycolytic (FOG), and slow oxidative (SO).[3,18,19] This nomenclature is based on the combination of reactions of cellular enzymes with substrates to identify myofibrillar ATPase activity (fast versus slow), succinate dehydrogenase activity (oxidative potential), and α-glycerophosphate dehydrogenase activity (glycolytic potential). It is often assumed that the two schemes are interchangeable; however, this may not be the case. There appears to be much overlap of metabolic enzyme activity between type IIA and type IIX. The fact that metabolic enzyme activity levels depend on the degree of training of the muscle suggests that these two schemes may not be the same. Another scheme, using immunohistochemical analysis (identification of portions of the myosin molecule using antibodies), has found that the type I, IIA, and IIX fibers correspond specifically to different types of myosin molecules (myosin heavy chain [MHC] I, MHC IIA, and MHC IIX).[18] The combination of this scheme and the myosin ATPase system provides an estimate of the contractile properties of the muscle. Whichever classification scheme is used, it should be remembered that there is actually a continuum of fiber types without exact distinctions between types.

Each skeletal muscle in the body is composed of a combination of each of the three types of fibers, but variations exist among individuals in the percentage of each fiber type

Table 3–1 **Characteristics of Skeletal Muscle Fibers**

	TYPE I (SLOW OXIDATIVE)	TYPE IIA (FAST OXIDATIVE GLYCOLYTIC)	TYPE IIX (FAST GLYCOLYTIC)
Diameter	Small	Intermediate	Large
Muscle Color	Red	Red	White
Capillarity	Dense	Dense	Sparse
Myoglobin Content	High	Intermediate	Low
Speed of contraction	Slow	Fast	Fast
Rate of Fatigue	Slow	Intermediate	Fast

in similar muscles. The variations in fiber types among individuals are believed to be genetically determined. In postmortem studies, the vastus lateralis, rectus femoris, and deltoid muscles contain about 50% type II and 50% type I fibers[2]; the gastrocnemius contains about 30% type II and 70% type I; and the hamstrings contain about 50% to 55% type II and 45% to 50% type I fibers.[20,21,22] In studies using muscle biopsy samples from younger subjects, the vastus lateralis tends to be about 54% type II fibers and 46% type I fibers.[23] Although the differences may be subtle, fiber type changes with age so that there is a decrease in the number and size of the type II fibers. This may account for the differences seen in many of the studies documenting fiber type percentages.

The soleus muscle, on the other hand, contains up to 80% type I fibers.[20,24] Muscles that have a relatively high proportion of type I fibers in relation to type II fibers, such as the soleus muscle, are able to carry on sustained activity because the type I fibers do not fatigue rapidly. These muscles are often called **stability** or **postural** muscles. The relatively small, slow motor units of the soleus muscle (with small cell bodies, small-diameter axons, and a small number of muscle fibers per motor unit) are almost continually active during erect standing in order to make the small adjustments in muscle tension that are required to maintain body balance and counteract the effects of gravity. Muscles that have a higher proportion of the type II fibers, such as the hamstring and gastrocnemius muscles, are sometimes designated as **mobility** or **nonpostural** muscles. These muscles are involved in producing a large range of motion (ROM) of the bony components.[20,21,22] Type II fibers produce slightly more force per fiber than type I fibers and are able to contract at a significantly higher velocity, thus producing a higher power output than type I fibers.[25] Although muscle fiber type is important in determining the function of a muscle, there are other aspects of muscle structure that also play an important role in determining muscle function.

CASE APPLICATION

Muscle Fiber Type Identification of Injured Muscle

case 3–3

Vik injured his plantar flexor muscles, which include the gastrocnemius and soleus muscles. However, because fiber type is related to muscle function, it is reasonable to make assumptions about which muscle may have been preferentially injured. Because the soleus muscle is composed primarily of type I fibers (postural control) and the gastrocnemius is composed primarily of type II fibers (power and mobility), the gastrocnemius was likely selectively recruited and therefore more likely injured.

Muscle Architecture: Size, Arrangement, and Length

Many human muscles have an approximately equal proportion of fast and slow fiber types. Therefore, the determination of muscle function should not be based solely on this single characteristic. In fact, the architecture of the whole muscle may be more important in determining muscle function than the fiber type.[26] The description of skeletal muscle architecture includes the arrangement of the fibers in relation to the axis of force **(amount of pennation), muscle fiber length, muscle length, muscle mass,** and the **physiological cross-sectional area (PCSA).** These structural variations affect not only the overall shape and size of the muscles but also the function of the skeletal muscles.

The two most important architectural characteristics that affect muscle function are the muscle fiber length and the PCSA. The fiber length (or the number of sarcomeres along the fiber) directly determines the amount of shortening or lengthening of the fiber. Consequently, a long muscle fiber, with more sarcomeres in series, is capable of shortening over a greater distance than a short muscle fiber. For example, if muscle fibers are capable of shortening to about 50% of resting length, a muscle fiber that is 6 cm long is able to shorten by 3 cm, whereas a fiber that is 4 cm long is able to shorten by only 2 cm. The significance of the preceding example is that a hypothetical muscle with long fibers is able to move the bony lever to which it is attached through a greater distance than is a muscle with short fibers. However, the relationship between the muscle fiber length and the distance that it is able to move a bony lever is not always a direct relationship. The arrangement of the muscle fibers and the length of the moment arm (MA) of the muscle affect the length-shortening relationship, and therefore, both fiber length and MA must be considered.

The PCSA is a measure of the cross-sectional area of the muscle perpendicular to the orientation of the muscle fibers. The amount of force that a muscle produces is directly proportional to the number of sarcomeres aligned side by side (or in parallel). Therefore, if there are a large number of fibers packed into a muscle (as in a pennate muscle) or if the fiber increases in size (addition of myofibrils), the ability to produce force will be increased.[26] A good example of the relationship between muscle architecture and function is the comparison between the quadriceps and hamstring muscles. The quadriceps muscles have a larger PSCA, and the hamstring muscles have longer fibers.[27,28] This architectural arrangement suggests that the quadriceps muscles are designed for force production and the hamstring muscles are designed for movements requiring a larger ROM. Because most of the hamstring muscles cross two joints (hip and knee), it would be expected that the muscle would need longer fibers for the greater excursion during movements of both the hip and the knee.

Arrangement of fascicles (muscle fiber groups) varies among muscles. The fasciculi may be parallel to the long axis of the muscle, as in fusiform-shaped muscles (Fig. 3–8A), or at an angle to the long axis in either a unipennate (Fig. 3–8B), bipennate (Fig. 3–8C), or multipennate (Fig. 3–8D) arrangement. Muscles that have a parallel fiber arrangement (parallel to the long axis and to each other) are designated as **fusiform** or **strap** muscles. In fusiform muscles such as the sternocleidomastoid or sartorius muscle, the fascicles are long and extend throughout the length of the muscle. However, in the

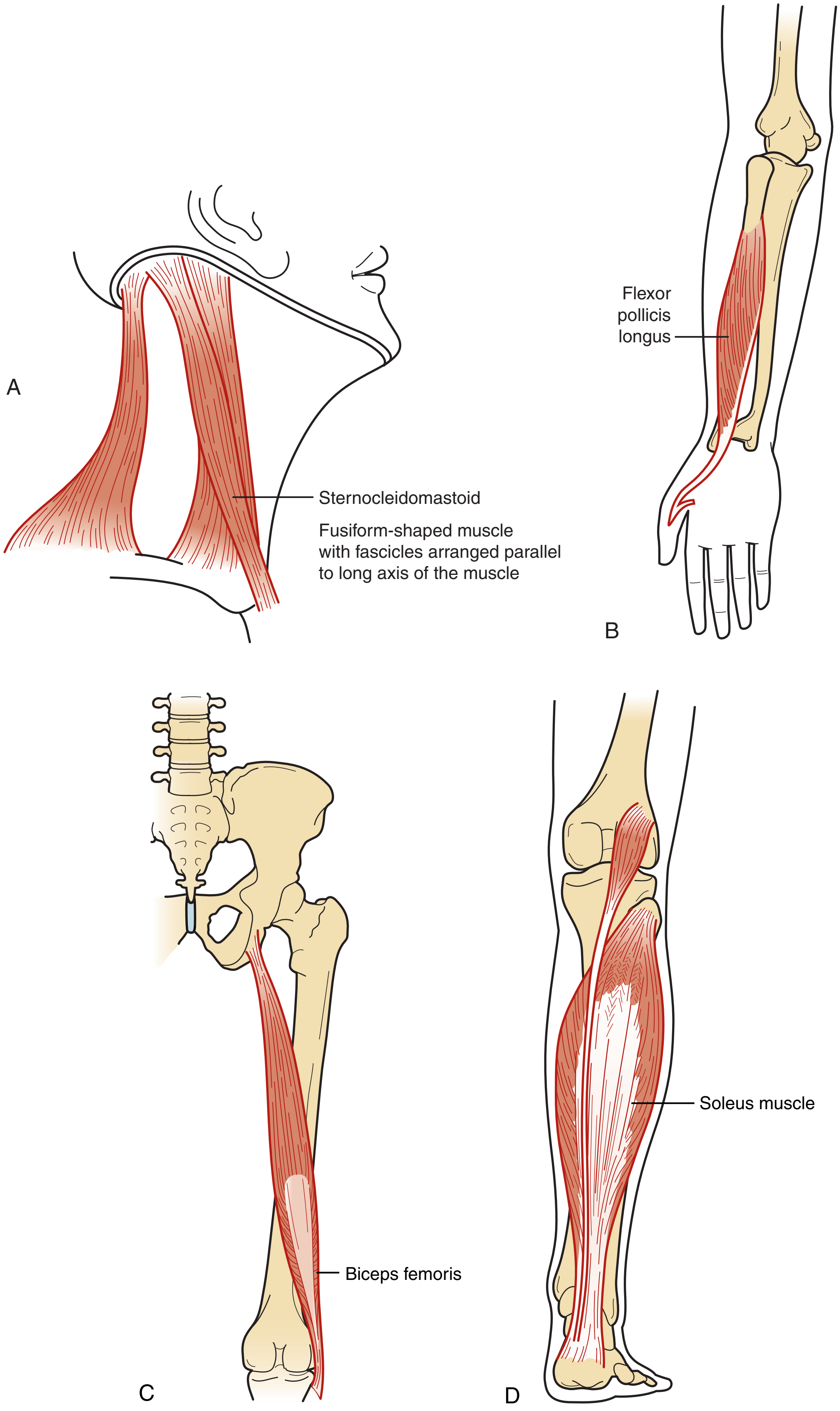


Figure 3–8 Arrangement of fasciculi in a muscle. **A.** Fusiform. **B.** Unipennate. **C.** Bipennate arrangement. **D.** Multipennate.

rectus abdominis, which also is considered a fusiform muscle, the fascicles are divided into short segments by fibrous intersections. In general, fusiform muscles with a parallel fiber arrangement have longer fibers and therefore produce a greater ROM of a bony lever than will muscles with a pennate fiber arrangement.

Muscles that have a fiber arrangement oblique to the muscle's long axis are called **pennate muscles** because the fiber arrangement resembles that found in a feather. The fibers that make up the fascicles in pennate muscles are usually shorter and more numerous than the fibers in many of the fusiform muscles. In **unipennate** muscles, such as the flexor pollicis longus, the obliquely set fascicles fan out on only one side of a central muscle tendon. In a **bipennate** muscle, such as the biceps femoris and the tibialis anterior, the fascicles are obliquely set on both sides of a central tendon. In a **multipennate** muscle, such as the soleus or subscapularis, the oblique fascicles converge on several tendons.

The oblique angle of the muscle fibers in a pennate muscle disrupts the direct relationship between the length of the muscle fiber and the distance that the total muscle can move a bony part and decreases the amount of force that is directed along the long axis of the muscle. Only a portion of the force of the pennate muscles goes toward producing motion of the bony lever. In fact, the more oblique a fiber lies to the long axis of the muscle, the less force the muscle is able to exert at the tendon. This decrease in muscle force is a function of the cosine of the pennation angle. Many human muscles have a pennation angle that is less than 30° at rest. Therefore, the muscle force at the tendon will be decreased by a maximum of 13% (cos 30° = 0.87).[3] When muscle shortens during muscle contraction and joint movement, the pennation angle becomes much more oblique, thus potentially affecting the tendon force even more.[9] This potentially decreased force at the tendon, however, is offset because pennate muscles usually have a large number of muscle fibers as a result of increased fiber packing, thus increasing PCSA. Therefore, despite the loss of force as a result of pennation, a pennated muscle, such as the soleus, is still able to transmit a large amount of force to the tendon to which it attaches.

Muscular Connective Tissue

Organization of Connective Tissue in Muscle

Muscles and muscle fibers, like other soft tissues in the body, are surrounded and supported by connective tissue. The sarcolemma of individual muscle fibers is surrounded by connective tissue called the **endomysium,** and groups of muscle fibers are covered by connective tissue called the **perimysium.** The myofibril is connected to the endomysium via specialized proteins. The endomysium and perimysium are continuous with the outer connective tissue sheath called the **epimysium,** which envelops the entire muscle (Fig. 3–9). The **myotendinous junction** is an intricate connection between muscle fibers and the connective tissue of the tendon. Many special proteins are arranged to make this junction strong. Tendons are attached to bones by Sharpey fibers, which become continuous with the periosteum.

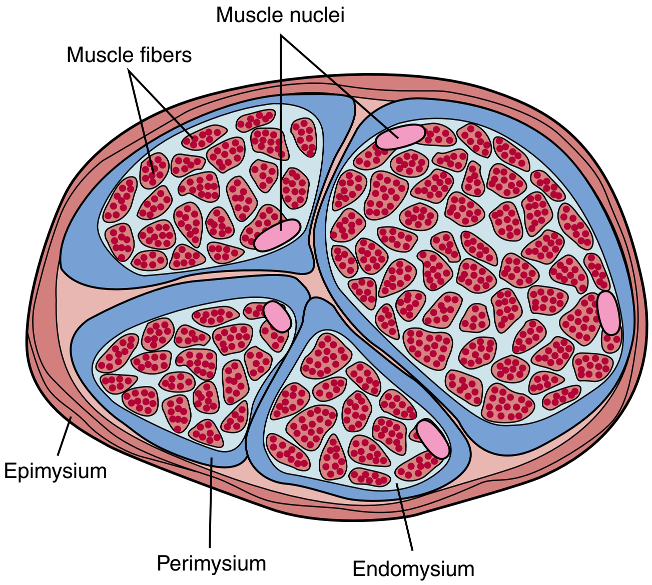


Figure 3–9 Muscular connective tissue. A schematic cross-sectional view of the connective tissue in a muscle shows how the perimysium is continuous with the outer layer of epimysium and the endomysium surrounds each muscle fiber.

CASE APPLICATION

Myotendinous Junction as a Possible Site of Injury

case 3–4

Although the myotendinous junction is designed to be strong and able to transmit the larger forces from the muscle to the tendon, the myotendinous junction is often the site of muscle strain injuries. This could be one explanation for the location of the injury in the case of our softball player patient.

Other connective tissues associated with muscles are in the form of fasciae, aponeuroses, and sheaths. Fasciae can be divided into two zones: superficial and deep. The zone of **superficial fasciae,** composed of loose tissue, is located directly under the dermis. This zone contributes to the mobility of the skin, acts as an insulator, and may contain skin muscles such as the platysma in the neck. The zone of **deep fasciae** is composed of compacted and regularly arranged collagenous fibers. The deep fasciae attach to muscles and bones and may form tracts or bands and retinacula. For example, the deep femoral fasciae in the lower extremity forms a tract known as the **iliotibial tract** or **band.** This tract transmits the pull of two of the lower-extremity muscles (the tensor fascia latae and the gluteus maximus) to the bones of the leg (Fig. 3–10). **Retinacula** are formed by localized transverse thickenings of the fasciae, which form a loop that is attached at both ends to bone (Fig. 3–11A). The tunnels formed by retinacula retain or prevent tendons from bowing out of position during muscle action (Fig. 3–11B). Sometimes deep

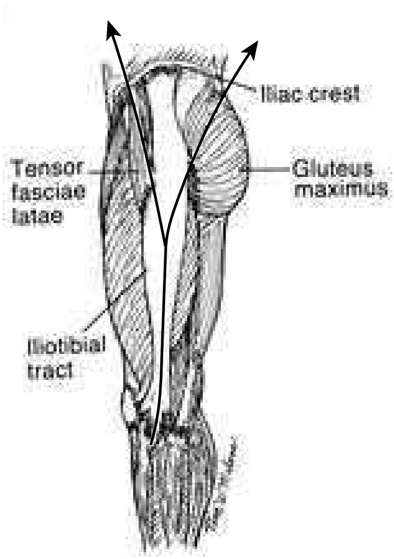


Figure 3–10 Iliotibial tract. A lateral view of the left lower limb showing the deep fascial iliotibial tract extending from the tubercle of the iliac crest to the lateral aspect of the knee. The right arrow represents the pull of the gluteus maximus. The left arrow represents the pull of the tensor fasciae latae.

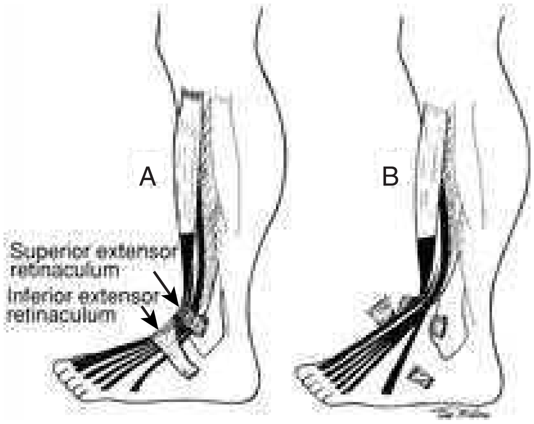


Figure 3–11 Retinacula. **A.** The superior and inferior retinacula are shown in their normal position, in which they form a tunnel for the tendons from the extensor muscles of the lower leg. **B.** When the retinacula are torn or removed, the tendons move anteriorly.

fasciae are indistinguishable from aponeuroses, which are sheets of dense, white, compacted collagen fibers that attach directly or indirectly to muscles, fasciae, bones, cartilage, and other muscles. Aponeuroses distribute forces generated by the muscle to the structures to which they are attached.[29]

Parallel and Series Elastic Components of Muscle

All of the connective tissue in a muscle is interconnected and constitutes the **passive elastic component** of a muscle. The connective tissues that surround the muscle, plus the sarcolemma, the elastic protein titin, and other structures (i.e., nerves and blood vessels), form the **parallel elastic component** of a muscle. When a muscle lengthens or shortens, these tissues also lengthen or shorten, because they function in parallel with the muscle contractile unit. For example, the collagen fibers in the perimysium of fusiform muscles are slack when the sarcomeres are at rest but straighten out and become taut as sarcomere lengths increase. As the perimysium is lengthened, it also becomes stiffer (resistance to further elongation increases). The increased resistance of perimysium to elongation may prevent overstretching of the muscle fiber bundles and may contribute to the tension at the tendon.[30,31] When sarcomeres shorten from their resting positions, the slack collagen fibers within the parallel elastic component **buckle (crimp)** even further. Whatever tension might have existed in the collagen at rest is diminished by the shortening of the sarcomere. Because of the many parallel elastic components of a muscle, the increase or decrease in passive tension can substantially affect the total tension output of a muscle. This relationship between length and tension will be addressed in the next section.

The tendon of the muscle is considered to function in **series** with the contractile elements. This means that the tendon will be under tension when the muscle actively produces tension. When the contractile elements in a muscle actively shorten, they exert a pull on the tendon. The pull must be of sufficient magnitude to take up the slack (compliance) in the tendon so that the muscle pull can be transmitted through the tendon to the bony lever (Fig. 3–12). Fortunately, the **compliance** (or **extensibility**) of the tendon is relatively small (about 3% to 5% in human muscles). Thus, most of the muscle force can be used for moving the bony lever and is not dissipated stretching the tendon. The tendon is also under tension when a muscle is controlling or braking the motion of the lever in an eccentric contraction. A tendon is under reduced tension only when a muscle is completely relaxed and in a relatively shortened position.

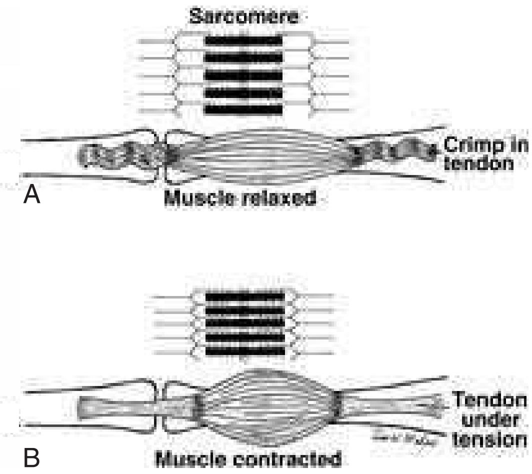


Figure 3–12 Series elastic component. **A.** The muscle is shown in a relaxed state with the tendon slack (crimping or buckling of collagen fibers has occurred). The sarcomere depicted above the muscle shows minimal overlap of thick and thin filaments and little cross-bridge formation. **B.** The muscle in an actively shortened position shows that the tendons are under tension and no crimp can be observed. The sarcomere depicted above the muscle shows extensive overlap of filaments and cross-bridge formation.

MUSCLE FUNCTION

Muscle Tension

The most important characteristic of a muscle is its ability to develop tension and to exert a force on the bony lever. Tension can be either active or passive, and the total tension that a muscle can develop includes both active and passive components. Total tension, which was identified in Chapter 1 as Fms, is a vector quantity that has (1) magnitude, (2) two points of application (at the proximal and distal muscle attachments), (3) an action line, and (4) direction of pull. The point of application, action line, and direction of pull were the major part of the discussion of muscle force in Chapter 1, but we now need to turn our attention to the determinants of the component called **magnitude of the muscle force,** or the **total muscle tension.**

Passive Tension

Passive tension refers to tension developed in the parallel elastic component of the muscle. Passive tension in the parallel elastic component is created by lengthening the muscle beyond the slack length of the tissues. The parallel elastic component may add to the active tension produced by the muscle when the muscle is lengthened, or it may become slack and not contribute to the total tension when the muscle is shortened. The total tension that develops during an active contraction of a muscle is a combination of the passive (noncontractile) tension added to the active (contractile) tension (Fig. 3–13).

Continuing Exploration 3-3:

Passive Muscle Stiffness

Passive muscle stiffness is an important property of skeletal muscle. The passive stiffness of an isolated muscle (not connected to bones and joints) is the slope of the passive length-tension relationship. The steeper the slope, the greater the stiffness in the muscle. The passive stiffness of a muscle attached to bone and crossing a joint is the slope of the torque-angle relationship. **Titin** is the primary component of the muscle that accounts for the stiffness of the muscle.[32] On the other hand, the connective tissues in and around the muscle (perimysium and endomysium) are responsible for the extent to which the muscle can be elongated.[30] This is often referred to as the muscle **extensibility** or **flexibility** (Fig. 3–14).

Active Tension

Active tension refers to tension developed by the contractile elements of the muscle. Active tension in a muscle is initiated by cross-bridge formation and movement of the thick and thin filaments. The amount of active tension that a muscle can generate depends on neural factors and mechanical properties of the muscle fibers. The neural factors that can modulate the amount of active tension include the frequency, number, and size of motor units that are firing. The mechanical properties of muscle that determine the active tension are the isometric length-tension relationship and the force-velocity relationship.

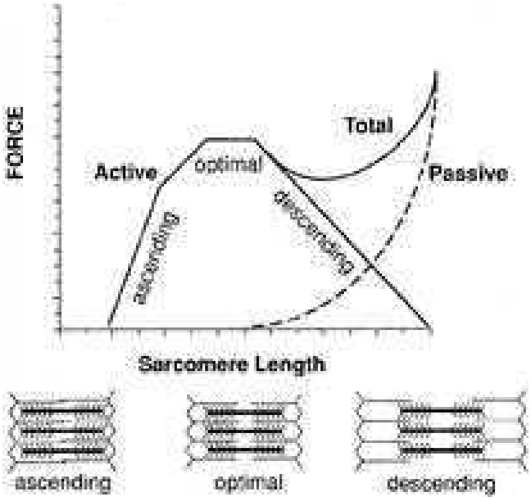


Figure 3–13 The skeletal muscle sarcomere length-tension relationship. Active, passive, and the total curves are shown. The plateau of the active curve signifies optimal sarcomere length at which maximum active tension is developed. Isometric tension decreases as the muscle is lengthened because fewer cross-bridges can be formed. Tension decreases as the muscle is shortened because of interdigitation of the thin filaments. The increase in passive tension with elongation of the muscle is shown by the dashed line. Passive plus active tension results in the total amount of tension developed by the muscle fiber.

Isometric Length-Tension Relationship

One of the most fundamental concepts in muscle physiology is the direct relationship between isometric tension development in a muscle fiber and the length of the sarcomeres in a muscle fiber.[33] The identification of this relationship was, and continues to be, the primary evidence supporting the sliding filament theory of muscle contraction. The isometric sarcomere length-tension relationship was experimentally determined with isolated single muscle fibers under very controlled circumstances. There is an optimal sarcomere length at which a muscle fiber is capable of developing maximal isometric tension (see Fig. 3–13). Muscle fibers develop maximal isometric tension at optimal sarcomere length because the thick and thin filaments are positioned so that the maximum number of cross-bridges within the sarcomere can be formed. If the muscle fiber is lengthened or shortened beyond optimal length, the amount of active tension that the muscle fiber is able to generate when stimulated decreases (see Fig. 3–13). When a muscle fiber is lengthened beyond optimal length, there is less overlap between the thick and thin filaments and consequently fewer possibilities for cross-bridge formation. However, the passive elastic tension in the parallel component may be increased when the muscle is elongated. This passive tension is added to the active tension, resulting in the total tension (see Fig. 3–13).

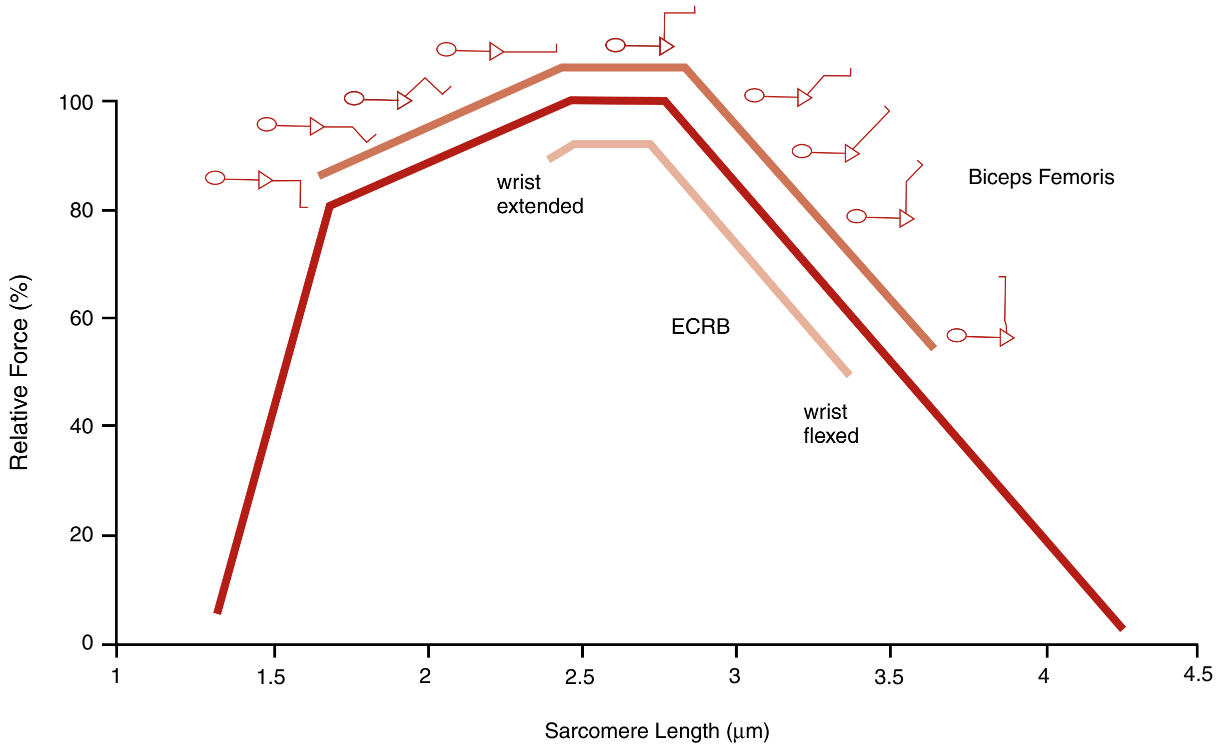


Figure 3–14 The sarcomere length-tension relationship for the human extensor carpi radialis brevis (ECRB) and the biceps femoris, long head. The data for the ECRB were determined from intraoperative laser diffraction measurement of the sarcomere length, and the data from the biceps femoris were calculated on the basis of ultrasound measurements of the change in fascicle length with joint position.[35] The dark line is the presumed length-tension relationship of human muscle, based on the known lengths of human thin and thick filaments. *(Adapted from Chleboun GS, France AR, Crill MT, et al: In vivo measurement of fascicle length and pennation angle of the human biceps femoris muscle. Cells Tissues Organs 169:401, 2001 with permission.)*

A similar loss of isometric tension or diminished capacity for developing tension occurs when a muscle fiber is shortened from its optimal sarcomere length. When the sarcomere is at shorter lengths, the distance between the Z discs decreases and there is interdigitation of the filaments. The interdigitation of the thick and thin filaments may interfere with the formation of cross-bridges from the myosin molecules, thus decreasing the active force.

It must be remembered that the sarcomere length-tension relationship was determined with isometric contractions and therefore should apply, in the strict sense, only to isometric muscle contraction. In addition, as we will see in the following section, the full range of sarcomere lengthening and shortening may be possible only in experiments with isolated muscle. Sarcomere length obviously changes during dynamic contractions (concentric and eccentric contractions) that affect the tension that can be developed in the muscle. However, during dynamic contractions, *the length-tension relationship must be combined with the force-velocity relationship to determine the effect that both length and velocity have on muscle tension.*

Application of the Length-Tension Relationship

In applying the length-tension relationship to whole muscle and ultimately to muscle-joint systems is not a simple matter. For example, sarcomere length is not homogeneous throughout the muscle, let alone between muscles with similar functions. This means that for any particular whole muscle length at a particular joint position, there may be sarcomeres at many different lengths corresponding to different points on the length-tension relationship. Also, when the muscle is acting at a joint, the torque produced is not only a function of the muscle force (which depends on muscle length) but also a function of the **moment arm (MA)** of the muscle. This means that at a certain joint angle, the muscle length may be short (which suggests that force will be low) but the MA may be relatively long, thus maintaining a higher joint torque. From these examples it is clear that the sarcomere length-tension relationship is important in our understanding of muscle physiology, but there are other important factors when whole muscle and joint systems are considered.

Only a few experiments have attempted to determine the isometric sarcomere length-tension relationship in intact human muscle.[34–38] In these experiments on human wrist muscles and thigh muscles, the range of sarcomere length change that is seen with normal joint motion is quite small and is located around optimal length. This design appears to be quite beneficial in that the muscle is not disadvantaged by being too long or too short. Figure 3–14 shows the estimated length-tension relationship for the extensor carpi radialis brevis and the biceps femoris, long head.[28,34]

One empirical application of the sarcomere length-tension relationship is the observation that a muscle has the diminished ability to produce isometric tension at the extremes of joint motion. This probably occurs only in muscles that cross more than one joint (two-joint or multijoint muscles), in which muscle length excursion is greater than in single-joint muscles. A decrease in the torque produced by the muscle may be encountered when the full ROM is attempted simultaneously at all joints crossed by a multijoint muscle. This decrease in torque is often referred to as **"active insufficiency."** Although the decrease in isometric torque can be conveniently explained by the length changes in the muscle that result in decreased muscle force, other factors, such as the change in MA and the passive restraint of the lengthened antagonists, also play a substantial role. Sarcomere length appears to stay close to the optimal length during joint movements; therefore, influence of the MA and passive restraint of the antagonists may be more important than once thought.[34–39] Therefore, the term *active insufficiency* refers to a concept that is much broader than just the active length-tension properties of the muscle.

Example 3-1

The finger flexors cross the wrist, carpometacarpal, metacarpophalangeal, and interphalangeal joints (Fig. 3–15A). When the finger flexors shorten, they will cause simultaneous flexion at all joints crossed. If all of the joints are allowed to flex simultaneously, the finger flexors will probably be shorter and, as a result, may develop less tension. In addition, the finger extensors (antagonists) may be restricting motion and limiting force production. Normally, when the finger flexors contract, the wrist is maintained in slight extension by the wrist extensor muscles (Fig. 3–15B). The wrist extensors prevent the finger flexors from flexing the wrist, and therefore an optimal length of the flexors is maintained.

Force-Velocity Relationship

Another factor that affects the development of tension within a muscle is the speed of shortening of the myofilaments. The speed of shortening is the rate at which the myofilaments are able to slide past one another and form and re-form cross-bridges. Remember that the speed of shortening is related to muscle fiber type as well as muscle fiber length. The force-velocity relationship describes the relationship between the velocity of the muscle contraction and the force produced, thereby providing an explanation for what happens during concentric and eccentric muscle contractions. From the experiments on isolated muscles, the force-velocity relationship states that the velocity of muscle contraction is a function of the load being lifted,[40] but, from a clinical perspective, it may also be stated with the variables reversed (the force generated is a function of the velocity of the muscle contraction) (Fig. 3–16). For example, in a *concentric muscle contraction, as the shortening speed decreases, the tension in the muscle increases. In an isometric contraction, the speed of shortening is zero and tension is greater than in a concentric contraction. In an eccentric contraction, as the speed of lengthening increases, the tension in the muscle increases and then plateaus.* Not only is this relationship seen in experimental conditions with isolated muscles lifting a load, but it is also seen, to some degree, in intact muscle moving bony levers.[41,42]

CASE APPLICATION

Force-Velocity Curve Prediction

case 3–5

Vik's plantar flexor muscles were most likely contracting eccentrically as he was planting his foot. Therefore, the force-velocity curve would predict that the muscles were producing high forces, which potentially led to the injury of Vik's muscle or tendon.

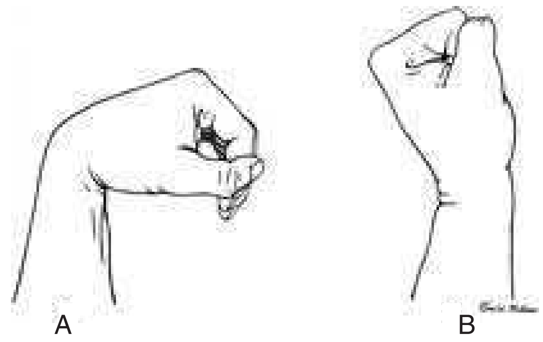


Figure 3–15 Decrease in active tension with muscle shortening. **A.** The individual is attempting to make a tight fist but is unable to do so because the finger flexors are shortened over both the flexed wrist and fingers. In addition, the finger extensors have become lengthened, potentially restricting motion. **B.** The length-tension relationship of both flexors and extensors has been improved by stabilization of the wrist in a position of slight extension. The individual, therefore, is able to form a tight fist.

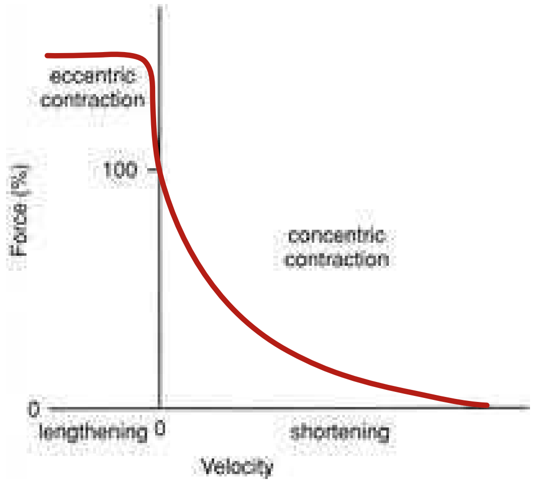


Figure 3–16 The skeletal muscle force-velocity curve. At maximum velocity of shortening, no force is produced (in other words, maximum velocity of shortening can be attained only with no load on the muscle). As shortening velocity decreases, the force that the muscle can develop increases. At zero velocity, the muscle contracts isometrically. Force increases dramatically and then plateaus when the muscle is lengthened actively.

Previously, it was mentioned that during dynamic contractions, the length-tension relationship must be combined with the force-velocity relationship because both sarcomere length and velocity of contraction affect the development of muscle tension. It cannot be assumed simply that as the muscle shortens, the tension developed in the muscle follows the isometric length-tension relationship. For example, at high shortening velocities, the muscle tension will be low, regardless of sarcomere length. The fact that most human movements do not occur at a constant velocity of contraction complicates the situation further because the force will vary with changing velocity and changing length.

Concept Cornerstone **3-3**

Factors Affecting Active Muscle Tension

In summary, active tension in the muscle can be modulated by several factors:

- Tension may be increased by increasing the frequency of firing of a motor unit or by increasing the number of motor units that are firing.
- Tension may be increased by recruiting motor units with a larger number of fibers.
- The greater the number of cross-bridges that are formed, the greater the tension.
- Muscles that have large physiological cross-sections are capable of producing more tension than are muscles that have small cross-sections.
- Tension increases as the velocity of active shortening decreases and as the velocity of active lengthening increases.

This basic understanding of the two most important mechanical properties of muscle, the length-tension relationship and the force-velocity relationship, can now be applied to clinical situations.

Types of Muscle Action

Muscle actions (or contractions) are described as **isometric contraction** (constant length) or dynamic contractions consisting of **concentric contraction** (shortening of the muscle under load) and **eccentric contraction** (lengthening of the muscle under load). The term *isotonic contraction* is not used here because it refers to equal or constant tension, which rarely, if ever, occurs during normal human movements. Therefore, the types of muscle actions that will be considered in the following section are isometric, concentric, and eccentric.

Previously, isometric, concentric, and eccentric muscle contractions were introduced in relation to the movement occurring at the sarcomere level. To review: when a muscle fiber is activated so that cross-bridges form, the sarcomeres in the fiber will either stay at constant length, shorten, or lengthen, depending on the load that is applied. An isometric contraction occurs when the muscle is activated and the sarcomere does not change length; a concentric contraction occurs when the sarcomere shortens; and an eccentric contraction occurs when the sarcomere lengthens (the load is greater than the force of the sarcomere).

We can apply this same idea to whole muscle that is attached to bone. When the whole muscle is activated and the bones it is attached to do not move, the muscle action is called an **isometric contraction** (Fig. 3–17). Holding the weight without changing the joint angle means that the muscle is contracting **isometrically.** During an isometric contraction, no work is being done because the joint is not moving. The formula for work is W = F × d, where W is work, F is the force created by the muscle, and d is the distance that the object is moved.

During a **concentric contraction,** the bones move closer together as the whole muscle shortens (Fig. 3–18). **Positive work** is being done by the muscle because the joint moves through a ROM. During an **eccentric contraction,** the bones move away from each other as the muscle tries to control the descent of the weight (Fig. 3–19). The muscle lengthens as the joint moves through the ROM. The work that is being done during an eccentric contraction is called **negative work** because the work is done *on* the muscle rather than *by* the muscle.

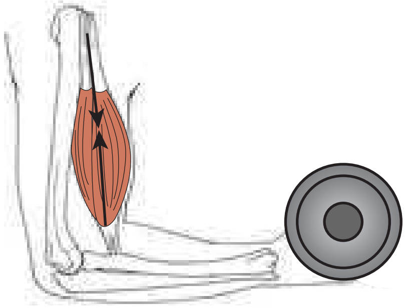


Figure 3–17 An isometric contraction. Both the distal and proximal bony levers are fixed, and no visible motion occurs when the muscle develops active tension.

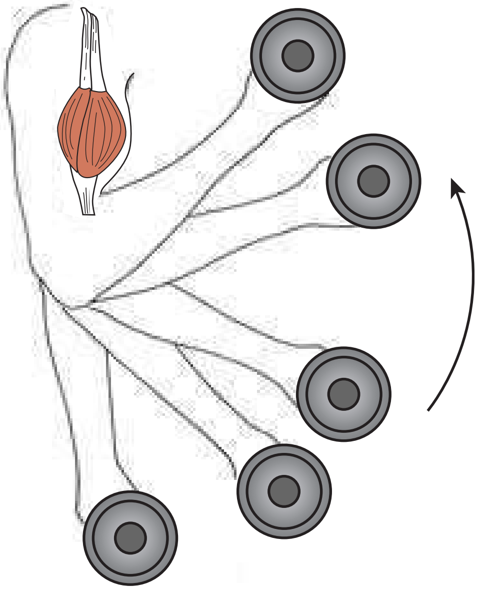


Figure 3–18 A concentric muscle contraction. The muscle shortens while continuing to produce active tension.

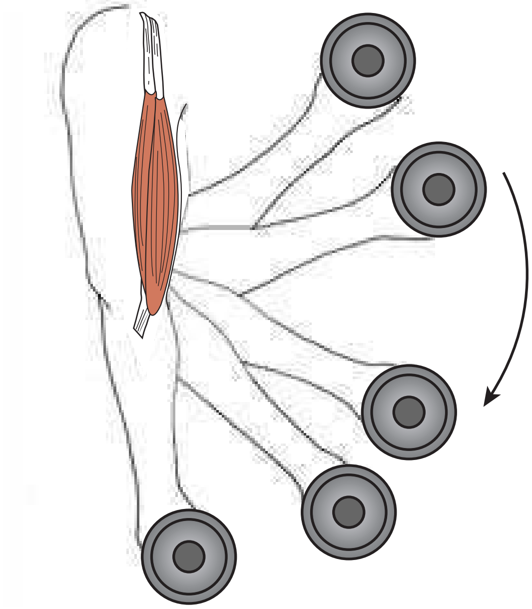


Figure 3–19 An eccentric contraction. The muscle elongates while continuing to produce active tension.

The amount of tension that can be developed in a muscle varies according to the type of contraction, as was seen in the force-velocity relationship. A greater amount of tension can be developed in an isometric contraction than in a concentric contraction.[43] In general, the tension developed in an eccentric contraction is greater than what can be developed in an isometric or concentric contraction.[44] However, this relationship may not hold true for all muscles at all points in a joint's ROM.[42,45] The greater tension development in a muscle during an eccentric contraction than during a concentric contraction may be due, in part, to either mechanical factors in the attachment and detachment of cross-bridges or to alterations in the neural activation of the muscle.[46]

The examples of concentric and eccentric muscle contractions in Figures 3–18 and 3–19 depict a common joint motion in which the distal segment (forearm) is free to move. Many times during normal functional activities, the proximal segment moves and the distal segment is stationary. For example, during a pull-up exercise (Fig. 3–20), the humerus moves and the forearm (ulna and radius) remain basically stationary. We sometimes refer to this as the **"reverse action"** of the muscle. Whether the distal segment or the proximal segment moves is dependent upon the desired motion, the effects of external forces, and the ability to stabilize other joints. Therefore, in the example of a pull-up, the biceps is shortening (concentric contraction) in order to raise the humerus (and the body).

Production of Torque

As clinicians, we often assess the patient's muscle strength. Whether we assess that strength by using an instrument (such as an isokinetic device) or by simple manual pressure, we are actually determining the amount of joint torque that the muscle can produce. In many physiological experiments on muscle, the muscle is isolated from the bone and the actual muscle force is measured as the muscle is activated. When the muscle is attached to bones in the body, the muscle still produces a force, but it now acts over a MA at the joint to produce a torque.

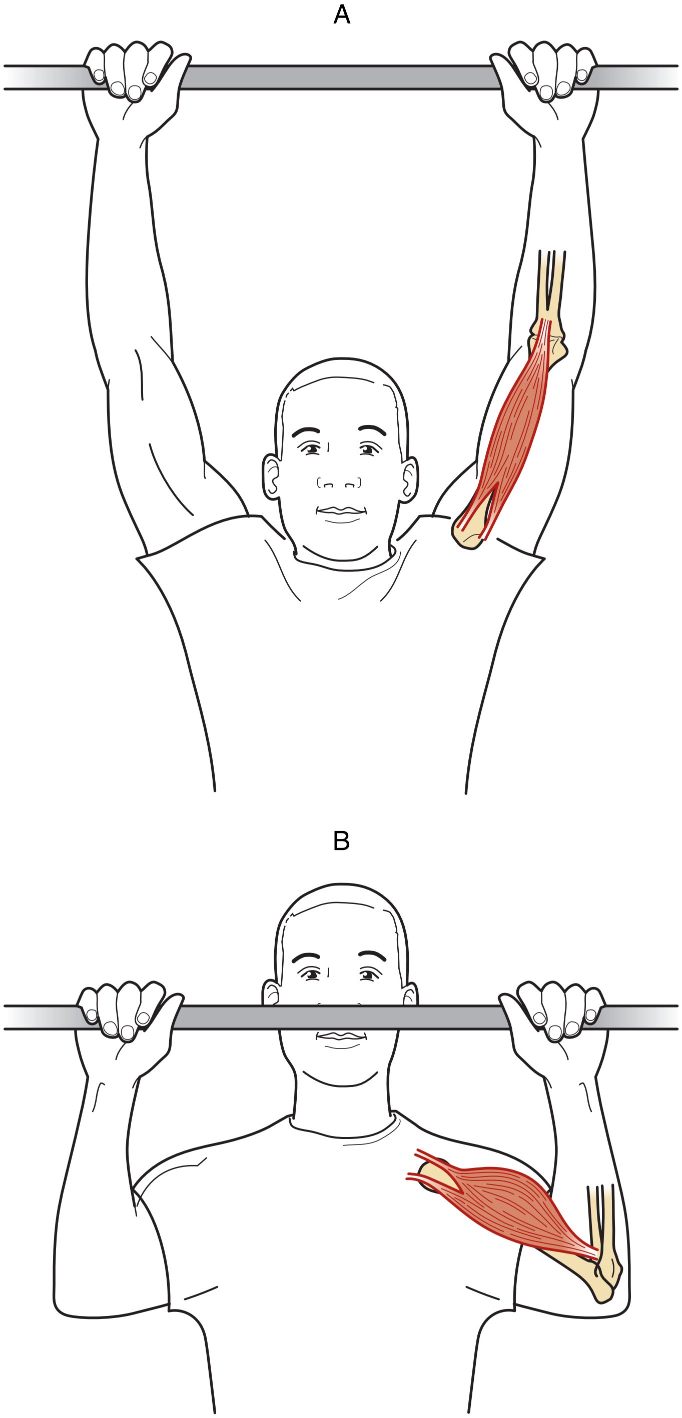


Figure 3–20 Pull-up exercise depicting the movement of the proximal segment (humerus) moving on the distal segment (forearm), which is sometimes referred to as the "reverse action" of the muscle. Movement from **A** to **B** is a concentric contraction of the elbow flexors.

The MA of the muscle can change with joint position, thereby affecting the torque being produced. For example, if the biceps brachii is activated to produce 10 pounds of force with a small MA at one joint position, the torque (muscle strength measured) will be less than if the MA of the muscle were larger at a different joint position (Fig. 3–21). Remember that as the joint moves, the muscle length changes. From the discussion of the length-tension relationship of the muscle, we know that the muscle force will vary as the muscle is lengthened or shortened. Therefore, at different joint positions, both the MA of the muscle and the length of the muscle affect the amount of joint torque that can be produced. In addition, during dynamic movements, the velocity of the shortening or lengthening affects the amount of force that the muscle produces, thus affecting the torque production.[3,42]

Interaction of Muscle and Tendon

The interaction of muscle and tendon (including the aponeurosis) during muscle contraction and movement has some important functional implications. Let us begin with a simple example. During an isometric contraction (as shown in Fig. 3–17), the muscle actually shortens slightly and the tendon lengthens slightly. In many muscles, the fibers may shorten and the tendon may lengthen by as much as 10% of their resting length during an isometric contraction (Fig. 3–22).[47] The compliance of the tendon (or ability to lengthen under load) is important in augmenting the torque production of the muscle. This is the basis for **plyometric exercises,** in which the muscle/tendon complex is stretched before a forceful concentric contraction. The stretch immediately before the concentric contraction helps produce a much greater torque during the concentric contraction. Although the exact mechanism for the increase in torque is debated, evidence shows that the muscle fibers tend to stay at constant length (isometric) while the tendon lengthens, storing energy to be used during the concentric contraction.[48–50]

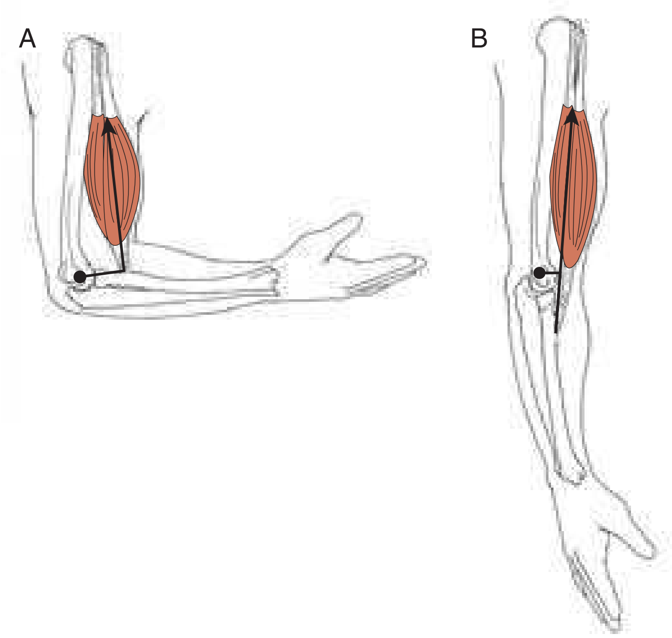


Figure 3–21 The torque generated by the muscle changes as the moment arm of the muscle changes during joint motion. In position **A,** the torque is greater than in position **B** because the moment arm of the biceps muscle force is greater (assuming that the force is the same in each position).

CASE APPLICATION

Achilles Tendon Involvement?

case 3–6

Because the Achilles tendon is one of the most compliant tendons in the human body, the patient may have caused undue stretching of the tendon when he stepped back. The high force of the eccentric contraction, coupled with the stretch of the tendon, could have easily caused a strain injury in the muscle and/or the tendon.

Isokinetic Exercise and Testing

Advances in technology have led to the development of testing and exercise equipment that provide for the manipulation and control of some of the variables that affect muscle function. In isokinetic exercise and testing[51] or isokinetic muscle contraction, the angular velocity of the bony component is preset and kept constant by a mechanical device throughout a joint ROM. The concept of an “isokinetic contraction” may not be so much a type of muscle action as it is a description of joint motion because the muscle fascicles do not shorten at constant velocity during an isokinetic movement.[52] Because the speed of joint motion is controlled by the isokinetic device, the resistance is directly proportional to the torque produced by the muscle at all points in the ROM. Therefore, as the torque produced by a muscle increases, the magnitude of the torque of the resistance increases proportionately. This provides an excellent means of testing muscle strength. Isokinetic devices such as a Biodex, Cybex, or Kin Com are commonly used in many clinics.

The proposed advantage of isokinetic exercise over free weight lifting through a ROM is that isokinetic exercise theoretically accommodates for the changing torques created by a muscle throughout the ROM. As long as the preset speed is achieved, the isokinetic device provides resistance that exactly matches the torque produced by a muscle group throughout the ROM. For example, the least amount of resistance is provided by an isokinetic device at the point in the ROM at which the muscle has the least torque-producing capability. The resistance provided is greatest at the point in the ROM at which the muscle has the largest torque-producing capability.[51]

The maximum isokinetic torques for concentric contractions obtained at high-angular velocities are less than at low-angular velocities. This decline in torque with increasing contraction velocity is expected on the basis of the force-velocity relationship of muscle. In fact, isometric torque values at any point in the ROM are higher than isokinetic concentric torque values at any velocity for a particular point in the joint ROM. Therefore, the closer the angular velocity of a concentric isokinetic contraction approaches to zero, the greater the isokinetic torque.[53,54]

Isokinetic equipment is used extensively for determining the amount of joint torque that a muscle can develop at different velocities, for strength training, and for comparing the

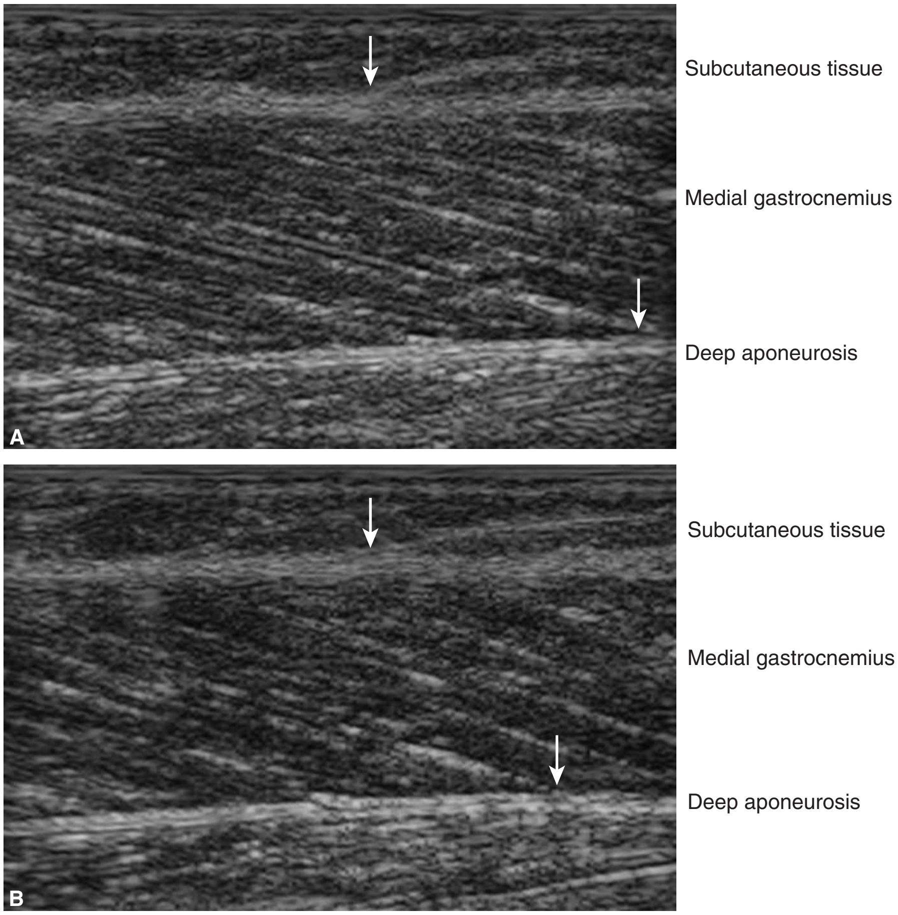


Figure 3–22 Ultrasound image of the medial gastrocnemius. **A.** Relaxed. **B.** Isometric contraction. The top arrow identifies the insertion of the fascicle into the aponeurosis of the tendon. Note how the insertion point (bottom arrow) moves to the left (proximal) relative to the top arrowhead. The top arrowhead in the subcutaneous tissue identifies a common reference structure in both images.

relative strength of one muscle group with another. Most isokinetic devices also permit quantification for testing eccentric muscle torque. Isokinetic evaluation of muscle strength has provided much important information about muscle function, and isokinetic exercise is effective for gaining strength. However, there are some limitations to isokinetic evaluation and exercise. For example, at higher speeds, the amount of ROM in which movement is at constant velocity is decreased, and functional movements are rarely performed at constant velocity. Some research shows that isokinetic testing or exercising may differentiate performance in functional tasks or may enhance the training effect for functional activities, and some research shows that it does not.[55–58]

Example 3-2

At various stages of rehabilitation, it is advantageous to limit the force that a muscle produces because we do not want to injure healing muscle or tendon tissue. The most obvious way to decrease muscle force is to decrease the resistance. However, based on the discussions in this section, the clinician may choose to increase the velocity of contraction. By exercising at a higher velocity on an isokinetic device (or even with a constant external resistance), the force will be decreased. Although this may not be a common choice for exercise training, it may prove useful in certain situations.

Summary of Factors Affecting Active Muscle Tension

Active muscle tension is affected by many factors. Some, such as the size and number of fibers, are intrinsic to the muscle, whereas other factors, such as the influence of the nervous system recruitment patterns, are extrinsic to the muscle.

Concept Cornerstone 3-4

Summary of Intrinsic and Extrinsic Factors Involving Active Muscle Tension

The velocity of muscle contraction is affected by:
- *recruitment order of the motor units:* Units with slow conduction velocities are generally recruited first.
- *type of muscle fibers in the motor units:* Units with type II muscle fibers can develop maximum tension more rapidly than units with type I muscle fibers; rate of cross-bridge formation, breaking, and re-formation may vary.
- *the length of the muscle fibers:* Long fibers have a higher shortening velocity than do shorter fibers.

The magnitude of the active tension is affected by:
- *size of motor units:* Larger units produce greater tension.
- *number and size of the muscle fibers in a cross-section of the muscle:* The larger the cross-section, the greater the amount of tension that a muscle may produce.

Continued

- *number of motor units firing:* The greater the number of motor units firing in a muscle, the greater the tension.
- *frequency of firing of motor units:* The higher the frequency of firing of motor units, the greater the tension.
- *sarcomere length:* The closer the length is to optimal length, the greater the amount of isometric tension that can be generated.
- *fiber arrangement:* A pennate fiber arrangement gives a greater number of muscle fibers and potentially a larger PCSA, and therefore a greater amount of tension may be generated in a pennate muscle than in a parallel muscle.
- *type of muscle contraction:* An isometric contraction can develop greater tension than a concentric contraction; an eccentric contraction can develop greater tension than an isometric contraction.
- *speed:* As the speed of shortening increases, tension decreases in a concentric contraction. As speed of active lengthening increases, tension increases in an eccentric contraction.

Classification of Muscles

Individual muscles may be named in many different ways, such as according to shape (rhomboids, deltoid), number of heads (biceps, triceps, quadriceps), location (biceps femoris, tibialis anterior), or a combination of location and function (extensor digitorum longus, flexor pollicis brevis). Groups of muscles are categorized on the basis of either the actions they perform or the particular role they serve during specific actions. When muscles are categorized on the basis of action, muscles that cause flexion at a joint are categorized as **flexors.** Muscles that cause either extension or rotation are referred to as **extensors** or **rotators,** respectively. When muscles are categorized according to role, individual muscles or groups of muscles are referred to as the **agonists, antagonists,** or **synergists** for a particular motion. In this section, we explore the classification of muscle based on the role of the muscle during movement as well as the classification based on muscle architecture and MA.

Based on the Role of the Muscle in Movement

The term **prime mover (agonist)** is used to designate a muscle whose role is to produce a desired motion at a joint. If flexion is the desired action, the flexor muscles are the prime movers and the extensor muscles that are directly opposite to the desired motion are called the **antagonists.** The desired motion is not opposed by the antagonists, but these muscles have the potential to oppose the action.

Ordinarily, when an agonist (for example, the biceps brachii) is called on to perform a desired motion (elbow flexion), the antagonist muscle (the triceps brachii) is inhibited. If, however, the agonist and the potential antagonist contract simultaneously, then **co-contraction** occurs (Fig. 3–23). Co-contraction of muscles around a joint can help to provide stability for the joint and represents a form of synergy that may be necessary in certain situations. Co-contraction of muscles with opposing functions can be undesirable when a desired motion is prevented by involuntary co-contraction, such as occurs in disorders affecting the control of muscle function (e.g., cerebral palsy).

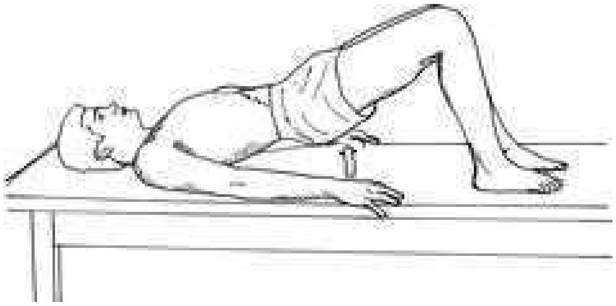


Figure 3–23 Co-contraction of an agonist and antagonist to stabilize the knees in a bridging exercise. *(From Kisner C, Colby LA: Therapeutic Exercise: Foundations and Techniques (ed. 5). Philadelphia, FA Davis, 2007, with permission.)*

Muscles that help the agonist to perform a desired action are called **synergists.**

Example 3-3

If flexion of the wrist is the desired action, the flexor carpi radialis and the flexor carpi ulnaris are referred to as the *agonists* or *prime movers* because these muscles produce flexion. The finger flexors are the synergists that might directly help the wrist flexors. The wrist extensors are the potential antagonists.

Synergists may assist the agonist directly by helping to perform the desired action, such as in the wrist flexion example, or the synergists may assist the agonist indirectly either by stabilizing a part or by preventing an undesired action.

Example 3-4

If the desired action is finger flexion, such as in the clenching of the fist, the finger flexors, which cross both the wrist and the fingers, cannot function effectively (a tight fist cannot be achieved) if they flex the wrist and fingers simultaneously. Therefore, the wrist extensors are used synergistically to stabilize the wrist and to prevent the undesired motion of wrist flexion. By preventing wrist flexion, the synergists are able to maintain the joint in a position that allows the finger flexors to develop greater torque, a combination of optimizing sarcomere length and MA.

Sometimes the synergistic action of two muscles is necessary to produce a pure motion such as radial deviation (abduction) of the wrist. The radial flexor, flexor carpi radialis, of the wrist acting alone produces wrist flexion and radial deviation. The radial extensor, extensor carpi radialis brevis and longus, acting alone produces wrist extension and radial deviation. When the wrist extensor and the wrist flexor work together as prime movers to produce radial deviation of the wrist, the unwanted motions are canceled and the pure motion of radial deviation results (Fig. 3–24). In this example, the muscles that are the potential antagonists of the desired motion are the wrist extensor and flexor on the ulnar side of the wrist (extensor carpi ulnaris and flexor carpi ulnaris, respectively).

Based on Muscle Architecture

Placing muscles into functional categories, such as flexor and extensor or agonists and antagonists, helps to simplify the task of describing the many different muscles and explaining their actions. However, muscles can change roles. A potential antagonist in one instance may be a synergist in another situation. An example of this can be found in the preceding discussion. The extensors and flexors on the ulnar side of the wrist are antagonists during the motion of radial deviation, but during ulnar deviation, these same muscles are synergists. Despite this apparent change in role, muscles that have similar functions also have similar architectural characteristics. This is intuitive, because muscle architecture plays such an important role in determining the potential force and velocity of muscle contraction, and several studies have confirmed that the functional groups of muscles have similar architecture. Examples of this may be seen in experiments with both animals and humans.[59–61] In the lower extremity, the knee extensors have a short fiber length and large PCSA, as opposed to the knee flexors, which have a longer fiber length and smaller PCSA (Fig. 3–25). The ankle plantar flexors typically have short fiber lengths and large PCSA, thus setting them apart from the ankle dorsiflexors, which have longer fiber lengths and smaller PCSA. In the upper extremity, the finger flexors have longer fiber lengths and greater PCSA, as opposed to the shorter and smaller finger extensors (Fig. 3–26). Although we can classify many muscles based on architecture, there are obvious exceptions. For example, the semimembranosus (a knee flexor) has a similar fiber length to the knee extensor muscles.

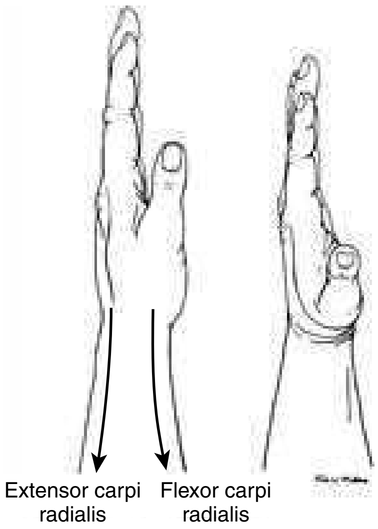


Figure 3–24 Synergistic muscle activity. When the flexor carpi radialis and the extensor carpi radialis work synergistically, they produce radial deviation of the wrist.

Based on Length of the Moment Arm

The orientation of the muscle to the joint has also been used to classify muscles into groups. The length of the muscle MA is an important component of determining the joint torque and, in combination with the fiber length, the ROM through which the muscle can move the joint.[3] The ratio of the fiber length to the MA provides a way of identifying which factor plays a greater role in the production of the joint torque and in determining the resulting ROM at the joint. For example, the ratio of fiber length to the MA is much higher in the wrist extensor muscles compared to the wrist flexor muscles. This suggests that fiber length plays a greater role than does the MA in the wrist extensors and that the MA plays a greater role than fiber length in the wrist flexors.[34]

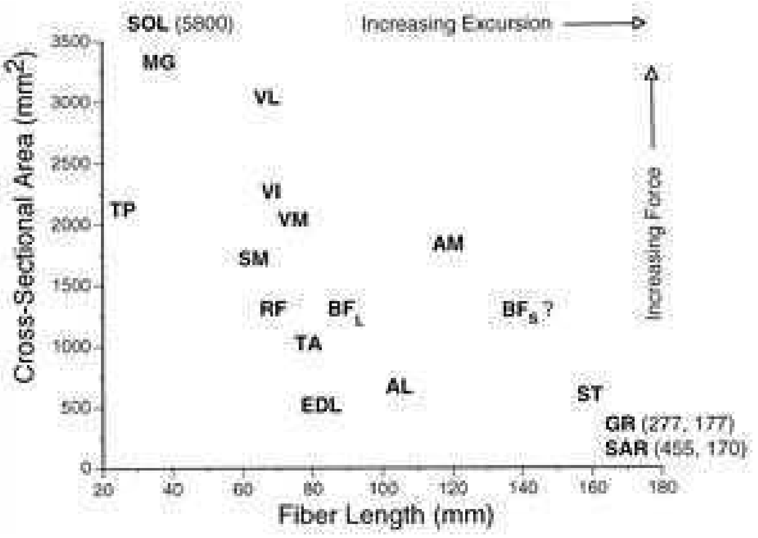


Figure 3–25 The relationship between PCSA and the fiber length of selected muscles of the lower extremity. Note the general grouping of muscles with similar function—for example, the soleus (SOL) and medial gastrocnemius (MG); the tibialis anterior (TA) and extensor digitorum longus (EDL); the vastus lateralis, vastus medialis, and vastus intermedius (VL,VM, VI); gracilis (GR); and sartorius (SAR) (Note: Data are only for fiber length of the biceps femoris short head [BFs].) *(Adapted from Lieber RL: Skeletal Muscle Structure and Function: Implications for Rehabilitation and Sports Medicine. Baltimore, Williams & Wilkins, 1992, with permission.)*

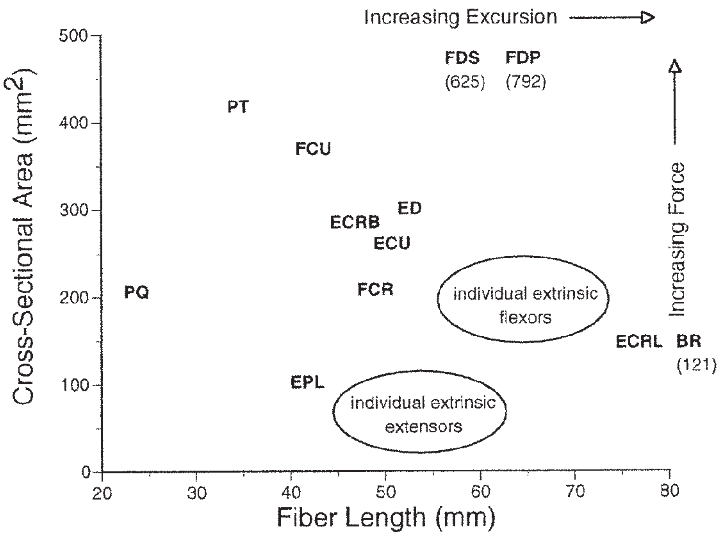


Figure 3–26 The relationship between PCSA and the fiber length of selected muscles of the upper extremity. Note the general grouping of muscles with similar function—for example, the flexor digitorum superficialis (FDS) and profundus (FDP); the extensor carpi radialis brevis (ECRB), extensor carpi ulnaris (ECU), and extensor digitorum (ED); and the individual finger flexors and extensors. *(Adapted from Lieber RL: Skeletal Muscle Structure and Function: Implications for Rehabilitation and Sports Medicine. Baltimore, Williams & Wilkins, 1992, with permission.)*

Although all skeletal muscles adhere to a general basic structural design, a considerable amount of variability exists among muscles in regard to the number, size, arrangement, and type of muscle fibers. Therefore, attempts to classify muscles into only a few groups may be inappropriate. According to the evidence that subpopulations of motor units from muscles, rather than groups of muscles, appear to work together for a particular motor task, a more appropriate way of describing muscle action might be in terms of motor units.[16] However, more research needs to be performed in this area before a motor unit classification system can be widely used and accepted.

Factors Affecting Muscle Function

In addition to the large number of factors affecting muscle function that were presented previously, a few other factors need to be considered:

- Types of joints and location of muscle attachments
- Number of joints crossed by the muscle
- Passive insufficiency
- Sensory receptors

Types of Joints and Location of Muscle Attachments

The type of joint affects the function of a muscle in that the structure of the joint determines the type of motion that will occur (flexion and extension) and the ROM. The muscle's location or line of action relative to the joint determines which motion the muscle will perform. In general, muscles that cross the anterior aspect of the joints of the upper extremities, trunk, and hip are flexors, whereas the muscles located on the posterior aspect of these joints are extensors. Muscles located laterally and medially serve as abductors and adductors, respectively, and may also serve as rotators. Muscles whose distal attachments are close to a joint axis are usually able to produce a wide ROM of the bony lever to which they are attached. Muscles whose distal attachments are at a distance from the joint axis, such as the brachioradialis, are designed to provide stability for the joint, because a large majority of their force is directed toward the joint that compresses the joint surfaces (Fig. 3–27). A muscle's relative contribution to stability will change throughout a motion as the rotatory and compressive components of the muscle's force vary indirectly with each other. A muscle provides maximum joint stabilization at the point at which its compressive component is greatest.

Usually one group of muscles acting at a joint is able to produce more torque than another group of muscles acting at the same joint. Disturbances of the normal ratio of agonist-antagonist pairs may create a muscle imbalance at the joint and may place the joint at risk for injury. For example, weakness of the hip external rotators causes a strength imbalance between hip external and internal rotation that may result in excess hip internal rotation during gait. Agonist-antagonist strength ratios for normal joints are often used as a basis for establishing treatment goals after an injury to a joint.

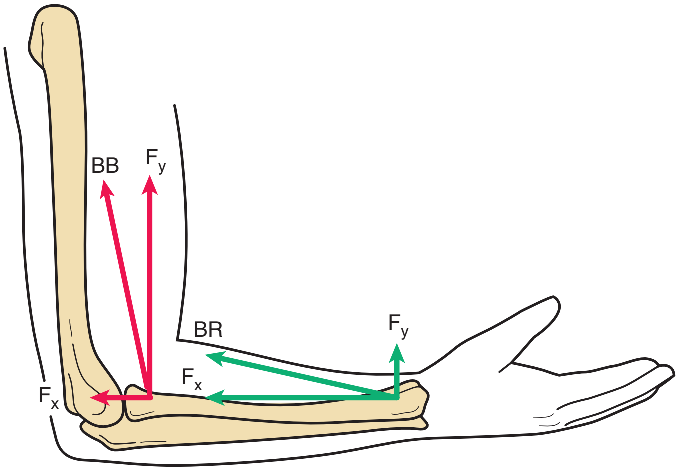


Figure 3–27 The biceps brachii (BB) and the brachioradialis (BR) force vectors with their rotational (Fy) and compressive (Fx) components. The compressive component of the brachioradialis is greater than the compressive component of the biceps.

Number of Joints

Many functional movements require the coordinated movement of several joints controlled by a combination of muscles that cross one or many of the joints. To produce a purposeful movement pattern, many authorities believe that the control of the movement is designed to minimize necessary muscle force to accomplish the task (least motor unit activity) and thus minimize muscle fatigue. These strategies of motor control attempt to ensure that movement is done efficiently.

One way of providing an efficient movement pattern is through the coordinated efforts of single-joint and multijoint muscles. In many ways, the number of joints that the muscle crosses determines the muscle function. Single-joint muscles tend to be recruited to produce force and work, primarily in concentric and isometric contractions. This recruitment strategy occurs primarily when a simple movement is performed at one joint, but it may also be used during movements involving multiple joints. Multijoint muscles, in contrast, tend to be recruited to control the fine regulation of torque during dynamic movements involving eccentric more than concentric muscle actions.[62,63] Multijoint muscles tend to be recruited during more complex motions requiring movement around multiple axes. For example, the movement of elbow flexion with concurrent supination uses the biceps brachii (a multijoint muscle) with added contribution of the brachialis (a single-joint muscle). This may seem obvious because of the attachment of the biceps brachii to the radius, which allows supination, whereas the brachialis attaches to the ulna and allows only flexion of the elbow.[64] If a single-joint motion is desired, a single-joint muscle is recruited because recruitment of a multijoint muscle may require the use of additional muscles to prevent motion from occurring at the other joint or joints crossed by the multijoint muscles. For example, elbow flexion with the forearm in pronation is accomplished primarily with the brachialis, not with the biceps brachii.

Single-joint and multijoint muscles may also work together in such a way that the single-joint muscle can assist in the movement of joints that it does not cross.[65] For example, the simple movement of standing up from a chair requires knee and hip extension. The hip extension is accomplished by activation of the single-joint hip extensor muscles (gluteus maximus) and the multijoint hip extensors (hamstrings). The concomitant knee extension is accomplished by activation of the single-joint knee extensor muscles (vastus muscles) and the multijoint knee extensors (rectus femoris). An interesting corollary is that the single-joint knee extensors may actually assist in hip extension in this movement of standing from a chair. If the hamstrings are active, the knee extension (produced by the vastus muscles) will pull on the active hamstring muscles (which act as a tie-rod), which results in hip extension (Fig. 3–28).

Passive Insufficiency

If a person's elbow is placed on a table with the forearm in a vertical position and the hand is allowed to drop forward into wrist flexion, the fingers tend to extend (Fig. 3–29A). Extension of the fingers is a result of the insufficient

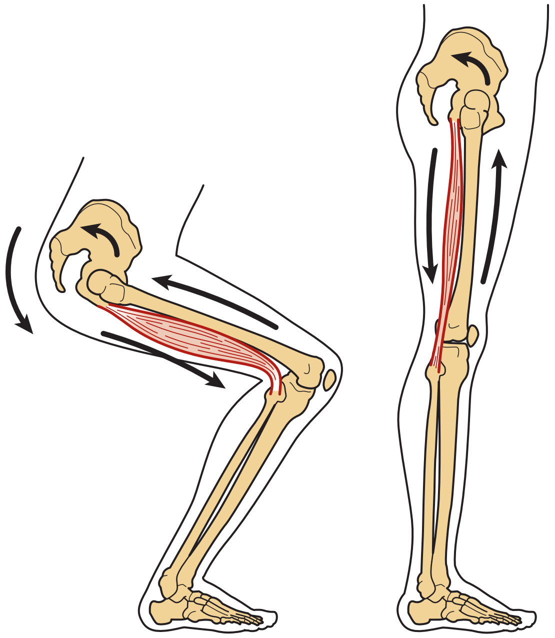


Figure 3–28 An example of the two-joint function of the hamstring muscles in transferring force of the single-joint knee extensors to extend the hip.

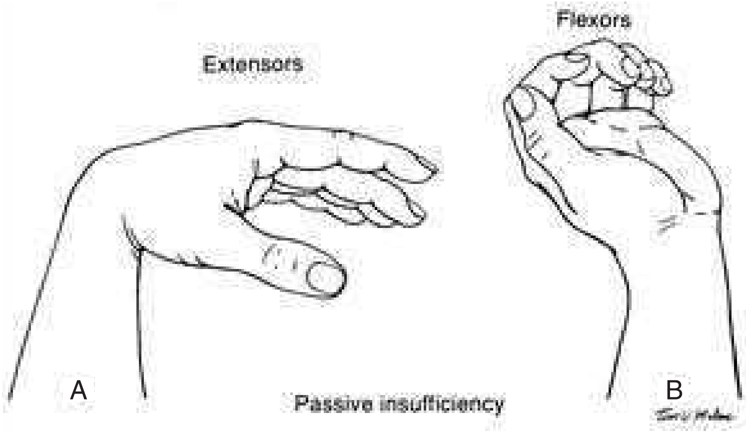


Figure 3–29 Passive insufficiency. **A.** The finger extensors become passively insufficient as they are lengthened over the wrist and fingers during wrist flexion. The passive tension that is developed causes extension of the fingers (tenodesis). **B.** The finger flexors become passively insufficient as they are lengthened over the wrist and fingers during wrist extension. The passive tension developed in the finger flexors causes the fingers to flex.

length of the finger extensors that are being stretched over the flexed wrist. The insufficient length is termed **passive insufficiency.** If the person moves his or her wrist backward into wrist extension, the fingers will tend to flex (Fig. 3–29B). Flexion of the fingers is a result of insufficient extensibility of the finger flexors as they are stretched over the extended wrist. The term *passive insufficiency* implies an abnormal situation—when, in fact, it may actually be quite normal.

Under normal conditions, one-joint muscles rarely, if ever, are of insufficient extensibility to allow full ROM at the joint. Two-joint or multijoint muscles, however, frequently are of insufficient extensibility to permit a full ROM to be produced simultaneously at all joints crossed by these muscles. The passive tension developed in these stretched muscles is sufficient to either cause motion, as in the wrist example above, or to limit motion, as in the following example.

Example 3-5

The finger flexors and extensors are an excellent example of this principle. When the finger flexors actively flex the wrist and fingers, the finger extensors are being lengthened over the wrist and finger joints, thereby limiting the amount of finger and wrist flexion.

In this example, at the same time that the finger flexors are shortened, the inactive finger extensors are being passively stretched over all of the joints that they cross. The extensors are providing a passive resistance to wrist and finger flexion at the same time that the finger flexors are having difficulty performing the movement (Fig. 3–30). Similar examples can be found in the rectus femoris and the hamstring muscles in the lower extremities. The rectus femoris may limit active knee flexion if the hip is in a position of extension, and the hamstrings may limit active knee extension if the hip is in a position of flexion. Such positions are not usually encountered in normal activities of daily living, but they may be encountered in sports activities.

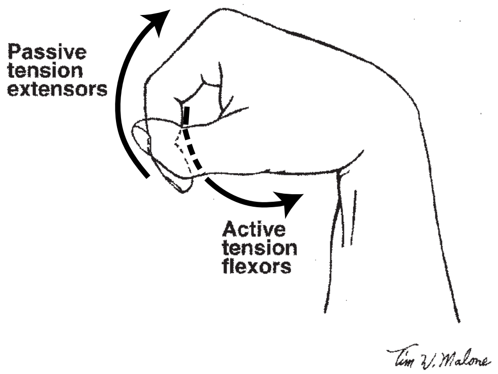


Figure 3–30 Increase in passive tension with passive muscle lengthening. When a person attempts to make a tight fist with the wrist fully flexed, the active shortening of the finger and wrist flexors results in passive lengthening of the finger extensors. The length of the finger extensors is insufficient to allow full range of motion at both the wrist and the fingers and therefore passively limits the ability of the finger flexors to make a tight fist.

Sensory Receptors

Normal motor control resulting in voluntary movements depends on the coordination of descending motor pathways from the cortex, muscle actions, and a constant flow of sensory information. When performing or participating in athletic events, highly skilled dancers and athletes always seem to make it look so easy. In order to achieve this level of skill, the movements were practiced frequently, requiring constant sensory information from many systems. But in the muscle, feedback comes from two important sensory receptors: the **Golgi tendon organ (GTO)** and the **muscle spindle.** The Golgi tendon organs, which are located in the tendon at the myotendinous junction, are sensitive to tension and may be activated either by an active muscle contraction or by an excessive passive stretch of the muscle. When the Golgi tendon organs are excited, they send a message to the nervous system to adjust the muscle tension (Fig. 3–31). The fine-tuning of muscle tension is not accomplished alone, however, because other reflex systems (muscle spindles) are also providing feedback to the muscle.

The **muscle spindles,** which consist of 2 to 10 specialized muscle fibers **(intrafusal fibers)** enclosed in a connective tissue sheath, are interspersed throughout the muscle. These spindle fibers are sensitive to the length and the velocity of lengthening of the muscle fibers **(extrafusal fibers).** They send messages to the brain (cerebellum) about the state of stretch of the muscle. When the muscle fiber shortens, the spindles stop sending messages because they are no longer stretched. When the signal decreases, the higher centers send a message to the intrafusal muscle fibers in the spindle to shorten so that they once again are able to respond to the length change in the muscle.

The muscle spindle is responsible for sending the message to the muscle in which it lies to contract when the tendon of the muscle is tapped with a hammer (Fig. 3–32). The quick stretch of the muscle caused by tapping the tendon activates the muscle spindles, and the muscle responds to the unexpected spindle message with a brief contraction. This response is called by various names: for example, **deep tendon reflex (DTR), muscle spindle reflex (MSR),** or simply **stretch reflex.** Both the Golgi tendon organs and the muscle spindles provide constant feedback to the central nervous system during movement so that appropriate adjustments can be made, and they help protect the muscle from injury by monitoring changes in muscle length.

The presence of the stretch reflex is beneficial for preventing muscle injury but presents a problem in treatment programs or fitness programs, in which stretching of a muscle is desirable for improving flexibility and restoring a full range of joint motion. Muscle contraction or reflex activation of motor units during intentional stretching of a muscle may create a resistance to the stretching procedure and makes stretching more difficult and possibly ineffective. Methods of stretching that may prevent reflex activity and motor unit activation during stretching have

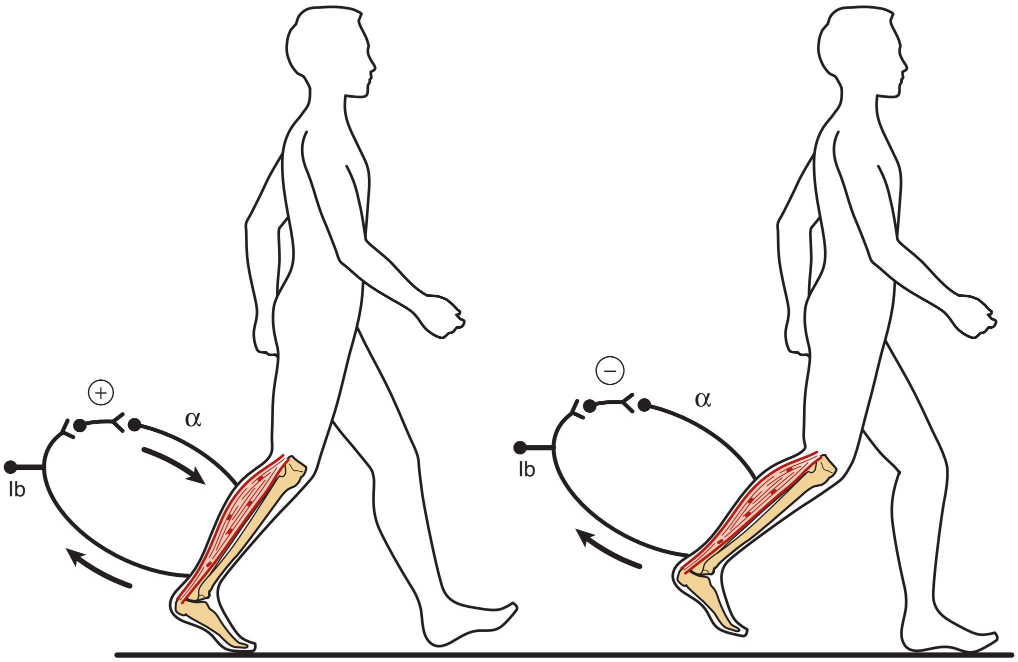


Figure 3–31 The function of the Golgi tendon organ during gait. *(Redrawn from Lundy-Eckman, L: Neuroscience: Fundamentals for Rehabilitation (ed. 3). Saunders, St. Louis, MO, 2007, with permission.)*

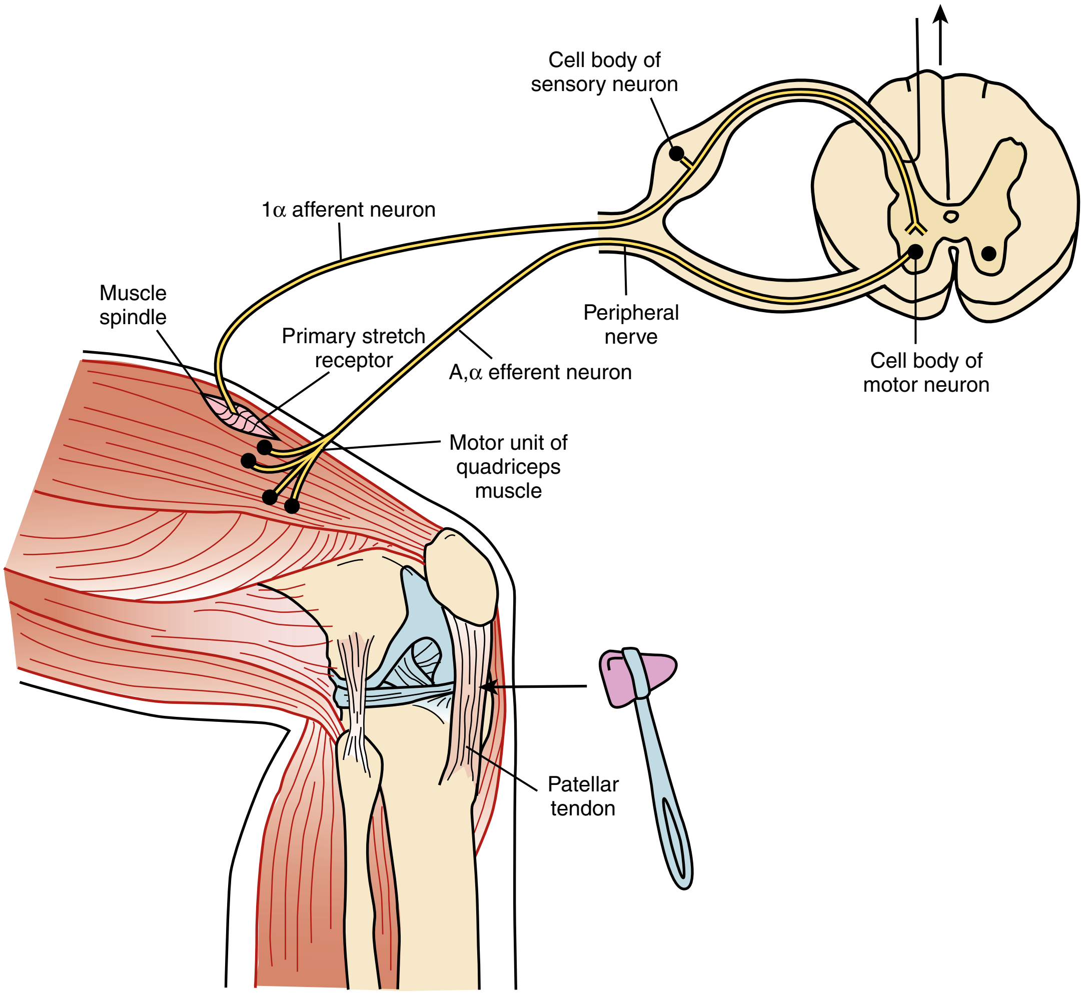


Figure 3–32 The stretch reflex. When the muscle spindle is stretched by the tap of the hammer, the 1α afferent neuron sends a message to the alpha motor neuron, which results in contraction of the stretched muscle. *(Adapted from Smith LK, Weiss EL, Lehmkuhl LD (eds): Brunnstrom's Clinical Kinesiology (ed. 5). Philadelphia, FA Davis, 1996, p 100, with permission.)*

been investigated.[66] The noncontractile components of a muscle also provide resistance to stretching and need to be considered when a muscle-stretching program is implemented.

Receptors that lie in joint capsules and ligaments may have an influence on muscle activity through their signals to the central nervous system. Swelling of the joint capsule and noxious stimuli, such as pinching of the capsule, will cause reflex inhibition of muscles.

Control of voluntary movements is not only accomplished via feedback systems such as Golgi tendon organs and the muscle spindles. **Feed-forward control** plays an important role in voluntary movement because it allows for anticipatory control of muscle actions. For example, in preparation for movements of the extremities, the abdominal muscles contract in order to stabilize the trunk.[67] The feed-forward control relies on sensory input from the visual and auditory systems in order to learn appropriate movement patterns as well as to anticipate the control of movement.

Example 3-6

Nociceptors and other receptors in and around the knee joint can have flexor excitatory and extensor inhibitory action. Even a small joint effusion that is undetectable by the naked eye can cause inhibition.[68–70]

The effects of the sensory receptors on muscle activity add an aspect of involuntary control of muscle function to the factors previously discussed. A review of the recent literature related to motor control or “movement science” is beyond the scope of this text, but some aspects of motor control will be presented in Chapter 13.

EFFECTS OF IMMOBILIZATION, INJURY, AND AGING

Immobilization

Immobilization affects both muscle structure and function. The effects of immobilization depend on *immobilization position (shortened or lengthened)*, *percentage of fiber types within the muscle*, and *length of the immobilization period.*

In Shortened Position

A muscle immobilized in a shortened position adapts to the new resting position through the following structural changes:

- Decrease in the number of sarcomeres with a compensatory increase in sarcomere length[71,72,73]
- Increase in the amount of perimysium[74]
- Thickening of endomysium[74]
- Collagen fibril orientation becoming more circumferential[75]
- Increase in ratio of connective tissue to muscle fiber tissue
- Loss of weight and muscle atrophy[72,76]

Changes in function that result from immobilization in the shortened position reflect the structural changes. The decrease in the number of sarcomeres, coupled with an increase in the length of sarcomeres, adjusts sarcomeres to a length at which the muscle is capable of developing maximal tension in the immobilized position. The loss of sarcomeres displaces the length-tension relationship of the muscle so that the maximum tension generated corresponds to the immobilized position. Therefore, the muscle is able to generate maximal tension in the shortened position. Although this altered capacity for developing tension may be beneficial while the muscle is immobilized in the shortened position, the muscle will not be able to function effectively at the joint it crosses immediately after cessation of the immobilization. The muscle that has adapted to its shortened state will resist lengthening passively, thus potentially limiting joint motion. Furthermore, the overall tension-generating capacity of the muscle is decreased and the increase in connective tissue in relation to muscle fiber tissue results in an increase in stiffness to passive stretch.

In Lengthened Position

Muscles immobilized in the lengthened position exhibit fewer structural and functional changes than do muscles immobilized in the shortened position. The primary structural changes are an increase in the number of sarcomeres, resulting in a decrease in sarcomere length at the lengthened position; increased endomyseal and perimyseal connective tissue; and muscle hypertrophy that may be followed by atrophy.[71,74,77] The primary functional changes in muscles immobilized in a lengthened rather than in a shortened position are an increase in maximum tension-generating capacity and displacement of the length-tension curve close to the longer immobilized position. Passive tension in the muscle approximates that of the muscle before immobilization.[72]

Prevention of the effects of immobilization in the shortened position may require only short periods of daily movement. With only 30 minutes of daily ROM activities out of the cast, the negative effects of immobilization in a shortened position were eliminated in animal models.[78] This beneficial effect of movement is not apparent when the frequency of movement is decreased to once per week over 3 weeks of immobilization.[79] If daily motion is not possible (e.g., due to fracture), then early and intensive re-mobilization after immobilization reverses the sarcomere number and connective tissue alterations that occur with immobilization.[80] In summary, a word of caution concerning the interpretation of the response of muscle to immobilization: the studies on sarcomere adaptation to immobilization have all been done with specific muscles in animals. Human skeletal muscle sarcomere numbers may be capable of adaptation similar to that of animals in situations of clinical bone lengthening to compensate for a short limb. When lengthened very slowly over several weeks in a case study, the vastus lateralis muscle adapted with an increase in number of sarcomeres.[81] Despite this one case study, it is not clear whether these changes are apparent in all human muscles and under what conditions.

Injury

Overuse

Overuse may cause injury to tendons, ligaments, bursae, nerves, cartilage, and muscle. The common etiology of these injuries is repetitive trauma that does not allow time for complete repair of the tissue. The additive effects of repetitive forces lead to microtrauma, which in turn triggers the inflammatory process and results in swelling. The tissue most commonly affected by overuse injuries is the musculotendinous unit.[82] Muscle and tendons can fatigue with repetitive submaximal loading, with rapidly applied loads, and when the active (contracting) musculotendinous unit is stretched by external forces.[83] Bursae may become inflamed with resultant effusion and thickening of the bursal wall as a result of repetitive trauma. Nerves can be subjected to compression injuries by muscle hypertrophy, decreased flexibility, and altered joint mechanics.[84]

Muscle Strain

Muscle strain injuries can occur from a single high-force contraction of the muscle while the muscle is lengthened by external forces (such as body weight). The muscle usually fails at the junction between the muscle and tendon.[21,85] Subsequently, there is localized bleeding and a significant acute inflammatory response, resulting in swelling, redness, and pain.[86]

CASE APPLICATION

Tissue Healing *case 3–7*

Because it is possible that the patient strained the plantar flexor muscles, there is probably some swelling and pain in the calf muscles. It would be best for Vik to rest the limb by decreasing his activities in order to allow the tissues to heal. As the tissues heal, he will need to begin a rehabilitation program to regain mobility and strength. He may not be able to return to strenuous activities until 4 to 8 weeks after the initial injury.

Eccentric Exercise-Induced Muscle Injury

Injuries to muscles may occur as a result of even a single bout of eccentric exercise.[87] After 30 to 40 minutes of eccentric exercise (walking downhill) or as few as 15 to 20 repetitions of high-load eccentric contractions, significant and sustained reductions in maximal voluntary contractions occur. Also, a loss of coordination, **delayed-onset muscle soreness (DOMS),** swelling, and a dramatic increase in muscle stiffness have been reported. The DOMS reaches a peak 2 to 4 days after exercise.[88–90] DOMS occurs in muscles performing eccentric exercise but not in muscles performing concentric exercise.[88] The search for a cause of DOMS is still under investigation. It is known to be related to the forces experienced by muscles and may be a result of mechanical strain in the muscle fibers or in their associated connective tissues.[89,92,93] Morphological evidence shows deformation of the Z disc (Z-disc streaming) and other focal lesions after eccentric activity that induces soreness.[94] Biomechanical and histochemical studies have demonstrated evidence for collagen breakdown and for other connective tissue changes.[88]

Aging

Fiber Number and Fiber Type Changes

As a person ages, skeletal muscle strength decreases as a result of **sarcopenia** (the loss of muscle mass). Sarcopenia occurs as result of a loss of muscle fibers and a decrease in the size of existing fibers. After the sixth decade of life, there is a loss of muscle fibers; some muscles (vastus lateralis) show a 25% to 30% loss of fibers in persons in their 70s and 80s.[95] It is commonly thought that there is a gradual decrease in the number and size of type II fibers, leaving the muscle with a relative increase in type I fibers. But more recent data using immunohistochemical methods of fiber typing (identifying the specific MHC content in the muscle) suggests that the proportion of type I and type II fibers remains relatively constant.[96] The maintenance of the proportion fiber types may occur to a greater degree in elderly people who stay active, whereas the elderly who are inactive may have a greater proportion of type I fibers.[96] There is also a decrease in the number of motor units, and the remaining motor units have a higher number of fibers per motor unit.[97,98]

Connective Tissue Changes

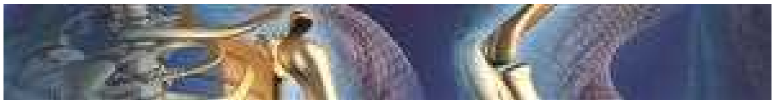
Aging also increases the amount of connective tissue within the extracellular matrix of the muscle (endomysium, perimysium, and epimysium) of the skeletal muscle. It is generally assumed that the increased connective tissue results in decreased ROM and increased muscle stiffness,[99,100] although there have been reports that muscle stiffness may not change or may decrease with aging.[101–103] All of these changes in the muscle result in decreased muscle strength and, more important, a loss in muscle power. This loss of muscle power, or the ability to contract the muscle with high force and high velocity, may be a cause of falls in the elderly.[104,105] Resistance exercise training in the elderly appears to have positive effects on aging muscles, causing an increase in the size of muscle fibers and an increase in strength and functional performance.[106–108] However, the response to resistance training is more limited in the elderly than in the young.[109]

SUMMARY

There are many factors that affect the function of the muscles. From the individual proteins and whole muscle architecture that determine the structure of the muscle to the neural and biomechanical relationships that determine performance of the muscle, the interrelationships between structure and function in muscles are complex and often indistinguishable. Muscles are more adaptable and, in many ways, more complex than the joints that they serve. Artificial joints have been designed and used to replace human joints, but it is as yet impossible to design a structure that can be used to replace a human muscle.

All skeletal muscles adhere to the general principles of structure and function that have been presented in this chapter. Muscles produce the forces that power an incredible array of movements. During human movements, muscles not only provide the force to move the limbs but also provide the force for stabilization. In the following chapters, the structure and function of specific muscles and the relationship between muscles and specific joints will be explored. The way in which muscles support the body in the erect standing posture and provide movement during walking will be examined in the last two chapters of this book.

STUDY QUESTIONS



1. What are the contractile and noncontractile elements of muscle?
2. What are the events that occur between the thick and thin filaments during isometric, concentric, and eccentric muscle contractions?
3. What is the function of titin in active muscle contraction and in passive muscle lengthening?
4. How is motor unit size related to the production of muscle force?
5. What is the fiber type distribution in the soleus muscle and how does this affect the functional characteristics of the soleus compared to the gastrocnemius muscle?
6. How is the function of the quadriceps similar to or different from the hamstrings muscles on the basis of the architectural characteristics of each muscle group?
7. What are the factors that could affect the development of active muscle tension? Suggest positions of the upper extremity in which each of the following muscles would not be able to develop maximal tension: biceps brachii, triceps brachii, and flexor digitorum profundus. What are the positions in which the same muscles would passively limit motion?
8. Which muscles are involved in lowering oneself into an armchair using one's arms? Is the muscle contraction eccentric or concentric? Explain your answer.
9. When testing strength using manual muscle testing, what exactly are we measuring? Beside changes in muscle length, why does muscle strength change at different points in the range of motion?
10. Diagram the changes in the MA of the biceps brachii muscle from full elbow extension to full flexion. How does the change in MA affect the function of the muscle?
11. What are the agonists, antagonists, and synergists in each of the following motions: abduction at the shoulder, flexion at the shoulder, and abduction at the hip?
12. Based on muscle architecture, what is the difference in function between the finger flexors and the finger extensors?
13. How does isokinetic exercise differ from other types of exercise, such as dynamic weight lifting and isometric exercise?
14. What are the effects of immobilization on muscles?
15. What adaptations occur in skeletal muscle with aging?

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Section 2

Axial Skeletal Joint Complexes

Chapter 4 **The Vertebral Column**
Chapter 5 **The Thorax and Chest Wall**
Chapter 6 **The Temporomandibular Joint**

Chapter 4

The Vertebral Column

Diane Dalton, PT, DPT, OCS

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INTRODUCTION

The vertebral column is an amazingly complex structure that must meet the seemingly contradictory demands of mobility and stability for the trunk and the extremities and of protection for the spinal cord. Although the pelvis is not considered part of the vertebral column, the pelvic attachment to the vertebral column through the sacroiliac joints will be included in this chapter because of the relationship of these joints and those of the lumbar region.

4-1 Patient Case

case

Our patient, Malik Johnson, is a 33-year-old male construction worker who for several months has been experiencing moderate to severe low back pain that radiates into his right lower extremity. He has pain with sitting, carrying, and all lifting activities, especially activities that involve lifting from a stooped position. He also has pain with upper extremity tasks such as hammering and using power tools. The pain is particularly severe when he first gets to work in the morning and when he performs any of these activities. He can relieve the pain somewhat if he lies down, but he has been able to tolerate work for only approximately four hours at a time. His history includes several episodes of low back pain that were severe but much shorter in duration, lasting for only a few days.

GENERAL STRUCTURE AND FUNCTION

Structure

The vertebral column resembles a curved rod, composed of 33 vertebrae and 23 intervertebral discs. The vertebral column is divided into the following five regions: cervical, thoracic, lumbar, sacral, and coccygeal (Fig. 4–1A). The vertebrae adhere to a common basic structural design but show regional variations in size and configuration that reflect the functional demands of a particular region. The vertebrae increase in size from the cervical to the lumbar regions and then decrease in size from the sacral to coccygeal regions. Twenty-four of the vertebrae in adults are distinct entities: Seven vertebrae are located in the cervical region, twelve in the thoracic region, and five in the lumbar region. Five of the remaining nine vertebrae are fused to form the sacrum, and the remaining four constitute the coccygeal vertebrae.

In the frontal plane, the vertebral column bisects the trunk when viewed from the posterior aspect (Fig. 4–1B). When viewed from the sagittal plane, the curves are evident (Fig. 4–1A). The curve of the vertebral column of a fetus exhibits one long curve that is convex posteriorly; secondary curves develop in infancy (Fig. 4–2A,B,C). However, in the column of an adult, four distinct anteroposterior curves are evident (Fig. 4–2D). The two curves (thoracic and sacral) that retain the original posterior convexity throughout life are called **primary curves,** whereas the two curves (cervical and lumbar) that show a reversal of the original posterior convexity are called **secondary curves.** Curves that have a posterior convexity (anterior concavity) are referred to as **kyphotic curves;** curves that have an anterior convexity (posterior concavity) are called **lordotic curves.** The secondary or lordotic curves develop as a result of the accommodation of the skeleton to the upright posture.

A curved vertebral column provides a significant advantage over a straight rod in that it is able to resist much higher compressive loads. According to Kapandji, a spinal column with the normal lumbar, thoracic, and cervical curves has a 10-fold ability to resist axial compression in comparison with a straight rod.[1]

The vertebral column functions as a closed chain with both the head and the ground. We can easily see how this occurs through contact of the feet to the ground, but we often forget the need for the head to remain in a somewhat stable position as we move to allow the sensory organs, particularly the eyes and ears, to be optimally positioned for function. Each of the many separate but interdependent components of the vertebral column is designed to contribute to the overall function of the total unit, as well as to perform specific tasks.

The first section of this chapter will cover the general components of the mobile segment, followed by regional variations and the sacroiliac joints. The second section of the chapter will cover the muscles of the vertebral column and pelvis.

The Mobile Segment

It is generally held that the smallest functional unit in the spine is the mobile segment; that is, any two adjacent vertebrae, the intervening intervertebral disc (if there is one), and all the soft tissues that secure them together.

A Typical Vertebra

The structure of a typical vertebra consists of two major parts: an anterior, cylindrically shaped vertebral body (Fig. 4–3A) and a posterior, irregularly shaped vertebral or neural arch (Fig. 4–3B). The vertebral body is designed to be the weight-bearing structure of the spinal column. It is suitably designed for this task, given its blocklike shape with generally flat superior and inferior surfaces. In order to minimize the weight of the vertebrae and allow dynamic load-bearing, the vertebral body is not a solid block of bone but a shell of cortical bone surrounding a cancellous cavity.[2] The cortical shell is reinforced by trabeculae in the cancellous bone, which help resist compressive forces.

The **neural arch** can be further divided into the pedicles and the posterior elements. The **pedicles** are the portion of the neural arch that lie anterior to the articular processes on either side and serve as the connection between the posterior elements and the vertebral bodies. The function of the pedicles is to transmit tension and bending forces from the posterior elements to the vertebral bodies. They are well designed for this function: they are short, stout pillars with thick walls. In general, the pedicles increase in size from the

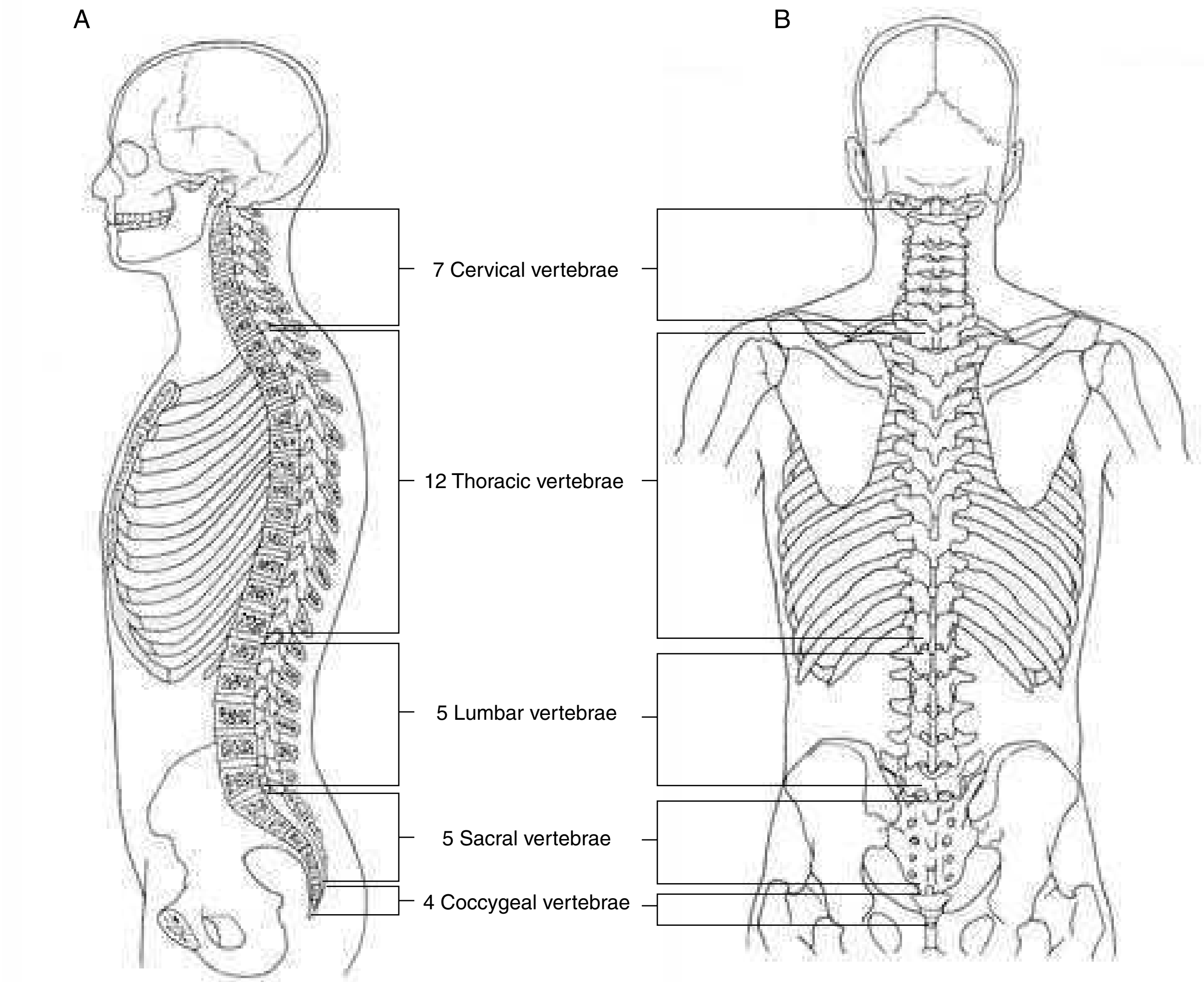


Figure 4–1 Five distinct regions of the vertebral column.

cervical to lumbar regions, which makes sense because greater forces are transmitted through the pedicles in the lumbar region.

The remaining posterior elements are the laminae, the articular processes, the spinous process, and the transverse processes (Fig. 4–4). The **laminae** are centrally placed and serve as origination points for the rest of the posterior elements. The laminae are thin, vertically oriented pieces of bone that serve as the "roof" to the neural arch, which protects the spinal cord. In addition, the laminae transmit forces from the posterior elements to the pedicles and, through them, to the vertebral body. This force transfer occurs through a region of the laminae called the **pars interarticularis.** The pars interarticularis, as its name suggests, is the portion of the laminae that is between the superior and inferior articular processes (Fig. 4–5). The pars interarticularis is subjected to bending forces that are transmitted from the vertically oriented lamina to the more horizontally oriented pedicles. The pars interarticularis is most developed in the lumbar spine, where the forces are the greatest in magnitude. Typically an increase in cortical bone occurs to accommodate the increased forces in this region. If the cortical bone is insufficient, the pars articularis is susceptible to stress fractures.[2]

Concept Cornerstone 4-1

Spondylolytic Spondylolisthesis

When stress fractures of the pars interarticularis occur bilaterally, the result may be **spondylolytic spondylolisthesis.**[1,2] In this condition, the posterior elements are completely separated from the remainder of the neural arch and the vertebral body. The vertebral body may then begin to slip forward on the vertebra below (Fig. 4–6). Although this can occur in any mobile segment, it occurs most frequently at the L5/S1 mobile segment because of the angulation of the segment and the anterior shear forces that exist there.

The altered location of the slipped vertebra changes its relationship to adjacent structures and creates excessive stress on supporting ligaments and joints. Overstretched ligaments may lead to a lack of stability or hypermobility of the mobile segment. Narrowing of the posterior joint space, which occurs with forward slippage of a vertebra, may cause compression on spinal nerves, the spinal cord, or the cauda equina. Pain may arise from excessive stress on any of the following pain-sensitive structures: anterior and posterior longitudinal ligaments, interspinous ligament, spinal nerves, dura mater, vertebral bodies, zygapophyseal joint capsules, synovial linings, or the muscles.

CASE APPLICATION

Spondylolisthesis as a Possible Cause of Pain

case 4–1

Spondylolytic spondylolisthesis could be a possible source of Malik's back pain because it can create excessive stress on the pain-sensitive structures listed previously. For Malik's pain in particular, the posterior ligaments, lumbar spinal nerves and dura mater, lumbar zygapophyseal joints, or the lumbar muscles that control anterior shear could all be producing his symptoms. The primary symptom of Malik's that is atypical of someone with spondylolytic spondylolisthesis, however, is pain in the sitting position. Flexion-based activities such as sitting are usually pain-free or much less painful in patients who have spondylolisthesis. This is due to the decreased anterior shear forces on the lumbar spine in the sitting position. Extension-based activities are typically more painful for those with spondylolytic spondylolisthesis.

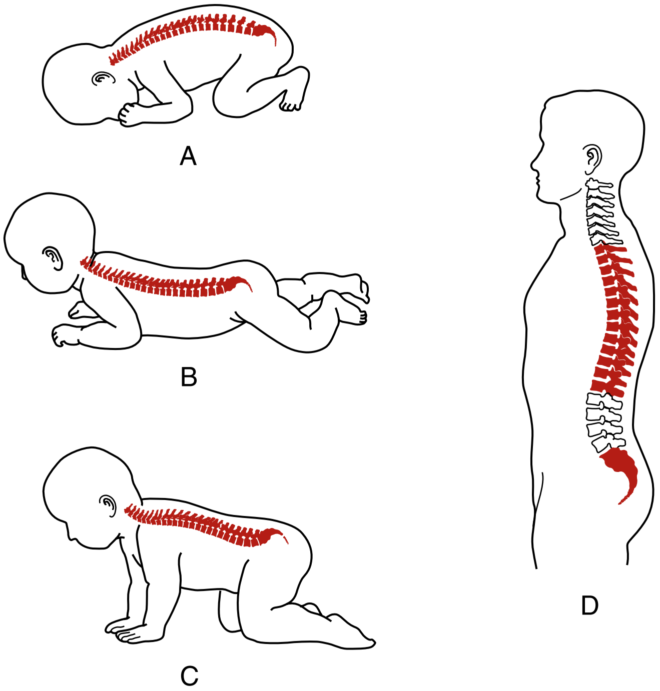


Figure 4–2 Primary and secondary curves. The colored areas represent the two primary curves. *(From McKinnis, LN: Fundamentals of Orthopedic Radiology, 1997, with permission from F.A. Davis Company.)*

In the typical vetebra, the **spinous processes** and two **transverse processes** are sites for muscle attachments and serve to increase the lever arm for the muscles of the vertebral column. The **articular processes** consist of two superior and two inferior facets for articulation with facets from the cranial and caudal vertebrae, respectively. In the sagittal plane, these articular processes form a supportive column,

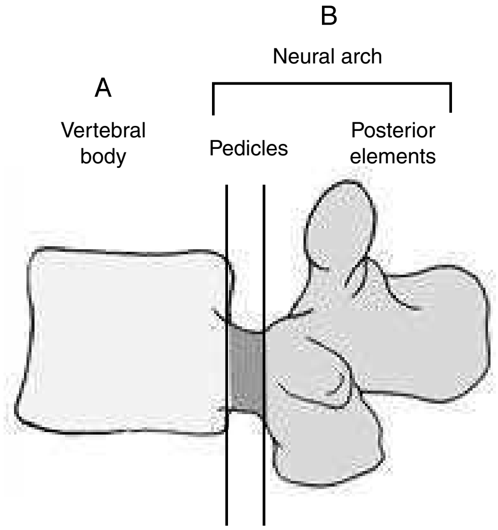


Figure 4–3 **A.** The anterior portion of a vertebra is called the **vertebral body. B.** The posterior portion of a vertebra is called the **vertebral** or **neural arch.** The neural arch is further divided into the **pedicles** and the **posterior elements.**

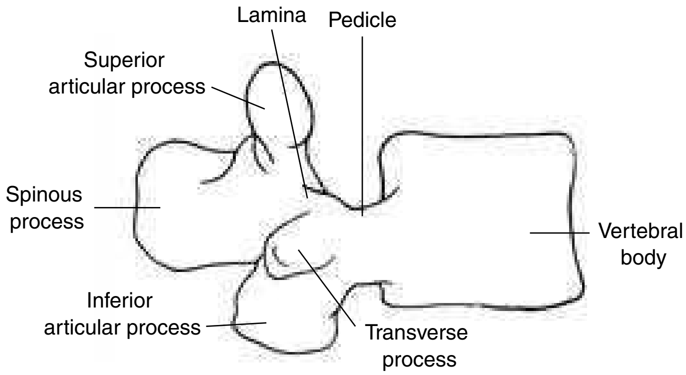


Figure 4–4 The posterior elements are the **laminae,** the **articular processes,** the **spinous process,** and the **transverse processes.**

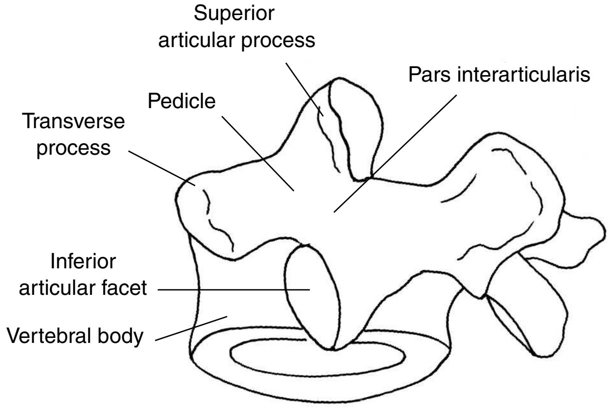


Figure 4–5 The **pars interarticularis** is the portion of the laminae between the articular processes.

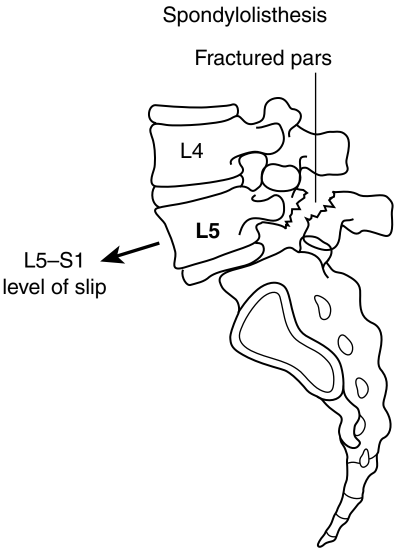


Figure 4–6 Spondylolisthesis. *(From McKinnis, LN: Fundamentals of Orthopedic Radiology, 1997, with permission from F.A. Davis Company.)*

frequently referred to as the **articular pillar.**[2] Table 4–1 summarizes the components of a typical vertebra and Table 4–2 summarizes the regional variations in vertebral structure.

The vertebrae are subjected to a wide variety of forces; however, they have a typical bony architecture that suggests a typical loading pattern.[3] Vertebral bone trabecular systems that develop in response to the stresses placed on the vertebral bodies and the neural arch are found within the spongy bone[4] (Fig. 4–7). The vertebrae have vertically oriented trabeculae with horizontal connections near the end plate and with denser bone areas near the pedicle bases.[3,5] The vertical systems within the vertebral bodies help support the body's weight and resist compression forces (Fig. 4–8). There are also fan-shaped trabeculae introduced into the vertebral body at the area of the pedicle in response to bending and shearing forces transmitted through this region.[3]

The Intervertebral Disc

The intervertebral disc has two principle functions: to separate two vertebral bodies, thereby increasing available motion, and to transmit load from one vertebral body to

Table 4–1 Components of a Typical Vertebra

	DESCRIPTION	FUNCTION
Body	Block of trabecular bone covered by a layer of cortical bone	To resist compressive loads
Arches		
Pedicle	Short, stout pillars with thick walls that connect the vertebral body to the posterior elements	To transmit the bending forces from the posterior elements to the vertebral body
Lamina	The vertical plate that constitutes the central portion of the arch posterior to the pedicles	To transmit the forces from the articular, transverse, and spinous processes to the pedicles
Transverse processes	Lateral projections of bone that originate from the laminae	Serve as muscle attachments and provide mechanical lever
Spinous process	Posterior projection of bone that originates from the central portion of the lamina, dividing it in two	Serves as muscle attachment and provides mechanical lever; may also serve as a bony block to motion
Vertebral foramen	Opening bordered by the posterior vertebral body and the neural arch	Combined with all segments, forms a passage and protection for the spinal cord

Table 4–2 Regional Variations in Vertebral Structure

PART	CERVICAL VERTEBRAE	THORACIC VERTEBRAE	LUMBAR VERTEBRAE
Body	The body is small, with a transverse diameter greater than its anteroposterior diameter. The anterior surface of the body is convex; the posterior surface is flat. The superior surface of the body is saddle-shaped because of the presence of uncinate processes on the lateral aspects of the superior surfaces.	The transverse and anteroposterior diameters of the bodies are equal. The anterior height is less than the posterior height. Two demifacets for articulation with the ribs are located on the posterolateral corners of the vertebral plateaus.	The body is massive, with a transverse diameter greater than the anteroposterior diameter and the height.

Continued

Table 4–2 Regional Variations in Vertebral Structure—cont'd

PART	CERVICAL VERTEBRAE	THORACIC VERTEBRAE	LUMBAR VERTEBRAE
Arches			
Pedicles	Project posterolaterally	Variable in shape and orientation	Short and thick
Laminae	Project posteromedially and are thin and slightly curved	Short, thick, and broad	Short and broad
Superior zygapophyseal facets	Face superiorly and medially	Thin and flat; face posteriorly, superiorly, and laterally	Vertical and concave; face posteromedially. Support mamillary processes on posterior borders.
Inferior zygapophyseal facets	Face anteriorly and laterally	Face anteriorly, inferiorly, and medially	Vertical, convex, and face anterolaterally
Transverse processes	Possess foramina for vertebral artery, vein, and venous plexus. Also have a gutter for the spinal nerve.	Large, with thickened ends. Possess paired oval facets for articulation with the ribs. Show a caudal decrease in length.	Long, slender, and extend horizontally. Support accessory processes on the posterior inferior surfaces of the root.
Spinous processes	Short, slender, and extend horizontally. Have bifid tips.	T1–T10 slope inferiorly. T11 and T12 have a triangular shape.	Broad, thick, and extend horizontally
Vertebral foramen	Large and roughly triangular	Small and circular	Triangular; larger than the thoracic but smaller than the cervical

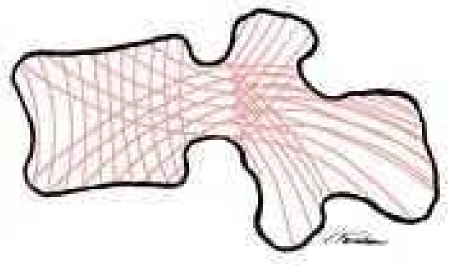


Figure 4–7 Schematic representation of the internal architecture of a vertebra. The various trabeculae are arranged along the lines of force transmission.

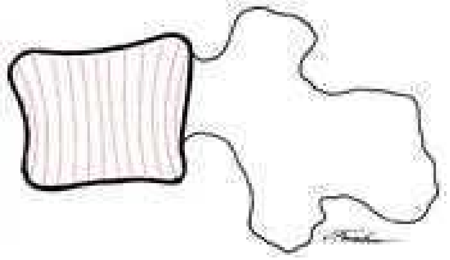
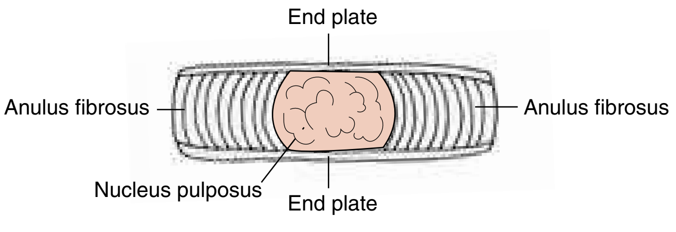


Figure 4–8 The vertical trabeculae of the vertebral bodies are arranged to resist compressive loading.

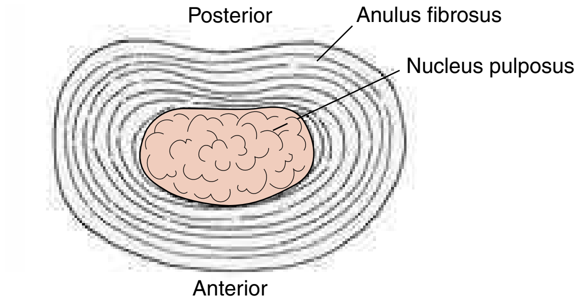
the next. Therefore, the size of the intervertebral disc is related to both the amount of motion and the magnitude of the loads that must be transmitted. The intervertebral discs, which make up about 20% to 33% of the length of the vertebral column, increase in size from the cervical to the lumbar regions.[6] The disc thickness varies from approximately 3 mm in the cervical region, where the weight-bearing loads are the lowest, to about 9 mm in the lumbar region, where the weight-bearing loads are the highest.[1] Although the discs are smallest in the cervical region and largest in the lumbar region, it is the ratio between disc thickness and vertebral body height that determines the available motion.[1] The greater the ratio, the greater the mobility. The ratio is greatest in the cervical region, followed by the lumbar region, and the ratio is smallest in the thoracic region. This reflects the greater functional needs for mobility in the cervical and lumbar regions and for stability in the thoracic region.

Most of what we know about the structure and function of the intervertebral discs has been gleaned from studies of the lumbar region. It was long thought that the discs of the cervical and thoracic regions had a structure similar to those of the lumbar region. It appears that this is not the case, particularly with regard to the intervertebral discs of the cervical region.[7–9] This section will describe the general structure and function of the intervertebral disc. Region-specific variations will be described in the regional structure sections of the chapter.

The intervertebral discs are composed of three parts: (1) the **nucleus pulposus,** (2) the **anulus fibrosus,** and (3) the **vertebral end plate** (Fig. 4–9). The nucleus pulposus is the gelatinous mass found in the center, the anulus fibrosus is the fibrous outer ring, and the vertebral end plate is the cartilaginous layer covering the superior and inferior surfaces of the disc, separating it from the cancellous bone of the vertebral bodies above and below. All three structures are composed of water, collagen, and proteoglycans; however, the relative proportions of each vary. Fluid and proteoglycan concentrations are highest in the nucleus and lowest in the outer anulus fibrosus and the outer vertebral end plate (closest to the vertebral body). Conversely, collagen concentrations are highest in the



Coronal section



Transverse section

Figure 4–9 A schematic representation of a lumbar intervertebral disc showing the **nucleus pulposus,** the **anulus fibrosus,** and the **vertebral end plate.**

vertebral end plate and outer anulus and lowest in the nucleus pulposus. Although the nucleus pulposus is clearly distinct from the anulus fibrosus in the center and the anulus fibrosus is clearly distinct from the nucleus in the outer rings, there is no clear boundary separating the two structures where they merge. They are distinct structures only where they are furthest apart.

Nucleus Pulposus

The nucleus pulposus is 70% to 90% water, depending on age and time of day.[2] Proteoglycans make up approximately 65% of the dry weight and have the ability to attract water molecules because of the presence of glycosaminoglycans; hence the high water content.[2] Collagen fibers contribute 15% to 20% of the dry weight, and the remainder of the dry weight is composed of collagen, chondrocytes elastin fibers, proteins, and proteolytic enzymes.[2] The nucleus pulposus has both type I and type II collagen; however, type II predominates because of its ability to resist compressive loads. In fact, very little, if any, type I collagen is present in the center of the nucleus pulposus.[2,10] The nucleus pulposus has been frequently likened to a water balloon. When compressed, it deforms, and the increased pressure stretches the walls of the nucleus pulposus in all directions (Fig. 4–10).

Anulus Fibrosus

In general, the anulus fibrosus is 60% to 70% water, like the nucleus pulposus, depending on age and time of day. Collagen fibers make up 50% to 60% of the dry weight, with proteoglycans contributing only 20% of the dry weight.[2] Clearly, the relative proportions of these components are different from the proportions in the nucleus pulposus, reflecting the difference in structure. The remainder of the dry weight is made up of approximately 10% elastin fibers and cells such as fibroblasts and chondrocytes.[2] Again, type I and type II collagen are present; however, type I collagen predominates in the anulus fibrosus, particularly in the outer portions.[2,10] This makeup reflects the need for the anulus fibrosus, rather than the nucleus pulposus, to resist greater proportions of tensile forces. The anulus fibers are attached by Sharpey fibers to the cartilaginous end plates on the inferior and superior vertebral plateaus of adjacent vertebrae and to the epiphyseal ring region.

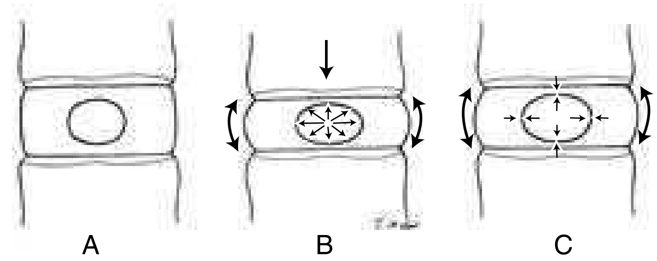


Figure 4–10 Compression of an intervertebral disc. **A.** In this schematic representation of a disc, the nucleus pulposus is shown as a round ball in the middle of the anulus fibrosus. **B.** Under compressive loading, pressure is exerted in all directions as the nucleus pulposus attempts to expand. Tension in the anulus fibrosus rises as a result of the nuclear pressure. **C.** A force equal in magnitude but opposite in direction is exerted by the anulus fibrosus on the nucleus pulposus, which restrains radial expansion of the nucleus pulposus and establishes equilibrium. The nuclear pressure is transmitted by the anulus fibrosus to the end plates.

Vertebral End Plates

The vertebral end plates are layers of cartilage 0.6 to 1 mm thick that cover the region of the vertebral bodies encircled by the ring apophysis on both the superior and inferior surfaces.[2] They cover the entire nucleus pulposus but not the entire anulus fibrosus. The vertebral end plate is strongly attached to the anulus fibrosus but only weakly attached to the vertebral body, which is why it is considered to be a component of the disc rather than of the vertebral body.[2,10] The vertebral end plates consist of proteoglycans, collagen, and water, as does the rest of the disc. In addition, there are cartilage cells aligned along the collagen. As in the other regions of the disc, there is a higher proportion of water and proteoglycans closest to the nucleus pulposus and a higher proportion of collagen closest to the anulus fibrosus and the subchondral bone of the vertebral body. The cartilage of the vertebral end plates is both hyaline cartilage and fibrocartilage. Hyaline cartilage is present closest to the vertebral body and is found mainly in young discs. Fibrocartilage is present closest to the nucleus pulposus and with increasing age becomes the major component of the vertebral end plate, with little or no hyaline cartilage remaining, reflecting the need to tolerate high compressive forces.

Innervation and Nutrition

The intervertebral discs are innervated in the outer one-third to one-half of the fibers of the anulus fibrosus.[2] In the cervical and lumbar regions, the intervertebral discs are innervated by branches from the vertebral and sinuvertebral nerves. The sinuvertebral nerve also innervates the peridiscal connective tissue and specific ligaments associated with the vertebral column.[2]

The intervertebral discs do not receive blood supply from any major arterial branches. The metaphyseal arteries form a dense capillary plexus in the base of the end plate cartilage and the subchondral bone deep to the end plate, and small branches from these metaphyseal arteries supply the outer surface of the anulus fibrosus.[2,11] The remainder of the disc receives its nutrition via diffusion.

Articulations

Two main types of articulations are found in the vertebral column: **cartilaginous joints of the symphysis type** between the vertebral bodies, including the interposed discs, and **diarthrodial,** or **synovial,** joints between the zygapophyseal facets located on the superior articular processes of one vertebra and the zygapophyseal facets on the inferior articular processes of an adjacent vertebra above. The joints between the vertebral bodies are referred to as the **interbody joints.** The joints between the zygapophyseal facets are called the **zygapophyseal** (**apophyseal** or **facet**) **joints** (Fig. 4–11). Synovial joints also are present where the vertebral column articulates with the ribs (see Chapter 5), with the skull, and with the pelvis at the sacroiliac joints.

Interbody Joints

Available movements at the interbody joints include **sliding, distraction and compression,** and **rotation** (also called tilt or rocking in the spine) (Fig. 4–12). Sliding motions can occur in the following directions: anterior to posterior, medial to lateral, and torsional. Tilt motions can occur in anterior to posterior and in lateral directions. These motions, together with distraction and compression, constitute six degrees of freedom.[6] The amounts of each of these motions are small and vary by region according to structural differences in the discs and the vertebral bodies, as well as in the ligamentous supports. In addition, the zygapophyseal joints influence the total available motion of the interbody joints.

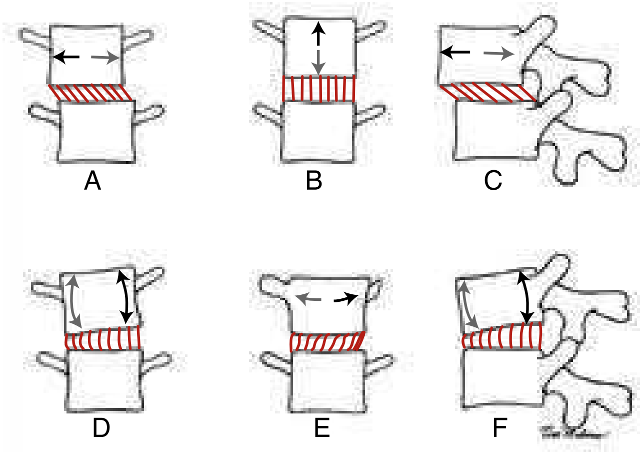


Figure 4–12 Translations and rotations of one vertebra in relation to an adjacent vertebra. **A.** Side-to-side translation (gliding) occurs in the frontal plane. **B.** Superior and inferior translation (axial distraction and compression) occur vertically. **C.** Anteroposterior translation occurs in the sagittal plane. **D.** Side-to-side rotation (tilting) in a frontal plane occurs around an anteroposterior axis. **E.** Rotation occurs in the transverse plane around a vertical axis. **F.** Anteroposterior rotation (tilting) occurs in the sagittal plane around a frontal axis.

Zygapophyseal Articulations

The zygapophyseal joints are composed of the articulations between the right and left superior articulating facets of a vertebra and the right and left inferior facets of the adjacent cranial vertebra. The zygapophyseal joints are diarthrodial joints and have regional variations in structure. Intra-articular accessory joint structures have been identified in the zygapophyseal joints.[2,12–14] These

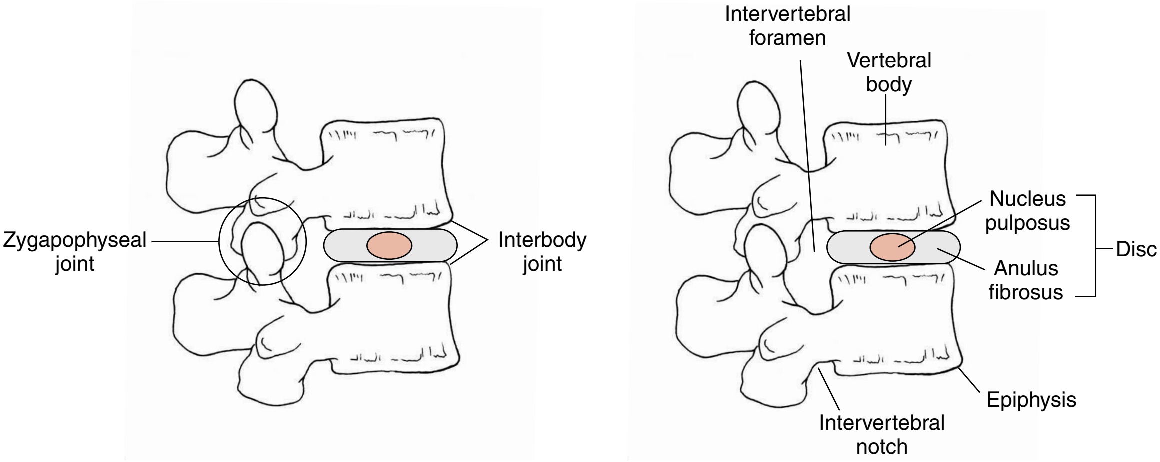


Figure 4–11 Interbody and zygapophyseal joints.

accessory structures appear to be of several types, but most are classified as either adipose tissue pads or fibroadipose meniscoids.[2] The structures are most likely involved in protecting articular surfaces that are exposed during flexion and extension of the vertebral column.

Ligaments and Joint Capsules

The ligamentous system of the vertebral column is extensive and exhibits considerable regional variability. Six main ligaments are associated with the intervertebral and zygapophyseal joints. They are the anterior and posterior longitudinal ligaments, the ligamentum flavum, and the interspinous, supraspinous, and intertransverse ligaments (Figs. 4–13 and 4–14).

Anterior and Posterior Longitudinal Ligaments

The anterior longitudinal ligament (ALL) and posterior longitudinal ligament (PLL) are associated with the interbody joints. The **anterior longitudinal ligament** runs along the anterior and lateral surfaces of the vertebral bodies from the sacrum to the second cervical vertebra. Extensions of the ligament from C2 to the occiput are called the anterior atlanto-occipital and anterior atlantoaxial ligaments. The anterior longitudinal ligament has at least two layers that are made up of thick bundles of collagen fibers.[15,16] The fibers in the superficial layer are long and bridge several vertebrae, whereas the deep fibers are short and run between single pairs of vertebrae. The deep fibers blend with the fibers of the anulus fibrosus and reinforce the anterolateral portion of the intervertebral discs and the anterior interbody joints. The ligament is well developed in the lordotic sections (cervical and lumbar) of the vertebral column but has little substance in the region of thoracic kyphosis. The anterior longitudinal ligament increases in thickness and width from the lower thoracic vertebrae to L5/S1.[15] The tensile strength of the ligament is greatest at the high cervical, lower thoracic, and lumbar regions, with the greatest strength being in the lumbar region.[17] It is reported to be twice as strong as the posterior longitudinal ligament.[17] The ligament is compressed in flexion (Fig. 4–15A) and stretched in extension (Fig. 4–15B). It may become slack in the neutral position of the spine when the normal height of the discs is reduced, as might occur when the nucleus pulposus is degenerated.[18]

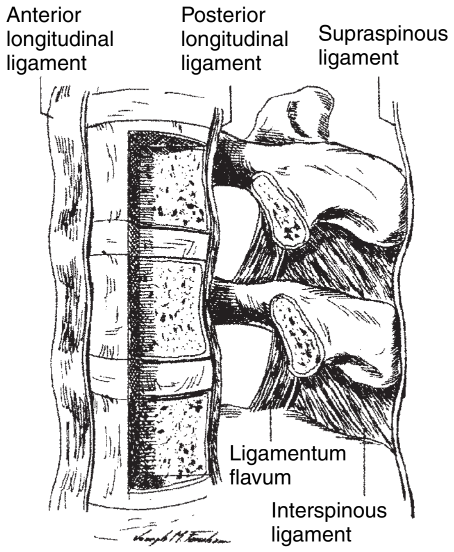


Figure 4–13 The anterior and posterior longitudinal ligaments are located on the anterior and posterior aspects of the vertebral body, respectively. The ligament flavum runs from lamina to lamina on the posterior aspect of the vertebral canal. Portions of the lamellae have been removed to show the orientation of the collagen fibers.

The **posterior longitudinal ligament** runs on the posterior aspect of the vertebral bodies from C2 to the sacrum and forms the ventral surface of the vertebral canal. It also consists of at least two layers: a superficial and a deep layer. In the superficial layer, the fibers span several mobile segments. In the deep layer, the fibers extend only to adjacent vertebrae, interlacing with the outer layer of the anulus fibrosus and attaching to the margins of the vertebral end plates in a manner that varies from segment to segment.[15] Superiorly, the ligament becomes the tectorial membrane from C2 to the occiput. In the lumbar region, the ligament narrows to a thin ribbon that provides little support for the interbody joints. The posterior longitudinal ligament's resistance to axial tension in the lumbar area is only one-sixth of that of the anterior longitudinal ligament.[17] The posterior longitudinal ligament is stretched in flexion (Fig. 4–16A) and slack in extension (Fig. 4–16B).

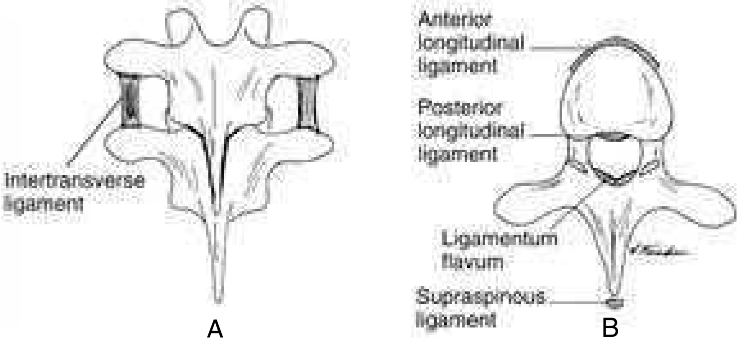


Figure 4–14 **A.** The intertransverse ligament connects the transverse processes. **B.** The relative positions of the other ligaments are shown in a superior view of the vertebra.

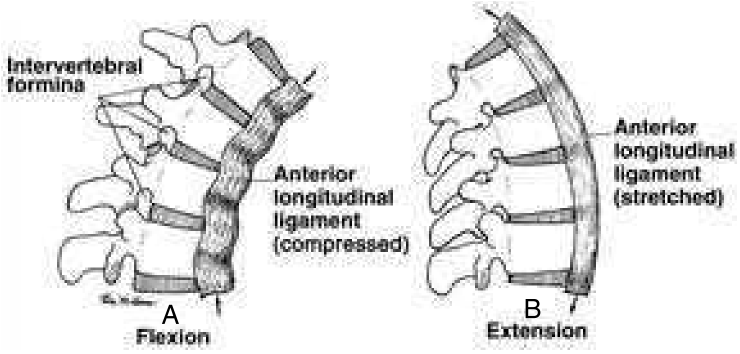


Figure 4–15 Anterior longitudinal ligament (ALL). **A.** The ALL is slack and may be compressed in forward flexion of the vertebral column. **B.** The ALL is stretched in extension of the vertebral column.

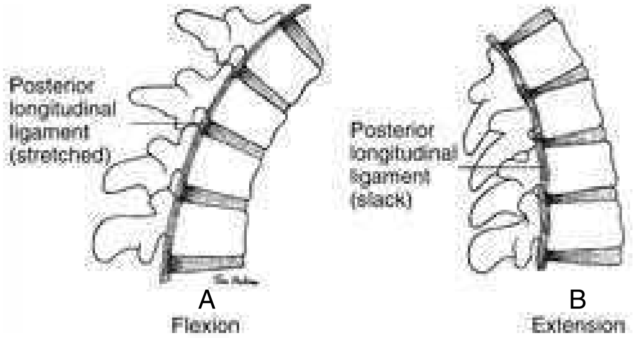


Figure 4–16 Posterior longitudinal ligament (PLL). **A.** The PLL is stretched during forward flexion of the vertebral column. **B.** The ligament is slack and may be compressed during extension.

CASE APPLICATION

Potential Role of the PLL in Low Back Injury

case 4–2

The narrow posterior longitudinal ligament in the lumbar region does not provide much support for the intervertebral discs, which is one of the factors contributing to the relatively high incidence of disc herniations in a posterolateral direction in the lumbar spine. A posterior disc herniation could be one of the causes of Malik's pain, especially because he has increased pain in a flexed position, which produces a large amount of stress on the area of the posterior longitudinal ligament. In addition, he has complaints of pain into his lower extremity, which may be a result of nerve root compression due to disc herniation.

Ligamentum Flavum

The **ligamentum flavum** is a thick, elastic ligament that connects lamina to lamina from C2 to the sacrum and forms the smooth posterior surface of the vertebral canal.[19] Some fibers extend laterally to cover the articular capsules of the zygapophyseal joints.[16] From C2 to the occiput, this ligament continues as the posterior atlanto-occipital and atlantoaxial membranes. The ligamentum flavum is strongest in the lower thoracic region and weakest in the midcervical region.[17] Although the highest strain in this ligament occurs during flexion when the ligament is stretched,[2,17] this ligament is under constant tension even when the spine is in a neutral position, because of its elastic nature.[16,20] This highly elastic nature serves two purposes. First, it creates a continuous compressive force on the discs, which causes the intradiscal pressure to remain high. The raised pressure in the discs makes the discs stiffer and thus better able to provide support for the spine in the neutral position.[21] Second, a highly elastic ligament in this location is advantageous because the ligament will not buckle on itself during movement. If the ligament did buckle on itself, it would compress the spinal cord in the vertebral canal, especially with any movement into flexion.

Interspinous Ligaments

The **interspinous ligament** connects spinous processes of adjacent vertebra. It is a fibrous sheet consisting of type I collagen, proteoglycans, and profuse elastin fibers.[22] The interspinous ligament, along with the supraspinous ligament, is the first to be damaged with excessive flexion.[23] The interspinous ligament is innervated by medial branches of the dorsal rami and is thought to be a possible source of low back pain.[24] The interspinous ligament has been found to contribute to lumbar spine stability and to degenerate with aging.[24]

Continuing Exploration 4-1:

Interspinous Ligament

The orientation of the fibers of the interspinous ligament has been subject to debate. Some authors have described the fibers as running predominantly parallel to the spinous processes, whereas others describe an oblique fiber orientation as well. Of those that describe an oblique orientation, the direction varies from anterior to posterior and from posterior to anterior.[22,24,25] The function of the ligament is also a subject of debate; however, most authors agree that it resists flexion. It may also resist end-range extension and posterior shear of the superior vertebra on the inferior one.[22,24,26] McGill suggests that the ligament also produces anterior shear during full flexion and that this should be considered in exercise prescription.[26] For example, individuals with pathologies such as spondylolytic spondylolisthesis are often prescribed full flexion exercises, which, McGill suggests, may in fact be contraindicated because of the increased anterior shear forces.[26]

Supraspinous Ligament

The **supraspinous ligament** is a strong, cordlike structure that connects the tips of the spinous processes from the seventh cervical vertebra to L3 or L4.[2,27] The fibers of the ligament become indistinct in the lumbar area, where they merge with the thoracolumbar fascia and insertions of the lumbar muscles. In the cervical region, the ligament becomes the **ligamentum nuchae.** The supraspinous ligament, like the interspinous ligament, is stretched in flexion, and its fibers resist separation of the spinous processes during forward flexion. During hyperflexion, the supraspinous ligament and the interspinous ligament are the first to fail.[28] The supraspinous ligament contains mechanoreceptors, and deformation of the ligament appears to play a role in the recruitment of spinal stabilizers such as the multifidus muscles.[29]

Intertransverse Ligaments

The structure of the **paired intertransverse ligaments** is extremely variable. In general, the ligaments pass between the transverse processes and attach to the deep muscles of the back. In the cervical region, only a few fibers of the ligaments are found. In the thoracic region, the ligaments consist of a few barely discernible fibers that blend with adjacent muscles. In the lumbar region, the ligaments consist of broad sheets of connective tissue that resemble a membrane. The membranous fibers of the ligament form part of the thoracolumbar fascia. The ligaments are alternately stretched and compressed during lateral bending. The ligaments on the right side are stretched and offer resistance during lateral bending to the left, whereas the ligaments on the left side are slack and compressed during this motion. Conversely, the ligaments on the left side are stretched during lateral bending to the right and offer resistance to this motion.

Zygapophyseal Joint Capsules

The **zygapophyseal joint capsules** assist the ligaments in providing limitation to motion and stability for the vertebral column. The roles of the joint capsules also vary by region. In the cervical spine, the facet joint capsules, although lax, provide the primary soft tissue restraint to axial rotation and lateral bending, but they provide little restraint to flexion and extension.[30] The zygapophyseal joint capsules of the lumbar spine, in addition to the anular fibers, also provide primary restraint to axial rotation,[31,32] but those of the thoracic spine do not.

The capsules are strongest in the thoracolumbar region and at the cervicothoracic junction[17] sites where the spinal configuration changes from a kyphotic to lordotic curve and from a lordotic to kyphotic curve, respectively, and the potential exists for excessive stress in these areas. The joint capsules, like the supraspinous and interspinous ligaments, are vulnerable to hyperflexion, especially in the lumbar region. It has been suggested that the joint capsules in the lumbar region provide more restraint to forward flexion than any of the posterior ligaments because they fail after the supraspinous and interspinous ligaments when the spine is hyperflexed.[33] Table 4–3 provides a summary of the ligaments and their functions.

Table 4–3 Major Ligaments of the Vertebral Column

LIGAMENTS	FUNCTION	REGION
Anulus fibrosus (outer fibers)	Resists distraction, translation, and rotation of vertebral bodies	Cervical, thoracic, and lumbar
Anterior longitudinal ligament	Limits extension and reinforces anterolateral portion of anulus fibrosus and anterior aspect of intervertebral joints	C2 to sacrum, but well developed in cervical, lower thoracic, and lumbar regions
Anterior atlantoaxial (continuation of the anterior longitudinal ligament)	Limits extension	C2 to the occipital bone
Posterior longitudinal ligament	Limits forward flexion and reinforces posterior portion of the anulus fibrosus	Axis (C2) to sacrum. Broad in the cervical and thoracic regions and narrow in the lumbar region.
Tectorial membrane (continuation of the posterior longitudinal ligament)	Limits forward flexion	Axis (C2) to occipital bone
Ligamentum flavum	Limits forward flexion, particularly in the lumbar area, where it resists separation of the laminae	Axis (C2) to sacrum. Thin, broad, and long in cervical and thoracic regions and thickest in the lumbar region.
Posterior atlantoaxial (continuation of the ligamentum flavum)	Limits flexion	Atlas (C1) and axis (C2)
Supraspinous ligaments	Limit forward flexion	Thoracic and lumbar (C7–L3 or L4). Weak in lumbar region.
Ligamentum nuchae	Limits forward flexion	Cervical region (occipital protuberance to C7)
Interspinous ligaments	Limit forward flexion	Primarily in lumbar region, where they are well developed

Continued

Table 4–3 **Major Ligaments of the Vertebral Column—cont'd**

LIGAMENTS	FUNCTION	REGION
Intertransverse ligaments	Limit contralateral lateral flexion	Primarily in lumbar region
Alar ligaments	Limit rotation of the head to the same side and lateral flexion to the opposite side	Atlas (C1 and C2)
Iliolumbar ligament	Resists anterior sliding of L5 and S1	Lower lumbar region
Zygapophyseal joint capsules	Resist forward flexion and axial rotation	Strongest at cervicothoracic junction and in the thoracolumbar region

Function

Kinematics

The motions available to the column as a whole are flexion and extension, lateral flexion, and rotation. These motions appear to occur independently of each other; however, at the level of the individual motion segment, these motions are often coupled. **Coupling** is defined as the consistent association of one motion about an axis with another motion around a different axis. The most predominant motions that exhibit coupled behaviors are lateral flexion and rotation. It appears that pure lateral flexion and pure rotation do not occur in any region of the spine. In order for either motion to occur, at least some of the other must occur as well.[6,34]

Coupling patterns, as well as the types and amounts of motion that are available, are complex, differ from region to region, and depend on the spinal posture and curves; the orientation of the articulating facets; the fluidity, elasticity, and thickness of the intervertebral discs; and the extensibility of the muscles, ligaments, and joint capsules.[34,35] It also appears that coupled motions vary widely among individuals and that there may be fewer patterns of coupled motions than was once believed.[36]

Motions at the interbody and zygapophyseal joints are interdependent. The *amount* of motion available is determined primarily by the size of the discs, whereas the *direction* of the motion is determined primarily by the orientation of the facets.

The intervertebral discs increase movement between two adjacent vertebrae. If the vertebrae were to lay flat against each other, the movement between them would be limited to translation alone.[2] The vertebrae are also allowed to rock or tilt on each other because the soft, deformable disc is between them. This arrangement adds tremendous range of motion (Fig. 4–17). The fibers of the anulus fibrosus behave as a ligamentous structure and act as restraints to motion.

The motions of flexion and extension occur as a result of the tilting and gliding of a superior vertebra over the inferior vertebra. As the superior vertebra moves through a range of motion, it follows a series of different arcs, each of which has a different instantaneous axis of rotation.[37,38] The nucleus pulposus acts like a pivot but is able to undergo greater distortion than a ball is because it behaves as a fluid.

Regardless of the magnitude of motion created by the ratio of disc height to width, a gliding motion occurs at the interbody and zygapophyseal joints as the vertebral body tilts (rotates) over the disc at the interbody joint. The orientation of the zygapophyseal facet surfaces, which varies from region to region, determines the direction of the tilting and gliding within a particular region. If the superior and inferior zygapophyseal facet surfaces of

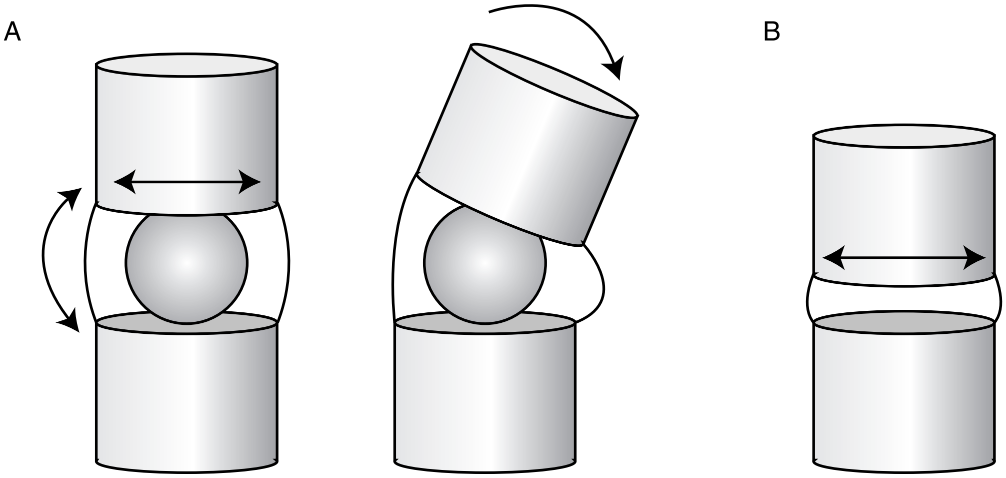


Figure 4–17 **A.** The addition of an intervertebral disc allows the vertebra to tilt, which dramatically increases ROM at the interbody joint. **B.** Without an intervertebral disc, only translatory motions could occur.

three adjacent vertebrae lie in the sagittal plane, the motions of flexion and extension are facilitated (Fig. 4–18A). On the other hand, if the zygapophyseal facet surfaces are placed in the frontal plane, the predominant motion that is allowed is lateral flexion (Fig. 4–18B).

Flexion

In vertebral flexion, anterior tilting and gliding of the superior vertebra occur and cause widening of the intervertebral foramen[39] and separation of the spinous processes (Fig. 4–19A). Although the amount of tilting depends partly on the size of the discs, tension in the supraspinous and interspinous ligaments resists separation of the spinous processes and thus limits the extent of flexion. Passive tension in the zygapophyseal joint capsules, ligamentum flavum, posterior longitudinal ligament, posterior anulus fibrosus, and the back extensors also imposes controls on excessive flexion. With movement into flexion, the anterior portion of the anulus fibrosus is compressed and bulges anteriorly, while the posterior portion is stretched and resists separation of the vertebral bodies.

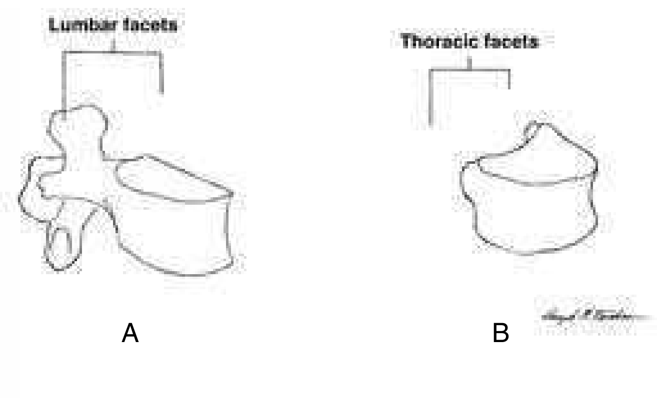


Figure 4–18 **A.** Sagittal plane orientation of the lumbar zygapophyseal facets favors the motions of flexion and extension. **B.** Frontal plane orientation of the thoracic zygapophyseal facets favors lateral flexion.

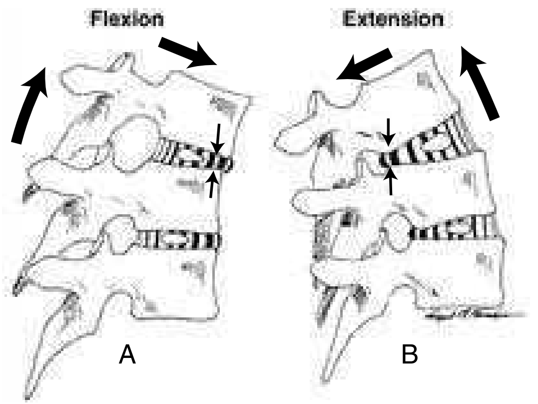


Figure 4–19 **A.** The superior vertebra tilts and glides anteriorly over the adjacent vertebra below during flexion. The anterior tilting and gliding cause compression and bulging of the anterior anulus fibrosus and stretching of the posterior anulus fibrosus. **B.** In extension, the superior vertebra tilts and glides posteriorly over the vertebra below. The anterior anulus fibers are stretched, and the posterior portion of the disc bulges posteriorly.

Extension

In extension, posterior tilting and gliding of the superior vertebra occur and cause narrowing of the intervertebral foramen,[39] and the spinous processes move closer together (Fig. 4–19B). The amount of motion available in extension, in addition to being limited by the size of the discs, is limited by bony contact of the spinous processes and passive tension in the zygapophyseal joint capsules, anterior fibers of the anulus fibrosus, anterior trunk muscles, and the anterior longitudinal ligament. In general, there are many more ligaments that limit flexion than ligaments that limit extension. The only ligament that limits extension is the anterior longitudinal ligament. This is likely the reason that this ligament is so strong compared to the posterior ligaments. The numerous checks to flexion follow the pattern of ligamentous checks to motion where bony limits are minimal. Fewer ligamentous checks to extension are necessary, given the presence of numerous bony checks.

Lateral Flexion

In lateral flexion, the superior vertebra laterally tilts, rotates, and translates over the adjacent vertebra below (Fig. 4–20). The intervertebral foramen is widened on the side contralateral to the lateral flexion and narrowed on the ipsilateral side.[39] The anulus fibrosus is compressed on the concavity of the curve and stretched on the convexity of the curve. Passive tension in the anulus fibers, intertransverse ligaments, and anterior and posterior trunk muscles on the convexity of the curve limits lateral flexion. The direction of rotation that accompanies lateral flexion differs slightly from region to region because of the orientation of the facets.

Rotation

Rotation is available in each spinal region, but due to the drastically different shapes of the zygapophyseal joints, the kinematics vary greatly by region and therefore will be discussed by region.

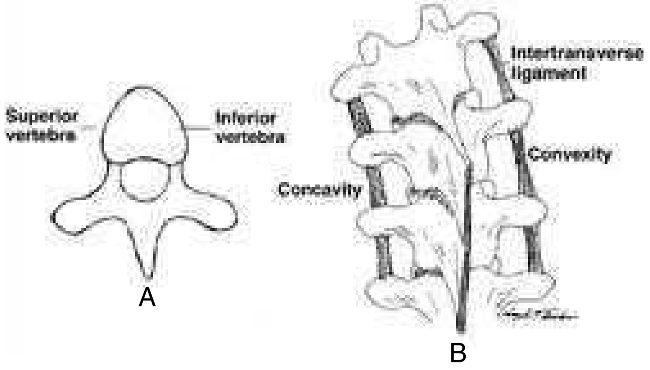


Figure 4–20 **A.** The superior vertebra tilts laterally and rotates over the adjacent vertebra below during lateral flexion. **B.** Lateral flexion and rotation of the vertebra are limited by tension in the intertransverse ligament on the convexity of the curve.

Kinetics

The vertebral column is subjected to axial compression, tension, bending, torsion, and shear stress, not only during normal functional activities but also at rest.[40] The column's ability to resist these loads varies among spinal regions and depends on the type, duration, and rate of loading; the person's age and posture; the condition and properties of the various structural elements (vertebral bodies, joints, discs, muscles, joint capsules, and ligaments); and the integrity of the nervous system.[41]

Axial Compression

Axial compression (force acting through the long axis of the spine at right angles to the discs) occurs as a result of the force of gravity, ground reaction forces, and forces produced by the ligaments and muscular contractions. The discs and vertebral bodies resist most of the compressive force, but the neural arches and zygapophyseal joints share some of the load in certain postures and during specific motions. The compressive load is transmitted from the superior end plate to the inferior end plate through the trabecular bone of the vertebral body and the cortical shell. The cancellous body contributes 25% to 55% of the strength of a lumbar vertebra before the age of 40, and the cortical bone carries the remainder. After age 40, the cortical bone carries a greater proportion of the load, since the trabecular bone's compressive strength and stiffness decrease with decreasing bone density.[42] Depending on the posture and region of the spine, the zygapophyseal joints carry from 0% to 33% of the compression load. The spinous processes also may share some of the load when the spine is in hyperextension.

The nucleus pulposus acts as a ball of fluid that can be deformed by a compression force. The pressure created in the nucleus pulposus is actually greater than the force of the applied load.[43] When a weight is applied to the nucleus pulposus from above, the nucleus pulposus exhibits a swelling pressure and tries to expand outward toward the anulus fibrosus and the end plates (see Fig. 4–10). As the nucleus attempts to distribute the pressure in all directions, stress is created in the anulus fibrosus, and central compressive loading occurs on the vertebral end plates. The forces of the nucleus pulposus on the anulus fibrosus and of the anulus fibrosus on the nucleus pulposus form an interaction pair. Normally, the anulus fibrosus and the end plates are able to provide sufficient resistance to the swelling pressure in the nucleus pulposus to reach and maintain a state of equilibrium. The pressure exerted on the end plates is transmitted to the superior and inferior vertebral bodies. The discs and trabecular bone are able to undergo a greater amount of deformation without failure than are the cartilaginous end plates or cortical bone when subjected to axial compression. The end plates are able to undergo the least amount of deformation and therefore will be the first to fail (fracture) under high compressive loading. The discs will be the last to fail (rupture).

The intervertebral discs, like all viscoelastic materials, exhibit **creep.** This phenomenon produces typical diurnal changes in disc composition and function. When the intervertebral discs are subjected to a constant load, they exhibit creep. Under sustained compressive loading such as that incurred in the upright posture, the rise in the swelling pressure causes fluid to be expressed from the nucleus pulposus and the anulus fibrosus. The amount of fluid expressed from the disc depends both on the size of the load and the duration of its application. The expressed fluid is absorbed through microscopic pores in the cartilaginous end plate. When the compressive forces on the discs are decreased, the disc imbibes fluid back from the vertebral body.[44] The recovery of fluid that returns the disc to its original state explains why a person getting up from bed is taller in the morning than in the evening. The average variation in height during the day has been demonstrated to be 19 mm, with a loss of approximately 1.5 mm (almost 20%) in height from each of the lumbar intervertebral discs.[45–47] Running is a form of dynamic loading that decreases disc height more rapidly than static loading. The height of the vertebral column is a widely used indicator of cumulative compression. In a study involving 31 men, Ahrens found that the men had a mean loss of 0.89 cm to 0.72 cm after a 6-mile run.[48]

Continuing Exploration 4-2:

Effects of Creep Loading on the Intervertebral Discs

Adams and colleagues reported mechanical changes in the disc with creep loading, such as loss of intervertebral disc height, increased bulging of the disc, increased stiffness in compression, and more flexibility in bending.[45] The result of these changes is that the neural arch and the ligaments, especially the zygapophyseal joints, are subjected to large compressive and bending forces. The authors stated that these normal changes cause different spinal structures to be more heavily loaded at different times of the day. In addition, Adams and colleagues reported that with prolonged compressive forces, there will be a shift in load from the nucleus pulposus to the anulus fibrosus, especially the posterior aspects.[45] This increased load can cause buckling or prolapse of the anulus fibrosus. Also, the decreased exchange of fluid causes a decrease in metabolism, thereby decreasing nutrition and healing.[45–47]

Bending

Bending causes both compression and tension on the structures of the spine. In forward flexion, the anterior structures (anterior portion of the disc, anterior ligaments, and muscles) are subjected to compression; the posterior structures are subjected to tension. The resistance offered to the tensile forces by collagen fibers in the posterior outer anulus fibrosus, zygapophyseal joint capsules, and posterior ligaments help to limit extremes of motion and thus provide stability in flexion. Creep occurs when the vertebral column is subjected to sustained loading, such as might occur in the fully flexed postures commonly assumed in gardening or the fully extended postures assumed in painting a ceiling. The resulting deformation (elongation or compression) of supporting structures such as ligaments, joint capsules, and intervertebral discs leads to an increase in the range of motion beyond normal limits and places the vertebral structures at risk of injury. If the creep deformation of tissues occurs within the toe region of the stress-strain curve, the structures will return to their original dimensions either minutes or hours after cessation of the activity.

In extension, the posterior structures generally are either unloaded or subjected to compression while the anterior structures are subjected to tension.[49] In general, resistance to extension is provided by the anterior outer fibers of the anulus fibrosus, the zygapophyseal joint capsules, passive tension in the anterior longitudinal ligament, and possibly by contact of the spinous processes. In lateral bending, the ipsilateral side of the disc is compressed; that is, in right lateral bending, the right side of the disc is compressed and the outer fibers of the left side of the disc are stretched. Therefore, the contralateral fibers of the outer anulus fibrosus and the contralateral intertransverse ligament help to provide stability during lateral bending by resisting extremes of motion.

Torsion

Torsional forces are created during axial rotation that occurs as a part of the coupled motions that take place in the spine. The torsional stiffness in flexion and lateral bending of the upper thoracic region from T1 to T6 is similar, but torsional stiffness increases from T7/T8 to L3/L4. Torsional stiffness is provided by the outer layers of both the vertebral bodies and intervertebral discs and by the orientation of the facets.[50] The outer shell of cortical bone reinforces the trabecular bone and provides resistance to torsion.[50] When the disc is subjected to torsion, half of the anulus fibrosus fibers resist clockwise rotations while fibers oriented in the opposite direction resist counterclockwise rotations. It has been suggested that the anulus fibrosus may be the most effective structure in the lumbar region for resisting torsion[51]; however, the risk of rupture of the disc fibers is increased when torsion, heavy axial compression, and forward bending are combined.[52]

Shear

Shear forces act on the midplane of the disc and tend to cause each vertebra to undergo translation (move anteriorly, posteriorly, or from side to side in relation to the inferior vertebra). In the lumbar spine, the zygapophyseal joints resist some of the shear force and the discs resist the remainder. When the load is sustained, the discs exhibit creep, and the zygapophyseal joints may have to resist all of the shear force. Table 4–4 summarizes vertebral function.

CASE APPLICATION

Effects of Creep Loading on Low Back Structures

case 4–3

Malik has a job that involves lifting and carrying heavy loads in a repetitive manner. Therefore, the intervertebral discs in his lumbar region will have experienced creep loading as a result of these loads. These repetitive loads, combined with the flexed postures that he sustains daily, may have caused damage to the posterior aspects of the anulus fibrosus and decreased fluid exchange to the disc. As a result, the neural arch , particularly the zygapophyseal joints, could be overloaded. In addition, with fluid loss, the capsuloligamentous support could be compromised as a result of the loss in disc height. (Recall, too, that the anular fibers in this region of the spine do not have as much support from the posterior longitudinal ligament.) Also, Malik may have increased mobility at one or more segments and decreased ability to resist bending and shear forces. Other possibilities are that the pain Malik is feeling is the result of tears in the anulus fibrosus, damage to posterior ligaments (such as the posterior longitudinal ligament or the interspinous ligament), or damage to the capsules or joint surfaces of the lumbar zygapophyseal joints, all of which are innervated and potential sources of pain.

Table 4–4 **Summary: Vertebral Function**

STRUCTURE	FUNCTION
Body	Resists compressive forces. Transmits compressive forces to vertebral end plates.
Pedicles	Transmit bending forces (exerted by muscles attached to the spinous and transverse processes) to the vertebral bodies.
Laminae	Resist and transmit forces (that are transmitted from spinous and zygapophyseal articular processes) to pedicles. Serve as attachment sites for muscles and ligaments.
Transverse processes	Serve as attachment sites for muscles and ligaments.
Spinous processes	Resist compression and transmit forces to laminae. Serve as attachment sites for ligaments and muscles.
Zygapophyseal facets	Resist shear, compression, tensile and torsional forces. Transmit forces to laminae.
Nucleus pulposus	Resists compression forces to vertebral end plates and translates vertical compression forces into circumferential tensile forces in anulus fibrosus.
Anulus fibrosus	Resists tensile, torsional, and shear forces.

REGIONAL STRUCTURE AND FUNCTION

The complexity of a structure that must fulfill many functions is reflected in the design of its component parts. Regional structures are varied to meet different but equally complex functional requirements. Structural variations evident in the first cervical and thoracic vertebrae, fifth lumbar vertebra, and sacral vertebrae represent adaptations necessary for joining the vertebral column to adjacent structures. Differences in vertebral structure are also apparent at the cervicothoracic, thoracolumbar, and lumbosacral junctions, at which a transition must be made between one type of vertebral structure and another. The vertebrae located at regional junctions are called **transitional vertebrae** and usually possess characteristics common to two regions. The cephalocaudal increase in the size of the vertebral bodies reflects the increased proportion of body weight that must be supported by the lower thoracic and lumbar vertebral bodies. Fusion of the sacral vertebrae into a rigid segment reflects the need for a firm base of support for the column. In addition to these variations, a large number of minor alterations in structure occur throughout the column. However, only the major variations are discussed here.

Structure of the Cervical Region

The cervical vertebral column consists of seven vertebrae in total. Morphologically and functionally, the cervical column is divided into two distinct regions: the upper cervical spine, or craniovertebral region, and the lower cervical spine (Fig. 4–21). The craniovertebral region includes the occipital condyles and the first two cervical vertebrae, **C1** and **C2,** or, respectively, the **atlas** and **axis.** The **lower cervical spine** includes the vertebrae of **C3 to C7.** The vertebrae from C3 to C6 display similar characteristics and are therefore considered to be the typical cervical vertebrae. The atlas, axis, and C7 exhibit unique characteristics and are considered the atypical cervical vertebrae. All of the cervical vertebrae have the unique feature of a foramen (transverse foramen) on the transverse process, which serves as passage for the vertebral artery.

Craniovertebral Region

Atlas

The atlas (C1) is frequently described as a washer sitting between the occipital condyles and the axis. The functions of the atlas are to cradle the occiput and to transmit forces from the occiput to the lower cervical vertebrae. These functions are reflected in the bony structure. The atlas is different from other vertebrae in that it has no vertebral body or spinous process and it is shaped like a ring (Fig. 4–22A). There are two large lateral masses on the atlas that have a vertical orientation to each occipital condyle, which reflects the function of transmitting forces (Fig. 4–22B). The lateral masses are connected by an anterior and a posterior arch, which together form the ring structure and also create large transverse processes for muscle attachments.[8] The transverse processes also contain a foramen for the passage of the vertebral artery. The lateral masses include four articulating facets: two superior and two inferior (Fig. 4–22C). The superior zygapophyseal facets are large, typically kidney-shaped, and deeply concave to accommodate the large, convex articular surfaces of the occipital condyles. There is, however, wide variation in the size and shape of these facets. The inferior zygapophyseal facets are slightly convex and are directed inferiorly for articulation with the superior zygapophyseal facets of the axis (C2). The atlas also possesses a facet on the internal surface of the anterior arch for articulation with the dens **(odontoid process)** of the axis.

Axis

The primary functions of the axis are to transmit the combined load of the head and atlas to the remainder of the cervical spine and to provide motion into axial rotation

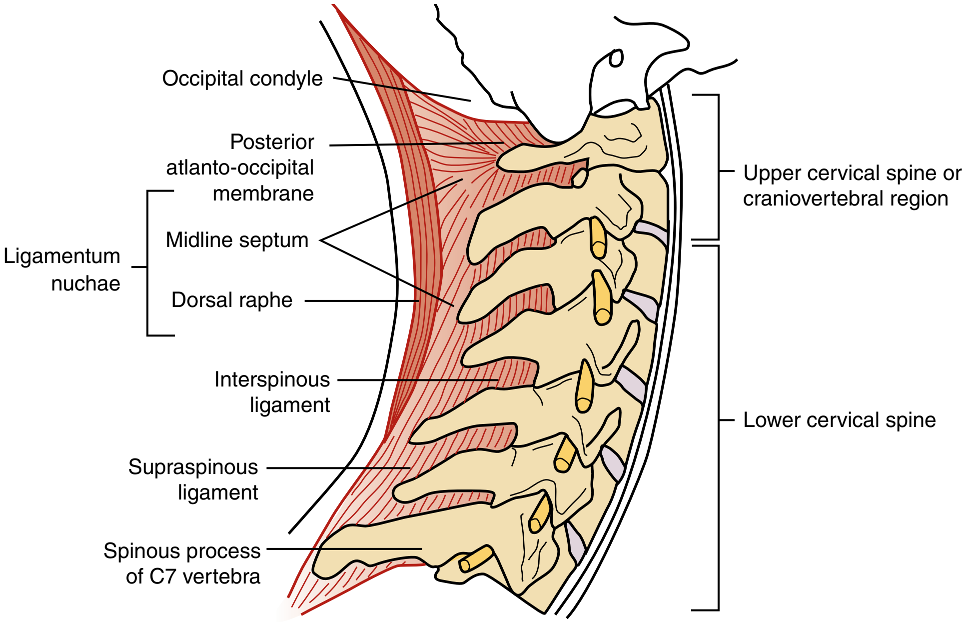


Figure 4–21 The cervical region consists of the upper cervical spine, or craniovertebral region, and the lower cervical spine.

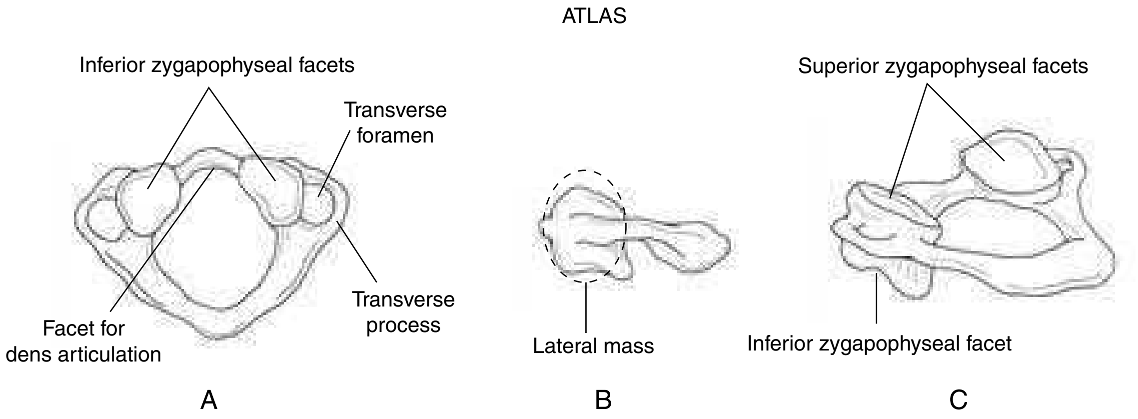


Figure 4–22 The atlas is a markedly atypical vertebra. **A.** The atlas is shaped like a ring and lacks a body and a spinous process. **B.** The lateral view demonstrates the lateral mass. **C.** The zygapophyseal facets.

of the head and atlas.[8] The axis is atypical in that the anterior portion of the body extends inferiorly and a vertical projection called the **dens** arises from the superior surface of the body (Fig. 4–23). The dens has an anterior facet for articulation with the anterior arch of the atlas and a posterior groove for articulation with the transverse ligament. The arch of the axis has inferior and superior zygapophyseal facets for articulation with the adjacent inferior vertebra and with the atlas, respectively. The spinous process of the axis is large and elongated, with a bifid (split into two portions) tip. The superior zygapophyseal facets of the axis face upward and laterally. The inferior zygapophyseal facets face anteriorly.[53]

Articulations

The two **atlanto-occipital joints** consist of the two concave superior zygapophyseal facets of the atlas articulating with the two convex occipital condyles of the skull. These joints are true synovial joints with intra-articular fibroadipose meniscoids and lie nearly in the horizontal plane.

There are three synovial joints that compose the **atlantoaxial joints**: the median atlantoaxial joint between the dens and the atlas and two lateral joints between the superior zygapophyseal facets of the axis and the inferior zygapophyseal facets of the atlas (Fig. 4–24). The median joint is a synovial trochoid (pivot) joint in which the dens of the axis rotates within an osteoligamentous ring formed by the anterior arch of the atlas and the **transverse ligament.** The two lateral joints appear, on the basis of bony structure, to be plane synovial joints; however, the articular cartilages of both the atlantal and axial facets are convex, rendering the zygapophyseal facet joints biconvex.[54] The joint spaces that occur as a result of the incongruence of the biconvex structure are filled with meniscoids.

Craniovertebral Ligaments

Besides the longitudinal ligaments mentioned earlier in this chapter, a number of other ligaments are specific to the cervical region. Many of these ligaments attach to the axis, atlas, or skull and reinforce the articulations of the upper two vertebrae. Four of the ligaments are continuations of the longitudinal tract system; the four remaining ligaments are specific to the cervical area.

The **posterior atlanto-occipital** and **atlantoaxial membranes** are the continuations of the ligamentum flavum (Fig. 4–25A). Their structure, however, varies from

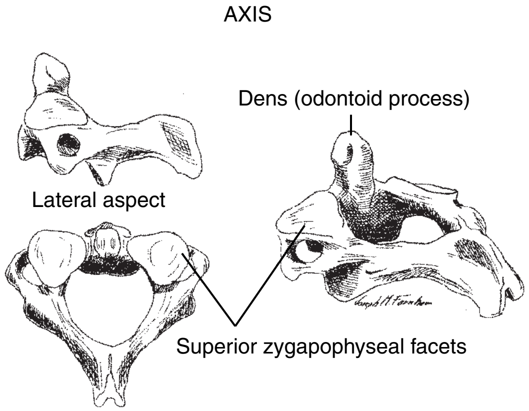


Figure 4–23 The dens (odontoid process) arises from the anterior portion of the body of the axis. The superior zygapophyseal facets are located on either side of the dens.

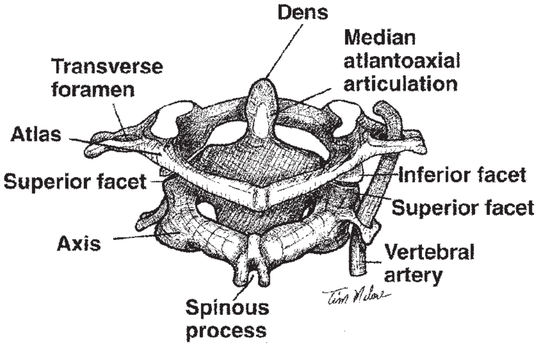


Figure 4–24 Atlantoaxial articulation. The median atlantoaxial articulation is seen, with the posterior portion (transverse ligament) removed to show the dens and the anterior arch of the atlas. The two lateral atlantoaxial joints between the superior zygapophyseal facets of the axis and the inferior facets of the atlas can be seen on either side of the median atlantoaxial joint.

the ligamentum flavum in that they are less elastic and therefore permit a greater range of motion, especially into rotation.[55] The **anterior atlanto-occipital** and **atlantoaxial membranes** are the continuations of the anterior longitudinal ligament (Fig. 4–25B). The **tectorial membrane** is the continuation of the posterior longitudinal ligament in the upper two segments and is a broad, strong membrane that originates from the posterior vertebral body of axis, covers the dens and its cruciate ligament, and inserts at the anterior rim of the foramen magnum[55] (Fig. 4–26).

Descriptions of the **ligamentum nuchae** (see Fig. 4–21) vary. Some describe it as an evolution of the supraspinous ligament with a thick, triangular, sheetlike structure, the base of which is attached to the skull from the external occipital protuberance and foramen magnum and the apex of which is attached to the tip of the C7 spinous process.[25,27] Its function is sometimes described as resisting flexion of the head and serving as a site for muscle attachments.[25,56,57] Mercer and Bogduk,[57] however, studied ten cadavers and found the ligamentum nuchae to be composed of two distinct parts: a dorsal raphe and a ventral midline septum. They describe the dorsal raphe as a narrow strip of collagenous tissues formed by the interlacing tendons of the upper trapezius, splenius capitis, and the rhomboid minor muscles. The dorsal raphe is firmly attached to the external occipital protuberance and the tip of the C7 spinous process, but it is freely mobile between these attachment sites. The fascial septum is composed of dense connective tissue running ventrally from the dorsal raphe and the external occipital crest to the tips of the cervical spinous processes.[57] It is continuous with the interspinous ligaments, the atlantoaxial and atlanto-occipital membranes, and the fascia of the semispinalis capitis and splenius capitis muscles.[57] This description disputes the notion that the ligamentum nuchae is a strong ligament securely attached to all cervical vertebrae and capable of playing a primary role in resisting flexion of the head.[57]

Transverse Ligament

The **transverse atlantal ligament** stretches across the ring of the atlas and divides the ring into a large posterior section for the spinal cord and a small anterior space for the dens. The transverse length of the ligament is about 21.9 mm.[58] The transverse ligament has a thin layer of articular cartilage on its anterior surface for articulation with the dens. Superior longitudinal fibers of the transverse ligament extend to attach to the occipital bone, and inferior fibers descend to the posterior portion of the axis. The transverse ligament and its longitudinal bands are sometimes referred to as the **atlantal cruciform ligament** (Fig. 4–27). The transverse portion of the ligament holds the dens in close approximation against the anterior ring of the atlas and serves as anarticular surface. Its primary role, however, is to prevent anterior displacement of C1 on C2. This ligament is critical in maintaining stability at the C1/C2 segment. Its superior and inferior longitudinal bands provide some assistance in this role. The transverse atlantal ligament is very strong, and the dens will fracture before the ligament will tear.[27] Integrity of the transverse ligament can be compromised, however,

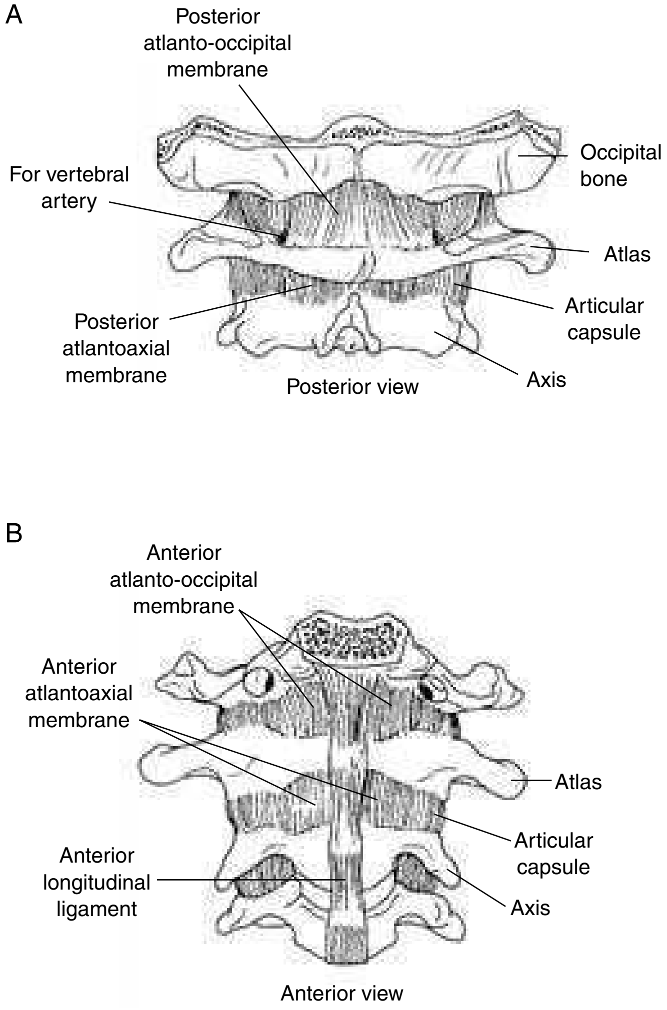


Figure 4–25 **A.** Posterior atlanto-occipital and atlantoaxial membranes. **B.** Anterior atlanto-occipital and atlantoaxial membranes.

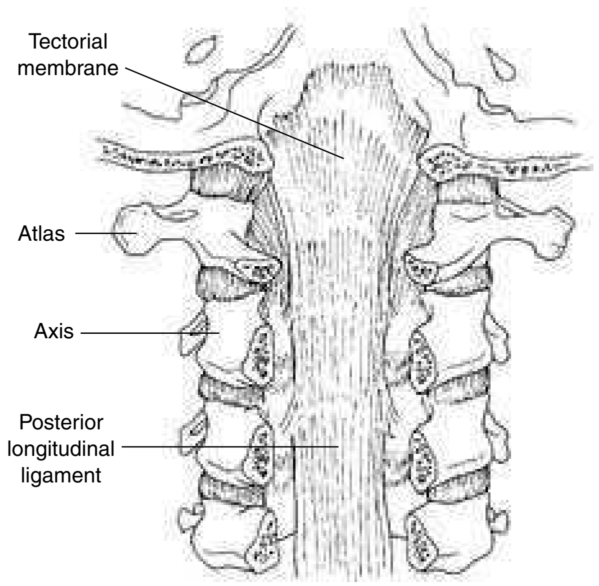


Figure 4–26 The **tectorial membrane** is a continuation of the posterior longitudinal ligament in the craniovertebral region.

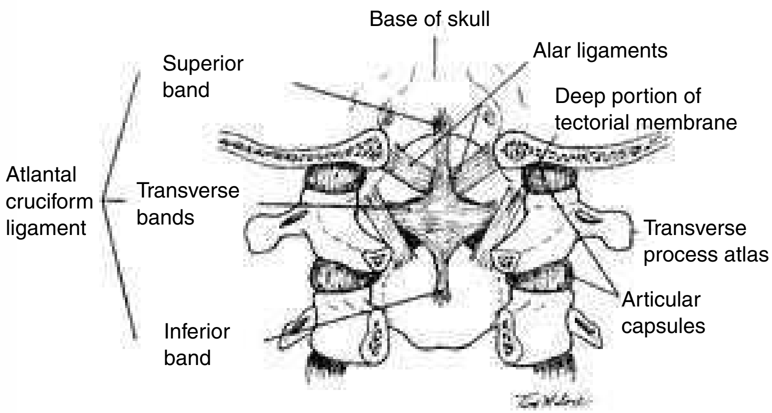


Figure 4–27 The transverse atlantal ligament. This is a posterior view of a vertebral column in which the posterior portion of the vertebrae (spinous processes and portion of the arches) has been removed to show the atlantal cruciform and alar ligaments.

particularly with diseases such as rheumatoid arthritis and conditions such as Down syndrome.

Alar Ligaments

The two alar ligaments are also specific to the cervical region (see Fig. 4–27). These paired ligaments arise from the axis on either side of the dens and extend laterally and superiorly to attach to roughened areas on the medial sides of the occipital condyles[59] and to the lateral masses of the atlas.[58] The ligaments are approximately 1 cm in length and about a pencil width in diameter and consist mainly of collagen fibers arranged in parallel.[59] These ligaments are relaxed with the head in neutral position and taut in neck flexion.[59] Axial rotation of the head and neck tightens both alar ligaments.[59] The right upper and left lower portions of the alar ligaments limit left lateral flexion of the head and neck.[6] These ligaments also help to prevent distraction of C1 on C2. The alar ligaments are weaker than the transverse atlantal ligament. The **apical ligament** of the dens connects the axis and the occipital bone of the skull. It runs in a fan-shaped arrangement from the apex of the dens to the anterior margin of the foramen magnum of the skull.[27]

The Lower Cervical Region

Typical Cervical Vertebrae

Body

The body (Fig. 4–28) of the cervical vertebra is small, with a transverse diameter greater than its anteroposterior diameter and height. The upper and lower end plates from C2 to C7 also have transverse diameters (widths) that are greater than the corresponding anteroposterior diameters. The transverse and anteroposterior diameters increase from C2 to C7, with a significant increase in both diameters in the upper end plate of C7.[60] The posterolateral margins of the upper surfaces of the vertebral bodies from C3 to C7 support **uncinate processes** that give the upper surfaces of these vertebrae a concave shape in the frontal plane. The uncinate processes are present prenatally, and between 9 to 14 years of age, they gradually enlarge.[61] The anterior inferior border of the vertebral body forms a lip that hangs down toward the vertebral body below, which produces a concave shape of the inferior surface of the superior vertebra in the sagittal plane.

Arches

Pedicles. The pedicles project posterolaterally and are located halfway between the superior and inferior surfaces of the vertebral body.

Laminae. The laminae are thin and slightly curved. They project posteromedially.

Zygapophyseal Articular Processes (Superior and Inferior). These articular processes support paired superior and inferior facets. The superior facets are flat and oval and face superiorly and posteriorly. They lie between the transverse and frontal planes. The width and height of the superior zygapophyseal facets gradually increase from C3 to C7. The

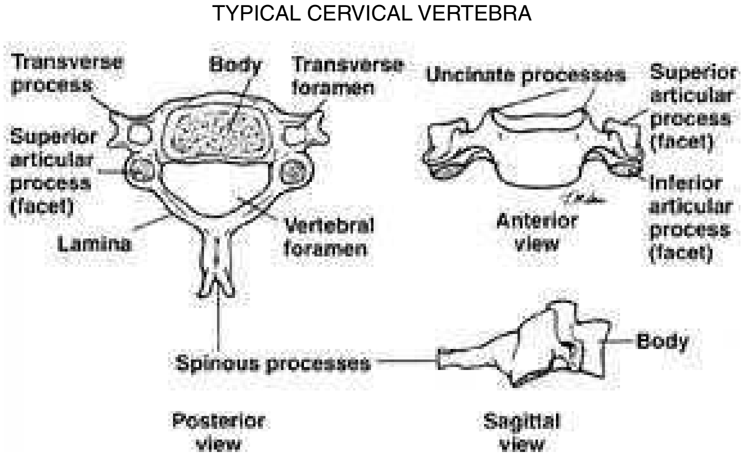


Figure 4–28 The body of a typical cervical vertebra is small and supports uncinate processes on the posterolateral superior and inferior surfaces.

paired inferior facets face inferiorly and anteriorly and lie closer to the frontal plane than do the superior facets.[27] The superior facets of C3 and C7 are more steeply oriented than the others.

Transverse Processes. A foramen is located in the transverse processes bilaterally for the vertebral artery, vein, and venous plexus. Also, there is a groove for the spinal nerves.

Spinous Processes. The cervical spinous processes are short, slender extend horizontally and have a bifid tip. The length of the spinous processes decreases slightly from C2 to C3, remains constant from C3 to C5, and undergoes a significant increase at C7.[60]

Vertebral Foramen. The vertebral foramen is relatively large and triangular to accommodate the large spinal cord.

Intervertebral Disc

The structure of the intervertebral disc in the cervical region is distinctly different from that in the lumbar region (Fig. 4–29). Mercer and Bogduk, in several works, contributed most of the information known about the structure of the cervical discs.[7,8,54] They reported that instead of a fibrous ring completely surrounding a gelatinous center, there is a discontinuous ring surrounding a fibrocartilaginous core.

The fibers of the anulus fibrosus are not arranged in alternating lamellar layers as they are in the lumbar region. In addition, they do not surround the entire perimeter of the nucleus pulposus. Instead, the anular fibers in this region have a crescent shape when viewed from above; they are thick anteriorly and taper laterally as they approach the uncinate processes[7,54] (see Fig. 4–29A). Anteriorly, the anulus fibrosus is thick with oblique fibers in the form of an inverted "V" whose apex points to the location of the axis of rotation on the anterior end of the upper vertebra.[7,54] Laterally, there is no substantive anulus fibrosus, and posteriorly, it is only a thin layer of vertically oriented fibers. Posterolaterally, the nucleus is contained only by the posterior longitudinal ligament.

Fissures in the disc develop along with the uncinate processes and become clefts by approximately 9 years of age (see Fig. 4–29B). These clefts become the joint cavity of what has been known as the **uncovertebral joints** or **"joints of Luschka."**[7,54]

Concept Cornerstone 4-2

Differences Between Cervical and Lumbar Discs

Damage to and pain from the cervical discs are unlikely to be similar in mechanism or pathoanatomy to discs of the lumbar region because of differences in the structure and function of discs between the two regions. Combined flexion and rotation movements do not damage the posterolateral fibers of the anulus fibrosus in the cervical region as they do in the lumbar region, because there are no posterolateral anular fibers. Disc herniations in the cervical region that cause spinal nerve compression will most likely also involve strain of the posterior longitudinal ligament.[9]

Interbody Joints of the Lower Cervical Region (C3 to C7)

The interbody joints of the lower cervical region are saddle joints (Fig. 4–30A). In the sagittal plane, the inferior surface of the cranial vertebra is concave and the superior surface of the caudal vertebra is convex because of the uncinate processes.[9] The motions that occur are predominantly rocking motions, although translatory motions remain

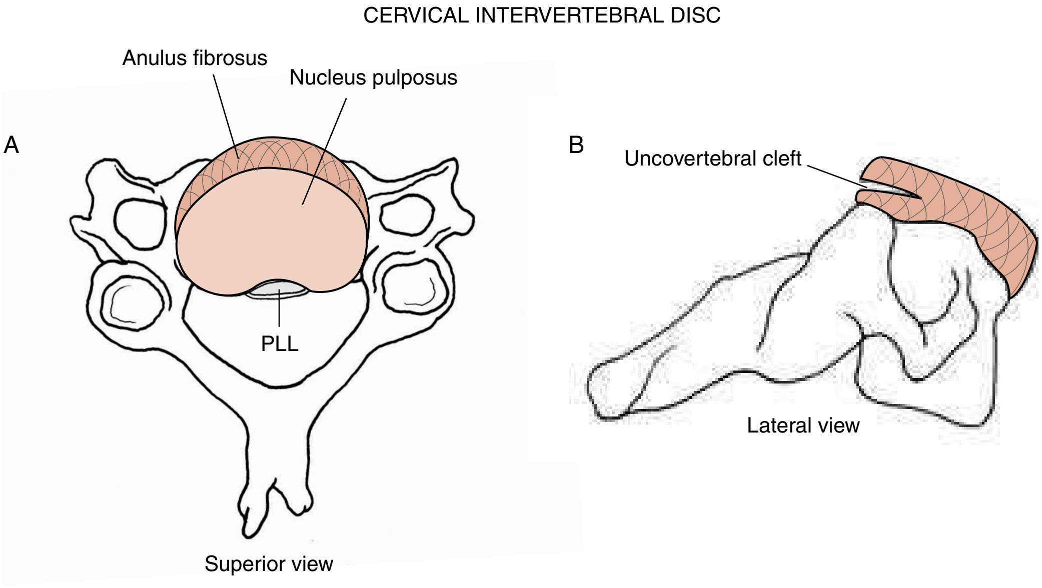


Figure 4–29 Cervical intervertebral disc. **A.** Superior view shows crescent-shaped anulus fibrosus. **B.** Lateral view shows uncovertebral cleft.

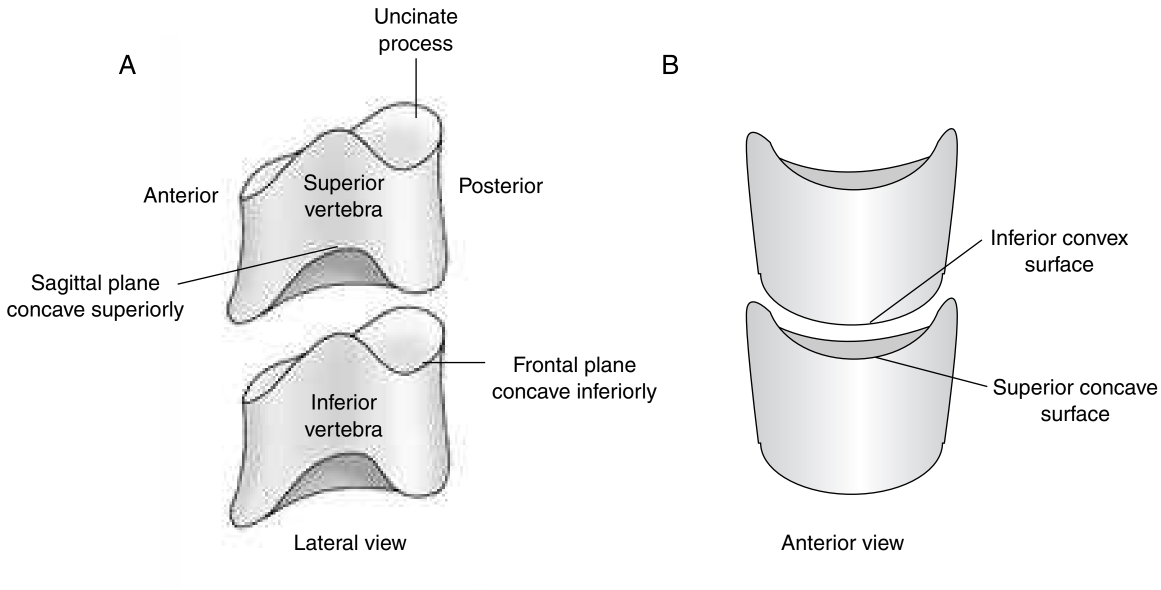


Figure 4–30 **A.** Lateral view of an interbody saddle joint of the lower cervical spine. **B.** Anterior view showing how the convex inferior surface of the superior vertebra fits into the concave superior surface of the inferior vertebra.

available.[9,54] In the frontal plane (Fig. 4–30B), the inferior surface of the cranial vertebra is convex and sits in the concave surface of the caudal vertebra created by the uncinate processes.

Zygapophyseal Joints

The zygapophyseal joints in the cervical spine, as in other regions, are true synovial joints and contain fibroadipose meniscoids.[8,9,54] The joint capsules are lax to allow a large range of motion; however, they do restrict motion at the end of the available ranges. The joints are generally described as oriented approximately 45° from the frontal and horizontal planes—in other words, they lie midway between the two planes. However, the orientation of the joints varies widely according to the individual, which contributes to the wide range of motion among individuals.[62]

Function of the Cervical Region

Although the cervical region demonstrates the most flexibility of any of the regions of the vertebral column, stability of the cervical region, especially of the atlanto-occipital and atlantoaxial joints, is essential for support of the head and protection of the spinal cord and vertebral arteries. The design of the atlas is such that it provides more free space for the spinal cord than does any other vertebra. The extra space helps to ensure that the spinal cord is not impinged on during the large amount of motion that occurs here. The bony configuration of the atlanto-occipital articulation confers some stability, but the application of small loads produces large rotations across the occipitoatlantoaxial complex[63,64] and also across the lower cervical spine.[63]

Kinematics

The cervical spine is designed for a relatively large amount of mobility. Normally, the neck moves 600 times every hour whether we are awake or asleep.[61] The motions of flexion and extension, lateral flexion, and rotation are permitted in the cervical region. These motions are accompanied by translations that increase in magnitude from C2 to C7.[65] However, the predominant translation occurs in the sagittal plane during flexion and extension.[66,67] Excessive anteroposterior translations are associated with damage to the spinal cord.[65]

The atlanto-occipital joints allow for primarily nodding movements between the head and the atlas,[68–70] although there is little agreement about the range of motion available at the atlanto-occipital joints. The combined range of motion for flexion-extension reportedly ranges from 10° to 30°.[63,68–70] In flexion, the occipital condyles roll forward and slide backward (Fig. 4–31A). In extension, the occipital condyles roll backward and slide forward (Fig. 4–31B). There are a few degrees of rotation and lateral flexion available at this segment as well.[71] The total motion available in both axial rotation and lateral flexion is extremely limited by tension in the joint capsules that occurs as the occipital condyles rise up the walls of the atlantal sockets on the contralateral side of either the rotation or lateral flexion.[59,72]

Motions at the atlantoaxial joint include rotation, lateral flexion, flexion, and extension. Approximately 55% to 58% of the total rotation of the cervical region occurs at the atlantoaxial joints[59,71,72] (Fig. 4–32). The atlas pivots about 45° to either side, or a total of about 90°. The alar ligaments limit rotation at the atlantoaxial joints. The remaining 40% of total rotation available to the cervical spine is distributed evenly in the lower joints.[72]

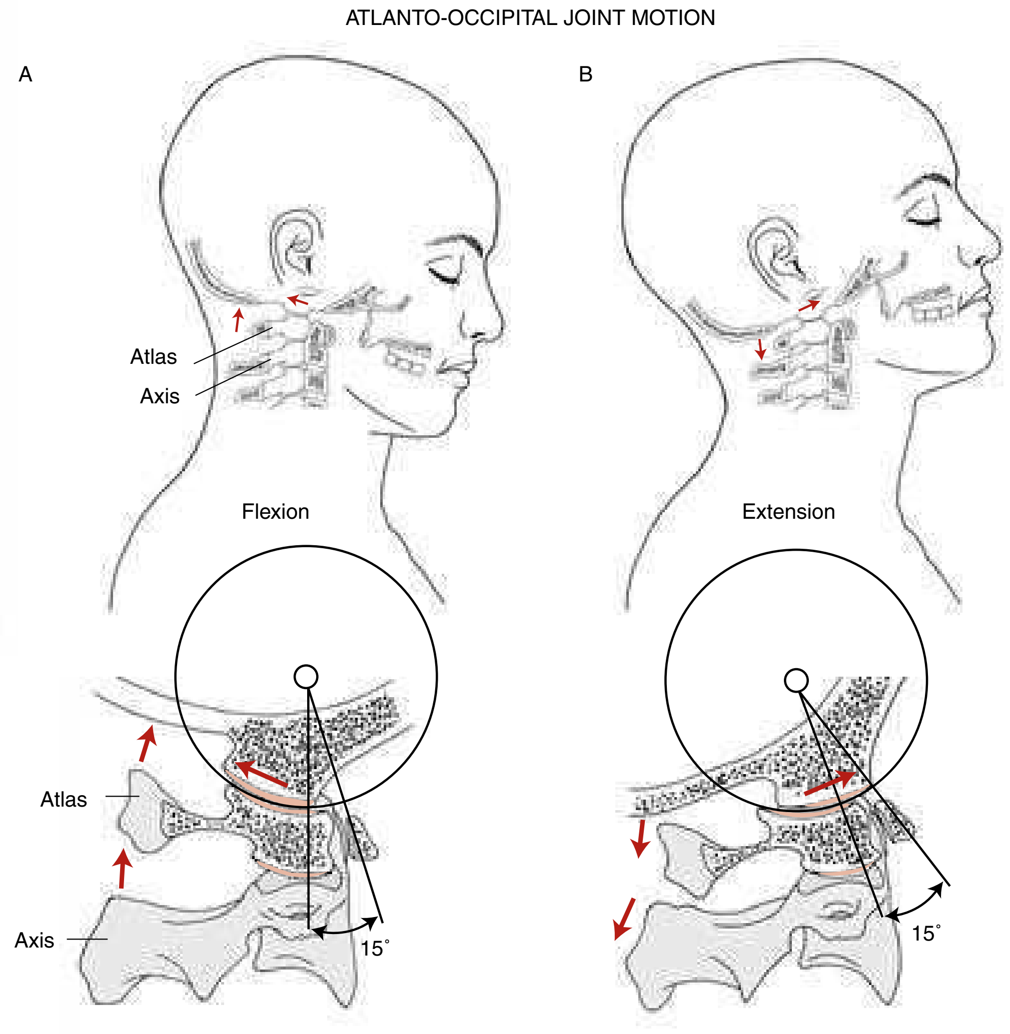


Figure 4–31 Nodding motions of the atlanto-occipital joints. **A.** Flexion. **B.** Extension.

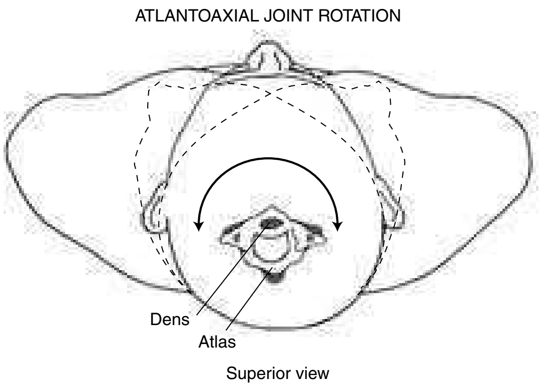


Figure 4–32 Superior view of rotation at the atlantoaxial joints: The occiput and atlas pivot as one unit around the dens of axis.

Lateral flexion and rotation are coupled motions. In the upper cervical segments, lateral flexion is coupled with contralateral rotation and rotation is coupled with contralateral lateral flexion.[71,73]

The shape of the zygapophyseal joints and the interbody joints dictates the motion at the lower cervical segments. Pure anterior translation does not occur, because it would cause the zygapophyseal joints to abut one another. Flexion of these segments must include anterior tilt of the cranial vertebral body coupled with anterior translation. Given the 45° slope, tilt of the vertebral body, in addition to anterior translation, is necessary to get full motion from these joints (Fig. 4–33). Extension includes posterior tilt of the cranial vertebral body coupled with posterior translation. Lateral flexion and rotation are also coupled motions, because movement of either alone would cause the zygapophyseal joints to abut one another and prevent motion. Lateral flexion is coupled with ipsilateral rotation, and rotation is coupled with ipsilateral lateral flexion. These motions are also a combination of vertebral tilt to the ipsilateral side and

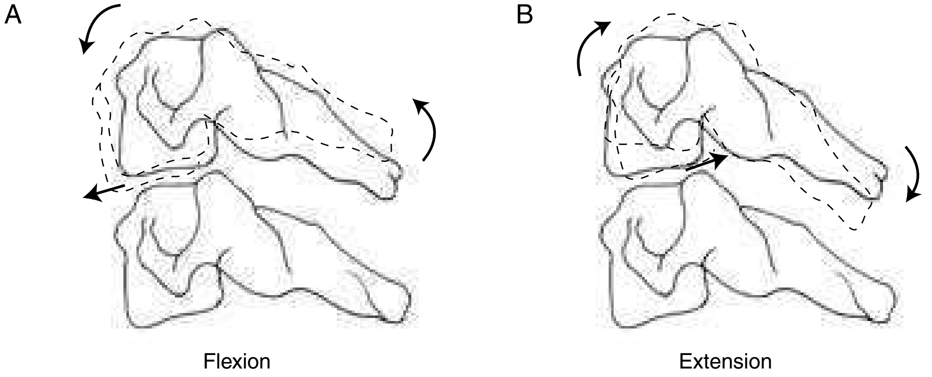


Figure 4–33 **A.** Flexion of the lower cervical spine combines anterior translation and sagittal plane rotation of the superior vertebra. **B.** Extension combines posterior translation with sagittal plane rotation.

translations at the zygapophyseal joints.[35,54] It appears, however, that coupling patterns can change with aging.[73–75]

Mercer and Bogduk[8,9,54] suggested that the notion of lateral flexion and horizontal rotation are an artificial construct. In their view, movement should be viewed as gliding that occurs in the plane of the zygapophyseal joints (Fig. 4–34). In this plane, the coupled motions are evident. Lower cervical segments generally favor flexion and extension motion; however, there is great variability in reported ranges of motion in the individual cervical segments. In general, the range for flexion and extension increases from the C2/C3 segment to the C5/C6 segment and decreases again at the C6/C7 segment.[9] The zygapophyseal joint capsules and the ligaments, in addition to the shape of the joints, dictate motions at all of the cervical segments. The zygapophyseal joint capsules are generally lax in the cervical region, which contributes to the large amount of motion available here. The height in relation to the diameter of the discs also plays an important role in determining the amount of motion available in the cervical spine. The height is large in comparison with the anteroposterior and transverse diameters of the cervical discs. Therefore, a large amount of flexion, extension, and lateral flexion may occur at each segment, especially in young people.

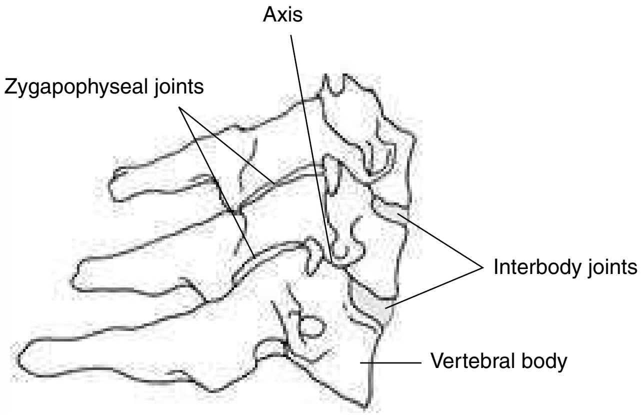


Figure 4–34 Motion at the lower cervical interbody joints occurs in the plane of the zygapophyseal joints about an axis perpendicular to the plane.

The total range of motion of the cervical spine is dependent on many factors, including age and sex; there is greater motion in those who are young and female.[76,77] A recent three-dimensional analysis of motion reports that the average range of motion in the cervical spine across all age groups is as follows: total flexion/extension: 126° (standard deviation [SD] = 22°), total lateral flexion: 87° (SD = 22°), and total rotation: 144° (SD = 23°), with wide variation among age groups.[78]

The disc at C5/C6 is subject to a greater amount of stress than other discs because C5/C6 has the greatest range of flexion-extension and is the area where the mechanical strain is greatest.[65]

Kinetics

Although the cervical region is subjected to axial compression, tension, bending, torsion, and shear stresses, as is the remainder of the spinal column, there are some regional differences. The cervical region differs from the thoracic and lumbar regions in that it bears less weight and is generally more mobile.

No discs are present at either the atlanto-occipital or atlantoaxial articulations; therefore, the weight of the head (compressive load) must be transferred directly through the atlanto-occipital joint to the articular facets of the axis. These forces are then transferred through the pedicles and laminae of the axis to the inferior surface of the body and to the two inferior zygapophyseal articular processes. Subsequently, the forces are transferred to the adjacent inferior disc. The laminae of the axis are large, which reflects the adaptation in structure that is necessary to transmit these compressive loads. The trabeculae show that the laminae of both the axis and C7 are heavily loaded, while the intervening ones are not. Loads diffuse into the lamina as they are transferred from superior to inferior articular facets.[79]

The loads imposed on the cervical region vary with the position of the head and body and are minimal in a well-supported reclining body posture. In the cervical region from C3 to C7, compressive forces are transmitted by three parallel columns: a single anterocentral column formed by the vertebral bodies and discs and two rodlike posterolateral columns composed of the left and right zygapophyseal joints. The compressive forces are transmitted mainly by the bodies and discs, with a little over one-third transmitted by the two posterolateral columns.[64,79,80] Compressive loads are relatively low during erect stance and sitting postures and high during the end ranges of flexion and extension.[21] The compressive and bending strength of the cervical spine was tested when subjected to complex, physiological loading. The cervical spine has approximately 45% of the compressive strength of the lumbar spine but only 20% of the bending strength.[81] There is greater bending strength in extension compared to flexion. Resistance to extension is provided primarily by the zygapophyseal joints and the intervertbral disc, while resistance to flexion is primarily provided by the posterior ligaments between the spinous processes.[81]

Structure of the Thoracic Region

The majority of the thoracic vertebrae adhere to the basic structural design of all vertebrae except for some minor variations. The 1st and 12th thoracic vertebrae are **transitional vertebrae** and therefore possess characteristics of the cervical and lumbar vertebrae, respectively. The **first thoracic vertebra** has a typical cervical-shaped body with a transverse diameter nearly twice the anteroposterior diameter. The spinous process of T1 is particularly long and prominent. The **12th thoracic vertebra** has thoracic-like superior zygapophyseal articular facets that face posterolaterally. The inferior zygapophyseal facets, however, are more lumbar-like and have convex surfaces that face anterolaterally to articulate with the vertical, concave, posteromedially facing superior zygapophyseal facets of the first lumbar vertebra. Additional differences in T1, T11, and T12 include the presence of full costal facets rather than demifacets, given that ribs 1, 11, and 12 articulate only with their corresponding vertebral bodies. The pedicles in the thoracic region are generally directed more posteriorly and less laterally than those in any other region, which creates a smaller vertebral canal in the thoracic region than in the cervical or lumbar regions. The laminae are short, thick, and broad. The end plates show a gradual increase in transverse and anteroposterior diameters from T1 to T12.

Typical Thoracic Vertebrae

Body

Figure 4–35 demonstrates the features of the typical thoracic vertebra. The body of a typical thoracic vertebra has equal transverse and anteroposterior diameters, which gives greater stability. The vertebral bodies are wedge-shaped, with posterior height greater than anterior height, the peak of which occurs at T7. This anterior wedging produces the normal kyphotic posture of the thoracic spine. **Demifacets** (or half facets) for articulation with the heads of the ribs are located on the posterolateral corners of the vertebral plateaus.

Arches

Pedicles. These generally face posteriorly with little to no lateral projection, creating a small vertebral canal.

Laminae. The laminae are short, thick, and broad.

Zygapophyseal Articular Processes. The superior zygapophyseal facets are thin and almost flat and face posteriorly, superiorly, and laterally. The inferior zygapophyseal facets face anteriorly, inferiorly, and medially. The facets lie nearly in the frontal plane (see Fig. 4–35A). The orientation of the facets changes at either T10 or T11 so that the superior facets face posterolaterally and the inferior facets face anterolaterally, and they lie closer to the sagittal plane.

Transverse Processes. The transverse processes have thickened ends that support paired large oval facets **(costotubercular facets)** for articulation with the tubercles of the ribs.

Spinous Processes. The spinous processes slope inferiorly and, from T5 to T8, overlap the spinous process of the adjacent inferior vertebra (see Fig. 4–35B). The spinous processes of T11 and T12 are triangular and project horizontally. For most of the thoracic spine, the tip of the spinous process lies at the level of the caudal vertebral body.

Vertebral Foramen. The vertebral foramen is small and circular (see Fig. 4–35C).

Intervertebral Discs

There has been little study of the structure of the thoracic intervertebral discs; however, the structure is generally held to be similar to that of discs in the lumbar region, with differences only in size and shape. Thoracic intervertebral discs are thinner than those in other regions, especially in the upper thoracic segments. Also, the ratio of disc size to vertebral body size is smallest in the thoracic region, which results in greater stability and less mobility for this region. The intervertebral discs are also somewhat wedge-shaped, with the posterior height greater than the anterior height, which contributes to the thoracic kyphosis.[82] The thoracic intervertebral discs are primarily restraints to movement and are considered the primary stabilizers of the mobile segment.[83]

Articulations

Interbody Joints

The interbody joints of the thoracic spine involve flat vertebral surfaces that allow for all translations to occur. The intervertebral disc allows for tipping of the vertebral bodies; however, its relatively small size limits the available motion.

Zygapophyseal Joints

The zygapophyseal joints are plane synovial joints with fibroadipose meniscoids present. These joints lie approximately

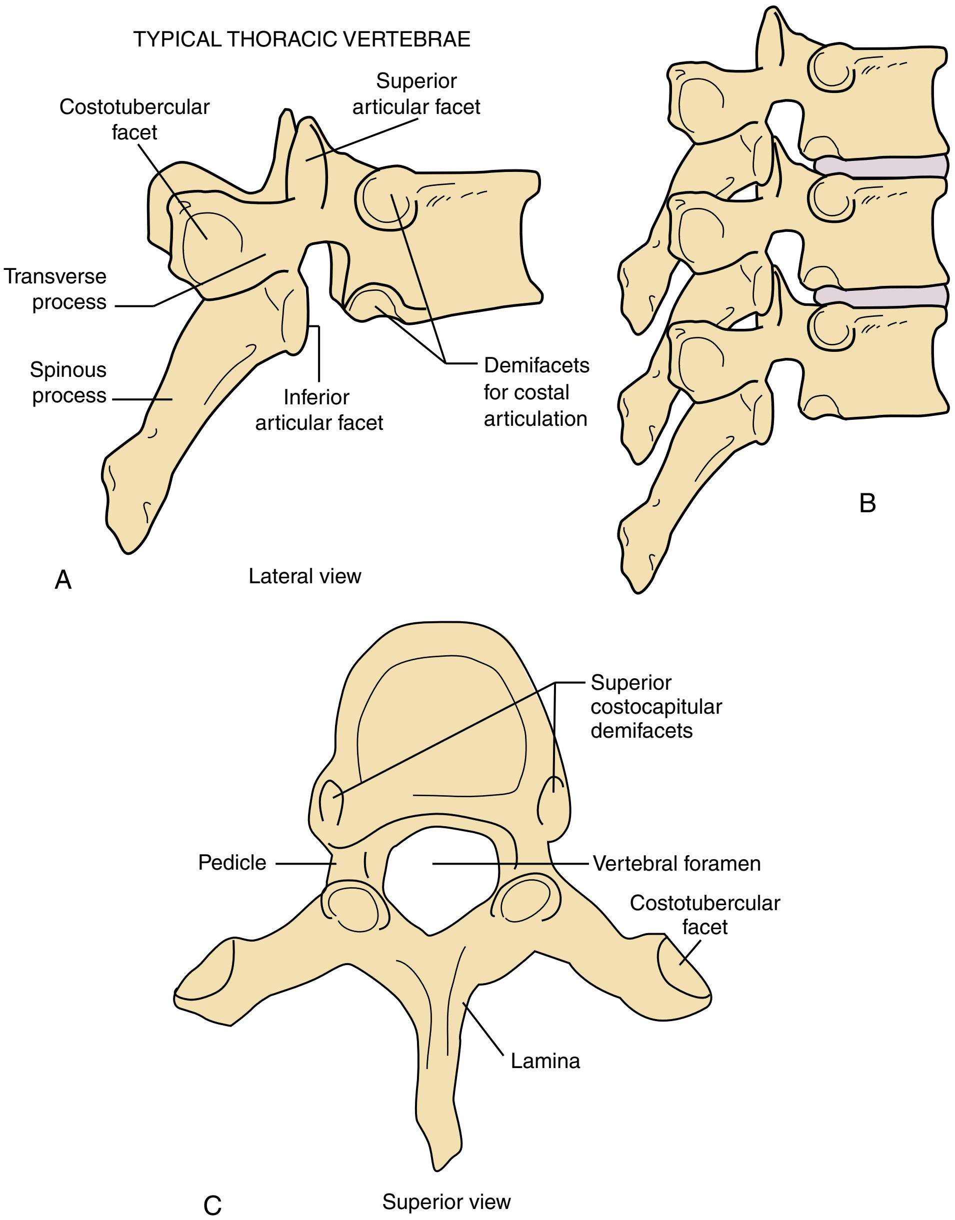


Figure 4–35 Typical thoracic vertebra **A.** Lateral view of the thoracic vertebra shows the superior and inferior facets of the zygapophyseal joints and the demifacets for articulation with the ribs. **B.** Overlapping of spinous processes in thoracic region. **C.** Superior view of a thoracic vertebra, showing the small, circular shape of the vertebral foramen, the costotubercular facets for articulation with the tubercles of the ribs, and the superior costocapitular facets for articulation with the heads of the ribs.

20° off the frontal plane, which allows greater range of motion into lateral flexion and rotation and less range of motion into flexion and extension (see Fig. 4–18B). The joint capsules are more taut than those of the cervical and lumbar regions, which also contributes to less available motion.

Ligaments

The ligaments associated with the thoracic region are the same as the ligaments described at the beginning of the chapter, except that the ligamentum flavum and anterior longitudinal ligaments are thicker in the thoracic region than in the cervical region.

Function of the Thoracic Region

The thoracic region is less flexible and more stable than the cervical region because of the limitations imposed by structural elements such as the rib cage, spinous processes, taut zygapophyseal joint capsules, the ligamentum flavum, and the dimensions of the discs and the vertebral bodies. Each thoracic vertebra articulates with a set of paired ribs by way of two joints: the **costovertebral** and the **costotransverse**

joints. The vertebral components of the costovertebral joints are the demifacets located on the vertebral bodies. The vertebral components of the costotransverse joints are the oval facets on the transverse processes. These joints are discussed in detail in Chapter 5.

Kinematics

All motions are possible in the thoracic region, but the range of flexion and extension is extremely limited in the upper thoracic region (T1 to T6) because of the rigidity of the rib cage and because of the zygapophyseal facet orientation in the frontal plane. In the lower part of the thoracic region (T9 to T12), the zygapophyseal facets lie closer to the sagittal plane, allowing an increased amount of flexion and extension. Lateral flexion and rotation are free in the upper thoracic region. The range of motion in lateral flexion is always coupled with some axial rotation. The amount of accompanying axial rotation decreases in the lower part of the region because of the change in orientation of the zygapophyseal facets at T10 or T11. In the upper part of the thoracic region, lateral flexion and rotation are coupled in the same direction, while rotation in the lower region may be accompanied by lateral flexion in the opposite direction.[16] The direction of coupled rotation may vary widely among individuals.[6]

Flexion in the thoracic region is limited by tension in the posterior longitudinal ligament, the ligamentum flavum, the interspinous ligaments, and the capsules of the zygapophyseal joints. Extension of the thoracic region is limited by contact of the spinous processes, laminae, and zygapophyseal facets and by tension in the anterior longitudinal ligament, zygapophyseal joint capsules, and abdominal muscles. Lateral flexion is restricted by impact of the zygapophyseal facets on the concavity of the lateral-flexion curve and by limitations imposed by the rib cage.[66] Rotation in the thoracic region also is limited by the rib cage. When a thoracic vertebra rotates, the motion is accompanied by distortion of the associated rib pair (Fig. 4–36). The posterior portion of the rib on the side to which the vertebral body rotates becomes more convex as the anterior portion of the rib becomes flattened. The amount of rotation that is possible depends on the ability of the ribs to undergo distortion and the amount of motion available in the costovertebral and costotransverse joints. As a person ages, the costal cartilages ossify and allow less distortion. This results in a reduction with aging in the amount of rotation available.

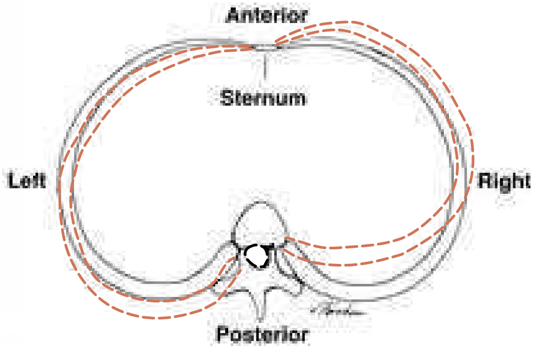


Figure 4–36 Rotation of a thoracic vertebral body to the left produces a distortion of the associated rib pair that is convex posteriorly on the left and convex anteriorly on the right.

Kinetics

The thoracic region is subjected to increased compression forces in comparison with the cervical region because of the greater amount of body weight that needs to be supported and the region's kyphotic shape. The line of gravity falls anterior to the thoracic spine. This produces a flexion moment on the thoracic spine that is counteracted by the posterior ligaments and the spinal extensors. The greatest flexion moment is at the peak of the kyphosis as a result of the increased moment arm of the line of gravity.[6]

Structure of the Lumbar Region

The first four lumbar vertebrae are similar in structure. The fifth lumbar vertebra has structural adaptations for articulation with the sacrum.

Typical Lumbar Vertebrae

Body

The body (Fig. 4–37) of the typical lumbar vertebra is massive, with a transverse diameter that is greater than the anteroposterior diameter and greater than the height. The size and shape reflect the need to support great compressive loads caused by body weight, ground reaction forces, and muscle contraction.

Arches

Pedicles. The pedicles are short and thick and project posterolaterally.

Laminae. The laminae are short and broad.

Zygapophyseal Articular Processes. According to Bogduk, both the superior and inferior zygapophyseal facets vary considerably in shape and orientation (see Fig. 4–37).[2] **Mamillary processes,** which appear as small bumps, are located on the posterior edge of each superior zygapophyseal facet[2] (see Fig. 4–37). The mamillary processes serve as attachment sites for the multifidus and medial intertransverse muscles.[27] The inferior zygapophyseal facets are vertical and convex and face slightly anteriorly and laterally.[27]

Transverse Process. The transverse process is long and slender and extends horizontally. **Accessory processes,** which are small, irregular bony prominences, are located on the posterior surface of each transverse process near its attachment to the pedicle[84] (see Fig. 4–37). The accessory processes serve as attachment sites for the multifidus and medial intertransverse muscles.

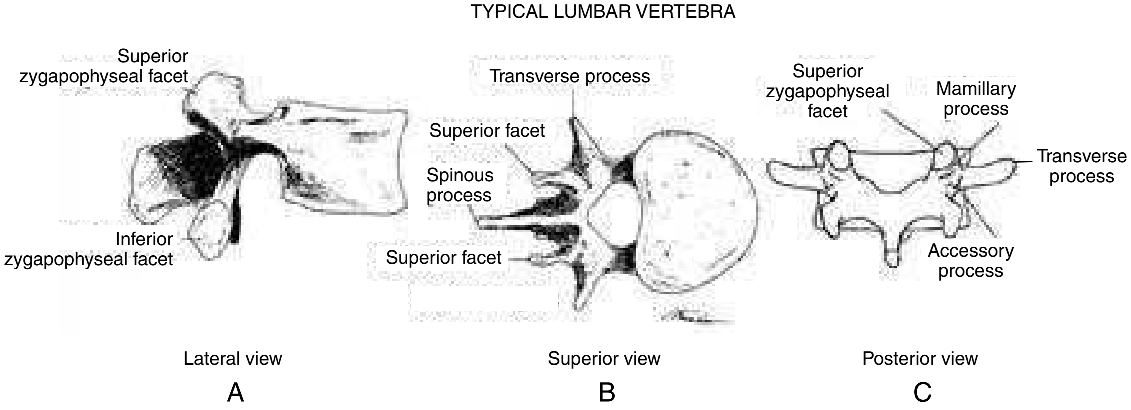


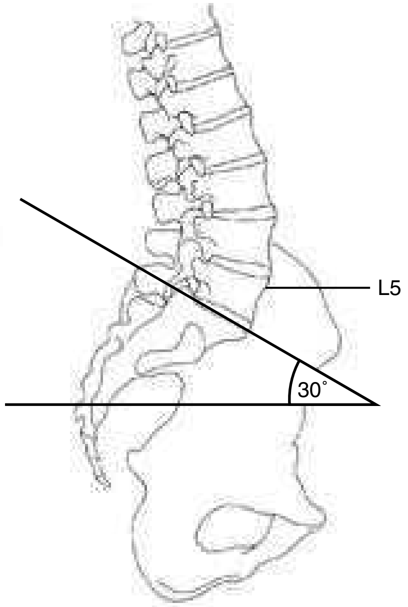
Figure 4–37 **A.** The lateral view of a typical lumbar vertebra shows the large body and zygapophyseal facets. **B.** The superior view of a typical lumbar vertebra shows transverse and spinous processes and superior zygapophyseal facets. **C.** The posterior view of a lumbar vertebra shows the location of the mamillary and accessory processes. The mamillary processes appear as small, smooth bumps on the posterior edges of each zygapophyseal facet. The accessory processes are easily recognizable as the bony prominences on the posterior surfaces of the transverse processes, close to the attachment of the transverse processes to the pedicles.

Spinous Process. The spinous process is broad and thick and extends horizontally.

Vertebral Foramen. The vertebral foramen is triangular and larger than the thoracic vertebral foramen but smaller than the cervical vertebral foramen.

The fifth lumbar vertebra is a transitional vertebra and differs from the rest of the lumbar vertebrae in that it has a wedge-shaped body whose anterior portion is of greater height than the posterior portion.[85] The L5/S1 lumbosacral disc also is wedge-shaped. The superior discal surface area of L5 is about 5% greater than the areas of discs at L3 and L4. The inferior discal surface area of L5 is smaller than the discal surface area at other lumbar levels. Also, the spinous process is smaller than other lumbar spinous processes, and the transverse processes are large and directed superiorly and posteriorly.

The **lumbosacral articulation** is formed by the fifth lumbar vertebra and first sacral segment. The first sacral segment, which is inclined slightly anteriorly and inferiorly, forms an angle with the horizontal called the **lumbosacral angle**[86] (Fig. 4–38). The size of the angle varies with the position of the pelvis and affects the superimposed lumbar curvature. An increase in this angle will result in an increase in lordosis of the lumbar curve and will increase the amount of shearing stress at the lumbosacral joint (Fig. 4–39).



Lumbosacral angle

Figure 4–38 The lumbosacral angle is determined by measuring the angle formed by a line drawn parallel to the superior aspect of the sacrum and a horizontal line.

Intervertebral Discs

Specific regional variations occur in the intervertebral discs of the lumbar region, which differ from the discs of the cervical region in that the collagen fibers of the anulus fibrosus are arranged in sheets called **lamellae** (Fig. 4–40). The lamellae are arranged in concentric rings that surround the nucleus. Collagen fibers in adjacent rings are oriented in opposite directions at 120° to each other.[6,10] The advantage of the varying fiber orientation by layer is that the anulus fibrosus is able to resist tensile forces in nearly all directions. The lumbar intervertebral discs are the largest in the body (as are the lumbar vertebral bodies). The shape of each disc is not purely elliptical but is concave posteriorly. This provides a greater cross-sectional area of anulus fibrosus posteriorly and

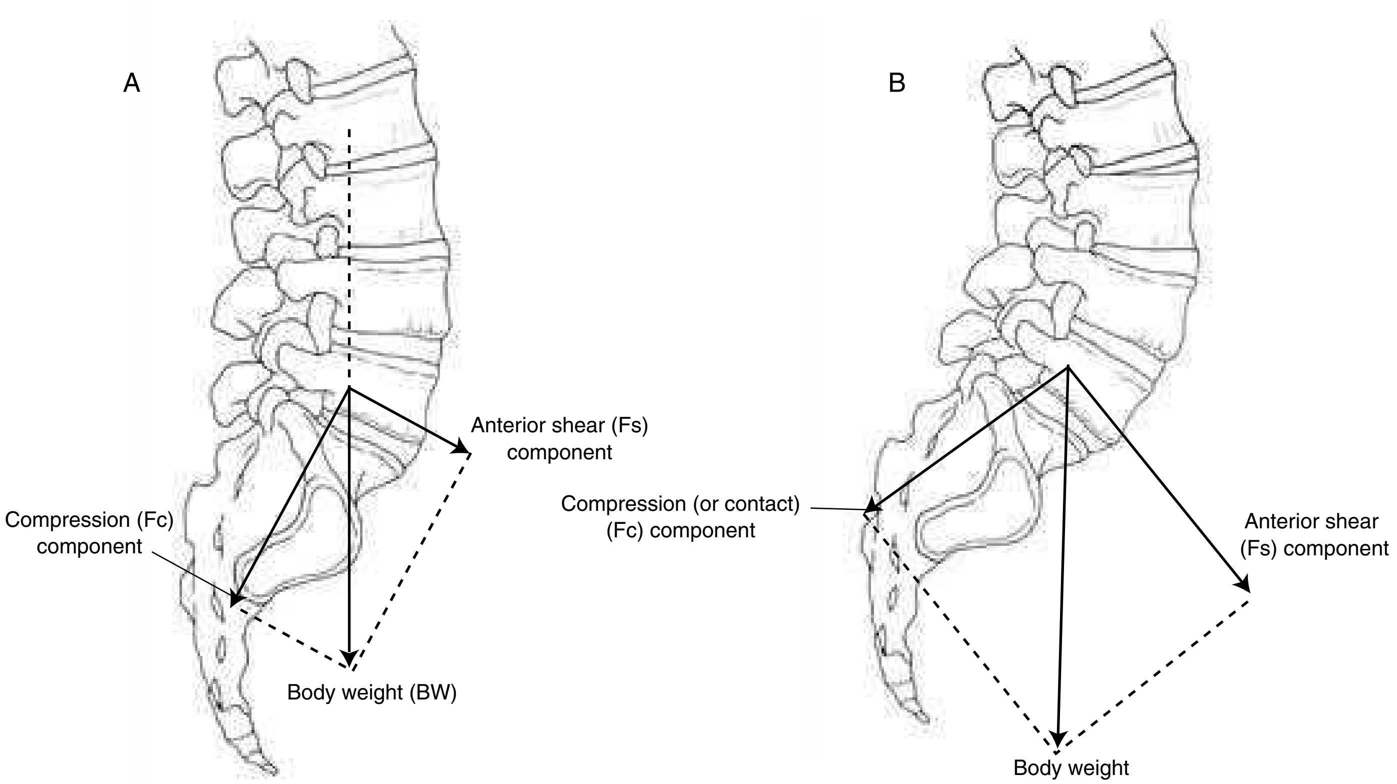


Figure 4–39 Shear stresses at the lumbosacral joint. **A.** Anterior shear with typical lumbosacral angle of 30°. Body weight acting on L5 results in L5's having both a compressive force (Fc) and an anterior shear force (Fs) in relation to the inclined surface of S1. **B.** With an increased lumbosacral angle of 45°, the force of the body weight acting on L5 results in a shear force (Fs) that is equal to or greater than the compressive force (Fc) in relation to the inclined surface of S1.

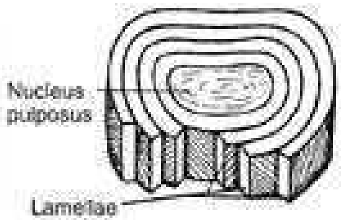


Figure 4–40 Schematic representation of an intervertebral disc, showing arrangement of lamellae in the anulus fibrosus. The collagen fibers in any two adjacent concentric bands or sheets (lamellae) are oriented in opposite directions.

thus increases the ability to resist the tension that occurs here with forward bending[2] (Fig. 4–41).

Articulations

Interbody Joints

The interbody joints of the lumbar region are capable of translations and tilts in all directions.

Zygapophyseal Joints

The zygapophyseal joints of the lumbar region, like all others, are true synovial joints and contain fibroadipose

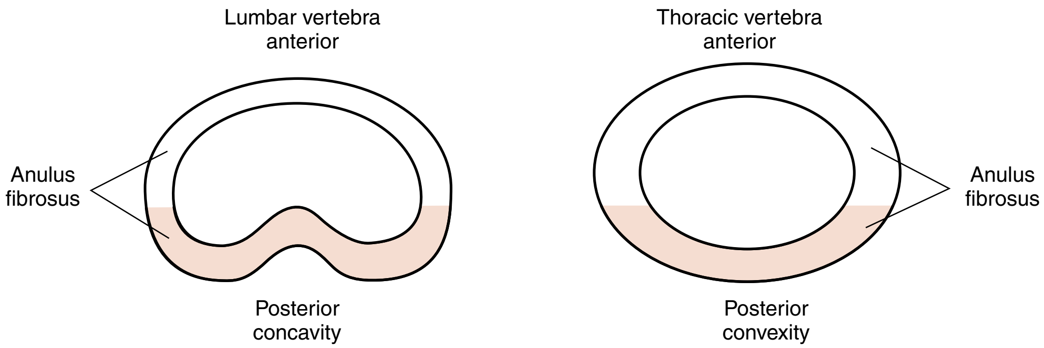


Figure 4–41 Lumbar intervertebral discs are concave posteriorly, which provides a greater portion of anulus fibrosus located posteriorly. This means more anulus fibrosus is available to resist the posterior stretch that occurs in flexion. *(From Bogduk, N: Clinical Anatomy of the Lumbar Spine and Sacrum (ed. 3), 1997, with permission from Elsevier.)*

meniscoid structures. The joint capsules are more lax than those in the thoracic region but more taut than those in the cervical region. The dorsal capsule has been demonstrated to be fibrocartilaginous, which suggests that this portion of the capsule is subject to compressive as well as tensile forces.[32]

In a newborn, the zygapophyseal joints in the lumbar region lie predominantly in the frontal plane in the presence of lumbar kyphosis. As the child develops and assumes an upright posture, the curve of the lumbar region changes to lordosis, and the orientation of the zygapophyseal joints changes as well. The orientations of the adult lumbar zygapophyseal joints display great variability both between individuals and within individuals; however, the majority of these joints have a curved structure that is biplanar in orientation. The anterior aspect of each joint remains in the frontal plane, and the posterior aspect lies close to or in the sagittal plane (Fig. 4–42). The degrees to which this happens vary. The frontal plane orientation provides resistance to the anterior shear that is present in the lordotic lumbar region. The sagittal plane orientation allows a great range of flexion and extension motion and provides resistance to rotation.

CASE APPLICATION

Variations in Zygapophyseal Joints

case 4–4

The shape of an individual's lumbar zygapophyseal joints may be a factor that predisposes some people to injury and protects others. For example, it may be that Malik has lumbar zygapophyseal joints at the L5/S1 segment that are oriented entirely in the sagittal plane, and that therefore offer little bony resistance to anterior shear forces.

Ligaments and Fascia

The majority of the ligaments associated with the lumbar region are the same ligaments described previously (ligamentum flavum, posterior longitudinal ligament, anterior longitudinal ligament, interspinous and supraspinous ligaments, and joint capsules). However, a few of these ligaments have variations specific to the lumbar region and need to be mentioned here before the iliolumbar ligaments and the thoracolumbar fascia are introduced.

The supraspinous ligament is well developed only in the upper lumbar region and may terminate at L3, although the most common termination site appears to be at L4. The ligament is almost always absent at L5/S1. The deep layer of the supraspinous ligament is reinforced by tendinous fibers of the multifidus muscle. The middle fibers of the supraspinous ligament blend with the dorsal layer of the thoracolumbar fascia. The intertransverse ligaments are not true ligaments in the lumbar area and are replaced by the iliolumbar ligament at L5.[84] The posterior longitudinal ligament is only a thin ribbon in the lumbar region, whereas the ligamentum flavum is thickened here.[87] In a study of 132 lumbar spine ligaments, Pintar and associates found that the interspinous ligament had the least overall stiffness and the joint capsules the highest. The anterior longitudinal ligament is strong and well developed in this region.[87]

Iliolumbar Ligaments

The iliolumbar ligaments consist of a series of bands that extend from the tips and borders of the transverse processes of L5 to attach bilaterally on the iliac crests of the pelvis (Fig. 4–43). There are three primary bands: the ventral (or anterior) band, which runs from the ventral caudal aspect of the transverse process of L5 to the ventral surface of the iliac crest; the dorsal (or posterior) band, which runs from the

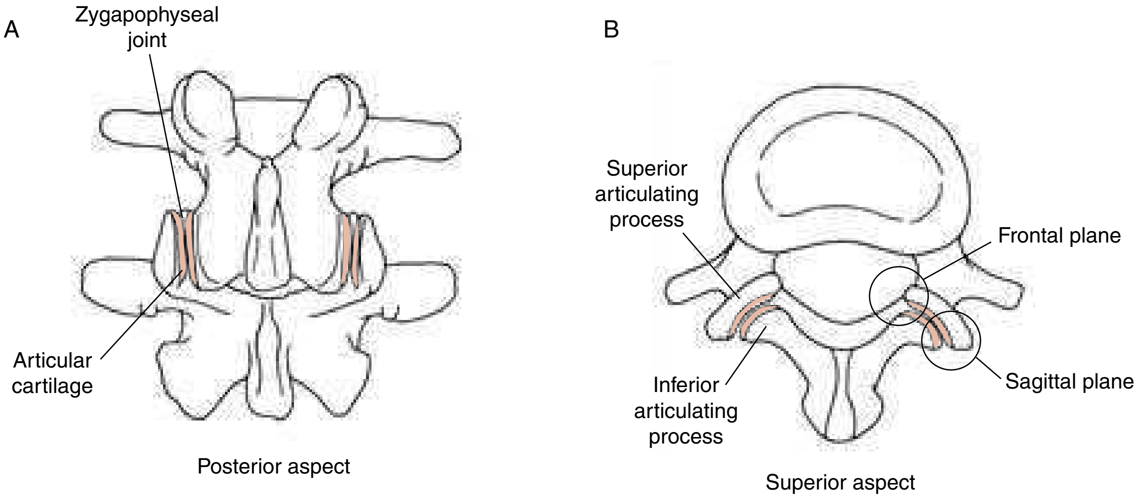


Figure 4–42 The biplanar orientation of the lumbar zygapophyseal joints. The posterior view **(A)** demonstrates the predominant sagittal plane orientation; however, a superior view **(B)** with the vertebral body of the cranial vertebra removed demonstrates the biplanar orientation. *(From Bogduk, N: Clinical Anatomy of the Lumbar Spine and Sacrum (ed. 3), 1997, with permission from Elsevier.)*

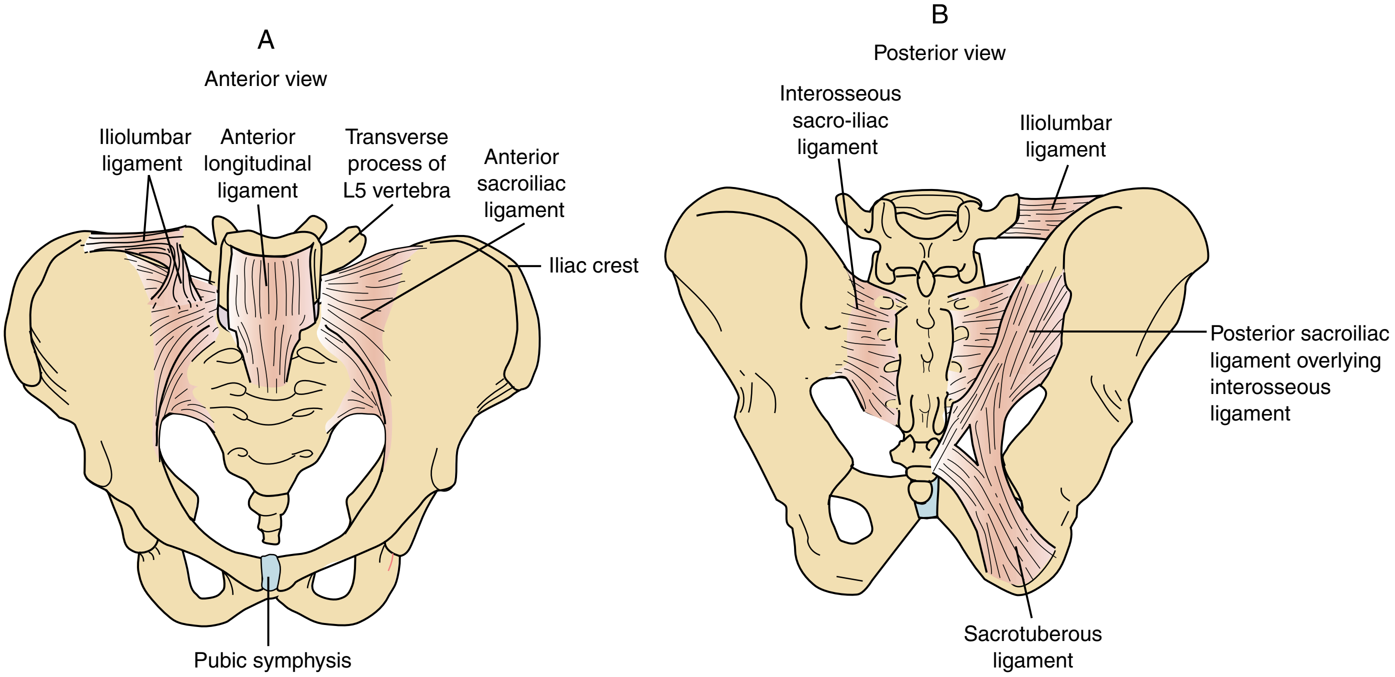


Figure 4–43 The iliolumbar ligaments. **A**. Anterior view. **B**. Posterior view.

tip of the transverse process of L5 to the more cranial part of the iliac crest; and the sacral band (sometimes called the lumbosacral ligament), which runs from the ventral aspect of the transverse process of L5 to the ala of the sacrum and the anterior sacroiliac ligaments.[88] The iliolumbar ligaments as a whole are very strong and play a significant role in stabilizing the fifth lumbar vertebra (preventing the vertebra from anterior displacement) and in resisting flexion, extension, axial rotation, and lateral bending of L5 on S1.[84,89–91] The iliolumbar ligaments are maximally loaded at the lumbosacral junction in the absence of muscle protection against flexion—for instance, when sitting in a relaxed, slouched posture. These ligaments can easily be a source of low back pain when subjected to creep in this position.[92]

Thoracolumbar Fascia

The thoracolumbar fascia (also called the lumbodorsal fascia) consists of three layers: the posterior, middle, and anterior (Fig. 4–44). The posterior layer is large, thick, and fibrous and arises from the spinous processes and supraspinous ligaments of the thoracic, lumbar, and sacral spines. The posterior layer gives rise to the latissimus dorsi cranially and is continuous cranially with the inferior tendinous border of the splenius capitis and lower fibers of the splenius cervicis.[93] The posterior layer travels caudally to the sacrum and ilium and blends with the fascia of the contralateral gluteus maximus. Deep fibers are continuous with the sacrotuberous ligament and connected to the posterior superior iliac spines, iliac crests, and long posterior sacroiliac ligament.[94] The posterior layer also travels laterally over the erector spinae muscles and forms the lateral raphe at the lateral aspect of the erector spinae. The internal abdominal oblique and the transversus abdominal muscles arise from the lateral raphe. The posterior layer becomes the middle layer and travels medially again along the anterior surface of the erector spinae and attaches back to the transverse processes and intertransverse ligaments of the lumbar spine. These two layers completely surround the lumbar extensor muscle group. The anterior layer of the thoracolumbar fascia is derived from the fascia of the quadratus lumborum muscle, where it joins the middle layer, inserts into the transverse processes of the lumbar spine, and blends with the intertransverse ligaments.[84]

McGill described the fascia as a stabilizing corset that forms a "hoop" around the abdomen along the abdominal muscles and their fascia[26] (see Fig. 4–44). Barker and colleagues support McGill's notion that the thoracolumbar fascia acts as a stabilizing corset by demonstrating that the fascia increases spinal stiffness.[95] Gracovetsky designated the anterior layer of the thoracolumbar fascia as the "passive part" and the posterior layer as the "active part."[96] According to Gracovetsky, the passive part serves to transmit tension produced by a contraction of the hip extensors to the spinous processes. The active portion is activated by a contraction of the transversus abdominis muscle, which tightens the fascia. The fascia transmits tension longitudinally to the tips of the spinous processes of L1/L4 and may help the spinal extensor muscles to resist an applied load.[96] Vleeming found that both the gluteus maximus and contralateral latissimus dorsi tensed the superficial layer and provided a pathway for the mechanical transmission of forces between the pelvis and the trunk.[94]

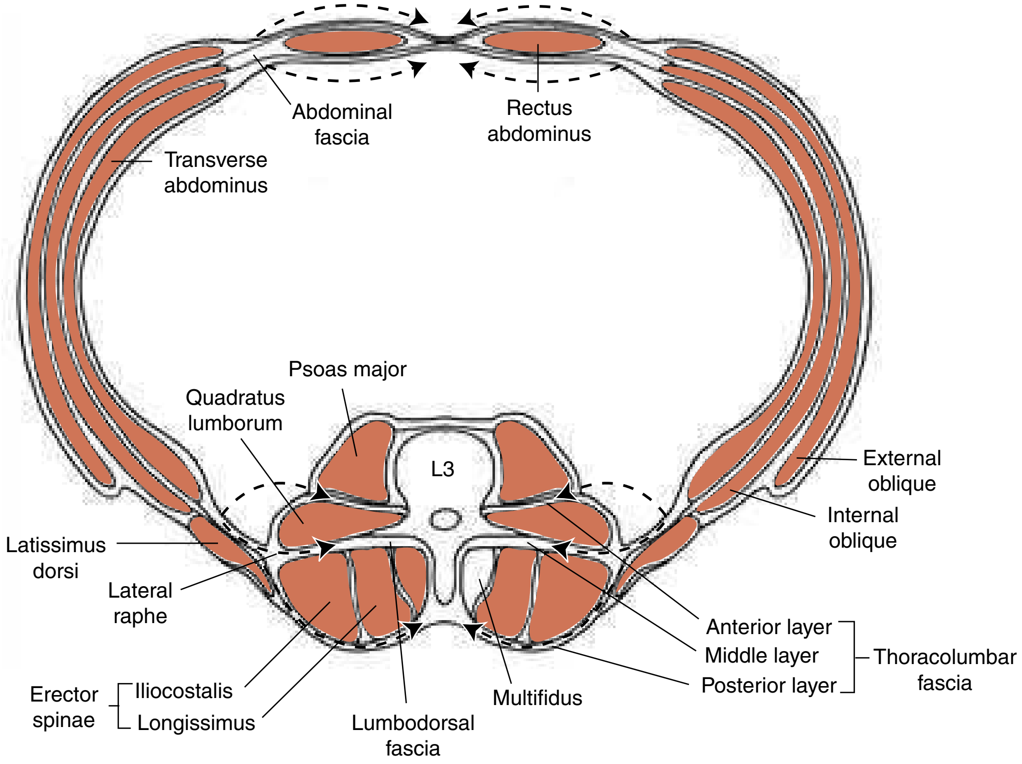


Figure 4–44 A superior view of a cross-section to identify the abdominal hoop. The anterior, middle, and posterior layers of the thoracolumbar fascia, along with the abdominal fascia, are the passive parts. The muscles are the active parts, which pull (dashed arrows) on the fascia to tighten the hoop.

CASE APPLICATION

Soft Tissue Structures as Possible Source of Pain

case 4–5

Given the tasks that Malik performs daily, he is continually experiencing large anterior shear forces. The iliolumbar ligaments, the posterior anulus fibrosus, the posterior longitudinal ligament, and the joint capsules are being subjected to stresses, which could lead to the failure of some or all of these structures. Each of these structures is innervated and may be a source of his pain.

Function of the Lumbar Region

Kinematics

The lumbar region is capable of movement in flexion, extension, lateral flexion, and rotation. The lumbar zygapophyseal facets favor flexion and extension because of the predominant sagittal plane orientation (see Fig. 4–18A). The amount of flexion varies at each interspace of the lumbar vertebrae, but most of the flexion takes place at the lumbosacral joint.[97,98] Lateral flexion and rotation are most free in the upper lumbar region and progressively diminish in the lower region. The largest lateral flexion range of motion and axial rotation occurs between L2 and L3.[98] Motion in this region is more limited because of the shape of the zygapophyseal joints (Fig. 4–45). The effectiveness of the zygapophyseal joints in resisting axial rotation depends on the extent that the superior facets face medially (in the sagittal plane). When the medial orientation of the joint surfaces is greater, the resistance to axial rotation is greater. The total amount of lumbar motion from a neutral lordotic position is reported to be as follows: flexion: 52° (SD = 9°), extension: 19° (SD = 9°), lateral flexion: 30° to each side (SD = 6°), and rotation: 32° to each side (+/–12°).[99]

In the lumbar region, coupled motions usually occur with lateral flexion and axial rotation. The pattern of coupled motion, however appears to be inconsistent. Legaspi and Edmond recently performed a critical review to determine if a consistent pattern of coupling exists in the literature. Of the 24 articles reviewed, there was little agreement about the specific characteristics of coupled motion in the lumbar spine.[36]

Nelson and coworkers studied lumbar-pelvic motion in 30 healthy women, age 19 to 35, who lifted and replaced a 9.5-kg weight on the floor. They found that lumbar and pelvic motion were variable among these individuals and tended to occur simultaneously during trunk flexion and more sequentially during trunk extension.[101] The use of a weight may have affected the lumbar-pelvic rhythm, but this study raises questions about exactly when and how trunk and pelvic motion occurs. McGill reported that he and his colleagues had never seen this strict sequence described by Calliet in any of the vast number of studies that they had done.[26]

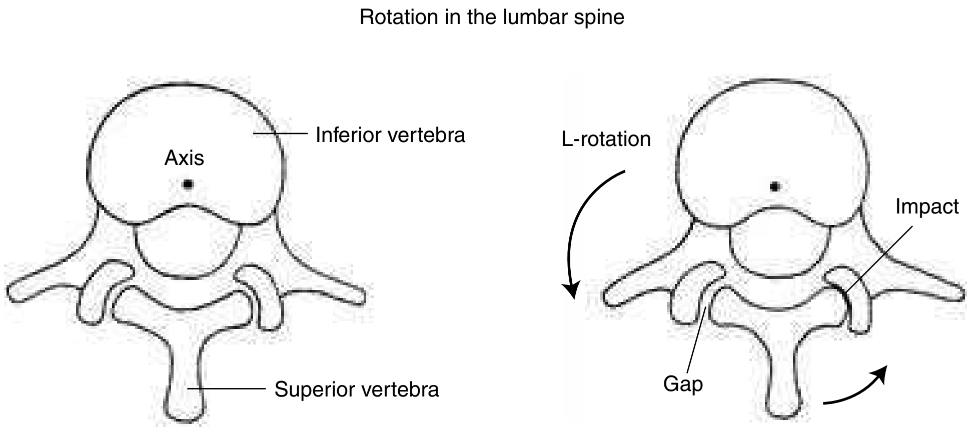


Figure 4–45 Zygapophyseal mechanics in the rotation of the lumbar vertebra. The sagittal plane orientation provides resistance to rotation. With left rotation, the right zygapophyseal joint will abut, limiting the ROM, and the left zygapophyseal joint will leave a gap.

Continuing Exploration 4-3:

Lumbar-Pelvic Rhythm

Cailliet described a specific instance of coordinated, simultaneous activity of lumbar flexion and anterior tilting of the pelvis in the sagittal plane during trunk flexion and extension. He called the combined lumbar and pelvic motion lumbar-pelvic rhythm. The activity of bending over to touch one's toes with knees straight depends on lumbar-pelvic rhythm.[100] According to Cailliet, the first part of bending forward consists of lumbosacral flexion (Fig. 4–46A), followed by anterior tilting of the pelvis at the hip joints (Fig. 4–46B). A return to the erect posture is initiated by posterior tilting of the pelvis at the hips, followed by extension of the lumbar spine. The initial pelvic motion delays lumbar extension until the trunk is raised far enough to shorten the moment arm of the external load, thus reducing the load on the erector spinae.

There is no argument, however, that the integration of motion of the pelvis about the hip joints with motion of the vertebral column not only increases the motion available to the total column but also reduces the amount of flexibility required of the lumbar region. Hip motion may even, as McGill suggested, eliminate the need for full lumbar flexion, which would serve a protective function by protecting the anulus fibrosus and posterior ligaments from being fully lengthened.[26]

The contribution to motion from multiple areas to produce a larger range of motion than could be accomplished by a single area is similar to what is found at the shoulder in scapulohumeral rhythm. A restriction of motion at either the lumbar spine or at the hip joints may disturb the rhythm and prevent a person from reaching the toes. Restriction of motion at one segment also may result in hypermobility of the unrestricted segment.

Kinetics

Compression

One of the primary functions of the lumbar region is to provide support for the weight of the upper part of the body in static as well as dynamic situations. The increased size of the lumbar vertebral bodies and discs in comparison with their counterparts in the other regions helps the lumbar structures support the additional weight. The lumbar region must also withstand the tremendous compressive loads produced by muscle contraction. Experimental

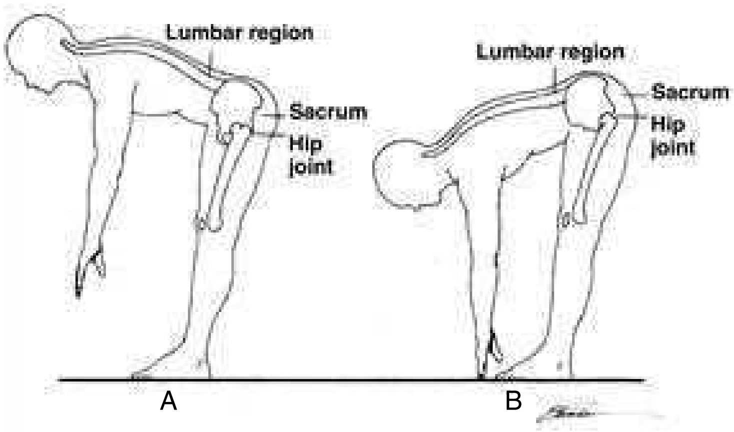


Figure 4–46 Lumbar-pelvic rhythm. The lumbar spine flexes **(A)** and the pelvis rotates anteriorly **(B)** in the sagittal plane.

testing of 10 cadaver spines subjected to 1,000-N compressive loading demonstrated that the lumbar interbody joints shared 80% of the load and the zygapophyseal facet joints in axial compression shared 20% of the total load.[102] This percentage can change with altered mechanics: With increased extension or lordosis, the zygapophyseal joints will assume more of the compressive load. Also, with degeneration of the intervertebral disc, the zygapophyseal joints will assume increased compressive load.

Khoo and colleagues compared lumbosacral loads (ground reaction forces and accelerations, plus forces generated by erector spinae and rectus abdominis muscle groups) at the center of the L5/S1 joint in static versus dynamic situations in 10 men. Lumbosacral loads in the erect standing posture were in the range of 0.82 to 1.18 times body weight, while lumbosacral loads during level walking were in the range of 1.41 to 2.07 times body weight, an increase of 56.3%.[103]

Changes in the position of the body will change the location of the body's line of gravity and thus change the forces acting on the lumbar spine. See Chapter 13 for a discussion of compressive loads on the lumbar spine with different positions.

Shear

In the upright standing position, the lumbar segments are subjected to anterior shear forces caused by the lordotic position, the body weight, and ground reaction forces (see Fig. 4–39). This anterior shear or translation of the vertebra is resisted by the direct impact of the inferior zygapophyseal facets of the cranial vertebra against the superior zygapophyseal facets of the subjacent (caudal) vertebra. The effectiveness of the zygapophyseal joint in providing resistance to anterior translation depends on the extent to which the caudal vertebra's superior facets lie in the frontal plane and face posteriorly. The more that the superior zygapophyseal facets of the caudal vertebra face posteriorly, the greater the resistance they are able to provide to forward displacement, because the posteriorly facing facets lock against the inferior facets of the cranial vertebra.

Concept Cornerstone **4-3**

Variations in Zygapophyseal Joints

The shape of the zygapophyseal joints, the zygapophyseal joint capsules, the fibers of the anulus fibrosus, and the iliolumbar ligaments in the lower segments provide structural resistance to anterior shear forces in the lumbar segments. Individual variation in joint structure, therefore, can be a contributing factor to pain in this region. If an individual has zygapophyseal joints oriented totally in the sagittal plane, the capsuloligamentous structures will be taxed, and eventually they may become lengthened and turn into a source of pain, because they are innervated structures. Even if an individual has zygapophyseal joints with a biplanar orientation, excessive anterior shear forces can cause damage. In this case, in addition to the lengthened capsuloligamentous structures, the zygapophyseal joints themselves can experience excessive compression in the anterior regions and produce pain. Fortunately, there is a dynamic restraint to anterior shear, the deep erector spinae muscles, which will be discussed later in the chapter.

CASE APPLICATION

Excessive Anterior Shear Forces

case 4–6

The shape of Malik's zygapophyseal joints may or may not be known from diagnostic tests. Regardless, it is likely that these joints have been under repetitive stress because of his job tasks. Excessive anterior shear forces may produce pain due to microtrauma of the zygapophyseal joint capsules and the ligaments, compression of the fibroadipose meniscoids, or degenerative changes to the joints. In any case, exercises to maximize the ability of the deep erector spinae to control the excessive anterior shear forces will be important to Malik's rehabilitation. In the meantime, changing Malik's activity to minimize the anterior shear forces that he encounters will likely decrease his symptoms. If his activity cannot be changed sufficiently, a lumbosacral brace or corset may be considered to provide proprioceptive input. This may help him maintain a more neutral posture and thereby decrease some of the anterior shear forces.

Structure of the Sacral Region

Five sacral vertebrae are fused to form the triangular or wedge-shaped structure that is called the sacrum. The base of the triangle, which is formed by the first sacral vertebra, supports two articular facets that face posteriorly for articulation with the inferior facets of the fifth lumbar vertebra. The apex of the triangle, formed by the fifth sacral vertebra, articulates with the coccyx. The lateral part of the sacrum, or ala, bears an auricular (ear-shaped) surface that articulates with the ilium. The ala also bears the sacral tuberosity that is posterior to the auricular surface and articulates with the iliac tuberosity.

Sacroiliac Articulations

The **sacroiliac joints** are weight-bearing, compound joints consisting of an anterior synovial joint between the auricular surfaces of the sacrum and ilium and a posterior syndesmosis between the tuberosities of the same bones[25] (Fig. 4–47). The role of the sacroiliac joints appears to be to relieve the stress on the pelvic ring imparted by movement of the trunk and lower limbs, muscle contraction, the weight of the body, and ground reaction forces. The joints must allow movement to absorb the twisting forces ascending from the lower limb yet be stable enough to

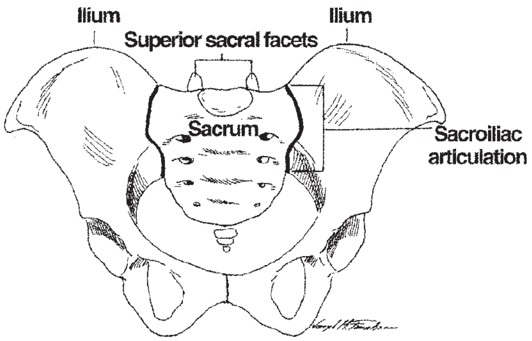


Figure 4–47 The sacroiliac joints consist of the articulations between the first three sacral segments and the two ilia of the pelvis.

transmit the forces from the vertebral column descending to the lower limbs.[2]

Articulating Surfaces on the Sacrum

The auricular (ear-shaped) articulating surfaces on the sacrum are located on the sides of the fused sacral vertebrae lateral to the sacral foramina from S1 to S3.[104] The articulating surfaces are multiplanar and curvy (Fig. 4–48A). The fetal and prepubertal surfaces are flat and smooth, while the postpubertal surfaces are marked by a central groove or surface depression that extends the length of the articulating surfaces.[104,105] Also, the articular surfaces are covered with hyaline cartilage. The overall mean thickness of the sacral cartilage which ranges from 1 to 3 mm is greater than that of the iliac cartilage.[106–109] The tuberosity is an irregular bony surface posterior to the auricular surface that articulates with the tuberosity of the ilium.

Articulating Surfaces on the Ilia

The auricular articulating surfaces on the ilia are covered with hyaline cartilage, although the hyaline of the ilia contains a denser aggregate of collagen fibers than that of the sacrum and is less than 1 mm thick.[104,110–112] The articular surfaces of the ilia are also multiplanar and curvy to match those of the sacrum (Fig. 4–48B). The fetal and prepubertal articular faces of the ilia are flat and smooth, while the postpubertal surfaces develop a central ridge that extends the length of the articulating surface and corresponds to the grooves on the sacral articulating surfaces.[106,113] The tuberosity of the ilia is an irregular bony surface posterior and superior to the auricular surface that articulates with the tuberosity of the sacrum via the sacroiliac interosseous ligament.

Sacroiliac Ligaments

The anterior, interosseous, and posterior (or dorsal) sacroiliac ligaments are directly associated with the sacroiliac joints. A separate portion of the posterior sacroiliac ligament is called either the long posterior sacroiliac ligament[27] or the long dorsal sacroiliac ligament.[114] The **iliolumbar ligaments,** which connect the fifth lumbar vertebra to the ilium, the sacrospinous ligaments, and the sacrotuberous ligaments, which connect the sacrum to the ischium, are indirectly associated with the sacroiliac joints. The iliolumbar ligaments were described previously in the lumbar region. The sacroiliac ligaments are reinforced by fibrous expansions from the quadratus lumborum, erector spinae, gluteus maximus, gluteus minimus, piriformis, and iliacus muscles, which contribute to the joints' stability. The fascial support is greater posteriorly than anteriorly because more muscles are located posteriorly.[105]

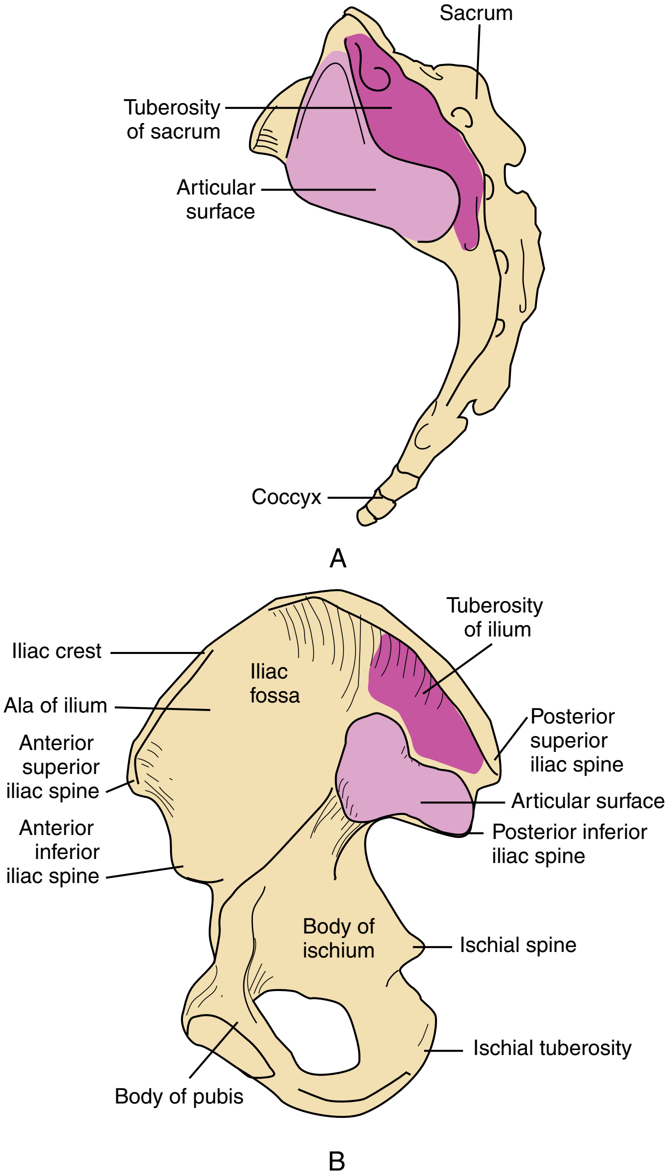


Figure 4–48 Articulating surfaces of the sacrum and ilium. **A.** Lateral view of sacrum. **B.** Medial view of the ilium.

The **anterior sacroiliac ligaments** (Fig. 4–49A) are considered to be capsular ligaments because of their intimate connections to the anteroinferior margins of the joint capsules.[27] According to Bogduk, the anterior sacroiliac ligaments cover the anterior aspects of the sacroiliac joints and join the ilia to the sacrum.[84]

The paired **long posterior** (or **dorsal**) **sacroiliac ligaments** (Fig. 4–49B) have superior attachments to the posterior superior iliac spines and adjacent parts of the ilium. Inferiorly, the ligaments are attached to the lateral crest of the third and fourth sacral segments. The medial fibers are connected to the deep lamina of the posterior layer of the thoracolumbar fascia and the aponeurosis of the erector spinae.[114]

The **sacrospinous ligaments** connect the ischial spines to the lateral borders of the sacrum and coccyx. The **sacrotuberous ligaments** connect the ischial tuberosities to the posterior spines at the ilia and the lateral sacrum and coccyx.

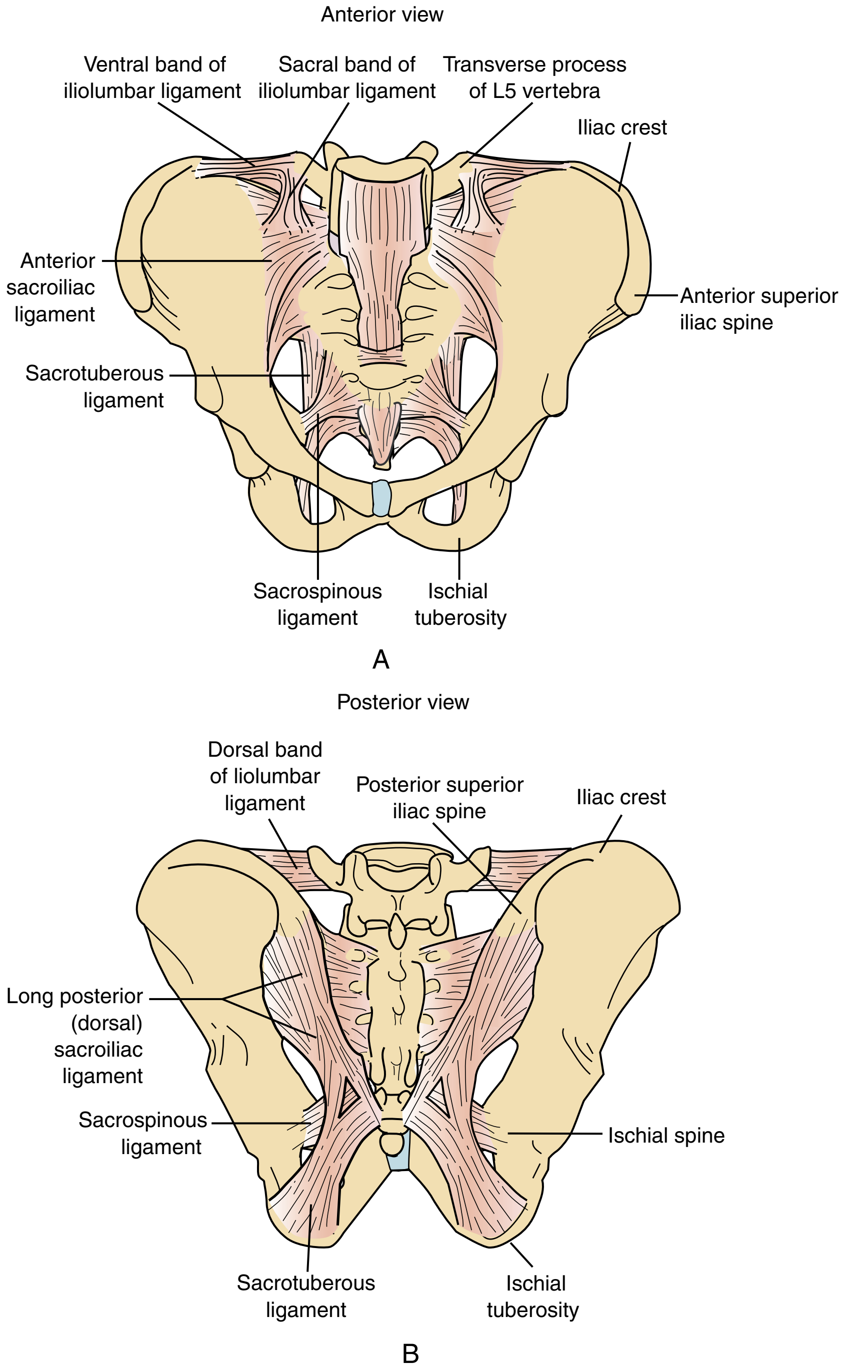


Figure 4–49 The sacroiliac ligaments. **A.** Anterior view. **B.** Posterior view.

The sacrospinous ligament forms the inferior border of the greater sciatic notch; the sacrotuberous ligament forms the inferior border of the lesser sciatic notch.[115,116]

The **interosseous sacroiliac ligaments** (Fig. 4–50), which constitute the major bonds between the sacrum and the ilia, are considered to be the most important ligaments directly associated with the sacroiliac joints.[27,84] These ligaments create the more posterior syndesmoses between the tuberosities of the sacrum and ilia.

Symphysis Pubis Articulation

The symphysis pubis is a cartilaginous joint located between the two ends of the pubic bones. The end of each pubic bone is covered with a layer of articular cartilage, and the joint is formed by a fibrocartilaginous disc that joins the hyaline cartilage–covered ends of the bones. The disc has a thin central cleft,[1] which in women may extend throughout the length of the disc.[117] The three ligaments that are associated with the joint are the **superior pubic ligament,** the **inferior pubic ligament,** and the **posterior ligament.**[1] The superior ligament is a thick and dense fibrous band that attaches to the pubic crests and tubercles and helps support the superior aspect of the joint. The inferior ligament arches from the inferior rami on one side of the joint to the inferior portion of the rami on the other side and thus reinforces the inferior aspect of the joint. The posterior ligament consists of a fibrous membrane that is continuous with the periosteum of the pubic bones.[1] The anterior portion of the joint is reinforced by aponeurotic expansions from a number of muscles that cross the joint (Fig. 4–51). Kapandji described the muscle expansions as forming an anterior ligament consisting of expansions of the transversus abdominis, rectus abdominis, internal obliquus abdominis, and adductor longus.[1]

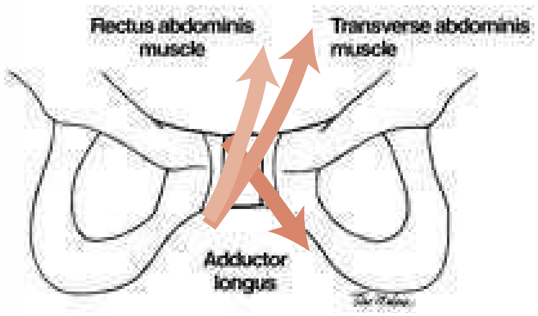


Figure 4–51 The aponeurotic extensions of the muscles crossing the anterior aspect of the symphysis pubis.

Function of the Sacral Region

Kinematics

The kinematics of the sacroiliac joint are complex and the source of considerable controversy, including the amount and type of motion available and the axes of motion. Over the years, a variety of methods to measure motion at the sacroiliac joints have been employed, which, in itself, is another source of controversy. For example, two-dimensional measurements, such as those done via radiographs and inclinometers, have been criticized for not capturing the three-dimensional nature of movement at the sacroiliac joints.[118] Three-dimensional analyses of motion using mathematical modeling, computerized modeling, and/or skin markers have also been questioned for validity.[119] Three-dimensional measures utilizing Roentgen Stereophotogrammetric Analysis (RSA) have been used for measuring small amounts of joint motion. More recent studies of sacroiliac joint kinematics have used this technique. Sturesson and colleagues have done several studies[120–122] using this technique and have examined motion at the sacroiliac joints with several

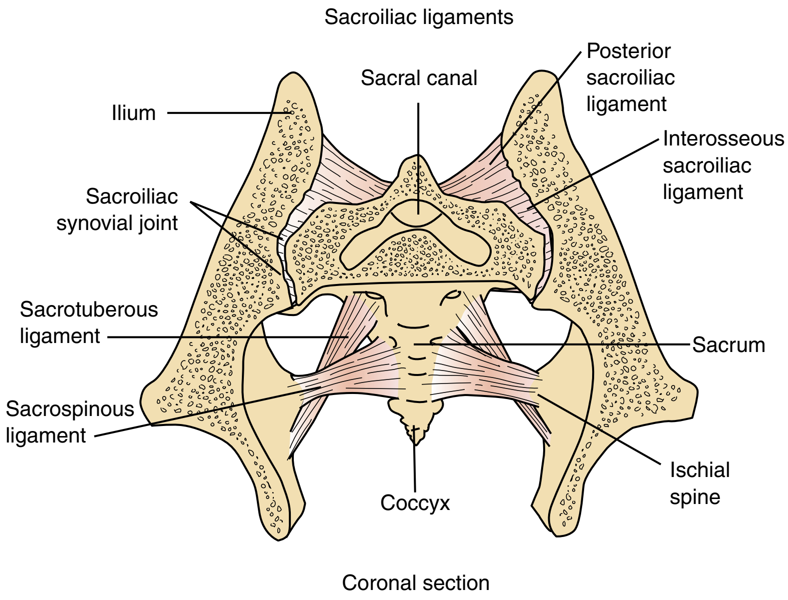


Figure 4–50 The sacroiliac ligaments in coronal section.

movements, including moving from prone to sitting,[120] performing the standing hip flexion test,[121] and moving into reciprocal straddle positions of the hip joints.[122] They found the mean amount of rotation at the sacroiliac joints to be 2.5°, less than 1°, and 2°, respectively, and the mean amount of translation to be 0.7 mm when moving from prone to sitting and 0.3 mm when performing the standing hip flexion test.[120–122] The amount of translation was not reported with movement into reciprocal straddle positions of the hips. A recent systematic review of the literature of all types of three-dimensional motion testing of the sacroiliac joints concluded: "motion at the sacroiliac joint is limited to minute amounts of rotation and of translation that may be sub-clinically detectable."[119]

In summary, it can be concluded that the sacroiliac joints do permit motion, but only in extremely small amounts. Although small in quantity, this motion is important for attenuating the forces transmitted via the pelvic ring to prevent sacral fracture.[2]

The motion that does occur at the sacroiliac joints is typically described as **symmetrical** (or **agonistic**) or **asymmetrical (antagonistic).**[123] Symmetrical motion is the movement of both innominate bones together, as a unit, in the same direction in relation to the sacrum. Asymmetrical motion is movement of the innominate bones in opposite directions. Because of the closed kinetic chain with the pubic symphysis, any asymmetrical motion at the sacroiliac joints results in motion at the pubic symphysis as well.[123]

Symmetrical motion at the sacroiliac joints can be described by naming the motion according to the sacral movement or according to the innominate movement. **Nutation** is the term commonly used to refer to movement of the sacrum whereby the sacral promontory moves anteriorly and inferiorly while the sacral apex moves posteriorly and superiorly (Fig. 4–52A). During the closed-chain task of forward bending from a standing position, for example, the sacrum will nutate on the two innominate bones immediately following flexion of the lumbosacral junction. **Counternutation** refers to the opposite movement, in which the sacral promontory moves posteriorly and superiorly while the sacral apex moves anteriorly (Fig. 4–52B). **Anterior pelvic tilt** is the term used to refer to the symmetrical movement of the innominate bones whereby the anterior superior iliac spines and the pubic symphysis move inferiorly (see Fig. 4–52B). During the open-chain task of bilateral hip extension in prone, for example, anterior pelvic tilt of the two innominate bones will occur on the sacrum immediately following maximum hip extension. **Posterior pelvic tilt** is the term commonly used to refer to the symmetrical movement of the innominate bones whereby the anterior superior iliac spines and the pubic symphysis move superiorly (see Fig. 4–52A).

Confusion regarding the description of sacroiliac motion is common as the methods of description are often misused. It is important to be clear about which bones are moving in order to avoid confusion. Take, for example, the task of bilateral hip extension in prone described above. Some describe the sacroiliac joint motion during this task as counternutation of the sacrum. What they are really describing, however, is the position of the sacrum in relation to the innominates, not the motion that is occurring; this is commonly described as "relative motion." The innominates moved into anterior pelvic tilt on the sacrum. If the position of the sacrum is described in relation to the innominates, it is called counternutation. The confusion when using this type of description is further compounded when considering motion that continues up the chain. Immediately following the anterior pelvic tilt of the innominates on the sacrum, extension will occur at the lumbosacral junction; it is movement of the sacrum on L5 that produces that extension. If the distinction between motion and relative position isn't clear to someone, he or she would conclude that the sacrum is moving into counternutation and would then be unable to conceptualize the lumbosacral extension.

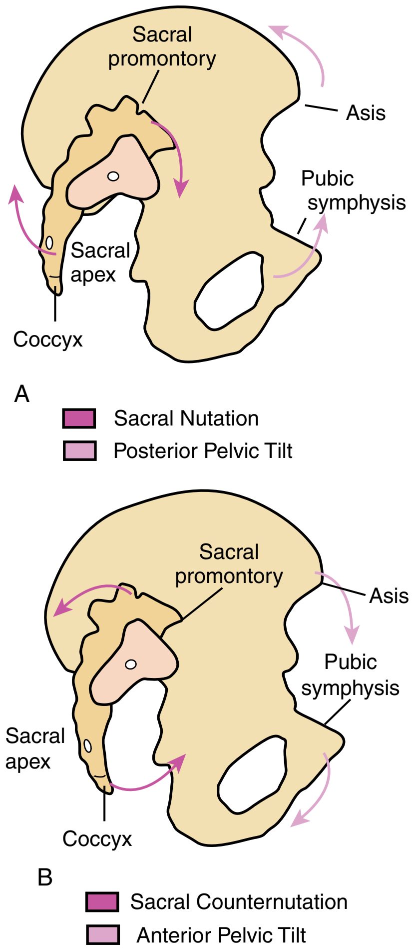


Figure 4–52 **A.** Sacral nutation/ posterior pelvic tilt. Nutation: The sacral promontory moves anteriorly and inferiorly while the sacral apex moves posteriorly and superiorly. Posterior pelvic tilt: The anterior superior iliac spine (ASIS) and the pubic symphysis move superiorly. **B.** Sacral counternutation/anterior pelvic tilt. Counternutation: The sacral promontory moves posteriorly and superiorly, and the sacral apex moves anteriorly. Anterior pelvic tilt: The ASIS and the pubic symphysis move inferiorly.

Asymmetrical motion at the sacroiliac joints is described as **pelvic torsion** of the innominates on the sacrum. Asymmetrical motion occurs during asymmetrical tasks, such as ambulation, in which one hip is in a flexed position while the opposite hip is in an extended position.[123] The innominate on the side of the hip flexion will be torsioned in a posterior direction while the innominate on the side of the hip extension will be torsioned in an anterior direction.[118,124] Pelvic torsions are always accompanied by motion at the pubic symphysis.[123]

Kinetics

Stability of the sacroiliac joints is extremely important because these joints must support a large portion of the body weight. In normal erect posture, the weight of the head, arms, and trunk is transmitted through the fifth lumbar vertebra and lumbosacral disc to the first sacral segment. The force of the body weight creates a nutation torque on the sacrum. Concomitantly, the ground reaction force creates a posterior tilt of the innominates. The countertorques of nutation of the sacrum and posterior tilt of the innominates are supported by the ligamentous tension and fibrous expansions from adjacent muscles, which reinforce the joint capsules and blend with the ligaments.[106]

In one study, Pool-Goudzwaard and colleagues investigated the role that the iliolumbar ligaments play in stabilizing the sacroiliac joints.[88] The authors demonstrated that the iliolumbar ligaments have a significant role in stabilizing the sacroiliac joints as well as the lumbosacral junction. The ventral band of the iliolumbar ligament is of particular importance in restricting sagittal plane sacroiliac joint mobility.

Also, tension developed in the sacrotuberous, sacrospinous, and anterior sacroiliac ligaments counteracts the nutation torques of the sacrum, although the sacrotuberous and sacrospinous ligaments have not been found to play a major role in pelvic stability.[125] However, the sacrotuberous and interosseous ligaments compress the sacroiliac joint during nutation.[114] The long posterior (or dorsal) sacroiliac ligament is under tension in counternutation and relaxed in nutation.[126] The interosseous sacroiliac ligament binds the ilia to the sacrum.[84]

The complementary ridges and depressions that develop in the cartilage of the sacral and ilial surfaces also contribute to the stability of the joint. Vertical load-bearing is facilitated by the development of these ridges and depressions, but motion is limited by them.[106,116,127,128]

Shearing forces are created at the sacroiliac joints and symphysis pubis during single-leg-support activities as a result of lateral pelvic tilting.[121] Under normal circumstances, the joints are capable of resisting the shearing forces, and no appreciable motion occurs. If, however, the pubic symphysis is dislocated, the pelvis becomes unstable during gait, with increased stress on the sacroiliac and hip joints as well as the vertebral column.

MUSCLES OF THE VERTEBRAL COLUMN

The Craniocervical/Upper Thoracic Regions

The muscles of the craniocervical region serve two primary roles: to hold the head upright against gravity and to infinitely position the head in space in order to optimally position the sensory organs. The muscles of the cervicothoracic region also serve two primary roles: again, to position the head and neck in space, and to stabilize the head and neck to allow and produce movement of the scapula. The line of gravity in an upright standing position passes anteriorly to the axis of rotation in the cervical region, producing a flexion moment (Fig. 4–53). The posterior muscles, along with the ligamentous structures previously discussed, counter this flexion moment. The need to position the head for the special sensory organs often includes rapid, coordinated movements, such as when a loud noise is heard and there is rapid turning of the head to locate the source of the sound. The muscular structure and function is complex in order to serve the demands for such great amounts of motion and yet provide sufficient stability to protect the spinal cord and allow for use of the upper extremities.

Posterior Muscles

We will examine the muscles from superficial to deep and begin with the posterior muscles (Figs. 4–54 and 4–55). The **trapezius muscle** is the most superficial of the posterior muscles. The trapezius spans from the occiput to the lower thoracic spine and contains a prominent tendinous region over the cervicothoracic junction.[55] The trapezius belongs predominantly to the shoulder region; however, when the upper extremities are fixated, the trapezius can produce extension of the head and neck. Acting unilaterally, the upper trapezius can produce ipsilateral lateral flexion and contralateral rotation of the head and neck.

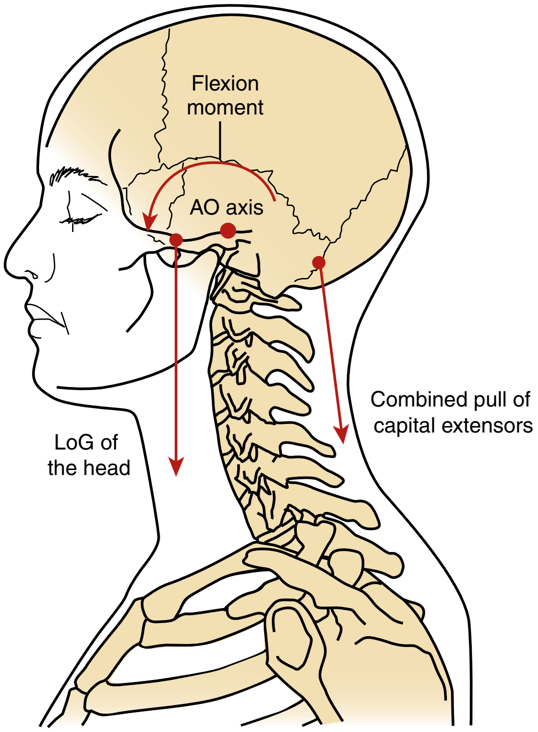


Figure 4–53 The line of gravity (LoG) in the cervical region passes anteriorly to the axis of rotation, producing a flexion moment. The extensor muscles must contract to counter this moment.

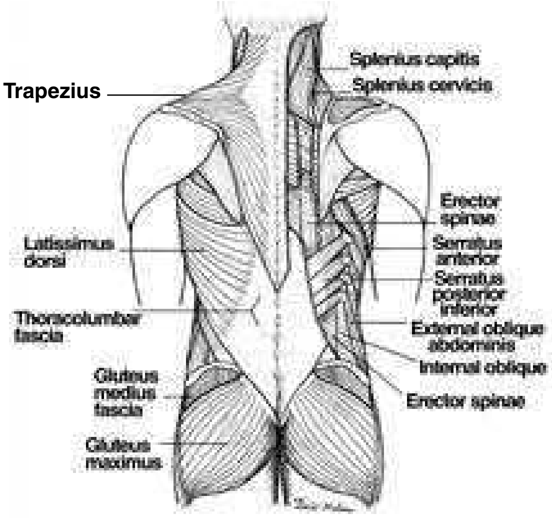


Figure 4–54 Posterior back muscles. The superficial muscles have been removed on the right side to show the erector spinae. The anterior layer of the thoracolumbar fascia is intact on the left side of the back.

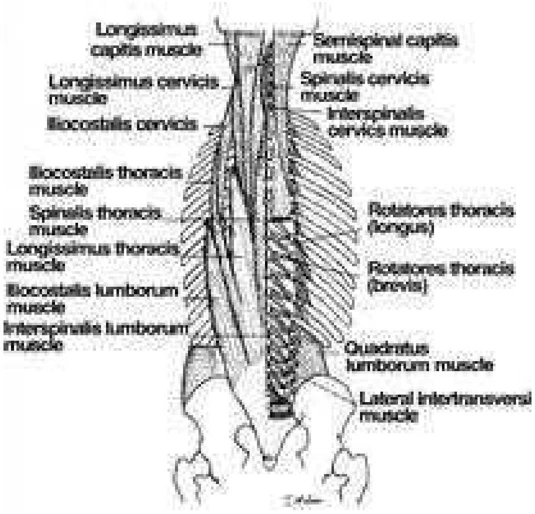


Figure 4–55 Erector spinae and deep back muscles. The erector spinae muscle has been removed from the right side of the neck to show the deep back muscles.

The **levator scapula** lies deep to the trapezius. It extends from its proximal attachments on the transverse processes of the first four cervical vertebrae to attach distally on the vertebral border of the scapula between the root of the spine and the superior angle. This muscle has a large cross-sectional area. The levator scapula is a scapular elevator and downward rotator when the neck is stable, but if the upper extremity is stabilized, it will produce ipsilateral lateral flexion and rotation of the cervical spine. In addition, the anterior inclination plays an important role in the mechanics of the cervical spine. The levator scapula is optimally aligned to produce a posterior shear force on the cervical spine.[55]

Porterfield and DeRosa likened the levator scapulae muscles to the deep erector spinae of the lumbopelvic area, which will be discussed later.[55] The cervical spine is subjected to constant anterior shear forces caused by gravity and the lordotic position of the spine in this region. The levator scapulae help resist these forces (Fig. 4–56). An increase in the cervical lordosis, as is often seen in excessive forward head posture, will further increase the anterior shear forces on the cervical vertebrae and may cause overactivity of the levator scapulae to resist these excessive anterior shear forces.

Continuing Exploration 4-4:

Treatment for Overactivity of the Levator Scapula Muscle

Porterfield and DeRosa have suggested that overactivity in the presence of a forward head posture may be the reason that so many people have pain and tenderness to palpation in the levator scapulae.[55] Conventional treatment will often involve stretching this strained muscle. They cautioned that stretching this muscle may actually worsen the situation and cause further irritation, because it will decrease the muscle's ability to control the anterior shear if it is overly lengthened. Rather, removal of the excessive anterior shear forces is necessary. In addition, the levator scapulae may need strength and endurance training.

The splenius capitis and splenius cervicis muscles lie deeper than the levator scapulae. The splenius muscles are large, flat muscles that have proximal attachments on the

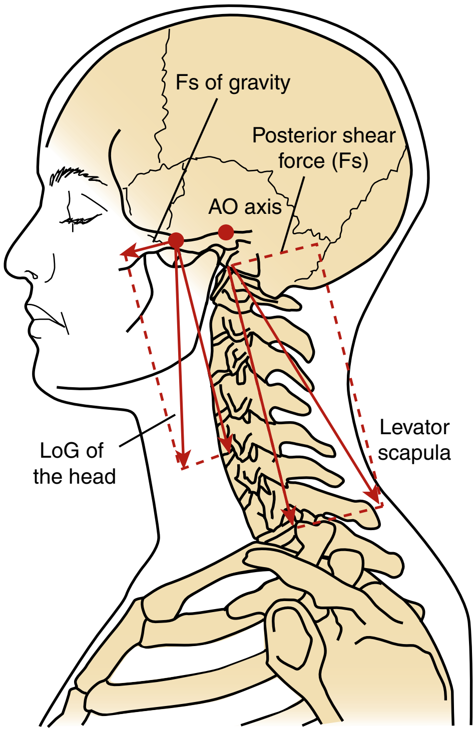


Figure 4–56 The cervical spine is subjected to anterior shear forces as a result of the lordosis and anterior line of gravity (LoG). The levator scapulae helps resist the anterior shear forces by producing posterior shear.

superior nuchal line mastoid process and the transverse processes of the upper first two or three cervical vertebrae and extend distally to attach to the spinous processes of the seventh cervical and first three to six thoracic vertebrae (see Fig. 4–54). These muscles serve as prime movers of the head and neck as a result of their large cross-sectional area and the large moment arm. They produce extension when working bilaterally and ipsilateral rotation when working unilaterally. However, these muscles show little electromyographic activity in normal stance.

The semispinalis capitis and semispinalis cervicis muscles are deeper than the splenius group. These muscles have the most optimal line of pull and a large moment arm to produce extension of the head and neck and an increase in the cervical lordosis. They run from the occiput to the cervical spinous processes (semispinalis capitis) and from the thoracic transverse processes to the articular processes of the lower cervical spine (semispinalis cervicis) (see Fig. 4–55). These muscles together form the cordlike bundle of muscles that can be palpated lateral to the cervical spinous processes. Their function is often likened to that of the multifidi in the lumbar region; like the multifidi in the lumbar regions, they have optimal alignment and moment arm for increasing the lordosis of the cervical spine (Fig. 4–57).[55] It is important to note that the greater occipital nerve pierces the semispinalis capitis muscle on its way to innervate the skull. This can be a site of nerve irritation and entrapment when the semispinalis capitis is overactive or shortened, as in a forward head posture. Occipital region headaches can result.

The longissimus capitis and longissimus cervicis are deeper than and lateral to the semispinalis group (see Fig. 4–55). Their deep position places them close to the axis of rotation for flexion and extension, rendering them ineffective extensors because of the small moment arm. They do, however, produce compression of the cervical segments. The lateral position allows them to produce ipsilateral lateral flexion when working unilaterally, and when working bilaterally, they serve as frontal plane stabilizers of the cervical spine.[55]

The suboccipital muscles are the deepest posterior muscles and consist of the rectus capitis posterior minor and major, inferior oblique, and superior oblique muscles. As a group, they run between the occiput and C2, allowing independent movement of the craniovertebral region on the lower cervical spine. Together, they produce occipital extension. Working alone, they produce ipsilateral rotation and lateral flexion. Given the small cross-sectional area of these muscles, some researchers have questioned their ability to generate force and produce movement; rather, they may serve primarily a proprioceptive role and produce small movements in order to fine-tune motion.[129]

Lateral Muscles

The scalene muscles are located on the lateral aspect of the cervical spine and serve as frontal plane stabilizers when acting as a group with the longissimus muscles posteriorly. In the sagittal plane, the anterior scalene muscles, which run from the first rib to the anterior tubercles of the transverse processes of C3 to C6, work with the levator scapulae to provide stability (Fig. 4–58). The anterior scalene muscles, when working bilaterally, will flex the cervical spine and produce an anterior shear. Unilaterally, the anterior scalene muscles will produce ipsilateral lateral flexion and contralateral rotation to the cervical spine. The middle scalene muscles run from the first rib to the posterior tubercles of the transverse processes of C3 to C7. The middle scalene muscles are more laterally placed than are the anterior scalene muscles, and their line of pull makes them excellent frontal plane stabilizers.[55] The posterior scalene muscles run from the second rib to the posterior tubercles of the transverse processes of C3 to C7. The posterior scalene muscles predominantly laterally flex the neck. The role of the scalene muscles in breathing will be discussed in Chapter 5. The

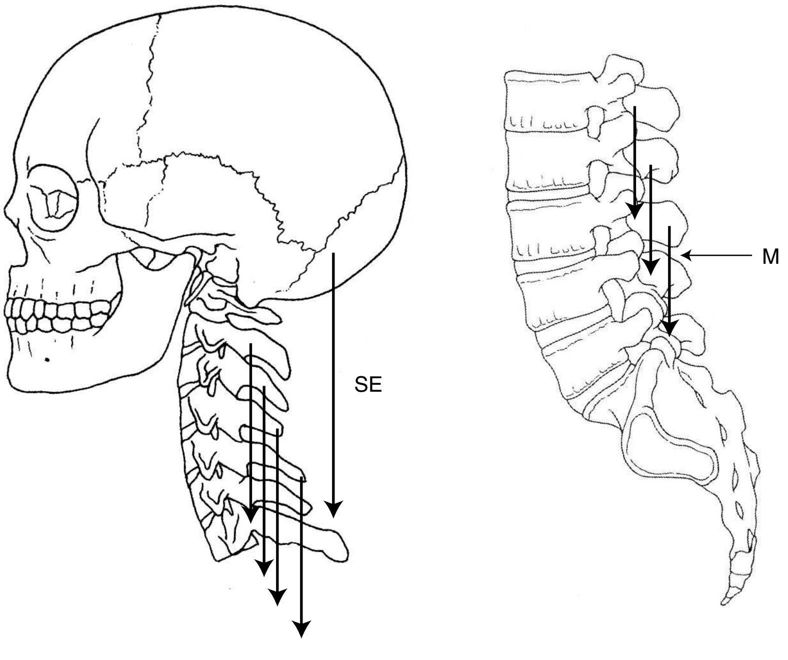


Figure 4–57 Function of the semispinalis capitis in comparison with the lumbar multifidus muscle. The semispinalis capitis has an optimal lever arm for cervical extension. M, multifidus muscle; SE, semispinalis capitis muscle. *(From Porter JA, DeRosa C: Mechanical Neck Pain: Perspectives in Functional Anatomy. Philadelphia, Saunders/Elsevier, 1995, with permission.)*

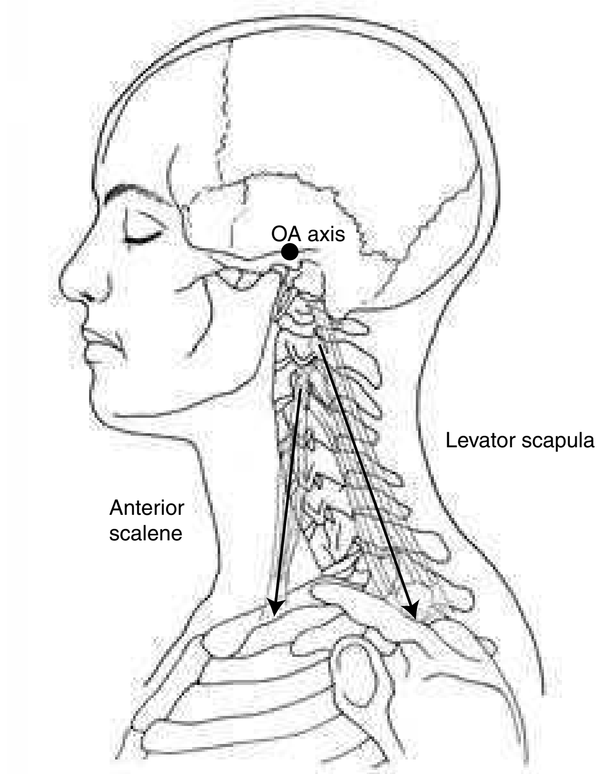


Figure 4–58 In the sagittal plane, the anterior scalene muscles work in synergy with the levator scapulae to provide stability for the cervical spine.

anterior and middle scalene muscles form a triangle through which the brachial plexus and the subclavian artery and vein pass (Fig. 4–59). This can be a site for compression of the neurovascular structures by the anterior scalene muscle: scalenus anticus syndrome, described by Cailliet. This can produce pain, numbness, and tingling in the arm.[70]

The sternocleidomastoid muscle runs from the manubrium and medial clavicle to the mastoid process. The angle of inclination is posterior, medial, and superior. It is unique in that, because of this orientation, it lies anterior to the axis of rotation in the lower cervical spine, producing flexion when acting bilaterally but posterior to the axis at the skull, producing extension of the head on the neck. Acting unilaterally, the sternocleidomastoid muscle will produce ipsilateral lateral flexion and contralateral rotation of the head and neck.

Anterior Muscles

The longus capitis runs from the anterior tubercles of the cervical transverse processes to the occiput. The longus colli runs from the thoracic vertebral bodies to the anterior tubercles of the cervical transverse processes and cranially from the anterior tubercles of the transverse processes to the atlas. These muscles lie close to the vertebral bodies and therefore are relatively close to the axis of rotation. Although they do have sufficient moment arm to produce flexion, they also produce a fair amount of compression. The longus capitis and longus colli work in synergy with the trapezius to stabilize the head and neck in order to allow the trapezius to upwardly rotate the scapula[55] (Fig. 4–60). Given that muscles always contract from both ends, were it not for the longus capitis and longus colli, the trapezius would extend the head and neck rather than upwardly rotate the scapula, in view of the vastly greater weight and moment arm of the upper extremity in comparison with the head.

The rectus capitis anterior and rectus capitis lateralis are able to produce flexion as a result of their line of pull; however, as with the suboccipital muscles, the small cross-sectional area and moment arms probably render them capable of serving a greater proprioceptive function rather than acting as a prime mover.

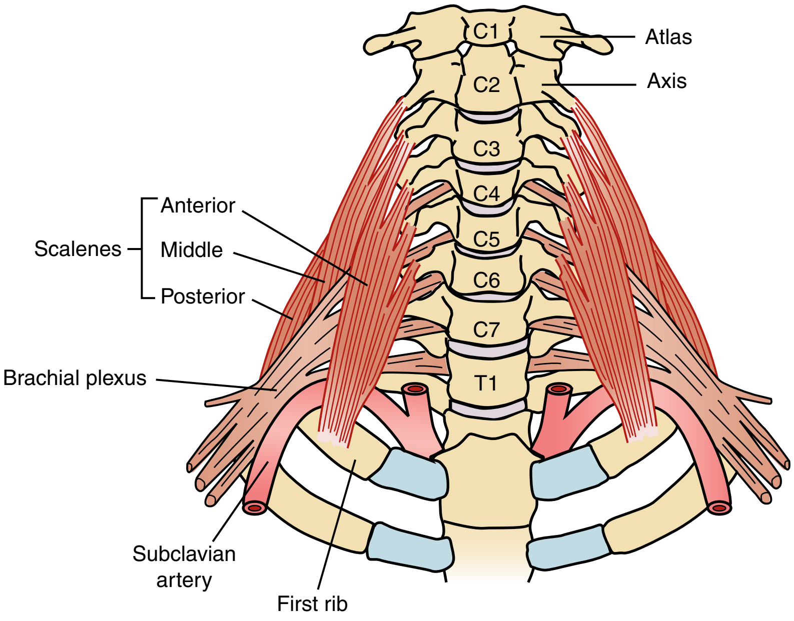


Figure 4–59 The brachial plexus and the subclavian artery and vein pass between the anterior and middle scalene muscles.

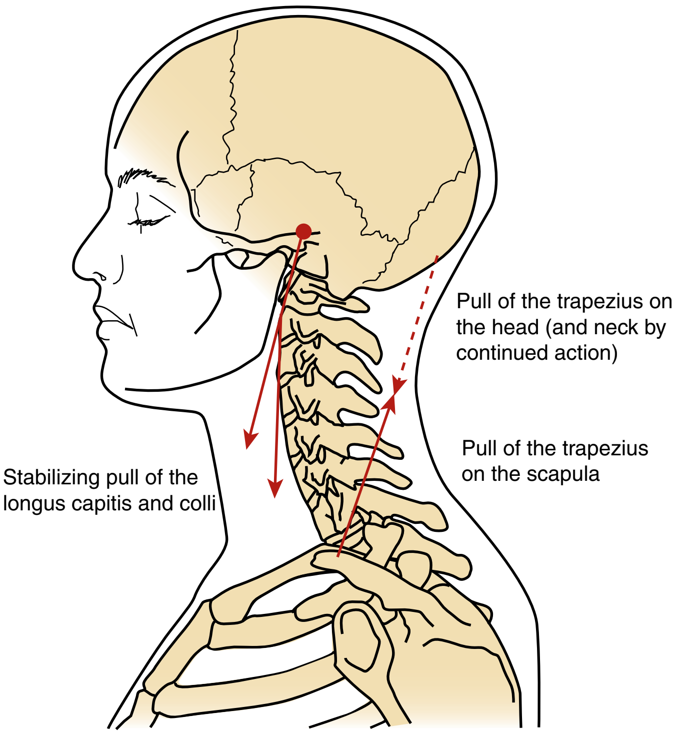


Figure 4–60 In the sagittal plane, the trapezius, longus capitis, and longus colli work in synergy to elevate the scapula.

Concept Cornerstone 4-4

Cervical Motion

Porterfield and DeRosa[55] suggested that this synergy between the longus capitis and longus colli muscles and the trapezius muscle helps explain why hyperextension injuries to the neck (whiplash) can result in pain and the inability to raise the arm overhead. If the longus capitis and colli are damaged, they will be unable to stabilize the head and neck and thus allow the trapezius to successfully rotate the scapula upward.

Lower Thoracic/Lumbopelvic Regions

Muscles of the lower spine regions produce and control movement of the trunk and stabilize the trunk for motion of the lower extremities. The muscles also assist in attenuating the extensive forces that affect this area.

Posterior Muscles

Again, we will examine the muscles from superficial to deep and begin with the posterior muscles. The thoracolumbar fascia is the most superficial structure. As discussed previously, several major muscle groups of this region are associated with the thoracolumbar fascia. The fascia gives rise to the latissimus dorsi, the gluteus maximus, the internal and external abdominal oblique, and the transversus abdominis. In addition, the fascia surrounds the erector spinae and the multifidus muscles of the lumbar region. These attachments are significant because tensile forces can be exerted on the thoracolumbar fascia through the contraction of these muscles. Tension on the thoracolumbar fascia will produce a force that compresses the abdominal contents. Like the contraction of the abdominal muscles, this compression is similar to that of an external corset. The coupled action of the latissimus dorsi, contralateral gluteus maximus, and tension through the thoracolumbar fascia will compress the lumbosacral region and make it stable[26,130] (Fig. 4–61).

The erector spinae consist of the longissimus and iliocostalis muscle groups. In general, these muscles are identified as extensors of the trunk. Bogduk[2] examined the function of the longissimus thoracis and the iliocostalis lumborum and further described these muscles as each having a lumbar portion (pars lumborum) and a thoracic portion (pars thoracis). The longissimus thoracis pars thoracis and the iliocostalis lumborum pars thoracis form the more superficial layer and the longissimus thoracis pars lumborum and the iliocostalis lumborum pars lumborum form a deeper layer.

Anatomically and functionally, therefore, it is easier to group the muscles together as the superficial layer and the deep layer. The **superficial layer** runs from the ribs and thoracic transverse processes to form muscle bellies that are laterally located in the thoracic region. The muscles have long tendons that join together to form the erector spinae aponeurosis, which inserts into the spinous processes of the lower lumbar spine, sacrum, and iliac crest (Fig. 4–62). This superficial layer, with its long moment arm and excellent line of pull, produces extension of the thoracic and lumbar regions when acting bilaterally. These muscles are considered to be the primary extensors of the trunk (Fig. 4–63). Acting unilaterally, they are

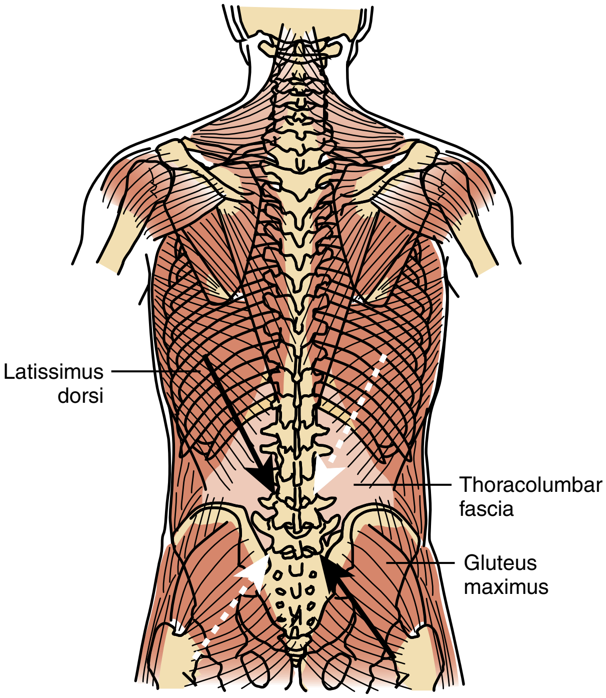


Figure 4–61 Coupled action of the latissimus dorsi, contralateral gluteus maximus, and tension through the thoracolumbar fascia will compress and stabilize the lumbosacral region.

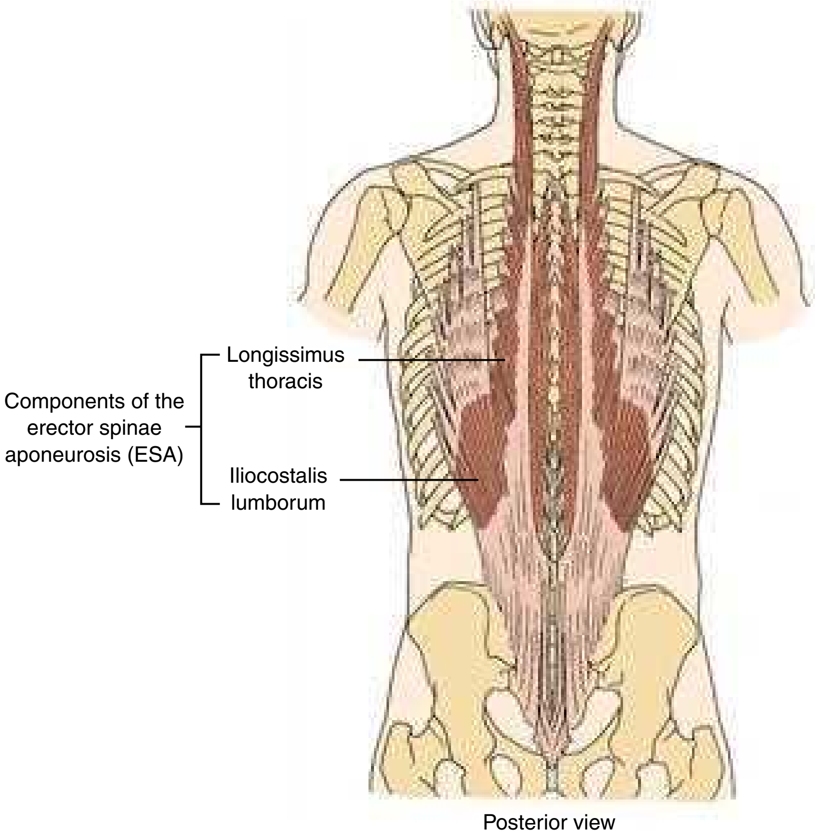


Figure 4–62 The superficial erector spinae with the erector spinae aponeurosis (ESA).

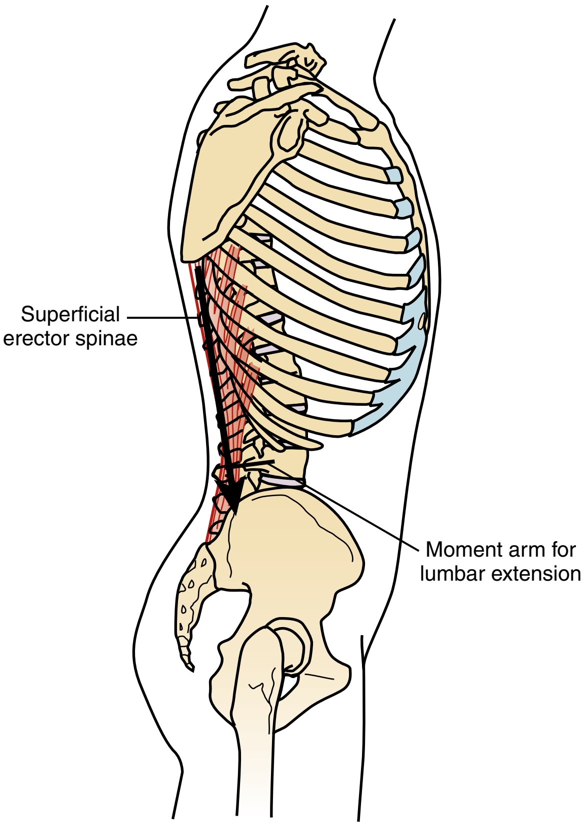


Figure 4–63 Sagittal view of the force vectors of the superficial erector spinae, demonstrating the excellent line of pull and large moment arm for extension.

able to laterally flex the trunk and contribute to rotation. During trunk flexion from a standing position, the erector spinae are responsible for contracting eccentrically to control the motion. The gravitational moment will produce forward flexion, but the extent and rate of flexion are controlled partially by eccentric contractions of the extensors with the erector spinae aponeurosis and partially by the thoracolumbar fascia and posterior ligamentous system. The erector spinae act eccentrically until approximately two-thirds of maximal flexion has been attained, at which point they become electrically silent.[84,91,96] This is called the **flexion-relaxation phenomenon,** which is thought to occur at the point when stretched and deformed passive tissues are able to generate the required moment. However, the extensor muscles may be relaxed only in the electrical sense because they may be generating force elastically through passive stretching.[126] According to Gracovetsky,[96] control of flexion becomes the responsibility of the passive elastic response of the erector spinae aponeurosis, the thoracolumbar fascia, and the posterior ligamentous system. The posterior ligaments (supraspinous and interspinous ligaments) have longer moment arms than do the extensor muscles and thus have a mechanical advantage over the extensors.

Bogduk and others[2,131,132] identified the **deep layer** of the erector spinae as being entirely separate from the superficial layer and consisting of individual fascicles with common tendinous insertions. Bogduk[2] reported that the fascicles arise from the ilium at the posterior superior iliac spine, just lateral on the iliac crest, and course superiorly, medially, and anteriorly to insert on the lumbar transverse processes

(Fig. 4–64). Some debate remains, however, as Daggfeldt and colleagues[131] reported some attachment to the erector spinae aponeurosis of the upper lumbar fascicles, which therefore may not function completely independently. Porterfield and DeRosa[130] described these deep erector spinae as similar in orientation and function to the levator scapulae in the cervical region. These muscles lie close to the axis of rotation and therefore do not have sufficient moment arm to be the prime movers into extension. Their oblique orientation, however, allows the muscles to exert a posterior shear force on the vertebrae. In addition to posterior shear, they also exert compressive forces (Fig. 4–65). Because of their oblique line of action and ability to produce posterior shear, these muscles provide an extremely important dynamic resistance to the constant anterior shear forces of the lumbar region caused by the lordotic position and the forces of gravity and ground reaction forces combined. These muscles, then, have great clinical significance. McGill[26] reported, however, that with full lumbar spine flexion, these muscles lose their oblique orientation and therefore lose their ability to resist anterior shear forces. A flexed spine, therefore, is unable to dynamically resist anterior shear forces, which can cause damage.

Continuing Exploration 4-5:

Negative Effects of Overstretching the Deep Erector Spinae

Like the levator scapulae in the cervical region, the deep erector spinae will become overworked and may be painful when subjected to excessive anterior shear forces. These muscles can become painful as a result of the excessive strain. Caution should be taken, however, in regard to stretching these muscles. Overstretching these muscles can remove the only dynamic restraint to the excessive anterior shear forces and either further load the noncontractile structures or, if they have already been damaged, remove the only remaining restraint. In either case, it is likely to worsen the symptoms. It is more likely that these muscles need greater endurance and strength training.

CASE APPLICATION

Deep Erector Spinae Muscle Strain

case 4–7

In addition to the noncontractile tissues previously discussed, Malik could also be experiencing pain that arises directly from the deep erector spinae muscles, which may have become strained as a result of continual attempts to control for excessive anterior shear forces. If this were the case, stretching and relaxing the deep erector spinae would be an inappropriate goal of rehabilitation for Malik, because this would cause only further stress to the noncontractile tissues. Rather, it would be important to develop greater endurance for these muscles and decrease the external anterior shear forces until the muscles can provide the necessary support. This may be an appropriate time to use a lumbar corset, for example.

Concept Cornerstone 4-5

Location and Function of the Erector Spine

NAME	LOCATION	FUNCTION
Deep Erector Spinae		
Longissimus thoracis pars lumborum (5 fascicles)	One from each lumbar TP to the PSIS	Ipsilateral lateral flexion Posterior shear: greatest at lower lumbar levels Assist with extension but close to axis of rotation; better MA at upper levels
Iliocostalis lumborum pars lumborum (4 fascicles)	One from each tip of the lumbar TP to the iliac crest	Ipsilateral lateral flexion Better rotators, due to greater MA Posterior shear, especially at lower levels Assist with extension but close to axis
Superficial Erector Spinae		
Longissimus thoracis pars thoracis (12 fascicles)	One from each thoracic TP and ribs via the ESA to L3–S3 SP	Extension of thoracic spine Extension of lumbar spine via increasing lordosis Assist with ipsilateral lateral flexion
Iliocostalis lumborum pars thoracis (7 or 8 fascicles)	One from each of the lower 7–8 ribs to PSIS and sacrum with contribution to the ESA	Extension of lumbar spine via increasing lordosis Assist with ipsilateral lateral flexion

ESA, erector spinae aponeurosis; MA, moment arm; PSIS, posterior superior sacroiliac spine; SP, posterior superior iliac spine; TP, transverse process.

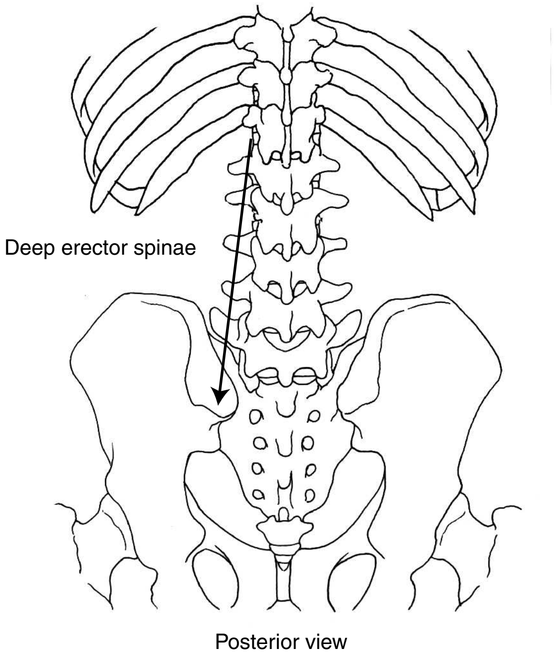


Figure 4–64 The fascicles of the deep erector spinae.

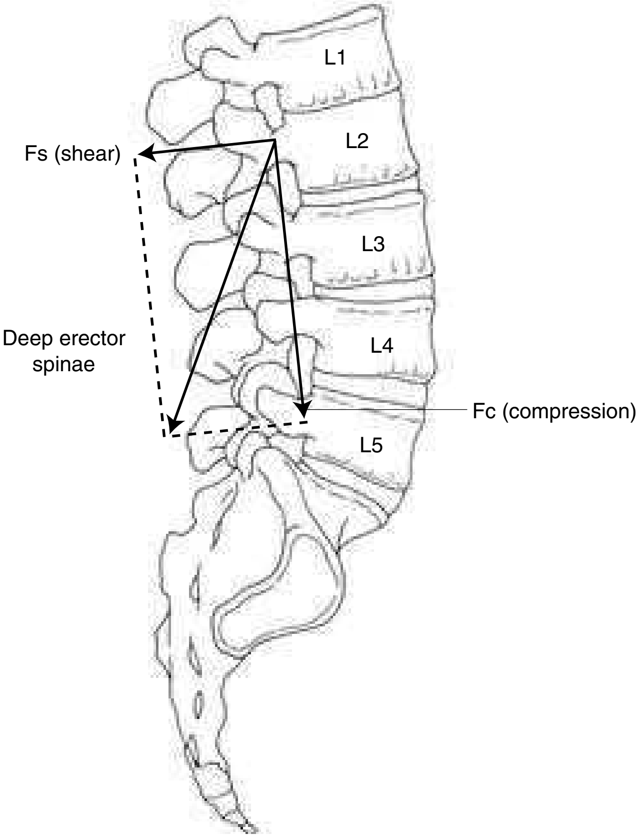


Figure 4–65 Sagittal view of the force vectors of the deep erector spinae, demonstrating the compression and posterior shear components.

The multifidus muscles of the spine are complex and demonstrate segmental and regional differences. The lumbar multifidi run from the dorsal sacrum and the ilium in the region of the posterior superior iliac spine to the spinous processes of the lumbar vertebrae and have separate fascicles.[2,133,135] In this region, the multifidus muscles are not transversospinales, as most anatomy texts depict. The lumbar multifidus muscles are a thick mass that fills the area of the sacral sulcus and are easily palpable there.[130,133] The most superficial fibers of each fascicle cross up to five segments and attach caudally to the sacrum and ilium. Their line of pull is vertically oriented, and they are distant from the axis of rotation and therefore have an effective moment arm for extension by increasing the lumbar lordosis (see Fig. 4–57).[135,136] The deep fibers of each fascicle attach from the lamina and spinous process and cross two segments to insert on the mamilary process and facet joint capsule. The deep fibers in the lumbar region have a small moment arm and therefore are not strong extensors. These fibers are ideally positioned to control segmental shear and torsion by producing compression.[136] The role of the lumbar multifidus muscles in rotation is to work in synergy with the abdominal muscles by opposing the flexion moment that the abdominal muscles produce.[2] McGill[134] has suggested that the role of the lumbar multifidus muscles is to produce extensor torque to allow correction of individual segments that are the foci of stress.

In the thoracic region, the multifidus muscles are transversospinales, as they are more laterally oriented with an oblique line of pull. They run from all of the transverse processes of the thoracic vertebrae to the spinous processes of the more cranial vertebrae, covering one to three segments. The thoracic multifidus muscles are activated with rotation but in a variable manner, suggesting that it too has a role in segmental control of motion rather than production of motion.[133,137,138]

Intertransversarii and rotatores muscles are frequently described as producing lateral flexion and rotation, respectively. Because of their small cross-sectional areas and small moment arms, however, it appears likely that these muscles serve more of a proprioceptive role.[26]

Lateral Muscles

The quadratus lumborum is deeper than the erector spinae and multifidus muscles. The quadratus lumborum, when acting bilaterally, serves as an important frontal plane stabilizer (Fig. 4–66A). Porterfield and DeRosa[130] described the quadratus lumborum as also serving an important role in stabilization in the horizontal plane. When acting unilaterally, the quadratus lumborum can laterally flex the spine (Fig. 4–66C). If lateral flexion occurs from erect standing, the force of gravity will continue the motion, and the contralateral quadratus lumborum will control the movement by contracting eccentrically. It can also control rotational

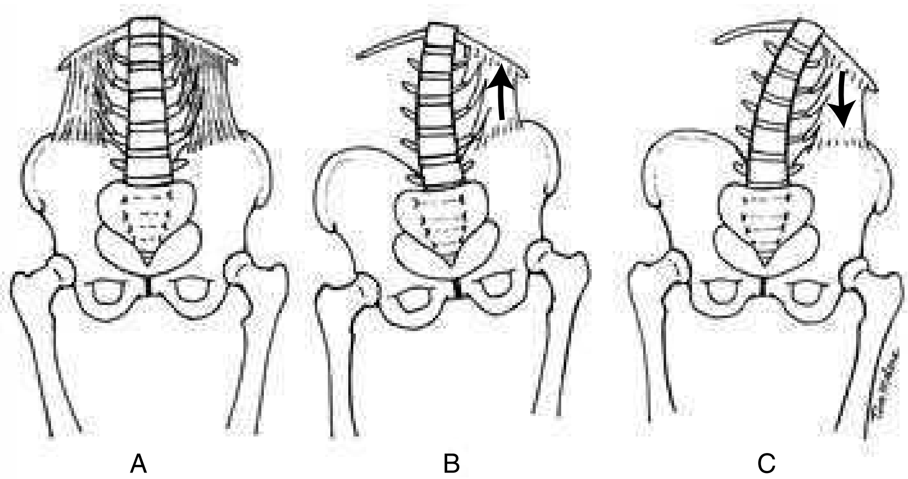


Figure 4–66 **A.** The illustration shows the attachments of the right and left quadratus lumborum muscles. **B.** A unilateral contraction of the left quadratus lumborum muscle will lift and tilt the left side of the pelvis and “hike the hip” when the trunk is fixed and the pelvis and leg are free to move. **C.** A unilateral contraction of the left quadratus lumborum muscle when the pelvis and left leg are fixed will cause ipsilateral trunk flexion.

motion, due to its attachments to the lumbar transverse processes. If the pelvis is free to move, the quadratus lumborum will "hike the hip," or laterally tilt the pelvis in the frontal plane (Fig. 4–66B).

Anterior Muscles

The rectus abdominis is the prime flexor of the trunk. It is contained within the abdominal fascia, which separates the rectus abdominis into sections and attaches the rectus abdominis to the aponeurosis of the abdominal wall. The abdominal fascia also has attachment to the aponeurosis of the pectoralis major. McGill[134] and Porterfield and DeRosa[130] have discussed the importance of these fascial connections as they transmit forces across the midline and around the trunk. Tension on this fascial system will provide stability, similar to that provided by a corset, around the trunk.

The abdominal wall consists of the external oblique, the internal oblique, and the transversus abdominis muscles. These muscles together form what McGill[134] called the "hoop" around the entire abdomen, with the abdominal wall as the anterior aspect and the thoracolumbar fascia and its muscle attachments as the posterior aspect (see Fig. 4–44). This hoop plays an important role in stability of the lumbopelvic region. The transversus abdominis has been shown to mechanically control the sacroiliac joints through significant compressive forces due to the transverse (perpendicular) orientation of the muscle to the joints.[139] Richardson and colleagues demonstrated that contraction of the transversus abdominis decreased laxity at the sacroiliac joints.[139] Cholewicki and colleagues[140] demonstrated that in a neutral spine posture in standing, there is trunk flexor-extensor muscle coactivation at a low level. Furthermore, they showed that this activation level increases with an external load and with decreased spinal stiffness, which supports the hypothesis that this "hoop" can provide stability to the lumbopelvic region and that increased muscle activity can compensate for loss of stiffness in the spinal column caused by injury.

CASE APPLICATION

Exercises to Stabilize Trunk

case 4–8

Knowledge of the function of the trunk musculature needs to be applied to the development of exercises for trunk stabilization for patients like Malik. The fascial connection to the pectoralis major is important because exercises that use the upper extremity in a functional manner can be used to target trunk stability, because they will produce tension on this fascia. Exercises can be functional and yet be done in such a way that trunk movement is minimized and is therefore less likely to produce pain, particularly in the early stages of rehabilitation. Exercises that activate this "hoop" both statically and dynamically will be critical for achieving spinal stabilization in states of pathology in the lumbopelvic region.

The psoas major runs from the lumbar transverse processes, the anterolateral vertebral bodies of T12 to L4, and the lumbar intervertebral discs to the lesser trochanter of the femur. It courses inferiorly and laterally, and the distal tendon merges with that of the iliacus. The primary role of the psoas major is flexion of the hip. McGill[134] reported that it is active only when there is active hip flexion. The iliacus runs from the iliac crest over the pubic ramus to the lesser trochanter with the tendon of the psoas major. The primary role of the iliacus is also hip flexion. The role of the psoas major at the lumbar spine appears to be to buttress the forces of the iliacus, which, when activated, causes anterior ilial rotation and thus lumbar spine extension.[134] The psoas major also provides stability to the lumbar spine during hip flexion activities by providing great amounts of lumbar compression during activation. Some anterior shear is also produced when it is activated.

Continuing Exploration 4-6:

Exercises for Low Back Pain

When developing therapeutic exercises for people with low back disorders, it is important to choose exercises that, while training the muscle groups that we have discussed, also impose the lowest possible loads through this region, especially in the early healing stages. Traditionally, this reasoning has not often been applied in rehabilitation programs.

Exercises to increase the strength of the back extensors are often performed in the prone position to take advantage of the resistance gravity provides to back, leg, and arm extension. Callaghan and colleagues[141] assessed loading of the L4/L5 segment in 13 male volunteers during commonly prescribed exercises. The authors found that the lowest compression forces at the L4/L5 segment were found in single-leg extension in the quadruped position (on hands and knees) (Fig. 4–67A). Raising an arm and leg simultaneously (right arm and left leg) (Fig. 4–67B) increased compression forces by 1,000 N and upper erector spinae muscle activity levels by 30% compared to single-leg extension in the hands and knees position. The right erector spinae and contralateral abdominal muscles were activated during single right-leg extension to maintain a neutral pelvis and spine posture and to balance internal moments and lateral shear forces.

The authors recommended that only single-leg extension exercises be performed because the lumbar posture is more neutral and the compression forces are relatively low (approximately 2,500 N). They further recommended that the exercise in which the subject raises the upper body and legs from a prone lying position never be prescribed for anyone at risk for low back injury because during this exercise, lumbar compression forces of up to 6,000 N are incurred. The extremely high compression forces are a result of bilateral muscle activity when the spine is hyperextended. In this posture, the facets are subjected to high loads, and the interspinous ligament is in danger of being crushed.[141]

Trunk sit-ups and curl-ups are often performed as methods of abdominal strengthening. McGill measured compression forces during these tasks, with both bent knees and straight knees. He found the compression forces to be approximately 3,300 N, without much variation between the types.[142] McGill suggested that given these large compressive loads, most people, let alone those who have sustained injury to this area, should not perform sit-ups of any type.[26]

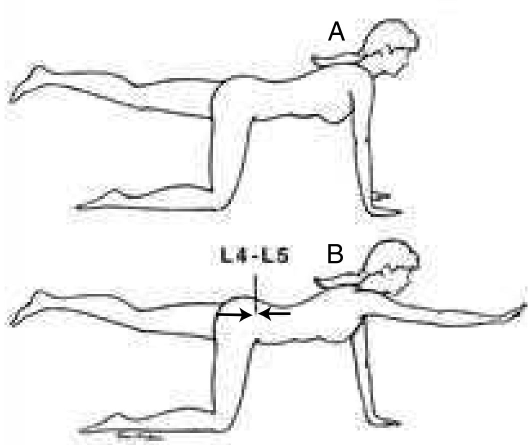


Figure 4–67 **A.** Single-leg extension in the quadruped position creates low compression forces at the L4/L5 segment. **B.** Raising the opposite leg and arm simultaneously increases compression forces at the L4/L5 segment by 1,000 N and upper erector spinae muscle activity by 30% in comparison with single-leg raising.

Injury Prevention With Lifting Tasks: Squat Lift Versus Stoop Lift

The prevalence of back problems in the general population and the difficulties of resolving these problems has generated a great deal of research, both to explain the mechanisms involved in lifting and to determine the best method of lifting so that back injuries can be prevented. A great deal of focus has been on the squat versus stoop lift (Fig. 4–68). During a stoop lift, trunk flexion is achieved primarily by thoracolumbar flexion, and there is little to no knee flexion. During a squat lift, the spine remains as erect as possible and trunk flexion is achieved primarily by hip and knee flexion.

Continuing Exploration 4-7:

Squat Lifting Versus Stoop Lifting

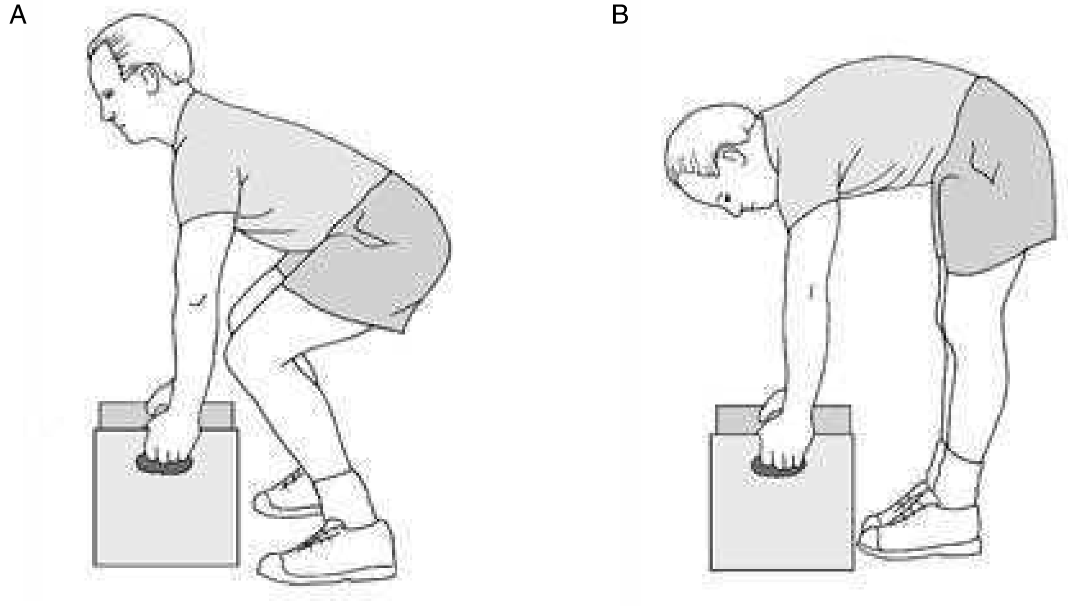
Controversy persists in the literature regarding whether there is biomechanical evidence in support of advocating the squat lifting technique over the stoop lifting technique to prevent low back pain. A review by van Dieen and coworkers in 1999 concluded that the literature does not support advocating squat lifting over stoop lifting.[143] However, the National Health Service Centre for Reviews and Dissemination cautioned that the review by van Dieen and coworkers contained methodological flaws that affected the authors' conclusions.[144] The points that follow appear to support the use of the squat lift; however, it is likely that the best posture for lifting is task-specific.[145]

The extensor muscles are at a disadvantage in the fully flexed position of the spine because of shortened moment arms in this position, a change in the line of pull, and the possibility of passive insufficiency resulting from the elongated state of the muscles. In a neutral lumbar spine posture, the deep layer of the erector spinae is capable of producing posterior shear, which will help to offset the tremendous amounts of anterior shear that occur with trunk flexion, particularly if the person is carrying an additional load.[26,134] The diminished capacity of the extensors to generate torque and to counteract the anterior shear forces in the forward flexed position are important reasons that stoop lifting is discouraged.[26,134]

Another factor involved is intradiscal pressures. Wilke and colleagues updated the classic works of Nachemson, studying in vivo measurements of intervertebral disc pressure.[146] Both Wilke and Nachemson found that intervertebral disc pressures were substantially higher when a load was held in a stooped position than in a squat position.[146,147] Takahashi and colleagues also measured intradiscal pressure in an upright standing position and in a stooped forward position, with and without external load, and found even higher loads in a stooped position than Wilke and Nachemson.[148]

In contrast, there is some evidence that squat lifting results in higher compression forces than does stoop lifting. However, damaging shear forces were two to four times higher for the stoop lift than for the squat lift.[143,149] Although excessive compressive loads can and will produce damage, the spine is better designed to tolerate compressive loads than shear forces. Preventing excessive shear forces, therefore, is critical in preventing injury.[134] Bazrgari and colleagues recently analyzed the dynamics of squat and stoop lifting and conclude that "net moments, muscle forces at different levels, passive forces and internal compression/shear forces are larger in stoop lifts than squat ones."[150]

Other, less controversial but still critical factors in lifting in any posture appear to be the distance from the body to the object to be lifted,[84] the velocity of the lift, and the degree of lumbar flexion.[151] The farther away the load is from the body, the greater the gravitational moment acting on the vertebral column. Greater muscle activity is required to perform the lift, and consequently greater pressure is created in the disc. The higher the velocity of the lift, the greater the amount of weight that can be lifted, but the greater the load on the lumbar discs. The relative spinal load and applied erector spinae force increase significantly with the velocity of trunk extension.[152]



Squat lift

Stoop lift

Figure 4–68 Squat lift **(A)** versus stoop lift **(B)**.

CASE APPLICATION

Instruction for Proper Lifting Techniques

case 4–9

An important aspect of rehabilitation for Malik will be teaching him appropriate bending and lifting mechanics. One of his primary complaints is pain with lifting from a stooped position. Lifting from this position causes greater anterior shear forces through the lumbar region and may be responsible for his pain. In treatment, therefore, he should be taught how to bend and lift in the squat position whenever possible, keeping a neutral lumbar spine position. This will remove some strain from the deep erector spinae and allow them to participate in control of the anterior shear forces.

Muscles of the Pelvic Floor

Structure

Although the **levator ani** and **coccygeus muscles** neither play a major supporting role for the vertebral column nor produce movement of the column, these muscles are mentioned here because of their proximity to the column and possible influence on the linkages that form the pelvis. The levator ani muscles comprise two distinct parts, the **iliococcygeus** and the **pubococcygeus,** which help to form the floor of the pelvis and separate the pelvic cavity from the perineum. The left and right broad muscle sheets of the levator ani form the major portion of the floor of the pelvis. The medial borders of the right and left muscles are separated by the visceral outlet, through which pass the urethra, vagina (in women), and anorectum. The pubococcygeal part of the muscle arises from the posterior aspect of the pubis and has attachments to the sphincter, urethra, walls of the vagina (in women), and the perineal body and rectum (in both genders). The iliococcygeal portion, which arises from the obturator fascia, is thin. Its fibers blend with the fibers of the anococcygeal ligament, form a raphe, and attach to the last two coccygeal segments. The coccygeus muscle arises from the spine of the ischium and attaches to the coccyx and lower portion of the sacrum. The gluteal surface of the muscle blends with the sacrospinous ligament (Fig. 4–69).

Function

Voluntary contractions of the levator ani muscles help constrict the openings in the pelvic floor (urethra and anus) and prevent unwanted micturition and defecation

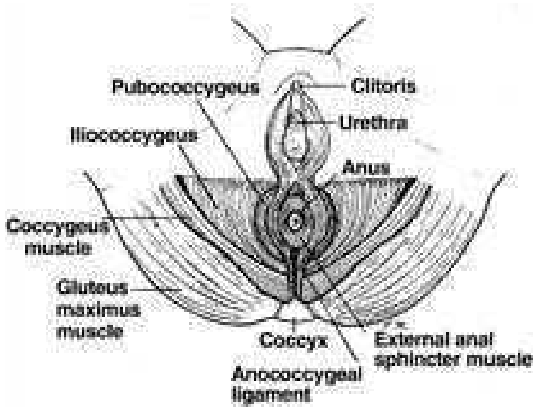


Figure 4–69 Muscles of the pelvic floor.

(stress incontinence). Involuntary contractions of these muscles occur during coughing or holding one's breath, when the intra-abdominal pressure is raised. In women, these muscles surround the vagina and help to support the uterus. During pregnancy the muscles can be stretched or traumatized, which can result in stress incontinence whenever the intra-abdominal pressure is raised. In men, damage to these muscles may occur after prostate surgery. The coccygeus muscle assists the levator ani in supporting the pelvic viscera and maintaining intra-abdominal pressure.

EFFECTS OF AGING

Age-Related Changes

Over the life span, the vertebral column is exposed to recurrent loads that change the morphology of the column. However, normal age-related changes also occur in the structures of the vertebral column.

The vertebral bone undergoes changes in the amount and form of the trabeculae. The numbers of both horizontal and vertical trabeculae decrease with age, and the horizontal trabeculae become significantly thinner.[153] This loss can decrease the loads that the vertebrae are able to withstand before failure.

Each of the structures of the intervertebral disc undergo changes that include loss in the amount of proteoglycans and change in the specific type of proteoglycan, with resultant loss of water content. In addition, there is an increase of collagen in these structures and loss of elastin. This results in a loss of the ability of the disc to transfer loads from one vertebra to another as the swelling ability of the nucleus decreases. The overall disc height will also decrease somewhat.

The vascularization of the disc also changes. In utero, blood vessels are present within the fibers of the anulus fibrosus.[154] By the end of the second year of life, these have predominantly degenerated. Thus, the disc relies on the diffusion of nutrients through the vertebral end plate. The vertebral end plate, with aging, gradually becomes more collagenous, and the process of diffusion is hindered. The fibers of the anulus fibrosus in the cervical spine of adults normally demonstrate lateral fissures that subdivide the disc into two halves at the uncovertebral joints. These fissures can first be observed in children at approximately 9 years of age.[154] After the formation of these fissures, joint pseudocapsules develop with vascularized synovial folds. The formation of these fissures appears to be load-related and is located predominantly in the regions of C3 to C5.

With large and/or repetitive loads, further changes occur in the discs. The discs demonstrate a dramatic decrease in their elasticity and proteoglycans.[154,155] Eventually, the intervertebral disc will become so dry that it begins to crumble. In the lumbar region, the inner layers of the anulus fibrosus begin to buckle outward, and the lamellae separate. Fissures and tears can occur within the anular fibers, which can decrease the ability of the disc to provide stiffness during movement.[155] The vertebral end plates may become ossified. The adjacent spongy bone of the vertebral body can begin to sclerose. On occasion, blood vessels grow into the discs and trigger ossification.[154] The disc can prolapse or protrude as a result of the pressure of the nucleus and the inability of the anulus fibrosus to sustain it. Schmorl's nodes are formed when the nuclear material prolapses through the vertebral end plate and into the cancellous bone of the vertebra. This material may cause an autoimmune response when it comes in contact with the blood supply in the cancellous bone.[130] This is typically labeled degenerative disc disease.

In this case of degenerative disc disease, there is a more substantial loss of disc height, which causes all ligaments to become slack. The ligamentous pre-stress normally provided by the ligamentum flavum will decrease, which in turn will impair spinal stiffness. This can also allow the ligament to buckle on itself with movement, potentially compressing the spinal cord. In addition, the ligamentum flavum begins to calcify with age, and this occasionally leads to ossification, which can also potentially cause compression of the spinal nerve in the vicinity of the zygapophyseal joints or compress the spinal cord within the canal.[156,157]

The zygapophyseal joints can also demonstrate age-related changes and eventual degeneration. Some authors have argued that these changes in the zygapophyseal joints must be secondary to disc degeneration, as a substantial amount of weight-bearing through these joints must occur to cause deterioration. This increase in weight-bearing may be due to the loss of disc height. However, this is not always the case.[154] There have been descriptions of degenerative zygapophyseal joints without disc degeneration. The mechanism of this is not as well understood. If, however, the discs degenerate and a substantive decrease in height occurs, what follows is hypermobility as a result of slackened capsules and longitudinal ligaments. A vertebra may also slip forward or backward on the vertebra below (**listhesis** or **retrolisthesis**). Excessive shear forces will be generated, and the zygapophyseal joints will also become subject to more load-bearing.

The result of these changes is the same as what happens to the larger joints of the extremities: damage of the cartilage, including fissures and cysts, and osteophyte formation. These changes can lead to localized pain or pressure on spinal nerves or the central canal or, in the cervical region, compression of the vertebral artery in the transverse foramen.[154]

The joints of Luschka, or uncovertebral joints, are frequent sites for age-related and degenerative changes. Osteophytes on the uncinate processes occur predominantly in the lower segments, C5/C6 or C6/C7. The motion of lateral bending becomes extremely limited when these osteophytes occur.

Concept Cornerstone 4-6

Osteophyte Formation

Clinicians should remember the frequency of osteophyte formation at the uncinate processes and subsequent lateral bending limitations when examining cervical range of motion. Restoration of lateral bending motion is often not a realistic expectation.

SUMMARY

In summary, it is extremely important to understand the normal structure and function, including normal variability, of the vertebral column in order to understand the structures at risk for injury and the best ways to treat people with dysfunction. Injury or failure occurs when the applied load exceeds the strength of a particular tissue. Repetitive strain causes injury by either the repeated application of a relatively low load or by application of a sustained load for a long duration (prolonged sitting or stooped posture).

The effects of injury, aging, disease, or development deficit on the vertebral column may be analyzed by taking the following points into consideration:

1. The normal function that the affected structure is designed to serve
2. The stresses that are present during normal situations
3. The anatomic relationship of the structure to adjacent structures
4. The functional relationship of the structure to other structures

STUDY QUESTIONS

1. Which region of the vertebral column is most flexible? Explain why this region has greater flexibility.
2. Describe the relationship between the zygapophyseal joints and the interbody joints.
3. What is the zygapophyseal facet orientation in the lumbar region? How does this orientation differ from that of other regions? How does the orientation in the lumbar region affect motion in that region?
4. Describe the relative strength of the longitudinal ligaments in the lumbar region. How does this differ in the other regions? Are some structures more susceptible to injury in this region on the basis of this variation?
5. Which structures would be affected if a person has an increased lumbar lordosis? Describe the type of stress that would occur, where it would occur, and how it would affect different structures.
6. Describe the function of the intervertebral disc during motion and in weight-bearing.
7. Describe the differences in structure between the cervical and lumbar intervertebral discs.
8. Identify the factors that limit rotation in the lumbar spine. Explain how the limitations occur.
9. Which muscles cause extension of the lumbar spine? In which position of the spine are they most effective?
10. Describe the forces that act on the spine during motion and at rest.
11. Explain how creep may adversely affect the stability of the vertebral column.
12. Describe how muscles and ligaments interact to provide stability for the vertebral column.
13. What role has been attributed to the thoracolumbar fascia in the stability of the lumbopelvic region?
14. Describe the dynamic and static restraints to anterior shear in the lumbar region.
15. Describe the dynamic restraints to anterior shear in the cervical region.

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Chapter 5

The Thorax and Chest Wall

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Introduction

General Structure and Function
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  - Kinematics of the Ribs and Manubriosternum
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INTRODUCTION

The thorax, consisting of the thoracic vertebrae, the ribs, and the sternum (Figs. 5–1A and B), has several important functions. It provides a base for the muscle attachment of the upper extremities, the head and neck, the vertebral column, and the pelvis. The thorax also provides protection for the heart, lungs, and viscera. Therefore, there needs to be a certain amount of inherent stability to the thorax. The structure of the rib cage significantly increases the stability of the thoracic spine in flexion/extension, lateral bending, and rotation.[1–4] Probably the most important function of the chest wall is its role in ventilation. The process of ventilation depends on the mobility of the bony rib thorax and the ability of the muscles of ventilation to move the thorax.[5,6]

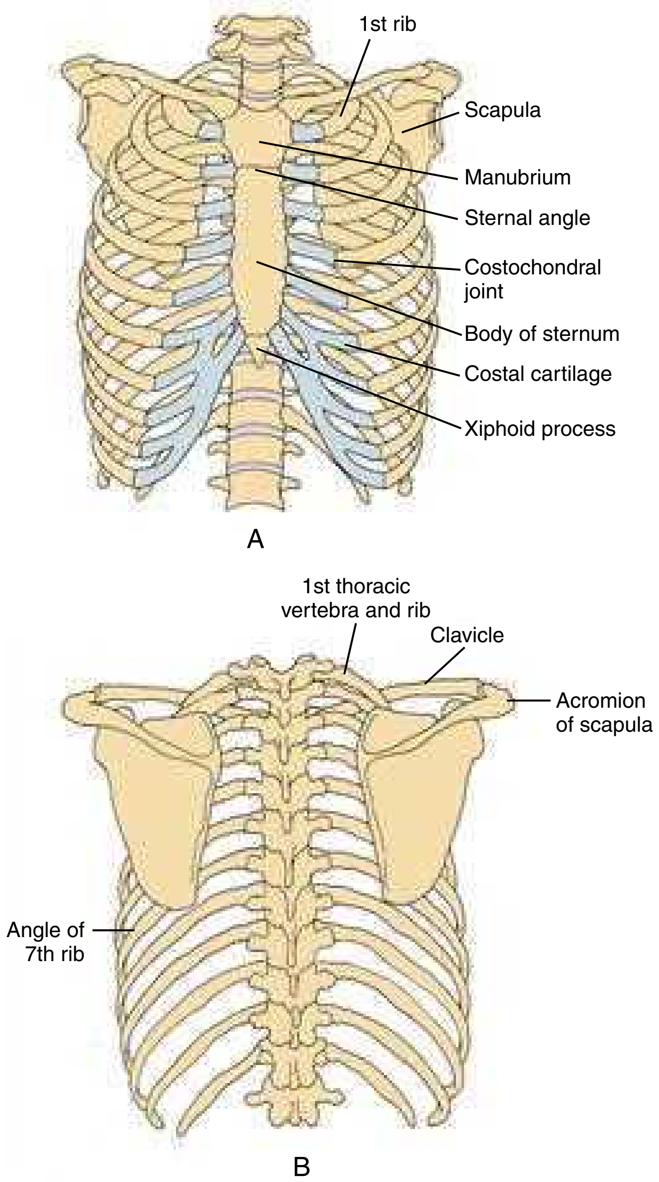


Figure 5–1 Anterior **(A)** and posterior **(B)** views of the thorax are shown, including its component parts: the sternum, 12 pairs of ribs and their costocartilages, and the thoracic vertebrae.

Function, especially ventilatory function, can be affected when pathology interferes with the structure of the bony thorax. For example, scoliosis is a pathological lateral curvature of the spine frequently associated with rotation of the vertebrae.[7] A right thoracic scoliosis (named for the convex side of the thoracic curve) results in left lateral flexion of the thoracic spine (Fig. 5–2A). The coupled rotation in a typical right thoracic scoliosis causes the bodies of the vertebrae to rotate to the right and the spinous processes to rotate to the left. The right transverse processes of the vertebrae rotate posteriorly, carrying the ribs with them (Fig. 5–2B). This is the mechanism that causes the classic posterior rib hump of scoliosis. On the concave side of the scoliotic curve, the effects are just the opposite. The transverse processes of the vertebrae move anteriorly, bringing the articulated ribs forward. The rib distortion that results from the vertebral rotation is evident bilaterally in Figure 5–2A and 5–2B. These musculoskeletal abnormalities limit the range of motion of the rib cage and the spine and, therefore, decrease ventilatory abilities.[8] The coupling and interaction of the bony thorax and the ventilatory muscles and their relationship to ventilation will be the focus of this chapter.

5-1 Patient Case

case

Mary Nasser is an otherwise sedentary 12-year-old female who recently joined her town's tennis team. This is the first time she has really played tennis since she took lessons in childhood. She began to complain of shortness of breath during portions of practice that involved a high level of exertion. She saw her primary care physician, who picked up evidence of a scoliosis (curvature of the spine) in her initial screening. Spine radiographs were done, and Mary was diagnosed with an idiopathic right thoracic scoliosis, with a 40° curve. A medical workup was negative for an acute pulmonary process. Mary was referred to an orthopedic physician and to physical therapy for management of the scoliosis and shortness of breath.

GENERAL STRUCTURE AND FUNCTION

Rib Cage

The rib cage is a closed chain that involves many joints and muscles. The anterior border of the rib cage is the sternum, the lateral borders are the ribs, and the posterior border is formed by the thoracic vertebrae. The superior border of the rib cage is formed by the jugular notch of the sternum, by the superior borders of the first costocartilages, and by the first ribs and their contiguous first thoracic vertebra. The inferior border of the rib cage is formed by the xiphoid process, the shared costocartilage of ribs 7 through 10, the inferior portions of the 11th and 12th ribs, and the 12th thoracic vertebra (see Fig. 5–1).

The sternum is an osseous protective plate for the heart and is composed of the **manubrium, body,** and **xiphoid process** (Fig. 5–3). The manubrium and the

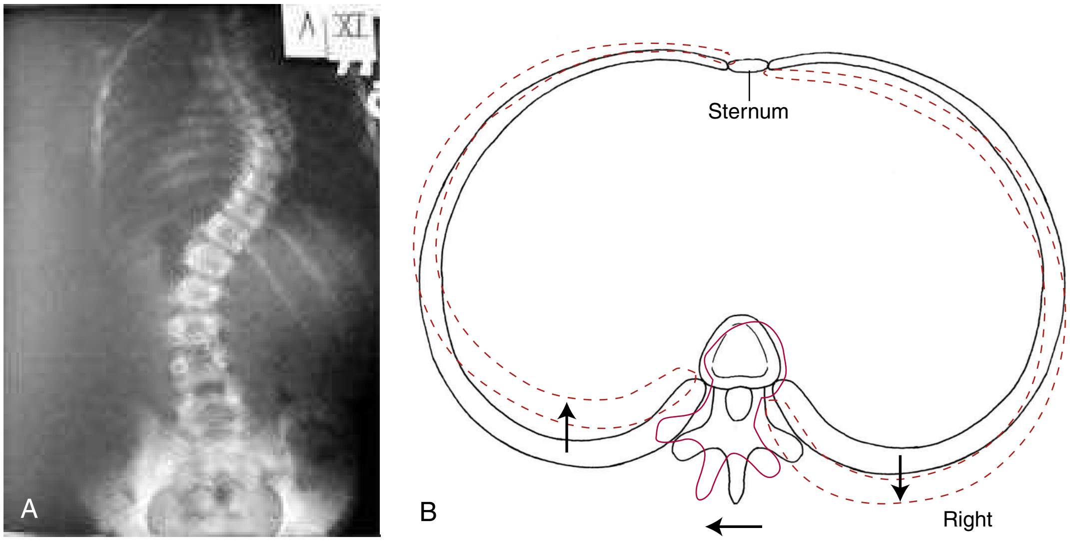


Figure 5-2 **A.** A right thoracic scoliosis (named for the side of the convexity) of 52° shows the evident rib distortion that results from accompanying rotation of the involved vertebrae. There is also a lumbar curve of 32°. **B.** The bodies of the thoracic vertebrae in a right scoliosis typically rotate to the right, resulting in posterior displacement of the right transverse process and the attached right rib, as well as anterior displacement of the opposite transverse process and left rib.

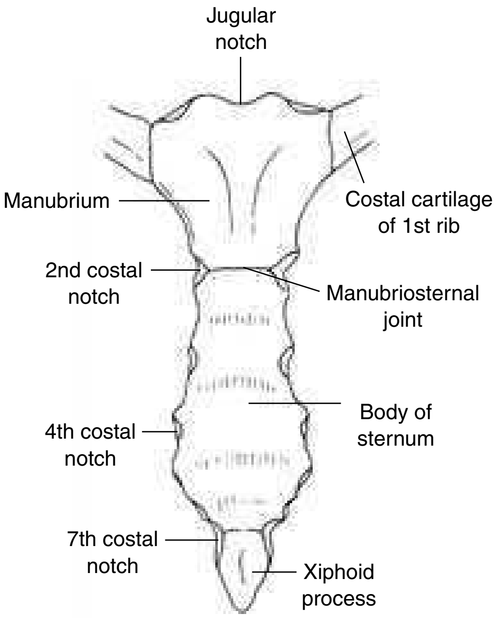


Figure 5–3 The sternum is composed of the manubrium, the body of the sternum, and the xiphoid process. The costal notches for the chondrosternal joints are also evident in this anterior view.

body of the sternum form a dorsally concave angle of approximately 160°. The xiphoid process often angles dorsally from the body of the sternum and may be difficult to palpate.

There are 12 thoracic vertebrae that make up the posterior aspect of the rib cage. One of the unique aspects of the typical thoracic vertebra is that the vertebral body and transverse processes have six costal articulating surfaces, four on the body (a superior and an inferior costal facet, or **demifacet,** on each side) and one costal facet on each transverse process (Fig. 5–4). The rib cage also includes 12 pairs of ribs. The ribs are curved flat bones that gradually increase in length from rib 1 to rib 7 and then decrease in length again from rib 8 to rib 12.[9] The posteriorly located head of each rib articulates with contiguous thoracic vertebral bodies. The costal tubercles of ribs 1 to 10 also articulate with the transverse processes of thoracic vertebrae (Fig. 5–5). Anteriorly, ribs 1 to 10 are joined either directly or indirectly to the sternum through their costal cartilages (Fig. 5–6). Ribs 1 through 7 are classified as **vertebrosternal** (or **"true"**) **ribs** because each rib, through its costocartilage, attaches directly to the sternum. The costocartilage of ribs 8 through 10 articulates with the costocartilage of the superior rib, indirectly articulating with the sternum via rib 7. These ribs are classified as **vertebrochondral** (or **"false"**) **ribs.** The 11th and 12th ribs are

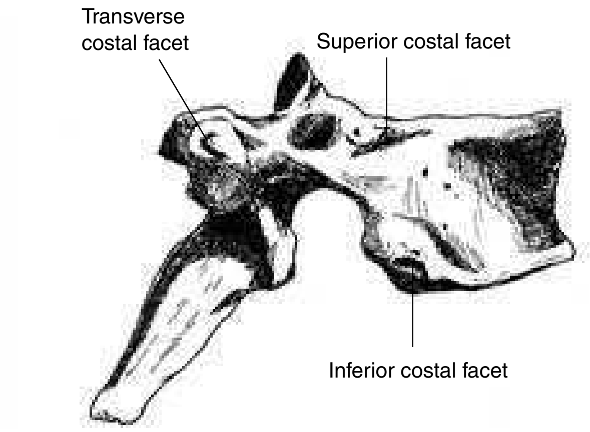


Figure 5–4 The costal facets on the typical thoracic vertebrae are found on the superior and inferior aspects of the posterior body and the anterior transverse processes.

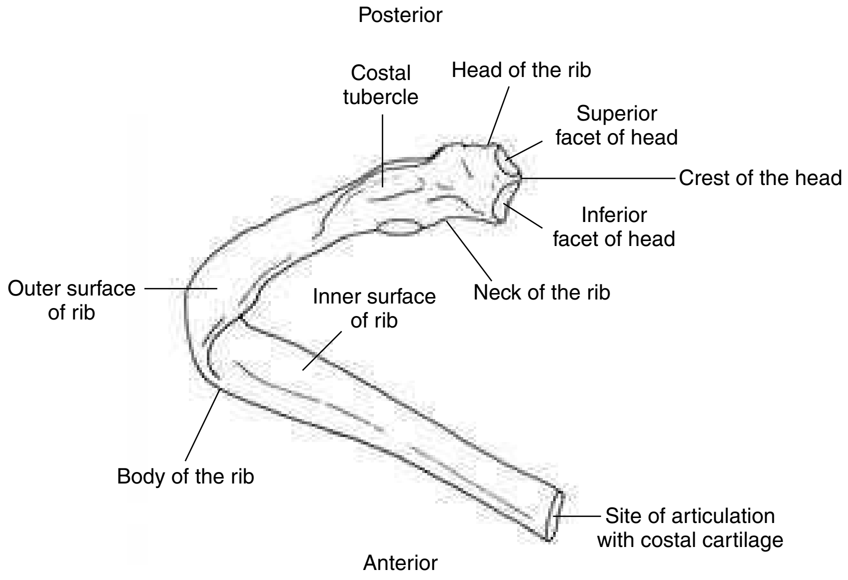


Figure 5–5 The typical rib (ribs 2 through 9) is a curved flat bone. The posteriorly located head of the rib has superior and inferior facets that are separated by a ridge called the crest of the head. The superior and inferior facets (also known as demifacets) articulate, respectively, with the inferior and superior costal facets on the bodies of adjacent vertebrae. The neck of the rib extends from the head of the rib to the costal tubercle. The facet on the costal tubercle articulates with the transverse process of the corresponding vertebra; the rib articulates anteriorly to the sternum via costocartilages.

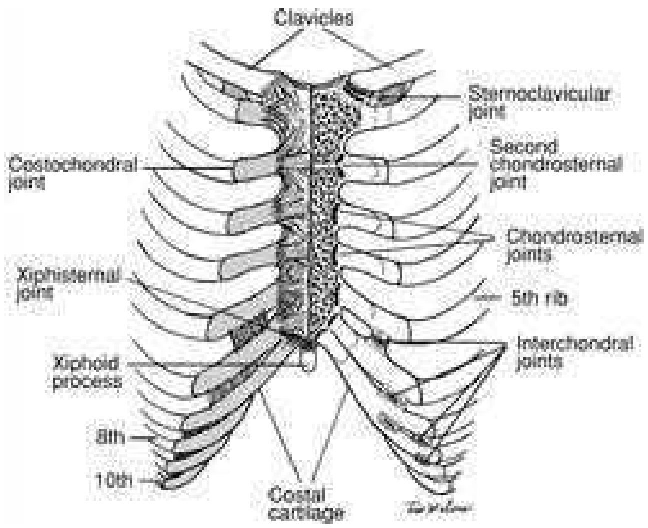


Figure 5–6 In this anterior view of the rib cage, the ribs articulate with the costal cartilages. The ribs join the costal cartilages at the costochondral joints. The costal cartilages of the first through the seventh ribs articulate directly with the sternum through the chondrosternal joints. The costal cartilages of the 8th through the 10th ribs articulate indirectly with the sternum through the costal cartilages of the adjacent superior rib at the interchondral joints.

called **vertebral** (or **"floating") ribs** because they have no anterior attachment to the sternum.[9]

Articulations of the Rib Cage

The articulations that join the bones of the rib cage include the **manubriosternal, xiphisternal, costovertebral, costotransverse, costochondral, chondrosternal,** and the **interchondral joints.**

Manubriosternal and Xiphisternal Joints

The manubrium and the body of the sternum articulate at the manubriosternal joint (see Fig. 5–3). This joint is also known as the sternal angle or the angle of Louis and is readily palpable as a horizontal ridge at the level of the second ribs' anterior attachments.[5,10] The manubriosternal joint is a synchondrosis. The manubriosternal joint has a fibrocartilaginous disc between the hyaline cartilage–covered articulating ends of the manubrium and the body of the sternum—it is structurally similar to the symphysis pubis of the pelvis. Ossification of the manubriosternal joint occurs in elderly persons.[10,11] The xiphoid process joins the inferior aspect of the sternal body at the xiphisternal joint. The xiphisternal joint is also a synchondrosis and tends to ossify by 40 to 50 years of age.[12]

Costovertebral Joints

The typical costovertebral joint is a synovial joint formed by the head of the rib, two adjacent vertebral bodies, and the interposed intervertebral disc. Ribs 2 through 9 have typical costovertebral joints, inasmuch as the heads of these ribs each have two articular facets, or so-called demifacets[10,13] (see Fig. 5–5). The demifacets are separated by a ridge called the crest of the head of the rib. The small, oval, and slightly convex demifacets of the ribs are called the **superior** and **inferior costovertebral facets.** Adjacent thoracic vertebrae have facets corresponding to those of the heads of the ribs that articulate with them. The heads of ribs 2 through 9 fit snugly into the angle formed by the adjacent vertebral demifacets and the intervening disc. Each rib's superior facet articulates with the inferior facet of the vertebrae above it. Each rib's inferior facet articulates with the superior facet of its own numbered vertebrae (Fig. 5–7). Take rib 4 as an example: the rib's superior facet articulates with vertebral body above (T3), and the rib's inferior facet articulates with the superior facet of the vertebral body below (T4). Ribs 1, 10, 11, and 12 are atypical ribs because they articulate with only one vertebral body and are numbered by that body.[9,12,13] The costovertebral facets of T10 through T12 are located more posteriorly on the pedicle of the vertebra.[10]

The typical costovertebral joint is divided into two cavities by the **interosseous** or **intra-articular ligament.**[12,13]

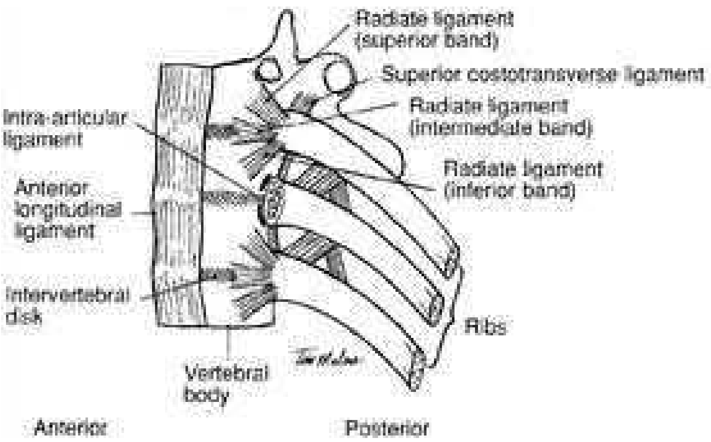


Figure 5–7 A lateral view of the costovertebral joints and ligaments. The three bands of the radiate ligament reinforce the costovertebral joints. The superior and inferior bands of the radiate ligament attach to the joint capsule (removed) and to the superior and inferior vertebral bodies, respectively. The intermediate band attaches to the intervertebral disc. The middle costovertebral joint is shown with the radiate ligament bands removed to reveal the intra-articular ligament that attaches the head of the rib to the anulus fibrosus.

This ligament extends from the crest of the head of the rib to attach to the anulus fibrosus of the intervertebral disc.[10,13] The **radiate ligament** is located within the capsule, with firm attachments to the anterolateral portion of the capsule. The radiate ligament has three bands: the **superior band,** which attaches to the superior vertebra; the **intermediate band,** which attaches to the intervertebral disc; and the **inferior band,** which attaches to the inferior vertebra[9,10,12] (see Fig. 5–7). A fibrous capsule surrounds the entire articulation of each costovertebral joint.

The atypical costovertebral joints of ribs 1, 10, 11, and 12 are more mobile than the other, more typical costovertebral joints because the rib head articulates with only one vertebra. The interosseous ligament is absent in these joints; therefore, they each have only one cavity.[13] The radiate ligament is present in these joints, with the superior band still attached to the superior vertebra. Both rotation and gliding motions occur at all of the costovertebral joints.[14]

Costotransverse Joints

The costotransverse joint is a synovial joint formed by the articulation of the costal tubercle of the rib with a costal facet on the transverse process of the corresponding vertebra[13] (Fig. 5–8). There are 10 pairs of costotransverse joints articulating vertebrae T1 through T10 with the rib of the same number. The costotransverse joints on T1 through approximately T6 have slightly concave costal facets on the transverse processes of the vertebrae and slightly convex costal tubercles on the corresponding ribs. This allows slight rotation movements between these segments. At the costotransverse joints of approximately T7 through T10, both articular surfaces are flat and gliding motions predominate. Ribs 11 and 12 do not articulate with their respective transverse processes of T11 or T12.

The costotransverse joint is surrounded by a thin, fibrous capsule. Three major ligaments support the costotransverse joint capsule. These are the **lateral costotransverse ligament,** the **costotransverse ligament,** and the **superior costotransverse ligament** (Fig. 5–9). The lateral costotransverse ligament is a short, stout band located between the lateral portion of the costal tubercle and the tip of the corresponding transverse process.[13,14] The costotransverse ligament is composed of short fibers that run within the

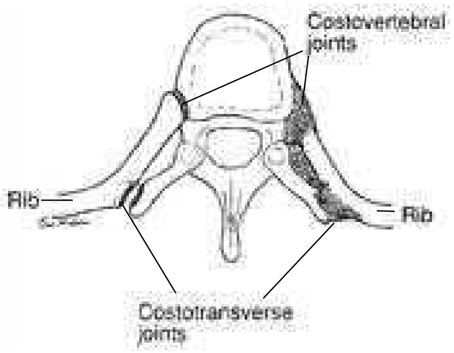


Figure 5–8 A superior view of the costovertebral and costotransverse joints shows the capsuloligamentous structures on the right. The joint capsules and ligaments are removed on the left to show the articulating surfaces.

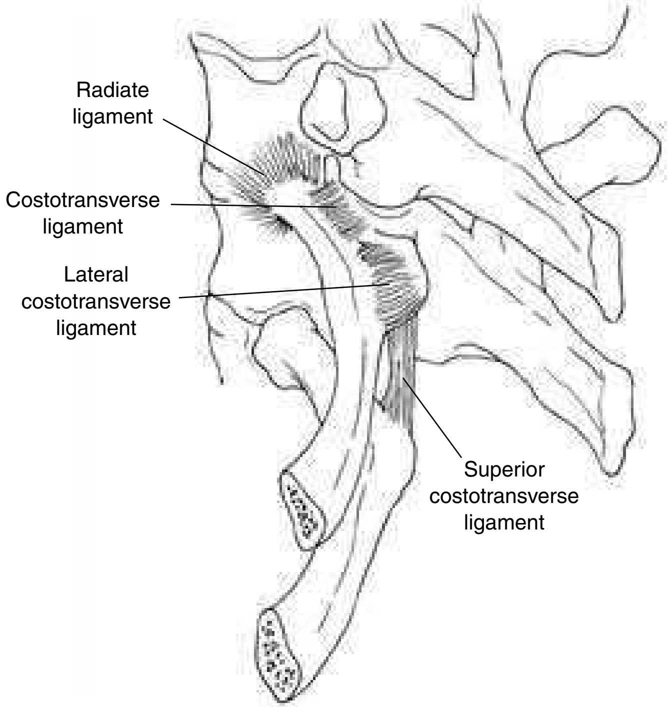


Figure 5–9 Ligaments supporting the costotransverse joint, including (1) the costotransverse ligament, (2) the lateral costotransverse ligament, and (3) the superior costotransverse ligament.

costotransverse foramen between the neck of the rib posteriorly and the transverse process at the same level.[10,13] The superior costotransverse ligament runs from the crest of the neck of the rib to the inferior border of the cranial transverse process.

Costochondral and Chondrosternal Joints

The costochondral joints are formed by the articulation of the 1st through 10th ribs anterolaterally with the costal cartilages (see Fig. 5–6). The costochondral joints are synchondroses.[10] The periosteum and the perichondrium are continuous, giving support to the union. The costochondral joints have no ligamentous support.

The chondrosternal joints are formed by the articulation of the costal cartilages of ribs 1 through 7 anteriorly with the sternum (see Fig. 5–6). Rib 1 attaches to the lateral facet of the manubrium, rib 2 is attached via two demifacets at the manubriosternal junction, and ribs 3 through 7 articulate with the lateral facets of the sternal body. The chondrosternal joints of ribs 1, 6, and 7 are synchondroses. The chondrosternal joints of ribs 2 through 5 are synovial joints.

The chondrosternal joints of ribs 1 through 7 have capsules that are continuous with the periosteum and support the connection of the cartilage as a whole.[13] Ligamentous support for the capsule includes **anterior** and **posterior radiate costosternal ligaments.** The **sternocostal ligament** is an intra-articular ligament, similar to the intra-articular ligament of the costovertebral joint, that divides the two demifacets of the second chondrosternal joint.[9,12,13] The chondrosternal joints may ossify with age.[9] The **costoxiphoid ligament** connects the anterior and posterior surfaces of the seventh costal cartilage to the front and back of the xiphoid process.

Interchondral Joints

The costal cartilages of ribs 7 through 10 each articulate with the cartilage immediately above them to form the interchondral joints. For ribs 8 through 10, this articulation forms the rib's only connection to the sternum, albeit indirectly (see Fig. 5–6). The interchondral joints are synovial joints and are supported by a capsule and interchondral ligaments. The interchondral articulations, like the chondrosternal joints, tend to become fibrous and fuse with age.

Concept Cornerstone 5-1

Rib Cage Summary

In summary, ribs 1 through 10 articulate posteriorly with the vertebral column by two synovial joints (the costovertebral and costotransverse joints) and anteriorly through the costocartilages to the manubriosternum, either directly or indirectly. These joints form a closed kinematic chain in which the segments are interdependent and motion is restricted. These articulations and their associated ligamentous support give the thoracic cage the stability necessary to protect internal organs and yet provide enough flexibility to maximize function.[13] Ribs 11 and 12 have a single costovertebral joint, no costotransverse joint, and no attachment anteriorly to the sternum. These ribs form an open kinematic chain, and the motion of these ribs is less restricted.

Kinematics of the Ribs and Manubriosternum

The movement of the rib cage is an amazing combination of complex geometrics that are governed by (1) the types and angles of the articulations, (2) the movement of the manubriosternum, and (3) the elasticity of the costal cartilages.

The costovertebral and costotransverse joints are mechanically linked, with a single axis of motion for elevation and depression passing through the centers of both joints.[6,12–14] The length, shape, and downward angle of each rib is unique, and therefore, the axis of rotation for each rib is slightly different. The axes of rotation for the upper ribs lie closest to the frontal plane, allowing the motion of those ribs to occur predominantly in the sagittal plane. The axes of rotation for the lower ribs move toward the sagittal plane, allowing the motion of those ribs to be closer to the frontal plane (Fig. 5–10). The axes of rotation for

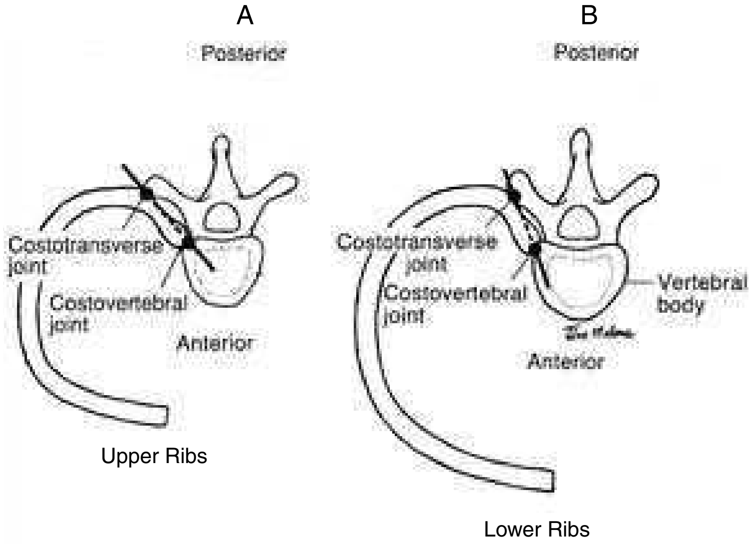


Figure 5–10 **A.** The common axis of motion for the upper ribs passes through the centers of the costovertebral and costotransverse joints and lies nearly in the frontal plane. **B.** The axis through the costovertebral and costotransverse joints for the lower ribs lies closer to the sagittal plane.

ribs 11 and 12 pass through the costovertebral joint only, because there is no costotransverse joint present. The axes of rotation for these last two ribs also lie close to the frontal plane.

The first rib is unique because its anterior articulation is larger and thicker than that of any other rib.[9] The first costal cartilage is stiffer than the other costocartilages. Also, the first chondrosternal joint is cartilaginous (synchondrosis), not synovial, and therefore is firmly attached to the manubrium. Finally, the first chondrosternal joint is just inferior and posterior to the sternoclavicular joint. For these reasons, there is very little movement of the first rib at its attachment on the manubrium. Posteriorly, the costovertebral joint of the first rib has a single facet, which increases the mobility at that joint. During inspiration, the costovertebral joint moves superiorly and posteriorly, elevating the first rib.

Ribs 2 through 7, which are attached to the body of the sternum, increase in length and mobility the more caudal the rib. In these upper ribs, most of the movement occurs at the anterior aspect of the rib, given the nearly coronal axis at the vertebrae. The costocartilage rotates upward, becoming more horizontal with inspiration.[13] The movement of the ribs pushes the sternum ventrally and superiorly. The manubrium moves less than the body of the sternum because the shortest and least mobile first rib is attached to the manubrium. This discrepancy in rib length increases the movement of the body of the sternum in relation to the manubrium and results in slight movement at the manubriosternal joint.[10] The greatest effect of the motion of the upper ribs and sternum is the increase in the anteroposterior (A-P) diameter of the thorax. This combined rib and sternal motion that occurs in a predominantly sagittal plane has been termed the **"pump-handle" motion** of the thorax (Fig. 5–11).

Elevation of ribs 8 through 10 occurs about an axis of motion lying more towards the sagittal plane. The lower ribs have a more angled shape (downward obliquity increases from rib 1 to rib 10) and an indirect attachment anteriorly to the sternum. These factors allow the lower ribs more motion at the lateral aspect of the rib cage. The greatest effect of the elevation of the lower ribs is the increase in the transverse diameter of the lower thorax. This motion that occurs in a more frontal plane has been termed the **"bucket-handle" motion** of the thorax (Fig. 5–12).

There is a gradual shift in the orientation of the ribs' axes of motion from cephalad to caudal; therefore, the intermediate ribs demonstrate a transitional zone, with qualities of both types of motion.[9,12–15] The 11th and 12th ribs each have only one posterior articulation with a single vertebra and no anterior articulation to the sternum; therefore, they do not participate in the closed-chain motion of the thorax.

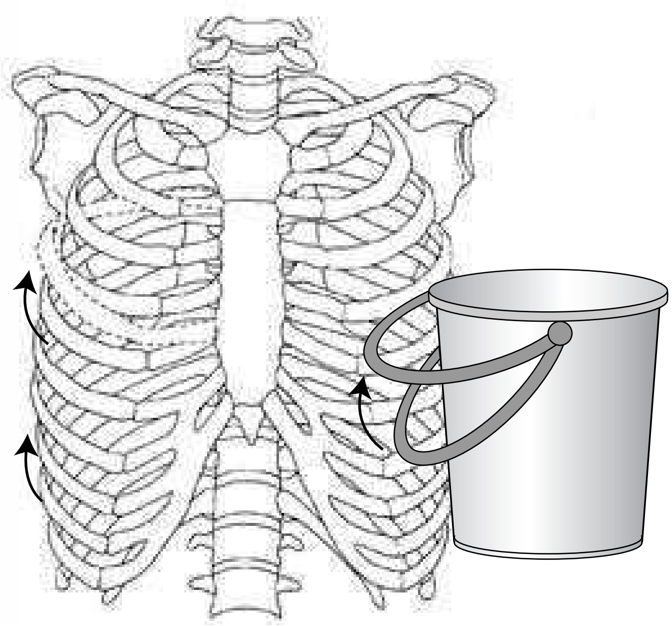


Figure 5–12 Elevation of the lower ribs at the costovertebral and costotransverse joints results in a lateral motion of the rib cage, referred to as "bucket-handle" motion of the thorax.

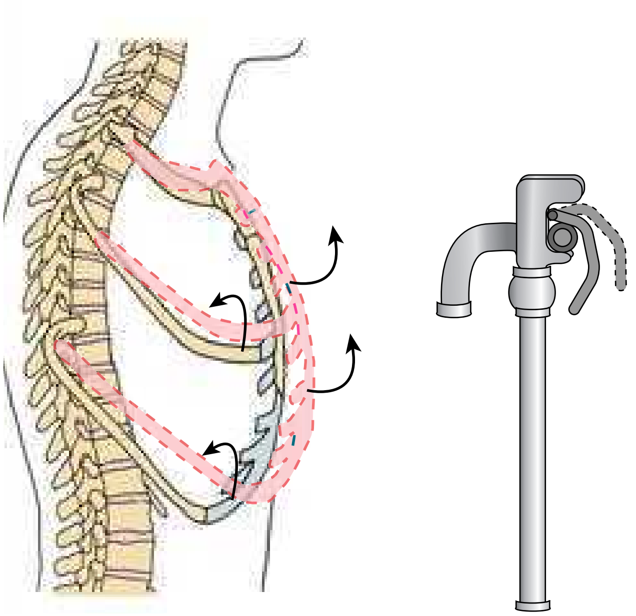


Figure 5–11 Elevation of the upper ribs at the costovertebral and costotransverse joints results in anterior and superior movement of the sternum (and accompanying torsion of the costal cartilages), referred to as the "pump-handle" motion of the thorax.

Continuing Exploration 5-1:

Effects of Scoliosis on the Rib Cage

The single axis of motion of the ribs is through the costovertebral and costotransverse joints. Therefore, changes in the alignment of these joints will change the mobility of the thorax. In scoliosis, the thoracic vertebrae not only laterally deviate but also rotate, altering the alignment of the costovertebral and costotransverse articulating surfaces (see Figs. 5–2A and B). The rib cage volume changes asymmetrically in scolioss, with the concave side of the thorax showing anterior rib distortion, a decrease in vertebral height, a narrowing of the intercostal spaces, and a decrease in lung volume. The convex side shows posterior rib distortion, widened intercostals spaces and an increased lung volume.[16] Figure 5–13A is a view of a normal thorax in a 4-year-old. Figure 5–13B is a view of the thorax of a 4-year-old with a congenital thoracic scoliosis and shows an extreme vertebral rotation and the accompanying rib distortion. The amount of ventilatory dysfunction found in patients with scoliosis depends on the angle of the deformity, the length of the deformity, the region of the deformity, the amount of rotation of the deformity, and the age at onset.[16–18]

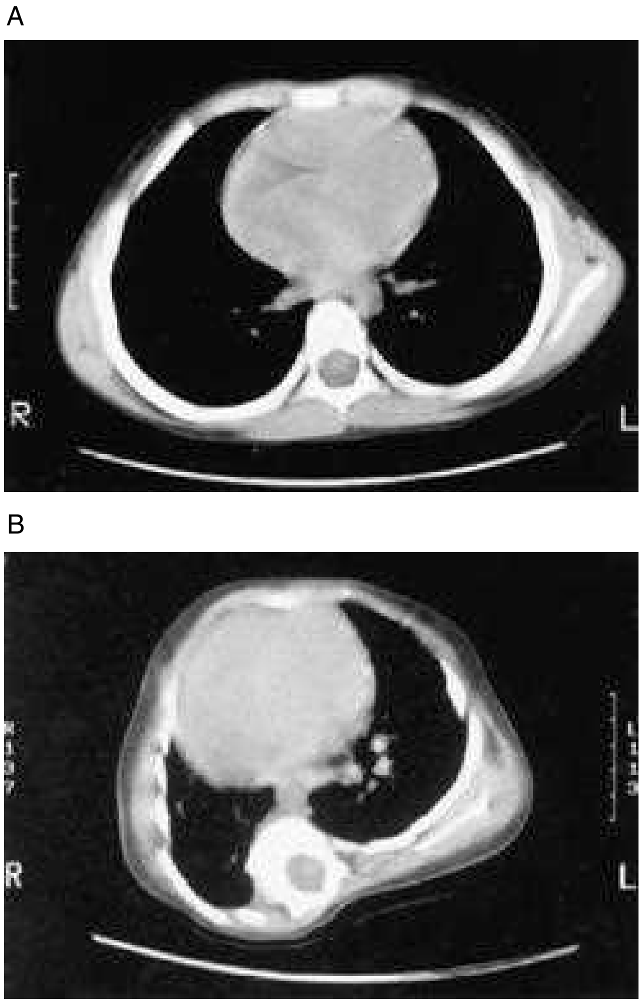


Figure 5–13 The rib cage volume changes in scoliosis. **A**. Normal ribcage. **B**. With scoliosis, the restriction is asymmetrically distributed, with the concave side of the thorax (left) decreasing in volume and the convex side (right) increasing in volume.[17] *(From Tobin MJ: Respiratory muscles in disease. Clin Chest Med 9:263, 1988, with permission.)*

CASE APPLICATION

Rib Distortion in Scoliosis

case 5–1

Mary is seen by an orthopedic physician, who confirms the measurement of her midthoracic scoliotic curve at 40°. This degree of scoliotic angulation in the midthoracic region is likely to be accompanied by rotation of the involved vertebrae and a decrease in her pulmonary reserve. This may be a contributing factor in Mary's shortness of breath while playing tennis.

Muscles Associated With the Rib Cage

The muscles that act on the rib cage are generally referred o as the **ventilatory muscles.** The ventilatory muscles are striated skeletal muscles that differ from other skeletal muscles in a number of ways: (1) they have increased fatigue resistance and greater oxidative capacity; (2) they contract rhythmically throughout life rather than episodically; (3) they work primarily against the elastic properties of the lungs and airway resistance rather than against gravitational forces; (4) neurological control of these muscles is both voluntary and involuntary; and (5) the actions of these muscles are life-sustaining.

Any muscle that attaches to the chest wall has the potential to contribute to ventilation. The recruitment of muscles for ventilation is related to the type of breathing being performed.[19] In quiet breathing that occurs at rest, only the primary inspiratory muscles are needed for ventilation. During active or forced breathing that occurs with, for example, increased activity or pulmonary pathologies, accessory muscles of both inspiration and expiration are recruited to help meet the increased demand for ventilation.

The ventilatory muscles are most accurately classified as either **primary** or **accessory muscles of ventilation.** A muscle's action during the ventilatory cycle, especially the action of an accessory muscle, is neither simple nor absolute, which makes the categorizing of ventilatory muscles as either inspiratory muscles or expiratory muscles inaccurate and misleading.

Primary Muscles of Ventilation

The primary muscles are those recruited for quiet ventilation. These include the **diaphragm,** the **intercostal**

muscles (particularly the **parasternal muscles**), and the **scalene muscles.**[20,21] These muscles all act on the rib cage to promote inspiration. There are no primary muscles for expiration because expiration at rest is passive.

Continuing Exploration 5-2:

Measures of Lung Volume and Capacity

All the air that is housed in the lungs can be divided into four segments. **Tidal volume** is the amount of air that is moved in or out during a resting breath. **Inspiratory reserve volume** is the amount of air that can be inhaled after a resting breath has already been inhaled. **Expiratory reserve volume** is the amount of air that can be exhaled after a resting breath has already been exhaled. Finally, **residual volume** is the amount of air that cannot be voluntarily exhaled no matter how hard someone tries. The term *capacity* is used when two or more lung volumes are added together. For example, **vital capacity** is a combination of inspiratory reserve volume, tidal volume, and expiratory reserve volume. Vital capacity is the volume of air that can be blown out of the lungs from a full inspiration to a full exhalation. **Inspiratory capacity** is a combination of inspiratory reserve volume and tidal volume; it is the volume of air that can be breathed in from resting exhalation. **Functional residual capacity** is a combination of expiratory reserve volume and reserve volume; it is the volume of air that remains in the lungs after a quiet exhalation. Total lung capacity is a combination of all four lung volumes. Table 5–1 summarizes the definitions graphically.

Diaphragm

The diaphragm is the primary muscle of ventilation, accounting for approximately 70% to 80% of inspiration force during quiet breathing.[20] The diaphragm is a circular set of muscle fibers that arise from the sternum, ribs, and vertebral bodies. The fibers travel cephalad (superiorly) from their origin to insert into a **central tendon.**[22,23] There is no bony insertion site, making the diaphragm unique. The boomerang-shaped central tendon forms the top of the domes of the right and left hemidiaphragms. Functionally, the muscular portion of the diaphragm is divided into dorsal and ventral segments. The ventral segment, called the **costal fibers,** arises from the sternum and ribs. The dorsal segment of the diaphragm, termed the **crural fibers,** arises from the vertebral bodies[24] (Fig. 5–14).

Table 5–1 **Lung Volumes and Lung Capacities**

LUNG VOLUMES	LUNG CAPACITIES		
Inspiratory reserve volume (IRV)	Vital capacity (VC)	Inspiratory capacity (IC)	Total lung capacity (TLC)
Tidal volume (TV)			
Expiratory reserve volume (ERV)		Functional residual capacity (FRC)	
Residual volume (RV)	Reserve capacity (RC)		

The costal fibers of the diaphragm originate from muscular slips attached to the posterior aspect of the xiphoid process, the inner surfaces of the lower six ribs, and the costal cartilages of those ribs.[22,23] The costal fibers of the diaphragm run vertically from their origin, in close apposition to the rib cage, and then curve to become more horizontal before inserting into the central tendon. The vertically traveling costal fibers of the diaphragm, which lie close to the inner wall of the lower rib cage, form a functional unit called the **zone of apposition**[6] (see Fig. 5–14A).

The crural fibers of the diaphragm originate from two tendonous structures, called the **right** and **left crus,** and from the aponeurotic **arcuate ligaments.** The right and left crus are tendons that arise from the L1 to L3 vertebrae. The tendons pass ventrally and medially, joining together to form an arch just ventral to the aorta.[9] The **medial arcuate ligament** arches over the upper anterior part of the psoas muscles and extends from the L1 or L2 vertebral body to the transverse process of L1, L2, or L3. The **lateral arcuate ligament** covers the quadratus lumborum muscles and extends from the transverse process of L1, L2, or L3 to the 12th rib[22,25] (see Fig. 5–14B).

During **tidal breathing,** the fibers of the zone of apposition of the diaphragm contract, causing a descent (but only a slight change in contour) of the dome of the diaphragm. As the dome descends, the abdominal contents are compressed, increasing intra-abdominal pressure.[25] With a deeper inhalation, the abdomen, now compressed, acts to stabilize the central tendon of the diaphragm (Fig. 5–15A). With a continued contraction of the costal fibers of the diaphragm against the stabilized central tendon, the lower ribs are now lifted and rotated outwardly in the bucket-handle motion[26–29] (see Fig. 5–15B).

The crural fibers of the diaphragm have an inspiratory effect on the central tendon of the diaphragm, but a less direct effect on the lower rib cage itself.[6,25] The action of the crural fibers of the diaphragm results in a descending of the central tendon, increasing intra-abdominal pressure. This increased pressure is transmitted across the apposed diaphragm to help the costal fibers of the diaphragm expand the lower rib cage.[6,24]

The thoracoabdominal movement during quiet inspiration is a result of the pressures that are generated by the contraction of the diaphragm. When the diaphragm contracts and the central tendon descends, there is an increase in thoracic size and a resultant decreased intrapulmonary pressure that is responsible for inspiration. There is also an increase in abdominal pressure, which causes the abdominal contents to be displaced anteriorly and laterally (Fig. 5–16). When active

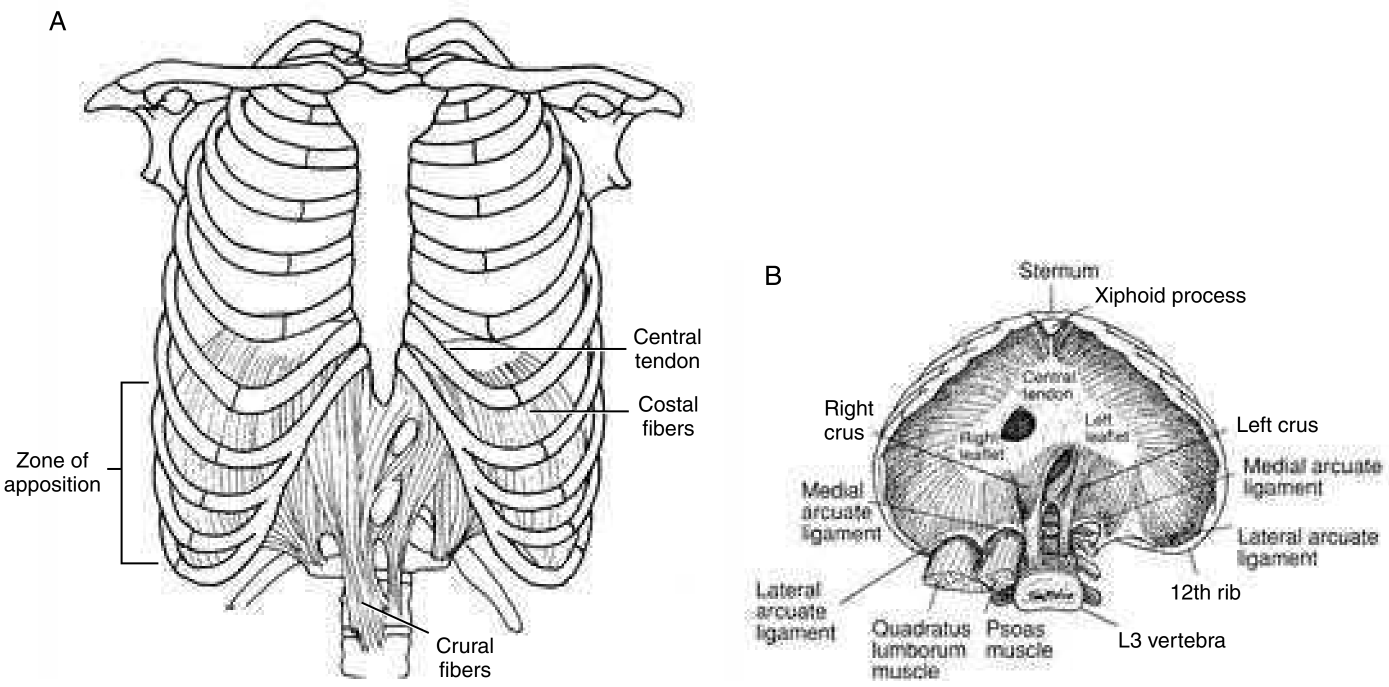


Figure 5-14 **A.** In an anterior view, the fibers of the diaphragm can be seen arising from the sternum, costocartilages, and ribs (costal fibers) and from the vertebral bodies (crural fibers). The costal fibers run vertically upward from their origin in close apposition to the rib cage and then curve and become more horizontal before inserting into the central tendon. **B.** An inferior view of the diaphragm shows the leaflets of the central tendon, the right crus, left crus, and the medial and lateral arcuate ligaments bilaterally.

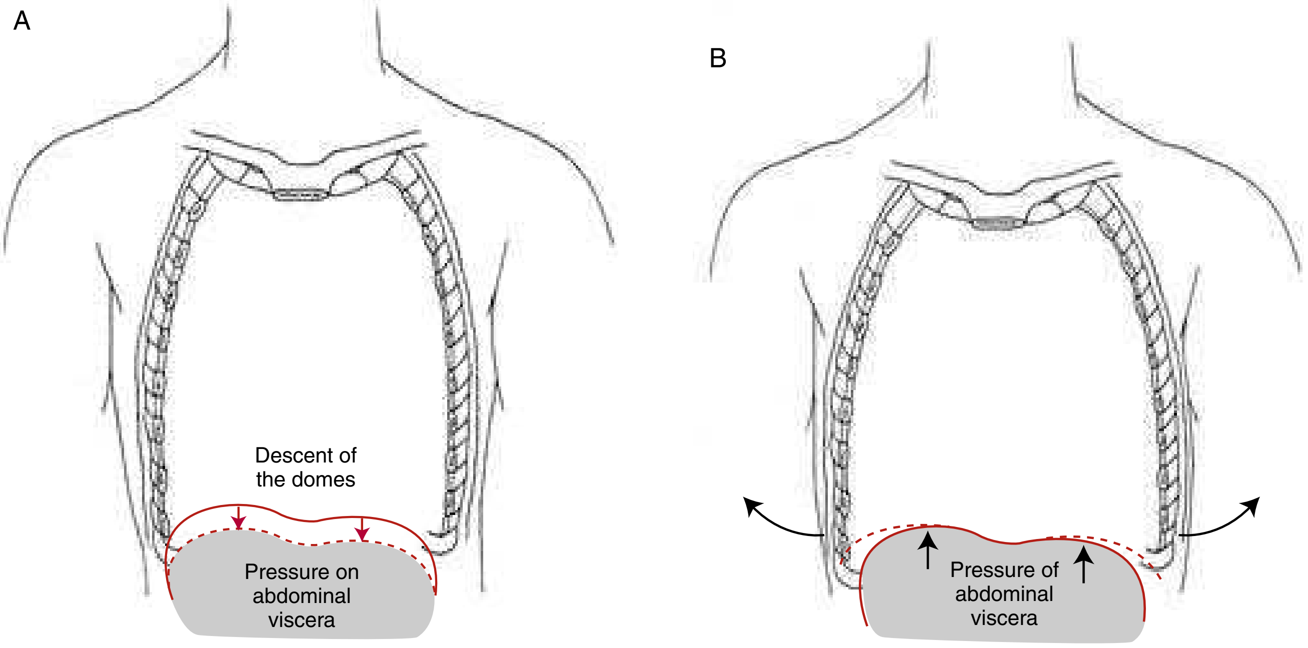


Figure 5–15 **A.** During tidal breathing, the diaphragm contracts, causing a descent of the dome of the diaphragm and an increase in pressure on the abdominal viscera. The increase in intra-abdominal pressure eventually limits further descent of (i.e., stabilizes) the central tendon of the diaphragm. **B.** Continued contraction of the costal fibers of the diaphragm on the stabilized central tendon results in expansion (bucket-handle motion) of the lower ribs.

muscle contraction of the diaphragm ceases, the domes returns to their resting position within the thorax. The thoracic volume decreases, intrapulmonary pressure increases, and exhalation occurs. The abdominal contents return to their starting position. In people with **chronic obstructive pulmonary disease (COPD),** chronic hyperinflation of the lungs results in a resting position of the diaphragm that is lower (more flattened) in the thorax than is found in healthy people. The more severe the disease, the lower and flatter the diaphragm. Eventually, even the

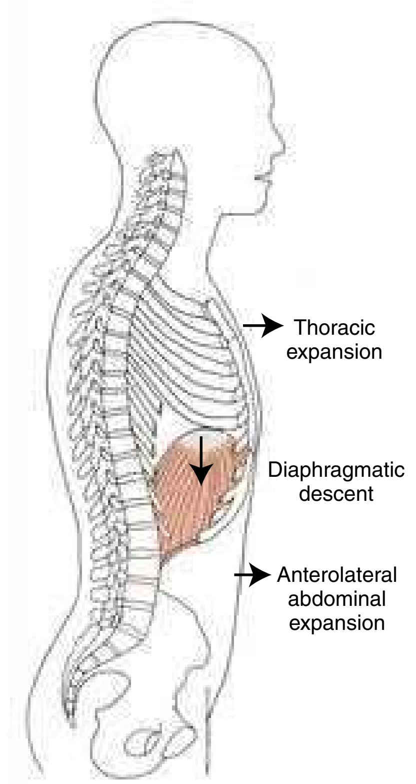


Figure 5–16 With quiet inspiration, the normal thoracoabdominal movement is caused by contraction of the diaphragm. The diaphragm descends, increasing thoracic size and displacing the abdominal viscera anteriorly and laterally. With passive exhalation, the thorax decreases in size, and the abdominal viscera return to their resting position.

zone of apposition fibers can become positioned more horizontally than vertically. An active contraction of a flattened diaphragm may pull the lower ribs inward, decreasing the thoracic size and making the diaphragm an ineffective primary muscle of inspiration (Fig. 5–17).

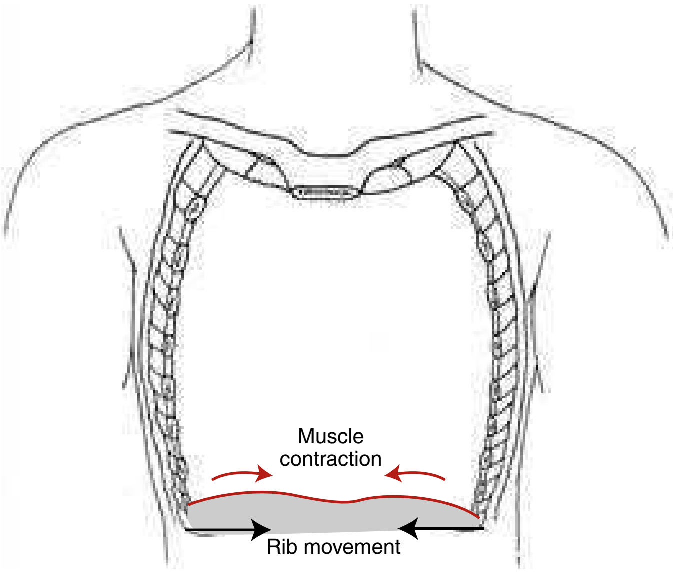


Figure 5–17 In patients with chronic obstructive pulmonary disease (COPD), the resting position of the diaphragm is flattened by hyperinflation. In severe disease, contraction of the fibers of the diaphragm pulls the lower rib cage inward.

Continuing Exploration 5-3:

Compliance

Compliance is a measurement of the distensibility of a structure or system. During diaphragm contraction, the abdomen cavity becomes the fulcrum for lateral expansion of the rib cage. Therefore, compliance of the abdomen is a factor in the inspiratory movement of the thorax.

$$\text{Compliance} = \Delta\ \text{volume}/\Delta\ \text{pressure}$$
$$\text{Compliance} = \text{change in volume per unit of pressure}$$

Increased compliance of the abdomen, as in spinal cord injury in which the abdominal musculature is no longer innervated, will affect the process of ventilation. Without the ability of the abdominal muscles to control anterior displacement of the abdominal contents, the central tendon cannot be stabilized and the costal fibers of the diaphragm cannot lift the lower ribs upward and outward. The ability to inhale fully is decreased. Decreased compliance of the abdomen, as in pregnancy, limits caudal diaphragmatic excursion and causes lateral and upward motion of the lower rib cage to occur earlier in the ventilatory cycle.

Intercostal Muscles

The **external** and **internal intercostal muscles** are categorized as ventilatory muscles. However, only the **parasternal muscles** (the portion of the internal intercostals immediately adjacent to the sternum) are considered primary muscles of ventilation. To provide a coordinated discussion of ventilatory musculature, the entire group of intercostal muscles will be described together in this section.

The internal and external intercostal and the **subcostales** muscles (Fig. 5–18) connect adjacent ribs to each other and are named according to their anatomic orientation and location. The internal intercostal muscles arise from a ridge on the inner surfaces of ribs 1 through 11 and insert into the superior border of the rib below. The fibers of the internal intercostal muscles lie deep to the external

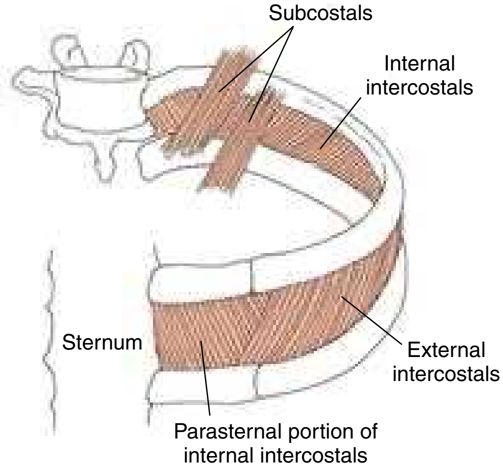


Figure 5–18 Intercostal muscles.

intercostal muscles and run caudally and posteriorly. The internal intercostals begin anteriorly at the chondrosternal junctions and continue posteriorly to the angles of the ribs, where they become an aponeurotic layer called the **posterior intercostal membrane.**

The external intercostal muscles originate on the inferior borders of ribs 1 through 11 and insert into the superior border of the rib below. The fibers run caudally and anteriorly at an oblique angle to the internal intercostal muscles.[6,30] The external intercostal muscles begin posteriorly at the tubercles of the ribs and extend anteriorly to the costochondral junctions, where they form the **anterior intercostal membrane.**

Given these attachments, only the internal intercostal muscles are present anteriorly from the chondrosternal junctions to the costochondral joints. These are the segments of the internal intercostal muscles that are referred to as the parasternal muscles and are found mainly in the first through fifth intercostal spaces.[30] There are only external intercostal muscles present posteriorly from the tubercle of the ribs to the angle of the ribs (see Fig. 5–18). Laterally, both internal and external intercostal muscle layers are present and may be referred to in this location as the **interosseous** or **lateral intercostal muscles.**[30]

The subcostal muscles (see Fig. 5–18) are also intercostal muscles but are generally found only in the lower rib cage. The subcostal muscles originate at the rib angles and may span more than one intercostal space before inserting into the inner surface of a caudal rib. Their fiber direction and action are similar to those of the internal intercostal muscles.

The functions of the intercostal muscles during ventilation are intricate and controversial. In 1749, Hamberger proposed an oversimplified theory that the external intercostal muscles tend to raise the lower rib up to the higher rib, which is an inspiratory motion, and the internal intercostal muscles tend to lower the higher rib onto the lower rib, which is an expiratory motion.[9] Electromyographic (EMG) studies have shown that, although the external intercostal muscles are active during inspiration and the internal intercostal muscles are active during exhalation,[31] both sets of intercostal muscles may be active during both phases of respiration as minute ventilation increases[32] (see the box titled "*Continuing Exploration*: Minute Ventilation"). Either set of intercostal muscles can raise the rib cage from a low lung volume or lower the rib cage from a high lung volume.[33] The activation of the intercostal muscles during the ventilatory cycle is from cranial to caudal, meaning that the recruitment of fibers begins in the higher intercostal spaces early in inspiration and moves downward as inspiration progresses. Activation of the lower intercostal muscles appears to occur only during deep inhalation.[34]

Continuing Exploration 5-4:

Minute Ventilation

Minute ventilation is the amount of air that is breathed in (or out) in one minute:

Minute ventilation (V_E) = [tidal volume] × [respiratory rate]

The parasternal muscles, the most anterior portion of the internal intercostal muscles, are considered primary inspiratory muscles during quiet breathing.[6,35] The action of the parasternal muscles appears to be a rotation of the costosternal junctions, resulting in elevation of the ribs and anterior movement of the sternum. The primary function of the parasternal muscles, however, appears to be stabilization of the rib cage.[36–38] This stabilizing action of the parasternal muscles opposes the decreased intrapulmonary pressure generated during diaphragmatic contraction, preventing a paradoxical, or inward, movement of the upper chest wall during inspiration.[38]

The function of the lateral (internal and external) intercostal muscles involves both ventilation and trunk rotation.[6,39,40] The lateral intercostal muscles, although active during the respiratory cycle, have a relatively small amount of activity in comparison with the parasternal muscles and the diaphragm.[41] The major role of the lateral intercostal muscles is in axial rotation of the thorax, with the contralateral internal and external intercostal muscles working synergistically to produce trunk rotation (e.g., right external and left internal intercostal muscles are active during trunk rotation to the left).[41]

Scalene Muscles

The scalene muscles are also primary muscles of quiet ventilation.[21–30] The scalene muscles attach to the transverse processes of C3 to C7 and descend to the upper borders of the first rib (scalenus anterior and scalenus medius) and second rib (scalenus posterior) (Fig. 5–19). Their action lifts the first two ribs, and therefore the sternum, in the pump-handle motion of the upper rib cage.[21,26,35] Activity of the scalene muscles begins at the onset of inspiration and increases as inspiration gets closer to total lung capacity. The length-tension relationship of the scalene muscles allows

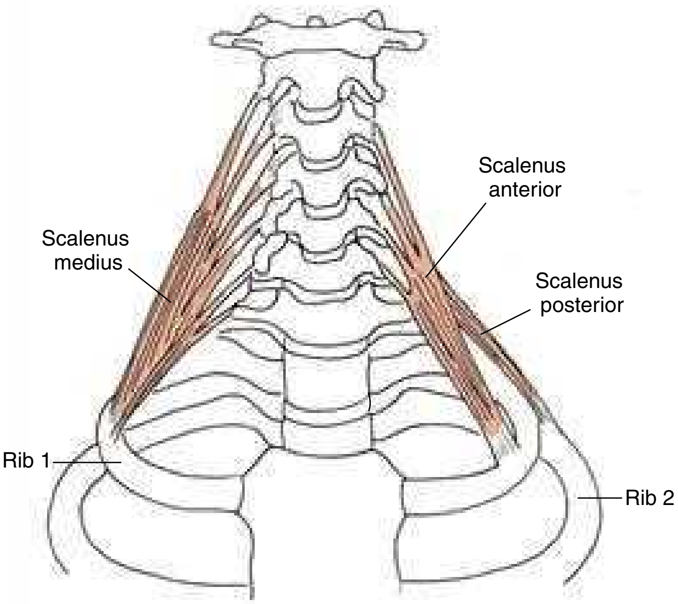


Figure 5–19 The scalenus anterior, scalenus medius, and scalenus posterior. Their action lifts the sternum and the first two ribs in the pump-handle motion.

them to generate a greater force late into the respiratory cycle, when the force from the diaphragm is decreasing. The scalene muscles also function as stabilizers of the rib cage. The scalene muscles, along with the parasternal muscles, counteract the paradoxical movement of the upper chest caused by a decrease in intrapulmonary pressure, which is created by the diaphragm's contraction.

Accessory Muscles of Ventilation

The muscles that attach the rib cage to the shoulder girdle, head, vertebral column, or pelvis may be classified as accessory muscles of ventilation. These muscles assist with inspiration or expiration in situations of stress, such as increased activity or disease.

When the thorax is stabilized, the accessory muscles of ventilation move the vertebral column, arm, head, or pelvis on the trunk. During times of increased ventilatory demand, the rib cage can become the mobile segment. The accessory muscles of inspiration, therefore, increase the thoracic diameter by moving the rib cage upward and outward.[26] The accessory muscles of expiration move the diaphragm upward and the thorax downward and inward. The most commonly described accessory muscles are shown in Figure 5–20 and are discussed in the following paragraphs.

The **sternocleidomastoid** muscle runs from the manubrium and superior medial aspect of the clavicle to the mastoid process of the temporal bone. The usual bilateral action of the sternocleidomastoid is flexion of the cervical spine. With the help of the **trapezius** muscle to stabilize the head, the bilateral action of the sternocleidomastoid muscles can pull the rib cage superiorly, expanding the upper rib cage in the pump-handle motion. The recruitment of this muscle seems to occur toward the end of a maximal inspiration.[42]

The sternocostal portion of the **pectoralis major** muscle can elevate the upper rib cage when the shoulders and the humerus are stabilized. The clavicular head of the pectoralis major can be either inspiratory or expiratory in action, depending on the position of the upper extremity. When the arms are positioned so that the humeral attachment of the pectoralis major is below the level of the clavicle, the clavicular portion acts as an expiratory muscle by pulling the manubrium and upper ribs downward. With the humeral attachment of the pectoralis major above the level of the clavicle, such as when the arms are raised overhead, the muscle becomes an inspiratory muscle, pulling the manubrium and upper ribs up and out. The **pectoralis minor** can help elevate the third, fourth, and fifth ribs during active inspiration. The **subclavius,** a muscle between the clavicle and the first rib, can also assist in raising the upper chest for inspiration.

Posteriorly, the fibers of the **levatores costarum** run from the transverse processes of vertebrae C7 through T11 to the posterior external surface of the next lower rib

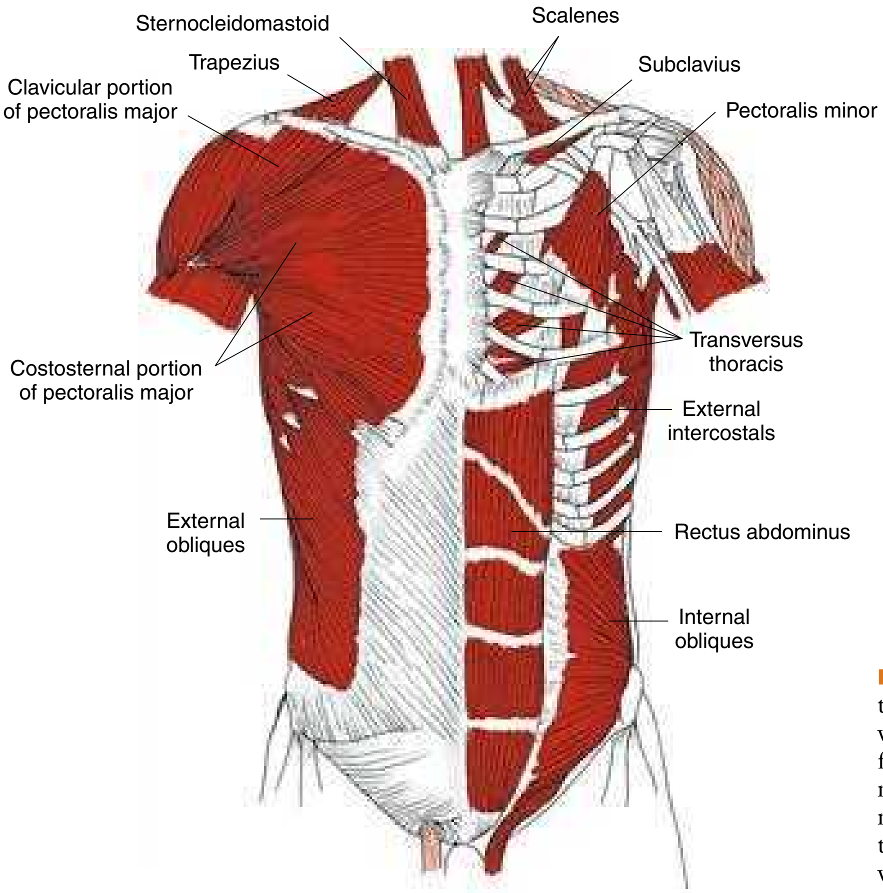


Figure 5–20 Accessory muscles of ventilation are those used during times of increased ventilatory demand. The right side of the figure shows some of the anterior superficial muscles of the thorax that can be accessory muscles of ventilation, and the left side of the thorax shows the deeper accessory muscles of ventilation.

between the tubercle and the angle and can assist with elevation of the upper ribs.[13,43] The **serratus posterior superior** has its superior attachment at the spinous processes of the lower cervical and upper thoracic vertebrae and attaches caudally via four thin bands just lateral to the angles of the second through fifth ribs. The serratus posterior superior and the **serratus posterior inferior** have been assumed to be accessory muscles of respiration based in large part on their anatomical origins and insertions. There is, to date, no EMG evidence to support a ventilatory role for these muscles; therefore they should not, at present, be considered to have a respiratory function.[44]

The abdominal muscles (**transversus abdominis, internal oblique abdominis, external oblique abdominis,** and **rectus abdominis**) are also accessory muscles of ventilation, as well as trunk flexors and rotators. The major function of the abdominal muscles in ventilation is to assist with forced expiration. The muscle fibers pull the ribs and costocartilages caudally, into a motion of exhalation. By increasing intra-abdominal pressure, the abdominal muscles can force the diaphragm upward into the thoracic cage, increasing both the volume and speed of exhalation.

Although usually considered accessory muscles of exhalation, the abdominal muscles play two significant roles during inspiration. First, the increased abdominal pressure created by lowering of the diaphragm in inspiration must be countered by tension in the abdominal musculature. Without sufficient compliance in the abdominal muscles, the central tendon of the diaphragm cannot be effectively stabilized and lateral chest wall expansion cannot occur. Secondly, the increased intra-abdominal pressure created by the active abdominal muscles during forced exhalation pushes the diaphragm cranially and exerts a passive stretch on the costal fibers of the diaphragm.[6] These changes prepare the respiratory system for the next inspiration by optimizing the length-tension relationship of the muscle fibers of the diaphragm. During periods of increased ventilatory needs, the increased muscular activity of the abdominal muscles assists in both exhalation and inhalation.[6,23]

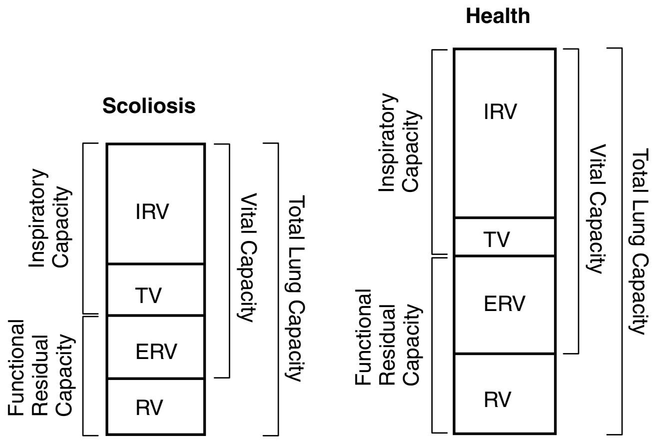
The **transversus thoracis (triangularis sterni)** muscles are a flat layer of muscle that runs deep to the parasternal muscles. The transversus thoracis muscles originate from the posterior surface of the caudal half of the sternum and run cranially and laterally, inserting into the inner surface of the costal cartilages of ribs 3 through 7.[6] These muscles are recruited, along with the abdominal muscles, to pull the rib cage caudally. The mechanical advantage of the triangularis sterni is greater the more caudal the intercostals space attachment.[30] Studies have shown that these muscles are primarily expiratory muscles, especially when expiration is active, as in talking, coughing, or laughing, or in forced exhalation into functional residual capacity.[45,46]

Gravity acts as an accessory force for ventilation in the supine position. Gravity, acting on the abdominal viscera, performs the same function as the abdominal musculature in stabilizing the central tendon of the diaphragm. In fact, in the supine position, the abdominal muscles and the trangularis sterni are silent on the EMG monitoring during quiet breathing.

Continuing Exploration 5-5:

Ventilatory Changes in Scoliosis

Not only do the anatomical changes that occur in scoliosis alter the alignment and motion of the thorax, but they also affect the length-tension relationship and the angle of pull of the muscles of ventilation. On the convex side of the scoliotic curve, with sufficient curvature, the intercostal space is widened and the intercostal muscles are elongated. On the concave side of the curve, the ribs are approximated and the intercostal muscles are adaptively shortened (see Fig. 5–2A). Lung volumes and capacities are reduced from those found in healthy people as a result of the altered biomechanics of the scoliotic thorax[47] (Fig. 5–21).



IRV = Inspiratory reserve volume; TV = Tidal volume;
ERV = Expiratory reserve volume; RV = Residual volume

Figure 5–21 Lung volumes and capacities of a healthy person and of a patient with scoliosis.

CASE APPLICATION

Treatment for Scoliosis

case 5–2

Accepted medical treatment for scoliosis when the curve is small, less than 20° to 25°, is usually only periodic monitoring.[48] Mary's curve was first diagnosed at 40°. A curve of this size is likely to cause a ventilatory restriction. Curves between 25° and 40° may initially be braced as the first line of intervention.[16,49] The purpose of bracing is to limit further progression of the curve.[50] The Boston Scoliosis Brace (Fig. 5–22) is a variation of a more standard thorocolumbosacral orthosis, either of which would be options for Mary at this time. Both braces place direct pressure on the rib cage in order to hold the scoliotic curve firmly, which decreases the thoracic mobility necessary for ventilation. These braces also have a tight-fitting abdominal

Continued

Figure 5–22 The Boston Scoliosis Brace consists of a firmly fitting pelvic section that extends upward anteriorly to approximately the level of rib 4 and posteriorly to the level of the scapula. The brace applies forces to the ribs in a way that helps limit the progression of the scoliotic curvature.

component that increases intra-abdominal pressure, restricting the descent of the diaphragm. Lung volumes and capacities are noted to be reduced by approximately 15% to 20% while the Boston Brace is worn.[51,52] This impairment, although significant, is reversible when the brace is removed.[53]

Mary, at age 12, is still skeletally immature, and her 40° curve may increase as she grows. If bracing is not successful in limiting progression of the curve, surgical correction will be the next line of intervention. The time to surgically intervene depends on a number of factors, including severity of the curve, progression of the curve, level of function, and pain. There is an increasing incidence of surgical correction for smaller curves because bracing is "both burdensome and of questionable effectiveness."[54] Another indication for surgical intervention is the amount of pulmonary restriction present. Patients whose pulmonary function is severely compromised are thought to be poor operative risks, and some patients may not be offered surgical correction as a result. However, Wazeka and colleagues found that surgical intervention, even in patients with very low vital capacities, was well tolerated.[55] In general, surgical treatment of scoliosis substantially reduces or corrects the lateral curvature of the spine. Pulmonary function tests show that any accompanying restrictions to ventilation are improved with surgical intervention, although they may not be fully normalized.[56,57] Failure to normalize pulmonary mechanics postoperatively may occur because of an incomplete correction of the lateral and spinal deviations, irreversible pulmonary parenchymal changes, continued rotation of the vertebrae, or decreased flexibility of the thoracic spine.[56]

Coordination and Integration of Ventilatory Motions

The coordination and integration of the skeletal and muscular chest wall components during breathing are complex and difficult to measure. Investigators have used EMG techniques, electrical stimulation, ultrasound, computed tomography scans, and computerized motion analysis techniques to analyze and describe chest wall motion and muscular actions.[31,39–41,43] Studies have confirmed the complexity of the coordinated actions of the many muscle groups involved even in quiet breathing. The recruitment of ventilatory muscles is dependent on the activities in which a person is participating, including not only sports, household, and job activities but also maintenance of posture, locomotion, speech, and defecation. A high and complex level of coordination is necessary for the primary and accessory muscles of ventilation to contribute to additional tasks while they continue to perform the necessary function of ventilation.

Concept Cornerstone 5-2

Summary of the Ventilatory Sequence During Breathing

Although the coordinated function and sequence of breathing are complex when activities are combined, the following sequence of motions and muscle actions is typical of a healthy person at rest during quiet breathing. The diaphragm contracts and the central tendon moves caudally. The parasternal and scalene muscles stabilize the anterior upper chest wall to prevent a paradoxical inward movement caused by the decreasing intrapulmonary pressure. As intra-abdominal pressure increases, the abdominal contents are displaced in such a way that the anterior epigastric abdominal wall is pushed ventrally. Further outward motion of the abdominal wall is countered by the abdominal musculature, which allows the central tendon to stabilize on the abdominal viscera. With continued shortening of the appositional (costal) fibers of the diaphragm, the lower ribs are pulled cephalad and laterally, which results in the bucket-handle movement of the lower ribs. With continued inspiration, the parasternal, scalene, and levatores costarum muscles actively rotate the upper ribs and elevate the manubriosternum, which results in an anterior motion of the upper ribs and sternum. The lateral motion of the lower ribs and anterior motion of the upper ribs and sternum can occur simultaneously. Expiration during quiet breathing is passive, involving the use of the recoil of the elastic components of the lungs and chest wall.

DEVELOPMENTAL ASPECTS OF STRUCTURE AND FUNCTION

Differences Associated With the Neonate

The compliance, configuration, and muscle action of the chest wall changes significantly from infants to the elderly. A newborn has a cartilaginous and therefore extremely compliant chest wall, which allows the distortion necessary for the infant's thorax to travel through the birth canal. The increased compliance of the rib cage is at the expense of thoracic stability. An infant's chest wall muscles must act as stabilizers, rather than mobilizers, of the thorax to counteract the reduced intrapulmonary pressure created by the lowered diaphragm during inspiration. Complete ossification of the ribs does not occur for several months after birth.

Whereas the ribs in the adult thorax slope downward and the diaphragm is elliptically shaped (Fig. 5–23A), the rib cage of an infant shows a more horizontal alignment of the ribs, with the angle of insertion of the costal fibers of the diaphragm also more horizontal than that of an adult (Fig. 5–23B). There is an increased tendency for these fibers to pull the lower ribs inward, thereby decreasing ventilation efficiency and increasing distortion of the chest wall.[58–59] There is very little motion of the rib cage during an infant's tidal breathing.

Only 20% of the muscle fibers of the diaphragm are fatigue-resistant fibers in a healthy newborn, compared to 50% in an adult. This discrepancy predisposes infants to earlier diaphragmatic fatigue.[59] Accessory muscles of ventilation are also at a disadvantage in the infant. Until infants can stabilize their upper extremities, head, and spine, it is difficult for the accessory muscles of ventilation to produce the action needed to be helpful during increased ventilatory demands.

As an infant ages and the rib cage ossifies, muscles can begin to mobilize rather than stabilize the thorax. As infants gain head control, they are also gaining accessory muscle use for increased ventilation. As a toddler assumes the upright positions of sitting and standing, the force of gravity and postural changes allow the anterior rib cage to angle obliquely downward over time. This elliptical thorax allows for a greater bucket-handle motion of the rib cage. The attachments for the muscles of ventilation move with the increasingly angled ribs, improving their action on the thorax. Throughout childhood, the numbers of alveoli and airways continue to increase.[60] In early adolescence, the sizes of the alveoli and airways continue to expand, as demonstrated by increases in pulmonary function test results.

Differences Associated With the Elderly

Skeletal changes that occur with aging affect pulmonary function. Many of the articulations of the chest wall undergo fibrosis with advancing age.[61,62] The interchondral and costochondral joints can fibrose, and the chondrosternal

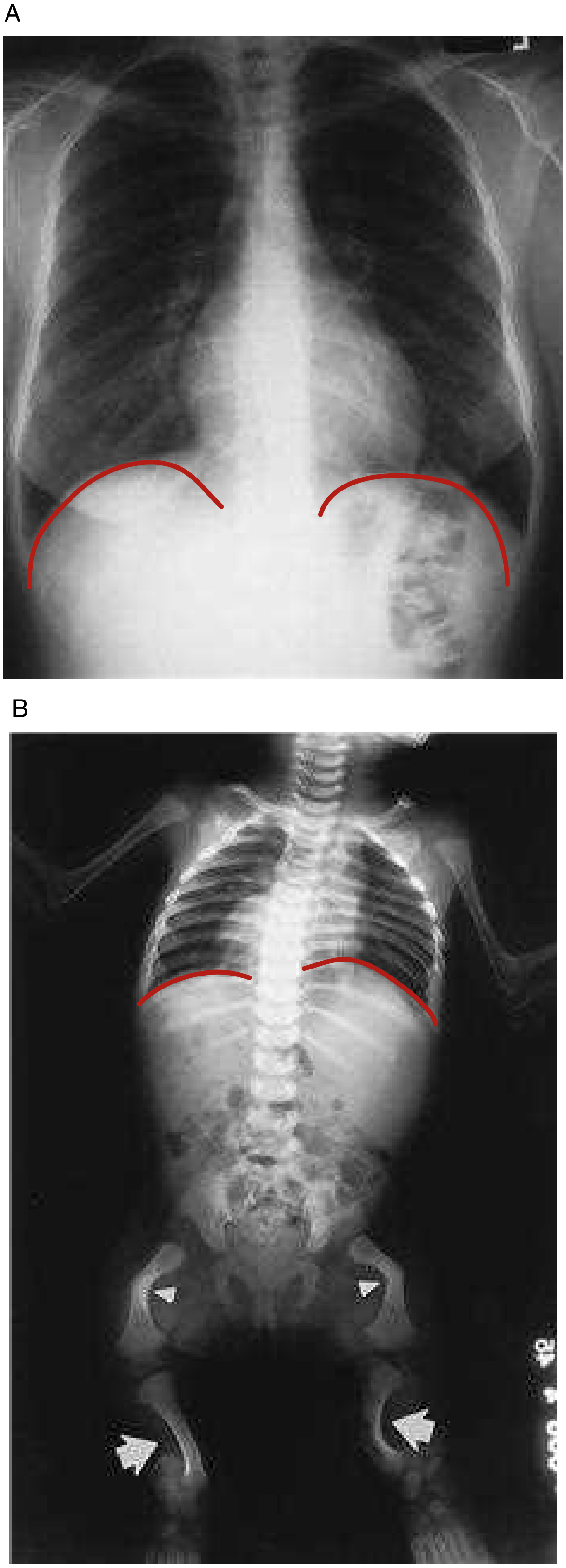


Figure 5–23 **A.** In an adult, the ribs slope downward, and the diaphragm has an elliptical shape. **B.** The rib cage of an infant shows a nearly horizontal alignment of the ribs, and the angle of insertion of the costal fibers of the diaphragm is also more horizontal.

joints may be obliterated. The xiphosternal junction usually ossifies after age 40. The chest wall articulations that are true synovial joints may undergo morphological changes associated with aging, which results in reduced mobility. The costal cartilages ossify, which interferes with their axial rotation.[14] Overall, chest wall compliance is significantly reduced with age. Reduction in diaphragm-abdomen compliance has also been reported and is at least partially related to the decreased rib cage compliance, especially in the lower ribs that are part of the zone of apposition.[63]

Aging also brings anatomical changes to the lung tissues that affect the function of the lungs. The airways narrow, the alveolar duct diameters increase, and the alveolar sacs become shallower. There is a reorientation and decrease of the elastic fibers. Overall, there is a decrease in elastic recoil and an increase in pulmonary compliance.[62] Because the resting position of the thorax depends on the balance between the elastic recoil properties of the lungs pulling the ribs inwardly and the outward pull of the bones, cartilage, and muscles, the reduced recoil property of the lung tissue allows the thorax to rest with an increased A-P diameter, a relatively increased inspiratory position. An increased kyphosis is often observed in older individuals, which decreases the mobility not only of the thoracic spine but also of the rib cage.

The result of these age-related skeletal and tissue changes is an increase in the amount of air remaining in the lungs after a normal exhalation (i.e., an increase in functional residual capacity). If the lungs retain more air at the end of exhalation, there will be a decrease in inspiratory capacity of the thorax. Functionally, the changes result in a decrease in the ventilatory reserve available during times of need, such as during an illness or increased activity.

Skeletal muscles of ventilation in the elderly have a documented loss of strength, fewer muscle fibers, a lower oxidative capacity, a decrease in the number or the size of fast-twitch type II fibers, and an increase in the time to peak tension.[62–64] The resting position of the diaphragm becomes less domed, with a decrease in abdominal tone in aging.[14] There is an early recruitment pattern for accessory muscles of ventilation. For example, the transverse thoracic muscles are active during quiet exhalation in older subjects in the standing position.[45]

Concept Cornerstone **5-3**

Summary of Rib Cage Changes With Aging

In the elderly, there is likely to be a decreased compliance of the bony rib cage, an increased compliance of the lung tissue, and an overall decreased compliance of the respiratory system as a result of the effects of aging. There is a decrease in the effectiveness of the ventilatory muscles, and ventilation becomes more energy-expensive with age. There is a decreased ventilatory reserve available during times of increased ventilatory need, such as increased activity or illness.

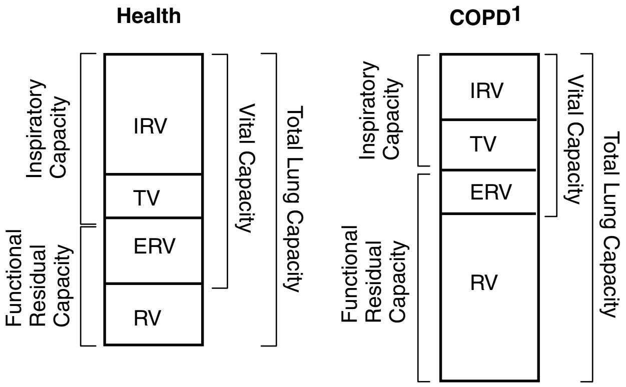
PATHOLOGICAL CHANGES IN STRUCTURE AND FUNCTION

In this chapter, the effects of the musculoskeletal system on ventilation have been discussed. In scoliosis, a change in the musculoskeletal structure renders a change to ventilation. It is interesting to note that the opposite can also be true; changes in the pulmonary system can affect the biomechanics of the thorax. A brief discussion of this relationship is presented, with chronic obstructive pulmonary disease as the framework.

Chronic Obstsructive Pulmonary Disease (COPD)

The major manifestation of COPD is damage to the airways and destruction of the alveolar walls. As tissue destruction occurs with disease, the elastic recoil property of the lung tissue is diminished. Passive exhalation is dependent upon this elastic recoil property, and in patients with COPD, exhalation becomes ineffective in removing an adequate amount of air from the thorax, leading to air trapping and hyperinflation.

The static or resting position of the thorax is a balance between the elastic recoil properties of the lungs pulling inward and the normal outward spring of the rib cage. In COPD, there is an imbalance in these two opposing forces that alters both lung volumes and ventilatory capacities (Fig. 5–24). As elasticity decreases and more air is left within the lungs, an increase in the A-P diameter of the hyperinflated thorax (it becomes more of a barrel shape) is apparent, along with flattening of the diaphragm at rest (Fig. 5–25).



IRV = Inspiratory reserve volume; TV = Tidal volume;
ERV = Expiratory reserve volume; RV = Residual volume

[1] COPD = Chronic obstructive pulmonary disease

Figure 5–24 Lung volumes and capacities of a healthy person and of a patient with COPD.

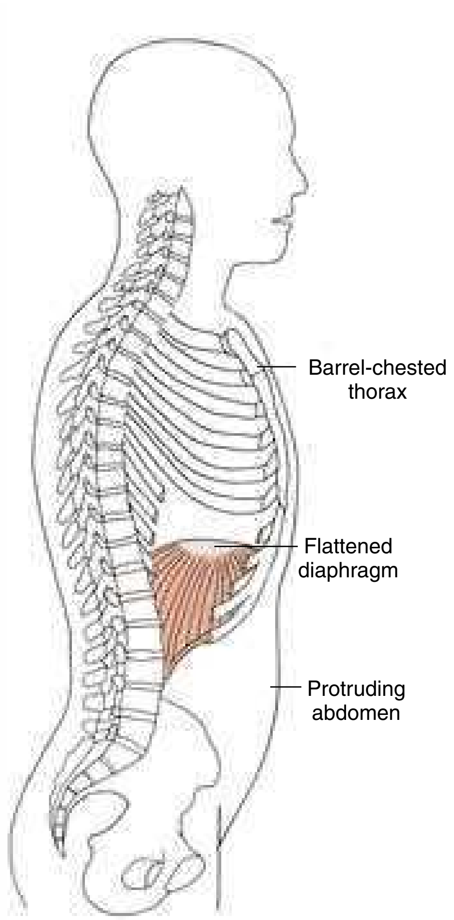


Figure 5–25 Resting position of a person with COPD. The thorax is barrel-shaped, the diaphragm is flattened from hyperinflation, and the abdomen protrudes as a result of increased intra-abdominal pressure.

The range of motion, or excursion, of the thorax is limited in obstructive pulmonary disease. Although the basic characteristic of COPD is an inability to exhale sufficiently, it is clear that inspiratory capacity is compromised as a result.

Hyperinflation affects not only the bony components of the chest wall but also the muscles of the thorax. The fibers of the diaphragm are shortened, decreasing the available range of contraction. The angle of pull of the flattened diaphragm fibers becomes more horizontal with a decreased zone of apposition. In severe cases of hyperinflation, the fibers of the diaphragm are aligned horizontally. Contraction of this very flattened diaphragm will pull the lower rib cage inward, actually working against lung inflation[65] (see Fig. 5–17).

With the compromise of the diaphragm in COPD, the majority of inspiration is performed by other inspiratory muscles, which are not as efficient as the diaphragm. The barrel-shaped and elevated thorax puts the sternocleidomastoid muscles in a shortened position, making them much less efficient. The parasternal and scalene muscles are able to generate a greater force as the lungs approach total lung capacity; consequently, hyperinflation has a less dramatic effect on them.[66] The diaphragm has a limited ability to laterally expand the rib cage, and so inspiratory motion must occur within the upper rib cage. With a forceful contraction of the functioning inspiratory muscles of the upper rib cage in a patient with an ineffective diaphragm, the diaphragm and the abdominal contents may actually be pulled upwards, into the thorax.[67] During exhalation, the abdomen is pushed back down and out. This paradoxical thorocoabdominal breathing pattern can be seen in patients with obstructive disease who are in respiratory distress (Fig. 5–26). The paradoxical pattern is a reflection of the maintained effectiveness of the upper inspiratory rib cage musculature and the reduced effectiveness of the diaphragm.[68] The disadvantages of these biomechanical alterations with hyperinflation are compounded by the increased demand for ventilation in COPD. More work is required of a less effective system. The energy cost of ventilation, or the work of breathing, is markedly increased in COPD.

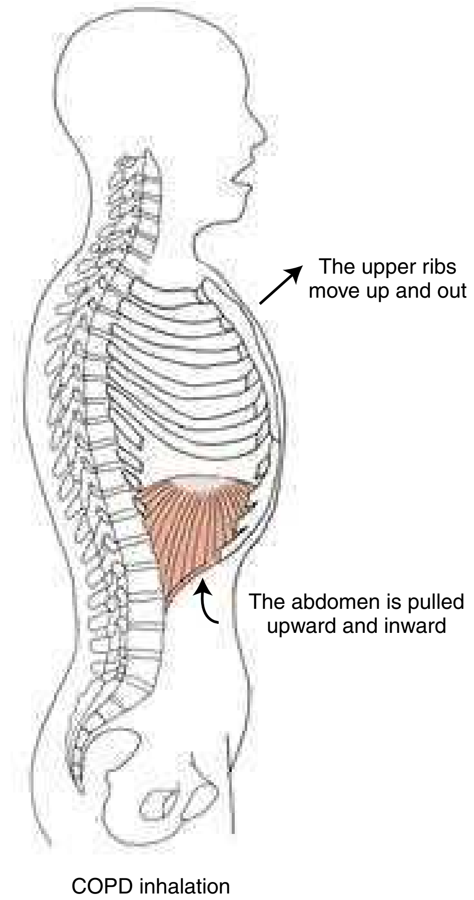
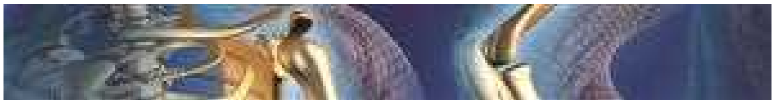


Figure 5–26 Paradoxical thorocoabdominal movement in COPD. With a strong pull of the accessory muscles of inspiration, there is an increase in the motion of the upper chest. Because the diaphragm is ineffective in descending, the abdominal viscera are pulled in and up. With exhalation, the thorax decreases in size, and the abdominal viscera return to their resting position.

SUMMARY

In this chapter, comprehensive coverage of the structure and function of the bony thorax and the ventilatory muscles has been provided. Additional information on the structure and function of accessory muscles of ventilation as these muscles may affect the shoulder complex will be presented in Chapter 7.

STUDY QUESTIONS



1. Describe the articulations of the chest wall and thorax, including the costovertebral, costotransverse, costochondral, chondrosternal, and manubriosternal joints.
2. What are the different motions of the chest wall motions during breathing? Explain where these motions occur.
3. What are the roles of the diaphragm, the intercostal muscles, and the abdominal muscles during breathing?
4. Describe the accessory muscles of ventilation and explain their functions.
5. Describe the inspiratory and expiratory functions of the abdominal muscles.
6. What effect does COPD have on the biomechanics of the thorax and the inspiratory muscles?
7. How does the aging process affect the structure and function of the thorax?

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Chapter 6

The Temporomandibular Joint

Pamela D. Ritzline, PT, EdD

Introduction

Joint Structure
- Articular Structures
- Accessory Joint Structures
- Capsule and Ligaments

Joint Function
- Joint Kinematics
  - Mandibular Depression and Elevation
  - Mandibular Protrusion and Retrusion
  - Mandibular Lateral Excursion
- Muscles
  - Primary Muscles
  - Secondary Muscles
  - Coordinated Muscle Actions
- Nerves
- Relationship to the Cervical Spine and Posture
- Dentition

Common Impairments and Pathologies
- Age-Related Changes in the TM Joint
- Inflammatory Conditions
  - Capsular Fibrosis
  - Osseous Mobility Conditions
  - Articular Disc Displacement
  - Degenerative Conditions

INTRODUCTION

The **temporomandibular (TM) joint** is unique in both structure and function. Structurally, the mandible is a horseshoe-shaped bone (Fig. 6–1) that articulates with the temporal bone at each posterior superior end and produces two distinct but highly interdependent articulations. Each TM joint contains a disc that separates the joint into upper and lower articulations. Functionally, mandibular movement involves concurrent movement in the four distinct joints, resulting in a complex structure that moves in all planes of motion to achieve normal function.

This chapter will introduce the TM joint. A discussion of the structure and function of the TM joint will allow you to appreciate and understand its unique features, its relationship with the cervical spine, and the impact of impairments and pathologies to the TM joint. A patient case scenario will provide the foundation for subsequent discussions. Although the purpose of this chapter is to discuss the normal function and structure of the TM joint, TM disorders are a common subgroup of orofacial pain disorders.[1] This chapter will also introduce some of the common problems that involve deviations of normal structure.

6-1 **Patient Case** *case*

Jill Smith is a 32-year-old single mother with three children under the age of 6. She works as an office manager in a busy law firm. Recently she experienced the onset of frequent headaches and intermittent pain in her right ear as well as intermittent pain with occasional "popping" at her right jaw when she opens her mouth. She describes the headaches as a dull throb that starts at the back of her head and radiates over the top of her head to just behind her right eye. Jill reports edema on the right side of her face anterior to her ear. Jill is right-handed. Her medical history is unremarkable except for a history of allergies and a fall from her bicycle when she was 12 years old, hitting her chin on the handle bars on the way down. Radiographs taken at the time of the accident were negative for any fractures. Jill reports that she first noticed occasional, intermittent headaches after the bicycle accident. She indicates that the intensity and frequency of the headaches have gotten progressively worse. The symptoms anterior to her right ear are activated when she attempts to eat something chewy, hard, or large.

Jill's physical examination reveals a forward head posture, with rounded shoulders, winging scapulae, increased thoracic kyphosis, increased lumbar lordosis, hyperextended knees, and pronated feet. Her right shoulder is elevated slightly. Active movements of the mandible are restricted when performing mandibular depression, protrusion, and left lateral excursion. The mandible deflects to the right with mandibular depression. Active range of motion of the cervical spine is limited, especially in the upper levels. Passive mobility of the right TM joint reveals limitations with distraction, anterior translation, and lateral glide. Passive mobility of the cervical spine is restricted. Palpation produces complaints of tenderness around the right mandibular condyle. Palpation of the suboccipital muscles reproduces her headache symptoms. The C1 and C2 vertebrae are rotated. Moderate muscle guarding is noted throughout the anterior and posterior neck and upper back. The pectoralis major and minor muscles, along with the latissimus dorsi muscles, are shortened, indicating decreased flexibility. Jill's strength and reflexes are within normal limits, although she has difficulty with motor control of her middle and lower trapezius muscles. Screening of the other body systems is negative. Diagnostic imaging of the head and neck is unremarkable. She has not had a cephalogram.

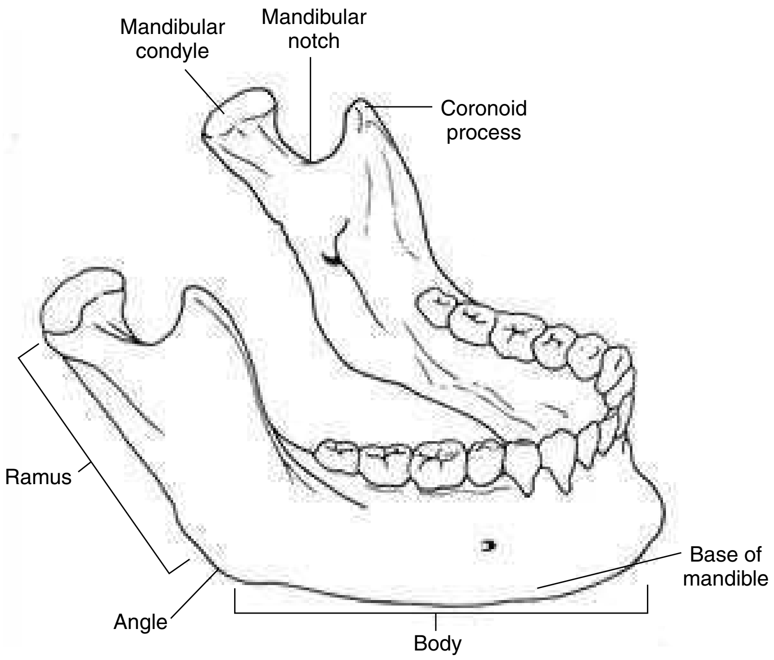


Figure 6–1 The mandible.

JOINT STRUCTURE

Articular Structures

Multiple bones merge to form the structure and contribute to the function of the TM joints. These bones include the **mandible, maxillae, temporal, zygomatic, sphenoid,** and **hyoid** bones[2] (Fig. 6–2). The proximal or stationary segment of the TM joint is the temporal bone. The condyles of the mandible sit in the **mandibular fossa** of the temporal bone (Fig. 6–3). The mandibular fossa is located between the **postglenoid tubercle** and the **articular eminence** of the temporal bone.[3,4]

Overall, the temporal bones and the mandible combine to create two separate yet connected TM joints. The two TM joints are part of a closed chain and must function together; however, each is an anatomically independent joint, and each can vary structurally as well as functionally. Structurally, the individual TM joints are considered to be synovial joints formed by the **condyle of the mandible** inferiorly and the articular eminence of the temporal bone superiorly.[2,5,6] Although the condyle of the mandible sits in the mandibular fossa, the bone in the fossa is thin and translucent and therefore is not at all appropriate as an articular surface. In contrast, the articular eminence contains a major area of trabecular bone and serves as the primary articular surface for the mandibular condyle**.** Thus, functionally, the mandibular condyle articulates with the articular eminence of the temporal bone. The articular eminence and the condyle are both convex structures, resulting in an incongruent joint[2–5] (Fig. 6–4).

The TM joint is classified as a synovial joint, although no hyaline cartilage covers the articular surfaces.[6,7] The articular surfaces of the articular eminence and the mandibular condyle are covered with dense, avascular, collagenous tissue that contains some cartilaginous cells.[8] Because some of the cells are cartilaginous, the covering is often referred to as fibrocartilage. The articular collagen fibers are aligned perpendicular to the bony surface in the deeper layers to withstand stresses. The fibers near the surface of the articular covering are aligned in a parallel arrangement to facilitate gliding of the joint surfaces.[2,7,9] The presence of fibrocartilage rather than hyaline cartilage is significant because fibrocartilage can repair and remodel itself.[6,7,9] Typically fibrocartilage is present in areas that have to withstand repeated and high-level stress. The TM joints are subjected to the repetitive stress of jaw motions as well as to tremendous bite forces, which have been measured at 597 N for women and 847 N for men.[10] The TM joint surfaces are amenable to some degree of adaptation, but no clear-cut point exists between adaptive and maladaptive changes.[11] Tanaka and Koolstra[6] acknowledged that loading is necessary to stimulate the remodeling that occurs with normal functional demands to ensure homeostasis within a joint. Although no

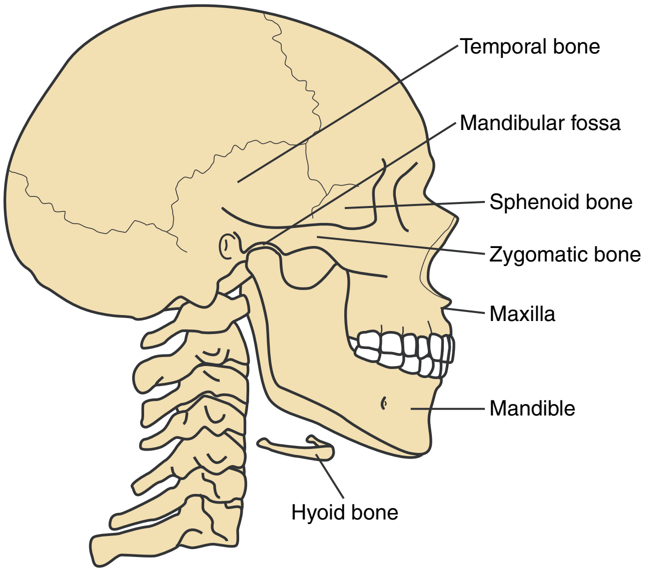


Figure 6–2 Lateral view of the mandible, maxilla, temporal bone, zygomatic bone, sphenoid bone, and hyoid bone.

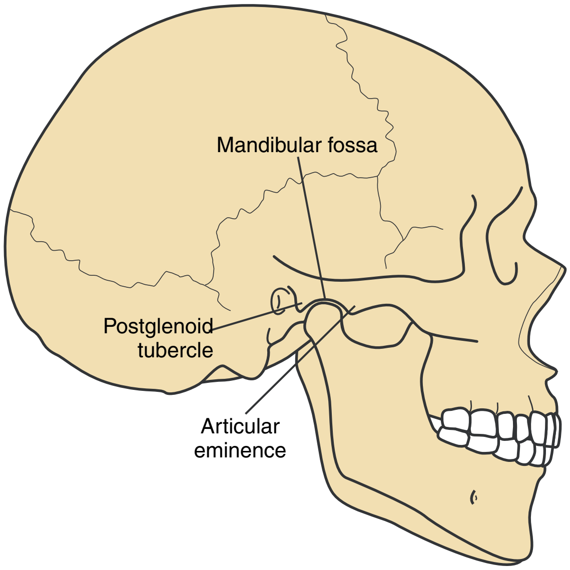


Figure 6–3 The bony composition of the temporomandibular joint.

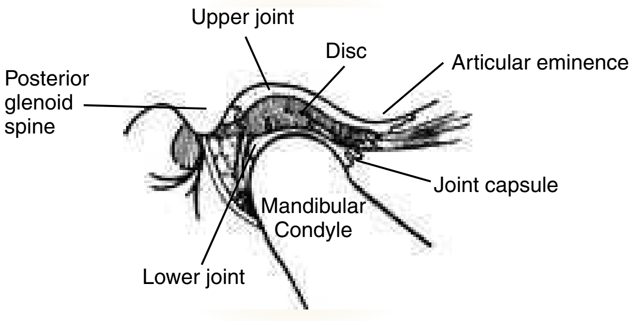


Figure 6–4 A cross-sectional lateral view of the TM joint shows the fibrocartilage-covered load-bearing surfaces on the condyle of the mandible and the articular eminence. The TM disc divides the articulation into an upper joint and a lower joint, each with its own synovial lining. The anterior and posterior attachments of the joint capsule to the disc are shown.

evidence exists to determine the appropriate load for the TM joint, it has been proposed that aging and some physical and mental conditions may disrupt normal load.[6]

The mandible is the largest of the facial bones and is highly mobile.[2] The mandible is arch-shaped and consists of a condyle at each posterior superior portion. Each mandibular condyle has a medial and lateral pole, and the lateral pole is readily palpable[2] (Fig. 6–5). The posterior aspect of the condyle can be palpated if a fingertip is placed into the external auditory meatus and the pad of the finger is pushed anteriorly[7] (Fig. 6–6). The shape of each condyle may vary among individuals and side to side in the same individual.[7,12] The condyles are positioned anterior to the external auditory meatus and sit within the respective mandibular fossa of each of the temporal bones. The **coronoid process** is located anterior to the mandibular condyle and serves as an attachment for the temporalis muscle[2] (Fig. 6–7). The mandible interacts with the maxillae by way of the teeth. The importance of the articulations between the mandible and the temporal bones and maxillae will become apparent when we discuss impairments of the TM joint.

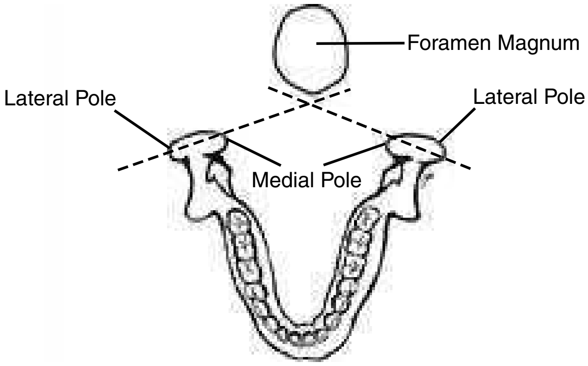


Figure 6–5 A superior view of the mandible (removed from the skull) shows the medial and lateral poles of the mandibular condyles. Mandibular rotation occurs around axes that pass through the medial and lateral poles of the right and left condyles, with the lines intersecting anterior to the foramen magnum of the skull.

Figure 6–6 Palpation of the posterior mandibular condyle through the external auditory meatus.

Accessory Joint Structures

The incongruence of the TM joint is addressed by a unique **articular disc.** A disc located within each TM joint separates the articulation into distinct superior and inferior joints with slightly different functions (see Fig. 6–4).[2,6] Thus, mandibular motion involves the simultaneous movement of four divergent joints. The **inferior TM joint** is formed by the mandibular condyle and the inferior surface of the disc and functions as a simple hinge joint. The **superior TM joint** is larger than the inferior joint and is formed by the articular eminence of the temporal bone and the superior surface of the disc; it functions as a gliding joint.[2] The articular disc of the TM joint is biconcave; that is, the superior and inferior surfaces are both concave. Styles and Whyte[13] describe the disc as having a "bowtie" appearance on magnetic resonance imaging (MRI) film, with the "knot" sitting at the thinnest portion. The thickness of the articular disc varies from 2 mm anteriorly to 1 mm in the middle to 3 mm posteriorly[14] (Fig. 6–8). The purpose of the disc is to allow the convex surfaces of the articular eminence and the mandibular condyle to remain congruent throughout the range of motion of the TM joint in all planes.[14] Sicher[16] described the TM joint as a hinge joint with movable sockets, and later investigators supported this description.[6,17] The articular disc, therefore, serves multiple purposes. It increases stability, minimizes loss of mobility, reduces friction, and decreases biomechanical stress on the TM joint.[7,18,19]

The disc within each TM joint has a complex set of attachments. The disc is firmly attached to the medial and lateral poles of the condyle of the mandible, but it is not attached to the TM joint capsule medially or laterally.[20] These attachments allow the condyle to rotate freely on the disc in an anteroposterior direction. The disc is attached to the joint capsule anteriorly, as well as to the tendon of the **lateral pterygoid muscle** (see Fig. 6–8).

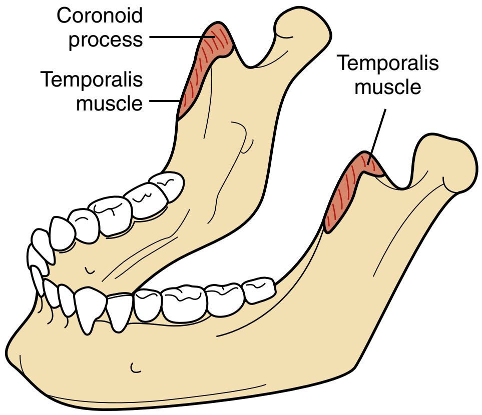


Figure 6–7 The temporalis muscle attaches to the coronoid process of the mandible.

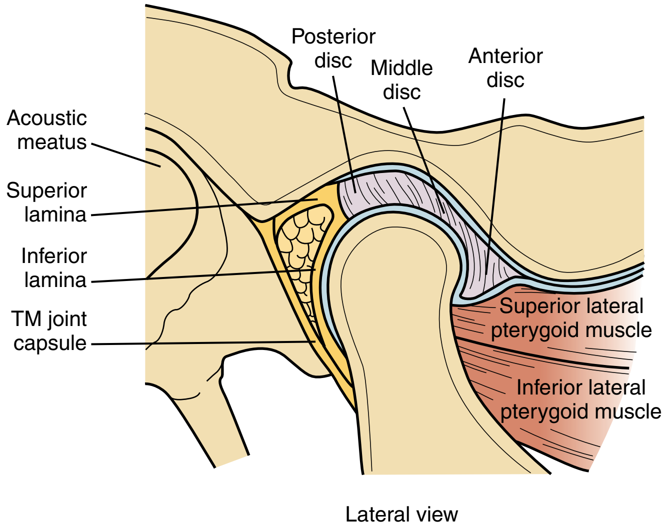


Figure 6–8 A cross-section of the temporomandibular joint showing the articular disc. The thickness of the disc varies from 2 mm anteriorly to 1 mm in the center to 3 mm posteriorly.

The anterior attachments restrict posterior translation of the disc. Posteriorly, the disc is attached to a complex structure, collectively called the **bilaminar retrodiscal pad.** The two bands (or laminae) of the bilaminar retrodiscal pad are both attached to the disc (see Fig. 6–8). The superior lamina is attached posteriorly to the tympanic plate (at the posterior mandibular fossa).[14,21] The superior lamina consists of elastic fibers that allow the superior band to stretch. The superior lamina allows the disc to translate anteriorly along the articular eminence during mandibular depression; its elastic properties assist in repositioning the disc posteriorly during mandibular closing. The inferior lamina is attached to the neck of the condyle and is inelastic. The inferior lamina serves as a tether on the disc, limiting forward translation, but does not assist with repositioning the disc during mandibular closing.[10,13,14] Neither of the laminae of the retrodiscal pad is under tension when the TM joint is at rest. Loose areolar connective tissue rich in arterial and neural supply is located between the two laminae.[10,14,19]

A healthy TM disc is viscoelastic and well suited for distribution of force, showing only minor changes in connective tissue fiber waviness even under significant stress.[22,23] The disc consists primarily of collagen, glycosaminoglycans (GAGs), and elastin. Collagen is largely responsible for maintaining the disc's shape. Elastin contributes to the disc's ability to regain its form during unloading. GAG composition maintains disc resiliency and resists mechanical compressive force. The biomechanical behavior of the disc may change according to changes in its composition.[23] Such changes in composition may occur as a result of aging, mechanical stress, or both.[8] Unlike the fibrocartilage on the mandibular condyles and articular eminences, the articular disc of the TM joint does not have the ability to repair and remodel itself.[21,23]

The anterior and posterior segments of the disc are vascular and innervated. The central segment of the disc is avascular and aneural.[2,14,15,22] The lack of vascularity and innervation is consistent with the fact that the middle portion of the disc is the force-accepting segment. The capsule, lateral TM ligament, and retrodiscal tissues contain mechanoreceptors and nociceptors, which are responsible for proprioception and pain sensation.[2]

Capsule and Ligaments

The elasticity of the joint capsule and ligaments determines the available motion at the TM joint in all planes. Motion can be enhanced or restricted depending on the flexibility of these structures. The portion of the capsule superior to the disc is quite lax, whereas the portion of the capsule inferior to the disc is taut.[8,24] Consequently, the disc is more firmly attached to the condyle below and freer to move on the articular eminence above. The capsule (Fig. 6–9) is thin and loose in its anterior, medial, and posterior aspects, but the lateral aspect is stronger and reinforced with long fibers (temporal bone to condyle).[20,24] The lack of strength of the capsule anteriorly and the incongruence of the bony articular surfaces predisposes the joint to anterior dislocation of the mandibular condyle.[16] The capsule is highly vascularized and innervated, which allows it to provide a great deal of information about position and movement of the TM joint.

The primary ligaments of the TM joint are the **TM ligament,** the **stylomandibular ligament,** and the **sphenomandibular ligament** (see Fig. 6–9). The TM ligament is a strong ligament composed of two parts, an outer oblique element and an inner horizontal element. The outer oblique element attaches to the neck of the condyle and the articular eminence (see Fig. 6–9). It serves as a suspensory ligament and limits downward and posterior motion of the mandible, as well as limiting rotation of the condyle during mandibular depression.[5,14,15,20] The inner horizontal component of the ligament is attached to the lateral pole of the condyle and posterior portion of the

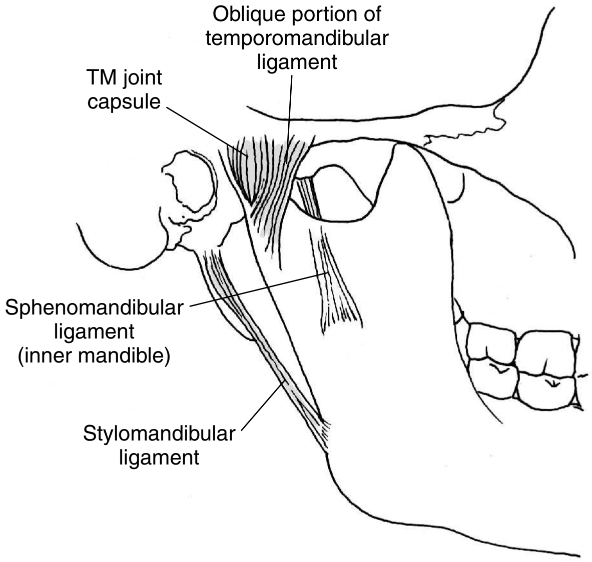


Figure 6–9 A lateral view of the TM joint capsule and ligaments.

disc and to the articular eminence. Its fibers are aligned horizontally to resist posterior motion of the condyle. Limiting the posterior translation of the condyle protects the retrodiscal pad.[14] The primary function of the TM ligament is to stabilize the lateral portion of the capsule.[5] Neither band of the TM ligament limits forward translation of the condyle or disc, but they do limit lateral displacement.[21]

The stylomandibular ligament is the weakest of the three ligaments and is considered a thickened part of the parotid sheath joining the styloid process to the angle of the mandible.[5] Some investigators have identified the function of this ligament as limiting the protrusion of the mandible,[14,15,17] but others have stated that it has no known function.[16,25,26]

The sphenomandibular ligament is described as the "strong" ligament that is the "swinging hinge" from which the mandible is suspended.[5,18] Some investigators have stated that it serves to protect the mandible from excessive anterior translation.[10,15,17] Others have stated that this ligament has no function.[24–26] The sphenomandibular ligament attaches to the spine of the sphenoid bone and to the middle surface of the ramus of the mandible. Abe and colleagues stated that the sphenomandibular ligament also has continuity with the disc medially.[27] Loughner and colleagues examined the structures surrounding the TM joint in 14 cadaver heads and found that the sphenomandibular ligament is not continuous with the medial capsule; rather, it is immediately adjacent to the capsule.[25] These investigators concluded that since the sphenomandibular ligament does not attach to the medial joint capsule, this ligament has no functional significance for the biomechanics of the TM joint. However, they suggested that the sphenomandibular ligament serves as an accessory ligament and, in concert with the TM ligament, provides structural support for the TM joint.[25]

JOINT FUNCTION

Joint Kinematics

The TM joint is one of the most frequently used and mobile joints in the body. It is engaged during mastication, swallowing, and speaking.[2,10] Most of the time, the TM joint movements occur without resistance from chewing or contact between the upper and lower teeth.[10] However, as a third-class lever, the TM joint is designed to maintain its structure in spite of significant forces acting on it.[28] As previously noted, the articular surfaces are covered with a pseudofibrocartilage that has the ability to remodel and repair and thus is able to tolerate repeated, high-level stress. Mastication requires tremendous power, while speaking requires intricate fine motor control. The musculature is designed to accomplish both these tasks.[10] Both **osteokinematic** and **arthrokinematic** movements are required for normal function of the TM joint. Osteokinematic motions include mandibular depression, elevation, protrusion, retrusion, and left and right lateral excursions. Arthrokinematic movements involve rolling, anterior glide, distraction, and lateral glide.[2] This section describes the function of the various components of the TM joint during osteokinematic and arthrokinematic movements. For the purposes of this chapter, only movements that occur without resistance will be discussed.

Mandibular Depression and Elevation

Mandibular depression and elevation are fundamental components of mastication.[2] Under normal circumstances, the motions of mandibular depression and elevation are relatively symmetrical, with each TM joint following a similar pattern. To accomplish mandibular depression and elevation, the mandibular condyle must roll and glide. The literature is contradictory as to whether the rolling and gliding occur sequentially or concurrently.[7,17,26,29–31] However, the literature is consistent in what rolling and gliding occur. During rotation, the mandibular condyle spins relative to the inferior surface of the disc in the lower joint (Fig. 6–10A). During translation, the mandibular condyle and disc glide together as a condyle-disc complex along the articular eminence. Translation occurs in the upper joint between the disc and the articular eminence[2,7,18] (Fig. 6–10B and Fig. 6–11).

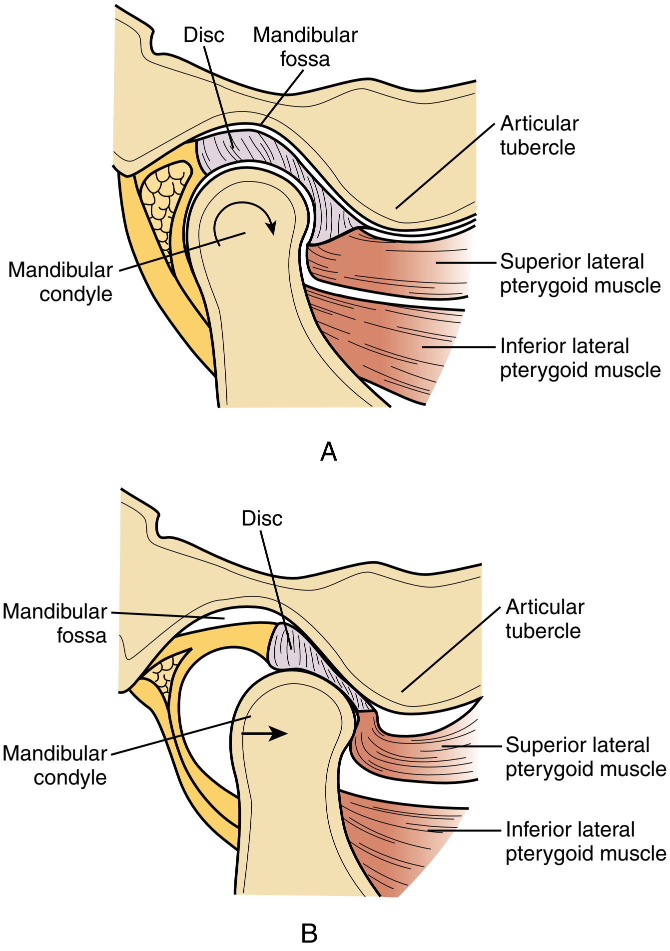


Figure 6–10 **A.** When the mouth begins to open, the motion at the TM joint may be limited to anterior rotation of the condyle on the disc. **B.** Anterior translation of the condyle and disc together on the articular eminence may occur in the later stages of mouth opening.

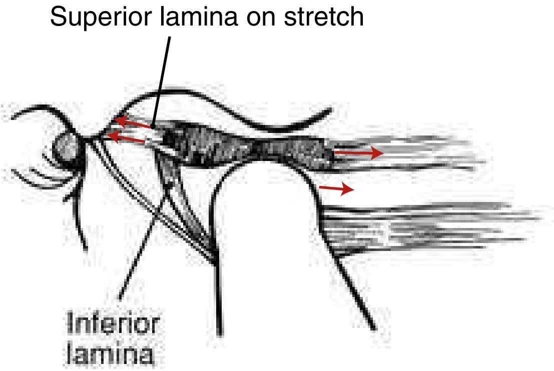


Figure 6–11 With full mouth opening, the disc and the condyle together translate anteriorly. The inferior lamina limits translation, and the elastic properties of the superior lamina both control anterior translation and assist with posterior translation during mouth closing.

Normal mandibular depression range of motion is 40 to 50 mm when measured between the incisal edges of the upper and lower front teeth.[2,7,18] Mastication requires approximately 18 mm of mandibular depression. Rolling occurs predominantly during the initial phase of mandibular depression with as little as 11 mm[24,29] or as much as 25 mm,[14] resulting from rotation of the condyle on the disc. The remaining motion results primarily from anterior translation of the condyle-disc complex along the articular eminence. The shape of the condylar head and the steepness of the articular eminence positively correlate with the amount of rotation.[29] Both the shape of the condylar head and the steepness of the articular eminence can be asymmetrical from one TM joint to the other, thus affecting the symmetry of motion.[7,12] As a quick screen, the clinician may use the adult knuckles (proximal interphalangeal joints) to assess the degree of mandibular depression. Two knuckles placed between the upper and lower incisors is considered functional, while three knuckles is considered normal[2,7] (Fig. 6–12). Gravity assists with mandibular depression. The mandibular elevators are thought to provide eccentric control of mandibular depression, although their contribution is unclear.[20]

Figure 6–12 Mandibular depression (mouth opening) is considered within normal limits if the proximal interphalangeal joints of two fingers can be inserted between the teeth.

Mandibular elevation is the reverse of mandibular depression. The mandibular condyle rotates posteriorly on the disc in the lower joint, and the condyle-disc complex translates posteriorly in the upper joint.

Control of the Disc During Mandibular Elevation and Depression

Active and passive control is exerted on the articular disc during mandibular depression and elevation. Passive control occurs through the capsuloligamentous attachments of the disc to the condyle. The lateral pterygoid muscle attaches to the anterior portion of the disc, producing active control, although evidence suggests that this attachment may not be consistently present[27,32] (Figs. 6–13 and 6–14). Bell proposed two other muscle segments that may assist with maintaining disc position during active movement.[10] These two muscle segments derive from the masseter muscle and attach to the anterolateral portion of the disc. They counteract the medial pull of the anteromedially directed lateral pterygoid.[10]

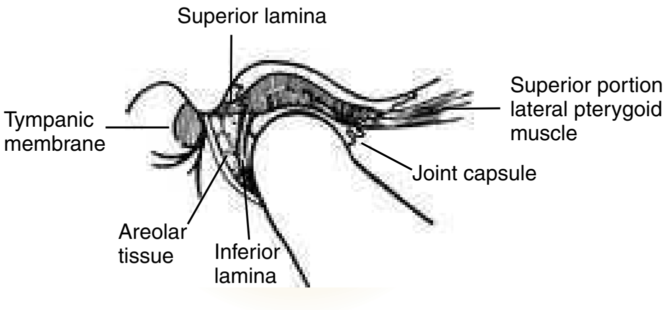


Figure 6–13 The TM disc attaches posteriorly to the joint capsule and to the superior and inferior laminae (segments of the bilaminar retrodiscal pad). The disc attaches anteriorly to the joint capsule and to the lateral pterygoid muscle.

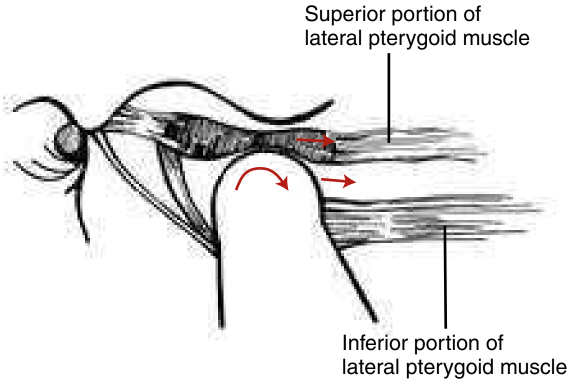


Figure 6–14 Another conceptual framework holds that condylar rotation on the disc and anterior translation of the disc and condyle on the articular eminence occur concomitantly during mouth opening.

During mandibular depression, the medial and lateral attachments of the disc to the condyle limit the motion between the disc and condyle to rotation. As the condyle translates, the biconcave shape of the disc causes it to track with the condyle without any additional active or passive assistance. However, the inferior retrodiscal lamina limits forward excursion of the disc. The superior portion of the lateral pterygoid muscle attaches to the disc and appears to be positioned to assist with anterior translation; however, no activity is noted during mandibular depression.[7,10]

During mandibular elevation, the elastic character of the superior retrodiscal lamina applies a posterior distractive force on the disc. In addition, the superior portion of the lateral pterygoid demonstrates activity that is assumed to eccentrically control the posterior movement of the disc, while maintaining the disc in an anterior position until the mandibular condyle completes posterior rotation to the normal resting position.[2,23,33–35] Abe and colleagues suggested that the sphenomandibular ligament also assists this action.[27] Again, the medial and lateral attachments of the disc to the condyle limit the motion to rotation of the disc around the condyle.[20]

Mandibular Protrusion and Retrusion

Mandibular protrusion and retrusion occur in the upper TM joint. The condyle-disc complex translates in an anterior inferior direction, following the downward slope of the articular eminence, during protrusion and returns along a posterior superior path.[2] Rotation is not present during protrusion and retrusion. The teeth are separated during these motions. Ideally, the lower teeth should surpass the upper teeth several millimeters (Fig. 6–15); however, protrusion is considered adequate when the upper and lower front incisal edges touch.[7] Protrusion is an important component necessary for maximal mandibular depression. Retrusion is an important component of mandibular elevation from a maximally depressed mandible.[2]

Control of the Disc During Mandibular Protrusion and Retrusion

During protrusion, the posterior attachments of the disc (the bilaminar retrodiscal tissue) stretch 6 to 9 mm to allow completion of the motion.[14] The degree of retrusion is limited by tension in the TM ligament as well as by compression of the soft tissue in the retrodiscal area between the condyle and the posterior glenoid spine. An estimated 3 mm of translation occurs during retrusion; however, this motion is rarely measured.[14]

Mandibular Lateral Excursion

Lateral excursion involves moving the mandible to the left and to the right. The degree of lateral excursion considered normal for an adult is 8 to 11 mm.[2,7] One functional screen to estimate whether this motion is normal is to observe whether the mandible can move the full width of one of the **central incisors** in each direction[7] (Fig. 6–16). Active lateral excursion is described as *contralateral* (the opposite side) or *ipsilateral* (the same side) relative to the primary muscle action.[2] To accomplish lateral excursion, the ipsilateral mandibular condyle spins around a vertical axis within the mandibular fossa, while the contralateral mandibular condyle translates anteriorly along the articular eminence. A slight degree of spin and lateral glide of the contralateral mandibular condyle is necessary to achieve maximal lateral excursion[7,8,24] (Fig. 6–17). Another normal asymmetrical

Figure 6–15 With maximum mandibular protrusion, the lower teeth should be in front of the upper teeth.

Figure 6–16 With normal lateral deviation of the mandible to the right, the midline of the lower teeth should move the full width of the right upper central incisor.

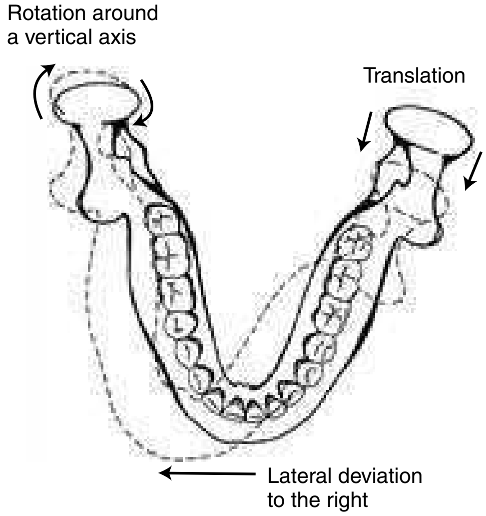


Figure 6–17 In this superior view of the mandible, lateral deviation of the mandible (chin) to the right occurs effectively as a rotation (spin) of the right condyle around a vertical axis, and the left condyle translates anteriorly.

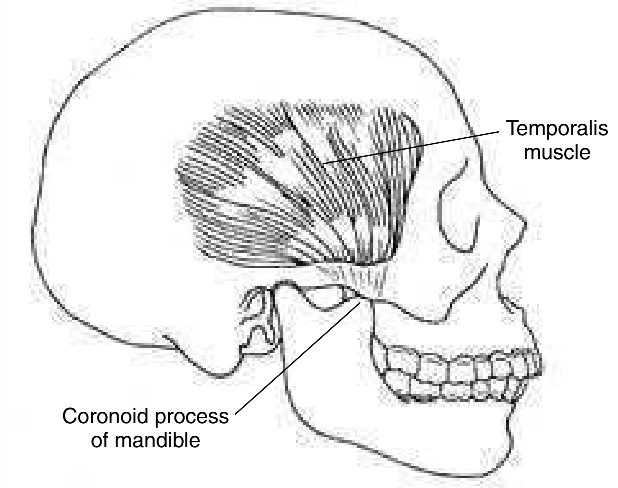


Figure 6–18 The temporalis muscle, with its attachment to the medial aspect of the coronoid process.

movement of the TM joint involves rotating one condyle around an anteroposterior axis while the other condyle depresses.[14] This movement results in a frontal plane motion of the mandible, with the chin moving downward and deviating from the midline toward the condyle that is spinning. These motions are generally combined into one complex motion used for chewing and grinding food.[14,36]

Deviations and **deflections** may be noted during osteokinematic movements of the mandible. A deviation is a motion that produces an "S" curve as the mandible moves away from the midline during mandibular depression or protrusion and returns to midline by the end of the movement. A deflection is a motion that creates a "C" curve, with the mandible moving away from midline during mandibular depression or protrusion but not returning to midline by the end of the movement. Deviations and deflections may result from mandibular condyle head shapes differing from right to left. If no other signs or symptoms accompany these asymmetries, then deviation or deflection is considered inconsequential. However, as will be discussed later in this chapter, deviations and deflections may indicate a pathology.[7]

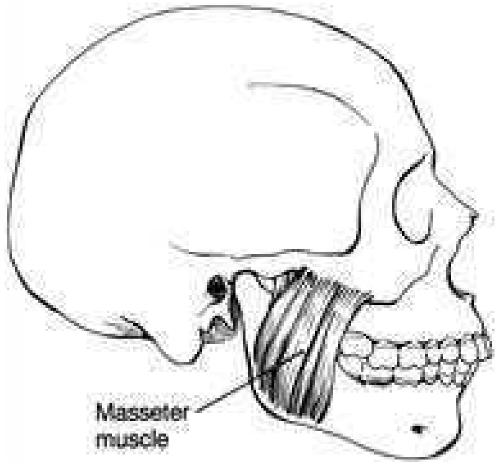


Figure 6–19 The masseter muscle.

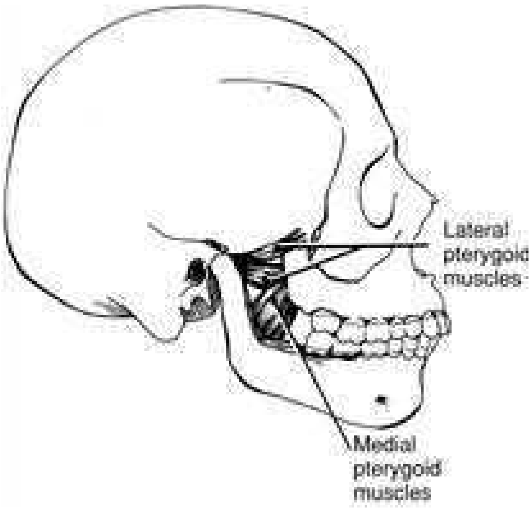


Figure 6–20 The medial and lateral pterygoid muscles.

Muscles

Primary Muscles

The muscles acting on the TM joint are divided into primary and secondary muscle groups. The primary muscles include the **temporalis** (Fig. 6–18), **masseter** (Fig. 6–19), lateral pterygoid, and **medial pterygoid**[5] (Fig. 6–20).

The temporalis is a flat, fan-shaped muscle that is wide at the proximal portion and narrow at the inferior portion. The superior fibers attach to the cranium while the inferior fibers attach to the coronoid process and the anterior edge and medial surface of the ramus of the mandible. The temporalis fills the concavity of the temporal fossa and can be palpated easily over the temporal bone.[2,5] The masseter is a thick, powerful muscle with its superior attachments on the zygomatic arch and zygomatic bone and its inferior attachment on the external surface of the ramus of the mandible. This muscle can be palpated easily superior to the angle of the mandible.[5] Van der Bilt and colleagues discovered that the bilateral masseters and anterior temporalis muscles were 30% more active during bilateral maximum voluntary bite force than during unilateral bite force.[11] Additionally, these investigators discovered that the muscle activity of the masseters was symmetrical with unilateral clenching. However, the muscle activity during unilateral clenching was significantly greater for the ipsilateral anterior temporalis muscles than for the contralateral anterior temporalis muscles.[11]

The lateral pterygoid consists of superior and inferior segments that travel in a horizontal direction and combine posteriorly to attach to the neck of the mandible, the articular disc, and the joint capsule.[2,5] The literature is contradictory as to whether this muscle can be palpated. The lateral pterygoid muscles are thought to assist with mandibular depression.[3,20,24,34] Bell, however, stated that only the inferior fibers are active; the superior fibers are silent.[10]

The medial pterygoid parallels the masseter in line-of-force and size. The superior fibers attach to the medial surface of the lateral pterygoid plate on the sphenoid bone, and the inferior attachment is on the internal surface of the ramus near the angle of the mandible.[2,5]

Secondary Muscles

The secondary muscles are smaller than the primary muscles and consist of the **suprahyoid** and **infrahyoid** groups (Fig. 6–21). The **digastric, geniohyoid, mylohyoid,** and **stylohyoid** comprise the suprahyoid group. The infrahyoid group includes the **omohyoid, sternohyoid, sternothyroid,** and **thyrohyoid** muscles.[2] The suprahyoid muscles assist with mandibular depression, while the infrahyoid muscles are responsible for stabilizing the hyoid. Both the suprahyoid and infrahyoid muscle groups are involved in speech, tongue movements, and swallowing.[2] The suprahyoid muscles attach between the base of the cranium, the hyoid, and the mandible. The superior fibers of the infrahyoid muscles attaches to the hyoid, while the inferior fibers attach to the thyroid cartilage, sternum, and scapula.

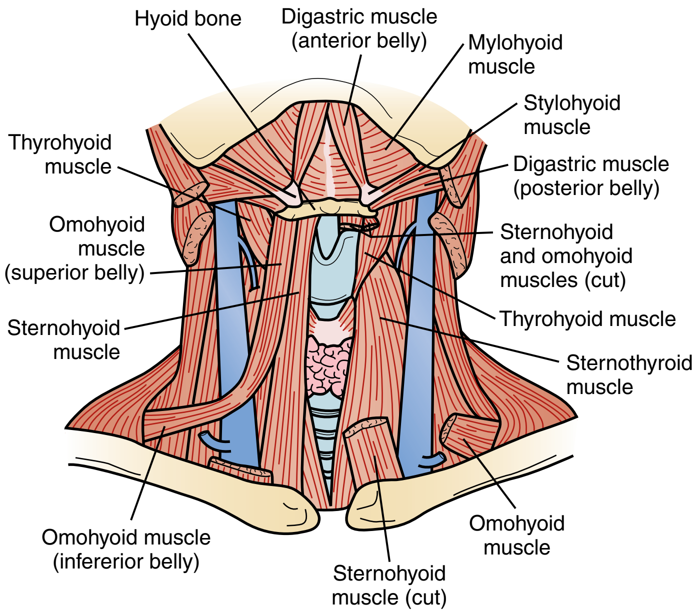


Figure 6–21 The suprahyoid and infrahyoid muscle groups shown from an anterior view of the neck.

The posterior fibers of the digastric muscle attach to the mastoid notch, while the anterior fibers attach to the inferior mandible. The tendon joining the anterior and posterior segments is connected to the hyoid bone in the neck by a fibrous loop[2] (Fig. 6–22). The digastric muscle is predominantly responsible for mandibular depression.[10] The hyoid bone has to be stabilized for the digastric muscle to depress the mandible. This stabilization is provided by the infrahyoid muscles.[2]

Coordinated Muscle Actions

Mandibular depression occurs from the concentric action of the bilateral digastric muscles in conjunction with the inferior portion of the lateral pterygoid muscles. Mandibular elevation results from the collective concentric action of the bilateral masseter, temporalis, and medial pterygoid muscles. The bilateral superior lateral pterygoid muscles eccentrically control the TM discs as the mandibular condyles relocate into the mandibular fossa with mandibular elevation.

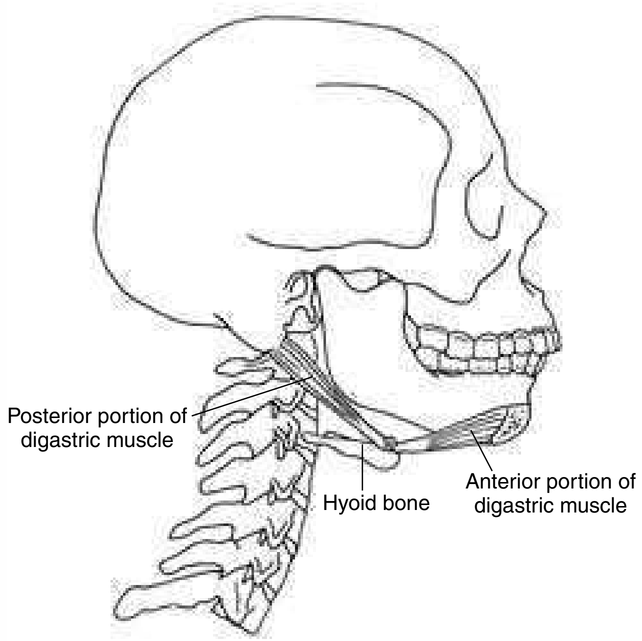


Figure 6–22 The posterior portion of the digastric muscle arises from the mastoid notch, and the anterior portion arises from the inferior mandible. The tendon that joins the anterior and posterior portions is connected by a fibrous loop to the hyoid bone in the neck.

The other mandibular motions of protrusion, retrusion, and lateral deviation are produced by the same muscles that elevate and depress the mandible, but in different sequences. Mandibular protrusion is produced by the bilateral action of the masseter, medial pterygoid,[7,26] and lateral pterygoid muscles.[14,37] Retrusion is generated through the bilateral action of the posterior fibers of the temporalis muscles, with assistance from the anterior portion of the digastric muscle.[24] Lateral deviation of the mandible is produced by the unilateral action of a selected set of these muscles. The medial and lateral pterygoid muscles each deviate the mandible to the opposite side.[14,24] The temporalis muscle can deviate the mandible to the same side. Although the temporalis and lateral pterygoid muscles on the left appear to create opposite motions of the mandible, concomitant contractions of the right lateral pterygoid and right temporalis muscles function as a force couple. The lateral pterygoid muscle is attached to the medial pole of the condyle and pulls the condyle forward. The temporalis muscle on the ipsilateral side is attached to the coronoid process and pulls it posteriorly. Together these muscles effectively spin the condyle to create deviation of the mandible to the left. Because the temporalis muscle is also an elevator of the mandible, this combination of muscular activity is particularly useful in chewing.

CASE APPLICATION

Palpation and Asymmetry of Motion

case 6–1

Jill Smith has a decreased active range of motion with mandibular depression, protrusion, and left lateral excursion. Passive mobility of the right TM joint is limited, with distraction, anterior translation, and lateral glide. Her mandible deflects to the right with mandibular depression. Palpation of the mandibular condyles reveals a larger space on the left than the right during active mandibular depression and protrusion. This information suggests that the left mandibular condyle is moving more than the right. These findings indicate that the right mandibular condyle and disc are not translating as far anteriorly as the left mandibular condyle. The conclusion may be either hypomobility on the right or hypermobility on the left.

Nerves

The primary muscles of mastication are innervated by the **mandibular nerve,** which is a branch of the **trigeminal nerve (cranial nerve V).** Sensation in the TM area occurs through two branches of the mandibular nerve, the **auriculotemporal** and the **masseteric.**[2,5] The deep temporal branches of the mandibular nerve supply the temporalis muscles; the masseteric nerve supplies the masseter; the lateral pterygoid nerve supplies the lateral pterygoids; and the medial pterygoid nerve supplies the medial pterygoids.[5] The digastric muscle is innervated by the **facial nerve (cranial nerve VII)** and the **inferior alveolar nerve,** which is a branch of the mandibular nerve. The geniohyoid is innervated by the first branch of the cervical plexus via the **hypoglossal nerve (cranial nerve V).** [2,38] The mylohyoid is innervated by the inferior alveolar nerve; the stylohyoid, by the facial nerve.[2] Three of the four infrahyoid muscles are innervated by the ventral rami of the first three branches of the cervical plexus. The fourth is innervated by the ventral rami of the first branch of the cervical plexus via **cranial nerve XII.**[2,38] The neurological evidence supports the possibility of inaccurate diagnoses of trigeminal neuralgia or an ear problem when in fact the person may have a TM joint condition. Loughner and colleagues[25] suggested that pain felt in the ear may be referred from the TM joint as a result of injury or inflammation.

Relationship to the Cervical Spine and Posture

The cervical spine and TM joint are intimately connected. A biomechanical relationship exists between the position of the head, the cervical spine, and the dentofacial structures.[12,39] The attachments of the primary and secondary muscles provide strong evidence of the relationship among the TM joint, cervical spine, throat, clavicle, and scapula. The impact of posture on the TM joint becomes apparent once the attachments of the musculature are examined. Given their attachments, muscles acting on the mandible may also impact the atlanto-occipital joint and cervical spine. Head and neck position may affect tension in the cervical muscles, which may in turn influence the position or function of the mandible.[4,39–43] Correct posture minimizes the forces produced by the cervical spine extensors as well as the other cervical muscles necessary to support the weight of the head. Over time, improper posture can lead to adaptive shortening or lengthening of the muscles around the head, cervical spine, and upper quarter (including all structures of the upper trunk from the scapulae superiorly). As a result of improper posture, there may be an alteration in range of motion, the ability to produce muscular forces, and joint morphology. Many of the symptoms associated with TM joint dysfunction are similar to the symptoms associated with primary cervical spine impairment. Therefore, the cervical spine and upper quarter should be thoroughly examined when clients present with TM joint complaints.[4,39–41,43]

Continuing Exploration 6-1:

TM/Cervical Joint Interrelationships

Pain often occurs concurrently in the head, neck, and jaw as the result of faulty posture. According to Kendall, a forward head posture involves mechanical changes that stimulate the nociceptors that produce pain. The forward head posture entails extension of the occiput and the cervical spine, leading to compensatory flexion of the cervicothoracic junction and upper thoracic spine to achieve a level head position.[44,45] With the head held in a forward head position, the cervical extensors are in a shortened

Continued

position and develop adaptive shortening over time. Concomitantly, the anterior cervical muscles are in an elongated position and develop stretch weakness.[44] Additionally, the occiput is extended on the atlas (C1), creating adaptive shortening of the suboccipital tissues. The suboccipital tissues include the anterior atlantoaxial and atlanto-occipital ligaments, the posterior belly of the digastric muscles, the stylohyoid muscles, the upper fibers of the upper trapezius, and the semispinalis capitis and splenius capitis muscles.[38] The forces necessary to maintain the head against gravity with faulty cervical posture and forward head result in muscle imbalances and altered movement patterns. Such alterations typically lessen the capacity of particular structures to meet the thresholds for adaptive responses to physical stresses. Increased tension from shortening of the suboccipital tissues may lead to headaches that originate in the suboccipital area, limitation in active range of motion of the upper cervical spine, and TM joint dysfunction. Furthermore, pain in the TM region may be referred from the cervical region.[45,46] Thus, cervical posture should be normalized to successfully treat dysfunction in the TM joint complex.[40,41,46] Andrade and colleagues[47] supported the relationship between the cervical spine muscles and TM dyfunction. These investigators found that individuals with TM dysfunction reported higher perceptions of pain with palpation of the cervical muscles.[47]

CASE APPLICATION

Posture and TM Joint Relationship

case 6–2

Jill Smith complains of frequent headaches and intermittent pain in her right ear, which may be the result of her faulty posture. The physical examination reveals a forward head posture (Fig. 6–23) with rounded shoulders, winging scapulae, increased thoracic kyphosis, increased lumbar lordosis, hyperextended knees, and overpronated feet. The noted elevation of her right shoulder may imply tightness of the suboccipitals and cervical musculature, consistent with her limited active range of motion when performing mandibular depression, protrusion, and left lateral excursion. Jill reports tenderness with palpation of the muscles at the base of her head, the right side of her face, and under her chin. Tenderness and muscle guarding may be related to the stresses placed on the tissues from improper positioning.[45]

Continuing Exploration 6-2:

TM/Respiratory/Cervical Dysfunction

Children with chronic breathing allergies may develop TM joint dysfunction as a result of dysfunctional growth and developmental patterns.[43,48] For example, a child with allergies who has difficulty breathing through the nose will often hyperextend the cervical spine, assuming the forward head position, to increase the diameter of the upper respiratory tract and improve ventilation.[43] This posture changes the dynamics of the TM joint. The upper and lower teeth contact each other and may affect the resting position of the TM joint. Thus, the muscles surrounding the TM joint complex expend greater energy to maintain this posture. Blocked nasal passages impede inspiration and promote the use of accessory muscles of respiration (scalene and sternocleidomastoid muscles) to assist with breathing. Use of accessory muscles may lead to a forward head posture.[48] Over time, a forward head posture contributes to a cycle of increasing musculoskeletal dysfunction, including repeated episodes of TM inflammation that can result in fibrosis of the TM joint capsule.[36]

Dentition

Occlusion, or contact of the teeth, is intimately involved in the function of the TM joint. Chewing is one of the functions of the TM joint. Although the teeth are together for approximately 15 minutes of each day, the presence and position of the teeth are critical to normal TM joint function. Contact of the upper and lower teeth limits motion of the TM joint during empty-mouth movements. The complexities of the TM joint and the interrelated issues with the teeth underscore the necessity of the comprehensive management of TM joint disorders. Normal adult dentition includes 32 teeth divided into four quadrants. The only teeth we will refer to by name are the upper and lower central incisors. These are the two central teeth of the maxilla and the two central teeth of the mandible.[49] When the central incisors are in firm approximation, the position is called **maximal intercuspation**[50] or the **occlusal position.**[24] This position is not the normal resting position of the mandible. Rather, 1.5 to 5 mm of **"freeway space"** between the upper and lower incisors is normally maintained.[10,14] By maintaining this freeway space, the intra-articular pressure within the TM joint is decreased, the stress on the articular structures is reduced, and the tissues of the area are able to rest and repair.[14]

The American Academy of Orofacial Pain and others have identified occlusion as a causative factor in TM disorders.[1] However, the literature has been contradictory and inconclusive. Lack of uniformity in research methods and techniques have been suggested as the source of these discrepancies. Ciancaglini and colleagues studied patients with unilateral TM disorders to determine whether an association exists between unilateral TM symptoms and occlusion.[41] They found a weak association between unilateral TM disorders and asymmetry of occlusal contacts in young adults with normal occlusion. Asymmetry of occlusal contacts was noted to be the rule rather than the exception in both patients and controls.[41]

Sonnesen and colleagues studied a sample of 96 children with severe malocclusion prior to orthodontic intervention.[42] These investigators identified a direct correlation between the occurrence of muscle tenderness in the masticatory muscles and a long-face craniofacial morphology. They provided two possible explanations: (1) the muscle tenderness could be

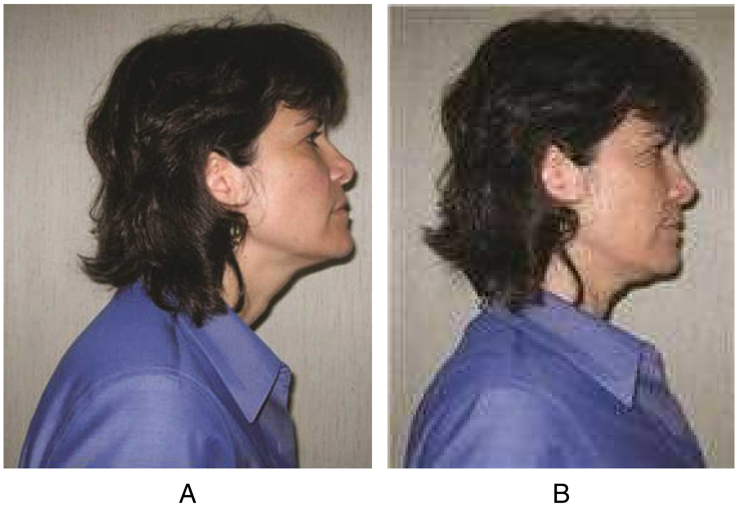


Figure 6–23 **A.** Poor cervical posture increases the physical demands on the suboccipital structures, contributing to TM joint dysfunction. **B.** Corrected cervical posture restores the muscles of the cervical spine and TM joint to a more balanced length-tension relationship.

the result of functional overloading of weak mandibular elevator muscles, or (2) the muscle tenderness may lead to temporary hypofunction of the masticatory muscles, resulting in decreased bite force. Additionally, the investigators confirmed an association in the studied sample between posture and TM dysfunction. Clicking (joint noise), locking of the mandible, and asymmetric mandibular depression were noted in children with a marked forward inclination of the cervical column and an increased craniocervical angle.[42,43]

COMMON IMPAIRMENTS AND PATHOLOGIES

TM dysfunction is a vague term that encompasses numerous clinical problems that involve the masticatory system. Mechanical stress is the most critical factor in the multifactorial etiology.[3,22,42,51] Dysfunction of either the muscles or the joint structure generally is at fault. Most clients with TM dysfunction will not fit into a specific category of dysfunction classification, which creates a clinical challenge.[20,52] Additionally, only 20% to 30% of individuals with internal derangement of the TM joint develop symptomatic joints.[20] These symptoms may progress or resolve spontaneously.[13] De Paiva Bertoli and colleagues identified the most common signs of TM dysfunction in children with headaches as (from most to least frequent): joint pain, muscular pain, joint noise, and mandibular deviation.[53] Pathomechanics of this joint may result from faulty posture, an isolated traumatic event such as a fall, cervical whiplash, or chronic inflammation of the TM joint resulting from indirect trauma. As with many disorders, the exact pathomechanics are often idiopathic. Although treatment of TM dysfunction is beyond the scope of this text, collaboration between dentists, physical therapists, and counselors provides the best functional outcomes.[43]

Determining whether a client has TM dysfunction requires an evidence-based diagnostic process. This process should include a thorough clinical assessment performed by a TM clinician, classification of patients according to the *Research Diagnostic Criteria for TM Disorders*, and MRI, the diagnostic gold standard.[52] Because of the complexity associated with TM joint dysfunction, clinicians should seek advanced education before attempting to manage this patient clientele. Common etiologies of TM joint dysfunction include aging, inflammatory conditions, capsular fibrosis, osseous mobility, articular disc displacement, and degenerative conditions.

Age-Related Changes in the TM Joint

The aging process affects the joints of the human body. The TM joint is no exception. However, degenerative changes are not always the result of the normal aging process. Degenerative changes may occur from a preexisting dysfunction. Furthermore, degenerative changes do not necessarily indicate disability. Rowe and Kahn[54] described successful aging as "multidimensional, encompassing the avoidance of disease and disability, the maintenance of high physical and cognitive function, and sustained engagement in social and productive activities." With progression through life, tissues become less supple, less elastic, and less able to withstand maximal forces, resulting in biomechanical changes in the musculoskeletal tissues. Pathology or biomechanical dysfunction will not necessarily result from these changes.[54]

Nannmark and colleagues examined cadaver mandibular condyles from 37 TM joints of people ages 55 to 99.[55] The investigators reported structural changes in 38% of the mandibular condyles examined. No signs of inflammatory cell infiltration were found, suggesting that the observed changes were secondary to biomechanical stresses, rather than the result of inflammation. Twenty-two (59%) of the articular discs had perforations or roughness or were thinned. However, only three (8%) of the discs were in an anterior position, and each of these was perforated. The investigators concluded that osteoarthritis may be expected in 14% to 40% of adults, with increased frequency with age in both men and women.[55]

DeLeeuw and colleagues performed radiography and MRI studies of 46 former patients 30 years after the diagnosis of osteoarthritis and internal derangement of the TM joint.[56] **Internal derangement** is described as the abnormal position and function of the disc and articulating surfaces.[20]

The patients studied by DeLeeuw and colleagues were between the ages of 50 and 70 years at the time of the follow-up study. The investigators incorporated into their study 22 age-matched controls without known TM joint dysfunction.[56] Signs noted on the radiographs were more common and more severe in the former patient group. MRI findings revealed a higher percentage of osteoarthritis and internal derangement in the dysfunctional TM joints as well as in the contralateral TM joints. However, degenerative changes in the contralateral TM joints appeared to have been asymptomatic, with only 25% reporting any symptoms and none of those seeking intervention. MRI evidence of the controls revealed infrequent osteoarthritis and internal derangement.[56] The work conducted by Nannmark and colleagues[55] and by deLeeuw and colleagues[56] indicates that TM joint degeneration does not necessarily occur with aging and that degenerative changes evident on radiograph or MRI are not necessarily associated with symptoms or dysfunction.

Inflammatory Conditions

Inflammatory conditions of the TM joint include **capsulitis** and **synovitis.** Capsulitis involves inflammation of the joint capsule, and synovitis is characterized by fluctuating edema caused by effusion within the synovial membrane of the TM joint. Rheumatoid arthritis is the most common cause of synovial membrane inflammation, but gout, psoriatic arthritis, ankylosing spondylitis, systemic lupus erythematosus, juvenile chronic arthritis, and calcium pyrophosphate dehydrate deposition may also contribute to inflammation of the synovia.[20] Individuals with inflammatory conditions experience pain and inflammation within the joint complex, which may diminish mandibular depression.[7] Unresolved inflammation can lead to adhesions that restrict the movement of the disc and limit the function of the TM joint.[13]

Rheumatoid arthritis is a chronic systemic condition with articular and extra-articular involvement. The primary symptoms of rheumatoid arthritis include pain, stiffness, edema, and warmth. This autoimmune disorder targets the joint capsule, ligamentous structures, and synovial lining of the joint complex, resulting in joint instability, joint deformity, or ankylosis.[55] Multiple bilateral joints are typically involved with this disease. A detailed discussion of rheumatoid arthritis is beyond the scope of this text; however, clinicians should be aware that rheumatoid arthritis often affects the TM joint.[55]

Capsular Fibrosis

Unresolved or chronic inflammation of the TM joint capsule stimulates overproduction of fibrous connective tissue, which creates **capsular fibrosis** of the TM joint complex.[7,55] The resultant fibrosis causes progressive damage and loss of tissue function[7,55] A thorough client history is the key to identifying this condition. The clinician should listen for reports of repeated episodes of capsulitis. Circumstances leading to chronic capsulitis with progression to capsular fibrosis may include prolonged periods of immobilization, direct or indirect trauma, or arthritis.[7] Physical examination will reveal limited or altered osteokinematic motions, suggestive of a decrease in translatory motion on the involved side. Active motion of the TM joint capsule will typically elicit pain.[7]

CASE APPLICATION

Trauma and TM Dysfunction

case 6–3

Jill Smith's musculoskeletal complaints and limited active range of motion of the mandible may be attributed to capsular fibrosis, as well as to her faulty posture. Most likely she has had repeated episodes of acute capsulitis of the right TM joint, with her initial episode following the bicycle accident when she was 12 years old. The trauma to her face from hitting her chin on the handlebars of her bicycle likely caused compression forces to both TM areas, resulting in an inflammatory process with edema and pain. Her faulty posture, which developed as a result of her allergies, and the stress of being a single mother trying to raise three young children while working may have exacerbated the inflammation without the opportunity for resolution, leading to a chronic and progressive capsular fibrosis.[12]

Osseous Mobility Conditions

Osseous mobility disorders of the TM joint complex include **joint hypermobility** and **dislocation.** Many similarities are noted in the client history and clinical findings for these two conditions. Hypermobility, or excessive motion, of the TM joint is a common phenomenon found in both symptomatic and nonsymptomatic populations. The hypermobility is a result of laxity of the joint capsules, tendons, and ligaments.[7,57] Joint hypermobility may be a generalized connective tissue disorder that involves all joints of the body, including the TM joints.[33,48,58–60] Individuals seen clinically for TM joint hypermobility typically report that the jaw "goes out of place," produces noises, or "catches" when the mouth is in the fully opened position. Physical examination of clients with TM joint hypermobility reveals an increased indentation posterior to the lateral pole. Joint noises occur at the end of mandibular depression and at the beginning of mandibular elevation. These noises may be heard by the person; however, the clinician often only can palpate the clicking or crepitus associated with the noises. Hypermobility of one TM joint results in the deflection of the mandible toward the contralateral side with mandibular depression. In addition, mandibular depression will exceed 40 mm.[7] Yang and colleagues examined MRI films of 98 patients diagnosed with TM joint hypermobility during jaw opening.[33] They found pathological changes (hypertrophy, atrophy, or contracture) in 77% of the lateral pterygoid muscles, with changes more common in the superior portion of the muscle. The investigators often found anterior disc displacement with reduction in the patients who reported more symptoms than were reported by persons with normal mobility or with disc displacement without reduction.[33]

Many aspects of the client history and physical examination of individuals with dislocation of the TM joint are similar to those of individuals with hypermobility. However, with TM joint dislocation, full mandibular depression results in deflection (lateral deviation) of the jaw to the contralateral side of the involved TM joint, and inability to close the mouth. The individual may experience pain with this condition. With dislocation, both the mandibular condyle and the disc have translated anteriorly beyond the articular crest of the tubercle of the temporal bone, thus "sticking" in the extreme end-range position.[7,13] Dislocation of the TM joint is usually temporary and resolves with joint mobilization.

Articular Disc Displacement

Articular disc displacement occurs when the articular disc subluxes beyond the articular eminence.[7,34] Two conditions can result: disc displacement with reduction and disc displacement without reduction.[7,33,34] Without intervention, disc displacement with reduction often advances to disc displacement without reduction.[7,13,33,34] Disc displacement is an internal derangement that may be identified by diagnostic imaging or during a physical examination.[13] MRI is the imaging modality of choice to identify disc displacement.[20]

Individuals exhibiting disc displacement with reduction experience "joint noise" at two intervals: during mandibular depression and during mandibular elevation. The joint noise is referred to as a **reciprocal click.**[7,13] The reciprocal click is the gold standard necessary to diagnose disc displacement with reduction. At rest the articular disc is anterior to the mandibular condyle; therefore, the mandibular condyle is in contact with the retrodiscal tissue rather than with the disc. During mandibular depression, the condyle translates anteriorly and captures the inferior surface of the disc to obtain a normal relationship with the disc. When the condyle slips under the disc, an audible click is often present. Once the condyle-disc relationship is restored, motion continues normally through full mandibular depression. As the mandible elevates to close the mouth, the condyle translate posteriorly and slips out from under the disc, producing a second audible click. The second click signifies that the condyle and disc have lost the normal condyle-disc relationship. When the click occurs early in opening and late in closing, the amount of anterior displacement of the disc is relatively limited. The later the click occurs in the opening phase, the more severe the disc dislocation.[7] Some evidence suggests that the timing of the clicks during opening and closing can determine treatment prognosis.[61]

Individuals exhibiting disc displacement with reduction may remain in this state or progress rapidly, within months, to an acute condition of disc displacement without reduction. The posterior attachments to the disc become overstretched and unable to relocate the disc during mandibular depression, which results in the loss of the reciprocal clicks.[7,13] On MRI, Yang and colleagues discovered a highly significant correlation between abnormalities of the lateral pterygoid muscles and TM joints with disc displacements both with and without reduction of the disc.[33] The abnormalities of the lateral pterygoid muscle included hypertrophy, contracture, and atrophy of the superior and inferior bellies of the lateral pterygoid muscles of the involved TM joint. Recall that the lateral pterygoid muscle attaches to the anterior portion of the disc and is normally active with mandibular elevation, presumably to eccentrically control the return to the resting position. Hypertrophy of this muscle indicates overactivity, which possibly leads to the excessive anterior translation of the articular disc.[33]

Whether acute or chronic, disc displacement without reduction indicates that the disc does not relocate onto the mandibular condyle. Thus, clients with acute disc displacement without reduction demonstrate limited mandibular motion as a result of the disc creating a mechanical obstruction to condylar motion rather than facilitating condylar translation. Individuals with disc displacement without reduction typically describe an inability to fully depress the mandible, as well as difficulty performing functional movements involving the jaw, such as chewing, talking, or yawning.

Degenerative Conditions

Two degenerative conditions may affect the TM joint: osteoarthritis and rheumatoid arthritis. Rheumatoid arthritis was discussed previously under inflammatory conditions. Hertling and Kessler stated that 80% to 90% of the population older than 60 years have some symptoms of osteoarthritis in the TM joint.[18] Yang and colleagues concurred, substantiating their findings with MRI evidence.[33] According to Mahan,[21] osteoarthritis usually occurs unilaterally (unlike rheumatoid arthritis, which usually presents bilaterally). The primary cause of osteoarthritis is repeated minor trauma to the joint, particularly trauma that creates an impact between the articular surfaces.[13,21] Styles and Whyte suggested that the radiographic features of degenerative changes in the TM joint, including joint space narrowing, erosions, osteophyte formation, sclerosis, and remodeling, are similar to those seen elsewhere in the body.[13] Loss of posterior teeth may also contribute to degenerative changes because simple occlusion of the remaining teeth alters the forces that occur between the TM joint forces.[7,21]

In a randomized control study of TM discs, Tanaka and colleagues found a cause-effect relationship between internal derangement of the TM joint and articular disc damage.[22] These investigators note that articular discs with severe internal derangement experienced more extensive degenerative changes than the articular discs from controls. The investigators described irregular patterns of collagen fibers in the articular discs of the patient group when compared to the control group. These structural changes were related to the diminished capacity of these tissues to withstand mechanical stresses.[22]

Concept Cornerstone **6-1**

Signs and Symptoms of TM Joint Dysfunction

Clinical manifestation of temporomandibular joint dysfunction can vary widely, depending on the extent of the condition and the presence of complicating factors. Signs and symptoms may include:

- Pain in the area of the jaw
- Edema around the mandibular condyle
- Increased or decreased active or passive range of motion
- Popping or clicking noises
- Difficulty with functional activities (e.g., eating, talking) or parafunctional activities (e.g., clenching, nail biting, pencil chewing) of the mandible
- Catching or locking of the jaw
- Forward head posture

CASE APPLICATION

Patient Summary

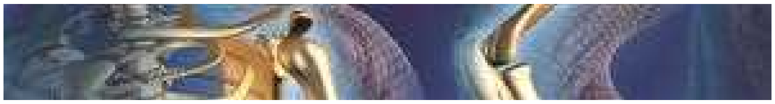
case 6–4

Jill Smith sought physical therapy intervention for frequent headaches and intermittent aching in the right jaw area. Her symptoms may be attributed to a number of potential sources in isolation—trauma to the TM joint, poor cervical posture, forward head position—or some combination of these sources. Factors such as stress from work and family life also may play a role in her clinical presentation. Although the research literature does not explicitly draw a direct link between these variables, it suggests that these biomechanical variables may play a role in a patient's pain presentation. Therefore, clinical interventions aimed at improving the structural balance of the head, neck, and thorax are important augmentations to any direct intervention at the TM joint. Task modification in her work environment and stress management strategies may be therapeutic adjuncts.

SUMMARY

The TM joints are unique both structurally and functionally. The magnitude and frequency of mandibular movement, the daily resistance encountered during mastication, the physical stress imposed by sustained sitting and standing postures, and the chronic adaptation of muscles around the TM joint complex make this joint particularly vulnerable to problems. The influence of the cervical spine and posture upon the TM joint must always be recognized. Intervention for clients with TM disorders presents many clinical challenges, and practitioners with interest in this clientele should seek advanced education beyond the entry level in this specialty area. As we proceed in subsequent chapters to examine the joint complexes of the appendicular skeleton, evidence will indicate that each complex has its own unique features. We will not see again, however, the complexity of intra-articular and discal motions observed at the TM joints.

STUDY QUESTIONS



1. Describe the articulating surfaces of the TM joint.
2. Although the TM joint is classified as a synovial joint, its articular surfaces are covered with fibrocartilage rather than hyaline cartilage. Discuss the significance of this difference.
3. What is the significance of the differing thicknesses and the differing vascularity of the disc?
4. How do the superior and inferior laminae of the retrodiscal area differ?
5. Describe the motions in the upper and lower joints during mouth opening and closing.
6. What limits posterior motion of the condyle? How is the motion limited?
7. What are the consequences of a left TM joint that does not translate?
8. What are the consequences of a right disc that does not rotate freely over the condyle?
9. Describe the control of the disc in moving from an open-mouth to a closed-mouth position.
10. What is the potential impact of the posture of the cervical spine on the function of the TM joint complex?
11. Discuss the effects of aging on the TM joint.
12. Compare and contrast the functional presentations of hypomobile and hypermobile TM joint complexes.

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Section 3

Upper Extremity Joint Complexes

Chapter 7 **The Shoulder Complex**
Chapter 8 **The Elbow Complex**
Chapter 9 **The Wrist and Hand Complex**

Chapter 7

The Shoulder Complex

Paula M. Ludewig, PT, PhD, and John D. Borstad, PT, PhD

Introduction

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  - Sternoclavicular Articulating Surfaces
  - Sternoclavicular Disc
  - Sternoclavicular Joint Capsule and Ligaments
  - Sternoclavicular Motions
  - Sternoclavicular Stress
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  - Acromioclavicular Articulating Surfaces
  - Acromioclavicular Joint Disc
  - Acromioclavicular Capsule and Ligaments
  - Acromioclavicular Motions
  - Acromioclavicular Stress
- Scapulothoracic Joint
  - Resting Position of the Scapula
  - Motions of the Scapula
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Integrated Function of the Shoulder Complex
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- Sternoclavicular and Acromioclavicular Contributions
  - Upward Rotators of the Scapula
- Structural Dysfunction

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- Supraspinatus Muscle Function
- Infraspinatus, Teres Minor, and Subscapularis Muscle Function
- Upper and Lower Trapezius and Serratus Anterior Muscle Function
- Rhomboid Muscle Function

Muscles of Depression
- Latissimus Dorsi and Pectoral Muscle Function
- Teres Major and Rhomboid Muscle Function

INTRODUCTION

The shoulder complex, composed of the clavicle, scapula, and humerus, is an intricately designed combination of three joints that links the upper extremity to the thorax. The articular structures of the shoulder complex are designed primarily for mobility, allowing us to move and position the hand through a wide range of space. The **glenohumeral (GH) joint,** which links the humerus and scapula, has greater mobility than any other joint in the body. Although the components of the shoulder complex constitute half of the mass of the entire upper limb,[1] they are connected to the axial skeleton by a single joint, the **sternoclavicular (SC) joint.** As a result, muscle forces serve as a primary mechanism for securing the shoulder girdle to the thorax and providing a stable base of support for upper extremity movements.

The contradictory requirements on the shoulder complex for both mobility and stability are met through active forces, or **dynamic stabilization,** a concept of which the shoulder complex is considered a classic example. In essence, dynamic stability exists when a moving segment or set of segments is limited very little by passive forces such as articular surface configuration, capsule, or ligaments and instead relies heavily on active forces or dynamic muscular control. Dynamic stabilization results in a wide range of mobility for the shoulder complex and provides adequate stability when the complex is functioning normally. However, the competing mobility and stability demands on the shoulder girdle and the intricate structural and functional design make the shoulder complex highly susceptible to dysfunction and instability.

7-1 Patient Case

Susan Sorenson is a 42-year-old dental hygienist who presents to the clinic with a chief complaint of right shoulder pain. She localizes the pain primarily at the lateral proximal humerus (C5 dermatome region) but also reports pain in the upper trapezius. Symptoms include pain and fatigue with elevating her arm and the inability to sleep on her right shoulder. Her medical history includes a diagnosis of early-stage breast cancer in the right breast 6 months ago. She had a lumpectomy with sentinel node biopsy, followed by radiation treatments for 5 weeks. She finished treatment 4 months ago. She reports feelings of tightness over the anterior chest region when she raises her right arm. Her history also includes a right acromioclavicular joint separation many years ago, for which she was immobilized in a sling for several weeks with no further treatment.

COMPONENTS OF THE SHOULDER COMPLEX

The osseous segments of the shoulder complex are the clavicle, scapula, and humerus (Fig. 7–1). These three segments are joined by three interdependent linkages: the sternoclavicular joint, the **acromioclavicular (AC) joint,** and the glenohumeral joint. The articulation between the scapula and the thorax is often described as the **scapulothoracic (ST) "joint,"** although it does not have the characteristics of a fibrous, cartilaginous, or synovial joint. Instead, scapula motion on the thorax is a function of sternoclavicular, acromioclavicular, or combined sternoclavicular and acromioclavicular joint motions. The scapulothoracic joint is frequently described in the literature as a "functional joint." An additional "functional joint" that is often considered part of the shoulder complex is the **subacromial** (or **suprahumeral**) **"joint."** This functional joint is formed by movement of the head of the humerus below the coracoacromial arch. Although the movement in this functional joint plays an important role in shoulder function and dysfunction, we will refer to it as the **subacromial space** and consider it a component of the glenohumeral joint rather than a separate joint.

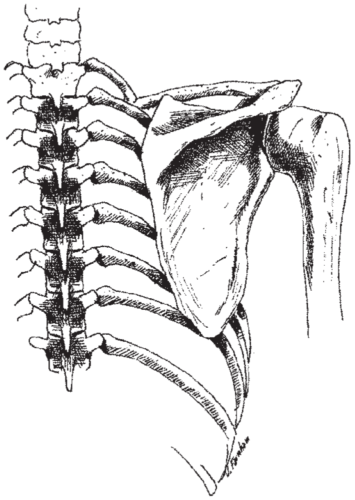


Figure 7–1 A posterior view of the three components of the shoulder complex: the humerus, the scapula, and the clavicle.

The joints that compose the shoulder complex can, in combination with trunk motion, contribute as much as 180° of elevation to the upper extremity. **Elevation** of the upper extremity refers to the combination of scapular, clavicular, and humeral motion that occurs when the arm is raised either *forward* or *to the side* (including sagittal plane flexion, frontal plane abduction, and all the motions in between). Motion of the scapula on the thorax normally contributes about one third of the total shoulder complex motion necessary for arm elevation, whereas the glenohumeral joint contributes about two thirds of the total motion. Although the integrated function of all three joints is of primary interest, each of the articulations and components of the shoulder complex must be examined individually before their integrated dynamic function can be appreciated.

Sternoclavicular Joint

The sternoclavicular joint serves as the only structural attachment of the shoulder complex and upper extremity to the axial skeleton. The sternoclavicular joint is a plane synovial joint with three rotary and three translatory degrees of freedom. The joint has a synovial capsule, a joint disc, and three major ligaments. Rotations at the sternoclavicular joint produce movement of both the clavicle and the scapula under conditions of normal function, because the scapula is linked with the lateral end of the clavicle at the acromioclavicular joint. Similarly, movement of the scapula often results in movement of the clavicle at the sternoclavicular joint.

Sternoclavicular Articulating Surfaces

The sternoclavicular articulation consists of two shallow, saddle-shaped surfaces, one at the medial end of the clavicle and one at the notch formed by the manubrium of the sternum and first costal cartilage (Fig. 7–2). The articular surfaces vary in morphology among people, and at least 10% of the population demonstrates side-to-side asymmetries in joint space and dimension.[2] The sternoclavicular joint is often classified as a plane synovial joint because the saddle shape of the articular surfaces is very subtle. The medial end of the clavicle and the manubrium are incongruent, with little direct contact between their articular surfaces. The superior portion of the medial clavicle does not contact the manubrium at all but serves as the attachment for the **sternoclavicular joint disc** and the **interclavicular ligament**. At rest, the sternoclavicular joint space is wedge-shaped and open superiorly. Movements of the clavicle in relation to the manubrium result in changes to the areas of contact between the clavicle, the sternoclavicular joint disc, and the manubriocostal cartilage.

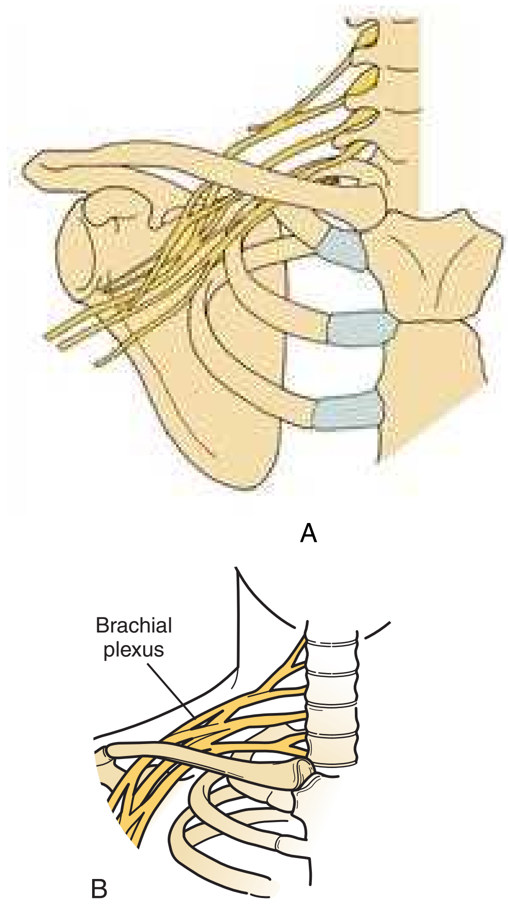


Figure 7–2 **A.** The sternoclavicular joint is the articulation of the medial clavicle with the manubrium and first costal cartilage. **B.** The saddle-shaped surfaces of the medial clavicle and manubrium are reciprocally convex and concave.

Sternoclavicular Disc

The sternoclavicular joint has a fibrocartilage disc, or meniscus, that increases congruence between the articulating surfaces. The upper portion of the sternoclavicular disc is attached to the posterosuperior clavicle, while the lower portion is attached to the manubrium and first costal cartilage, as well as to the anterior and posterior aspects to the fibrous sternoclavicular capsule.[3] The disc transects the sternoclavicular joint space diagonally (Fig. 7–3), dividing the joint into two separate cavities.[1] During shoulder motion, the disc acts like a hinge or pivot point for the medial end of the clavicle.

During **elevation** and **depression** of the clavicle, the medial articular surface rolls and slides on the relatively stationary disc, with the upper attachment of the disc serving as a pivot point. During **protraction** and **retraction** of the clavicle, the sternoclavicular disc and medial articular surface roll and slide together on the manubrial facet, with the lower attachment of the disc serving as a pivot point.[1] The disc, therefore, is considered part of the manubrium during elevation and depression and part of the clavicle during protraction and retraction. The sternoclavicular disc provides stability by increasing joint congruence and by absorbing forces transmitted along the clavicle from its lateral end to the sternoclavicular joint. In Figure 7–3, note that the unique diagonal attachment of the sternoclavicular disc helps limit medial movement of the clavicle, which might otherwise cause the medial articular surface of the clavicle to override the shallow manubrial facet. The disc also has a substantial area of contact with the medial clavicle, which helps dissipate medially directed forces, protecting the small manubrial facet from high concentrations of pressure. Although one might think that medially directed forces on the clavicle

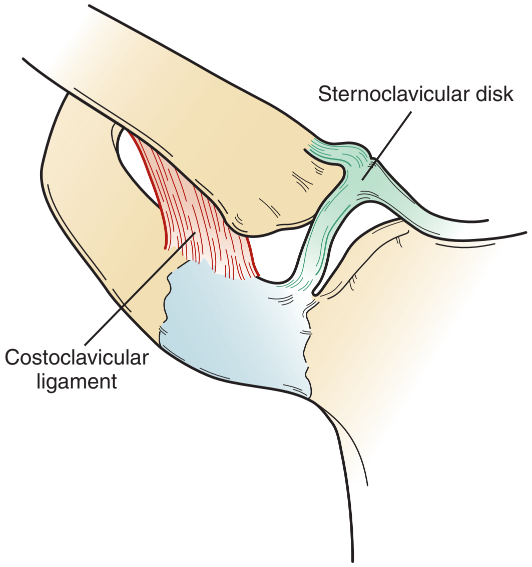


Figure 7–3 The sternoclavicular disc transects the joint into two separate joint cavities.

are infrequent and minimal, we shall see that this is not the case when we examine the function of the acromioclavicular joint, the **upper trapezius muscle,** and the **coracoclavicular ligament.**

Continuing Exploration 7-1:

Three-Compartment Sternoclavicular Joint

Anatomic examination of the sternoclavicular articulation has led to the proposal that there are three, rather than two, functional units of the sternoclavicular joint: a lateral compartment between the disc and clavicle for elevation and depression, a medial compartment between the disc and manubrium for protraction and retraction, and a costoclavicular joint for anterior and posterior long axis rotation. Anterior and posterior rotation are thought to occur between a portion of the disc over the first rib and a "conus" on the anteroinferior edge of the articular surface of the medial clavicle.[4]

Sternoclavicular Joint Capsule and Ligaments

The sternoclavicular joint is surrounded by a fairly strong fibrous capsule but depends on three ligament complexes for the majority of its support: the **anterior** and **posterior sternoclavicular ligaments,** the bilaminar **costoclavicular ligament,** and the interclavicular ligament (Fig. 7–4). The anterior and posterior sternoclavicular ligaments reinforce the capsule and function primarily to check anterior and posterior translatory movement of the medial end of the clavicle. The posterior capsule provides the primary restraint to both anterior and posterior translations of the medial clavicle on the sternum.[5] The costoclavicular ligament is a very strong ligament between the clavicle and the first rib and has two segments, or *laminae*. The anterior lamina has fibers that are directed laterally from the first rib to the clavicle, whereas the fibers of the posterior lamina are directed medially from the rib to the clavicle.[3,6] Both laminae limit elevation of the lateral end of the clavicle and, when taut, may contribute to the inferior gliding of the medial clavicle on the manubrium that occurs with clavicular elevation.[7] The costoclavicular ligament also limits the superiorly directed forces applied to the clavicle by the sternocleidomastoid and sternohyoid muscles. The posterior lamina will resist medial movement of the clavicle, absorbing some of the force that would otherwise be imposed on the sternoclavicular disc. The interclavicular ligament limits excessive depression of the distal clavicle and superior gliding of the medial clavicle on the manubrium.[8,9] The limitation of clavicular depression by the interclavicular ligament is critical to protecting structures such as the brachial plexus and subclavian artery, which pass under the clavicle and over the first rib (see Fig 7–2).

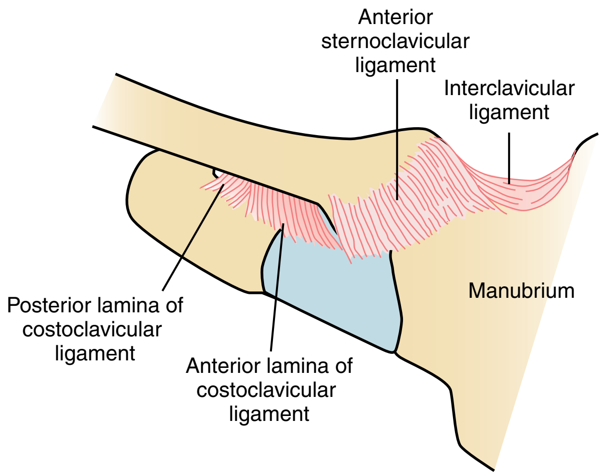


Figure 7–4 The sternoclavicular joint ligaments. The axes for sternoclavicular joint motion appear to occur at the location of the costoclavicular ligament.

Sternoclavicular Motions

The three rotary degrees of freedom at the sternoclavicular joint are most commonly described as elevation/depression, protraction/retraction, and **anterior/posterior rotation** of the clavicle. Motions of any joint are typically described by identifying the direction of movement of the portion of the lever that is farthest from the joint. The horizontal alignment of the clavicle (rather than the vertical alignment of most of the appendicular levers of the skeleton) can sometimes create confusion and impair visualization of the clavicular motions. The motions of elevation/depression (Fig. 7–5) and protraction/retraction (Fig. 7–6) should be visualized by referencing movement of the lateral end of the

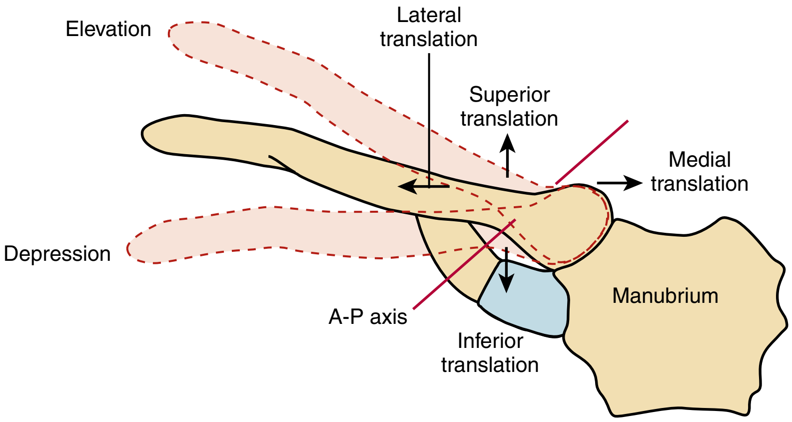


Figure 7–5 Clavicular elevation/depression at the sternoclavicular joint occurs as movement of the lateral clavicle about an A-P axis. The medial clavicle also has small magnitudes of medial/lateral translation and superior/inferior translation at the sternoclavicular joint.

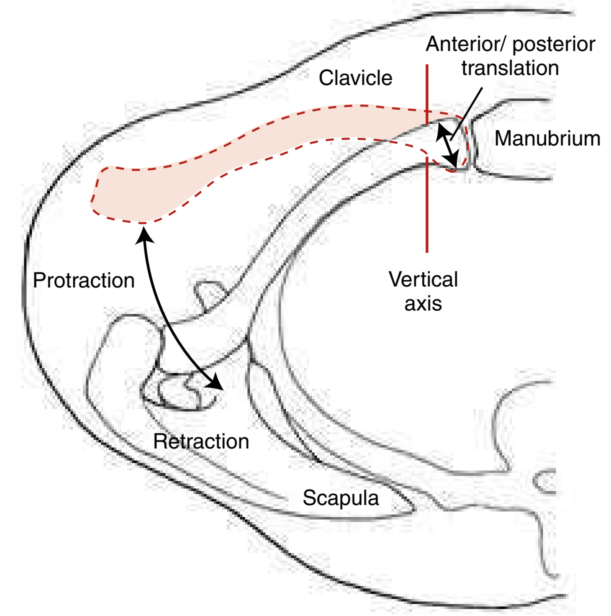


Figure 7–6 Shown in a superior view, clavicular protraction/retraction at the sternoclavicular joint occurs as movement of the lateral clavicle (and attached scapula) around a vertical axis. The medial clavicle also has a small magnitude of anterior/posterior translation at the sternoclavicular joint.

clavicle. Clavicular anterior/posterior rotations are long-axis rolling motions of the entire clavicle (Fig. 7–7). Three degrees of translatory motion at the sternoclavicular joint can also occur, although they are very small in magnitude. Translations of the medial clavicle on the manubrium are usually defined as occurring in anterior/posterior, medial/lateral, and superior/inferior directions (see Figs. 7–5 and 7–6).

Elevation and Depression of the Clavicle

The motions of elevation and depression occur around an approximately anteroposterior (A-P) axis (see Fig. 7–5) between a convex clavicular surface and a concave surface formed by the manubrium and the first costal cartilage. With elevation, the lateral clavicle rotates upward; with depression, the lateral clavicle rotates downward. The sternoclavicular joint axis is described as lying lateral to the joint at the costoclavicular ligament. The location of this functional (rather than anatomic) axis relatively far from the joint results in a larger intra-articular motion of the medial clavicle. The cephalocaudal shape of the articular surfaces and the location of the axis indicate that the convex surface of the clavicle must slide inferiorly on the concave manubrium and first costal cartilage during elevation, in a direction opposite to movement of the lateral end of the clavicle. The range of available clavicular elevation has been described as up to 48°, whereas passive depression is limited, on average, to less than 15°.[10] The full magnitude of the available range of elevation is generally not utilized during functional ranges of arm elevation.[11,12]

Protraction and Retraction of the Clavicle

Protraction and retraction of the clavicle occur at the sternoclavicular joint around an approximately vertical (superoinferior) axis that also appears to lie at the costoclavicular ligament (see Fig. 7–6). With protraction, the lateral clavicle moves anteriorly, and with retraction, the lateral clavicle moves posteriorly. The configuration of joint surfaces in this plane is the opposite of that for elevation/depression; the medial end of the clavicle is concave, and the manubrial side of the joint is convex. During protraction, the medial clavicle is expected to slide anteriorly on the manubrium and first costal cartilage. There is about 15° to 20° protraction and 30° or greater retraction of the clavicle available.[10,12–14]

Anterior and Posterior Rotation of the Clavicle

Anterior/posterior, or long axis, rotation of the clavicle (see Fig. 7–7) occurs as a spin between the saddle-shaped surfaces of the medial clavicle and manubriocostal facet. Unlike many joints, which can rotate in either direction from resting position of the joint, the clavicle rotates primarily in only one direction from its resting position. The clavicle rotates posteriorly from neutral, bringing the inferior surface of the clavicle to face anteriorly. This has also been referred to as backward or upward rotation rather than posterior rotation.[1] From its fully rotated position, the clavicle can rotate anteriorly again to return to neutral. Available anterior rotation past neutral is very limited, generally described as less than 10°.[1] The range of available clavicular posterior rotation may be as much as 50°.[11] The axis of rotation runs longitudinally through the clavicle, intersecting the sternoclavicular and acromioclavicular joints.

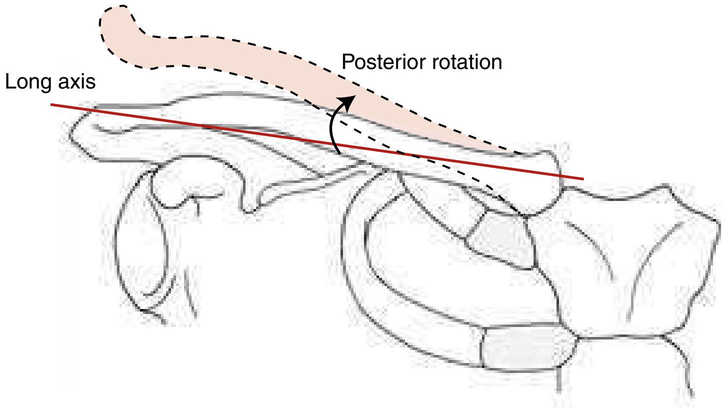


Figure 7–7 Clavicular rotation at the sternoclavicular joint occurs as a spin of the entire clavicle around a long axis that has a medial/lateral orientation. As the clavicle posteriorly rotates, the lateral end flips up; anterior rotation is a return to resting position.

Sternoclavicular Stress

The bony morphology, capsule, ligaments, and disc of the sternoclavicular joint combine to create an articulation that contributes to the great mobility of the arm while remaining a stable link to the axial skeleton. Although the sternoclavicular joint is considered incongruent, it does not undergo the degree of degenerative change common to the other joints of the shoulder complex.[15,16] Strong force-dissipating structures such as the sternoclavicular disc and the costoclavicular ligament minimize articular stresses and also prevent excessive intra-articular motion, which could lead to subluxation or dislocation. Dislocations of the sternoclavicular joint represent only 1% of joint dislocations in the body and only 3% of shoulder girdle dislocations.[17]

Acromioclavicular Joint

The acromioclavicular joint attaches the scapula to the clavicle. It is generally described as a plane synovial joint with three rotational and three translational degrees of freedom. It has a joint capsule, two major ligaments, and a joint disc that may or may not be present. The primary function of the acromioclavicular joint is to allow the scapula to rotate in three dimensions during arm movement so that upper extremity motion is increased, the glenoid is positioned beneath the humeral head, and the scapula remains congruent and stable on the thorax. The acromioclavicular joint also allows transmission of forces from the upper extremity to the clavicle.

Acromioclavicular Articulating Surfaces

The acromioclavicular joint consists of the articulation between the lateral end of the clavicle and a small facet on the acromion of the scapula (Fig. 7–8A). The articular facets are considered to be incongruent and vary in configuration. They may be flat, reciprocally concave-convex, or reversed (reciprocally convex-concave).[6] The vertical inclination of the articulating surfaces varies from individual to individual. Depalma[15] described three joint types in which the angle of inclination of the contacting surfaces varied between 16° and 36° from vertical and suggested that the closer the surfaces are to vertical, the more prone the joint is to the wearing effects of shear forces. However, greater arthritic changes are not apparent in acromioclavicular joints with more vertical orientations.[18]

Acromioclavicular Joint Disc

The disc of the acromioclavicular joint (Fig. 7–8B) may vary in size between individuals, within an individual as they age, and between shoulders of the same individual. Through 2 years of age, the joint is actually a fibrocartilaginous union. With use of the upper extremity, a joint space develops at each articulating surface that may leave a “meniscoid” fibrocartilage remnant within the joint.[16]

Acromioclavicular Capsule and Ligaments

The capsule of the acromioclavicular joint is weak and cannot maintain integrity of the joint without the reinforcement of the **superior** and **inferior acromioclavicular ligaments** and the coracoclavicular ligaments (Fig. 7–9). The acromioclavicular ligaments assist the capsule in apposing the articular surfaces[19] while the superior acromioclavicular is the main ligament limiting movement caused by anterior forces applied to the distal clavicle.[20] The fibers of the superior acromioclavicular ligament are reinforced by aponeurotic fibers of the **trapezius** and **deltoid muscles,** making the superior joint support stronger than the inferior.

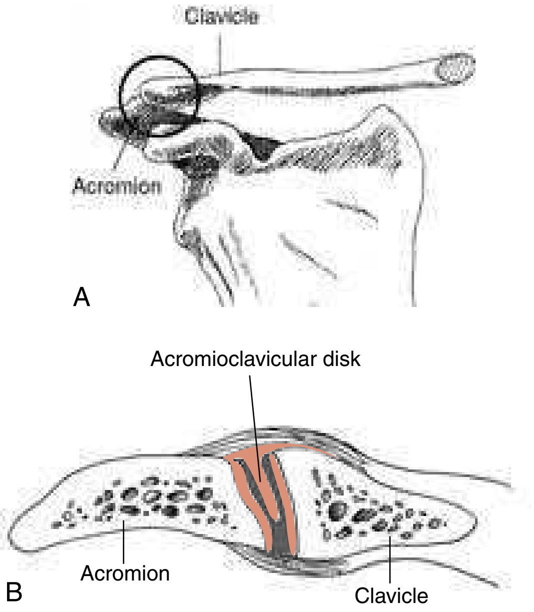


Figure 7–8 **A.** The acromioclavicular joint. **B.** A cross-section of the acromioclavicular joint shows the disc and the angulation of the articular surfaces.

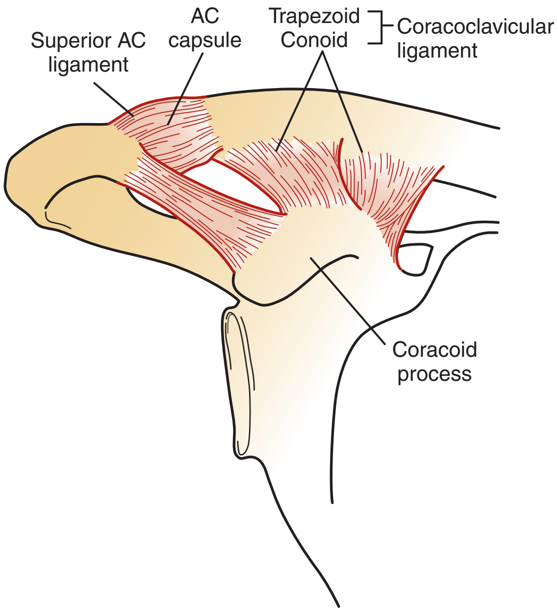


Figure 7–9 The acromioclavicular joint capsule and ligaments, including the coracoclavicular ligament with its conoid and trapezoid portions.

The coracoclavicular ligament, although not belonging directly to the anatomic structure of the acromioclavicular joint, firmly unites the clavicle and scapula and provides much of the joint's superior and inferior stability.[19] This ligament is divided into a medial portion, the **conoid ligament,** and a lateral portion, the **trapezoid ligament.** The conoid ligament, medial and slightly posterior to the trapezoid, is more triangular and vertically oriented. The trapezoid ligament is quadrilateral in shape and is nearly horizontal in orientation.[6] The two portions are separated by adipose tissue and a large bursa.[9] Both ligaments attach to the undersurface of the clavicle and influence the biomechanics of the shoulder in a way described in the Integrated Function of the Shoulder Complex section. Although the acromioclavicular capsule and ligament can resist small rotary and translatory forces at the acromioclavicular joint, restraint of larger translations is credited to the coracoclavicular ligament.

The conoid portion of the coracoclavicular ligament provides the primary restraint to translatory motion caused by superior-directed forces applied to the distal clavicle (or, conversely, to translatory motion caused by inferior-directed forces applied to the acromion).[20] The trapezoid portion of the coracoclavicular ligament provides more restraint than the conoid portion to translatory motion caused by posterior-directed forces applied to the distal clavicle.[21,22] In addition, both portions of the coracoclavicular ligament limit upward rotation of the scapula at the acromioclavicular joint. When medially directed forces on the humerus are transferred to the glenoid fossa of the scapula, medial displacement of the scapula on the clavicle is prevented by the coracoclavicular ligament complex, in particular the horizontal trapezoid portion. These medial forces are transferred to the clavicle and then to the strong sternoclavicular joint (Fig. 7–10). One of the most critical roles played by the coracoclavicular ligament in integrated shoulder function is to couple the posterior rotation of the clavicle to scapula rotation during arm elevation.

Acromioclavicular Motions

The articular facets of the acromioclavicular joint are small, afford limited motion, and have a wide range of individual differences. For these reasons, studies are inconsistent in identifying the movement and axes of motion for this joint. The primary rotary motions that take place at the acromioclavicular joint are **internal/external rotation, anterior/posterior tilting** or **tipping,** and **upward/downward rotation.** These motions occur around axes that are oriented to the **plane of the scapula** rather than to the cardinal planes. Although internal/external rotation occurs around an essentially vertical axis, anterior/posterior tilting occurs around an oblique "coronal" axis, and upward/downward rotation around an oblique "A-P" axis (Fig. 7–11). Terminology for the acromioclavicular motions, as well as for motions of the scapula on the thorax, varies widely. The acromioclavicular joint also influences and is influenced by rotation of the clavicle around its long axis. In addition, translatory motions at the acromioclavicular joint can occur, although, as in the case of the sternoclavicular joint, these motions are typically small in magnitude. These translations are usually defined as anterior/posterior, medial/lateral, and superior/inferior.

Internal and External Rotation

Internal/external rotation of the scapula in relation to the clavicle occurs around an approximately vertical axis through the acromioclavicular joint. Internal and external rotation at the acromioclavicular joint can best be visualized

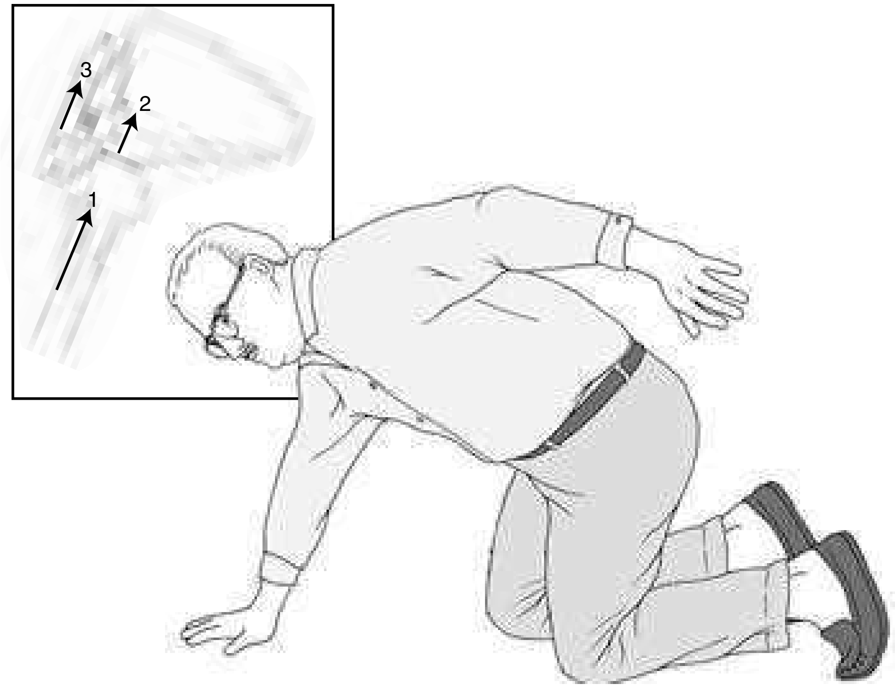


Figure 7–10 When a person bears weight on an arm, a medially directed force up the humerus (1) is transferred to the scapula (2) through the glenoid fossa and then to the clavicle (3) through the coracoclavicular ligament.

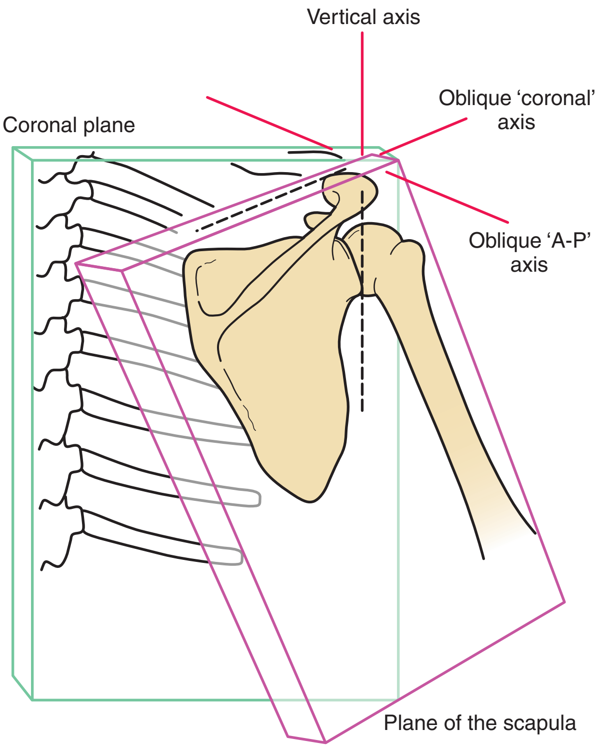


Figure 7–11 The acromioclavicular rotary axes of motion are oriented in relation to the plane of the scapula, rather than in relation to the cardinal planes.

as bringing the glenoid fossa of the scapula anteromedially and posterolaterally, respectively (Fig. 7–12A). These motions occur in part to maintain contact of the scapula with the horizontal curvature of the thorax as the clavicle protracts and retracts, sliding the scapula around the thorax in scapular protraction and retraction. These motions also "aim" the glenoid fossa toward the plane of humeral elevation (Fig. 7–12B). The orientation of the glenoid fossa is important for maintaining congruency with the humeral head; maximizing the function of glenohumeral muscles, capsule, and ligaments; maximizing the stability of the glenohumeral joint; and maximizing available motion of the arm. The available range of motion (ROM) at the acromioclavicular joint is difficult to measure. Dempster provided a range of 30° for combined internal and external ROM in cadaveric acromioclavicular joints separated from the thorax.[1] Smaller values (20° to 35°) have been reported in vivo during arm motions, although up to 40° to 60° may be possible with full-range motions reaching forward and across the body.[10,12,13,14]

Anterior and Posterior Tilting

The second acromioclavicular motion is anterior/posterior tilting or tipping of the scapula in relation to the clavicle around an oblique "coronal" axis through the joint. Anterior tilting results in the acromion tilting forward and the inferior angle tilting backward (Fig. 7–13A). Posterior tilting rotates the acromion backward and the inferior angle forward. Scapular tilting, like internal/external rotation of the scapula, occurs in order to maintain the contact of the scapula with the contour of the rib cage and to orient the glenoid fossa. As the scapula moves upward or downward on the rib cage in elevation or depression, the scapula can adjust its position to maintain contact with the vertical curvature of the ribs (Fig. 7–13B). Elevation of the scapula on the thorax, such as occurs with a shoulder shrug, can result in anterior tilting. The scapula does not always follow the curvature of the thorax precisely. During normal flexion or abduction of the arm, the scapula posteriorly tilts on the thorax as the scapula is upwardly rotating. Available passive

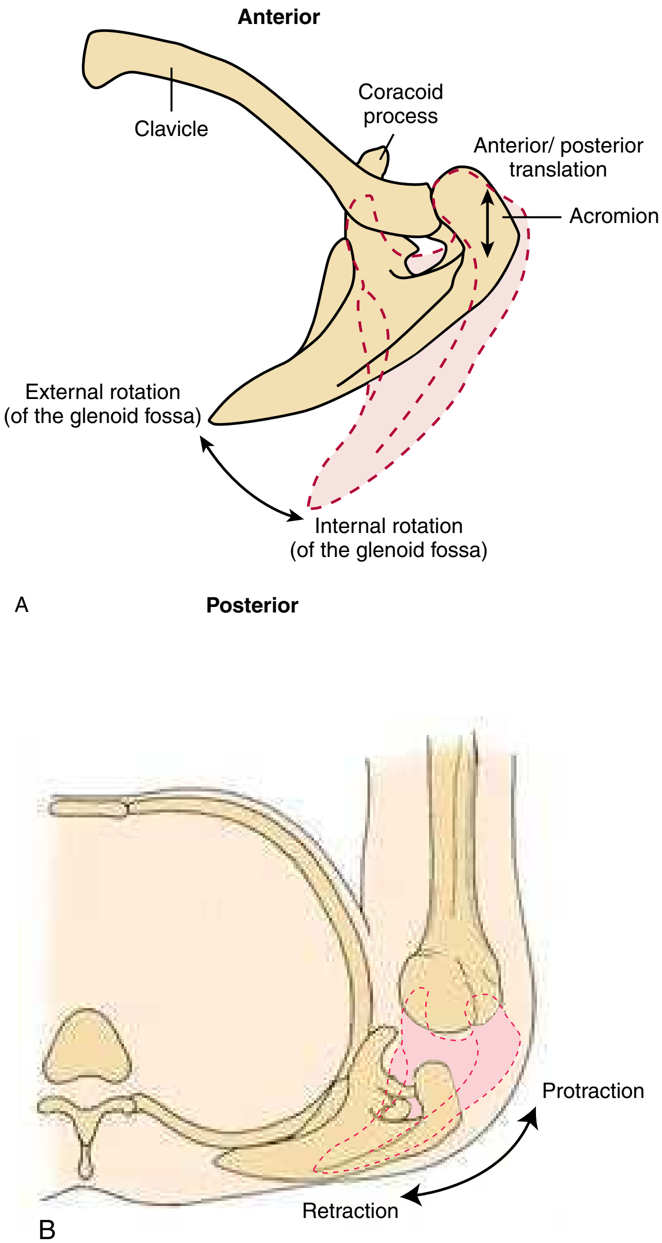


Figure 7–12 **A.** A superior view of scapular internal and external rotation at the acromioclavicular joint. Although the directional arrows are drawn at the vertebral border, the motions are named with the glenoid fossa of the scapula as the reference. The acromion also has small amounts of anterior and posterior translatory motions that can occur. **B.** Protraction and retraction of the scapula require internal and external rotation for the scapula to follow the convex thorax and orient the glenoid fossa with the plane of elevation.

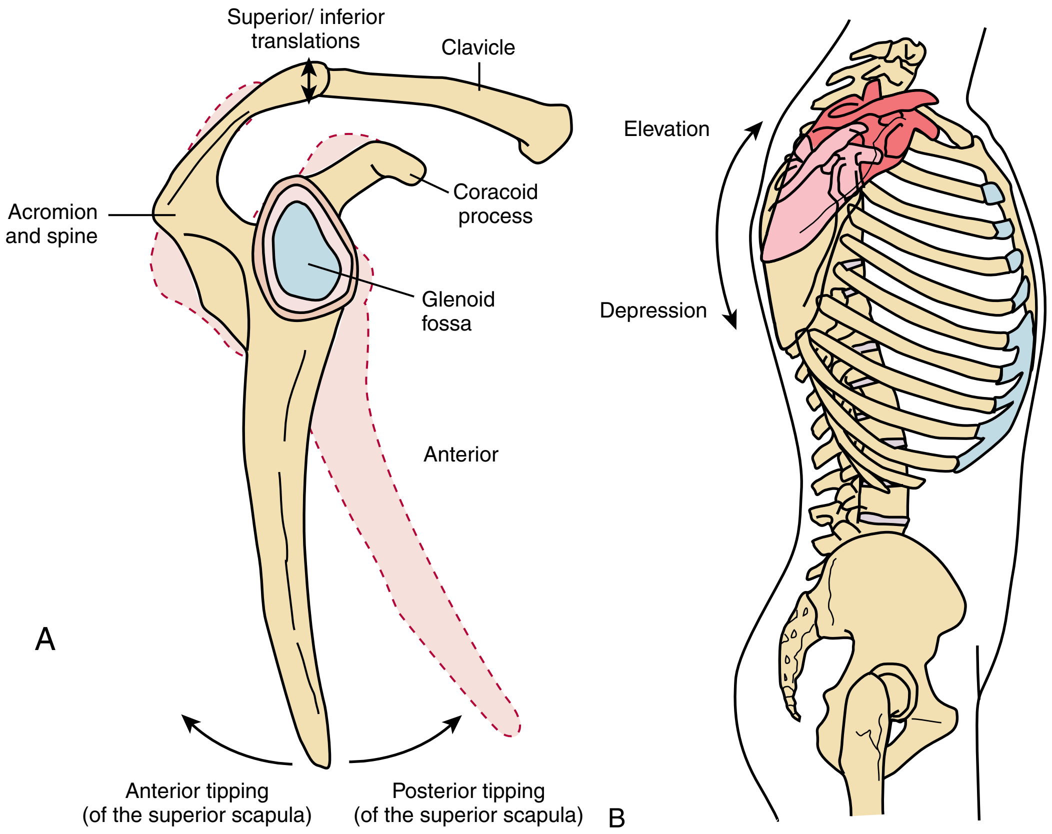


Figure 7–13 **A.** A lateral view of scapular anterior and posterior tilting at the acromioclavicular joint. Although the directional arrows are drawn at the inferior angle, the motions are named with the superior aspect of the scapula as the reference. The acromion also has small amounts of anterior and posterior translatory motions that can occur. **B.** Elevation and depression of the scapula can include anterior and posterior tilting, respectively, for the scapula to follow the convex thorax.

motion into anterior/posterior tilting at the acromioclavicular joint is 60° in cadaveric acromioclavicular joint specimens separated from the thorax.[1] The magnitude of anterior/posterior tilting during in vivo arm elevation has been quantified as approximately 20°, although up to 40° or more may be possible in the full range from maximum flexion to extension.[14,23]

Upward/Downward Rotation

The third acromioclavicular joint motion is upward/downward rotation of the scapula in relation to the clavicle about an oblique "A-P" axis approximately perpendicular to the plane of the scapula, passing midway between the joint surfaces of the acromioclavicular joint. Upward rotation tilts the glenoid fossa upward (Fig. 7–14), and downward rotation is the opposite motion. The amount of available passive motion into upward/downward rotation specifically at the acromioclavicular joint is limited by the attachment of the coracoclavicular ligament. In order for upward rotation to occur at the acromioclavicular joint, the coracoid process and superior border of the scapula need to move inferiorly away from the clavicle, a motion restricted by tension in the coracoclavicular ligaments. However, Dempster described 30° of available passive ROM into upward/downward rotation.[1] The amount of available upward rotation is dependent in part on clavicular long axis rotation. Because of the attachment of the coracoclavicular ligaments to the undersurface and posterior edge of the clavicle, posterior rotation of the clavicle releases tension on the coracoclavicular ligaments and "opens up" the acromioclavicular joint, allowing

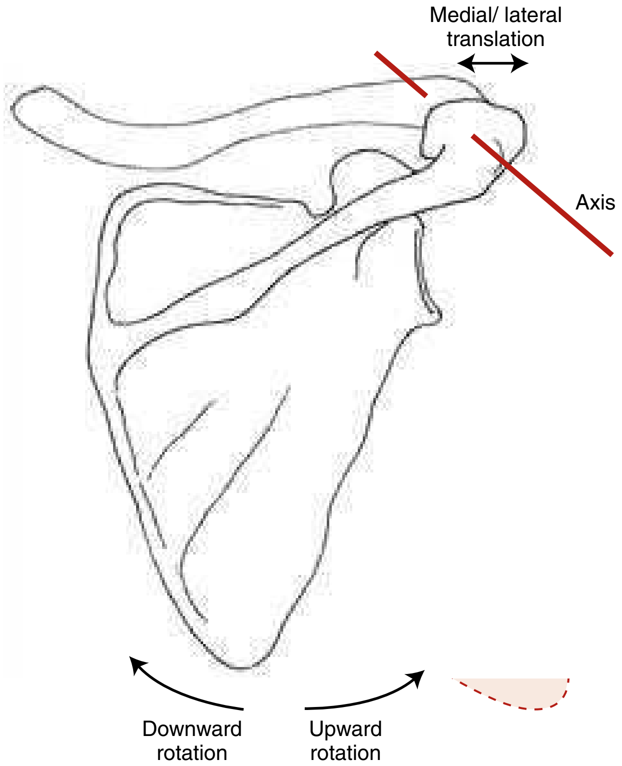


Figure 7–14 Upward/downward rotation of the scapula at the acromioclavicular joint occurs around an approximately A-P (perpendicular to the plane of the scapula) axis. Although the directional arrows are drawn at the inferior angle, the motions are named with the glenoid fossa of the scapula as the reference. The acromion also has a small magnitude of medial and lateral translatory motions that can occur.

upward rotation to occur.[11] Conway describes 30° of upward rotation and 17° of downward rotation actively at the acromioclavicular joint in vivo.[10] Others have described 15° to 16° of upward rotation occurring during active humeral abduction.[14,23]

Acromioclavicular Stress

Unlike the sternoclavicular joint, the acromioclavicular joint is susceptible to both trauma and degenerative changes. Trauma to the acromioclavicular joint most often occurs in the first three decades of life, during either contact sports or a fall on the shoulder with the arm adducted. Typically the result of high inferior forces on the acromion, trauma results in acromioclavicular joint disruption ranging from sprains and subluxations to dislocations. Degenerative change is common from the second decade on,[16] with the joint space frequently narrowed by the sixth decade.[24] acromioclavicular joint degeneration is likely due to small and incongruent articular surfaces that result in high forces per unit area.

Continuing Exploration 7-2:

Classifying Acromioclavicular Dislocations

The acromioclavicular joint is susceptible to traumatic injury through accidents or contact sports. These acromioclavicular dislocations are graded by the relationship of the displacement between the acromion and clavicle and by the amount of this displacement. Various classification schemes exist; most commonly, type I injuries consist of a sprain to the acromioclavicular ligaments only, type II injuries consist of ruptured acromioclavicular ligaments and sprained coracoclavicular ligaments, and type III injuries result in the rupture of both sets of ligaments, with a 25% to 100% increase in coracoclavicular space. Type II and III acromioclavicular dislocations involve inferior displacement of the acromion in relation to the clavicle due to loss of support from the coracoclavicular ligaments. Type IV injuries have a posteriorly displaced lateral clavicle, often pressing into the trapezius posteriorly, with complete rupture of both the acromioclavicular and coracoclavicular ligaments. Type V injuries also involve an inferior displacement of the acromion and complete rupture of both sets of ligaments, but unlike type III, they exhibit between three and five times greater coracoclavicular space than normal. Type VI injuries have an inferiorly displaced clavicle in relation to the acromion, with complete ligament rupture and displacement of the distal clavicle into a subacromial or subcoracoid position. Types IV, V, and VI are much rarer injuries, and each requires surgical management.[25]

CASE APPLICATION

Acromioclavicular Joint Injury

case 7–1

Our patient, Ms. Sorenson, reports a past acromioclavicular joint separation treated by immobilization with the arm at the side in a sling. This treatment is consistent with an injury ranging from type I to type III in severity, which are frequently not surgically stabilized. With a type I injury, she may have healed well and may have normal acromioclavicular joint function. If the past injury was a type III injury, she may still have a substantial instability or disruption of the claviculoscapular linkage. The position and prominence of the distal clavicle on the right in comparison with her left, uninvolved side can provide some insight into her past injury. A prominent distal clavicle with a step down to the acromion (step sign) would be consistent with inferior scapular (or superior clavicle) positioning related to a past type II or III injury. Although this injury is commonly not surgically stabilized because many surgeons believe it unnecessary, follow-up studies indicate rates of residual symptoms in persons with past acromioclavicular joint injuries, from 36% in type I injuries to 69% in type III injuries.[26] One residual problem is scapular malposition and dyskinesis following type III injuries, a finding in nearly 60% of subjects in a recent analysis.[27] These reports suggest that more aggressive treatment and rehabilitation may be warranted following acromioclavicular joint injuries.

Scapulothoracic Joint

The scapulothoracic "joint" is formed by the articulation of the scapula with the thorax. It is not a true anatomic joint because it is not a union of bony segments by fibrous, cartilaginous, or synovial tissues. By contrast, the articulation of the scapula with the thorax depends on the integrity of the anatomic acromioclavicular and sternoclavicular joints. The sternoclavicular and acromioclavicular joints are interdependent with scapulothoracic motion because the scapula is attached by its acromion process to the lateral end of the clavicle through the acromioclavicular joint; the clavicle, in turn, is attached to the axial skeleton at the manubrium of the sternum through the sternoclavicular joint. Any movement of the scapula on the thorax must result in movement at the acromioclavicular joint, the sternoclavicular joint, or both. This makes the functional scapulothoracic joint part of a true closed chain with the acromioclavicular and sternoclavicular joints and the thorax. Observation and measurement of individual sternoclavicular and acromioclavicular joint motions are more difficult than observing or measuring motions of the scapula on the thorax. Consequently, scapulothoracic position and motions are described and measured far more frequently than are the sternoclavicular and acromioclavicular joint motions upon which scapulothoracic motions are dependent.

Resting Position of the Scapula

The scapula rests on the posterior thorax approximately 5 cm from the midline between the second through seventh ribs (see Fig. 7–1). The scapula is internally rotated 35° to 45° from the coronal plane (Fig. 7–15A), is tilted anteriorly approximately 10° to 15° from vertical (Fig. 7–15B), and is upwardly rotated 5° to 10° from vertical.[14,28] This magnitude of upward rotation has as its reference a "longitudinal" axis perpendicular to the axis running from the root of the scapular spine to the acromioclavicular joint (Fig. 7–15C). If the vertebral (medial) border of the scapula is used as the reference axis, the magnitude of upward rotation at rest is usually described as 2° to 3° from vertical.[29] Although these "normal" values for the resting scapula are cited, substantial individual variability exists in scapular rest position, even among healthy subjects.[29]

Motions of the Scapula

The motions of the scapula from the resting position include three rotations that have already been described

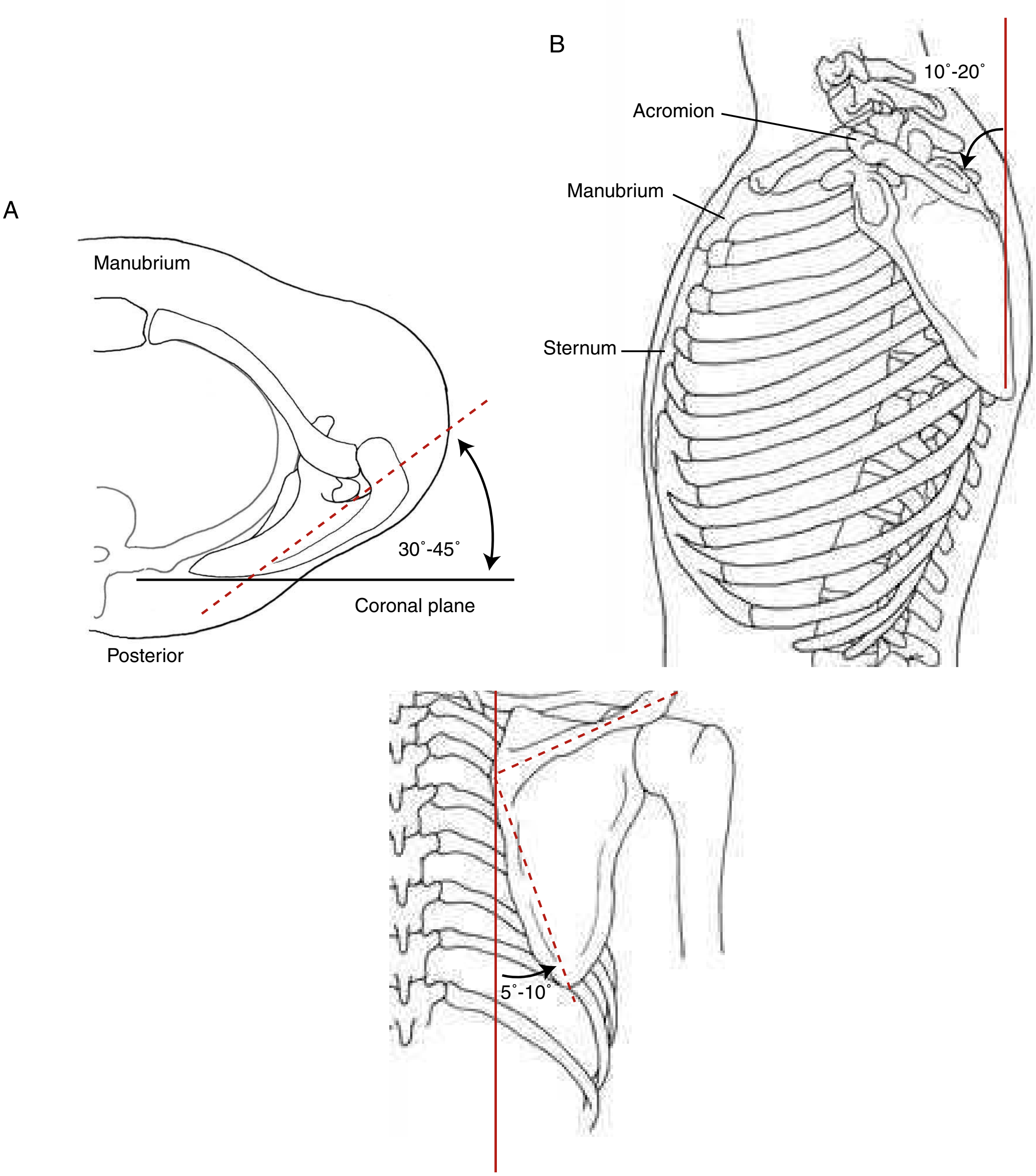


Figure 7–15 The resting position of the scapula on the thorax. **A.** In this superior view, it can be seen that the scapula rests in an internally rotated position 35° to 45° anterior to the coronal plane. **B.** In this side view, it can be seen that the scapula at rest is tilted anteriorly approximately 10° to 15° to the vertical plane. **C.** In this posterior view, the "longitudinal" axis of the scapula (90° to an axis through the spine) is upwardly rotated 5° to 10° from vertical.

because they occur at the acromioclavicular joint. These are upward/downward rotation, internal/external rotation, and anterior/posterior tilting. Of these three acromioclavicular joint rotations, only upward/downward rotation is easily observable at the scapula, and it is therefore considered for our purposes to be a “primary” scapular motion. Internal/external rotation and anterior/posterior tilting are normally difficult to observe and are therefore considered for our purposes to be “secondary” scapular motions. The scapula presumably also has available the translatory motions of scapular **elevation/depression** and **protraction/retraction.** These “primary” (readily observable) scapular motions are typically described as if they occur independently of each other. However, the linkage of the scapula to the acromioclavicular and sternoclavicular joints actually prevents scapular motions from occurring in isolation and from occurring as true translatory motions. Instead, scapular motions on the thorax must occur in combinations, such as the simultaneous upward rotation, external rotation, and posterior tilting that occur when the arm is abducted.

Upward/Downward Rotation

Upward rotation of the scapula on the thorax (Fig. 7–16) is the principal motion of the scapula observed during active elevation of the arm and plays a significant role in increasing the arm’s range of elevation overhead. Approximately 50° to 60° of upward rotation of the scapula on the thorax is typically available. Given the closed-chain relationship between the sternoclavicular, acromioclavicular, and scapulothoracic joints, differing proportions of upward/downward rotation of the scapula are contributed by sternoclavicular joint posterior/anterior rotation, by sternoclavicular joint elevation/depression, and by acromioclavicular joint upward/downward rotation.[30] Most often, scapular upward/downward rotation results from a combination of these sternoclavicular and acromioclavicular motions.[30]

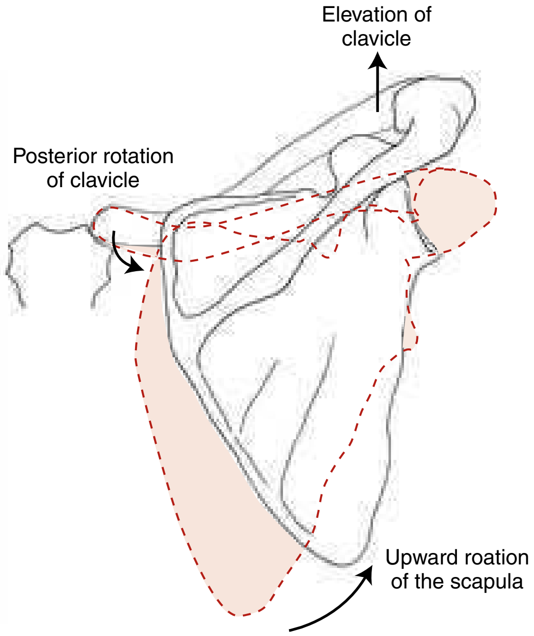


Figure 7–16 Upward rotation of the scapula is produced by clavicular posterior rotation and somewhat by elevation at the sternoclavicular joint, and by upward rotation at the acromioclavicular joint.

Because the axes of the sternoclavicular and scapulothoracic joints are not parallel, the relationships between sternoclavicular and acromioclavicular joint motions and scapulothoracic motion are more challenging to visualize. If the long axis of the clavicle were parallel to the plane of the scapula such that acromioclavicular joint internal rotation was 0° (Fig. 7–17A), *sternoclavicular joint elevation* would directly result in or couple with scapulothoracic upward rotation. If the long axis of the clavicle were perpendicular to the plane of the scapula such that acromioclavicular joint rotation was 90° (Fig. 7–17B), *sternoclavicular joint posterior rotation* would directly result in or couple with scapulothoracic upward rotation. The average angle of acromioclavicular joint internal rotation is 58° to 68° (Fig. 7–17C), or about two thirds of a 90° acromioclavicular joint internal rotation position.[14,30] Consequently, two thirds of the motion of *sternoclavicular joint posterior rotation* will translate into or couple with scapulothoracic upward rotation, while only one third of *clavicular elevation* will result in scapulothoracic upward rotation.[30]

Concept Cornerstone 7-1

Terminology

In describing upward and downward rotation of the scapula, we define upward and downward motion by the upward and downward movement of the glenoid fossa, respectively. Others use the inferior angle of the scapula as the referent, with upward and downward rotation described as movement of the inferior angle away from the vertebral column (upward rotation) or movement of the inferior angle toward the vertebral column (downward rotation). Some also refer to upward/downward rotation of the scapula (regardless of reference point on the scapula) by the names *abduction/adduction* or *lateral/medial rotation*, respectively.[1,31]

Elevation/Depression

Scapular elevation and depression can be isolated (relatively speaking) by shrugging the shoulder up and depressing the shoulder downward. Elevation and depression of the scapula on the thorax are commonly described as translatory motions in which the scapula moves upward (cephalad) or downward (caudal) along the rib cage from its resting position. Scapular elevation, however, occurs through elevation of the clavicle at the sternoclavicular joint and may include subtle adjustments in anterior/posterior tilting and internal/external rotation at the acromioclavicular joint in order to keep the scapula in contact with the thorax (Fig. 7–18).

Protraction/Retraction

Protraction and retraction of the scapula on the thorax are often described as translatory motions of the scapula away from or toward the vertebral column, respectively. These theoretical translatory motions have also been termed *scapular abduction and adduction.* However, if protraction or abduction of the scapula on the thorax occurred as a pure translatory movement, the scapula would move directly away from the vertebral column, and the glenoid fossa would face laterally. Only the vertebral border of the scapula would

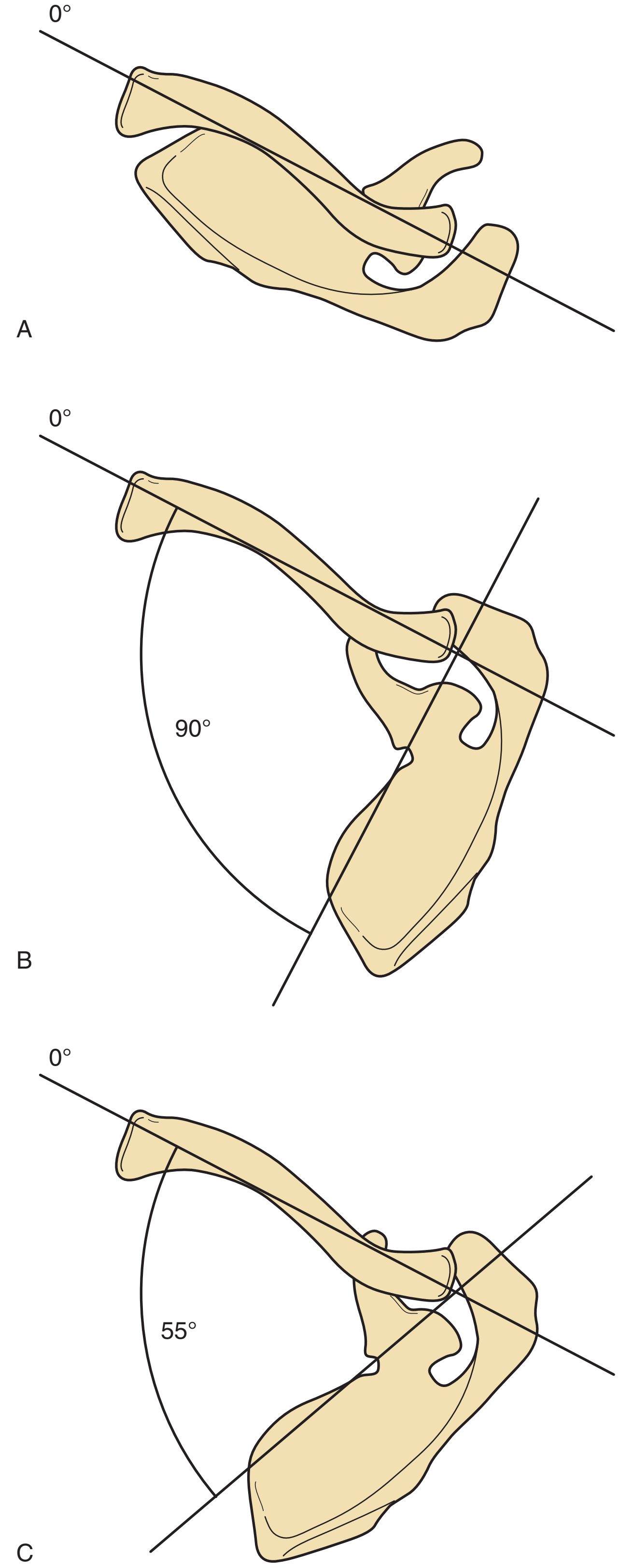


Figure 7–17 Coupling of sternoclavicular joint rotations and scapular motion on the thorax. **A.** Theoretical acromioclavicular joint internal rotation of 0°. **B.** Theoretical acromioclavicular joint internal rotation of 90°. **C.** Typical acromioclavicular joint internal rotation alignment of 55° to 68°.

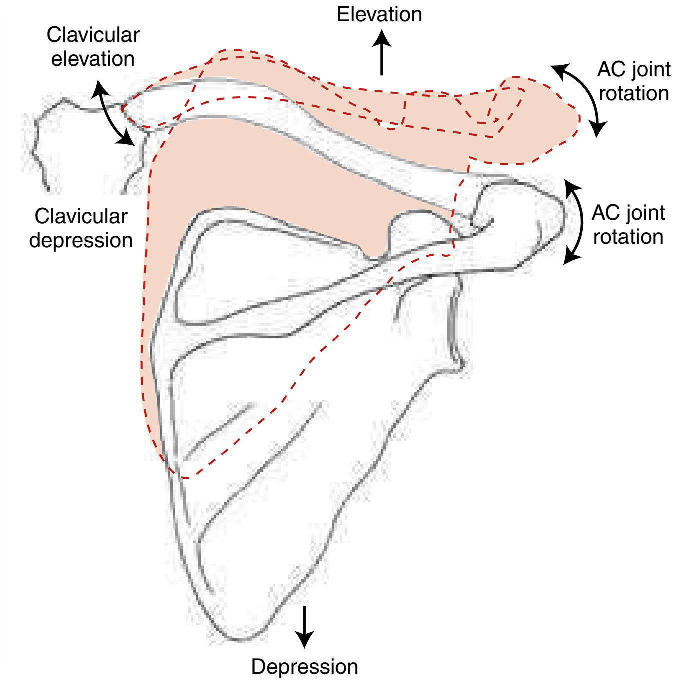


Figure 7–18 Elevation and depression of the scapula are produced by elevation/depression of the clavicle at the sternoclavicular joint and by rotations at the acromioclavicular joint.

remain in contact with the rib cage. In reality, full scapular protraction results in the glenoid fossa facing anteriorly, with the scapula in contact with the rib cage. The scapula protracts and retracts through sternoclavicular joint protraction and retraction and follows the contour of the ribs by rotating internally and externally at the acromioclavicular joint (Fig. 7–19).

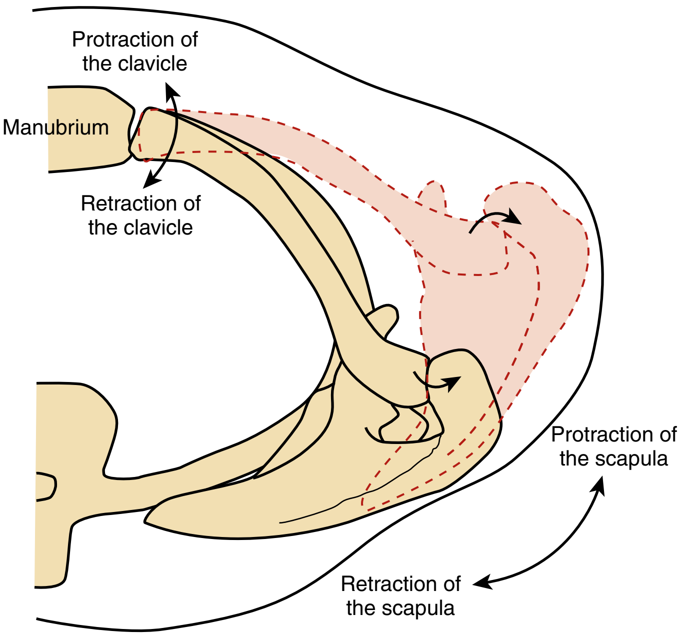
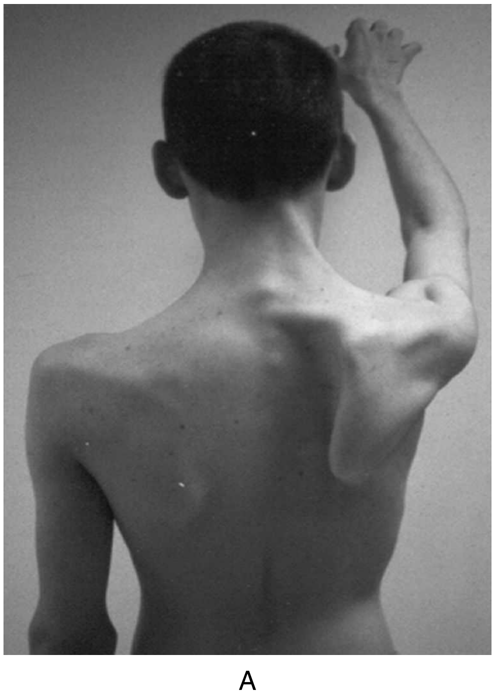


Figure 7–19 Protraction and retraction of the scapula are produced by protraction/retraction of the clavicle at the sternoclavicular joint and by rotations at the acromioclavicular joint.

Internal/External Rotation

The scapular motions of internal and external rotation are normally not overtly identifiable on physical observation but are critical to movement of the scapula along the curved rib cage. Internal/external rotation of the scapula on the thorax should normally accompany protraction/retraction of the clavicle at the sternoclavicular joint. Approximately 15° to 16° of internal rotation occurs at the acromioclavicular joint during normal elevation of the arm.[14,23] A larger amount of internal rotation of the scapula on the thorax that is isolated to (or occurs excessively at) the acromioclavicular joint results in prominence of the vertebral border of the scapula as a result of loss of contact with the thorax. This is often referred to clinically as **scapular winging** (Fig. 7–20A,B). Excessive internal rotation may be indicative of pathology or poor neuromuscular control of the scapulothoracic muscles.



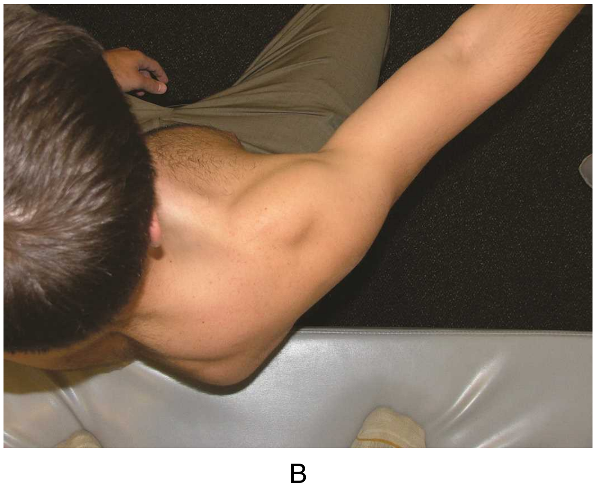


Figure 7–20 Posterior **(A)** and superior **(B)** views of scapula “winging.” Excessive internal rotation of the scapulae at the acromioclavicular joint causes prominence of the medial border of the scapula with attempted elevation of the arms.

Anterior/Posterior Tilting

As is true for internal/external rotation, anterior/posterior tilting is normally not overtly obvious on clinical observation and yet is critical to maintaining contact of the scapula against the curvature of the rib cage. Anterior/posterior tilting of the scapula on the thorax occurs at the acromioclavicular joint.[14] Because of the differing sternoclavicular and acromioclavicular axis alignment as described above, scapulothoracic anterior/posterior tilting can also couple with elevation/depression of the clavicle at the sternoclavicular joint.[30] Anterior tilting beyond resting values will result in prominence of the inferior angle of the scapula (Fig. 7–21). An anteriorly tilted scapula may occur in pathologic situations (poor neuromuscular control) or in abnormal posture.

Scapulothoracic Stability

Stability of the scapula on the thorax is provided by the structures that maintain integrity of the linked acromioclavicular and sternoclavicular joints. The muscles that attach to both the thorax and scapula maintain contact between these surfaces while producing the movements of the scapula. In addition, stabilization is provided by the scapulothoracic musculature, which pulls or compresses the scapula to the thorax.[31]

The ultimate functions of scapular motion are to orient the glenoid fossa for optimal contact with the humeral head of the maneuvering arm, to add range to elevation of the arm, and to provide a stable base for the controlled motions between the humeral head and glenoid fossa. The scapula, with its associated muscles and linkages, can perform these mobility and stability functions so well that it serves as the premier example of dynamic stabilization in the human body.

Figure 7–21 This scapula is anteriorly tilted with attempted elevation of the arm, causing the inferior angle of the scapula to lift off the thorax and become visually prominent.

Glenohumeral Joint

The glenohumeral joint is a ball-and-socket synovial joint with three rotary and three translatory degrees of freedom. It has a capsule and several associated ligaments and bursae. The articulation is composed of the large head of the humerus and the smaller glenoid fossa (Fig. 7–22). Because the glenoid fossa of the scapula is the proximal segment of the glenohumeral joint, any motions of the scapula will influence glenohumeral joint function. The glenohumeral joint has sacrificed articular congruency to increase the mobility of the upper extremity and hand and is therefore susceptible to degenerative changes, instability, and derangement.

Glenohumeral Articulating Surfaces

The glenoid fossa of the scapula serves as the proximal articular surface for the glenohumeral joint. The orientation of the shallow concavity of the glenoid fossa in relation to the thorax varies with the resting position of the scapula on the thorax and with motion at the sternoclavicular and acromioclavicular joints. In addition, the orientation of the glenoid fossa may vary with the form of the scapula itself. The glenoid fossa may be tilted slightly upward or downward when the arm is at the side,[32–34] although representations most commonly show a slight upward tilt. Similarly, the fossa also does not always lie in a plane perpendicular to the plane of the scapula; it may be anteverted or retroverted up to 10°, with 6° to 7° of retroversion most typical.[33] With anteversion, the glenoid fossa faces slightly anterior in relation to the plane or body of the scapula and, with retroversion, slightly posterior. The curvature of the surface of the glenoid fossa is greater in the vertical direction than in the horizontal direction, with substantial variability between subjects.[35] The concavity of the fossa is increased by articular cartilage that is thinner in the middle and thicker on the periphery, which improves congruence with the humeral head.[36]

The humeral head is the distal articular surface of the glenohumeral joint and has an articular surface area that is larger than that of the glenoid articular surface, forming one third to one half of a sphere. In an anatomical position, the head faces medially, superiorly, and posteriorly with regard to the shaft of the humerus and the humeral condyles. The **angle of inclination** is formed by an axis through the humeral head and neck in relation to a longitudinal axis through the shaft of the humerus and is normally between 130° to 150° in the frontal plane (Fig. 7–23A). The **angle of torsion** is formed by an axis through the humeral head and neck in relation to an axis through the humeral condyles. This transverse plane angle varies but is approximately 30° posterior (Fig. 7–23B). The posterior orientation of the humeral head with regard to the humeral condyles is also called **posterior torsion, retrotorsion,** or **retroversion** of the humerus. When the arms hang dependently at the side, the two articular surfaces of the glenohumeral joint have little contact; the inferior surface of the humeral head rests on only a small inferior portion of the glenoid fossa[37,38] (see Fig. 7–22).

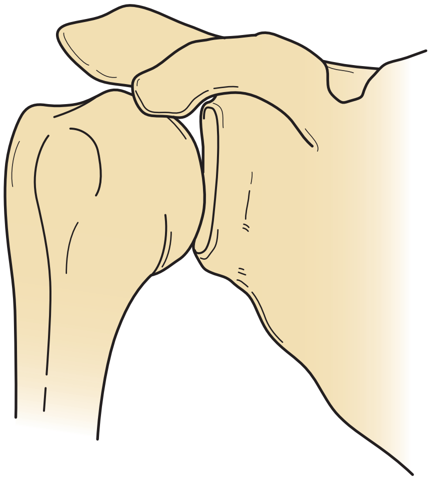


Figure 7–22 The glenohumeral joint.

Continuing Exploration 7-3:

Humeral Retroversion

Because of the internally rotated resting position of the scapula on the thorax, normal retroversion of the humeral head increases congruence of the glenohumeral joint by orienting the humeral head toward the articular surface of the glenoid fossa (Fig. 7–24). A larger humeral retroversion angle orients the humeral head more posteriorly on the glenoid surface when the humeral condyles are aligned in the coronal plane (anatomic neutral) (Fig. 7–25). This increased humeral retroversion may result in increased range of lateral rotation of the humerus and reduced range of medial rotation. Increased glenohumeral lateral rotation and decreased glenohumeral medial rotation have been demonstrated in the dominant arm of throwing athletes, and evidence suggests that increased humeral retroversion may be one contributing mechanism for this ROM adaptation.[39,40] While not as common, decreased humeral retroversion (or anteversion) may increase medial rotation range of motion and decrease lateral rotation range of motion (Fig 7–26).

Glenoid Labrum

The total available articular surface of the glenoid fossa is enhanced by the **glenoid labrum.** This accessory structure surrounds and is attached to the periphery of the glenoid fossa (Fig. 7–27), enhancing the depth or concavity of the fossa by approximately 50%.[41,42] The core of the labrum is composed of densely packed fibrous connective tissue covered by a fine superficial mesh consistent with cartilaginous tissue, with fibrocartilage at the attachment of the labrum to the periphery of the fossa.[43] The composition of the labrum allows it to perform a variety of functions, including resistance to humeral head translations, protection of the bony edges of the labrum, reduction of joint friction, and dissipation of joint contact forces.[43] The glenoid labrum serves as the attachment site for the glenohumeral ligaments and the tendon of the **long head of the biceps brachii.**[44]

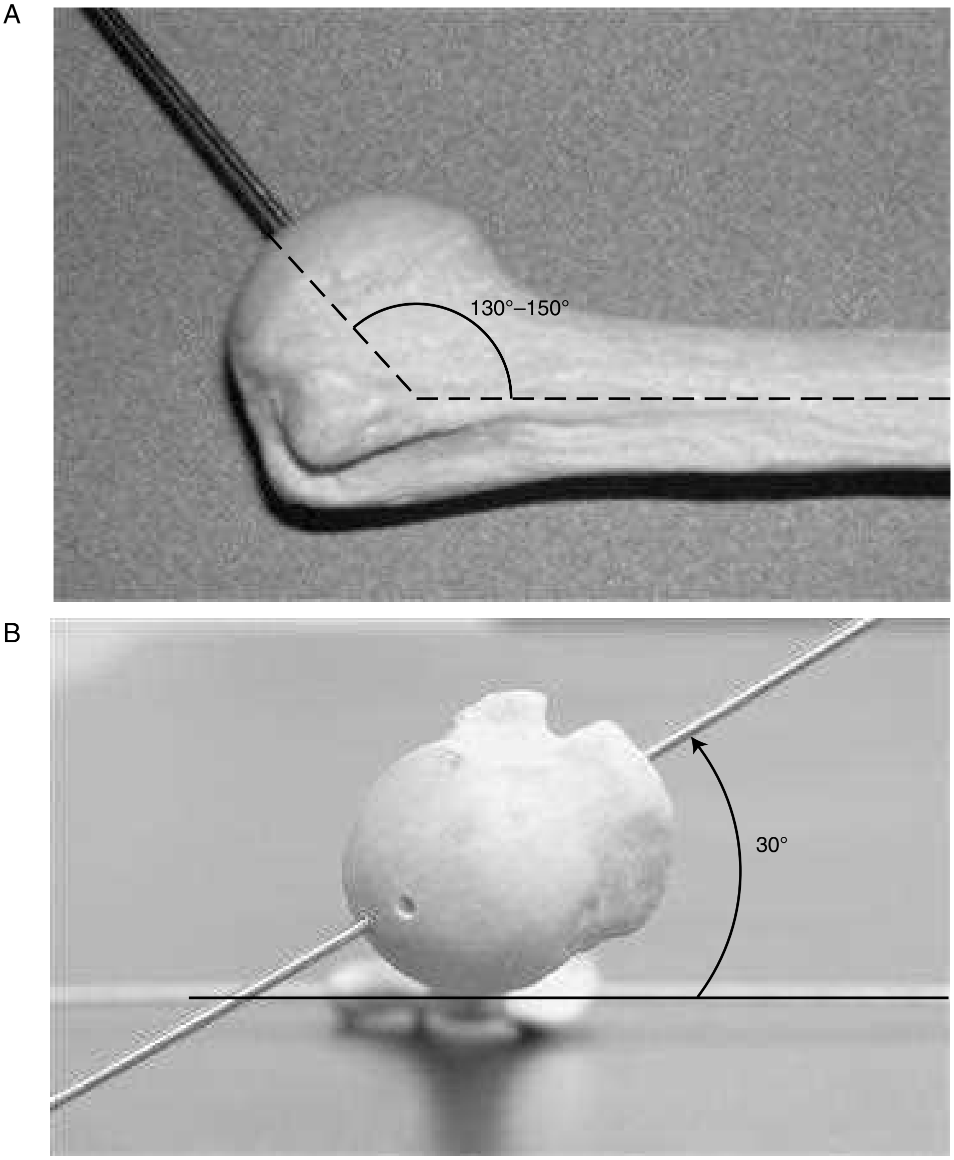


Figure 7–23 **A.** The normal angle of inclination (the angle between the humeral head and the shaft) varies between 130° and 150°. **B.** The humeral head is normally angled posteriorly approximately 30° (angle of torsion) with regard to an axis through the humeral condyles.

Figure 7–24 The slightly retroverted angle of torsion turns and effectively centers the humeral head on the glenoid fossa of the scapula when the scapula is in its internally rotated resting position on the thorax and the humerus is in neutral rotation relative to the scapula.

Figure 7–25 Retroversion of the humerus places the humeral head posteriorly with regard to the glenoid fossa of the scapula when the scapula is in its internally rotated resting position on the thorax and the humerus is in neutral rotation relative to the scapula.

Figure 7–26 Reduced retroversion or anteversion of the humerus places the humeral head anteriorly with regard to the glenoid fossa of the scapula when the scapula is in its internally rotated resting position on the thorax and the humerus is in neutral rotation relative to the scapula.

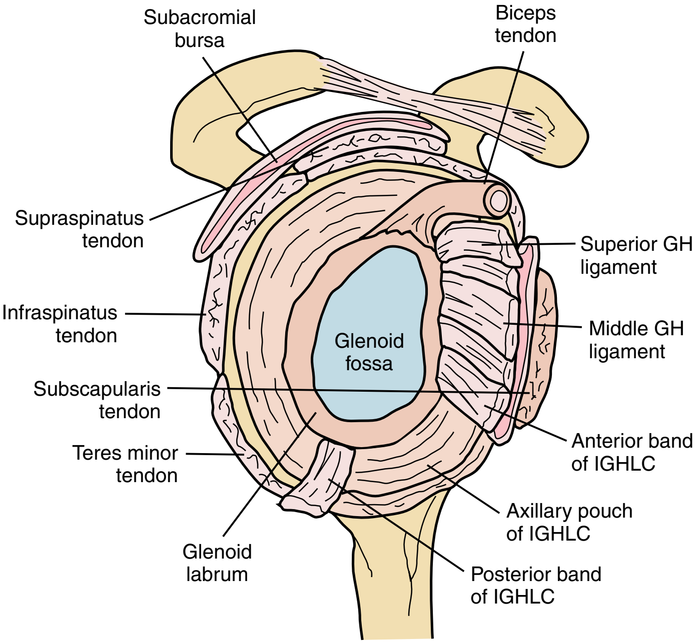


Figure 7–27 In a direct view into the glenoid fossa (humerus removed), it can be seen that the glenoid labrum increases the articular area of the glenoid fossa and serves as the attachment for the glenohumeral (GH) capsule and capsular ligaments. IGHLC, inferior glenohumeral ligament complex.

Glenohumeral Capsule and Ligaments

The glenohumeral joint is surrounded by a large, loose capsule that is taut superiorly and slack anteriorly and inferiorly with the arm dependent at the side (Fig. 7–28). The capsule tightens when the humerus is abducted and laterally rotated, making this the close-packed position for the glenohumeral joint.[45] The capsular surface area is twice that of the humeral head, and more than 2.5 cm of distraction of the head from the glenoid fossa is possible in the loose-packed position.[6] The relative laxity of the glenohumeral capsule is necessary for the large excursions of the joint but provides little stability without the reinforcement of ligaments and muscles. The capsule is reinforced by the **superior, middle,** and **inferior glenohumeral ligaments** and by the **coracohumeral ligament** (Fig. 7–29). The superior, middle, and inferior glenohumeral ligaments are thickened regions within the capsule tissue itself. In addition to these static capsular reinforcements, the **rotator cuff muscles** and their

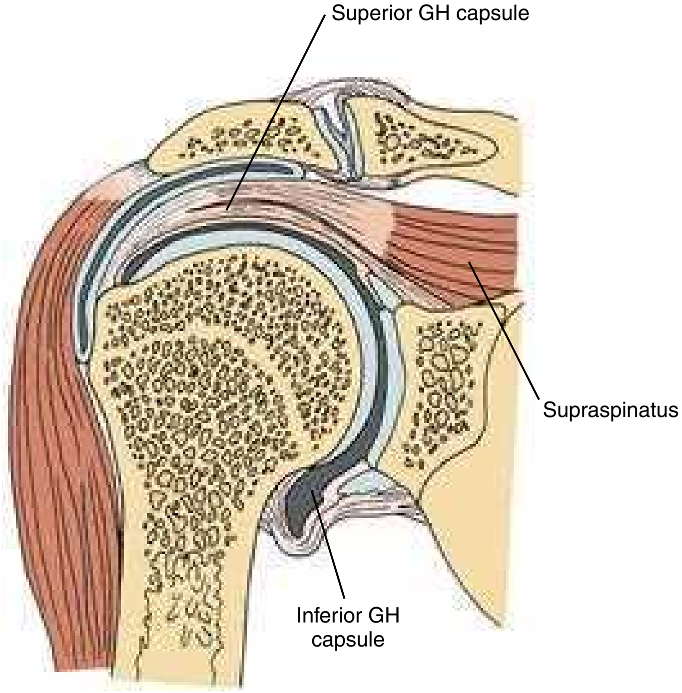


Figure 7–28 When the arm is at rest at the side, the superior capsule is taut while the inferior capsule is slack. GH, glenohumeral.

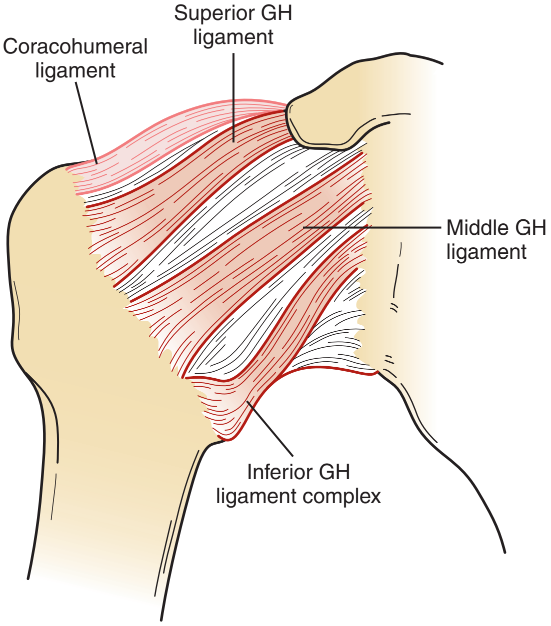


Figure 7–29 The ligamentous reinforcements to the glenohumeral (GH) capsule: the superior, middle, and inferior glenohumeral ligaments, along with the coracohumeral ligament.

tendons provide dynamic reinforcement to the capsule through their anatomical proximity to the joint and because the tendons insert directly onto and blend into the glenohumeral capsule. Even with these reinforcements, the glenohumeral joint is vulnerable to dislocations, particularly in the anterior direction. The three capsular glenohumeral ligaments (superior, middle, and inferior) vary considerably in size, extent, and attachment sites between individuals.[46] Figure 7–27 shows the three ligaments as they appear on the interior surface of the joint capsule.

The superior glenohumeral ligament passes from the superior glenoid labrum to the upper neck of the humerus deep to the extracapsular coracohumeral ligament. Harryman and colleagues described the superior glenohumeral ligament, the superior capsule, and the coracohumeral ligament as interconnected structures that bridge the space between the **supraspinatus** and **subscapularis** muscle tendons and form the **rotator interval capsule (RIC)**[47] (Fig. 7–30). The middle glenohumeral ligament runs obliquely from the superior anterior labrum to the anterior aspect of the proximal humerus below the superior glenohumeral ligament attachment (see Figs. 7–27 and 7–29). Ide and colleagues found this ligament to be absent in up to 30% of subjects and also described it as cord-like in nearly 20% of subjects.[46] The inferior glenohumeral ligament is described as having three components and thus has been termed the **inferior GH ligament complex (IGHLC)**.[48] The three components of the complex are the anterior and posterior ligament bands and the axillary pouch in between (see Fig. 7–27). The IGHLC shows position-dependent variability in function,[49] as well as variations in viscoelastic behavior.[42]

Study of the movement restraint provided by the glenohumeral ligaments indicates that each ligament contributes differently to glenohumeral stability. The superior glenohumeral ligament and its associated rotator interval capsule structures contribute most to anterior and inferior joint stability by limiting anterior and inferior translations of the humeral head when the arm is at the side (0° abduction) (Fig. 7–31A). The middle glenohumeral ligament contributes primarily to anterior joint stability by limiting anterior humeral translation with the arm at the side and up to 60° of abduction (Fig. 7–31B).[50] With abduction beyond 45° or with combined abduction and rotation (Fig. 7–31C), the IGHLC plays the major role of joint stabiliztion.[42,48,49,51,52] With abduction, the axillary (inferior

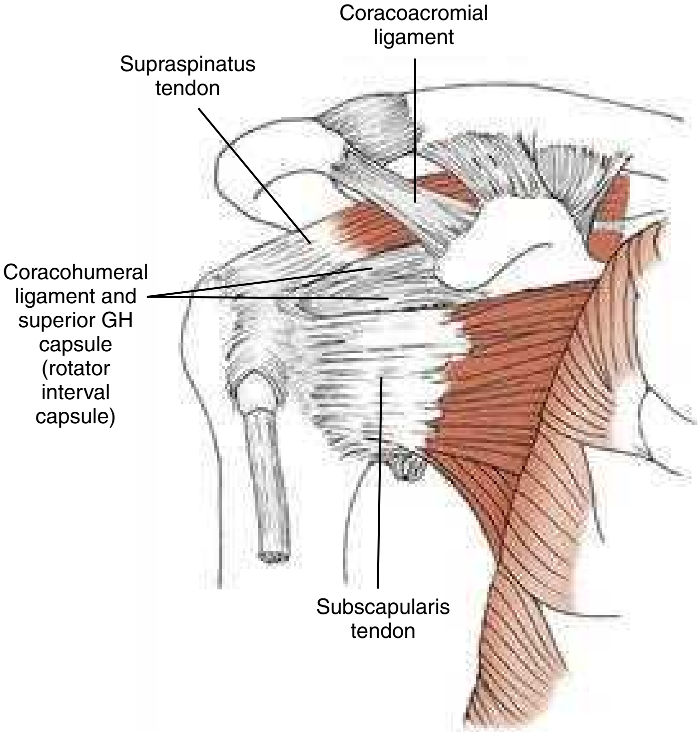


Figure 7–30 The rotator interval capsule is made up of the superior glenohumeral (GH) capsule, superior glenohumeral ligament, and coracohumeral ligament. Together, these structures bridge the gap between the supraspinatus and subscapularis muscle tendons.

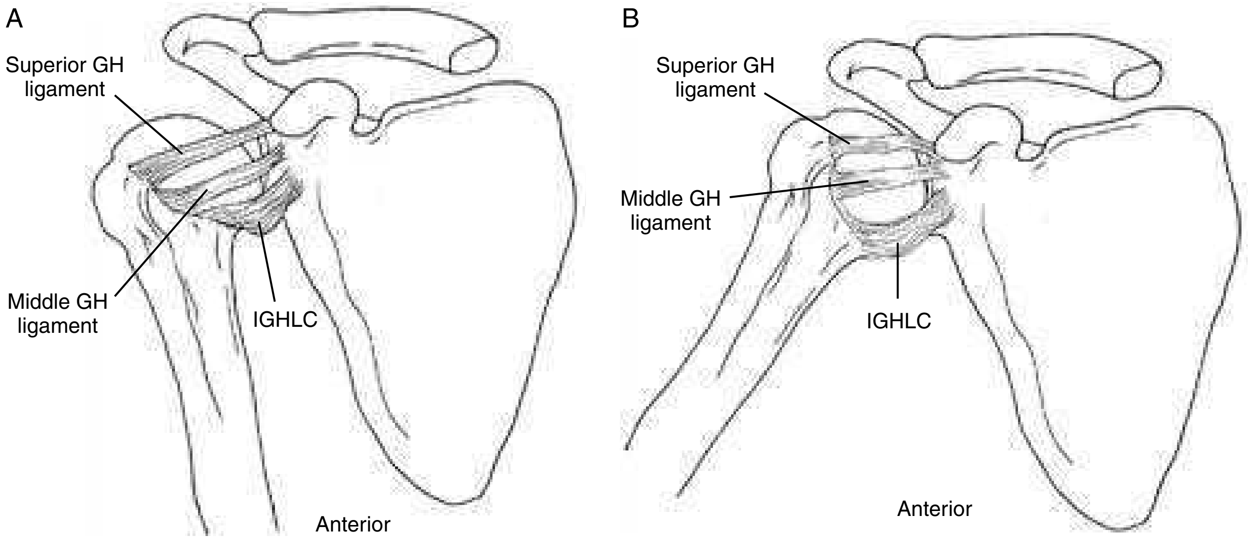


Figure 7–31 The glenohumeral (H) ligaments at rest **(A),** at 45° humeral abduction and neutral rotation **(B),** at 90° humeral abduction and neutral rotation **(C),** at 90° humeral abduction and external rotation **(D),** and at 90° humeral abduction and medial rotation **(E)**. IGHLC, inferior glenohumeral ligament complex.

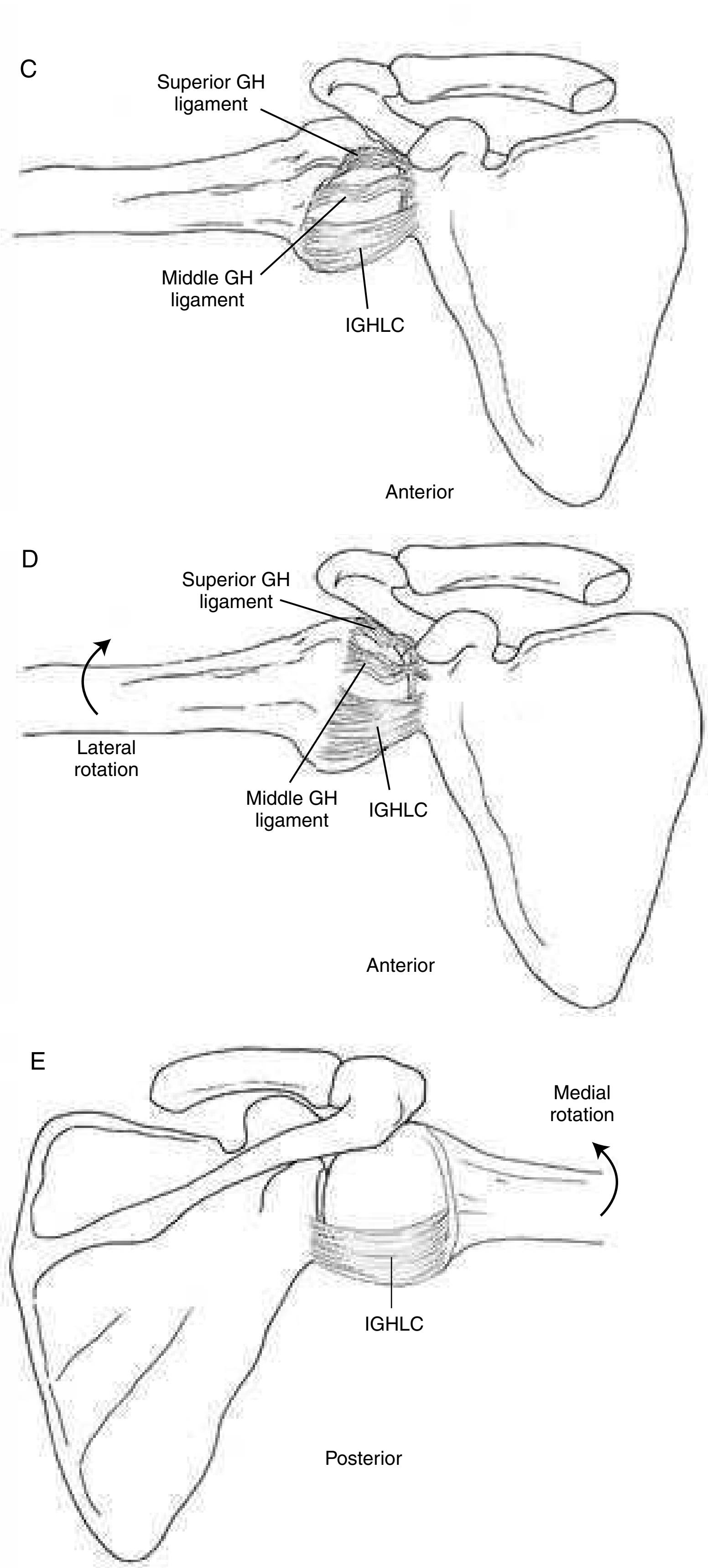


Figure 7–31—cont'd

capsule) slack is taken up, and the IGHLC resists inferior humeral head translation. If humeral lateral rotation is added (Fig. 7–31D), the anterior band of the IGHLC fans out to provide anterior joint stability and resistance to anterior humeral head translation. If humeral medial rotation is added (Fig. 7–31E), the posterior band of the IGHLC fans out and provides posterior joint stability and resistance to posterior humeral head translation.[53] In all positions of humeral abduction, lateral or medial rotation of the humerus tightens the capsule and glenohumeral ligaments and increases glenohumeral joint stabilization.[54]

The coracohumeral ligament (see Fig. 7–29 and 7–30) originates from the base of the coracoid process and has two bands. The first band inserts into the edge of the supraspinatus and onto the greater tubercle, joining the superior glenohumeral ligament. The second band inserts into the subscapularis and lesser tubercle.[47,55] The two bands form a tunnel through which the tendon of the long head of the biceps brachii passes.[56] As part of the rotator interval capsule, the coracohumeral ligament limits inferior translation of the humeral head in the dependent arm position. In addition, the coracohumeral ligament resists humeral lateral rotation with the arm adducted. The ligament may also assist in preventing superior translation of the humerus, especially when the dynamic stabilizing force of the rotator cuff muscles is impaired.[55]

Coracoacromial Arch

The **coracoacromial** (or **suprahumeral**) **arch** is formed by the coracoid process, the acromion, the **coracoacromial ligament,** and the inferior surface of the acromioclavicular joint (Fig. 7–32). The coracoacromial arch forms an osteoligamentous vault over the humeral head and the region between the arch and the humeral head is called the *subacromial space.* The **subacromial bursa,** the rotator cuff tendons, and a portion of the tendon of the long head of the biceps brachii lie within the subacromial space and are protected superiorly from direct trauma by the coracoacromial arch. Such trauma could occur through simple daily tasks such as carrying a heavy bag over the shoulder. The arch also acts as a physical barrier to superior translatory forces acting on the humeral head, preventing it from dislocating superiorly. Although beneficial to joint stability, contact of the humeral head with the undersurface of the arch can simultaneously cause painful impingement or mechanical abrasion of the structures within the subacromial space. The supraspinatus tendon is particularly vulnerable because of its location beneath all of the potentially impinging structures except the coracoid process (see Fig. 7–32).

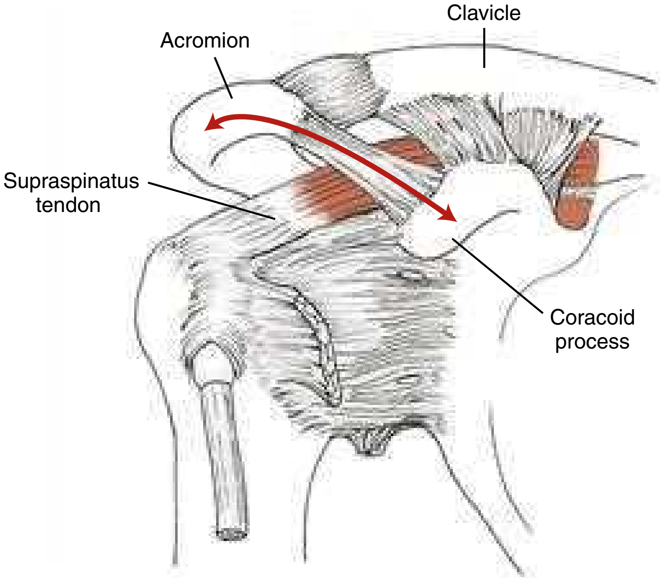


Figure 7–32 The coracoacromial arch is formed by the coracoid process anteriorly, the acromion posteriorly, and the coracoacromial ligament superiorly. Together, these structures form an osteoligamentous arch over the humeral head.

The subacromial space, also referred to as the **suprahumeral space** or **supraspinatus outlet,** has been quantified by measuring the superior-to-inferior **acromiohumeral interval** on radiographs. This interval averages 10 mm in healthy subjects with the arm adducted at the side and decreases to about 5 mm during elevation of the arm.[57,58] As the acromiohumeral interval decreases, it must accommodate the soft tissue structures within it, as well as the articular cartilage and the capsuloligamentous structures. For this reason, Flatow and colleagues suggested that even during normal motion into humeral elevation, there is some contact of the rotator cuff with the coracoacromial arch.[58] However, recent 3-D imaging has determined that anatomical approximation of the supraspinatus tendon with the acromion occurs at humeral elevation angles below 60°, while the smallest acromiohumeral interval is between the acromion and the greater tuberosity of the humerus at approximately 90° of elevation.[59] This anatomical relationship suggests that normal decreases in the acromiohumeral distance during arm elevation may not impact the rotator cuff at elevation angles higher than 60°.

When the subacromial space decreases even more than what has been measured in healthy subjects, the likelihood of impingement of the rotator cuff tendons and **subacromial bursa** during elevation of the arm increases. The space can decrease by anatomical factors such as changes in the shape or slope of the acromion, acromial bone spurs, acromioclavicular joint osteophytes, a large coracoacromial ligament, or a disproportionately large humeral head.[60,61] Abnormal scapular or humeral motions can also functionally reduce the size of the subacromial space. Inadequate posterior tilting or inadequate upward rotation of the scapula during arm elevation or excessive superior or anterior translation of the humeral head on the glenoid fossa are believed to bring the humeral head in closer proximity to the acromion, increasing the risk of impingement.[28,62,63] Finally, repetitive impingement may lead to inflammation, fibrosis, and thickening of the soft tissues, further reducing the subacromial space during arm elevation. Abnormal anatomic factors and motion abnormalities have been identified in persons with impingement,[28,61–63] as has a decrease in the subacromial space during arm elevation.[64]

CASE APPLICATION

Symptoms of Impingement

case 7–2

Ms. Sorenson reports shoulder pain localized at the proximal lateral humerus. This localization of pain is consistent with pain originating from the rotator cuff tendons, the long head of the biceps tendon, or the subacromial bursa. Her pain may be related to a rotator cuff or biceps tendonopathy and possible shoulder impingement. Repetitive impingement can create tendonopathy and progress to partial- and full-thickness rotator cuff tears.[61] In addition, she reports pain when sleeping on the right shoulder. This is a common complaint of persons with pain originating from the subacromial space. Additional compression of the humeral head into the subacromial space is experienced while lying on the affected side.

Bursae

Several bursae are associated with the shoulder complex in general and with the glenohumeral joint specifically, reflecting the presence of frictional forces between anatomical structures. Although all bursae at the shoulder contribute to function, the most important are the subacromial and **subdeltoid bursae** (see Fig. 7–28). These bursae separate the supraspinatus tendon and head of the humerus from the acromion, coracoid process, coracoacromial ligament, and deltoid muscle. These bursae may be separate but are commonly continuous with each other and are collectively known as the *subacromial bursa.* The subacromial bursa reduces friction to permit smooth gliding between the humerus and supraspinatus tendon and the surrounding structures. Interruption or failure of this gliding mechanism is a common cause of pain and decreased glenohumeral motion, although it rarely occurs as a primary problem. Subacromial bursitis is most commonly secondary to inflammation or degeneration of the supraspinatus tendon.[16] In the absence of inflammation, bursae are merely layers of synovial tissue in contact with each other with a very thin layer of fluid between. However, when inflamed, the subacromial bursa may decrease the subacromial space available for rotator cuff tendon clearance.

Glenohumeral Motions

The glenohumeral joint is usually described as having three rotational degrees of freedom: **flexion/extension, abduction/adduction,** and **medial/lateral rotation.** The range of each of these motions occurring solely at the glenohumeral joint varies considerably. Flexion and extension occur about a coronal axis passing through the center of the humeral head. The glenohumeral joint is often considered to have 120° of flexion and about 50° of extension.[65] However, more recent work measuring three-dimensional glenohumeral motion reported peak humeral flexion of only 97° in relation to the scapula in a small sample of subjects averaging 50 years of age.[66] This lesser value of glenohumeral motion is also supported by more recent work in young healthy subjects.[14] The higher values classically attributed to glenohumeral flexion may not have fully isolated glenohumeral motion from trunk and scapular motion.

Medial and lateral rotation occur about a long axis parallel to the shaft of the humerus and passing though the center of the humeral head. The range of medial/lateral rotation of the humerus varies with position. With the arm at the side, medial and lateral rotation may be limited. Abducting the humerus to 90° frees the arc of rotation, with glenohumeral values of 130° or greater.[65] The restricted arc of medial/lateral rotation when the arm is at the side may be related to different alignment of the greater and lesser tubercles, which creates a mechanical block, or to different areas of capsular or muscular tightness when the arm is adducted versus abducted.

Abduction/adduction of the glenohumeral joint occur around an A-P axis passing through the humeral head center. The maximum range of abduction at the glenohumeral joint is the topic of much disagreement. There is general consensus, however, that the range of abduction of the humerus in the frontal plane (whether the abduction is active or passive) will be diminished if the humerus is maintained in neutral or medial rotation. The restriction to abduction in medial or neutral rotation is commonly attributed to impingement of the greater tubercle on the coracoacromial arch. When the humerus is laterally rotated 35° to 40°,[67,68] the greater tubercle will pass under or behind the arch so that abduction can continue.

The ROMs for abduction of the glenohumeral joint (if impact of the greater tubercle is avoided) are reported to be anywhere from 90° to 120°.[1,16,66,69,70] Inman and coworkers[11] found active abduction to be limited to 90° when the scapula does not participate in the motion but claim that 120° of motion was available passively. Further increasing the variability between investigations, some studies examined the range of abduction in the traditional frontal plane, while others have investigated elevation in the plane of the scapula (30° to 45° anterior to the frontal plane). The available passive range for abduction in the scapular plane may be slightly greater than for abduction in the frontal plane.[68] When the humerus is elevated in the plane of the scapula (referred to as *abduction in the plane of the scapula*, *scapular abduction*, or **scaption** in clinical jargon), there is presumably less restriction to motion because the capsule is less twisted than when the humerus is brought further back into the frontal plane. An,[67] however, found maximum elevation not in the plane of the scapula but 10° to 37° anterior to that plane. Although it has been proposed that abduction in the scapular plane does not require concomitant lateral rotation to achieve maximal range, this premise has also been disputed.[67,68] During active arm elevations in all planes of elevation, glenohumeral lateral rotation has been demonstrated.[14]

Intra-articular Contribution to Glenohumeral Motions

Full ROM of the glenohumeral joint is, to a reasonable degree, a function of the intra-articular movement of the incongruent articular surfaces. The convex humeral head is a substantially larger surface and may have a different radius of curvature than the shallow concave fossa. Given this incongruence, rotations of the joint around its three axes do not occur as pure spin but have changing centers of rotation and shifting contact patterns within the joint. There is a somewhat surprising lack of consensus on the extent and direction of movement of the humeral head on the fossa.[42] However, elevation of the humerus requires that the articular surface of the humeral head slide inferiorly (caudally) in a direction opposite to movement of the shaft of the humerus. Failure of the humeral articular surface to slide downward in abduction of the humerus would cause superior (cephalad) rolling of the humeral head surface on the fossa. The large humeral head would soon run out of glenoid surface, and the head of the humerus would impinge upon the overhanging coracoacromial arch (Fig. 7–33A). If, as it should, the articular surface of the head of the humerus slides inferiorly while the head rolls up the fossa, full ROM can be achieved (Fig. 7–33B).

There is consensus that inferior sliding of the humeral head's articular surface is necessary to minimize upward rolling of the humeral head. However, it appears that the humeral head as a whole (its center of rotation) still moves somewhat superiorly (translates upwardly) on the glenoid fossa in spite of the downward sliding (Fig. 7–34), although the magnitude of reported upward shift differs among investigators.[42,62,68,71,72] The humeral articular surface may also slide anteriorly or posteriorly and medially or laterally on the glenoid fossa. The humeral head's center is believed to move slightly superior (1–2 mm of translation) until about 60° of active elevation motion.[34,62,71,72] With further elevation, the humeral head's center is believed to remain relatively stable and centered on the glenoid fossa. Less agreement exists regarding the anterior and posterior translations of the humeral head's center. Slight anterior positioning and translation (1–2 mm) has been reported during active elevation.[71] Other investigators have reported slight posterior translations early in the range or slight anterior translations early in the range and posterior translations later in the range of active elevation.[62,72,73] All studies of active translations show smaller magnitudes of motion (less than 5 mm) during active motions than is available in a passive laxity examination, in which translations up to 20 mm have been reported.[74] These data support the premise that rotator cuff forces help to stabilize and center the humeral head on the glenoid fossa. Most investigators also agree that many variables determine the patterns of movement of the humeral head on the glenoid fossa, including articular geometry, capsuloligamentous influences, influences of arm position, and muscle forces.

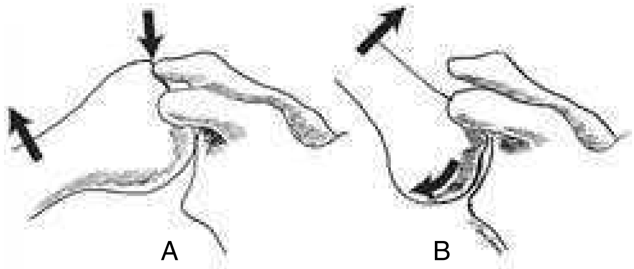


Figure 7–33 **A.** Without downward sliding of the articular surface of the humeral head, the humeral head will roll up the glenoid fossa and impinge upon the coracoacromial arch. **B.** With downward sliding of the humeral head's articular surface as the humeral abducts, a full range of motion can occur.

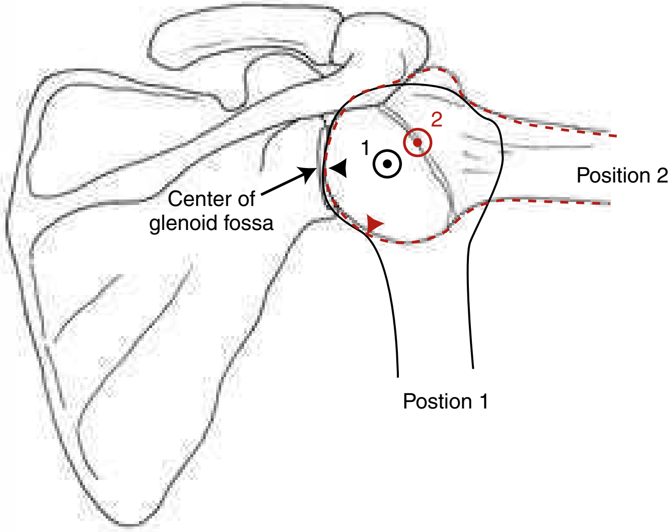


Figure 7–34 Slight superior translation of the center of the humeral head can still occur during humeral abduction despite inferior sliding of the head's articular surface.

Much of the confusion surrounding reported motions of the humeral head on the glenoid fossa may be attributed to the differences in the point on the humerus that is being followed (combinations of rolling and sliding vs. translations), as well as to the small magnitudes of these motions and the limitations of currently available measurement systems.

Continuing Exploration 7-4:

The Role of the Capsule

The glenohumeral capsule and its associated glenohumeral ligaments provide stability to the glenohumeral joint by limiting anterior, inferior, or posterior humeral head translation on the glenoid fossa. The stabilizing function of these structures is minimal at less than 90° of humeral elevation when only the superior segment of the capsule is under any significant tension. At these lower elevation angles, the rotator cuff muscles and tendons actively stabilize the glenohumeral joint. Not only does their orientation across the joint provide stability, but because their tendons insert into the capsule, rotator cuff muscle contractions tense the capsule and ligaments to increase stability. Toward the end range of humeral motions, however, the capsule becomes tight passively, and this tension restricts glenohumeral translation. At end ranges, this tension begins to produce rather than restrict humeral head center translations.[75] With asymmetrical tightening of the capsule, this obligate translation occurs in a direction away from the tight and toward the loose capsular tissue.[75] For example, with increasing medial rotation, the posterior capsule becomes tight and produces anterior translation of the humeral head center that is not restricted by the relatively slack anterior capsule. A tight posterior capsule is, therefore, one potential mechanism for shoulder impingement, inasmuch as it may produce increased anterior humeral head translation and minimize the subacromial space.[62]

Static Stabilization of the Glenohumeral Joint in the Dependent Arm

Given the incongruence of the glenohumeral articular surfaces, bony geometry alone cannot maintain joint stability with the arm relaxed at the side, requiring the contribution of other mechanisms. With the humeral head resting on the fossa, gravity imparts a caudally directed translatory force on the humerus. To maintain equilibrium, a cranially directed force is needed and could be supplied by active contraction or passive tension of muscles such as the deltoid, supraspinatus, or the long heads of the biceps brachii and **triceps brachii.** However, Basmajian and Bazant[32] and MacConaill and Basmajian[45] reported that all muscles of the shoulder complex are electrically silent in the relaxed, unloaded limb, even when the limb is tugged vigorously downward. The mechanism of joint stabilization, therefore, appears to be passive, and the structures of the rotator interval capsule (superior capsule, superior glenohumeral ligament, and coracohumeral ligament) that are taut when the arm is at the side possess both the magnitude and orientation for this function.[47,49,51] The resultant vector formed from the gravity and rotator interval capsule vectors creates a force that compresses the humeral head into the lower portion of the glenoid fossa and prevents inferior humeral head translation (Fig. 7–35).

In addition to passive tension from the rotator interval capsule, two other mechanisms help provide static stability to the glenohumeral joint with the arm at the side. In a healthy glenohumeral joint, the capsule is airtight and there is negative intra-articular pressure. This negative pressure creates a relative vacuum that resists the inferior humeral translation caused by the force of gravity.[49] Loss of intra-articular pressure, produced by venting the capsule or tears in the glenoid labrum, results in large increases in inferior humeral translations.[76,77] The degree of glenoid inclination also influences glenohumeral joint stability with the arm in the dependent position.[52] A slight upward tilt of the glenoid fossa, either anatomically in the structure of the scapula or through scapular upward rotation, will produce a partial bony block against humeral inferior translation.

When these passive forces are inadequate for glenohumeral joint stabilization, as may occur in the heavily loaded arm, the supraspinatus is recruited to provide active assistance.[32] This is not surprising, given that the supraspinatus tendon has attachments to the rotator interval capsule.[55] In fact, the role of the supraspinatus may be more critical than its activity as measured by electromyography (EMG) indicates because paralysis or dysfunction in the supraspinatus may lead to gradual inferior subluxation of the glenohumeral joint. Without the reinforcing passive tension of the intact supraspinatus muscle, the sustained load on the structures of the rotator interval capsule apparently causes these structures to gradually stretch (become plastic), which results in a loss of joint stability. Inferior glenohumeral subluxation is commonly encountered in patients with diminished rotator cuff function caused by stroke or other brain injury.

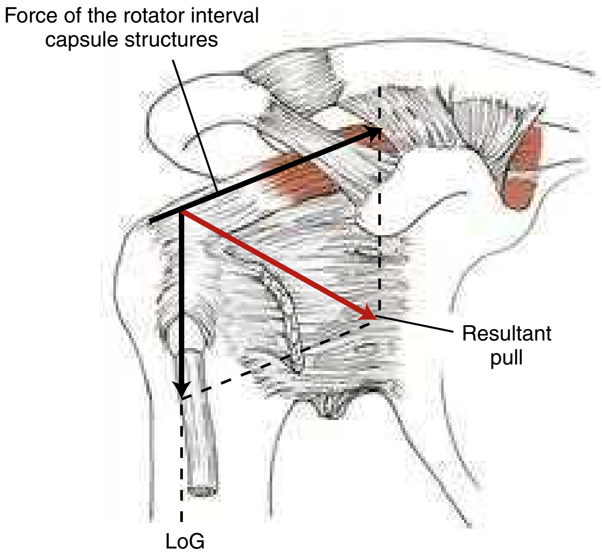


Figure 7–35 Mechanism for stabilization of the dependent arm. With the arm relaxed at the side, the downward pull of gravity on the arm (vector extended from the center of gravity of the upper extremity) is opposed by the passive tension in the rotator interval capsule. The resultant of these forces stabilizes the humeral head on the glenoid fossa.

Dynamic Stabilization of the Glenohumeral Joint

The Deltoid and Glenohumeral Stabilization

It is generally accepted that the deltoid muscle is a prime mover (along with the supraspinatus) for glenohumeral abduction. The anterior deltoid is also considered the prime mover in glenohumeral flexion. Both abduction and flexion are elevation activities with many biomechanical similarities. The segment or segments of the deltoid that participate in elevation will vary with role and function.[78,79] However, examination of the resultant line of action of the deltoid muscle in abduction can be used to highlight the stabilization needs of the glenohumeral joint in elevation activities. Figure 7–36 shows the line of action of the deltoid muscle with the arm at the side. The force vectors of the three segments of the deltoid acting together coincide with the fibers of the middle deltoid. When the muscle force vector (F_D) is resolved into its parallel (Fx) and perpendicular (Fy) components in relation to the long axis of the humerus, the parallel component directly cephalad (superiorly) is by far the larger of the two components. That is, the majority of the force of contraction of the deltoid from this initial position causes the humerus and humeral head to translate superiorly;

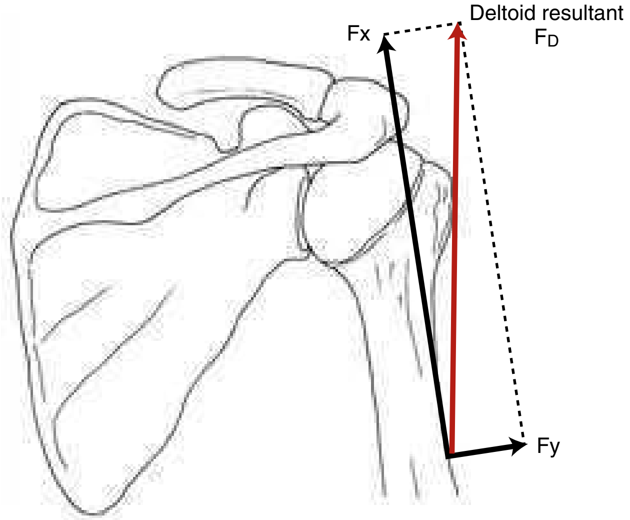


Figure 7–36 The action line of all three segments of the deltoid follows the line of pull of the middle deltoid. The resultant (F_D) resolves into a very large translatory component (Fx) and a small rotary component (Fy) so that an isolated contraction of the deltoid would cause the deltoid to produce more superior translation than rotation of the humerus.

only a small proportion of force is applied perpendicular to the humerus and directly contributes to rotation (abduction) of the humerus with a large moment arm.

At many other joints, a force component parallel to the long bone has a stabilizing effect because the parallel component contributes to joint compression. However, when the arm is at the side, the glenoid fossa is not in line with the shaft of the humerus. Consequently, the force (Fx) applied parallel to the long axis of the bone in this position creates a shear force (approximately parallel to the contacting articular surfaces) rather than a stabilizing (compressive) effect. The large superiorly directed force of the deltoid, if unopposed, would cause the humeral head to impact the coracoacromial arch before much abduction had occurred. The rotary torque produced by the relatively small perpendicular component of the deltoid (Fy) will not be particularly effective until the translatory forces are in equilibrium. If the humeral head migrated upward into the coracoacromial arch, the inferiorly directed contact force of the arch would offset the Fx component of the deltoid, theoretically permitting rotation of the humeral head to continue. However, pain from impinged structures in the subacromial space is likely to prevent much motion. The inferior pull of gravity cannot offset the Fx component of the deltoid, because the resultant force of the deltoid must exceed that of gravity before any rotation can occur. As a result, the deltoid cannot independently abduct (elevate) the arm. Another force or set of forces must be introduced to work synergistically with the deltoid for the deltoid to work effectively to produce the desired rotation. This is the role of the rotator **(musculotendinous)** cuff.

The Rotator Cuff and Glenohumeral Stabilization

The supraspinatus, **infraspinatus, teres minor,** and subscapularis muscles and tendons compose the rotator cuff (also referred to by the acronym **SITS muscles**). These muscles are considered to be part of a "cuff" because the inserting tendons of each muscle of the cuff blend with and reinforce the glenohumeral capsule. Also, all have lines of action that significantly contribute to the dynamic stabilization of the glenohumeral joint. The resultant force vectors of the four segments of the rotator cuff (the superiorly located supraspinatus, posteriorly located infraspinatus and teres minor, and the more anteriorly located subscapularis muscles) are shown in Figure 7–37. If any one (or all three) of the vector pulls of the infraspinatus, teres minor, or subscapularis muscles is resolved into its components (Fig. 7–38), it can be seen that the

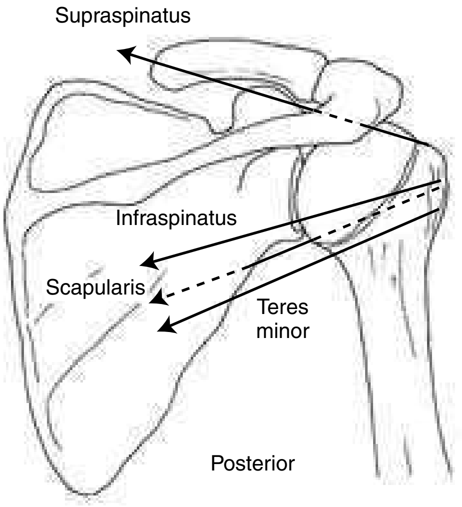


Figure 7–37 The action lines of the four segments of the rotator cuff: the supraspinatus, infraspinatus, teres minor, and subscapularis muscles.

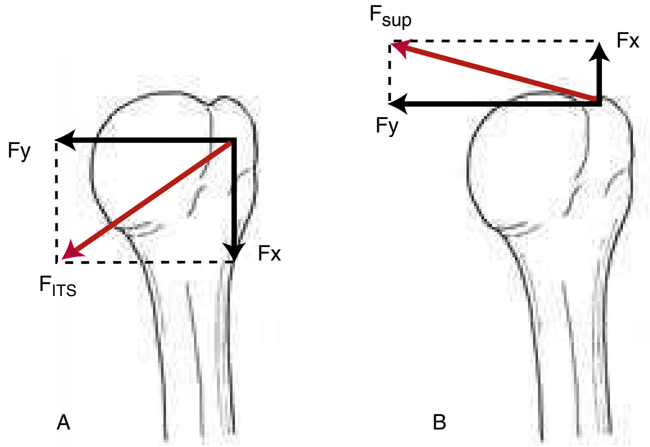


Figure 7–38 **A.** The infraspinatus, teres minor, and subscapularis muscles individually and together (ITS) have a similar line of pull. The perpendicular component (Fy) compresses as well as rotates, and the parallel component (Fx) helps offset the superior translatory pull of the deltoid. **B.** The supraspinatus (sup) has a superiorly directed parallel translatory component (Fx) and a perpendicular component (Fy) that is more compressive than that of the other rotator cuff muscles and can independently abduct the humerus.

perpendicular force component (Fy) not only tends to cause at least some rotation of the humerus, given its orientation to the long axis of the bone, but it also compresses the head into the glenoid fossa. This is due to the fact that the articular surface of the humerus lies nearly perpendicular to the shaft and provides a clear illustration of how a rotary component may do more than "rotate" a bone around a joint axis.

Although the infraspinatus, teres minor, and subscapularis muscles of the rotator cuff are important glenohumeral joint compressors, equally (or perhaps more) critical to the stabilizing function of these particular muscles is the inferior (caudal) translatory pull (Fx) of the muscles. The sum of the three negative (inferior) translatory components of these three muscles of the rotator cuff nearly offsets the superior translatory force of the deltoid muscle. Sharkey and Marder[80] showed that abduction without the infraspinatus, teres minor, and subscapularis muscles resulted in substantial superiorly directed shifts in humeral position in cadaver models.

The teres minor and infraspinatus muscles, in addition to their stabilizing role, contribute to abduction of the arm by providing the lateral rotation that typically occurs with elevation of the humerus to help clear the greater tubercle from beneath the acromion. Although the weak adduction force of the teres minor muscle and the medial rotary force of the subscapularis muscle appear to contradict their role in elevation of the arm, Otis and colleagues[81] found the effectiveness of these muscles in their contradictory functions to be diminished during abduction of the arm. That is, the infraspinatus and subscapularis muscles add to the abduction torque, whereas the teres minor muscle adds to the lateral rotary torque. The medial and lateral rotary forces also help center the humeral head in an anterior/posterior direction, with increased anterior and posterior displacements evident when rotator cuff forces are reduced.[82] Saha[35] referred to these cuff muscles as "steerers."

The action of the deltoid and the combined actions of the infraspinatus, teres minor, and subscapularis muscles approximate a force couple. The nearly equal and opposite superior/inferior forces for the deltoid and these three rotator cuff muscles acting on the humerus approximate an *almost* perfect rotation of the humeral head around a relatively stable axis of rotation with minimal translation.

The Supraspinatus and Glenohumeral Stabilization

Although the supraspinatus muscle is part of the rotator cuff, the line of action of the supraspinatus muscle, unlike the action lines of the other three rotator cuff muscles, has a superior (cephalad) translatory component, rather than the inferior (caudal) component found in the other muscles of the cuff (see Fig. 7–38B). Given its line of pull, the supraspinatus is not able to offset the upward dislocating action of the deltoid.[83] The supraspinatus is still an effective stabilizer of the glenohumeral joint, however. Because of the more superior location of the supraspinatus results in a line of action that lies farther from the glenohumeral joint axis than the action lines of the other rotator cuff muscles, the larger supraspinatus moment arm is capable of independently producing a full or nearly full range of glenohumeral joint abduction while simultaneously stabilizing the joint.[45] Gravity acts as a stabilizing synergist to the supraspinatus by offsetting the small upward translatory pull of the muscle.

Continuing Exploration 7-5:

The Supraspinatus as an Independent Abductor

The supraspinatus can, at least theoretically, independently produce abduction of the arm through most or all of its range, whereas the deltoid cannot. The resultant of the force vectors of a supraspinatus contraction and gravity is essentially a vector identical to the resultant vector that we saw in Figure 7–35. The line of action of the supraspinatus is the same as that of the rotator interval capsule with which it blends (and to which it contributes passive tension at rest). With a concentric contraction of the supraspinatus, the proportionally small superiorly directly translatory force is offset by the inferiorly directed force of gravity, which results in translatory equilibrium but effective rotation. The resultant of the gravitational and supraspinatus force vectors contributes to an inferior gliding of the humeral head surface during abduction of the arm, allowing full articulation of the joint surfaces and preventing abnormal superior displacement. The supraspinatus can also contribute small amounts of either medial or lateral rotation torque, depending on the position of the arm, although its moment arm for long axis rotation is very small.[84]

The Long Head of the Biceps Brachii and Glenohumeral Stabilization

The long head of the biceps brachii runs superiorly from the anterior shaft of the humerus through the bicipital groove between the greater and lesser tubercles to attach to the supraglenoid tubercle and superior labrum. It enters the glenohumeral joint capsule through an opening between the supraspinatus and subscapularis muscles, where it penetrates the capsule but not the synovium (Fig. 7–39). Within the bicipital groove, the biceps tendon is enveloped by a tendon sheath and tethered there by the **transverse humeral ligament** that runs between the greater and lesser tubercles. The long head of the biceps brachii, because of its position at the superior capsule and its connections to structures of the rotator interval capsule,[55] is sometimes considered to be part of the reinforcing cuff of the glenohumeral joint. The biceps muscle is capable of contributing to the force of flexion and can, if the humerus is laterally rotated, contribute to the force of abduction.[11] Although elbow and shoulder position may influence its function, the long head appears to contribute to glenohumeral stabilization by centering the head in the fossa and by reducing vertical (superior and inferior) and anterior translations.[85–88] Pagnani and colleagues hypothesized that the long head may produce its effect

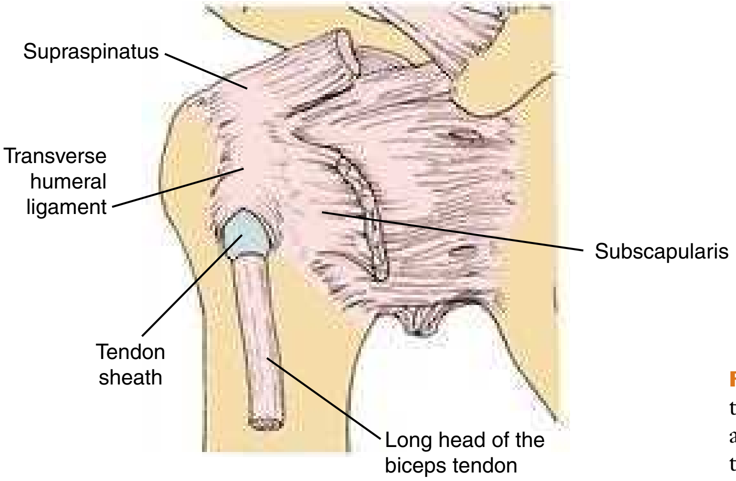


Figure 7–39 The long head of the biceps brachii passes through a fibro-osseous tunnel formed by the bicipital groove and the transverse humeral ligament. It is protected within the tunnel by a tendon sheath.

by tightening the relatively loose superior labrum and transmitting increased tension to the superior and middle glenohumeral ligaments.[88] This idea follows from their observation that lesions of the anterosuperior labrum did not affect stability of the glenohumeral joint unless the attachment of the long head of the biceps brachii was also disrupted.[89] The overall contribution of the long head to glenohumeral stabilization is supported by the observation that the tendon hypertrophies with rotator cuff tears.[88]

Concept Cornerstone **7-2**

Dynamic Stabilization

Given what we know about the glenohumeral joint thus far, we can summarize that dynamic stabilization at any point in the range is a function of (1) the force of the prime mover or movers, (2) the force of gravity, (3) the force of the muscle stabilizers, (4) articular surface geometry, and (5) passive capsuloligamentous forces. Inman and coworkers[11] appropriately added the factors of (6) the force of friction and (7) the joint reaction force, because any shear force within the glenohumeral joint creates some friction across its joint surfaces and because all forces that compress the head into the glenoid fossa must be opposed by an equal force from the glenoid fossa in the opposite direction (joint reaction force). Due to muscular compression forces, joint reaction forces can reach magnitudes of 9 to 10 times the weight of the upper extremity as the arm is elevated.[11,41] When the medially directed resultant muscular forces have slightly superior or inferior components, shear forces are the result. The greatest shear forces during humeral elevation typically occur between 30° and 60° of elevation.[41]

Costs of Dynamic Stabilization of the Glenohumeral Joint

When all stabilization forces and factors are intact and properly functioning, the head of the humerus rotates into flexion or abduction around a relatively stable axis with minimal translation. Over time, however, even normal stresses resulting from the complex dynamic stabilization process may lead to degenerative changes or dysfunction at the glenohumeral joint. Any disruption in the synergistic action of the dynamic stabilization factors may accelerate degenerative changes in or around the joint.

The supraspinatus muscle is a particularly key structure in dynamic stabilization. The supraspinatus is either passively stretched or actively contracting when the arm is at the side (depending on load); it also participates in humeral elevation throughout the ROM. Consequently, the tendon is under tension most of a person's waking hours and is vulnerable to tensile overload and chronic overuse. Mechanical compression and impingement of the stressed supraspinatus tendon may occur on either the superior or inferior surface when the subacromial space is reduced by osteoligamentous factors, when there is increased superior or anterior translation of the humeral head center with less favorable glenohumeral mechanics, when the scapula does not posteriorly tilt or upwardly rotate adequately during humeral elevation, or when occupational factors require heavy lifting or sustained overhead arm postures. The supraspinatus tendon is the most vulnerable of the cuff muscles. However, the overuse and potential impingement issues also apply to the other cuff muscles. Symptomatic and asymptomatic partial or full thickness rotator cuff tears are seen in many people over the age of 70, with the supraspinatus likely to show lesions before the other tendons of the cuff.[90] Rotator cuff tendonopathy or tears typically produce pain between 60° and 120° of humeral elevation in relation to the trunk. This range constitutes what has been described as the **painful arc.** It is within this ROM that the tendons of the rotator cuff were previously described as passing beneath the coracoacromial arch in a cadaveric investigation.[58] However, more recent investigation of active motion in human subjects has revealed that beyond 60° of humerothoracic abduction, the supraspinatus tendon has rotated past the overlying acromion.[59]

CASE APPLICATION

Supraspinatus Tendon Tears

case 7–3

We have already noted that Susan Sorenson's symptoms are consistent with and may be related to subacromial impingement. She reports pain with elevation of the arm. Her job as a dental hygienist requires sustained elevation of her arms in the lower range of the painful arc (60°–80°). Rates of shoulder pain consistent with rotator cuff involvement are higher than the norm in occupational groups that need to sustain such shoulder postures. Ms. Sorenson's past breast cancer treatment may also have altered the position of the scapula and the dynamic stabilization of the glenohumeral joint. Even a small amount of superior translation of the humeral head may contribute to changes that occur in the pressure within the subacromial bursa as the humerus elevates, especially if the subacromial space is reduced by other factors. The increased subacromial bursa pressures are related to both arm position and load, with greater pressures in the bursa evident as the arms are loaded and maintained in an elevated position.[91,92] The increased pressure of the subacromial bursa, especially with a concomitant supraspinatus contraction as the arm elevates, may produce further narrowing of the subacromial space and may decrease blood supply to the supraspinatus tendon, where small anastomosing vessels are responsible for tendon nutrition.[16] Such restriction of blood supply may be one factor contributing to an increasing incidence of supraspinatus tendon tears from minor trauma with increasing age.[93] Supraspinatus tendon tears, however, are not attributable to the aging process alone. The etiology of these tears is considered multifactorial.[94]

Degenerative changes in the acromioclavicular joint may result in pain in the same area of the shoulder as pain from supraspinatus or rotator cuff lesions. Pain due to acromioclavicular degeneration is more typically found when the arm is raised beyond the painful arc or when the arm is adducted across the body, compressing the acromioclavicular joint surfaces.[95] The long head of the biceps brachii similarly can produce pain in the anterosuperior shoulder. Because the long head of the biceps tendon also passes directly beneath the impinging structures of the coracoacromial arch, it is subject to some of the same degenerative changes and the same trauma seen in the tendons of the rotator cuff. Whether the biceps is actively contributing to elevation of the arm, to joint stabilization, or is passive, the tendon of the biceps must slide within the bicipital groove and under the transverse humeral ligament as the humerus moves. If the bicipital tendon sheath is worn or inflamed, or if the tendon is hypertrophied (as often seen with rotator cuff tears), the gliding mechanism may be interrupted and pain produced. A tear in the transverse humeral ligament may result in the tendon of the long head popping in and out of the bicipital groove with rotation of the humerus, a potentially wearing and painful microtrauma.

CASE APPLICATION

Acromioclavicular Joint Degenerative Changes

case 7–4

In addition to or instead of rotator cuff or biceps tendonopathy or a partial rotator cuff tear, Ms. Sorenson's shoulder pain may be related to acromioclavicular joint degeneration. Because of her history of an acromioclavicular joint separation, she is more likely to have secondary acromioclavicular joint degeneration.[26] If she has a painful arc of motion between 60° and 120° of elevation, it may be more indicative of a rotator cuff or biceps primary source of pain, whereas pain later in the motion may indicate that acromioclavicular degeneration is the primary source of pain.

Mechanical deviations in glenohumeral stabilization factors may result in injury to other structures of the joint besides the rotator cuff (e.g., the glenoid labrum) and to subluxation of the glenohumeral joint. Dislocation of the glenohumeral joint can also occur; in fact, it is the most frequently dislocated of all the joints in the body. Capsuloligamentous and muscle reinforcement to the glenohumeral joint are weakest inferiorly, but it is most common for the glenohumeral joint to dislocate anteriorly, or anteriorly and inferiorly, because of the type of forces to which it is exposed. Although the subscapularis and the glenohumeral ligaments reinforce the capsule anteriorly, a force applied to an abducted, laterally rotated arm can force the humeral head beyond the limits of the anterior glenoid. A predisposition to glenohumeral subluxation or dislocation is multifactorial. Saha suggested that people are most susceptible when their individual structural variations are in the direction of (1) anterior tilt of the glenoid fossa in relation to the scapular plane, resulting in less of a mechanical block of the glenoid fossa to anterior translation; (2) excessive retroversion of the humeral head; or (3) weakened rotator cuff muscles.[35] Alternatively, Weiser suggested that scapular internal rotation results in less anterior humeral head translation and greater tension in the anterior capsule.[96]

INTEGRATED FUNCTION OF THE SHOULDER COMPLEX

The shoulder complex acts in a coordinated manner to provide the smoothest and greatest ROM possible to the upper limb. Motion available to the glenohumeral joint alone would not account for the full range of elevation (abduction or flexion) available to the humerus. The remainder of the range is contributed by the scapula on the thorax through the sternoclavicular and acromioclavicular joints. Combined **scapulohumeral motion** (1) distributes the motion between the joints, permitting a large ROM with less compromise of stability than would occur if the same range occurred at one joint; (2) maintains the glenoid fossa in an optimal position in relation to the head of the humerus, increasing joint congruency while decreasing shear forces; and (3) permits muscles acting on the humerus to maintain a good length-tension relationship while minimizing or preventing active insufficiency of the glenohumeral muscles.

Scapulothoracic and Glenohumeral Contributions

The scapula on the thorax contributes to elevation (flexion and abduction) of the humerus by upwardly rotating the glenoid fossa 50° to 60° from its resting position.[12,14] If the humerus were immobile at the glenohumeral joint, the scapula alone would theoretically result in up to 60° of elevation of the humerus relative to the thorax. The humerus, of course, is not immobile but can move independently on the glenoid fossa. The glenohumeral joint contributes 100° to 120° of flexion and 90° to 120° of abduction. The combination of scapular and humeral movement results in a maximum range of elevation of 150° to 180° (Fig. 7–40).[66,97] Some of the variability in ranges reported by investigators is due to individual structural variations (especially for the glenohumeral joint); another factor in variability may be the extent to which trunk contributions were isolated from humeral motions during the measurement. If trunk motions are not included in the measurements, the overall range for most people is closer to 150° than 180°.[12,66] An *overall* ratio of 2° of glenohumeral to 1° of scapulothoracic motion during arm elevation is commonly described, and this combination of concomitant glenohumeral and scapulothoracic motion is most commonly referred to as **scapulohumeral rhythm.** According to the 2-to-1 ratio framework, flexion or abduction of 90° in relation to the thorax would be accomplished through approximately 60° of glenohumeral and 30° of scapulothoracic motion. It must also be recognized, however, that elevation of the arm is accompanied not only by elevation of the humerus but also by lateral rotation of the humerus in relation to the scapula. During flexion, an average of 51° of lateral rotation has been reported.[14] In addition, the plane of elevation varies for the humerus relative to the scapula, depending on the plane of elevation relative to the thorax.[14] When asked to actively elevate in a scapular plane (40° anterior to the frontal plane), subjects on average positioned the humerus 5° anterior to the scapular plane.[14] During flexion, the glenohumeral plane of elevation increased up to 30° anterior to the scapula plane, and in abduction, the glenohumeral plane of elevation was up to 20° posterior to the scapular plane.[14]

The 2-to-1 ratio of glenohumeral to scapulothoracic contribution to elevation of the arm is acknowledged to be an oversimplification, with substantial variability in scapular and humeral contributions at different points in the ROM and among individuals. The distinction must also be made between elevation of the arm from vertical and elevation of the arm relative to the trunk. The trunk may laterally flex or extend to gain additional range for the arm. However, we will consistently refer to elevation of the arm *in relation to the trunk* unless otherwise stated. As long as the trunk does *not* participate in the motion, the degree of elevation of the arm from vertical and elevation of the arm relative to the trunk will be the same.

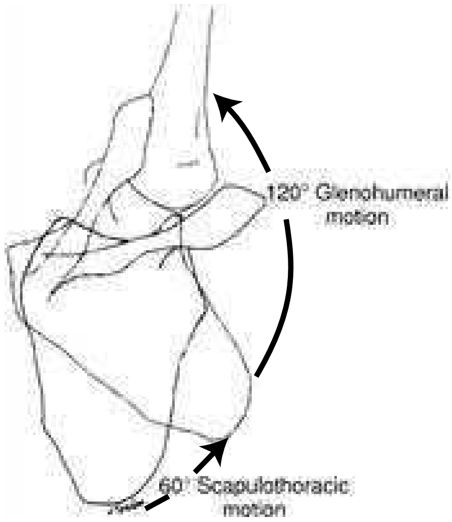


Figure 7–40 When the total range of elevation is considered to be 180°, it is common to attribute 120° of the ROM to the humerus at the glenohumeral joint and 60° to the scapula on the thorax—with the two segments moving concomitantly rather than sequentially.

Concept Cornerstone 7-3

Variations in Scapulohumeral "Rhythm"

Inman and coworkers reported that during the initial 60° of flexion or the initial 30° of abduction of the humerus, an inconsistent amount and type of scapular motion occurred in relation to glenohumeral motion.[11] The scapula has been described as seeking a position of stability in relation to the humerus during this period (setting phase).[11,79,98] In this early phase, motion occurs primarily at the glenohumeral joint, although stressing the arm may increase the scapular contribution.[70] A number of studies have investigated the scapulohumeral "rhythm," with ratios reported varying between 1.25:1 and 2.69:1.[34,35,69,70,98,99] Ratios are often described as nonlinear, indicating changing ratios during different portions of the ROM for elevation of the arm. The rhythm varies among individuals and may vary with external constraints.[99] Synchronous upward rotation of the scapula with glenohumeral elevation is certainly an important concept. However, the utility of the term *rhythm* seems limited because there does not appear to be a definitive scapulohumeral "rhythm" and because scapulohumeral "rhythm" provides limited insight into pathologies.

The scapula contributes to elevation of the arm not only by upwardly rotating on the thorax but also through other scapulothoracic motions. The exact magnitudes of different scapular motions vary across studies. The general patterns of scapular motions, however, are relatively consistent. As the arm is elevated into flexion, scapular plane abduction, or frontal plane abduction, the scapula posteriorly tilts on the thorax.[12,14,28,29,31] As the arm moves from the side to 150° of elevation, the magnitude of this motion is about 20° to 30°.[12,14] This posterior tilting allows the inferior angle of the scapula to move anteriorly and stay in contact with the thorax as it rotates upward and around the rib cage. Posterior tilting of the scapula also has the effect of bringing the anterior acromion up and back. This may serve to minimize reduction in the subacromial space as the humerus elevates.

During elevation, the scapula is more variable in its internal/external rotation both within and between subjects.[12,14,28] In general, at the end ranges of elevation the scapula is externally rotating on the thorax.[12,14,28,31] However, early in the range of motion for flexion, slight internal rotation of the scapula on the thorax occurs. Prevention of excess internal rotation of the scapula is important for keeping the scapula, particularly the medial border, in contact with the thorax as the scapula upwardly rotates during arm elevation. McClure and colleagues reported about 25° of external rotation of the scapula during abduction of the humerus in the plane of the scapula, most of which occurred at more than 90° of motion.[12] However, a more consistent scapular internal rotation position during abduction in the scapular plane has been reported in other studies.[14,28] In flexion, the scapula initially protracts and internally rotates to orient the glenoid fossa anteriorly (in the sagittal plane).[12,14] Structural limitations or inability of muscles to appropriately stabilize the scapula may result in anterior tilting, internal rotation, and downward rotation of the scapula with attempted flexion of the arm (Fig. 7–41).

Sternoclavicular and Acromioclavicular Contributions

Elevation of the arm in any plane involves motion of the sternoclavicular and acromioclavicular joints to produce scapulothoracic motion. Given the complexity of the linkages, there is limited description in the literature on the relative contributions of the sternoclavicular and acromioclavicular joints to the potential 60° arc of upward rotation of the scapula on the thorax as the scapula moves through its full ROM. However, recent investigations report consistent average values for sternoclavicular and acromioclavicular joint motions during active elevation despite varying measurement methods.[12,14,23]

The initiation of scapulothoracic upward rotation as the arm is flexed or abducted appears to couple with clavicular posterior rotation and elevation at the sternoclavicular joint.[79] This scapular upward rotation occurs around an oblique A-P axis, passing through the costoclavicular ligament (sternoclavicular joint motion) and projecting backward through the root of the scapular spine (scapulothoracic motion) (Fig. 7–42). As elevation of the arm progresses, the scapulothoracic axis of rotation gradually shifts laterally, reaching the acromioclavicular joint in the final range of scapular upward rotation[79] (Fig. 7–43). This major shift in the axis of rotation happens because the scapulothoracic motion can occur only through a combination of motions at the sternoclavicular and

Figure 7–41 Structural limitations or the inability of muscles to appropriately stabilize the scapula may result in anterior tilting, internal rotation, and downward rotation of the scapula during elevation of the arm, lifting the inferior and medial angles of the scapula off the thorax.

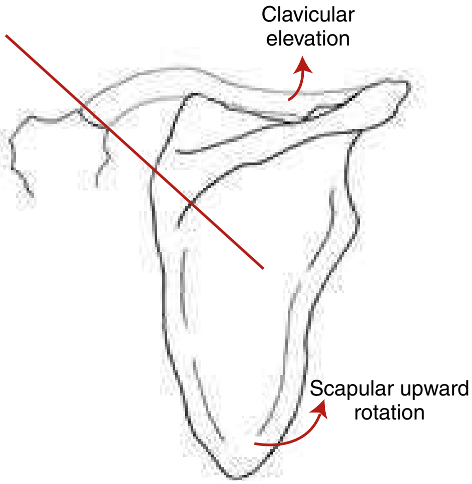


Figure 7–42 With elevation of the arm, the scapulothoracic upward rotation contribution begins as posterior rotation and elevation at the sternoclavicular joint around an axis that appears to pass posteriorly from the costoclavicular ligament to the root of the spine of the scapula.

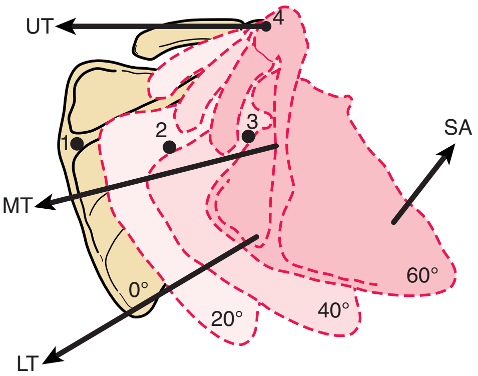


Figure 7–43 With the exception of the upper trapezius (UT) attaching to the clavicle, the action lines of the middle trapezius (MT), lower trapezius (LT), and serratus anterior (SA) muscles combine to produce upward rotation of the scapula on the thorax. The axis of scapular upward rotation progresses from its initial position (1) near the root of the scapular spine, laterally (2 and 3) to its final location near the acromioclavicular joint (4) at the end of the motion.

acromioclavicular joints. When the axis of scapular upward rotation is near the root of the scapular spine, scapulothoracic motion is primarily a function of sternoclavicular joint motion. When the axis of scapular upward rotation is at the acromioclavicular joint, acromioclavicular joint motions predominate; and when the axis of scapular upward rotation is in an intermediate position, both the sternoclavicular and acromioclavicular joints are contributing to scapulothoracic motion.

Inman and coworkers in 1944 described the relative contributions of the sternoclavicular and acromioclavicular joints to scapulothoracic upward rotation during arm elevation to be about 50% from sternoclavicular elevation and 50% from acromioclavicular upward rotation (20°–30° each to obtain 50°–60° upward rotation).[11] However, current three-dimensional descriptions of clavicular motion show only about 10° or less of clavicular elevation during arm elevation.[14,23] In addition, as described previously (see Fig. 7–17), only about one third of this elevation (about 3°) will couple with scapular upward rotation.[30] In order for upward rotation of the scapula to occur at the acromioclavicular joint (which has been described in three-dimensional studies as 15°–20° of acromioclavicular joint upward rotation),[14,23] the limitation to acromioclavicular motion imposed by the coracoclavicular ligament must be overcome. Tension in the coracoclavicular ligament (especially the conoid portion) is produced as the coracoid process of the scapula gets pulled downward with muscle forces attempting to upwardly rotate the scapula at the acromioclavicular joint. The tightened conoid ligament pulls its posteroinferior clavicular attachment forward and down as the coracoid process drops, causing the clavicle to posteriorly rotate. Posterior rotation of the clavicle around its longitudinal axis will result in additional scapulothoracic upward rotation (see Figs. 7–7 and 7–17). The scapulothoracic upward rotation occurs both as the lateral end of the S-shaped clavicle flips up, coupling about two thirds of this posterior sternoclavicular joint rotation to scapulothoracic upward rotation[30], and as the tension in the coracoclavicular ligament is reduced, permitting upward rotation at the acromioclavicular joint. The magnitude of posterior rotation of the clavicle may be anywhere from 30° to 50°,[11,14] contributing 20° or more to scapulothoracic upward rotation. Subsequently, sternoclavicular and acromioclavicular joint rotations work cumulatively to produce overall scapular upward rotation on the thorax during arm elevation.

Throughout elevation of the arm (flexion, scapular plane abduction, or frontal plane abduction), the clavicle also retracts at the sternoclavicular joint, typically 20° to 30°, which couples with external rotation of the scapula on the thorax.[12,14,23] However, at the same time, the acromioclavicular joint is internally rotating 10° to 15° throughout arm elevation.[14,23] The magnitude of this internal rotation is slightly greater in flexion and less in frontal plane abduction.[14] As a result, the acromioclavicular joint internal rotation offsets a large portion of the scapulothoracic external rotation that would occur from sternoclavicular joint retraction. The magnitudes of acromioclavicular joint internal rotation can be expected to differ with variations in scapular resting position, rib cage configuration, and muscle dynamics. Although the range varies somewhat, the component motions of the sternoclavicular (retraction) and acromioclavicular (internal rotation) joints are similar regardless of whether the elevation motion is performed in the sagittal, scapular, or frontal planes. The other difference between the performances of sagittal plane and frontal plane elevation is that the clavicle and scapula begin flexion in less retraction and more internal rotation in order to bring the glenoid fossa forward, keeping the fossa in line with the shaft of the elevating humerus.[14]

The final component motion of the scapula on the thorax occurring during arm elevation in all planes is posterior tilting. Although about one third (about 10°) of sternoclavicular posterior rotation of the clavicle will couple with scapulothoracic posterior tilting, two thirds of sternoclavicular elevation (about 6°) will couple with scapulothoracic anterior titlting.[30] This is because the acromioclavicular joint internal rotation angle is about two thirds of the way to a 90° alignment between the clavicular long axis and the scapular plane (see Fig. 7–17). In a 90° alignment, clavicular elevation at the acromioclavicular joint would produce scapulothoracic anterior tilting. As a result, the rotations of the sternoclavicular joint offset one another with regard to scapulothoracic tilting and minimal scapulothoracic posterior tilting is produced. The predominance of scapulothoracic posterior tilting during elevation of the arm occurs with acromioclavicular joint posterior tilting, which averages 20° or more.[14,23]

Upward Rotators of the Scapula

There is agreement that the motions of the scapula are primarily produced by a balance of the forces between the trapezius and **serratus anterior** muscles through their attachments on the clavicle and the scapula (see Fig. 7–43). The upper portion of the trapezius muscle is attached to the clavicle and is positioned to contribute directly to the initial elevation of the clavicle as well as to the sternoclavicular joint retraction that occurs during normal arm elevation.[100]

The serratus anterior muscle makes its contribution to the combined clavicular and scapular motion through its action on the scapula. When the location of the axis for scapulothoracic upward rotation is at the acromioclavicular joint, the lower trapezius may contribute to upward rotation, although the lower serratus anterior muscle has a much greater moment arm.

There are substantial implications for the large shift in the axis of rotation for scapular upward rotation with regard to muscle function. As the axis of rotation shifts laterally toward the acromioclavicular joint (toward axis position 4 in Fig. 7–43), the middle trapezius has a progressively smaller moment arm for scapulothoracic upward rotation; in the latter stages of elevation motion, it may be a downward rotator of the scapula. The torque capabilities of the lower trapezius will also change across the range of elevation as the axis of rotation shifts.[100] The serratus anterior muscle maintains a large moment arm for scapulothoracic upward rotation throughout the entire range of elevation.

Despite lines of action and moment arms that are not as effective for producing scapulothoracic upward rotation, the trapezius is clearly active during arm elevation and plays a role in the balance of forces that move and control the scapula on the thorax.[11] If the serratus anterior muscle acted in isolation, the lateral line of action would result in substantial lateral translation and less effective upward rotation of the scapula. The medial translatory action of the trapezius offsets the lateral translatory action of the serratus and results in more effective upward rotation by the serratus.

Continuing Exploration 7-6:

Trapezius Function Reconsidered

Johnson and colleagues[100] proposed lines of action for the trapezius muscle that differ from the traditional anatomic descriptions of muscle fiber orientation. Typically, the upper trapezius is shown with a superior and medial line of action on the scapula and the lower trapezius with an inferior medial line of action (Fig. 7–44). Johnson and colleagues suggested, on the basis of cadaveric dissection, that the upper trapezius fibers act minimally on the scapula and more through elevation and retraction of the clavicle, thus indirectly minimally upwardly rotating the scapula on the thorax. The middle trapezius would be capable of upwardly rotating the scapula with a small moment arm; this role would decrease as the axis of rotation approaches the acromioclavicular joint later in elevation (see Fig. 7–43).[100] Johnson and colleagues also suggested, on the basis of actual fiber orientation and cross-sectional area, that the lower trapezius action line should be directed more medially and less inferiorly than traditionally depicted.[100] Consequently, this muscle would have a lesser moment arm to contribute to scapular upward rotation on the thorax than has been classically described (see Fig. 7–43). These descriptions are consistent with Dvir and Berme's[79] model of shoulder function, which identifies the lower serratus anterior muscle as the prime mover of the scapula and the trapezius as the prime stabilizer.

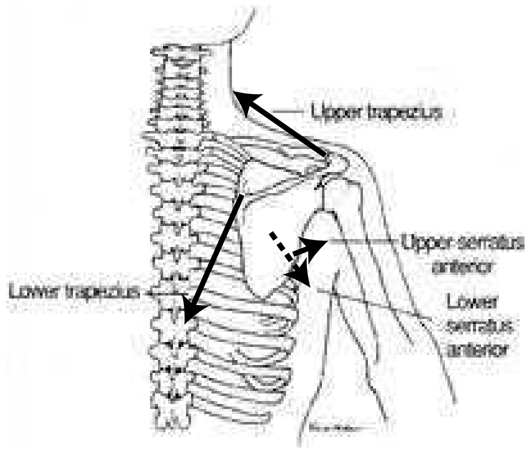


Figure 7–44 Classically described action lines for the upward rotators of the scapula.

The contribution of scapulothoracic muscles to producing and controlling other scapulothoracic motions (anterior/posterior tilting and internal/external rotation) is not well described in the literature. The middle and lower serratus anterior muscles, with their insertions into the inferior angle and medial border of the scapula, play a primary role in stabilizing the scapula to the thorax. Although the function of the serratus anterior muscle is traditionally considered to be the production of scapular protraction, its line of action is capable of producing acromioclavicular joint external rotation as it pulls the scapula laterally on the thorax.[101] Any scapular protraction the serratus anterior might help to produce would be through sternoclavicular joint protraction. In fact, paralysis of the serratus anterior muscle classically presents with scapular "winging." The scapular winging includes internal rotation and anterior tilting of the scapula, produced by the remaining muscles without the stabilizing external rotation and posterior tilting influence of the middle and lower serratus. The lower serratus anterior muscle also has a large moment arm to produce posterior tilting of the scapula. The middle and lower trapezius also can contribute to external rotation torques of the scapula at the acromioclavicular joint, and the upper trapezius to clavicular retraction torque at the sternoclavicular joint.[100,101]

Structural Dysfunction

Completion of the range of elevation of the arm depends on the ability of the glenohumeral, sternoclavicular, and acromioclavicular joints each to make the needed contribution. Disruption of movement in any of the participating joints can result in a loss of ROM. Once restrictions to function are introduced, the concept of scapulohumeral rhythm is altered; that is, a reduction in glenohumeral joint range may *not* result in a proportional decrease in scapulothoracic range. The ratio of movement is no longer consistent because the body will likely recruit remaining motion at other joints.[102] Although it is not necessarily predictable, the restriction of motion at any one joint in the shoulder complex may result in the development of some hypermobility (and reduced stability) in remaining articulations.

Example 7-1

Glenohumeral Joint Hypomobility

If motion at the glenohumeral joint is restricted by pain or disease, the total ROM available to the humerus will be reduced. Whatever portion of the motion remains at the glenohumeral joint will still be accompanied by scapulothoracic motion. A restriction of the humerus, limiting glenohumeral joint abduction to approximately 60°, theoretically can combine with up to 60° of scapulothoracic motion to provide a total available range of 120° as the arm is raised from the side. Recent research has also provided evidence of increased scapulothoracic upward rotation, or scapular "substitution" in cases where glenohumeral mobility is reduced, such as glenohumeral osteoarthritis or adhesive capsulitis.[102]

Example 7-2

Sternoclavicular Joint Hypomobility

Hypothetical fusion of the sternoclavicular joint would substantially minimize scapulothoracic movement. Because clavicular elevation, rotation, and retraction occur through the sternoclavicular joint, sternoclavicular fusion would eliminate both contributors of scapular upward rotation and result in scapulothoracic internal rotation. The arm would elevate only at the glenohumeral joint, with limited contribution of acromioclavicular joint upward rotation, because the clavicle could not posteriorly rotate to reduce coracoclavicular ligament tightness. It should be noted, however, that fixation of the very stable sternoclavicular joint rarely occurs. In such an unusual instance, one would expect over time to develop hypermobility and increased instability of the acromioclavicular joint.

Example 7-3

Acromioclavicular Joint Hypomobility

Although hypermobility and instability of the acromioclavicular joint are more common, fusion of the joint generally occurs only through surgical fixation. With limited or no acromioclavicular joint mobility within the closed chain of the acromioclavicular, sternoclavicular, and scapulothoracic joints, motions of the scapula on the thorax (and therefore motions of the clavicle at the sternoclavicular joint) could be limited. If the clavicle attempted to protract with a fused acromioclavicular joint (and a fixed scapuloclavicular relationship), the clavicular protraction would bring the scapula into the thorax, and further motion would be limited. Clavicular protraction (and scapular internal rotation at the initiation of flexion of the arm) is also dependent on the ability of the acromioclavicular joint to allow the scapula to internally rotate to orient the glenoid fossa anteriorly and to accommodate to the curvature of the thorax.

MUSCLES OF ELEVATION

Elevation and depression have been described as the two primary patterns of shoulder complex function. Elevation activities are those that require muscles to overcome or control the weight of the limb and its load. The completion of normal elevation depends on not only the freedom of movement and integrity of the sternoclavicular, acromioclavicular, and glenohumeral joints, but also the appropriate strength and function of the muscles producing and controlling movement. A closer look at the activity of these muscles should enhance an understanding of normal function, as well as contribute to an understanding of the deficits seen in pathologic situations.

Deltoid Muscle Function

The deltoid is at resting length (optimal length-tension) when the arm is at the side. When at resting length, the deltoid's angle of pull results in a predominance of superior translatory pull on the humerus with an active contraction (see Fig. 7–36). With an appropriate synergistic inferior pull from the infraspinatus, teres minor, and subscapularis muscles, the rotary component of the deltoid muscle is an effective primary mover for elevation. While the anterior deltoid is the prime mover for flexion, it can assist with abduction after 15° of glenohumeral motion.[81] During abduction in the plane of the scapula, the anterior and middle deltoid segments are optimally aligned to produce elevation of the humerus.[40] The line of action of the posterior deltoid has too small a moment arm (and too small a rotary component) to contribute effectively to frontal plane abduction; it serves primarily as a joint compressor[37,78] and in functions such as horizontal abduction and extension.

As the humerus elevates, the translatory component of the deltoid diminishes its superior dislocating influence as its resultant force vector shifts increasingly toward the glenoid fossa. At the same time, the rotary component of the deltoid must counteract the increasing torque of gravity as the arm moves toward horizontal. Analysis of EMG shows gradually increasing activity in the deltoid, peaking at 90° of humeral abduction with a plateau for the remainder of the motion (Saha[35] found a peak at 120° with a drop-off to moderate activity at 180°). The peak activity in flexion does not occur until the end of the range, and there is less total activity.[11,35] Although the moment arm of the deltoid gets larger as the humerus elevates[38] and the torque of gravity diminishes once the arm is above the horizontal, the high activity level of the deltoid continues. The shortening deltoid is not able to produce as much active tension, and passive tension is diminished. As a result, a greater number of motor units must be recruited to maintain force output. The multipennate structure and considerable cross-sectional area of the deltoid help compensate for its relatively small moment arm, low mechanical advantage, and less-than-optimal length-tension relationship as elevation progresses.

Maintenance of an appropriate length-tension relationship of the deltoid is strongly dependent on simultaneous scapular movement or scapular stabilization. When the scapula is restricted and cannot upwardly rotate, the loss of tension in the deltoid with increased shortening has been reported to result in reduced glenohumeral abduction (whether the supraspinatus is available for assistance or not).[11] If the scapular upward rotators (serratus anterior and trapezius muscles) are absent, the middle and posterior fibers of the activate deltoid (originating on the acromion and spine of the scapula) will act not on the heavier arm but on the lighter scapula; that is, without the stabilizing force of the upward rotators, the middle and posterior deltoid will downwardly rotate the scapula (Fig. 7–45). Although the deltoid can still achieve the glenohumeral motion attributed to it when the scapular motion is restricted, the glenohumeral motion occurs on a downwardly rotated scapula. The net effect of attempted abduction by the deltoid in the presence of trapezius and serratus anterior muscle paralyses is that the arm will rise from the side only about 60° to 75° (see Fig. 7–20A).[103]

As we discussed earlier, effective deltoid activity also depends on intact rotator cuff muscles. With complete derangement of the cuff, a contraction of the deltoid results in a shrug of the shoulder (clavicular elevation at the sternoclavicular joint and upward translation of the humerus) rather than in abduction of the humerus from the side. Stimulation of the axillary nerve (innervating the deltoid and teres minor muscles alone) produces approximately 40° of abduction.[104] Partial tears in or partial paralysis of the cuff will weaken the elevation produced by the deltoid.

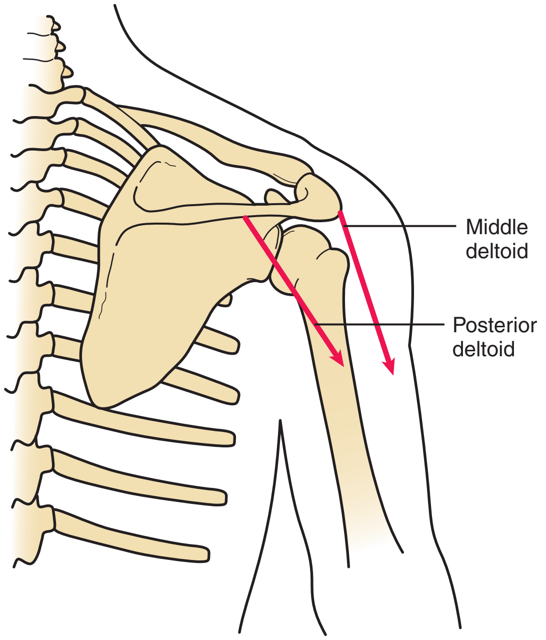


Figure 7–45 Without the trapezius, the scapula rests in a downwardly rotated position as a result of the unopposed effect of gravity on the scapula. When abduction of the arm is attempted, the middle and posterior fibers of the activated deltoid—unopposed by the trapezius—will act on the lighter scapula to increase the downward rotary pull on the scapula.

Supraspinatus Muscle Function

The supraspinatus muscle is considered an abductor of the humerus. Like the deltoid muscle, it functions in all planes of elevation of the humerus. Its role, according to MacConaill and Basmajian,[45] is quantitative rather than specialized. The pattern of activity of the supraspinatus is essentially the same as that found in the deltoid.[11] The moment arm of the supraspinatus is fairly constant throughout the ROM and is larger than that of the deltoid for the first 60° of shoulder abduction.[37] When the deltoid is paralyzed, the supraspinatus alone can bring the arm through most, if not all, of the glenohumeral range, but the motion will be weaker. With a suprascapular nerve block paralyzing the supraspinatus and the infraspinatus, the strength of elevation in the plane of the scapula is reduced by 35% at 0° and by 60% to 80% at 150°.[105]

The secondary functions of the supraspinatus are to compress the glenohumeral joint, to act as a "steerer" for the humeral head, and to assist in maintaining the stability of the dependent arm. With isolated and complete paralysis of the supraspinatus muscle, or an isolated supraspinatus tear, some loss of abduction force is evident, but most of its functions can be performed by remaining musculature. Isolated paralysis of the supraspinatus is unusual, however, because its innervation is the same as the infraspinatus and related to that of the teres minor muscle. Most commonly, tears of the rotator cuff muscles do not remain isolated to the supraspinatus but extend to the infraspinatus or subscapularis, producing a more extensive deficit than is seen with paralysis of the supraspinatus alone.

Infraspinatus, Teres Minor, and Subscapularis Muscle Function

When Inman and coworkers[11] assessed the combined actions of the infraspinatus, teres minor, and subscapularis muscles, EMG activity indicated a nearly linear rise in action potentials from 0° to 115° elevation. Activity dropped slightly between 115° and 180°. Total activity in flexion was slightly greater than that in abduction. In abduction, an early peak in the activity of these muscles appeared at 70° of elevation. As noted earlier, the medial rotary function of the subscapularis muscle diminishes with abduction,[81] serving instead to steer the head of the humerus horizontally while continuing to work with the other cuff muscles to compress and stabilize the joint.[35]

Upper and Lower Trapezius and Serratus Anterior Muscle Function

The upper trapezius, along with the levator scapula muscle, supports the shoulder girdle against the downward pull of gravity. Although support of the scapula in the pendant limb is passive in many individuals, loading the limb will produce activity in these muscles.[11,45] As previously discussed, the trapezius and lower serratus anterior muscles work synergistically to produce upward rotation of the scapula on the thorax. When activity of the upper and lower trapezius and serratus anterior muscles was monitored by EMG during humeral elevation, the curves were similar and complementary. Activity in the trapezius rises linearly to 180° in abduction, with more undulating activity in flexion. The serratus anterior muscle shows a linear increase in action potentials to 180° in flexion, with undulating activity in abduction.[11] Saha found that the upper and lower trapezius activity peaked and reached plateau before the end of the range, with some decrease in activity at maximal elevation.[35] The middle trapezius muscle is also active during elevation (especially abduction) and may contribute to upward rotation of the scapula early in the ROM, as well as contributing to medial stabilization against the lateral pull of the serratus anterior.

Continuing Exploration 7-7:

EMG and Muscle Function

EMG activity must be interpreted as only an indirect representation of muscle force production. If the length of the muscle is not held constant, an increase in EMG activity is not necessarily indicative of increased force production but, rather, may be compensating for muscle shortening, as is proposed to be the rationale for increased end-range activity of the deltoid. Higher activity levels are commonly seen in muscles at the end ranges of motion, despite the necessity for peak torque production near the midrange of motion, when gravitational resistance torques are often greatest. In addition, for increased EMG activity to relate to increased muscle force, the velocity of contraction and type of contraction (concentric, eccentric, isometric) must not change across comparison conditions.

The EMG activity of a muscle during a specific motion does not define the function of the muscle. Often, muscles are active to offset unwanted translatory components of another muscle force, rather than to produce a primary rotational torque. This was discussed previously with trapezius function and will also be discussed as it occurs with rhomboid function during arm elevation.

In active abduction of the arm, the force of the trapezius seems more critical to scapulothoracic motion than in active flexion. When the trapezius is intact and the serratus anterior muscle is paralyzed, full active abduction of the arm can occur, although it is weakened, whearas flexion is more difficult. With EMG analysis, the trapezius has been found to be more active in abduction than in flexion.[101] This may be related to the increased scapulothoracic and acromioclavicular joint internal rotation noted in flexion as compared to abduction.[14] Without the trapezius (with or without the serratus anterior muscle), the scapula rests in a downwardly rotated position as a result of the unopposed effect of gravity on the scapula.

CASE APPLICATION

Trapezius Overuse

case 7–5

Excess activation of the trapezius has been identified in some patients with shoulder impingement symptoms.[28] This can result in an imbalance of forces between the trapezius and serratus. The result can be a shoulder shrug type of motion when trying to elevate the arm and less efficient upward rotation of the scapula on the thorax. Prolonged overuse of the upper trapezius can result in muscle fatigue and pain in this muscle. The pain over the upper trapezius region that Ms. Sorenson reports may be related to an upper trapezius overuse pattern with arm elevation.

Although the trapezius is more active in abduction, the serratus anterior muscle seems to be the more critical of the two muscles in producing scapular upward rotation during flexion of the arm. If the serratus anterior muscle is intact, trapezius muscle paralysis results in a loss of force of shoulder flexion but a fairly normal range of flexion. If the serratus anterior muscle is paralyzed (even in the presence of a functioning trapezius), flexion will be both diminished in strength and limited in range. When the scapular retraction component of the trapezius is unopposed by the serratus anterior muscle, the trapezius is unable to effectively upwardly rotate the scapula.

The role of the serratus anterior muscle in normal shoulder function appears to be essential in many aspects. This includes it being the only muscle capable of producing simultaneous scapular upward rotation, posterior tilting, and external rotation, the three component motions of the scapula on the thorax that have been identified as occurring during elevation of the arm. The serratus also has the largest moment arms of any of the scapulothoracic muscles, regardless of the changing scapulothoracic axis of rotation. The serratus is the primary stabilizer of the inferior angle and medial border of the scapula to the thorax. Finally, reduced serratus anterior muscle activity has been identified in patients with shoulder impingement,[28] which further suggests the importance of this muscle to normal shoulder function.

The serratus anterior is the prime mover for upward rotation of the scapula. The serratus and trapezius are also synergists for the deltoid during abduction at the glenohumeral joint. The trapezius and serratus anterior muscles stabilize the scapula and prevent the undesired downward rotary movement of the scapula by the middle and posterior deltoid segments that are attached to the scapula (see Fig. 7–45). The trapezius and serratus anterior muscles maintain an optimal length-tension relationship with the deltoid and permit the deltoid to carry its heavier distal lever through its full ROM. Thus, the role of the scapular forces of the trapezius and the serratus anterior muscles is both agonistic to scapular movement and synergistic with glenohumeral movement.

Rhomboid Muscle Function

The **rhomboid major** and **minor** muscles are active in elevation of the arm, especially in abduction. These muscles serve a critical function as stabilizing synergists to the muscles that upwardly rotate the scapula. If the rhomboids, downward rotators of the scapula, are active during upward rotation of the scapula, these muscles must be working eccentrically to control the change in position of the scapula produced by the trapezius and the serratus anterior muscles. Paralysis of these muscles causes disruption of the normal scapulohumeral rhythm and may result in diminished ROM. Like the middle and lower trapezius, the rhomboid muscles act primarily to offset the lateral translation component of the serratus anterior muscle and help prevent excess internal rotation of the scapula at the acromioclavicular joint by stabilizing the medial or vertebral border of the scapula to the thorax.

MUSCLES OF DEPRESSION

Depression is the second of the two primary patterns of shoulder complex function. Depression involves the *forceful* downward movement of the arm in relation to the trunk. If the arm is fixed by weight-bearing or by holding on to an object (e.g., a chinning bar), shoulder depression will move the trunk upward in relation to the arm. In depression activities, the scapula tends to rotate downward and adduct during the humeral motion, but there is not a consistent or overall ratio of movement of one segment in comparison with the other.

Latissimus Dorsi and Pectoral Muscle Function

When upper extremity motion is unrestricted, the **latissimus dorsi** muscle may produce adduction, extension, or medial rotation of the humerus. Through its attachment to both the scapula and humerus, the latissimus dorsi can also adduct and depress the scapula and shoulder complex. The latissimus dorsi also contributes to glenohumeral joint stability by compressing the joint when the arm is abducting.[106] However, when the upper extremity is weight-bearing or restricted, the latissimus dorsi muscle will pull the pelvis upward toward the scapula and humerus. When the hands are bearing weight on the handles of a pair of crutches, a contraction of the latissimus dorsi will unweight (lift) the feet as the trunk rises beneath the fixed scapula, allowing the legs to swing forward through the crutches.

The **sternal portion** of the **pectoralis major** is a key depressor of the shoulder complex. The action of the pectoralis major parallels that of the latissimus dorsi muscle (adduction, extension, or medial rotation of the humerus), with the pectoralis major located anterior to the glenohumeral joint rather than posterior. Pectoralis major activity during arm elevation increases glenohumeral joint stability by contributing to higher joint reaction forces,[106] but it also increases the magnitude of anterior humeral head translation.[107] In activities involving weight-bearing on the hands, the pectoralis major and the latissimus dorsi muscles combine to depress the shoulder complex while synergistically offsetting anterior/posterior translation of the humerus and protraction/retraction of the shoulder girdle. The **pectoralis minor** muscle assists the latissimus dorsi and pectoralis major by directly depressing the scapula through its attachment on the coracoid process. The pectoralis minor is oriented to internally rotate, downwardly rotate, and anteriorly tilt the scapula so the length of this muscle may influence full scapula movement during arm elevation.[108]

Teres Major and Rhomboid Muscle Function

The **teres major** muscle, like the latissimus dorsi, adducts, medially rotates, and extends the humerus. The teres major muscle is active primarily during resisted activities but may also be active during unresisted extension and adduction activities behind the back.[109] The function of the teres major muscle is strongly dependent on the activity of the rhomboid muscles. The teres major muscle originates on the scapula and attaches to the humerus. Consequently, its proximal segment is lighter than the segment to which it attaches distally. The proximal scapular segment must be stabilized to permit the teres major muscle to act effectively as an extensor and adductor of the distal humeral segment. Without stabilization, the teres major muscle would upwardly rotate the lighter scapula rather than move the heavier humerus (Fig. 7–46). The rhomboid muscles, as downward rotators of the scapula, offset the undesired upward rotary torque of the teres major muscle. By fixing the scapula as the teres major muscle contracts, the rhomboids allow the teres major muscle to move the heavier humerus. The rhomboids are assisted in stabilization of the scapula during humeral extension or adduction by the anteriorly located pectoralis minor muscle.

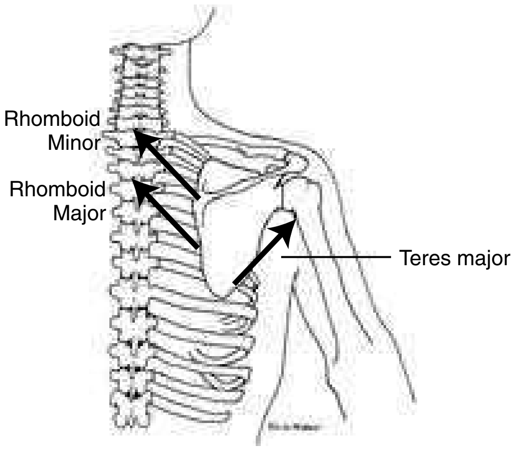


Figure 7–46 In order for the teres major muscle to extend the heavier humerus rather than upwardly rotate the lighter scapula, the synergy of the rhomboid muscles is necessary to stabilize the scapula.

Continuing Exploration 7-8:

Breast Cancer and Radiation

Standard treatment for breast cancer treated by breast-conserving therapy (lumpectomy) includes using two-field tangential radiation of the whole breast and chest wall.[110,111] Because of the contour of the rib cage, the ipsilateral pectoral muscles are generally included in the field. Radiation may be associated with the release in exposed tissue cells of certain cytokines and growth factors that stimulate radiation-induced fibroblast proliferation, collagen deposition, and fibrosis,[112] with the potential to result in secondary muscular and soft tissue fibrosis.[111,113] A clinical consequence of muscular fibrosis may be increased passive resistance to stretch, including the pectoralis major and minor muscles. There is evidence of reduced shoulder ROM and impaired mobility at higher rates in patients whose breast cancer was treated with radiation than in patients who underwent treatments that did not include radiation.[114,115]

CASE APPLICATION

Patient Summary case 7–6

In addition to her contributing history of acromioclavicular joint separation and her job requirements, Ms. Sorenson's treatment for breast cancer may be a contributing factor to her shoulder pain. The pectoralis major muscle is capable of producing medial rotation of the humerus and, indirectly, clavicular protraction. The pectoralis minor is capable of producing scapular downward rotation, internal rotation, and anterior tilting on the thorax.[108] These motions are all antagonistic to the motions of the scapula and humerus that must occur during normal arm elevation. Because of the attachment of the pectoralis minor muscle to the coracoid process and rib cage (Fig. 7–47), fibrotic changes and decreased extensibility as a result of radiation treatment could cause the muscle to limit scapular upward rotation, posterior tilting, and external rotation.[108] Decreased extensibility in the pectoralis major could limit her ability to laterally rotate the humerus and retract the clavicle as she elevates her arm. The reduced muscle extensibility may limit ROM. A subtle but potentially important effect may also be to increase the risk of impingement of the rotator cuff tendons and long head of the biceps brachii in elevation of the arm. The risk may be increased as a result of a decreased subacromial space because of the inability of the scapula to posteriorly tilt and externally rotate to clear the acromion during elevation. A reduction in scapular upward rotation may also result in hypermobility of the glenohumeral joint, which increases the likelihood that the humerus will impinge on the coracoacromial arch. Furthermore, a restriction in lateral rotation of the humerus from pectoral major tightness may not allow the greater tubercle to adequately clear the acromion. These risk factors from potential muscle fibrosis, Ms. Sorenson's potential acromioclavicular joint degenerative changes, and her need to maintain elevation of her arms for sustained periods of time during her workday may be multifactorial contributors to subacromial impingement and potential rotator cuff tearing.

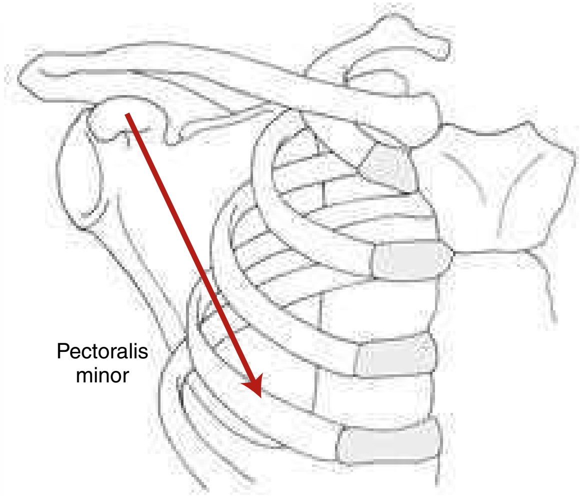


Figure 7–47 The attachment of the pectoralis minor muscle to both the coracoid process of the scapula and the rib cage may limit the ability of the scapula to upwardly rotate, posteriorly tilt, and externally rotate on the thorax if there is excess active or passive tension in this muscle during elevation of the arm.

SUMMARY

In this chapter, we laid the foundation for understanding more distal upper extremity joint function by exploring the intricate dynamic stabilization of the shoulder complex. The more distal joints of the upper extremity depend on the dual mobility/stability roles of the shoulder complex. Whereas function in the hand, for instance, can continue on a limited basis with loss of shoulder mobility, loss of shoulder stability can render the remaining function in the hand unusable. In the next chapter, we will explore the elbow as the intermediary between the shoulder and the hand.

STUDY QUESTIONS

1. Identify the intra-articular motions of the sternoclavicular joint for elevation/depression, protraction/retraction, and axial rotation.
2. What are the roles of the costoclavicular and interclavicular ligaments at the sternoclavicular joint?
3. Discuss the relevance of the sternoclavicular disc to sternoclavicular joint congruency, joint motion, and joint function.
4. Identify the scapular movements that take place at the acromioclavicular joint.
5. Discuss the relevance of the coracoclavicular ligament to acromioclavicular joint function.
6. Discuss the anatomical configuration of the humerus and the glenoid fossa as they relate to glenohumeral joint stability. What role do the glenoid labrum and joint capsule play in joint stability?
7. What is the most frequent direction of glenohumeral dislocation? Why?
8. Compare the relative stability and tendency toward degenerative changes in the glenohumeral, acromioclavicular, and sternoclavicular joints.
9. What are the advantages of the coracoacromial arch? What are the disadvantages?
10. What intra-articular motions must occur at the glenohumeral joint for full abduction to occur? What is the normal range of motion?
11. What muscle is the prime mover in shoulder glenohumeral flexion and abduction? What synergy is necessary for normal function of this muscle? Why?
12. Why is the supraspinatus able to abduct the shoulder without additional muscular synergy?
13. What accounts for the static stabilization of the glenohumeral joint when the arm is at the side? What happens if you excessively load the hanging (dependent) limb?
14. Identify five factors that play a role in the dynamic stabilization of the glenohumeral joint in either flexion or abduction.
15. What is the total ROM available to the humerus in elevation? How is this full range achieved?
16. How does the shape of the clavicle contribute to elevation of the arm?
17. What muscles are necessary to produce the normal scapular and humeral movements in elevation of the arm?
18. If the scapulothoracic joint were fused in neutral position, what active range of elevation would still be available to the upper extremity?
19. What is the most common traumatic problem at the acromioclavicular joint? What deficits is a person with this disability likely to encounter?
20. What are the consequences of a rupture of the coracoclavicular ligaments?
21. If the glenohumeral joint were immobilized by osteoarthritis, what range of elevation would be available to the upper extremity?
22. If isolated paralysis of the supraspinatus were to occur, what would be the likely functional deficit?
23. If the muscles of the rotator cuff are paralyzed, what is the effect when abduction of the arm is attempted?
24. When there is paralysis of the trapezius and the serratus anterior muscles, what is the functional deficit when abduction of the arm is attempted?
25. If the deltoid alone is paralyzed, what happens with attempted abduction of the arm? With attempted flexion of the arm?
26. What is the role of the rhomboids in elevation of the arm?
27. What differences do you see in attempted abduction if the trapezius alone is paralyzed, compared to the differences you see if both the trapezius and the serratus anterior muscles are paralyzed?
28. What muscular synergy does the teres major muscle require to perform its function?
29. Describe why electromyographic activity of the deltoid in normal abduction shows a gradual rise in activity to between 90° and 120°, with a plateau thereafter.
30. Which of the joints of the shoulder complex is most likely to undergo degenerative changes over time? Which is least likely?

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Chapter 8

The Elbow Complex

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INTRODUCTION

The joints and muscles of the elbow complex are designed to serve the hand. They provide mobility for the hand in space by shortening and lengthening the upper extremity. This allows the hand to be brought close to the face for eating and grooming or to be placed at a distance from the body equal to the length of the entire upper extremity. Rotation at the elbow complex provides additional mobility for the hand. In conjunction with providing mobility for the hand, the elbow complex structures also provide stability for skilled or forceful movements of the hand when performing activities with tools or implements. Many of the 15 muscles that cross the elbow complex[1] also act at either the wrist or shoulder, and therefore the wrist and shoulder are linked with the elbow in enhancing the function of the hand.

The elbow complex includes the elbow joint (humeroulnar and humeroradial joints) and the proximal and distal radioulnar joints. The elbow joint is considered to be a compound joint that functions as a modified or loose hinge joint. One degree of freedom is possible at the elbow, permitting the motions of flexion and extension, which occur in the sagittal plane around a coronal axis. A slight bit of axial rotation and side-to-side motion of the ulna occurs during flexion and extension, and that is why the elbow is considered to be a modified or loose hinge joint rather than a pure hinge joint.[2] Two major ligaments and five muscles are directly associated with the elbow joint. Three of the muscles are flexors that cross the anterior aspect of the joint. The other two muscles are extensors that cross the posterior aspect of the joint.

The proximal and distal radioulnar joints are linked and function as one joint. The two joints acting together produce rotation of the forearm and have 1 degree of freedom of motion. The radioulnar joints are diarthrodial uniaxial joints of the pivot (trochoid) type and permit rotation (supination and pronation), which occurs in the transverse plane around a longitudinal axis. Six ligaments and four muscles are associated with these joints. Two muscles are for supination, and two are for pronation. The elbow joint and the proximal radioulnar joint are enclosed in a single joint capsule but constitute distinct articulations.

8-1 Patient Case

James Daly, a 40-year-old carpenter, has come into the clinic complaining of pain in his right lateral forearm. He says that he has experienced numerous episodes of pain in the same area over the past few years. Usually the pain lessens, but does not entirely disappear, after a short rest period of 1 or 2 days. The current episode of pain is more severe than those he has experienced in the past; it has continued for a much longer period of time and has not been relieved by a short period of rest. He reports that the pain is worse when he attempts to hammer, saw, or lift the lumber used in his work.

We notice that James is holding his right elbow in slight flexion. Palpation reveals tenderness in the area over the patient's right lateral epicondyle, common extensor tendon, and the muscle belly of one of the wrist extensors. Some tenderness also appears to be present along the path of the radial nerve. Active wrist and/or finger extension increases the pain in the lateral elbow area. A test of the patient's grip strength was attempted but not completed because of the degree of discomfort that James was experiencing.

Our task is to determine which structures are involved in producing the patient's pain. Once we identify the structure involved and make a diagnosis, we need to select the most appropriate treatment by examining the evidence regarding the effectiveness of various treatment options.

STRUCTURE: ELBOW JOINT (HUMEROULNAR AND HUMERORADIAL ARTICULATIONS)

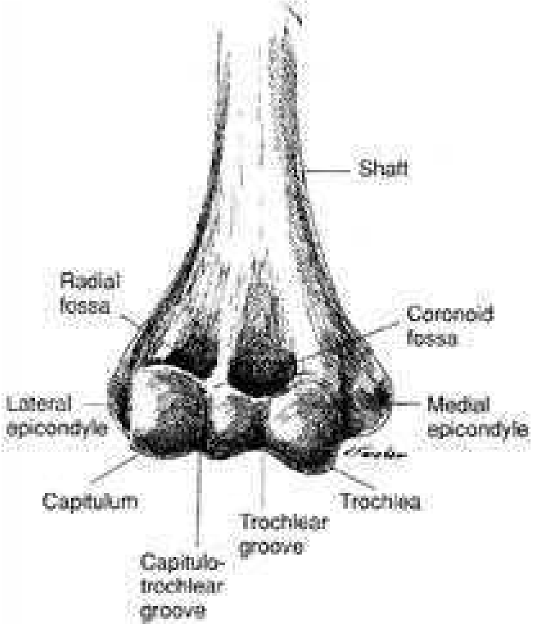
Articulating Surfaces on the Humerus

The articulating surfaces on the anterior aspect of the distal humerus are the hourglass-shaped trochlea and the spherical capitulum (Fig. 8–1). These structures are situated between the medial and lateral humeral epicondyles. The **trochlea,** which forms part of the humeroulnar articulation, is set at an angle on the medial aspect of the distal humerus and lies slightly anterior to the humeral shaft. A groove called the **trochlear groove** spirals obliquely around the trochlea and divides it into medial and lateral portions. The medial portion of the trochlea projects distally more than the lateral portion and results in a valgus angulation of the forearm called the *carrying angle*.

The indentation in the humerus located just above the trochlea is called the **coronoid fossa** and is designed to receive the coronoid process of the ulna at the end of the elbow flexion range of motion. The **capitulum,** which is part of the humeroradial articulation, is located on the anterior lateral surface of the distal humerus. The capitulum, like the trochlea, lies anterior to the shaft of the humerus. A groove called the **capitulotrochlear groove** separates the capitulum from the trochlea (see Fig. 8–1). The indentation located on the humerus just above the capitulum is called the **radial fossa** and is designed to receive the head of the radius in elbow flexion. Posteriorly, the distal humerus is indented by a deep fossa called the **olecranon fossa,** which is designed to receive the olecranon process of the ulna at the end of the elbow extension ROM (Fig. 8–2).

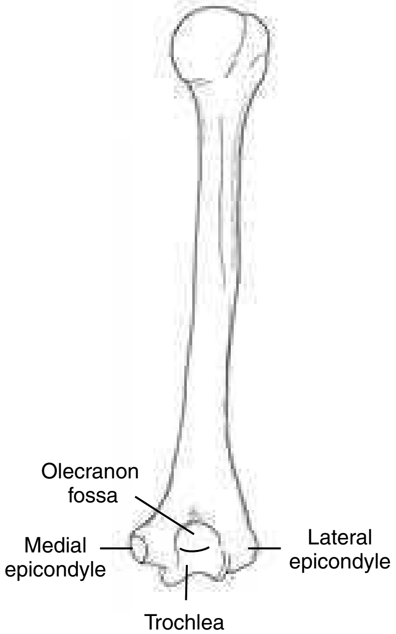
Articulating Surfaces on the Radius and Ulna

The articulating surfaces of the ulna and radius correspond to the humeral articulating surfaces (Fig. 8–3A and B). The ulnar articulating surface of the humeroulnar joint is a deep, semicircular, concave surface called the **trochlear notch**[3] (Fig. 8–4A and B). The proximal portion of the notch is divided into two unequal parts by the **trochlear ridge,**



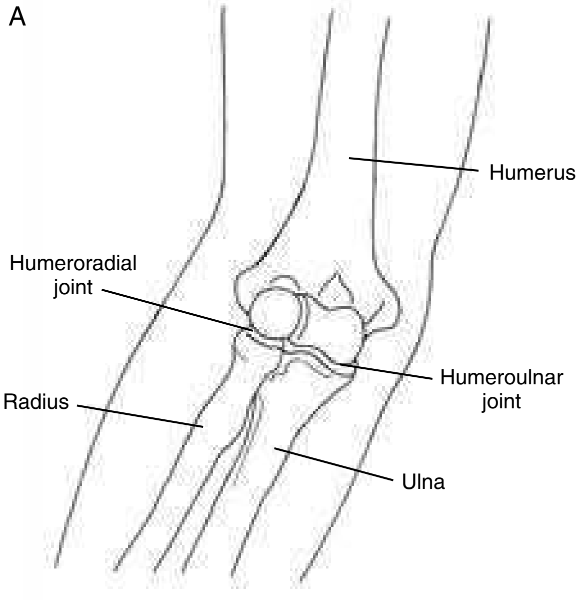
Anterior view

Figure 8–1 Articulating surfaces on the anterior aspect of the right distal humerus.

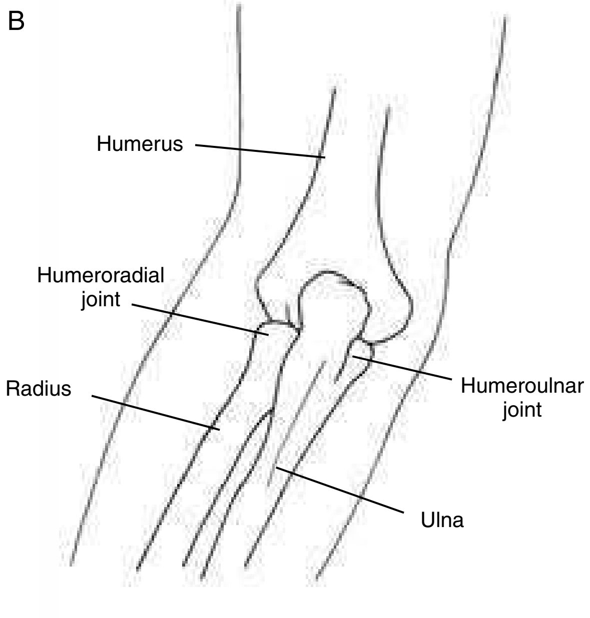


Right humerus
Posterior view

Figure 8–2 The olecranon fossa on the posterior aspect of the distal humerus.



Anterior view



Posterior view

Figure 8–3 **A.** An anterior view of the right elbow showing the humeroulnar and humeroradial joints. **B.** A posterior view of the right elbow showing the humeroulnar and humeroradial joints.

which corresponds to the trochlear groove on the humerus. The **ulnar coronoid process** forms the distal end of the notch, while the **olecranon process** projects over the proximal end of the notch (see Fig. 8–4B). The radial articulating surface of the humeroradial joint is composed of the proximal end of the radius, known as the **head of the radius** (Fig. 8–5A). The radial head has a slightly cup-shaped concave surface called the **fovea** that is surrounded by a rim (Fig. 8–5B). The radial head's convex rim fits into the **capitulotrochlear groove.**

Articulation

Articulation between the ulna and humerus at the humeroulnar joint occurs primarily as a sliding motion of the ulnar trochlear ridge on the humeral trochlear groove. In extension, sliding continues until the olecranon process enters the olecranon fossa (Fig. 8–6A). In flexion, the trochlear ridge of the ulna slides along the trochlear groove

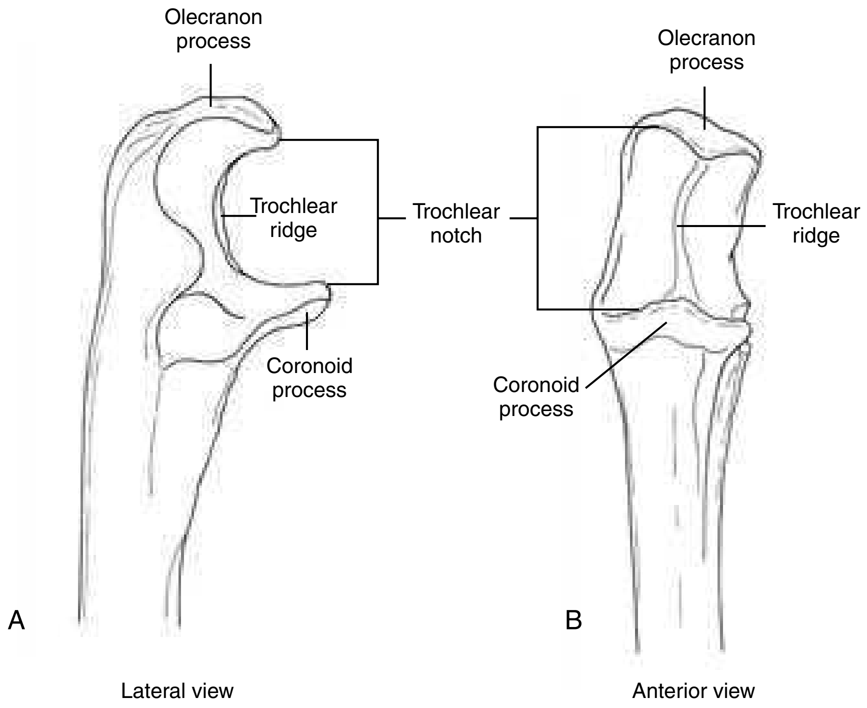


Figure 8–4 **A.** Lateral view of the trochlear notch and ridge. The coronoid process forms the distal end of the trochlear notch and the olecranon process projects over the proximal end. **B.** Anterior view of the trochlear notch and ridge.

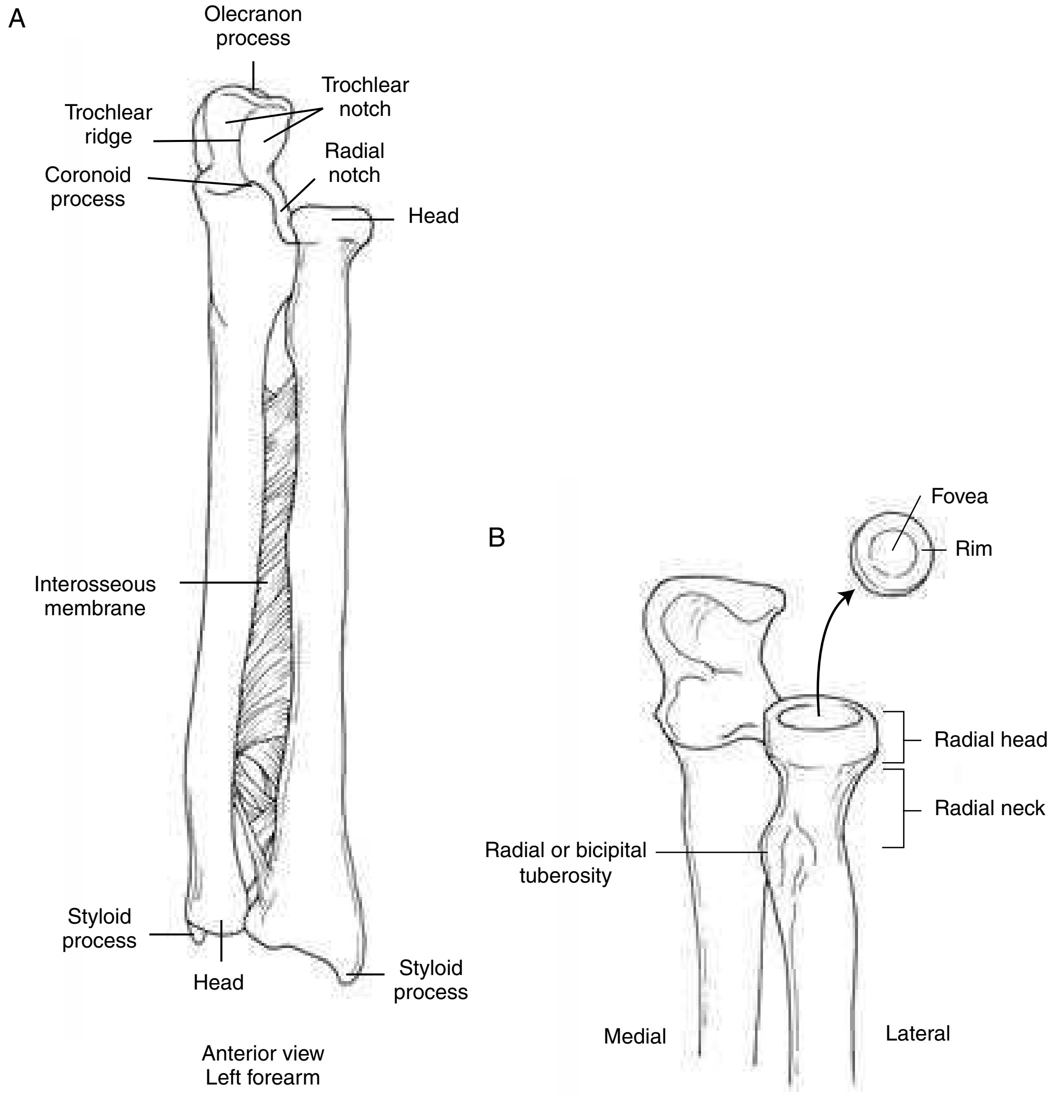


Figure 8–5 **A.** Head of the radius. **B.** Fovea and rim.

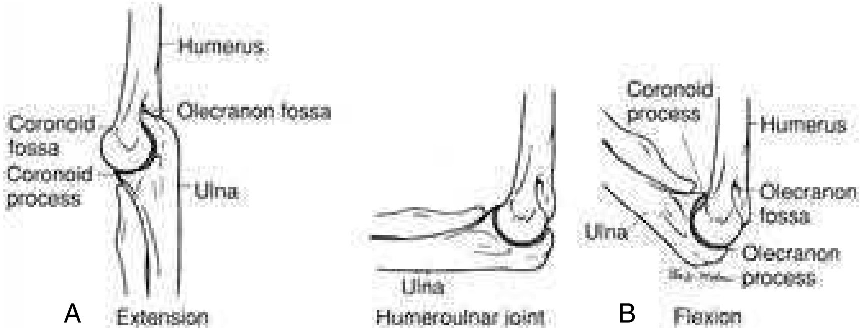


Figure 8–6 Schematic representation of motions of the ulna on the humerus at the humeroulnar joint. **A.** In extension, the olecranon process enters the olecranon fossa. **B.** In flexion, the coronoid process reaches the coronoid fossa.

until the coronoid process reaches the floor of the coronoid fossa in full flexion[3] (Fig. 8–6B).

Although the opposing articulating surfaces of the trochlea and trochlear notch appear to be completely congruent, experiments with cadaveric specimens have shown that the articulating surface of the trochlea does not contact the articulating surface at the bottom center of the notch unless the joint is heavily loaded.[4–7]

For example, Eckstein and colleagues[4] determined that at loads of 25 N (simulating resisted elbow extension), with the elbow positioned at 30° increments up to 120° of flexion, no surface contact occurred between the trochlea and the depths of the trochlear notch in all six elbow specimens studied (Fig. 8–7A). At a load of 500 N (approximately 112 lb), the articulating surface contact areas expanded from the sides toward the depths of the notch (Fig. 8–7B).

Articulation between the radial head and the capitulum at the humeroradial joint involves sliding the shallow concave radial head over the convex surface of the capitulum. The humeral capitulum is slightly smaller than the corresponding radial fovea, so the joint surfaces are slightly incongruent.[8] In full extension, no contact occurs between the articulating surfaces (Fig. 8–8A). In flexion, the rim of the radial head slides in the capitulotrochlear

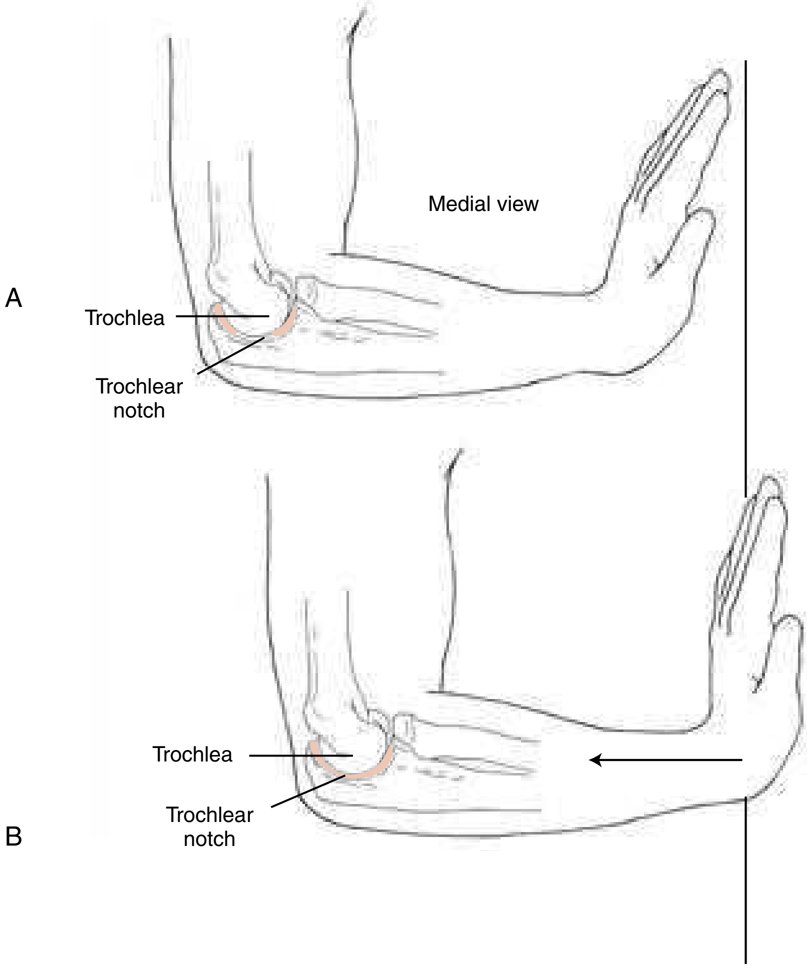


Figure 8–7 **A.** No surface contact occurs between the trochlea and the center of the troclear notch from 30° to 120° of flexion. Contact is primarily on the sides of the notch under no-load conditions. **B.** Contact areas expand from the sides toward the center when a load is applied.

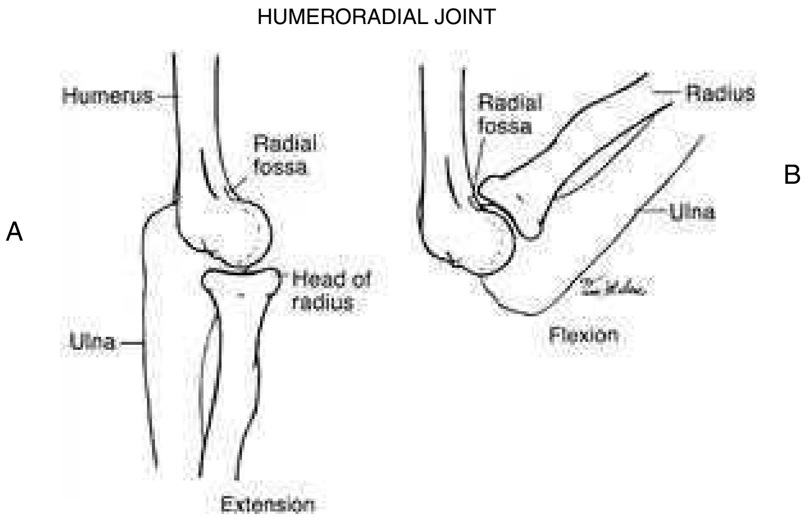


Figure 8–8 Schematic representation of motions of the radius at the humeroradial joint. **A.** In full extension, there is no contact between the capitulum and the radial head. **B.** During flexion, the rim of the radius slides in the capitulotrochlear groove, and in full flexion, it reaches the radial fossa on the humerus.

groove and enters the radial fossa as the end of the flexion range is reached[9] (Fig. 8–8B).

Joint Capsule

The humeroulnar, humeroradial, and superior radioulnar joints are enclosed in a single joint capsule. As shown in the anterior view in Figure 8–9, the proximal humeral attachment of the capsule is just above the coronoid and radial fossae. Distally, the capsule attaches into the ulna along the margin of the coronoid process and blends with the proximal border of the annular ligament. Medially and laterally, the capsule is continuous with the collateral ligaments.[3] Posteriorly, the capsule is attached to the humerus along the upper edge of the olecranon fossa and to the back of the medial epcondyle.[3] The capsule passes just below the annular ligament to attach to the posterior and inferior margins of the neck of the radius.[3]

The capsule is fairly large, loose, and weak anteriorly and posteriorly, and it contains folds that are able to expand to allow for a full range of elbow motion. Fat pads are located between the capsule and the synovial membrane adjacent to the olecranon, coronoid, and radial fossae.[3] Laterally and medially, the capsule is reinforced by the collateral ligaments.

The capsule's synovial membrane lines the coronoid, radial, and olecranon fossae. It also lines the flat medial trochlear surface and the lower part of the annular ligament. A triangular synovial fold inserted between the proximal radius and ulna partly divides the elbow joint into two joints.[3] Duparc and colleagues[10] found that in the majority of joints examined in 50 adult cadavers, the

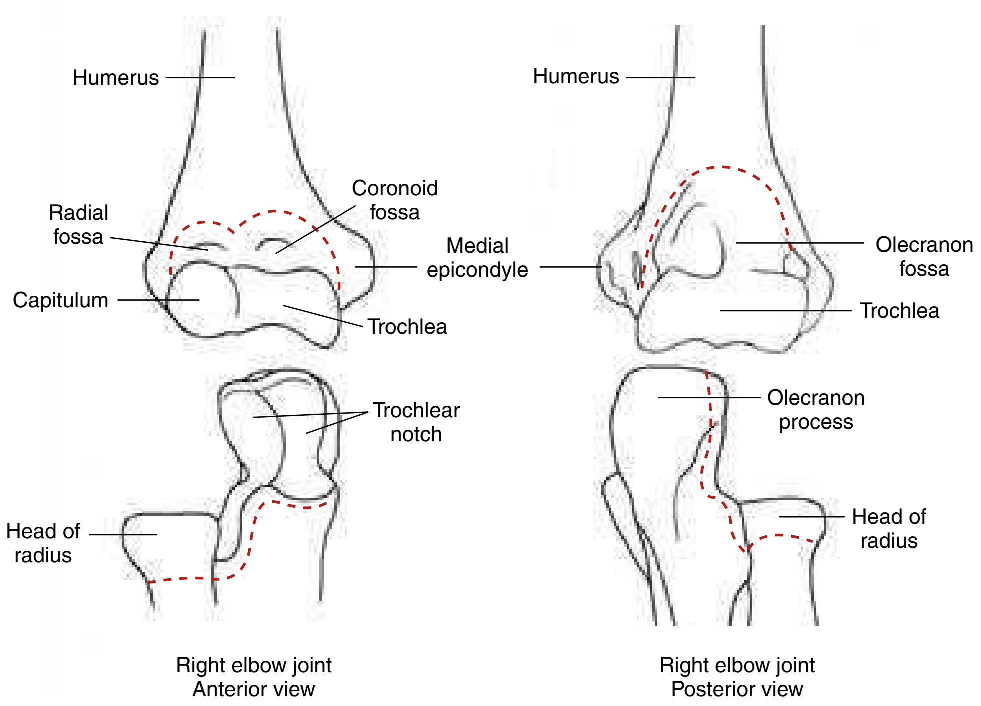


Figure 8–9 The red dashed lines show the anterior and posterior attachments of the elbow joint capsule.

triangular synovial fold was located at the proximal radioulnar joint near the junction of the annular ligament and the joint capsule. The synovial folds varied from 1 to 4 mm in thickness, from 9 to 51 mm in length, and contained fat pads and nerve fibers.[10]

CASE APPLICATION

Hypertrophied Synovial Folds

case 8–1

Hypertrophied triangular synovial folds have been identified as a source of pain in cases of lateral epicondylalgia (pain in the region of the lateral epicondyle).[10] Therefore, it is possible that the patient's lateral elbow pain arises from an irritation of the nerve fibers in a hypertophied triangular synovial fold.

Ligaments

Our knowledge of ligamentous functioning stems primarily from tests on cadaveric joint specimens that are subjected to various stresses. In one type of testing, intact ligaments are sectioned or transected to determine the effects of cutting on joint stability. In another type of testing, the length changes of intact ligaments are measured in different joint positions to determine when a ligament is providing stability. Researchers use the results of these types of ligamentous tests to make judgments about the role or roles that a particular ligament plays at a joint.

Most hinge joints in the body have collateral ligaments, and the elbow is no exception. Collateral ligaments are located on the medial and lateral sides of hinge joints to provide medial/lateral stability and to keep joint surfaces in apposition. The two main ligaments associated with the elbow joints are the **medial (ulnar)** and **lateral (radial) collateral** ligaments.

Medial (Ulnar) Collateral Ligament

The medial collateral ligament consists of either two parts, anterior and posterior,[11–13] or three parts, anterior, transverse, and posterior[14–17] (Fig. 8–10A). The anterior and posterior parts may be referred to as either the **anterior** and **posterior oblique ligaments** or simply **anterior** and **posterior bundles.** We will use the term *bundles* rather than *oblique ligaments*. The bundles are separated into anterior and posterior bands.[18,19] When the medial collateral ligament is dissected free from other structures, the proximal enthesis of the ligament is described as a round area on the flat portion of the anterior and inferior aspect of the medial humeral epicondyle.[18,20] Milz and colleagues[20] determined that the enthesis of the medial collateral ligament was essentially fused with the common flexor tendon as well as part of the humeral articular cartilage, where the ligament wraps around the articular cartilage of the edge of the trochlea.

The distal enthesis of the medial collateral ligament, which is on the coronoid process of the ulna, is broad proximally and tapers distally, with fibers of the ligament crossing the coronoid process and inserting further distally on the proximal and medial ulna.[18] This type of spread-out insertion at an enthesis is also a mechanism for distribution of stress.[21] Mechanoreceptors (Golgi organs, Ruffini terminals, Pacini corpuscles, and free nerve endings) are densely distributed near the ligament's humeral and ulnar attachments.[22]

The anterior bundle of the medial collateral ligament is considered to be the primary ligamentous restraint of valgus stress from 20° to 120° of elbow flexion.[23,24] It is overlaid by the **flexor carpi ulnaris, pronator teres,** and **flexor digitorum sublimus muscles.** Callaway and

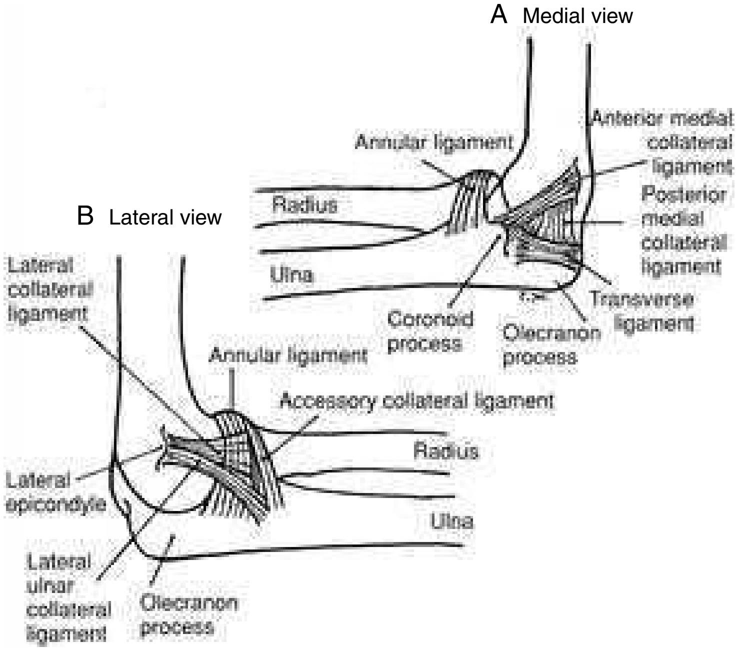


Figure 8–10 **A.** Three parts of the medial (ulnar) collateral ligament are shown on the medial aspect of the right elbow. The musculature and joint capsule have been removed to show the ligament's attachments. **B.** The lateral collateral ligament complex inlcudes the lateral (radial) collateral ligament, lateral ulnar collateral ligament, and annular ligament. The musculature and the joint capsule have been removed to show the ligaments' attachments.

colleagues[17] described the anterior bundle as consisting of an anterior band and a posterior band that tighten in a reciprocal manner as the elbow flexes and extends. The anterior band of the anterior bundle of the medial collateral ligament was found by these investigators to be the primary restraint of valgus at 30°, 60°, and 90° of flexion and the coprimary restraint up to 120°.[17] The anterior capsule and radial head also make contributions to medial joint stability, but these structures fail to provide stability against valgus stress when the anterior bundle is cut.[25]

The posterior bundle of the medial collateral ligament is not as distinct as the anterior bundle, and sometimes its fibers blend with the fibers from the medial portion of the joint capsule. The posterior bundle extends from the posterior aspect of the medial epicondyle of the humerus to attach to the ulnar coronoid and olecranon processes. It limits elbow extension but plays a less significant role than the anterior bundle in providing valgus stability for the elbow.[23,24] In Figure 8–10A, one can see that the oblique (transverse) fibers of the medial collateral ligament extend between the olecranon and ulnar coronoid processes. This portion of the ligament appears to provide little valgus stability but may help to keep the joint surfaces in approximation.

Safran[26] investigated the effects of both elbow flexion and forearm rotation on valgus laxity in 12 intact cadaveric specimens with the forearm in neutral, supination, and pronation at 30°, 50°, and 70° of flexion. The greatest degree of valgus laxity occurred with the forearm in neutral rotation at all elbow flexion angles tested. Repeated valgus stress on the medial collateral ligament occurs during many activities, but injury to the ligament occurs most frequently during the backswing portion of pitching, with the shoulder in external rotation and the elbow flexed.

Concept Cornerstone **8-1**

Functional Summary for Medial (Ulnar) Collateral Ligament

1. Stabilizes against valgus torques at the medial elbow[17,23–27]
2. Limits extension at the end of the elbow extension range of motion[11,12]
3. Guides joint motion throughout flexion range of motion[11]
4. Provides some resistance to longitudinal distraction of joint surfaces.

Lateral Collateral Ligamentous Complex

The **lateral collateral ligamentous complex** includes the **lateral (radial) collateral ligament (LCL),** the **lateral ulnar collateral ligament (LUCL),**[11,13,28] and the **annular ligament**[29] (Fig. 8–10B). The lateral radial collateral ligament is a fan-shaped structure that extends from the inferior aspect of the lateral epicondyle of the humerus to merge with the annular ligament (the ligament encircling the head of the radius). Ligamentous tissue extending from the lateral humeral epicondyle to the lateral aspect of the ulna and the annular ligament is referred to as the *lateral ulnar collateral ligament*.[12] This ligament adheres closely to the supinator, extensor, and anconeus muscles that lie just posterior to the lateral collateral ligament.[30]

Continuing Exploration 8-1:

Effect of Forearm and Elbow Position on Posterolateral Elbow Stability

Milz[20] found that the entheses of the lateral collateral ligament complex and the common extensor tendon were fused at the lateral epicondyle. In a study of five unembalmed specimens, Wavreille[31] determined that the positions of both the forearm and the elbow affect the length of the lateral collateral ligaments and therefore affect the posterolateral stability of the elbow. For example, when the forearm was in neutral rotation, the lateral collateral ligament was long between 0° and 30° and at 90° of flexion. When the forearm was in pronation, the lateral collateral ligament was long at 0°, 60°, and 120° of flexion and short at about 30°. In contrast to the effects of forearm rotation on the lateral collateral ligament, forearm rotation had little effect on the length of the lateral ulnar collateral ligament. The anterior bundle of the lateral collateral ligament was observed to be longest at 90° and shortest between 120° and 150° of elbow flexion, where the posterior bundle was the longest.[31]

Certain functions are attributed to individual ligaments in the complex while other functions are thought to be the result of the entire complex. The lateral radial collateral ligament provides reinforcement for the humeroradial articulation, offers some protection against varus stress in some positions of the elbow, and assists in providing resistance to longitudinal distraction of the joint surfaces.[32] Some fibers of the lateral radial collateral ligament remain taut throughout the flexion ROM when either a varus or valgus moment is applied.[12] O'Driscoll[33] described the lateral radial collateral ligament as a key structure that is always disrupted in elbow dislocations.

Olsen and colleagues[32] concluded that the lateral radial collateral ligament is the primary soft tissue restraint and the lateral ulnar collateral ligament and the annular ligament are secondary restraints to combined forced varus and supination stresses and forced valgus stress. The investigators found that sectioning either the lateral ulnar collateral ligament or annular ligament resulted in either no or minor (2°) laxity during forced varus stress and supination and a 4° laxity in forced valgus stress. However, sectioning of the lateral radial collateral ligament led to a maximum laxity of 15.4° during forced varus stress and supination and 23° in forced valgus stress.[34]

It appears that the lateral ulnar collateral ligament has the potential for assisting the lateral radial collateral ligament in resisting varus stress at the elbow and assisting in providing lateral support to the elbow joint.[35] Imatani and colleagues[30] suggested that the lateral ulnar collateral ligament is not a major restraint but contributes to posterolateral stability by securing the ulna to the humerus. Also, it may provide support to the annular ligament. Kim and associates[36] suggested that the lateral ulnar collateral ligament is not a static stabilizer but acts as a dynamic stabilizer together with related muscles.

Continuing Exploration 8-2:

Controversy Regarding the Roles of the Lateral Collateral Ligament, the Lateral Ulnar Collateral Ligament, and the Annular Ligament

Dunning and coworkers[37] found that when the annular ligament was intact, either the lateral collateral ligament or the lateral ulnar collateral ligament could be transected without causing posterolateral rotatory instability. In contrast, Hannouche and Begue[38] found that subluxation of the humeroulnar joint occurred when either the anterior and medial bundles of the lateral collateral ligament were sectioned at their humeral attachment or when the medial bundle and annular ligament were sectioned at their ulnar insertion. The researchers concluded that posterolateral stability is largely maintained by the anterior and medial bundles of the lateral collateral ligament and the annular ligament.

Concept Cornerstone **8-2**

Functional Summary for Lateral Collateral Ligamentous Complex

1. Stabilizes elbow against varus torque[28,29,32,34]
2. Stabilizes against combined varus and supination torques[28,29,34]
3. Reinforces humeroradial joint and helps provide some resistance to longitudinal distraction of the articulating surfaces[32]
4. Stabilizes radial head, thus providing a stable base for rotation[34]
5. Maintains posterolateral rotatory stability[29,30,35,37–39]
6. Prevents subluxation of humeroulnar joint by securing ulna to humerus
7. Prevents the forearm from rotating off of the humerus in valgus and supination during flexion from the fully extended position

CASE APPLICATION

Lateral Collateral Ligament Complex

case 8–2

It is possible that either the lateral collateral ligament or the lateral ulnar collateral ligament has been overstretched and incurred some microtrauma, which could be a source of the patient's pain. Both ligaments are attached to the joint capsule, which has free nerve endings and mechanoreceptors distributed near the humeral and ulnar capsular attachments.[22] Isolated tears of the lateral collateral ligament are uncommon, but chronic insufficiency may lead to symptomatic posterolateral joint subluxation.[39] Complete disruption of the lateral collateral ligament complex is most often seen in fracture dislocations and fractures involving the coronoid or radial head.[40] If the patient, James, was experiencing painful clicking or locking of the elbow, we would suspect posterolateral instability and conduct the appropriate manual tests, as well as request stress x-rays.[33] However, we have no reason to suspect posterolateral instability, a fracture, or a dislocation, because thr patient has not complained of any painful clicking or locking of the elbow. Therefore, we can probably rule out complete disruption of the lateral collateral ligament as a source of his pain. Furthermore, complete disruptions of the lateral ligaments usually occur as a result of a fall in which the radius is fractured, and our patient has no history of a fall. Some amount of stretching and microtrauma involving one of the structures in the lateral collateral ligament complex could still be present and difficult to diagnose because microsopic tears and partial disruption of fibers might not cause observable instability.

Muscles

Nine muscles cross the anterior aspect of the elbow joint, but only three of these muscles (the brachialis, biceps brachii, and brachioradialis) have primary functions at the elbow joint. The supinator teres and pronator teres have major functions at the radiolunar joints. The remaining four muscles (flexor carpi radialis, flexor carpi ulnaris, flexor digitorum superficialis, and palmaris longus), which arise by a common tendon from the medial epicondyle of the humerus, have primary functions at other joints, including the wrist, hand, and fingers, and are considered to be weak flexors of the elbow (Fig. 8–11A).

The major flexors of the elbow are the **brachialis,** the **biceps brachii,** and the **brachioradialis.** The brachialis muscle arises from the anterior surface of the lower portion of the humeral shaft and attaches by a thick, broad tendon to the **ulnar tuberosity** and **coronoid process.** The biceps brachii arises from two heads, one short and the other long. The short head arises as a thick, flat tendon from the coracoid process of the scapula, and the long head arises as a long, narrow tendon from the scapula's supraglenoid tubercle. The muscle fibers arising from the two tendons unite in the middle of the upper arm to form the prominent muscle bulk of the upper arm. Muscle fibers from both heads insert by way of the strong flattened tendon on the rough posterior area of the tuberosity of the radius. Other fibers of the biceps brachii insert into the bicipital aponeurosis that extends medially to blend with the fascia that lies over the forearm flexors.[3] The brachioradialis muscle arises from the lateral supracondylar ridge of the humerus and inserts into the distal end of the radius just proximal to the radial styloid

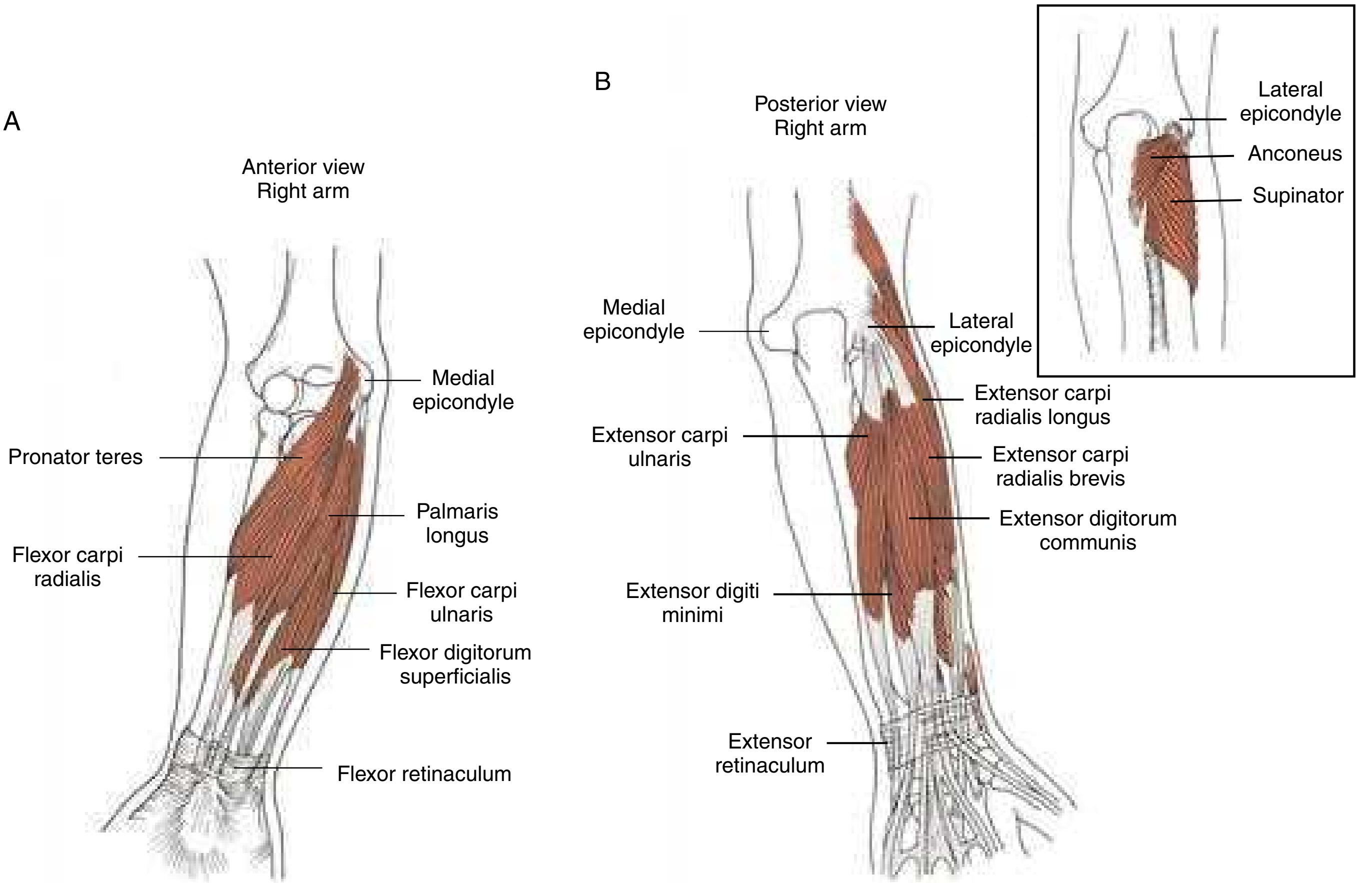


Figure 8–11 **A.** Insertion of the flexor muscles on the medial epicondyle of the humerus. **B.** Insertion of the extensor muscles on the lateral epicondyle of the humerus.

process. Both the biceps and brachioradialis insert into the radius.

The two extensors of the elbow are the triceps brachii and the anconeus. The **triceps brachii** has three heads: long, medial, and lateral. The long head crosses both the glenohumeral joint at the shoulder as well as the elbow joint. It arises from the infraglenoid tubercle of the scapula by a flattened tendon that blends with the glenohumeral joint capsule. The medial and lateral heads cross only the elbow joint. The medial head covers an extensive area as it arises from the entire posterior surface of the humerus. In contrast, the lateral head arises from only a narrow ridge on the posterior humeral surface. The three heads insert via a common tendon into the olecranon process. The **anconeus** is a small triangular muscle that arises from the posterior surface of the lateral epicondyle of the humerus and extends medially to attach to the lateral aspect of the olecranon process and the adjacent proximal quarter of the posterior surface of the ulna[3] (see Fig. 8–11).

In addition to the anconeus muscle, a number of muscles with primary actions at the wrist and fingers insert into the lateral humeral epicondyle by way of a common extensor tendon. These muscles include the **extensor carpi radialis longus, extensor carpi radialis brevis, extensor digitorum communis, extensor carpi ulnaris,** and **extensor digiti minimi** (see Fig. 8–11B).

CASE APPLICATION

Muscles and Tendons

case 8–3

The extensor carpi radialis longus and brevis and the extensor carpi ulnaris are active in gripping, hammering, and sawing activities. Therefore, the repetitive pull of these muscles during the patient's workday could have injured the common extensor tendon or another tendon, either in its substance or at the enthesis. The muscles also could have been strained. Therefore, it is possible that James's elbow pain could come from (1) the common extensor tendon, (2) the tendon's attachment site (enthesis) on the lateral humeral epicondyle, or (3) one of the muscles. Disorders at the enthesis (enthesopathies) are commonly seen in tennis elbow and jumper's knee.[41] Also, because James has had numerous episodes of similar pain over a number of years, we might suspect that this is a chronic condition and that, therefore, there may be degenerative changes affecting the tendon if it is the source of pain.

The types of changes that might be seen in either our patient's tendon or his muscle are presented in Table 8–1. These changes are from examinations of biopsy material from patients with chronic lateral elbow pain previously diagnosed as lateral epicondylitis (inflammation of the lateral epicondyle and surrounding tissues). A shift in understanding of the disease process involved in tennis elbow has led to the diagnosis of epicondylitis being abandoned by some and replaced with a diagnosis of epicondylosis or insertional tendinopathy.[46,47] The reason for this change in diagnosis is that many of the changes that appear in Table 8–1 appear to be more suggestive of a degenerative process than of a simple inflammatory process and are similar to changes observed in aged supraspinatus tendons.[42,46]

According to Benjamin,[21] fibrocartilagenous tendon insertional problems rarely show evidence of inflammation; instead, tendons show thinning, disruption of collagen fibers, increased vascularity and cellularity, granulation tissue, and increased proteoglycan content in the extracellular matrix. Faro and Wolf [46] suggested that a failed reparative process might be the cause of the tissue changes, but they indicated that our understanding of the exact disease process involved is still incomplete. Coombs[48] also confirmed that the process is noninflammatory, but he could not differentiate between a degenerative pathological process and a dysfunctional healing process. However, Coombs and colleagues proposed a model for tennis elbow that consists of local tendon pathology, changes in the pain system, and impairment in the motor system. [48]

The fact that the enthesis of the lateral ligament and the common extensor tendon may be fused makes it difficult to identify which structure is involved (muscle, tendon, ligament). Mackay and colleagues[49] used magnetic resonance imaging (MRI) to identify signs of edema, thickening, and tears in common extensor tendons and peritendon edema in patients with lateral elbow pain. Savnik[50] and associates, also using MRI, found separation of the extensor radialis brevis tendon from the lateral collateral ligament. Edema is more suggestive of an inflammatory process than are some of the changes identified in Table 8–1.

FUNCTION: ELBOW JOINT (HUMEROULNAR AND HUMERORADIAL ARTICULATIONS)

Axis of Motion

Traditionally, the axis for flexion and extension has been described as a relatively fixed axis that passes horizontally through the center of the trochlea and capitulum and bisects the longitudinal axis of the shaft of the humerus[51,52] (Fig. 8–12). However, some studies have found that the axis is not as fixed as previously thought.[53–55]

An exact determination of the axis of motion at the elbow is important because of the need to position elbow

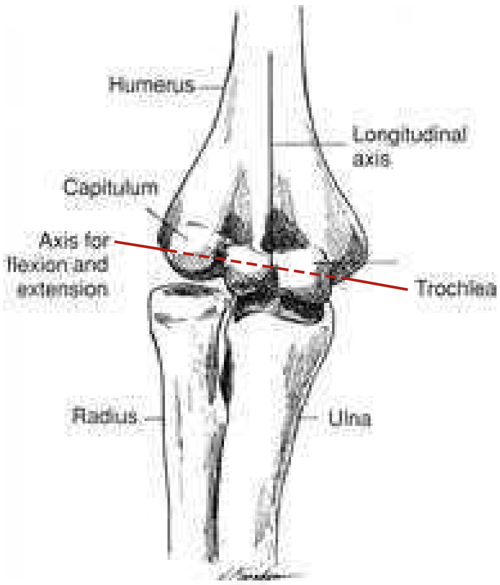


Figure 8–12 The axis of motion for flexion and extension. The axis of motion is centered in the middle of the trochlea on a line that intersects the longitudinal (anatomic) axis of the humerus.

Table 8–1 Tennis Elbow: Changes in Tendons and Muscles

CHARD ET AL[42]: BIOPSIES (N = 20 PATIENTS, 27–56 YR)	**GALLIANI ET AL[43]: BIOPSIES (N = 11 PATIENTS, 38–54 YR)**	**STEINBORN ET AL[44]: BIOPSIES (N = 23 PATIENTS, 29–58 YR)**	**LJUNG ET AL[45]: BIOPSIES (N = 20 PATIENTS)**
Common Extensor Tendon	*Common Extensor Tendon Insertion*	*Common Extensor Tendon*	*Extensor Carpi Radialis Brevis Muscle*
Loss of tenocytes	Loss of tenocytes	Fatty degeneration	Moth-eaten fibers
Calcification	Calcifying processes	Intratendinous cartilage formation	Fiber necrosis
Glycosaminoglycan infiltration	Biochemical and spatial degeneration of collagen	Fibrosclerotic degeneration	Fiber degeneration
Fibrocartilaginous transformation	Hyaline degeneration Fibrocartilage metaplasia	Fibrovascular proliferation	Increased percentage of fast-twitch type 2A fibers

prostheses in such a way that they correctly mimic elbow joint motion. In the past, elbow prostheses that were modeled as pure hinge devices often became loose during motion. Variations found in the instantaneous axis inclination support the hypothesis that activity of the various muscles may influence the pattern of motion during active flexion, and differences in contours of the joint surfaces may explain interindividual differences during passive motion.[53] Intraindividual and interindividual variations in the axes appear to be greater in the frontal plane than in the horizontal plane.[53]

For example, Ericson and coworkers,[53] using a radiostereometric analysis technique and x-rays taken at 30° intervals up to 120° of active elbow flexion, found that the orientation of the instantaneous axes varied (between subjects) within the arc of flexion from 2.1° to 14.3° in the frontal plane and from 1.6° to 9.8° in the horizontal plane. However, the inclination of the mean axis in the horizontal plane differed little from a line through the centers of the trochlea and capitulum (Fig. 8–13).

Bottlang and colleagues[55] found that the envelope for valgus-varus laxity was greatest between 0° and 40° of flexion and decreased considerably when flexion exceeded 100°. Like Ericson and coworkers,[53] however, Bottlang and colleagues[55] found that all instantaneous rotation axes nearly intersected on the medial facet of the trochlea.

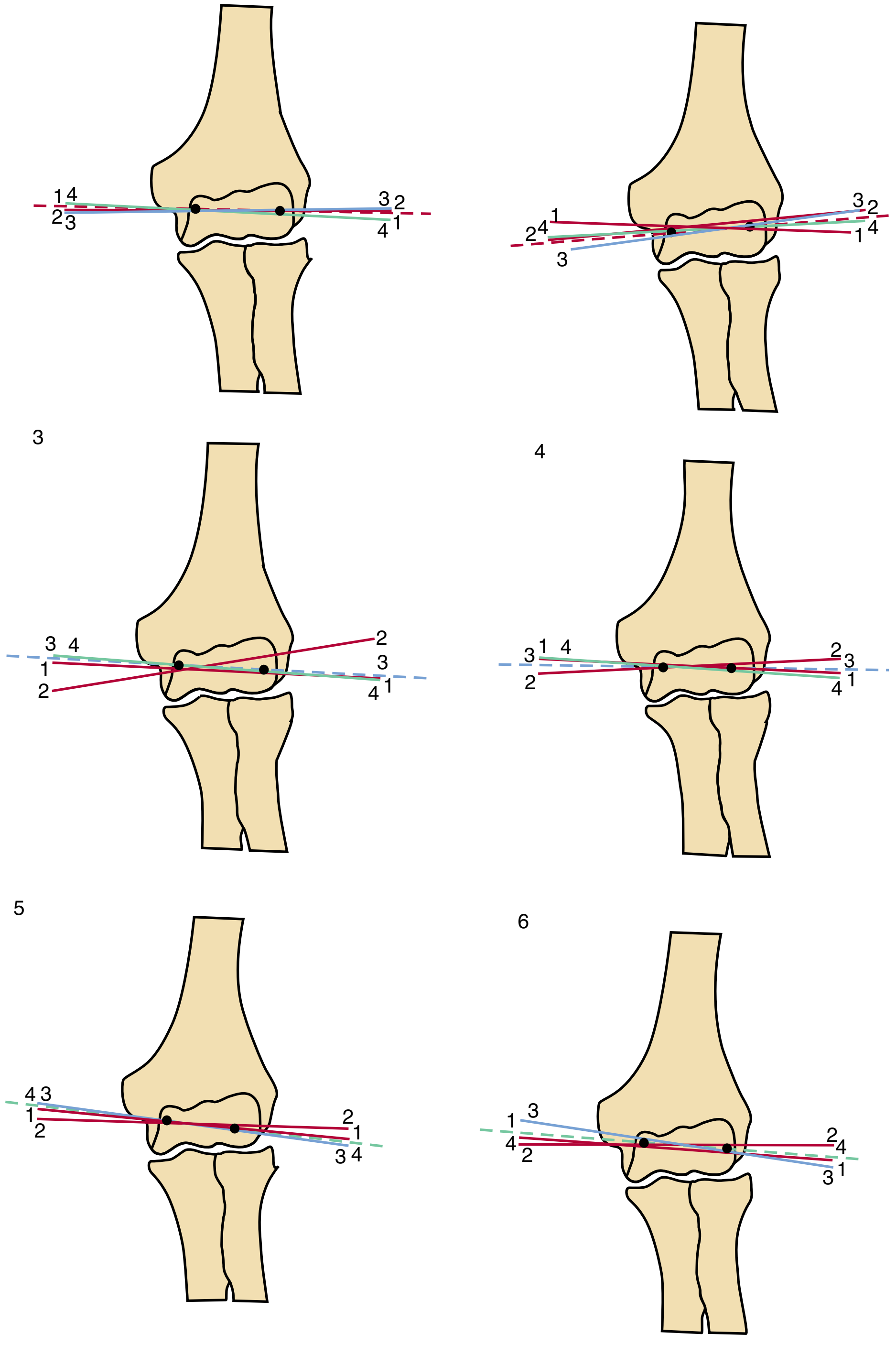


Figure 8–13 Variations in the instantaneous axis for flexion and extension. (*From Ericson A, Arndt A, Stark A, et al: Variation in the position and orientation of the elbow flexion axis. J Bone Joint Surg Br 85:539, 2003.*)

Long Axes of the Humerus and Forearm

When the upper extremity is in the anatomical position (shoulder in external rotation, elbow in extension and fully supinated), the long axis of the humerus and the long axis of the forearm form an acute angle medially when they meet at the elbow. The angulation in the frontal plane is caused by the configuration of the articulating surfaces at the humeroulnar joint. The medial aspect of the trochlea extends more distally than does the lateral aspect, which shifts the medial aspect of the ulna trochlear notch more distally and results in a lateral deviation (or valgus angulation) of the ulna in relation to the humerus. This normal valgus angulation is called the **carrying angle** or **cubitus valgus** (Fig 8–14A).

Functional use of the carrying angle results from a combination of shoulder lateral rotation, elbow extension, and forearm supination, which enables a person to carry a bucket in one hand in such a manner as to avoid contact between the carried load and lower limb on the same side. This position also helps in leading the hand towards a position above the center of mass of the weight.[56]

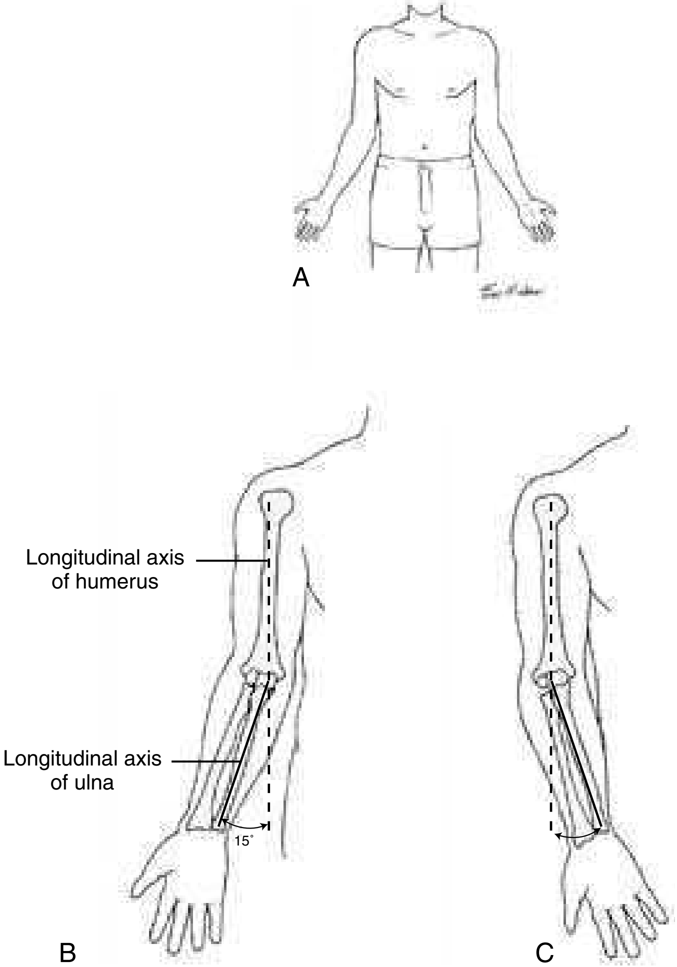


Figure 8–14 The carrying angle of the elbow. **A.** The forearm lies slightly lateral to the humerus when the elbow is fully extended in the anatomic position. **B.** The long axis of the humerus and the long axis of the forearm form the carrying angle. **C.** Cubitus varus.

Khare[57] found that in the first year of life there is no difference in the carrying angle between males and females. However, the mean carrying angles have been found to be significantly larger in female children than in male children in the same age group.[57–60] As shown in Table 8–2, the carrying angle is larger in young adult females than in males[61] and in other age groups. An overall increase in the carrying angle and a considerable amount of variability in the angle occurs up to age 14 or 15, which corresponds to the age of epiphysis closure around the elbow.[60,62] After age 15, the angle appears to decrease slightly but shows less variability. The angle in full elbow extension is generally about 15° but may vary from about 8° to 15° (Fig. 8–14B). Yilmaz[62] found a slight but significant difference in the carrying angle between dominant and nondominant arms, with the dominant arm having a slightly larger angle than the nondominant arm. An increase in the carrying angle valgus beyond the average is considered to be abnormal, especially if it occurs unilaterally. A varus angulation at the elbow is referred to as **cubitus varus** and is usually abnormal (Fig. 8–14C).

Continuing Exploration 8-3:

Why Females Have Larger Carrying Angles Than Males

Khare[57] has proposed that the proximal end of ulna angulates more and the medial flange of the trochlea grows longer in a shorter person than in a taller person. Consequently, the shorter the forearm bones, the greater the carrying angle. Because the average height of women is usually less than that of men, the carrying angle is greater in women than it is in men. Zampagni and colleagues[63] measured flexion as well as the carrying angle and found that the flexion angle was a significant factor in carrying angle values: the mean flexion angle of 5.26° in women was significantly smaller than that in men, indicating that women reach a greater natural physiological extension than men, leading to a larger carrying angle. Golden[59] measured 600 elbows in a pediatric population (4 months–18 years) and found that girls had a larger ROM in extension than boys and that the increased elbow extension ROM in girls could account for girls' larger carrying angle.

Normally, the carrying angle disappears when the forearm is pronated and the elbow is in full extension and when the supinated forearm is flexed against the humerus in full elbow flexion.[9] Van Roy found statistically significant differences in the mean carrying angles in men and women from 0° to 30° of flexion, where the carrying angle disappears at flexion angles beyond 30°.[56]

The configuration of the trochlear groove determines the pathway of the forearm during flexion and extension. In the most common configuration of the groove, the ulna is guided progressively medially from extension to flexion, so that in full flexion, the forearm comes to rest in the same plane as the humerus[9] (Fig. 8–15A). In extension,

Table 8–2 Carrying Angle

	POPULATION	AGE	*N*	GENDER	MEANS	RANGE
KHARE[57]	Healthy	0–20 yr	2,050	—	13.6 M, 17 F	—
GOLDEN[59]	Healthy pediatric population	4 mo–18 yr	n-300 (600 elbows)	155 M, 145 F	9.3 M, 11.5 F	—
GOLDEN[58]	Healthy	2–18 yr	113 (226 elbows)	73 M, 40 F	9.4 M, 11.0 F	—
BALASUBORAMIAN[60]	South Indian children	5–18 yr	300	150 M, 150 F	10.8 M, 12.9 F	—
PARASKEVSAS[61]	Healthy students	15–28 yr	600	320 M, 280 F	11.0 M, 15.1 F	3–19 M, 3–25 F
VAN ROY[56]	Healthy volunteers	20–46 yr	20 (40 elbows)	10 M, 10 F	11.6 M, 16.7 F	4–15 M, 12–21 F
ZAMPAGNI[63]	Healthy adults	45–81 yr	28	15 M, 13 F	11.2 M, 11.8 F	—
YILMAZ[62]	Healthy volunteers	2–91yr	1,275	631 M, 644 F	10.5 M, 12.0 F*	—

* Right arm dominant.

the forearm moves laterally until it reaches a position slightly lateral to the axis of the humerus in full extension. Variations in the direction of the groove will alter the pathway of the forearm, so that when the elbow is passively flexed, the forearm will come to rest either medial[9,51] (Fig. 8–15B) or lateral (Fig. 8–15C) to the humerus[9] in full flexion.

Mobility and Stability

A number of factors determine the amount of motion that is available at the elbow joint. These factors include the **type of motion (active or passive),** the **position of the forearm (relative pronation-supination), body mass index,** and the **position of the shoulder.** The range of active flexion at the elbow is usually less than the range of passive motion, because the bulk of the contracting flexors on the anterior surface of the humerus may interfere with the approximation of the forearm with the humerus. The active range of motion for elbow flexion with the forearm supinated is typically considered to be from about 135° to 145°, whereas the range for passive flexion is between 150° and 160°.[9] The position of the forearm also affects the flexion range of motion. When the forearm is either in pronation or in neutral (midway between supination and pronation), the range of motion is less than it is when the forearm is supinated. A body mass index (BMI) that is high (indicating overweight) has been identified as a factor limiting elbow range of motion; however, this finding is controversial. Golden[58] measured the range of motion of 226 elbows in 113 healthy children between the ages of 2 and 18. The authors found a decrease in elbow range of motion of 2° to 3° for each Z score increase (indicating an increase in weight)—so that an AZ body mass index score of 5.6 would limit the elbow range of motion by 11° to 17°.

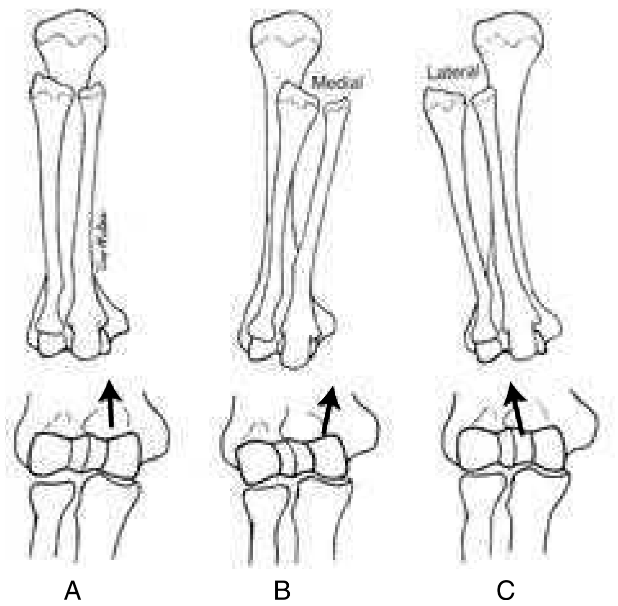


Figure 8–15 Position of the forearm in passive flexion. **A.** In the most common configuration of the trochlear groove, the ulna is guided progressively medially from extension to flexion so that in full flexion the forearm comes to rest in the same plane as the humerus. **B.** The forearm comes to rest slightly medially to the humerus in passive flexion. **C.** The forearm comes to rest slightly laterally in the least common configuration of the trochlear groove.

The position of the shoulder may affect the range of motion available to the elbow because of the two joint muscles that cross both the shoulder and elbow. These muscles, the biceps brachii and the triceps brachii, may limit range of motion at the elbow if a full range of motion is attempted at both joints simultaneously.

Concept Cornerstone **8-3**

Two-Joint Muscle Effects on Elbow Range of Motion

Two or multijoint muscles do not have sufficient length to allow a simultaneous full range of motion at all joints crossed. For example, in **passive motion,** passive tension in the triceps brachii may limit full elbow flexion when the shoulder is simultaneously moved into full flexion (Fig. 8–16A). In **active motion,** torque produced by the long head of the biceps brachii may diminish as the muscle is excessively shortened over both joints in full active shoulder and elbow flexion (Fig. 8–16B).

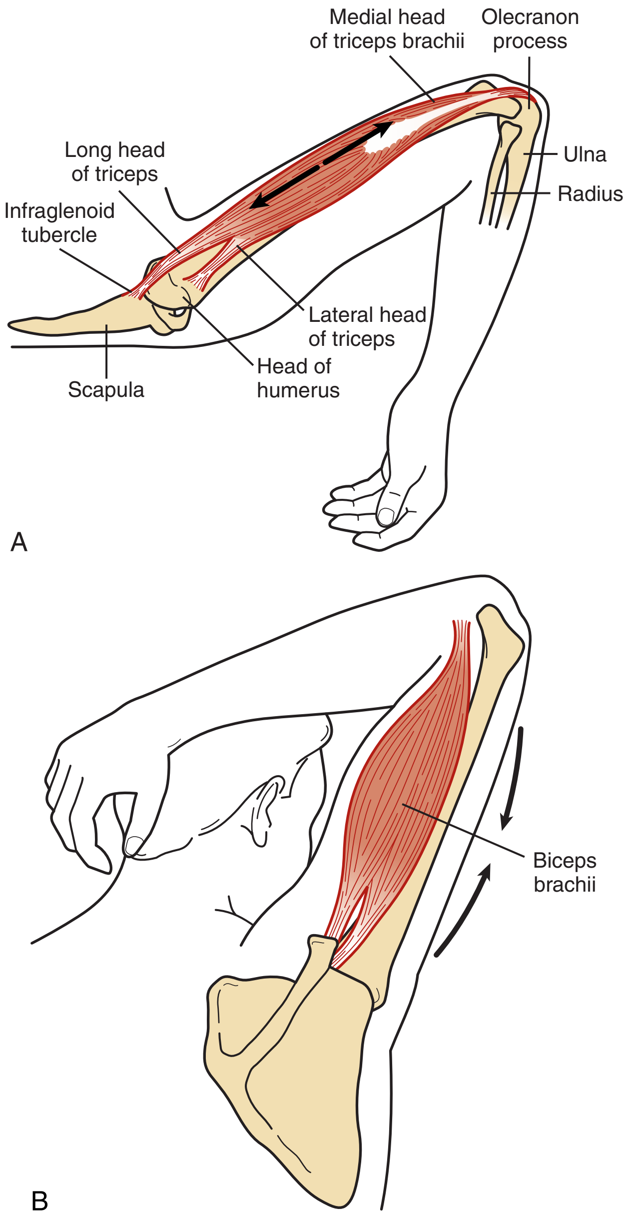


Figure 8–16 **A.** Passive tension created in the long head of the triceps by passively stretching the muscle over both the shoulder and elbow joints may limit elbow flexion. **B.** Active tension in the long head of the biceps brachii may drcrease and therefore limit the active range of motion in elbow flexion because the muscle is excessively shortened by contracting over both the elbow and shoulder.

Other factors that limit the range of motion but help to provide stability for the elbow are the configuration of the joint surfaces, the ligaments, and the joint capsule. The elbow has inherent articular stability at the extremes of extension and flexion.[24,32] In full extension, the humeroulnar joint is in a **close-packed position.** In this position, bony contact of the olecranon process in the olecranon fossa limits the end of the extension range, and the configuration of the joint structures helps provide valgus and varus stability. The bony components, medial collateral ligament, and anterior joint capsule contribute equally to resist valgus stress in full extension.[32] The flexor carpi ulnaris and the pronator teres overlay the anterior bundle of the medial collateral ligament and contribute to medial support of the elbow.[19] The bony components provide half of the resistance to varus stress in full extension, and the lateral collateral complex and joint capsule provide the other half of the resistance.[32] Resistance to joint distraction in the extended position is provided entirely by soft tissue structures. The anterior portion of the joint capsule provides the majority of the resistance to anterior displacement of the distal humerus out of the trochlear notch; the medial and lateral collateral ligaments contribute only slightly.[24,32]

Approximation of the coronoid process with the coronoid fossa and of the rim of the radial head in the radial fossa limits extremes of flexion. In 90° of flexion, the anterior part of the medial collateral ligament provides the primary resistance to both distraction and valgus stress. If the anterior portion of the medial collateral ligament becomes lax through overstretching, medial instability will result when the elbow is in flexed positions. The majority of the resistance to varus stress when the elbow is flexed to 90° is provided by the osseous structures of the joint; only a slight amount is provided by the lateral collateral ligament and the joint capsule. The anterior joint capsule contributes only slightly to varus and valgus stability and provides little resistance to distraction when the elbow is flexed.[24,32] Co-contractions of the flexor and extensor muscles of the elbow, wrist, and hand help to provide stability for the elbow during forceful motions of the wrist and fingers and in activities in which the arms are used to support the body weight. During pulling activities, such as when a person grasps and attempts to pull a fixed rod toward the body, the elbow joints are compressed by the contractions of muscles that cross the elbow and act on the wrist and hand.[64]

Swelling and/or pain also may limit the range of elbow motion. McGuigan and Bookout[65] investigated the effects of intra-articular fluid on range of motion. They found that the flexion arc of motion decreased 2.1° for every millimeter of injected fluid.

Muscle Action

A great deal of our information regarding muscle action comes from studies using electromyography (EMG). This

CASE APPLICATION

Swelling

case 8–4

When we first met James, he was holding his right elbow in slight flexion. It is possible but not probable that excess fluid has accumulated within the joint, which could stretch the joint capsule and cause pain. To reduce the pain, patients will often assume a position in which stretching of the joint capsule is at a minimum. Elbow flexion of about 80° is considered to be the elbow position at which the least amount of tension is present in the joint capsule.[66] Therefore, it is a position of relative comfort for patients with interarticular swelling.

Concept Cornerstone **8-4**

Summary of Factors Affecting Elbow Muscle Activity

The role that the elbow muscles play in motion at the elbow is determined by a number of factors, including:

- Number of joints crossed by the muscle (one-joint or two-joint muscles)
- Physiological cross-sectional area (PCSA)
- Location in relation to joint axis
- Position of the elbow and adjacent joints
- Position of the forearm
- Magnitude of the applied load
- Type of muscle action (concentric, eccentric, isometric, isokinetic)
- Speed of motion (slow or fast)
- Moment arm at different joint positions
- Fiber types

technique is used to monitor the electrical activity that is produced by the firing of motor units. With electromyography, it is possible to determine the relative proportion of motor units that are firing in a particular muscle during a specific muscle contraction. In addition, the muscle activation patterns of agonists and antagonists, as well as synergistic activity among both agonists and antagonists, may be identified during the performance of different tasks.

Flexors

Elbow Flexors

The brachialis is considered a mobility muscle because its insertion is close to the elbow joint axis. Also, the muscle has a large strength potential in that it has a large physiological cross-sectional area (PCSA) and a large work capacity (volume).[1] Its moment arm (MA) is greatest at slightly more than 100° of elbow flexion,[67] at which point its ability to produce torque is greatest (Fig. 8–17). Because the brachialis is inserted on the ulna, it is unaffected by changes in the forearm position brought about by rotation of the radius. Being a one-joint muscle, it is not affected by the position of the shoulder. According to EMG studies, the brachialis muscle works in flexion of the elbow in all positions of the forearm, with and without resistance. It also is active in all types of contractions (isometric, concentric, and eccentric) during slow and fast motions.[68] The fact that the brachialis works in all conditions may be related to the finding that the central nervous system (CNS) appears to favor using one-joint muscles over two-joint muscles to perform an isometric activity.[69]

The **biceps brachii,** like the brachialis, is also considered to be a mobility muscle because of its insertion close to the elbow joint axis. The long head of the biceps brachii has the largest volume among the flexors, but the muscle has a relatively small physiological cross-sectional area.[1] The moment arm of the biceps is largest between 80° and 100° of elbow flexion, and therefore the biceps is capable of producing its greatest torque in this range[67] (Fig. 8–18A). The moment arm of the biceps is rather small when the elbow is in full extension, and most of the muscle force is translatory and toward joint compression (Fig. 8–18B). Therefore, the biceps is less effective as an elbow flexor when the elbow is fully extended than when the elbow is flexed to 90°. When the elbow is flexed beyond 100°, the translatory component of the muscle force is directed away from the elbow joint and therefore acts as a distracting or dislocating force.

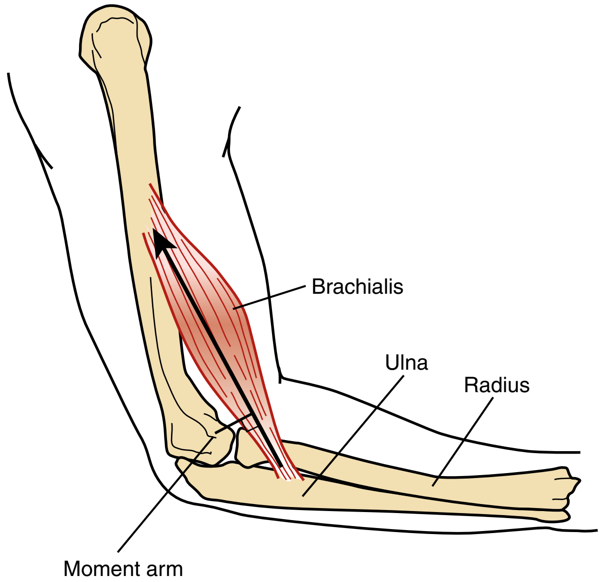


Figure 8–17 Moment arm of the brachialis at 100° of elbow flexion.

The functioning of the biceps is affected by the position of the shoulder because both heads of the muscle cross both the shoulder and the elbow. If full flexion of the elbow is attempted with the shoulder in full flexion, especially when the forearm is supinated, the muscle's ability to generate torque is diminished (see Fig. 8–16B). Also, the activation of the biceps was found to be significantly affected by elbow joint angle during concentric and isometric contractions but not during eccentric or isokinetic contraction.[70] In an EMG study of the flexors and extensors, the biceps brachii was active for supination torques.[71] Subjects in a study by Naito and associates were asked to maintain the elbow in flexion while performing alternating motions of supination and pronation at slow and fast speeds. EMG activity increased in the biceps during slow supination and decreased during pronation.[72]

The biceps brachii is active during unresisted elbow flexion with the forearm supinated and when the forearm is midway between supination and pronation in both concentric and eccentric contractions, but it tends *not* to be active when the forearm is pronated. However, when the magnitude of the resistance increases much beyond limb weight, the biceps is active in all positions of the forearm.[70] Bremer and colleagues[73] found that the moment arms of all major supinators exhibit peak values

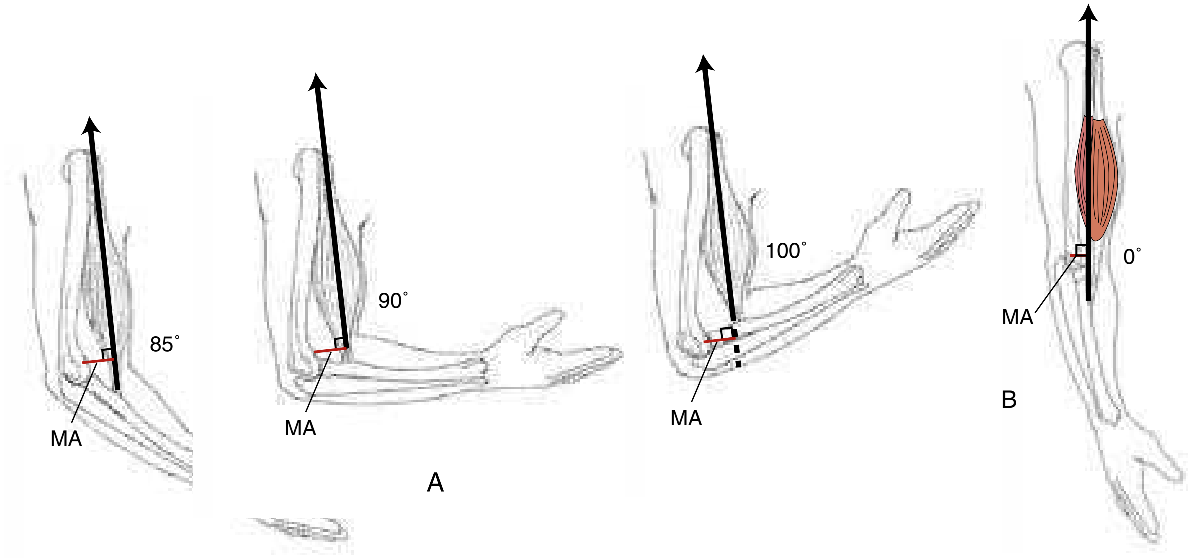


Figure 8–18 **A.** Moment arm (MA) of the biceps at 85° to 100° of elbow flexion. **B.** The moment arm of the biceps at full extension is relatively small, and most of the muscle force is toward joint compression.

in 40° to 50° of pronation. The biceps brachii and the supinator act as supinators through the entire range of forearm rotation.[73]

The **brachioradialis** is inserted at a distance from the joint axis, and therefore the largest component of muscle force goes toward compression of the joint surfaces and hence toward stability. The brachioradialis has a relatively small mean physiological cross-sectional area (1.2 cm) but a relatively large average peak moment arm (7.7 cm) in comparison with other elbow flexors.[67] The peak moment arm for the brachioradialis occurs between 100° and 120° of elbow flexion.[67] The brachioradialis does not cross the shoulder and therefore is unaffected by the position of the shoulder. The position of the elbow joint was found to affect brachioradialis muscle activity only during voluntary maximum eccentric contractions. Elbow joint angle had no effect on concentric, isometric, or isokinetic maximum voluntary contractions.[70]

The brachioradialis shows no electrical activity during eccentric flexor activity when the motion is performed slowly with the forearm supinated.[74] Also, the brachioradialis shows no activity during slow, unresisted, concentric elbow flexion. When the speed of the motion is increased, the brachioradialis shows moderate activity if a load is applied and the forearm is either in a position midway between supination and pronation or in full pronation.[68] In an electromyography experiment on the effects of forearm motion on muscle activity in which nine healthy subjects maintained their elbows in 90° flexion while pronating and supinating the forearm, the brachioradialis showed high levels of activity during rapid alternating supination/pronation motions. Higher levels of activity were noted when the forearm was pronated than when it was supinated.[72] Bremer suggested that the brachioradialis acts as a pronator in supination and as a supinator in pronation.[73]

The pronator teres, as well as the palmaris longus, flexor digitorum superficialis, flexor carpi radialis, and flexor carpi ulnaris, is a weak elbow flexor with primary actions at the radioulnar and wrist joints.[3]

Extensors

The effectiveness of the **triceps brachii** as a whole is affected by changes in the position of the elbow but not by changes in position of the forearm, because the triceps attaches to the ulna and not the radius. Activity of the long head of the triceps is affected by changing shoulder joint positions because the long head crosses both the shoulder and the elbow. The long head's ability to produce torque may diminish when full elbow extension is attempted with the shoulder in hyperextension. In this instance, the muscle is shortened over both the elbow and shoulder simultaneously.

The medial and lateral heads of the triceps, being one-joint muscles, are not affected by the position of the shoulder. The medial head is active in unresisted active elbow extension,[68] but all three heads are active when heavy resistance is given to extension or when quick extension of the elbow is attempted in the gravity-assisted position. Maximum isometric torque is generated at a position of 90° of elbow flexion.[75,76] However, the total amount of extensor torque generated at 90˚ varies with the position of the shoulder and the body.[77] The triceps is active eccentrically to control elbow flexion as the body is lowered to the ground in a push-up (Fig. 8–19A). The triceps is active concentrically to extend the elbow when the triceps acts in a closed kinematic chain, such as in a push-up (Fig. 8–19B). The triceps may be active during activities requiring stabilization of the elbow. For example, it acts as a synergist to prevent flexion of the elbow when the biceps is acting as a supinator. The other extensor of the elbow, the anconeus, assists in elbow extension and apparently also acts as a stabilizer during supination and pronation.

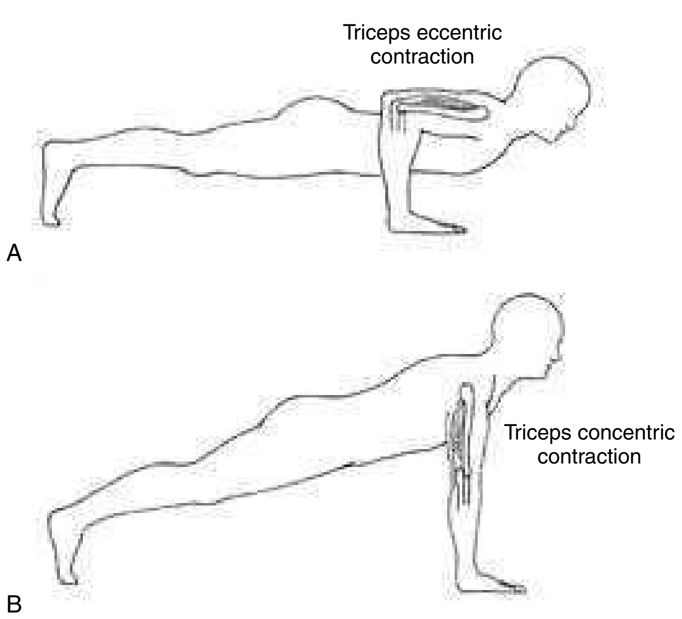


Figure 8–19 Action of the triceps in a push-up. **A.** The triceps muscle works eccentrically in reverse action to control elbow flexion during the lowering phase of a push-up. **B.** The triceps works concentrically in reverse action to produce the elbow extension that raises the body in a push-up.

Synergistic actions of elbow flexor and extensor muscles have been investigated during isometric contractions in response to a variety of stresses, including varus stress, valgus stress, flexion, and extension.[78] Some flexor muscle pairs, such as the brachialis and brachioradialis, and the extensor pairs of the anconeus and medial head of the triceps brachii are coactivated in a similar manner for all stresses. However, the synergistic patterns of other muscles at the elbow are complex and vary with the **joint angle, direction of the stress,** and the **type of muscle contraction.** For example, the brachialis and the long head of the biceps brachii work synergistically during isometric contractions only from 0° to 45° of flexion. In a no-load situation in which subjects held their elbows at 90° of flexion while they supinated and pronated their forearms, reciprocal activity among the elbow flexors permitted the biceps to work to produce supination without increasing the amount of elbow flexion.[72]

In addition to the fact that synergies are affected by the direction and variety of stress, synergistic activity also appears to be affected by the type of muscle contraction being used (isometric, concentric, eccentric).[72] Nakazawa and associates found that activation patterns in the biceps brachii and the brachioradialis varied with the type of muscle contraction during elbow flexion against a load.[74] The synergistic actions of the muscles of the elbow and their relation to elbow and wrist function are still being investigated. Dounskaia and coworkers, in an electromyographic study of arm cycling, suggested that a hierarchical organization of control for elbow-wrist coordination is operative in this activity. Muscles of the elbow are responsible for movement of the entire linkage, and the wrist muscles are responsible for making the corrections to the movement that were necessary to complete the task.[79] Zhang and Nuber[69] found that in voluntary isometric extension, the uniarticular lateral and medial heads of the triceps provide 70% to 90% of the total elbow extension moment. The anconeus muscle contributes about 15% of the extension moment. In contrast, the biarticular long head of the triceps contibutes significantly less. The authors of the study concluded that this was an example of the fact that the central nervous system selectively recruits uniarticular muscles rather than two-joint muscles to complete a task. In another study, Prodoehl and colleagues[80] demonstrated that muscle activation patterns change with the force requirements of the task and the amount of available mucle force.

Concept Cornerstone 8-5

Summary of Muscle Activation Patterns

Muscle activation patterns appear to be affected by the following:

1. Number of joints crossed
2. Type of muscle action (concentric, eccentric, isometric, isokinetic)
3. Speed of motion
4. Resistance
5. Requirements of the task
6. Direction of the stress
7. Activity of other muscles

STRUCTURE: PROXIMAL AND DISTAL RADIOULNAR ARTICULATIONS

Proximal (Superior) Radioulnar Joint

The articulating surfaces of the proximal radioulnar joint include the ulnar radial notch, the annular ligament, head of the radius, and the humeral capitulum. The **ulnar radial notch** is located on the lateral aspect of the proximal ulna directly below the trochlear notch (Fig. 8–20A). The surface of the radial notch is concave and covered with articular cartilage. A circular ligament called the **annular ligament** is attached to the anterior and posterior edges of the notch. The ligament is lined with articular cartilage, which is continuous with the cartilage lining of the radial notch. The annular ligament encircles the rim of the radial head, which is also covered with articular cartilage (Fig. 8–20B). Mechanoreceptors are evenly distributed throughout the ligament.[22] The capitulum and the proximal surface of the head of the radius are actually part of the elbow and have already been discussed in the section on the elbow joint.

Distal (Inferior) Radioulnar Joint

The articulating surfaces of the distal radioulnar joint include the ulnar notch of the radius, the articular disc, and the head of the ulna (Fig. 8–21). The **ulnar notch**

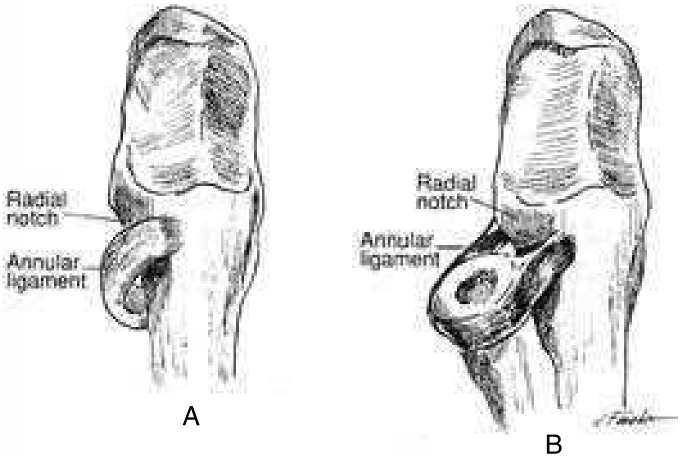


Figure 8–20 The annular ligament. **A.** Attachments of the annular ligament. **B.** The head of the radius has been pulled away from its normal position adjacent to the radial notch to show how the ligament partially surrounds the radial head.

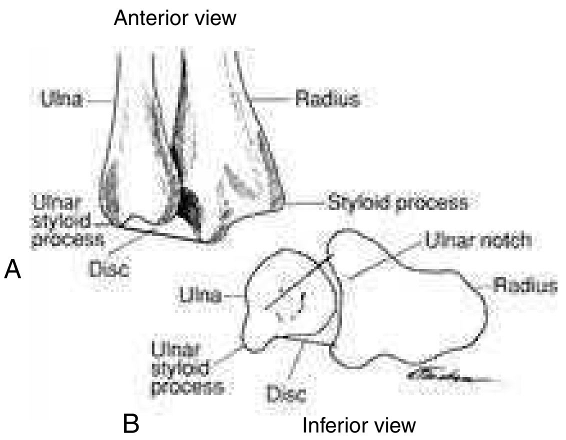


Figure 8–21 The inferior radioulnar joint of a left forearm. **A.** An anterior view of the inferior radioulnar joint shows the disc in its normal position in a supinated left forearm. **B.** An inferior view of the disc shows how the disc covers the inferior aspect of the distal ulna and separates the ulna from the articulation at the wrist.

of the radius is located at the distal end of the radius along the interosseous border. The radius of the curvature of the concave ulnar notch is larger than that of the ulnar head. The **articular disc** is sometimes referred to as either the **triangular fibrocartilage (TFC),** because of its triangular shape, or as a part of the **triangular fibrocartilage complex (TFCC),** because of its extensive fibrous connections.

The disc has been described as resembling a shelf whose medial border is embedded in a wedge of vascular connective tissue containing fine ligamentous bands that join the disc to the ulna and articular capsule.[81] The base of the articular disc is attached to the distal edge of the ulnar notch of the radius. The apex of the articular disc has two attachments. One attachment is to the fovea on the ulnar head. The other is to the base of the ulnar styloid process.[82,83] Medially, the articular disc is continuous with the fibers of the ulnar collateral ligament, which arises from the sides of the styloid process.[83] The margins of the articular disc are thickened[83,84] and are either formed by or are integral parts of the dorsal and palmar capsular radioulnar ligaments (Fig. 8–22). The ligaments are firmly attached to the radius; the ulnar attachments are somewhat less firmly attached. The thickness of the dorsal and palmar margins and of the apex of the disc is approximately 3 to 6 mm,[83,84] in contrast with the central area of the articular disc, which is often so thin that it is transparent.[83] Also, the central area may be perforated, and the number of perforations increases with age from 7% in the third decade to 53.1% in the sixth decade and beyond.[83] Chidgey and colleagues, in a study of 12 cadaver wrists, found that the articular discs had a high collagen content with sparsely but equally distributed elastin fibers. The same authors found that 80% of the central portion of the articular disc was avascular, in comparison with the peripheral area, which was only 15% to 20% avascular. The radioulnar ligaments were well vascularized.[85] Ohmori and Azuma found free nerve endings in the ulnar side of the articular disc, particularly around the periphery. The authors suggested that, in view of their findings, the disc may be a source of wrist pain.[86]

The articular disc has two articulating surfaces: the proximal (superior) surface and the distal (inferior) surface. The proximal surface of the disc articulates with the ulnar head at the distal radioulnar joint, whereas the distal surface articulates with the carpal bones as part of the radiocarpal joint.[3] Both the proximal and distal surfaces of the articular disc are concave. The superior surface of the articular disc is deepened to accommodate the convexity of the ulnar head; the distal surface is adapted to accommodate the carpal bones.[83] The peripheral parts of both the ulnar and carpal disc surfaces are covered by synovium from their respective joint capsules.[83]

The **ulnar head** is convex and is covered with articular cartilage distally.[87,88] The head has two articular surfaces, the pole and the seat, which articulate with the articular disc and the ulnar notch of the radius, respectively. The convex

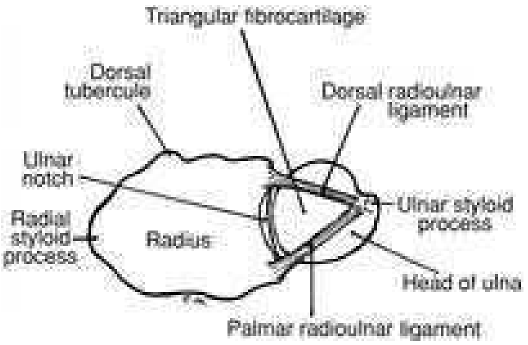


Figure 8–22 The illustration includes the distal aspects of a left radius and ulna, as well as the articular disc and articulating surfaces of the distal radioulnar joint. The articular disc is shown with the dorsal and palmar radioulnar ligaments bordering the sides of the disc.

pole is U-shaped and faces the disc. The convex seat faces the ulnar notch of the radius.[82]

Articulations

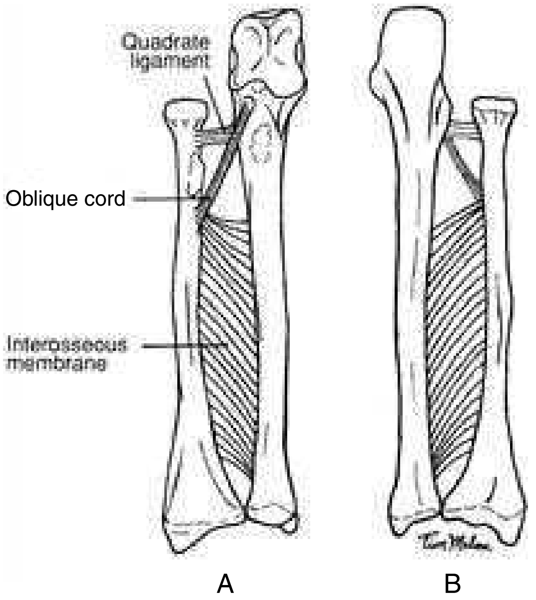
The proximal and distal radioulnar joints are mechanically linked; therefore, motion at one joint is always accompanied by motion at the other joint. The distal radioulnar joint is also considered to be functionally linked to the wrist in that compressive loads are transmitted through the distal radioulnar joint from the hand to the radius and ulna.[88]

Pronation of the forearm occurs as a result of the radius's crossing over the ulna at the superior radioulnar joint. During pronation and supination, the rim of the head of the radius spins within the osteoligamentous enclosure formed by the radial notch and the annular ligament. At the same time, the surface of the head spins on the capitulum of the humerus. At the distal radioulnar joint, the concave surface of the ulnar notch of the radius slides around the ulnar head, and the disc follows the radius by twisting at its apex and sweeping along beneath the ulnar head. Joint surface contact is optimal only with the forearm in a neutral position between supination and pronation. In maximal pronation and supination, the articulating surfaces have only minimal contact.[89] In full supination, the seat of the ulnar head rests on the palmar aspect of the ulnar notch, whereas in full pronation, it rests against the dorsal lip of the ulnar notch.[82,90]

Ligaments

The interosseous membrane provides stabilization at both the proximal and distal radioulnar joints (Fig. 8–23). The three ligaments associated specifically with the proximal radioulnar joint are the annular and quadrate ligaments and the oblique cord. The **annular ligament** is a strong band that forms four fifths of a ring that encircles the radial head (see Fig. 8–20B). The inner surface of the ligament is covered with cartilage and serves as a joint surface. The proximal border of the annular ligament blends with the joint capsule, and the lateral aspect is reinforced by fibers from the lateral collateral ligament.[3] The **quadrate ligament** extends from the inferior edge of the ulna's radial notch to insert in the neck of the radius (see Fig. 8–23A, B). The quadrate ligament reinforces the inferior aspect of the joint capsule and helps maintain the radial head in apposition to the radial notch. The quadrate ligament also limits the spin of the radial head in supination and pronation. The **oblique cord** is a flat fascial band on the ventral forearm that extends from an attachment just inferior to the radial notch on the ulna to insert just below the bicipital tuberosity on the radius (see Fig.8–23A). The fibers of the oblique cord are at right angles to the fibers of the interosseous membrane.[3] The functional significance of the oblique cord is not clear, but it may assist in preventing separation of the radius and ulna.

The **dorsal** and **palmar radioulnar ligaments,** as well as the interosseous membrane, reinforce the distal radioulnar joint. The dorsal and palmar ligaments are formed by longitudinally oriented collagen fiber bundles originating from the dorsal and palmar aspects of the ulnar notch of the radius.[89] The two ligaments extend along the margins of the articular disc to insert on the ulnar fovea and base of ulnar styloid process[82] (see Fig. 8–22). The palmar radioulnar ligament is at least 2 mm longer than the dorsal radioulnar ligament.[91] According to Linscheid, the dorsal radioulnar ligament averages 18 mm in length, whereas the palmar radioulnar ligament averages 22 mm.[82]



A Anterior view

B Posterior view

Figure 8–23 Ligamentous structures that provide stability for the proximal and distal radioulnar joints. The head of the radius has been slightly separated from the ulna, and the annular ligament has been removed to show the quadrate ligament. **A.** The anterior view of the radius and ulna are shown with the right forearm in a supinated position. The quadrate ligament is shown extending from the inferior edge of the radial notch to attach on the neck of the radius. The ventral oblique cord extends from below the radial notch to attach just below the bicipital tuberosity. **B.** A posterior view of the right radius and ulna in the supinated position. The interosseous membrane is shown extending between the radius and ulna for a considerable portion of their length. The dorsal oblique cord is not shown in this figure.

The interosseous membrane, which is located between the radius and the ulna, is a complex structure consisting of the following three components: a central band, a thin membranous portion, and a dorsal oblique cord (see Figs. 8–5 and 8–23A, B). The **central band** is described as being a strong, thick, ligamentous[92,93] or tendinous structure[94] consisting of bundles of fibers that run obliquely from the radius to the ulna. The central band has a very high collagen content arranged in fibrillar structures surrounded by elastin. The collagen content is more abundant in proximal bundles than it is in distal bundles.[95] When the tensile strength of the central band was compared with that of the patellar tendon, investigators[93] found that the ultimate tensile strength of the central band was 84% of the strength of the patellar tendon. In contrast to the central band, the **membranous portion** is described as a soft and thin structure that lies adjacent proximally and distally to the central band.[94] The **dorsal oblique cord** is considered part of the interosseous membrane and should not be confused with the oblique cord located on the ventral aspect of the forearm, which is not considered part of the interosseous membrane. The dorsal oblique cord extends from the proximal quarter of the ulna to the middle region of the radius.[92,94] Its fibers run counter to the central band.

The **interosseous membrane** maintains space between the radius and ulna during forearm rotation,[96] and according to a magnetic resonance imaging study by Nakamura and associates,[97] the central band remains taut throughout forearm rotation, apparently to keep the radius and ulna from splaying apart. In contrast, the membranous portion of the interosseous membrane evidenced wavy deformations at maximum supination and in the neutral position. Deformations also occurred around the oblique cord at maximum pronation. The interosseous membrane protects the proximal radioulnar joint by transferring some of the compressive loads at the distal radius to the proximal ulna.[98] The interosseous membrane maintains transverse stability of the forearm during compressive load transfer from the hand to the elbow[99] (see Fig. 8–28).

Maximum strain in the fibers of the central band was found to occur when the forearm was in a neutral position (midway between supination and pronation). Force in the interosseous membrane that depends on elbow flexion angle and forearm rotation ranges from a minimum of 8 N in full elbow extension with neutral forearm rotation to a maximum of 43 N at 30° of elbow flexion and with the forearm supinated. The largest of all forces was found in supination in all flexion angles.[100] The average proportion of total load in each bone in supination was 68% in the distal radius, 32% in the distal ulna, 51% in the proximal radius, and 49% in the proximal ulna. The interosseous membrane transfers loads from the wrist to the proximal forearm via fibers that run from the distal radius to the proximal ulna.The fibers in the central band are relaxed in both full supination and full pronation.[3,92]

A reciprocal relationship exists between the radius and ulna in axial force transmission. An axial force transmitted through the radius was found to be least at the extremes of supination and increased as the forearm moved from supination to pronation. Whereas the axial force transmitted through the ulna was least at the extreme of pronation and gradually increased as the forearm moved into supination.[101]

A tract extends from the interosseus membrane and inserts in the distal radioulnar joint capsule between the tendon sheaths of the extensor digiti minimi and the extensor carpi ulnaris muscles. The tract's deep fibers insert directly into the articular disc (triangular fibrocartilage). The tract of the interosseus membrane is taut in pronation and loose in supination.[102] The articular disc also provides stability for the inferior radioulnar joint by binding the distal radius and ulna together. The distal radioulnar joint capsule, which is a separate entity from the triangular fibrocartilage, can be a source of limitation of motion when it is invaded by scar tissue after wrist injuries.[103]

Muscles

The primary muscles associated with the radioulnar joints are the pronator teres, pronator quadratus, biceps brachii, and supinator. The **pronator teres** has two heads: a humeral head and an ulnar head. The humeral head comes from the common flexor tendon on the medial epicondyle of the humerus. The smaller ulnar head arises from the medial aspect of the coronoid process of the ulna. Both heads attach distally to the surface of the lateral side of the radius at its greatest convexity. The **pronator quadratus**, which is located at the distal end of the forearm, also has two heads (superficial and deep). Both of these heads arise from the ulna and cross the interosseous membrane anteriorly to insert on the radius. The fibers of the superficial head pass transversely across the interosseous membrane, whereas the fibers of the deep head extend obliquely across the interosseous membrane to insert on the radius.[104] The biceps brachii has been discussed previously. The **supinator** is a short, broad muscle that arises from the lateral epicondyle of the humerus, the radial collateral ligament, the annular ligament, and the lateral aspect of the ulna. The muscle crosses the posterior aspect of the interosseous membrane to insert into the radius just medial and inferior to the bicipital tuberosity.

Other muscles that are active during supination and pronation, especially when gripping is involved and during resisted motion, include the **flexor carpi ulnaris** and **extensor carpi ulnaris,** the **brachioradialis,** and the **flexor carpi radialis** and **extensor carpi radialis brevis (ECRB)** (see Fig. 8–11A, B). The anconeus muscle may also play a role in supination and pronation.

CASE APPLICATION

Role of the Extensor Carpi Radialis Brevis

case 8–5

The extensor carpi radialis brevis may play a role in James's pain because it exerts a pull on the lateral epicondyle and is active during gripping. Repetitive pulling during hammering and using a screwdriver and chain saw may have damaged the enthesis of either the common extensor tendon or the extensor carpi radialis brevis tendon.

Two tests that we performed on our patient were indicative of a diagnosis of tennis elbow. A third test we performed that is used to determine whether pain is coming from the extensor carpi radialis brevis tendon involves applying resistance to the end of our patient's extended third finger. James had pain over the lateral epicondyle when we performed this test, so now we have three tests indicating a diagnosis of tennis elbow with involvement of the extensor carpi radialis brevis. A sensorimotor test involving upper-limb reaction time and speed of movement showed that our patient had deficits when compared to a control group of healthy subjects.[105]

FUNCTION: RADIOULNAR JOINTS

Axis of Motion

The axis of motion for pronation and supination is a longitudinal axis extending from the center of the radial head to the center of the ulnar head.[9,106] In supination, the radius and ulna lie parallel to one another, whereas in pronation, the radius crosses over the ulna (Fig. 8–24). Motion of the distal ulna is of less magnitude than that of the radius and opposite in direction to motion of the radius.[88] The ulnar head moves distally and dorsally in pronation and proximally and medially in supination. Therefore, at the distal radioulnar joint, the ulnar head slides in the ulnar notch of the radius from the dorsal lip of the ulnar notch in pronation to a position on the palmar aspect of the ulnar notch in full supination.[82] From supination to pronation an ulnar valgus shift is the predominant motion, which according to Kasten[107] is an average of 5.84° in 90° of pronation. From pronation to supination the ulna undergoes a 8.31° varus or lateral shift totally in the coronal plane.

Range of Motion

A total range of motion of 150° has been ascribed to the radioulnar joints.[3,87,106] In the clinic, the range of motion of pronation and supination is assessed with the elbow held against the trunk in 90° of flexion (Fig. 8–25). This position of the elbow stabilizes the humerus so that radioulnar joint rotation may be distinguished from rotation that is occurring at the shoulder joint. When the elbow is fully extended, active supination and pronation occur in conjunction with shoulder rotation. Limitation of pronation when the elbow is extended may be caused by passive tension in the biceps brachii. Pronation in all elbow positions is limited by bony approximation of the radius and ulna and by tension in the dorsal radioulnar ligament and the posterior fibers of the medial collateral ligament of the elbow.[24] Supination is limited by passive tension in the palmar radioulnar ligament and the oblique cord. The quadrate ligament limits spin of the radial head in both pronation and supination, and the annular ligament helps maintain stability of the proximal radioulnar joint by holding the radius in close approximation to the radial notch.

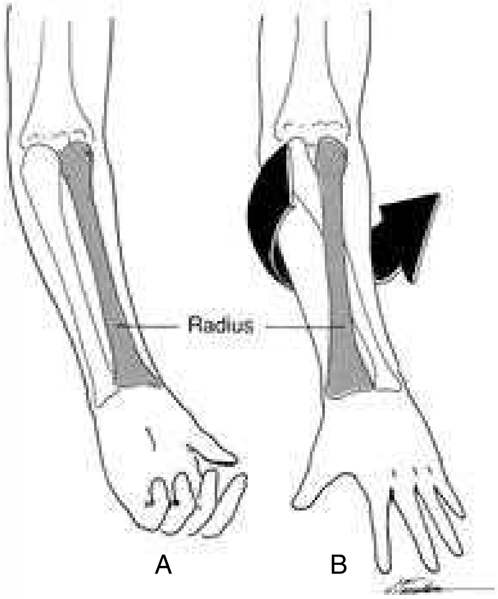


Figure 8–24 Supination and pronation in the left upper extremity. **A.** The radius and ulna are parallel to each other in the supinated position of the forearm. **B.** In the pronated position, the radius crosses over the ulna.

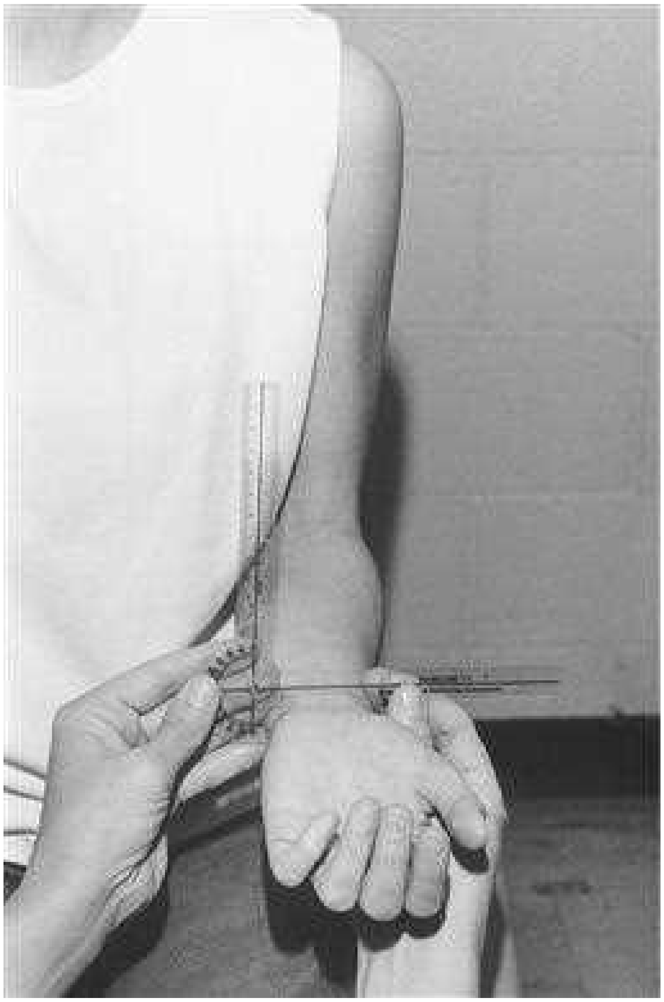


Figure 8–25 Clinical measurement of the range of motion in supination with a goniometer. The subject's left elbow is flexed to 90° and the upper arm is held close to the trunk. The same position is used for measuring the range of motion in pronation. *(From Norkin CC, White DJ: Measurement of Joint Motion: A Guide to Goniometry (ed. 4). Philadelphia, FA Davis, 2009, with permission.)*

Muscle Action

The pronators produce pronation by exerting a pull on the radius, which causes its shaft and distal end to turn over the ulna (Fig. 8–26A, B). The pronator teres has its major action at the radioulnar joints, but the long head, as a two-joint muscle, plays a slight role in elbow flexion. The pronator teres contributes some of its force toward stabilization of the proximal radioulnar joint, inasmuch as the muscle's

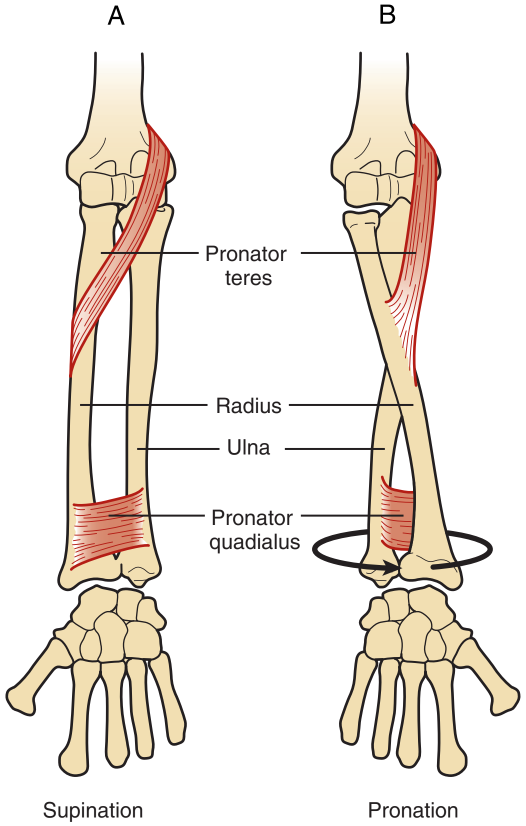


Figure 8–26 Pronation of the right forearm **A.** The pronator teres and the pronator quadratus are shown in the supinated position of the forearm. **B.** The two pronator muscles produce pronation by pulling the radius over the ulna.

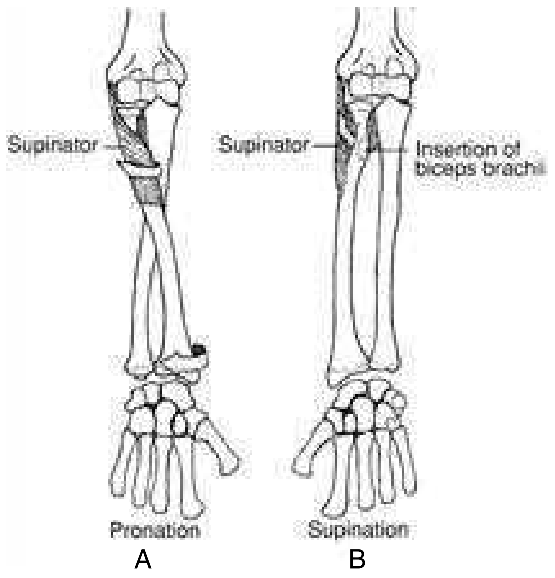


Figure 8–27 Supination of the right forearm. **A.** In the pronated position, the supinator muscle wraps around the proximal radius. A contraction of the supinator, the biceps, or both pulls the radius over the ulna. **B.** The supinator muscle and the insertion site of the biceps are shown in the supinated position.

translatory component helps the radial head maintain contact with the capitulum.

The pronator quadratus, a one-joint muscle, is unaffected by changing positions at the elbow. The pronator quadratus is active in unresisted and resisted pronation and in slow and fast pronation. The deep head of the pronator quadratus is active during both resisted supination and resisted pronation and is thought to act as a dynamic stabilizer to maintain compression of the distal radioulnar joint.[104,106]

In a mechanical study on cadavers, the pronators were found to be most efficient around the neutral position of the forearm when the elbow was flexed to 90°.[108] Bremer,[73] in another study on cadavers, determined that the pronating effect of the pronator teres and pronator quadratus reached a maximum between 40° of supination and 40° of pronation with a decline at the extremes of pronation and supination. In a study by Gordon and colleagues in which the supinators and pronators were tested in the absence of gripping but against resistance, no significant differences were found between supination and pronation torques with the forearm in a neutral position. However, supination torque generation was greatest with the forearm in pronated positions, and pronation torque generation was greatest with the forearm in supinated positions.[109]

The supinators, like the pronators, act by pulling the shaft and distal end of the radius over the ulna (Fig. 8–27A, B). The supinator muscle may act alone during unresisted slow supination in all positions of the elbow or forearm. It can also act alone during unresisted fast supination when the elbow is extended. However, activity of the biceps is always evident when supination is performed against resistance and during fast supination when the elbow is flexed to 90°. As the forearm moves into pronation, its supination torque increases and reaches a maximum at about 20° of pronation. Bremer found that the moment arms of all major supinators exhibited peak torque values in 40° to 50° of pronation.[73] The biceps brachii has been found to exert four times as much supination torque with the forearm in the pronated position than with other forearm positions.[108] A mean maximum supination torque of 16 Nm was recorded with the forearm 75% pronated, in comparison with a mean maximum supination torque of 13.1 Nm for the neutral forearm position with the elbow at 45° of flexion.[71] The anconeus muscle is active in supination and pronation, and an elbow stabilization role has been suggested to explain this activity. As determined by isometric testing, the supinators are stronger than the pronators.[110]

Stability

Muscular support of the distal radioulnar joint is attributed to the pronator quadratus[91,104,106,111,112] and the extensor carpi ulnaris tendon.[82,92,113] The deep head of the pronator quadratus is active throughout supination and pronation and therefore is thought to provide dynamic stabilization for the distal radioulnar joint.[110] Activity in the extensor carpi ulnaris muscle exerts a depressive force on the dorsal aspect of the ulnar head as the tendon is stretched over the head during supination. Tension in the tendon helps to maintain the position of the ulnar head during both supination and pronation.[113]

The extensor carpi radialis brevis also provides support for the forearm, as evidenced by the maximum voluntary effort (MVE) in the extensor carpi radialis brevis that occurs in both supination (26% to 43% MVE) and pronation torques (27% to 55% MVE). The extensor carpi radialis brevis also appears to act both as a stabilizer to the forearm for gripping during pronation torques (depending on forearm angle) and as a prime mover for wrist extension for supination torques.[71]

CASE APPLICATION

Link Between Gripping During Forearm Rotations and High Muscle Activity

case 8–6

The direct link found by O'Sullivan and Gallwey[71] between gripping during forearm rotations and high muscular activity in the extensor carpi radialis brevis not only helps explain the mechanism of injury but also has implications for the prognosis for James. A poor prognosis in cases of tennis elbow is associated with manual job employment with a high level of physical strain at work and a high level of pain at baseline.[114]

Nonmuscular support of the distal radioulnar joint is provided by the dorsal and palmar radioulnar ligaments, the interosseous membrane and its tract, and the articular disc. The dorsal radioulnar ligament becomes taut in pronation, whereas the palmar radioulnar ligament becomes taut in supination.[83,89,91,106,111,115] According to Schiend,[90] the radioulnar ligaments have limited cross-sectional areas and low structural stiffness, but they are able to prevent separation of the radius from the ulna during loading and also allow for force transmission from the radius to the ulna through the distal radioulnar joint.[90] However, these ligaments do not augment longitudinal stability. They allow approximately 5 mm of play between the radius and ulna before providing resistance to further distraction.[82] The radioulnar ligaments, the articular disc, and the pronator quadratus maintain the ulna within the ulnar notch and prevent the ulna from subluxating or dislocating. However, these ligaments allow a high degree of mobility. The interosseous membrane provides stability for the distal joint by binding the radius and ulna together. Also, according to Skahen and coworkers, the interosseous membrane in combination with the triangular fibrocartilaginous complex provide important longitudinal stabilization.[92,116]

Markolf and associates studied radioulnar load sharing at the wrist and elbow with the elbow in varus, valgus, and neutral positions.[117] When the elbow was in the varus position (no contact between the radial head and capitulum), force was transmitted from the distal radius through the interosseous membrane to the proximal ulna (Fig. 8–28). When the elbow was in the valgus position (contact between the radial head and the capitulum), the force was transmitted through the radius. When the forearm was in the neutral position, the mean force in the distal end of the ulna averaged 7% of the applied wrist load, while the force in the proximal ulna averaged 93% of the load applied to the wrist.[117] The tract associated with the interosseous membrane is taut in pronation and loose in supination. During pronation, the tract protects the ulnar head in a sling. It also provides stability for the joint by reinforcing the dorsal aspect of the joint capsule.[102]

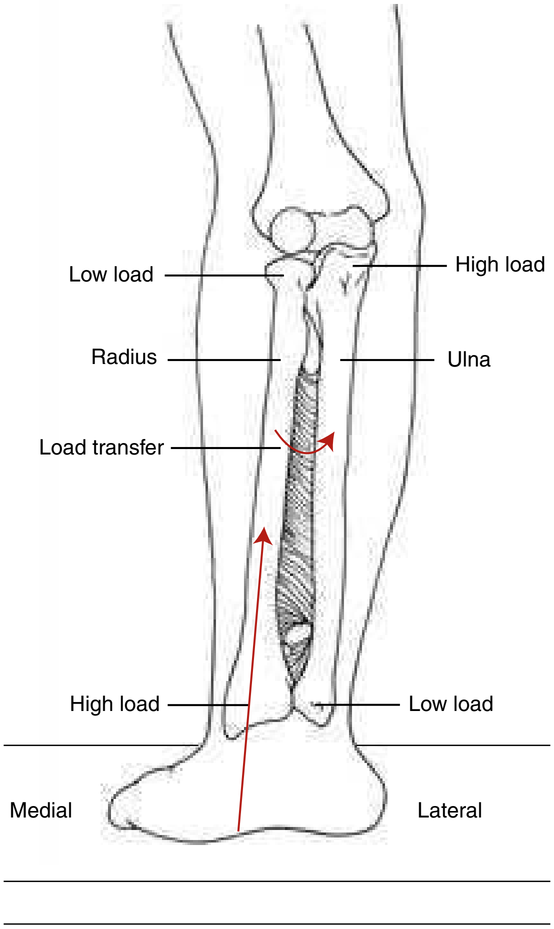


Figure 8–28 Interosseous membrane force transmission from radius to the ulna.

The articular disc acts as a cushion in allowing compression force transmission from the carpals to the ulna and acts as a stabilizer of the ulnar side of the carpals.[88] Also, the disc assists in the transmission of compressive forces from the radius to the ulna.[79,117,118] Adams and Holley[115] used a distractive force to simulate the effects of the separation of articulating surfaces that accompanies a power grip. These authors found that strain distribution in the disc was dependent on forearm position.[115] Tension across the entire disc decreased in supination and increased in the radial portion of the disc in pronation. The authors concluded that the articular disc regularly bears both compressive and tensile strains.[115] According to Mikic, compressive forces are transmitted through the central portion of the disc, and some of the load is converted to tensile loading within the peripheral margins.[83] A summary of ligamentous and muscular support for both radioulnar joints is presented in Table 8–3.

MOBILITY AND STABILITY: ELBOW COMPLEX

Functional Activities

The joints and muscles of the elbow complex are used in almost all activities of daily living, such as dressing, eating, carrying, and lifting. They are also used in tasks such as splitting firewood, hammering nails, and in sports such as tennis, golf, basketball, and baseball. Most of the activities of daily living require a combination of motion at both the elbow and radioulnar joints. Morrey and associates measured elbow and forearm motion in 33 healthy subjects during 15 activities.[119] The authors concluded that a total arc of about 100° of elbow flexion (between 30° and 130°) and about 100° of forearm rotation (50° supination and 50° pronation) is sufficient to accomplish simple tasks such as eating, drinking, brushing hair, brushing teeth, and dressing (Fig. 8–29). For example, about 40° of pronation and 20° of supination are necessary to use a telephone.[119] Therefore, mobility of the complex is necessary for normal functioning in most areas of activity. As can be seen in Table 8–4,[119,120,121,122] among the 10 activities listed, using the telephone requires the largest arc of motion in flexion (92.8°) and pronation/supination (63.5°). Cutting with a knife requires the smallest arc of flexion and of pronation/supination.

Figure 8–29 According to Safaee-Rad[121] (see Table 8–4), drinking from a cup requires a range of elbow flexion between 72° and 129° and a range of pronation/supination between 3° and 31°. (*From Norkin CC, White DJ: Measurement of Joint Motion: A Guide to Goniometry (ed. 4). Philadelphia, FA Davis, 2009, with permission.*)

Relationship to the Hand and Wrist

The design of the radioulnar joints enhances the mobility of the hand. In primitive mammalian species, the ulna was a major weight-bearing structure and was connected directly to the carpals through a dense immobile syndesmosis.[106] The complete separation of the ulna from the carpals by the articular disc and the formation of a true diarthrodial joint lined with articular cartilage are features that permit pronation and supination to occur in every position of the hand to the forearm. Pronation and supination of the forearm, when the elbow is flexed at 90°, rotates the hands so that the palm faces either superiorly or inferiorly. The mobility afforded the hand is achieved at the expense of stability: The movable forearm is unable to provide a stable base for attachment of the wrist and hand muscles. Therefore, many of the muscles that act on the wrist and hand are

Table 8–3 Ligamentous and Muscular Contributions to Stability at the Proximal and Distal Radioulnar Joints

JOINT	LIGAMENTOUS	MUSCULAR
Proximal radioulnar joint	Annular and quadrate ligaments[11] Oblique cord[21] (limits supination) Interosseous membrane	Passive tension in the biceps brachii in the fully extended elbow position Pronator teres (helps maintain contact of radial head and capitulum)
Distal radioulnar joint	Interosseous membrane[49,50] Dorsal radioulnar ligament (limits pronation)[39,46,56] Palmar radioulnar ligament (limits supination)[39,46,56] Triangular fibrocartilage[39,40,47,59] Joint capsule	Pronator quadratus[39,52,57] Anconeus Extensor carpi ulnaris[39,48,58] Pronator teres

Table 8–4 Elbow and Forearm Motion During Functional Activities: Mean Values in Degrees

ACTIVITY	FLEXION			PRONATION AND SUPINATION			SOURCE
	Min	*Max*	*Arc*	*Pronation Max*	*Supination Max*	*Arc*	
Combing hair	112	157	45	−54	−143	−89	Magermans[119]
Wash axilla	104	132	18	−42	−124	−82	Magermans[119]
Use telephone	42.8	135.6	92.8	40.9	22.6	63.5	Morrey[120]
	75	140	65				Packer[122]
Rise from chair	20.3	94.5	74.2	33.8	−9.5*	24.3	Morrey[120]
	15	100	85				Packer[122]
Open door	24.0	57.4	33.4	35.4	23.4	58.8	Morrey[120]
Read newspaper	77.9	104.3	26.4	48.8	−7.3*	41.5	Morrey[120]
Pour from a pitcher	35.6	58.3	22.7	42.9	21.9	64.8	Morrey[120]
Put glass to mouth	44.8	130.0	85.2	10.1	13.4	23.5	Morrey[120]
Drink from cup	71.5	129.2	57.7	−3.4[†]	31.2	27.8	Safaee-Rad[121]
Cut with knife	89.2	106.7	17.5	41.9	−26.9*	15.0	Morrey[120]
Eat with fork	85.1	128.3	43.2	10.4	51.8	62.2	Morrey[120]
	93.8	122.3	28.5	38.2	58.8	97.0	Safaee-Rad[121]
Eat with spoon	101.2	123.2	22.0	22.9	58.7	81.6	Safaee-Rad[121]
	117	143	26	−33	−127	−94	Magermans[119]
	70	115	45				Packer[122]

*The minus sign indicates pronation.

[†] The minus sign indicates supination.

Source: Adapted from Norkin CC, White DJ: Measurement of Joint Motion: A Guide to Goniometry (ed. 4). Philadelphia, FA Davis, 2009.

attached on the distal end of the humerus rather than on the forearm.

The location of the hand and wrist muscles at the elbow and the fact that these muscles cross the elbow create close structural and functional relationships between the elbow and wrist/hand complexes. Anatomically, the hand and wrist muscles help reinforce the elbow joint capsule and contribute to stability of the elbow complex. In a study of 11 cadaveric specimens, Davidson and coworkers[123] found that the humeral head of the flexor carpi ulnaris muscle is the only muscle that lies directly over the anterior portion of the medial collateral ligament at elbow flexion positions between 90° and 120°. Because the medial elbow is subjected to the largest valgus stress during the cocking and acceleration phases of throwing, which occur between 80° and 120° of elbow flexion, the flexor carpi ulnaris muscle has the potential to provide significant reinforcement for the medial collateral ligament during throwing activities.[123] Lin and colleagues[124] suggested that the flexor carpi ulnaris, flexor digitorum sublimus, and flexor carpi radialis function as dynamic stabilizers, with the flexor carpi ulnaris being the primary stabilizer for elbow valgus stability. The flexor carpi ulnaris, flexor dititotum sublimus, and flexor carpi radialis significantly reduced medial collateral ligament strain at 45° and 90°. All extensor muscles increased the strain in the medial collateral ligament.[124]

Continuing Exploration 8-4:

The Role of Muscles in Joint Stability

During muscle contractions, the wrist muscles may contribute to the torque production of the elbow muscles. However, the muscles may have a more important functional role: producing compression of the articulating surfaces at the elbow. The importance of compression or stabilization of the elbow can be seen in the work of Amis and associates,[64] who investigated the effect of tensile loads on the forearm during a pulling activity. They found that both the humeroradial and humeroulnar articulations are subjected to compressive forces during pulling activities and that the medial collateral ligament was heavily loaded. Andersson and Schultz found that during a pulling task, the flexors, at an elbow position of 90° of flexion, exerted a flexor force of 6,000 N.[125] In an investigation of interarticular compressive stress of the elbow in extension in 30 cadavers with intact medial and lateral collateral ligaments, Chantelot and colleagues determined that compressive stress at the humeroulnar joint is unaffected by pronation and supination.[27] The investigators also found that the ulnar coronoid process is located in an area that recieved 52% to 65% of the stress. At the humeroradial joint, the compressive stress on the radial head varied from a low of 6% in full supination to 23% in the neutral position. The anterior portion of the radial head absorbed most of the compressive stress, and the authors of the study concluded that the radial head does not appear to play a major role in elbow stability if an intact ulnar collateral ligament exists.[27]

EFFECTS OF AGE, GENDER, AND INJURY

Like other joints in the body, the joints and muscles of the elbow complex may be subject to the effects of age, injury, and immobilization.

Age and Gender

As can be seen in the sampling of experimental findings presented in Table 8–5, the decrease in muscle strength that accompanies increasing age appears to be affected by the type of muscle action involved (eccentric/concentric), the muscle group involved, and gender, along with other factors such as level of physical activity.[126–131] Also, the elderly have been found to have smaller pennation angles and decreased fascicle lengths, which may acount for as much of 50% of the loss of muscle function. Fortunately, resistance training may be able to increase fasicicle length and therefore the number of sarcomeres.[132]

Continuing Exploration 8-5:

Elbow Torque in Children and Adults

Wood[133] investigated isokinetic elbow torque development in children over a 3-year period. The average age in the first year of the study was 13 years for 17 boys and 14 girls. Isokinetic concentric and eccentric elbow extensor peak torques did not differ significantly at any test occasion for boys and girls. However, eccentric elbow flexor torques were significantly greater than concentric elbow flexor torques.

Neu[134] did find gender differences: muscle and grip force in boys were higher than in girls in a group of 6- and 7-year-olds. Grip force per muscle cross-sectional area, adjusted by forearm length, increased by almost a half between 6 and 20 years in boys and girls. Deigham[135] measured the maximum cross-sectional area of the elbow extensors in children (ages 9 and 10), teenagers (16 and 17), and adults (over 21). Significant effects of gender and age group on physiological cross-sectional area were found for each muscle group. A greater physiological cross-sectional area was found in males than in females except in the 9- and 10-year-olds, and a greater physiological cross-sectional area with increasing age was also found in males compared to females. Percentage increases in physiological cross-sectional area for the extensors between the youngest and oldest age groups were 207% for males and 65% for females. The percentage increases in mean physiological cross-sectional area for the flexors was 210 % for the males and 78% for the females. Tonson and colleagues[136] also found that mean physiological cross-sectional area ratios were significantly higher in adults than in children and adolescents. According to these researchers, the maximal strength exerted by the forearm muscles is proportional to their size, and muscle volume is the best index of muscle size during growth.

Significant differences between young and old groups have been found in the location where peak torque is produced in a range of motion.[128] John[137]determined that younger subjects produce significantly greater relative joint torques than older subjects across all effort levels except for the light effort level. Valour and Pousson found that maximal isometric force and series elastic component compliance of the elbow flexors were significantly less in the elderly than in younger groups, but the antagonist coactivation was similar for both groups.[138] In contrast, John[137] found that for both isometric elbow flexion and extension tasks, older subjects showed significantly greater antagonist co-contraction than younger subjects.

Table 8–5 **Effects of Aging on Elbow Muscles**

HUGHES ET AL[127]	LYNCH ET AL[128]	GALLAGHER ET AL[129]	RUNNELS[142]
n = 68 women *n = 52 men*	*n = 339 women* *n = 364 men*	*n = 60 men*	*n = 75*
Age 1st eval, 47–78 yr *Age 2nd eval, 56–88 yr*	*Age 19–93 yr*	*Age 20–60 yr*	*Age 20–83 yr*
Isokinetic strength in the elbow flexors and extensors declined by 2% per decade for women and 12% per decade for men.	Muscle quality (peak torque per unit of muscle mass) for concentric peak torque showed a 28% decrease in men and a 20% decrease in women. Eccentric peak torque showed a 25% decline in men, but the decline was not significant in women.	Active elbow flexion/extension peak torque, power, and angle of peak torque production measured bilaterally and showed highly significant differences between young and old. However, no age-related differences occurred in supination and pronation.	Isokinetic elbow flexor and extensor peak torque was less in men 60 to 83 years than in men 20 to 59 years. Maximal isometric force production also declined at age 60.

Klass Baudry and Duchateau[139] attributed these conflicting findings to differences in the sensitivity of the methods used to assess voluntary activation, characteristics of the study population, muscle group being tested, and type of contraction being performed.

Muscular Effort in Young and Old

John[137] determined that older people (65–70 years) require significantly greater muscular effort than do younger subjects (20–40 years) during isometric elbow flexion and extension torque production tasks. The elderly also made more severe errors in judgment about the amount of effort needed to accomplish a motor task. The younger subjects were able to produce significantly larger torques for both flexion and extension tasks than the older subjects at all effort levels except for the lowest effort level. Klein and associates found that the area of type I fibers in the biceps brachii muscle and maximum voluntary strength of the elbow flexors is lower in the elderly than in young persons, but the percentages of type II fibers and type I fiber areas are not different between young and old persons.[140]

Aging Effects on Forearm Muscles

Toji[141] concluded that muscle force and shortening velocity may be affected more by muscle function than by physiological cross-sectional area and that the effects of aging on muscle shortening velocity becomes apparent after age 50. Isokinetic testing conducted by Runnels[142] in a group of 75 volunteers ages 20 to 83 years demonstrated that the elbow flexor and extensor muscle performance remained relatively constant from age 20 to age 59 before declining at all angular velocities. The pattern of decline began in the 60- to 69-year-old group, with the rate of decline being greatest at age 60. Chronological age affected the performance of muscles independent of muscle mass and regardless of contraction type; however, isometric contraction was the least affected in 75 volunteers ages 20 to 83 years.[142]

Injury

Injuries to the elbow are fairly common, and in early adolescence the elbow is one of the most common sites for apophysitis or strains at the apophysis.[143] An understanding of the mechanisms of elbow injuries and their relation to elbow joint structures is necessary for determining the effects of the injuries on joint function.

Compression Injuries

Resistance to longitudinal compression forces at the elbow is provided mainly by the contact of bony components; therefore, excessive compression forces at the elbow often result in bony failure. Falling on the hand when the elbow is in a close-packed (extended) position may result in the transmission of forces through the bones of the forearm to the elbow (Fig. 8–30A). If the forces are transmitted through the radius, as may happen with a concomitant valgus stress, a fracture of the radial head may result from impact of the radial head on the capitulum (Fig. 8–30B). A fracture of the radial head maybe accompanied by a tear of the central band of the interosseous membrane, which provides the majority of the membrane's strength. Unfortunately, the central band has little physiological ability to heal.[143] If the force from the fall is transmitted to the ulna, a fracture of either the coronoid process or olecranon process may occur from the impact of the ulna on the humerus. If neither the radius nor the ulna absorbs the excessive force by fracturing, then the force may be transmitted to the humerus, which may result in a fracture of the supracondylar area.

Muscle contractions also may cause high compression forces at the elbow. For example, during the acceleration and deceleration phases of baseball pitching, the compression

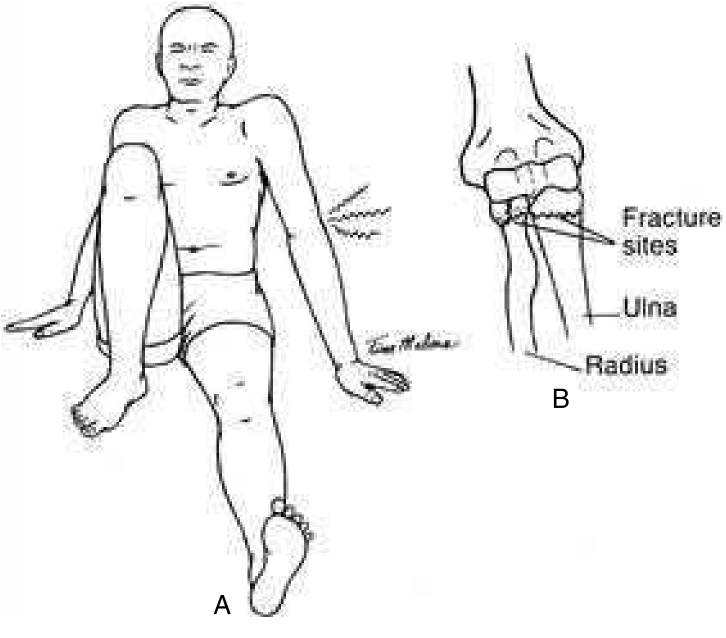


Figure 8–30 A fall on the hand with the elbow in a close-packed position may involve the transmission of forces through the bones of the forearm to the elbow. **A.** The transmission of forces from the hand to the elbow may occur through either the radius or ulna or through both. **B.** Impact of the radial head on the capitulum may cause a fracture of the radial head, the neck, or both. A fracture of the coronoid process, olecranon process, or both may result from forces transmitted through the ulna.

forces at the elbow can attain 90% of body weight.[144] Nerve compression, bony fracture, or dislocation may also result from muscle contractions. Repetitive forceful contractions of the flexor carpi ulnaris muscle may compress the ulnar nerve as it passes through the cubital tunnel between the medial epicondyle of the humerus and olecranon process of the ulna[145–147] (Fig. 8–31). According to Chen and colleagues,[148] the ulnar nerve may be subjected not only to compression but also to traction and friction stresses during flexion and extension. These stresses can cause an injury called *cubital tunnel syndrome*, in which motion of the fourth and fifth fingers is impaired. Even in a magnetic resonance imaging examination of 20 normal fresh-frozen elbow specimens, the ulnar nerve changed in area as much as 50% during elbow flexion and extension.[149]

Distraction Injuries

Ligaments and muscles provide for resistance of the joints of the elbow complex to longitudinal traction. A tensile force of sufficient magnitude exerted on a pronated and extended forearm may cause the radius to be pulled inferiorly out of the annular ligament. This injury is common in children younger than 5 years[149] and rare in adults.[150] Lifting a small child up into the air by one or both hands or yanking a child by one hand is the usual causative mechanism, and therefore the injury is referred to as either *nursemaid's elbow* or *pulled elbow*[149] (Fig. 8–32).

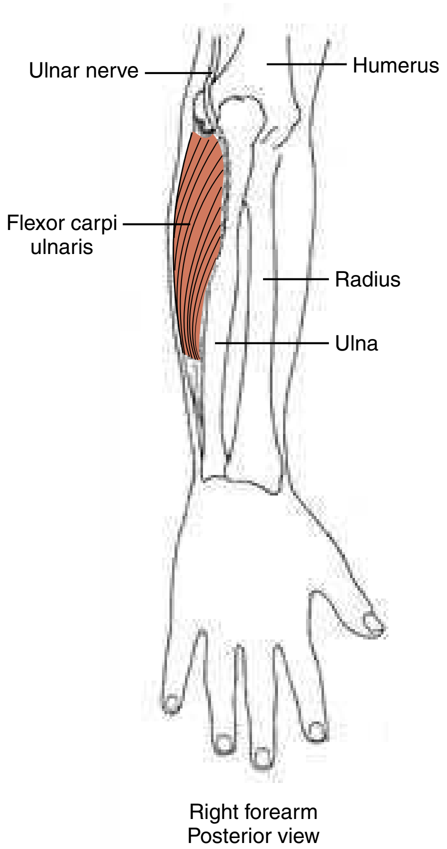


Figure 8–31 Location of the ulnar nerve as it passes through the cubital tunnel. A contraction of the flexor carpi ulnaris muscle can cause compression of the ulnar nerve between the two heads of the muscle, which are located on either side of the ulnar nerve at the elbow.

Varus/Valgus Injuries

Distraction and compression forces are created if either one of the collateral ligaments is overstretched or torn. If one side of the joint is subjected to abnormal tensile stresses, the other side is subjected to abnormal compressive forces (Fig. 8–33).

For example, the medial collateral ligament is subjected to tensile stress during the backswing or "cock-up" portion of throwing a ball (Fig. 8–34). If the stress on the medial collateral ligament is repetitive, such as in baseball pitching, the ligament may become lax and unable to reinforce the medial aspect of the joint.[151–153] The resulting medial instability may cause an increase in the normal carrying angle and excessive compression on the lateral aspect of the joint, so that the radial head impacts on the capitulum. If the abnormal compression forces on the articular cartilage are prolonged, these forces may interfere with the blood supply of the cartilage and result in **avascular necrosis** of the capitulum.

In a study of 40 uninjured professional baseball pitchers, Ellenbacher and colleagues found increased elbow laxity in players' pitching arms.[152] In another study, full-thickness tears of the medial collateral ligament were found in over half of the elbows tested. In addition, 30 loose bodies were detected in the elbows of 14 subjects, and cartilaginous damage was present in 21 elbows.[153]

Other conditions that may occur in the throwing elbow include ulnar neuritis, flexor-pronator muscle strain or tendinitis, and medial epicondylitis.[154] Medial tendinitis and medial epicondylitis may be caused by forceful repetitive contractions of the pronator teres, the flexor carpi radialis, and, occasionally, the flexor carpi ulnaris. These muscles are

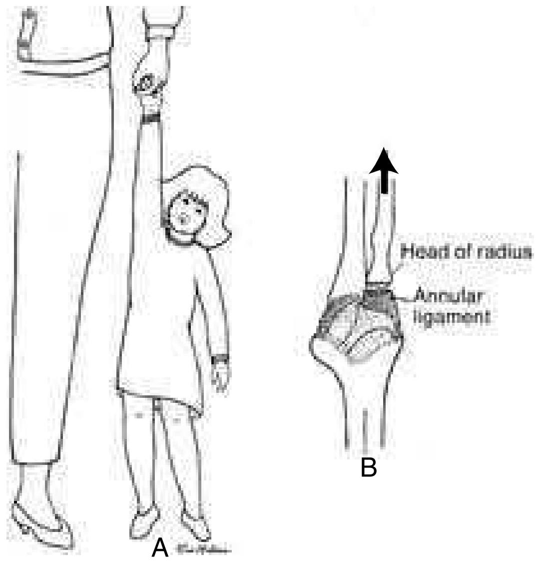


Figure 8–32 Nursemaid's elbow. **A.** A pull on the hand creates tensile forces at the elbow. **B.** The radial head is shown being pulled out of the annular ligament.

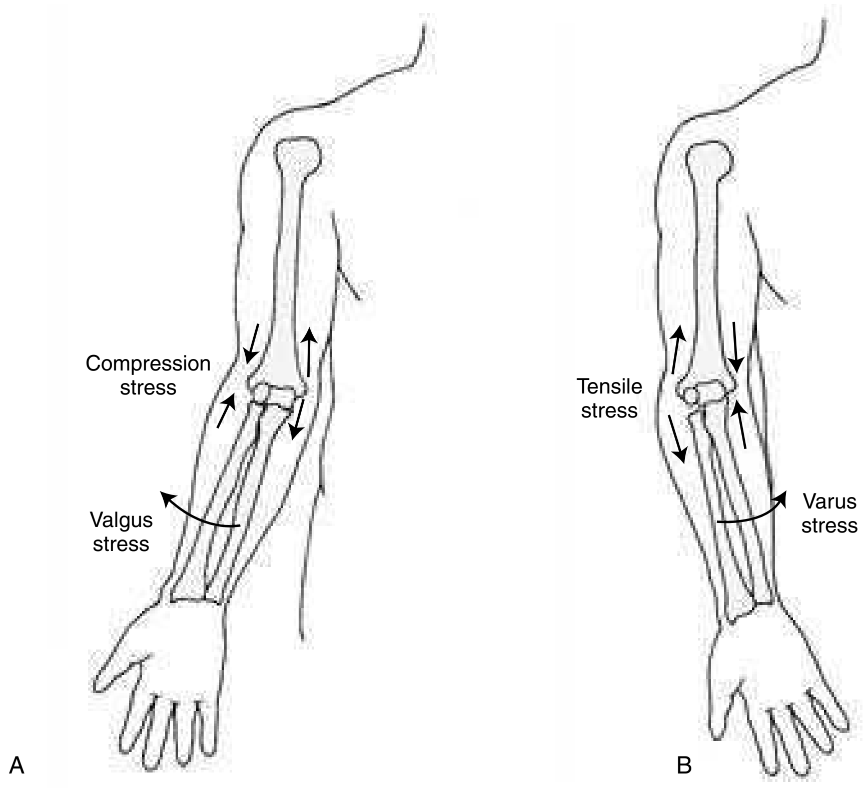


Figure 8–33 **A.** The application of a valgus stress to the forearm produces compression on the lateral aspect of the elbow joint and tensile stress on the medial joint aspect. **B.** The application of a varus stress to the forearm produces tensile stress on the lateral aspect of the elbow joint and compression on the medial joint aspect.

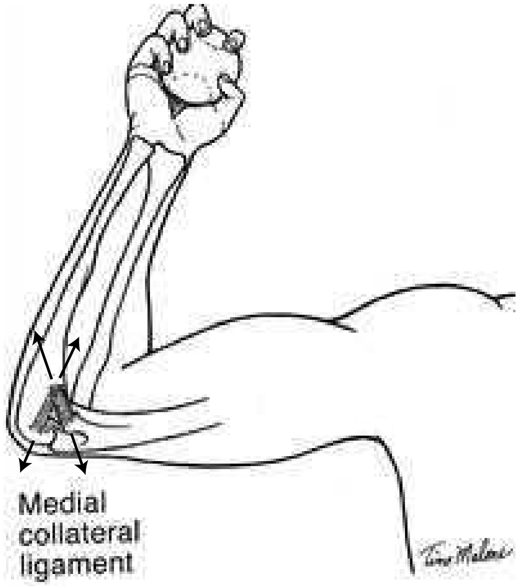


Figure 8–34 Stretching of the medial collateral ligament during throwing.

involved in the tennis serve when the combined motion of elbow extension, pronation, and wrist flexion is used. High-speed video analysis shows that the elbow moves from 116° to 20° of flexion during serving. Ball impact occurs at an average of 35° of flexion. The forearm is in about 70° pronation at full impact.[155] Medial epicondylitis, or golfer's elbow, is thought to result from traction-based insults to the elbow from the wrist and hand flexors and forearm pronators at their insertion into the medial epicondyle. A sudden deceleration of the golf club head hitting the ground instead of the ball is the usual cause.[156]

CASE APPLICATION

Diagnosis and Treatment Options

case 8–7

We have tentatively diagnosed James as having "tennis elbow" even though we know that he is not a tennis player. A person does not have to be a tennis player to develop tennis elbow. Any repetitive activity that causes tensile stresses on the lateral epicondyle may cause tennis elbow. We also reviewed the evidence for the change in diagnosis for tennis elbow from lateral epicondylitis to lateral epicondylosis or tendinopathy and are aware of the model proposed by Coombs[48] that includes not only local tendon pathology but also changes in the pain system and impairment in the motor system.

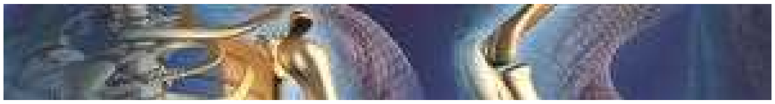
Now we are faced with the challenge of determining the best treatment for James. The choice of treatment is somewhat controversial, and many options are available but with little supporting scientific evidence. Some treatment options include splinting, forearm support bands and taping,[157–164] ultrasound,[165,166] low-level laser therapy, Bjordal[167] manipulation, exercise, and mobilization techniques,[168,169] active release technique, and acupuncture for short-term pain relief.[170] Botulinum toxin injection and extracorporeal shock-wave treatments also have been employed.[171–173] Although many different treatment options are available, more studies need to be performed to provide us with sufficient evidence of the success of one method over another.

SUMMARY

The interrelationship between the elbow complex and the wrist and hand complex makes normal functioning of the elbow vitally important. If elbow function is impaired, function of the hand also may be impaired. For example, if the elbow cannot be flexed, it is impossible for the hand to bring food to the mouth. Because many important vascular and neural structures that supply the hand are closely associated with the elbow, it is important to prevent excessive stress and to protect the elbow from injury. If the radial nerve is injured at the level of the epicondyle, the wrist extensors, supinator, and thumb and finger extensors will be affected. If the median nerve is injured at the level of the elbow, the pronators, flexor carpi radialis, finger flexors, thenar muscles, and lumbricales will be affected. In the following chapter, we will discuss the specific functions of the hand muscles and will be better able to appreciate the significance of injury to some of the muscles associated with the elbow complex.

Some of the interrelationships between the structure and function of elbow, shoulder, wrist, and hand have been introduced in this chapter. Muscles that have their primary actions at the wrist and hand also cross the elbow and contribute to its stability and function, while the stability and range of motion at the shoulder and elbow help to enhance the function of the wrist and hand. Compensations at the elbow complex often are necessary when the range of motion is limited at the shoulder or wrist. New relationships for the joints and muscles of the upper extremity will be introduced in the detailed study of the wrist and hand that follows in the next chapter.

STUDY QUESTIONS



1. Name and locate all of the articulating surfaces of the joints of the elbow complex and describe the method of articulation at each joint, including axes of motion and degrees of freedom.
2. Describe the articulation at the humeroradial joint between the radial head, capitulum, and annular ligament in supination and pronation with the elbow flexed and extended.
3. Explain the stabilizing function of the brachioradialis by diagramming the translatory and rotatory components at different joint angles.
4. Explain why active elbow flexion is more limited than passive flexion. Which structures limit extension?
5. Describe the "carrying angle" and explain why it is present and why females have been found to have larger angles than males.
6. Which structures limit supination and pronation?
7. If slow pronation of the forearm is attempted without resistance, which muscle will be used?
8. What does the term *concave incongruity* mean? Where is this condition found?
9. How does the structure and function of the annular ligament differ from that of the medial collateral ligament?
10. Describe the activity of the biceps brachii during a chin-up and the activity of the triceps during a push-up.
11. Describe the mechanism of injury in tennis elbow.
12. Explain why the term *epicondylosis* may be a more appropriate diagnostic term than the term *epicondylitis* for tennis elbow.
13. Compare the medial and lateral collateral ligaments on the basis of structure and function.
14. Compare the biceps brachii and the brachialis on the basis of structure and function.
15. Describe the mechanism of injury involved in cubital tunnel syndrome.
16. Explain the function of the interosseous membrane.
17. At what age would you expect elbow flexor and extensor muscle function to decline?
18. According to Table 8–4, what activity requires the most pronation?
19. What ligaments provide support the distal radioulnar joint?
20. Can a full range of passive elbow motion be accomplished if the shoulder is also in full flexion? Explain your answer.
21. What is the role of the articular disc (triangular fibrocartilage) at the distal radioulnar joint?

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Chapter 9

The Wrist and Hand Complex

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Functional Position of the Wrist and Hand

INTRODUCTION

The human hand may well surpass all body parts but the brain as a topic of universal interest. The human hand has been characterized as a symbol of power,[1] an extension of intellect,[2] and the seat of the will.[3] The symbiotic relation of the mind and hand is exemplified by sociologists' claim that while the brain is responsible for the design of civilization, the hand is responsible for its formation. The hand cannot function without the brain to control it; likewise, the encapsulated brain needs the hand as a tool of expression. The entire upper limb is subservient to the hand. Any loss of function in the upper limb, regardless of the segment, ultimately translates into diminished function of its most distal joints. The significance of this potential loss has led to the detailed study of the finely balanced intricacies of the normal upper limb and hand.

THE WRIST COMPLEX

The wrist **(carpus)** consists of two compound joints: the **radiocarpal** and the **midcarpal joints,** referred to collectively as the **wrist complex** (Fig. 9–1A, B). Each joint proximal to the wrist complex serves to broaden the placement of the hand in space and to increase the degrees of freedom available to the hand. The shoulder serves as a dynamic base of support; the elbow allows the hand to approach or extend away from the body; and the forearm adjusts the approach of the hand to an object. The carpus, unlike the more proximal joints, serves placement of the hand in space to only a minor degree. The major contribution of the wrist complex is to control length-tension relationships in the multiarticular hand muscles and to allow fine adjustment of grip.[4,5] The wrist muscles appear to be designed for balance and control rather than for maximizing torque production.[6] The adjustments in the length-tension relationship of the extrinsic hand muscles that occur at the wrist cannot be replaced by compensatory movements of the shoulder, elbow, or forearm (radioulnar joint). The wrist has been called the most complex joint of the body, from both an anatomical and physiological perspective.[7] The intricacy and variability of the interarticular and intra-articular relationships within the wrist complex are such that the wrist has received a large amount of attention with agreement on relatively few points. Two points on which there appears to be consensus are (1) that the structure and biomechanics of the wrist, as well as of the hand, vary tremendously from person to person; and (2) that even subtle variations can produce differences in the way a given function occurs. The intent of this chapter, therefore, is less to provide details on what is "normal" and more to describe the wrist complex and hand in such a way that general structure is clear and a conceptual framework is developed within which normal function and pathology can be understood.

The wrist complex as a whole is considered biaxial, with motions of **extension/flexion** around a coronal axis and **ulnar deviation/radial deviation** around an anteroposterior axis. Some investigators argue that some degree of **pronation/supination** may also be found, especially at the radiocarpal joint.[8] The ranges of motion (ROMs) of the entire complex are variable and reflect the differences in carpal kinematics that arise from such factors as ligamentous laxity, the shape of articular surfaces, and the constraining effects of muscles.[9] Normal ranges are cited as a varying 65° to 85° of flexion, 60° to 85° of extension, 15° to 21° of radial deviation, and 20° to 45° of ulnar deviation.[10–13] The ranges are contributed in various proportions by the compound radiocarpal and midcarpal joints. Gilford and colleagues proposed that the two-joint, rather than single-joint, system of the wrist complex (1) permitted large ROMs with less exposed articular surface and tighter joint capsules, (2) had less tendency for structural pinch at extremes of ranges, and (3) allowed for flatter multijoint surfaces that are more capable of withstanding imposed pressures.[14]

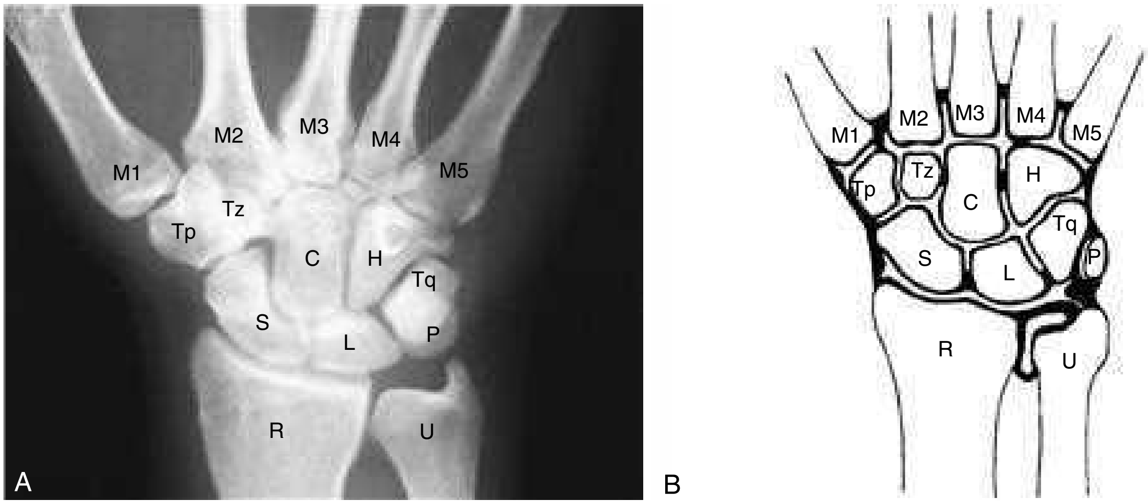

Figure 9–1 Wrist complex as shown on radiograph **(A)** and in a schematic representation **(B).** The radiocarpal joint is composed of the radius and the radioulnar disc, with the scaphoid (S), lunate (L), and the triquetrum (Tq). The midcarpal joint is composed of the scaphoid, lunate, and triquetrum with the trapezium (Tp), the trapezoid (Tz), the capitate (C), and the hamate (H).

Concept Cornerstone 9-1

Nomenclature

As is true of many other joints of the body, there are variations in nomenclature for the wrist and hand. *Flexion/extension* of the wrist may also be termed *volar (palmar) flexion/dorsiflexion*, respectively. Radial/ulnar deviation of the wrist may also be called *abduction/adduction*, respectively. At both the wrist and with joints and structures in the hand, the terms *volar* and *palmar* are used virtually interchangeably, whereas reference to the posterior aspect of the hand is more consistently referred to as the *dorsum*. The terms *medial* and *lateral* may be used in lieu of *ulnar* and *radial*. We will use *flexion/extension* and *radial/ulnar deviation* for the wrist motions, although coronal plane motions of the fingers are referred to most commonly (and we will follow this convention) as *abduction/adduction*. The terms *volar* and *palmar* will be used interchangeably in order to accurately represent terms found in the cited literature.

Radiocarpal Joint Structure

The radiocarpal joint is formed by the **radius** and **radioulnar disc** as part of the **triangular fibrocartilage complex (TFCC)** proximally and by the **scaphoid, lunate,** and **triquetrum** distally (see Fig. 9–1A, B).

Proximal and Distal Segments of the Radiocarpal Joint

The distal radius has a single, continuous, biconcave curvature that is long and shallow from side to side (in the frontal plane) and shorter and sharper anteroposteriorly (in the sagittal plane). The proximal joint surface is composed of (1) the lateral radial facet, which articulates with the scaphoid; (2) the medial radial facet, which articulates with the lunate; and (3) the triangular fibrocartilage complex, which articulates predominantly with the triquetrum, although it also has some contact with the lunate in the neutral wrist. The radioulnar disc, a component of the triangular fibrocartilage complex, also serves as part of the distal radioulnar joint, as discussed in the previous chapter. As a whole, the compound proximal radiocarpal joint surface is oblique and angled slightly volarly and ulnarly. The average inclination of the distal radius is 23°. This inclination occurs because the radial length (height) is 12 mm greater on the radial side than on the ulnar side[15] (Fig. 9–2A). The distal radius is also tilted 11° volarly[15] (Fig. 9–2B), with the posterior radius slightly longer than the volar radius.

The triangular fibrocartilage complex consists of the radioulnar disc and the various fibrous attachments that provide the primary support for the distal radioulnar joint (Fig. 9–3).[16] Although the attachments attributed to the triangular fibrocartilage complex vary somewhat, Mohiuddin and Janjua,[17] Benjamin and colleagues,[18] and Palmer[19] provided descriptions that represent a reasonable consensus. The articular disc is a fibrocartilaginous continuation of the articular cartilage of the distal radius. The disc is connected medially via two dense, fibrous connective tissue laminae. The upper laminae include the **dorsal** and **volar radioulnar ligaments,** which attach to the ulnar head and ulnar styloid. The lower lamina has connections to the sheath of the **extensor carpi ulnaris (ECU)** tendon and to the triquetrum, **hamate,** and the base of the fifth metacarpal through fibers from the **ulnar collateral ligament.** The so-called **meniscus homolog** is a region of irregular connective tissue that lies within and is part of the lower lamina, which traverses volarly and ulnarly from the dorsal radius to insert on the triquetrum. Along its path, the meniscus homolog has fibers that insert into the ulnar styloid and contribute to the formation of the **prestyloid recess.**[20] The medial (ulnar) connective tissue structures may exist in lieu of more extensive fibrocartilage because connective tissue is more compressible than fibrocartilage and thus are less likely to limit ROM.[17] Overall, the triangular fibrocartilage complex should be considered to function at the wrist as an extension of the distal radius, just as it does at the distal radioulnar joint.

The scaphoid, lunate, and triquetrum compose the proximal carpal row (see Fig. 9–1A, B). The proximal carpal row articulates with the distal radius. These bones are interconnected by two ligaments that, like the carpals themselves,

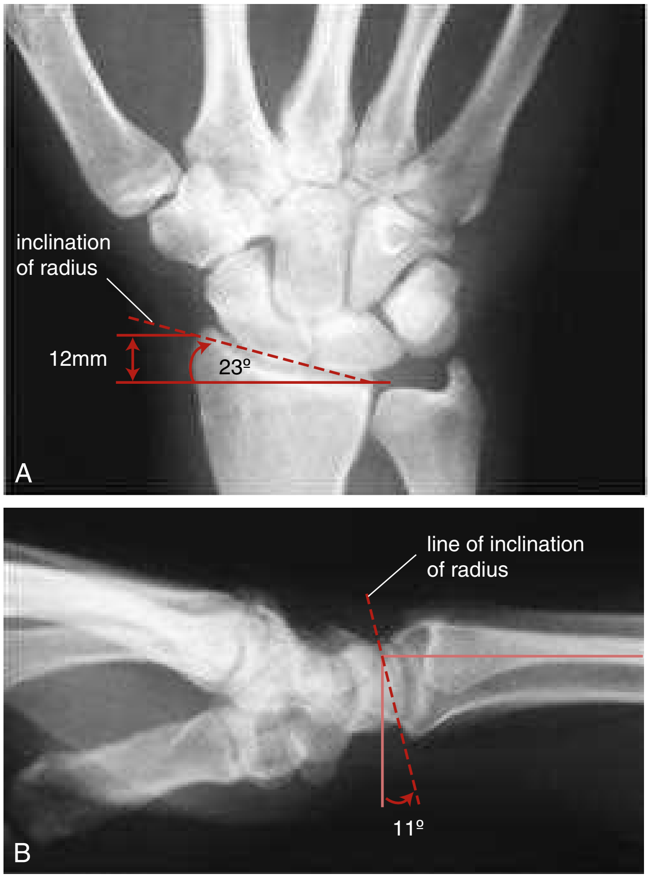

Figure 9–2 **A.** A normal angle of 23° of inclination of the radius in the frontal plane, with the distal radius about 12 mm long on the radial side than on the ulnar side. **B.** A normal angulation of inclination of about 11° of the radius volarly in the sagittal plane.

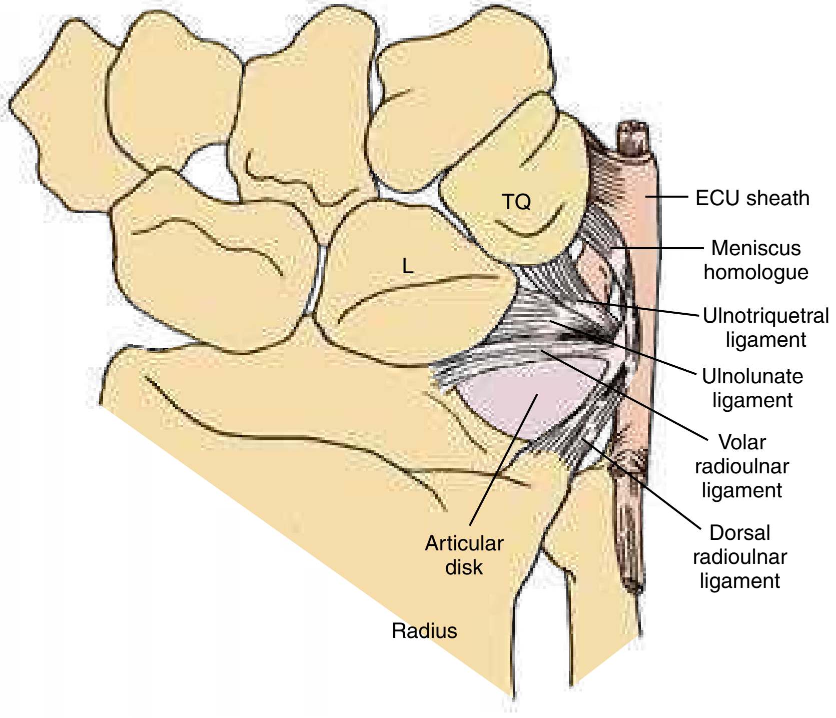

Figure 9–3 The triangular fibrocartilage complex, including the articular disc with its various fibrous attachments, which provide support to the distal radioulnar joint. L, lunate; TQ, triquetrum.

are covered with cartilage proximally.[21] These are the **scapholunate interosseous** and the **lunotriquetral interosseous ligaments.** The proximal carpal row and ligaments together appear to be a single biconvex cartilage-covered joint surface that, unlike a rigid segment, can change shape somewhat to accommodate to the demands of space between the forearm and hand.[22] The **pisiform,** anatomically part of the proximal row, does not participate in the radiocarpal articulation. The pisiform functions entirely as a sesamoid bone, presumably to increase the moment arm of the **flexor carpi ulnaris (FCU)** tendon that envelops it. The curvature of the distal radiocarpal joint surface is sharper than the proximal joint surface in both the sagittal and coronal planes, which makes the joint somewhat incongruent. The concept of articular incongruence is supported by the finding that the overall contact between the proximal and distal radiocarpal surfaces is typically only about 20% of available surface, with never more than 40% of available surface in contact at any one time.[23] Joint incongruence and the angulation of the proximal joint surface result in a greater range of flexion than extension[24] and in greater ulnar deviation than radial deviation for the radiocarpal joint.[21] The total range of flexion/extension is greater than the total range of radial/ulnar deviation. Incongruence and ligamentous laxity may account for as much as 45° of combined passive pronation/supination at the radiocarpal and midcarpal joints together,[8] although this motion is rarely considered to be an additional degree of freedom available to the wrist complex.

Not only do the curvature and inclination of the radiocarpal surfaces affect function, but the length of the ulna in relation to the radius is also a factor.[25,26] **Ulnar negative variance** is described as a short ulna in comparison with the radius at the distal end, whereas in **ulnar positive variance,** the distal ulna is long in relation to the distal radius (Fig. 9–4).[27] An ulnar positive variance has been associated with changes in the triangular fibrocartilage complex thickness.[28] When an axial (longitudinal compressive) load is applied to the wrist, the scaphoid and lunate receive approximately 80% of the load, whereas the triangular fibrocartilage complex receives approximately 20%.[16,23,29,30] At the distal radius, 60% of the contact is made with the scaphoid and 40% with the lunate.[23]

9-1 Patient Case *case*

Distal Radius Fracture

Gail Angeles sustained a right distal radius fracture after a fall on an outstretched hand (known by the acronym FOOSH). The posteroanterior (P-A) view in the radiograph illustrates how there is a loss in length of the radius and the normal radial inclination is diminished (Fig. 9-5A). The normal volar inclination of the distal radius is now dorsally angulated in the postreduction lateral radiograph (see Fig. 9-5B). These changes would be likely to result in a loss of ROM and the likelihood of future joint degeneration. Restoring the articular surfaces to near-anatomical position (and correcting the relative lengths of the radius and ulna) would most likely require open reduction and internal fixation (ORIF) with plate and screws.

With an ulnar positive variance, there is a potential for impingement of the triangular fibrocartilage complex structures between the distal ulna and the triquetrum.[25] Palmer and colleagues found an inverse relationship between the thickness of the triangular fibrocartilage complex and ulnar variance, with positive ulnar variance associated with a thinner triangular fibrocartilage complex and negative ulnar variance with a relatively thicker triangular fibrocartilage

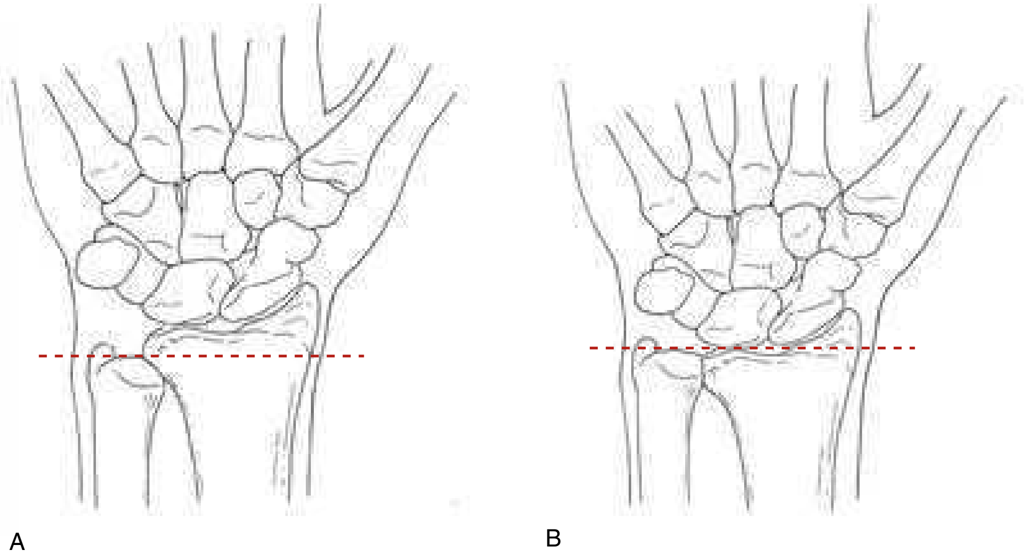

Figure 9–4 Ulnar variance: negative **(A)** and positive **(B).**

complex.[31] A relatively "long" ulna may be present after a distal radius fracture (see Fig. 9–5A) that healed in a shortened position. Pain is commonly present with end-range pronation and ulnar deviation because these motions increase the likelihood of impingement of the ulnar structures. Surgical intervention may include a joint-leveling procedure such as ulnar shortening to unload the ulnar side of the wrist.[32]

In contrast to ulnar positive variance, ulnar negative variance (a relatively short ulna) may result in abnormal force distribution across the radiocarpal joint with potential degeneration at the radiocarpal joint.[26] **Avascular necrosis** of the lunate, **Kienbock's disease** (Fig. 9–6), has been associated with ulnar negative variance.[32,33] Treatment options include unloading of the radiocarpal joint by lengthening the ulna, shortening the radius, or fusing select carpal bones.[34]

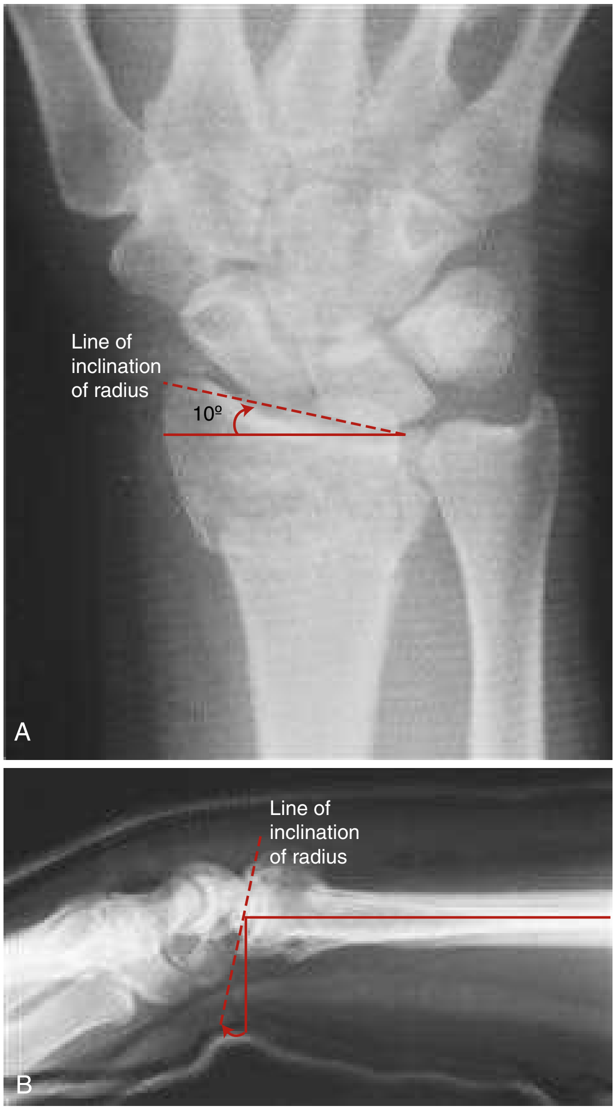

Figure 9–5 A radial fracture from a fall on an outstretched hand resulting in diminished angulation (and length) of the distal radius **(A).** Relative shortening of the radius results in an increased ulnar variance, as well as a reversal of the normal volar inclination of the radius **(B).**

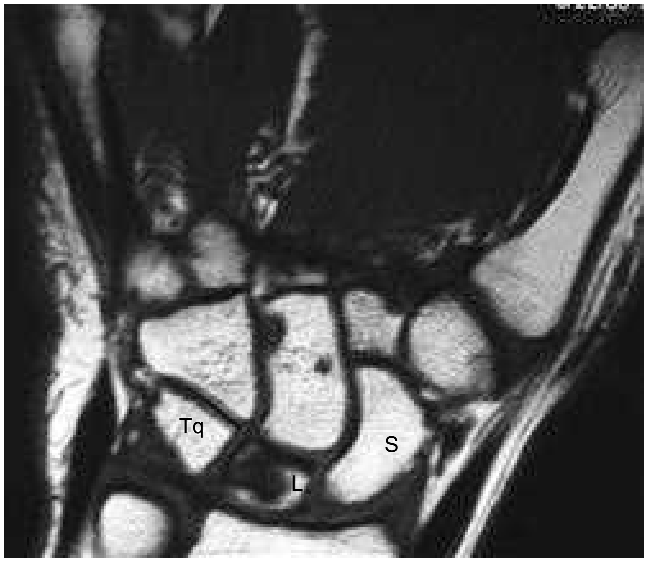

Figure 9–6 Avascular necrosis of the lunate seen in this magnetic resonance image (MRI) is known as Kienbock's disease and has been associated with negative ulnar variance. L, lunate; S, scaphoid; Tq, triquetrum.

Radiocarpal Capsule and Ligaments

The radiocarpal joint is enclosed by a strong but somewhat loose capsule and is reinforced by capsular and intracapsular ligaments. Most ligaments that cross the radiocarpal joint also contribute to stability at the midcarpal joint, and so all the ligaments will be presented together after introduction of the midcarpal joint. Similarly, the muscles of the radiocarpal joint also function at the midcarpal joint. In fact, the radiocarpal joint is not crossed by any muscles that act on the radiocarpal joint alone. The flexor carpi ulnaris is the only muscle that crosses the radiocarpal joint and attaches to any of the bones of the proximal carpal row. Although fibers of the flexor carpi ulnaris tendon end on the pisiform, the pisiform is only loosely connected to the triquetrum below.[35] Consequently, forces applied to the pisiform by the flexor carpi ulnaris muscle are translated not to the triquetrum on which it sits but to the hamate and fifth metacarpal via pisiform ligaments. Motions occurring at the radiocarpal joint are a result of forces applied by the abundant passive ligamentous structures and by muscles that are attached to the distal carpal row and metacarpals. Consequently, movements of the radiocarpal and midcarpal joints must be examined together.

Midcarpal Joint Structure

The midcarpal joint is the articulation between the scaphoid, lunate, and triquetrum proximally and the distal carpal row composed of the **trapezium, trapezoid, capitate,** and hamate (see Fig. 9–1B). The midcarpal joint is a functional rather than an anatomical unit because it does not form a single uninterrupted articular surface. However, it is anatomically separate from the radiocarpal joint and has a fibrous capsule and synovial lining that is continuous with each intercarpal articulation and may be continuous with some of the **carpometacarpal (CMC) joints.**[21] The midcarpal joint surfaces are complex, with an overall reciprocally concave-convex configuration. The complexity of surfaces and ligamentous connections, however, forces simplification of the conceptualization of its movements. Functionally, the carpals of the distal row (with their attached metacarpals) move as an almost fixed unit. The capitate and hamate are most strongly bound together with, at most, a small amount of play between them.[36–38] The union of the distal carpals also results in nearly equal distribution of loads across the scaphoid-trapezium-trapezoid, the scaphoid-capitate, the lunate-capitate, and the triquetrum-hamate articulations.[23,39] Together the bones of the distal carpal row contribute two degrees of freedom to the wrist complex, with varying amounts of radial/ulnar deviation and flexion/extension credited to the joint. The excursions permitted by the articular surfaces of the midcarpal joint generally favor the range of extension over flexion and radial deviation over ulnar deviation—the opposite of what was found for the radiocarpal joint.[21,24,40] The functional union of the distal carpals with each other and with their contiguous metacarpals not only serve the wrist complex but also are the foundation for the transverse and longitudinal arches of the hand, which will be addressed in detail later.[37]

Ligaments of the Wrist Complex

The tremendous individual differences that exist in the structure of the carpus can perhaps best be appreciated after a review of the ligaments of the wrist. There are substantive differences in names, anatomical descriptions, and ascribed functions from investigator to investigator.[41–44] We will present the work of Taleisnik to organize and describe the wrist ligamentous anatomy.[45,46] Although there may not be universal agreement on the structure and function of individual ligaments, there is consensus that the ligamentous structure of the carpus is responsible not only for articular stability but also for guiding and checking motion between and among the carpals.[47] When we examine the function of the wrist complex, we shall see that the variability of ligaments will, among other factors, translate into substantial and widely acknowledged differences among individuals in movement of the joints of the wrist complex. In general, the dorsal wrist ligaments are described as thin, whereas the more numerous volar ligaments are thicker and stronger.[45,46]

The ligaments of the wrist complex are designated either extrinsic or intrinsic.[41,45,46] The **extrinsic ligaments** are those that connect the carpals to the radius or ulna proximally or to the metacarpals distally; the **intrinsic ligaments** are those that interconnect the carpals themselves and are also known as intercarpal or interosseous ligaments. Nowalk and Logan found the intrinsic ligaments to be stronger and less stiff than the extrinsic ligaments.[41] They concluded that the intrinsic ligaments lie within the synovial lining and, therefore, must rely on synovial fluid for nutrition rather than contiguous vascularized tissues, such as the extrinsic ligaments. The extrinsic ligaments, therefore, are more likely to fail but also have better potential for healing and help protect the slower to heal intrinsic ligaments by accepting forces first.[41]

Volar Carpal Ligaments

On the volar surface of the wrist complex, the numerous intrinsic and extrinsic ligaments are variously described by either composite or separate names, depending on the investigator. Taleisnik organized the volar extrinsic ligaments into two groupings: the radiocarpal and the ulnocarpal ligaments. The composite ligament known as the **volar radiocarpal ligament** is described most commonly as having three distinct bands: the **radioscaphocapitate (radiocapitate)**, short and long **radiolunate (radiolunotriquetral)**, and **radioscapholunate ligaments** (Fig. 9–7A).[45–47] The radioscapholunate ligament was once described as the most important stabilizer of the proximal pole of the scaphoid, and disruption of it may lead to issues of scaphoid instability[46]; however, current research reveals that this structure offers little support to the joint but acts as a conduit for neurovascularity to the scapholunate joint.[47] The **radial collateral ligament** may be considered an extension of the volar radiocarpal ligament and capsule.[48] Nowalk and Logan[41] identified the radiocapitate as an extrinsic ligament, whereas Blevens and colleagues[43] identified it as part of the "palmar intracapsular radiocarpal ligaments." The **ulnocarpal ligament complex** is composed of the

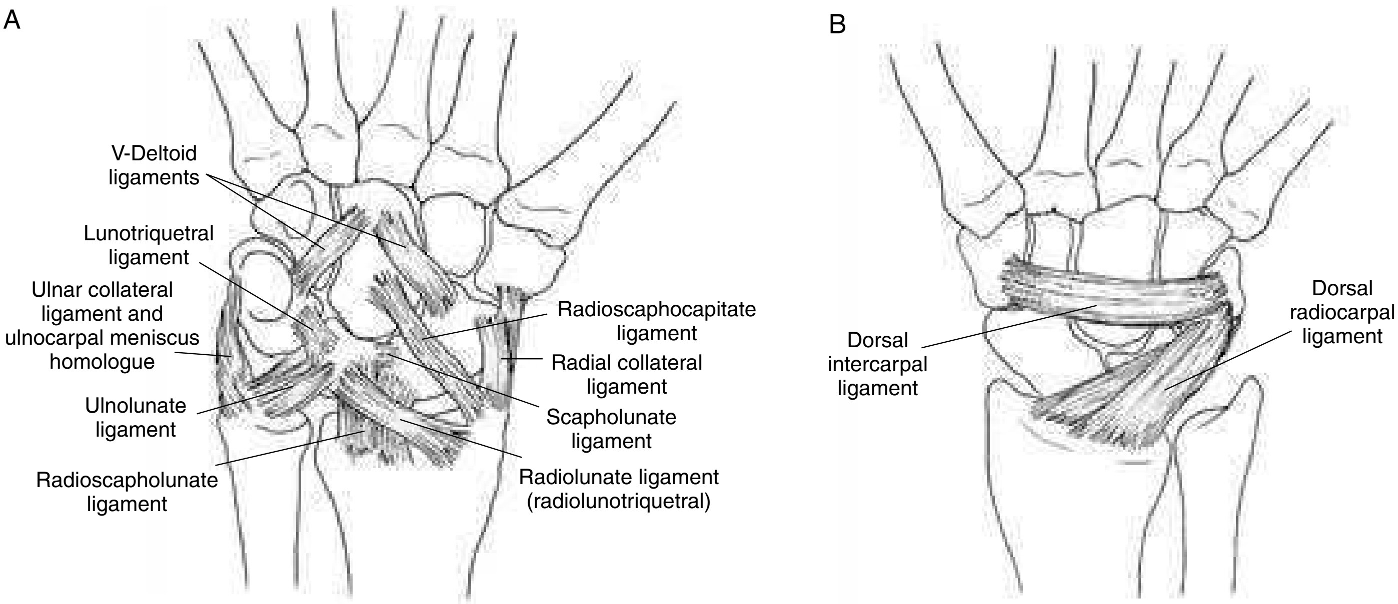

Figure 9–7 **A.** Volar ligaments of the wrist complex, including the three bands of the volar radiocarpal ligament: radioscaphocapitate, radiolunate, and radioscapholunate. The two intrinsic ligaments (scapholunate and lunotriquetral) are credited with maintaining scaphoid stability. **B.** Dorsal wrist ligaments form a horizontal V, adding to radiocarpal stability.

triangular fibrocartilage complex (including the articular disc and meniscus homolog), the **ulnolunate ligament,** and the ulnar collateral ligament.[20,48]

Two volar intrinsic ligaments have received particular attention and acknowledgment of their importance to wrist function. The first of these, the scapholunate interosseous ligament, is generally, although not universally,[49] credited with being a key factor in maintaining scaphoid stability and, therefore, stability of much of the wrist.[43,50–52] Studies have shown that the dorsal portion of this ligament is the most important in terms of contributing to stability.[47] Injury to this ligament appears to contribute largely to scaphoid instability and, therefore, to one of the most common wrist problems.[53] As an intrinsic ligament, however, the scapholunate interosseous ligament is largely avascular and, therefore, may be susceptible to degenerative change.[54] The second key intrinsic ligament is the lunotriquetral interosseous ligament. This ligament is credited with maintaining stability between the lunate and triquetrum. Injury to this ligament appears to contribute to lunate instability, another problematic wrist pathology.[55,56] However, this instability pattern will most likely not occur without concomitant injury to the extrinsic ligaments. In general, the volar wrist ligaments are placed on stretch with wrist extension.[57]

Dorsal Carpal Ligaments

Dorsally, the major wrist ligament is the **dorsal radiocarpal ligament** (Fig. 9–7B). This ligament, as is true of the volar radiocarpal, varies somewhat in description but is obliquely oriented.[45,46] Essentially, the ligament as a whole converges on the triquetrum from the distal radius, with possible attachments along the way to the lunate and lunotriquetral interosseous ligament.[44,58,59] Garcia-Elias suggested that the obliquity of the volar and dorsal radiocarpal ligaments helps offset the sliding of the proximal "carpal condyle" on the inclined radius.[60] A second dorsal ligament is the **dorsal intercarpal ligament,** which courses horizontally from the triquetrum to the lunate, scaphoid, and trapezium.[48,58] The two dorsal ligaments together form a horizontal V that contributes to radiocarpal stability, notably stabilizing the scaphoid during wrist ROM.[45,58,59] The dorsal wrist ligaments are taut with wrist flexion.[57]

Concept Cornerstone 9-2

Summary of Ligaments

- Extrinsic Ligaments
  - Radiocarpal
    - Radial collateral
    - Volar collateral
      - Superficial
      - Deep
        - Radioscaphocapitate
        - Radiolunate (radiolunotriquetral)
        - Radioscaphoid-lunate
    - Ulnocarpal
      - Triangular fibrocartilage complex
      - Meniscus homolog
      - Ulnolunate
      - Ulnar collateral
    - Dorsal radiocarpal (radiotriquetral)
- Intrinsic Ligaments
  - Short
    - Volar
    - Dorsal
    - Interosseous

Continued

Intermediate
  Lunotriquetral
  Scapholunate
  Scaphotrapezium
Long
  Volar intercarpal (v-ligament, deltoid)
  Dorsal intercarpal

Function of the Wrist Complex

Movements of the Radiocarpal and Midcarpal Joints

Motions at the radiocarpal and midcarpal joints are caused by a rather unique combination of active muscular and passive ligamentous and joint reaction forces. Although there are abundant passive forces on the proximal carpal row, no muscular forces are applied directly to the articular bones of the proximal row, given that the flexor carpi ulnaris muscle applies its force via the pisiform to the more distal bones. The proximal carpals, therefore, are effectively a mechanical link between the radius and the distal carpals and metacarpals to which the muscular forces are actually applied. Gilford and colleagues suggested that the proximal carpal row is an **intercalated segment,** a relatively unattached middle segment of a three-segment linkage.[14] Ruby and associates concurred, hypothesizing that the proximal carpal row functions as an intercalated segment between the distal radius/triangular fibrocartilage complex and the relatively immobile distal row.[61] When compressive forces are applied across an intercalated segment, the middle segment tends to collapse and move in the opposite direction from the segments above and below. For example, application of compressive muscular extensor forces across the biarticular wrist complex would cause an unstable proximal scaphoid to collapse into flexion while the distal carpal row extended. An intercalated segment requires some type of stabilizing mechanism to normalize combined midcarpal/radiocarpal motion and prevent collapse of the middle segment (the proximal carpal row). The stabilization mechanism appears to involve the scaphoid's functional and anatomical (ligamentous) connections both to the adjacent lunate and to the distal carpal row.

Garcia-Elias supported the hypothesis that the stability of the proximal carpal row depends on the interaction of two opposite tendencies when the carpals are axially loaded (compression across a neutral wrist); the scaphoid tends to flex, whereas the lunate and triquetrum tend to extend.[60] These counterrotations within the proximal row are prevented by the ligamentous structure (including the key scapholunate interosseous and lunotriquetral interosseous ligaments). Linking the scaphoid to the lunate and triquetrum through ligaments will, according to Garcia-Elias, cause the proximal carpals to "collapse synchronously" into flexion and pronation, whereas the distal carpals move into extension and supination.[60] Garcia-Ellis proposed that the counterrotation between proximal and distal carpal rows and the resulting ligamentous tension increase coaptation of midcarpal articular surfaces and add to stability.

Although the carpal stability mechanism proposed by Garcia-Elias appears to hold as a conceptual framework, findings of other investigators differ in detail if not in substance. Advances in technology, including computer modeling, suggest that intercarpal motion is far more complex and individualistic than was once thought.[62,63] There is general agreement that the three bones of the proximal carpal row do not move as a unit but that motions of the three carpals vary both in magnitude and in direction with axial loading, with radiocarpal flexion/extension, and with radial/ulnar deviation.[7,46,60,64,65] In fact, Short and colleagues[66] found that carpal motions differed not only with individual osteoligamentous configuration and position but also with direction of motion; that is, relations in the carpus differed when the wrist reached neutral position, depending on whether the position was reached from full flexion, full extension, or deviation.

Controversy remains in terms of the existence of an actual "center of rotation" of the wrist complex. Much of the literature proposes that the head of the capitate, frequently referred to as the "keystone" of the wrist, may serve as the location of the coronal axis for wrist extension/flexion and the A-P axis for radial/ulnar deviation,[40] as well as providing the rigid center of the fixed carpal arch.[57] Also of importance is the scaphoid and its movement with the capitate, most notably with wrist extension.[67] Neu and associates studied the kinematics of the capitate with wrist motion in both planes and concluded that the axes of motion are not constant, which further supports the premise that carpal kinematics are complex and vary depending on the individual.[68]

Flexion/Extension of the Wrist

During flexion/extension of the wrist, the scaphoid seems to show the greatest motion of the three proximal carpal bones, whereas the lunate moves least.[7,38] Some investigators found that flexion and extension of the radiocarpal joint occurs almost exclusively as isolated flexion and extension of the proximal carpal row.[51,66] Others, however, found simultaneous but lesser amounts of radial/ulnar deviation and pronation/supination of two or all three proximal carpal bones during radiocarpal flexion/extension.[7,21,57] Motion of the more tightly bound distal carpals and their attached metacarpals during midcarpal flexion/extension appears to be a fairly simple flexion and extension, with movement of the distal segments proportional to movement of the hand.[64]

In view of the apparent variability of findings, a conceptual framework for flexion/extension of the wrist is in order. The following sequence of events (Fig. 9–8A) was proposed by Conwell[69] and provides an explanation of the relative motions of the various segments and of their interdependence. It can easily be appreciated, however, that the conceptual framework is oversimplified and ignores some of the simultaneous interactions that occur among the key carpal bones.

1. The motion in this conceptual framework begins with the wrist in full flexion. Active extension is initiated at the distal carpal row and at the firmly attached metacarpals by

Figure 9–8 **A.** As wrist extension is initiated from full flexion, (1) the distal carpal row moves on the proximal carpal row; (2) the scaphoid and distal row move on the lunate/triquetrum; and (3) the carpals move as a unit on the radius and triangular fibrocartilage complex to achieve full wrist extension **(B)**. C, capitate; L, lunate; S, scaphoid.

the wrist extensor muscles attached to those bones. The distal carpals (capitate, hamate, trapezium, and trapezoid) glide on the relatively fixed proximal bones (scaphoid, lunate, and triquetrum). Although the surface configurations of the midcarpal joint are complex, the distal carpal row effectively glides in the same direction as motion of the hand. When the wrist complex reaches neutral (the long axis of the third metacarpal in line with the long axis of the forearm), the ligaments spanning the capitate and scaphoid draw the capitate and scaphoid together into a close-packed position.

2. Continued extensor force now moves the combined unit of the distal carpal row and the scaphoid on the relatively fixed lunate and triquetrum. At approximately 45° of extension of the wrist complex, the scapholunate interosseous ligament brings the scaphoid and lunate into close-packed position. This unites all the carpals and causes them to function as a single unit.
3. Completion of wrist complex extension (Fig. 9–8B) occurs as the proximal articular surface of the carpals move as a relatively solid unit on the radius and triangular fibrocartilage complex. All ligaments become taut as full extension is reached and the entire wrist complex is close packed.[70]

Wrist motion from full extension to full flexion occurs in the reverse sequence. In the context of this conceptual framework, the scaphoid (through mediation of the wrist ligaments) participates at different times in scaphoid-capitate, scaphoid-lunate, or radio-scaphoid motion. Crumpling of the proximal carpal row (intercalated segment) is prevented, and full ROM is achieved. Interestingly, computer modeling and cadaver study of radiocarpal intra-articular contact patterns showed that radiocarpal extension is accompanied by increased contact dorsally. One would expect extension of the hand to be accompanied by sliding of the convex proximal carpal surface volarly in a direction opposite to hand motion. If this contact pattern exists in vivo, it likely reflects the complexity of radiocarpal motion and may contradict assumptions about movement between convex and concave surfaces.[23]

Radial/Ulnar Deviation of the Wrist

Radial and ulnar deviation of the wrist seems to be an even more complex, but perhaps less varied, motion than is flexion/extension. The proximal carpal row displays a unique "reciprocal" motion with radial and ulnar deviation.[12] In radial deviation, the carpals slide ulnarly on the radius (Fig. 9–9A). The carpal motion not only produces reciprocal deviation of the proximal and distal carpals with radial deviation, but simultaneous flexion of the proximal carpals and extension of the distal carpals (with observations of accompanying pronation/supination components varying among investigators).[7,22,29,66,71] The opposite motions of the proximal and distal carpals occur with ulnar deviation (Fig. 9–9B). During radial/ulnar deviation, the distal carpals, once again, move as a relatively fixed unit while the magnitude of motion between the bones of the proximal carpal row may differ.[7,38] Garcia-Elias and colleagues found that the magnitude of scaphoid flexion during radial deviation (and extension during ulnar deviation) was related to ligamentous laxity.[9] Volunteer subjects with ligamentous laxity showed more scaphoid flexion/extension and less radial/ulnar deviation than did others. Ligamentous laxity was more common among women than among men. The investigators proposed that ligamentous laxity led to less binding of the scaphoid to the distal carpal row and, therefore, more out-of-plane motion for the scaphoid.

In full radial deviation, both the radiocarpal and midcarpal joints are in close-packed position.[40,72–74] The ranges of wrist complex radial and ulnar deviation are greatest when the wrist is in neutral flexion/extension.[75] When the wrist is extended and is in close-packed position, the carpals are all locked, and very little radial or ulnar deviation is possible. In wrist flexion, the joints are loose packed and the bones are splayed. Further movement of the proximal row cannot occur, and, as in extreme extension, little radial or ulnar deviation is possible in the fully flexed position.[76]

Continuing Exploration 9-1:

Functional Range of Motion

What appears to be a redundancy in function at the midcarpal and radiocarpal joints ensures maintenance of the minimum ROM required for activities of daily living. Brumfield and Champoux found that a series of hand activities necessary for independence required a functional

Continued

Figure 9–9 With radial deviation of the wrist **(A),** the flexion of the scaphoid makes the scaphoid appear shorter than when the scapoid extends during ulnar deviation **(B).** C, capitate; L, lunate; S, scaphoid.

wrist motion of 10° of flexion and 35° of extension.[77] Ryu and colleagues included a wide range of hand functions in their test battery and determined that all could be completed with minimum wrist motions of 60° extension, 54° flexion, 40° ulnar deviation, and 17° radial deviation.[13] There is consensus that wrist extension and ulnar deviation are most important for wrist activities. Wrist extension and ulnar deviation were also found to constitute the position of maximum scapholunate contact.[23] Given the key role of the scaphoid in wrist stability—acting as a link between the proximal and distal carpal rows—this extended and ulnarly deviated wrist position will provide a stable base that allows for maximum hand function distally. When deciding on the position of fusion for the wrist, the surgeon commonly chooses an optimal functional position of approximately 20° of extension and 10° of ulnar deviation.[57] This extended position also positions the long digital flexors for maximal force generation in prehension activities.

Wrist Instability

Injury to one or more of the ligaments attached to the scaphoid and lunate may diminish or remove the synergistic stabilization of the lunate and scaphoid.[78,79] When this occurs, the scaphoid behaves as an unconstrained segment, following its natural tendency to collapse into flexion on the volarly inclined surface of the distal radius (potentially including some out-of-plane motion as well). The base of the flexed scaphoid slides dorsally on the radius and subluxes. Released from scaphoid stabilization, the lunate and triquetrum together act as an unconstrained segment, following their natural tendency to extend. The muscular forces that bypass the proximal carpals and apply force to the distal carpals cause the distal carpals to flex on the extended lunate and triquetrum. The flexed distal carpals glide dorsally on the lunate and triquetrum, accentuating the extension of the lunate and triquetrum. This zigzag pattern of the three segments (the scaphoid, the lunate/triquetrum, and the distal carpal row) is known as *intercalated segmental instability*.[22,29] When the lunate assumes an extended posture, the presentation is referred to as **dorsal intercalated segmental instability (DISI)** (Fig. 9–10A). The scaphoid subluxation may be dynamic, occurring only with compressive loading of the wrist with muscle forces, or the subluxation may become fixed or static.[80] With subluxation of the scaphoid, the contact pressures between the radius and scaphoid increase because the contact occurs over a smaller area.[23,43] A dorsal intercalated segmental instability problem, therefore, may result over time in degenerative changes at the radioscaphoid joint and then, ultimately, at the other intercarpal joints.[42] With sufficient ligamentous laxity, the capitate may sublux dorsally off the extended lunate or, more commonly, migrate into the gap between the flexed scaphoid and extended lunate. The progressive degenerative problem from an untreated dorsal intercalated segmental instability is known as **scapholunate advanced collapse** (**SLAC** wrist).[43,81] The progressive stages have been identified radiographically on the basis of the time lapse from injury.[81,82] Although it is arguable whether the load between the scaphoid and the lunate increases or decreases with dorsal intercalated segmental instability,[23,35,51] there is agreement that the radiolunate articulation is less likely to show degenerative changes than is the radioscaphoid joint. The lesser tendency toward degenerative changes in the radiolunate joint has been

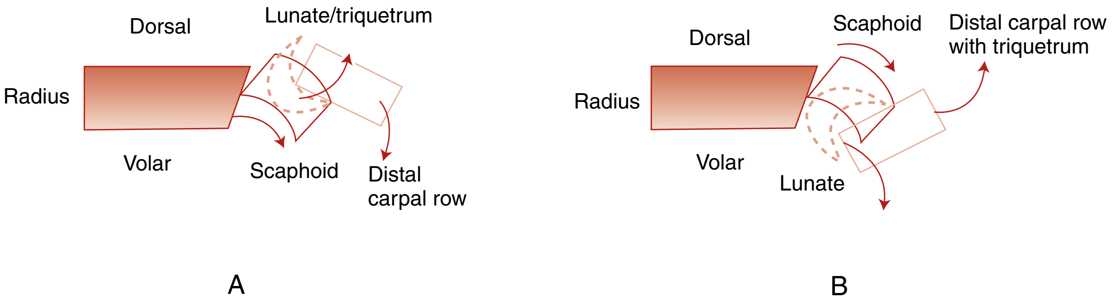

Figure 9–10 **A.** Dorsal intercalated segmental instability. The lunate, released from the flexed scaphoid, extends on the radius. The capitate moves in the opposite direction (flexion) on top of the lunate. **B.** Volar intercalated segmental instability. The lunate and scaphoid flex on the radius, whereas the triquetrum extends. The distal carpal row (capitate shown) follows the triquetrum into extension.

attributed to a more spherical configuration of the radiolunate facets that better center applied loads across the articular surfaces.[43]

The other common form of carpal instability occurs when the ligamentous union of the lunate and triquetrum is disrupted through injury.[22,80] The lunate and triquetrum together normally tend to move toward extension and offset the tendency of the scaphoid to flex. When the lunate is no longer linked with the triquetrum, the lunate and scaphoid together fall into flexion, and the triquetrum and distal carpal row extend (Fig. 9–10B). This ulnar perilunate instability is known as **volar intercalated segmental instability (VISI).**[46] This condition is not as common as dorsal intercalated segmental instability. The problems of volar intercalated segmental instability and dorsal intercalated segmental instability illustrate the importance of proximal carpal row stabilization to wrist function and of maintenance of the scaphoid as the bridge between the distal carpal row and the two other bones of the proximal carpal row.

9-2 Patient Case

Scapholunate Ligament Injury

Jeff O'Brien, playing in a men's softball league, sustained a fall on an outstretched hand (FOOSH) that resulted in pain and swelling on the dorsum of the wrist. A radiograph taken in a walk-in clinic a week later showed a separation between his scaphoid and lunate (Fig. 9–11A) that was indicative of ligamentous damage, including tear of the scapholunate interosseous ligament. It was recommended that Mr. O'Brien see a hand specialist. A follow-up radiograph with the hand surgeon showed dorsal intercalated segmental instability (DISI) (Fig. 9–11B). Jeff made the decision not to pursue any kind of treatment beyond a period of immobilization. The problem appeared to resolve with time. Five years later, however, pain in Jeff's wrist increased to the point at which he sought medical attention from the hand surgeon once again. The hand surgeon diagnosed scapholunate advanced collapse (SLAC). Repetitive loading of the wrist (grasping, lifting) caused degenerative bony changes in the wrist complex, wrist instability marked by proximal migration of the capitate into the space between the scaphoid and lunate, and degenerative changes in the radioscaphoid and capitate-lunate joints (Fig. 9–11C). A partial wrist fusion (scaphocapitate arthrodesis) was recommended to improve stability (removing one source of pain) while allowing limited wrist mobility to minimize loss of function.

Muscles of the Wrist Complex

The primary role of the muscles of the wrist complex is to provide a stable base for the hand while permitting positional adjustments that allow for an optimal length-tension relationship in the long finger muscles.[4,57] Information on a muscle's cross-sectional area and length of moment arm will help facilitate understanding of a muscle's specific action, force, and torque potential. Many researchers investigated the peak force that could be exerted at the **interphalangeal (IP) joints** of the fingers by the long finger flexors during different wrist positions. Some studies found that the greatest interphalangeal flexor force occurs with ulnar deviation of the wrist (neutral flexion/extension), whereas the least force occurred with wrist flexion (neutral deviation).[2,83] Other studies concluded that 20° to 25° of wrist extension with 5° to 7° of ulnar deviation was the optimal range to maximize grip strength output.[84,85] The muscles of the wrist, however, are not structured merely to optimize the force of finger flexion. If optimizing finger flexor force outweighed other concerns, one might expect the wrist extensors to be stronger than the wrist flexors. Rather, the work capacity (ability of a muscle to generate force per unit of cross-section) of the wrist flexors is more than twice that of the extensors. Again contrary to expectation if optimizing finger flexor force was the goal, the work capacity of the radial deviators slightly exceeds that of the ulnar deviators.[86] The function of the wrist muscles cannot be understood by looking at any one factor or function; it should be assessed by electromyography (EMG) in various patterns of use against the resistance of gravity and external loads. We will describe the wrist muscles here, but their function is best understood in the context of later discussion of the synergies between hand and wrist musculature.

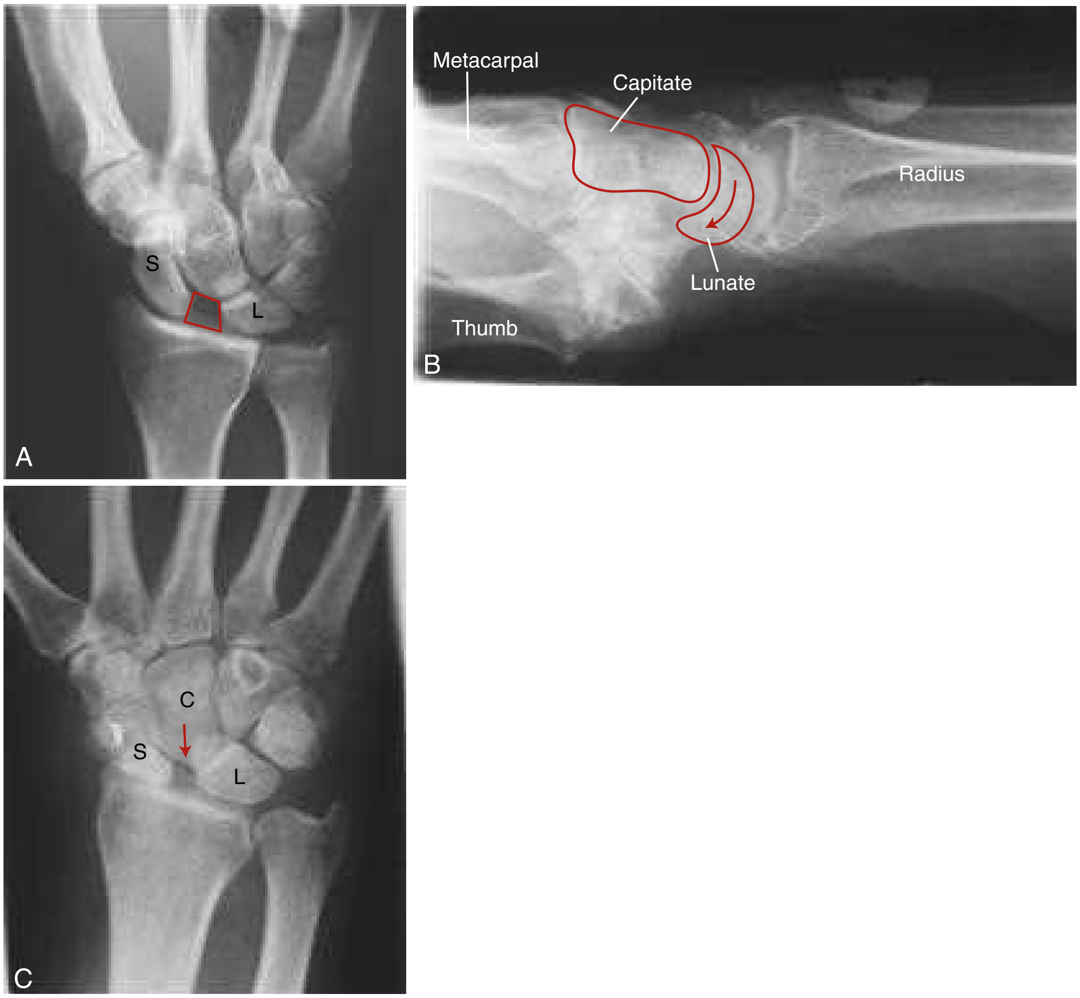

Figure 9–11 **A.** With disruption of the scapholunate ligaments through trauma, the scaphoid and lunate migrate apart, leaving a gap (diastasis). **B.** Dorsal intercalated segmental instability (DISI) results in dorsal tilt of the lunate (shown), as well as less evident volar tilt of the scaphoid and capitate. **C.** Scapholunate advance collapse (SLAC) with migration of the capitate proximally and erosion of the radioscaphoid and capitate-lunate joints.

Volar Wrist Musculature

Six muscles have tendons crossing the volar aspect of the wrist and, therefore, are capable of creating a wrist flexion movement (Fig. 9–12A). These are the **palmaris longus (PL),** the **flexor carpi radialis (FCR),** the flexor carpi ulnaris, the **flexor digitorum superficialis (FDS),** the **flexor digitorum profundus (FDP),** and the **flexor pollicis longus (FPL)** muscles. The first three of these muscles are primary wrist muscles. The last three are flexors of the digits with secondary actions at the wrist. At the wrist level, all of the volar wrist muscles pass beneath the **flexor retinaculum** along with the median nerve except the palmaris longus and the flexor carpi ulnaris muscles (Fig. 9–12B). The flexor retinaculum prevents bowstringing of the long flexor tendons, thereby contributing to maintaining an appropriate length-tension relationship. The flexor retinaculum is often considered to have a proximal portion and a distal portion, with the distal portion more commonly known as the **transverse carpal ligament (TCL).**

The positions of the flexor carpi radialis and flexor carpi ulnaris tendons in relation to the axis of the wrist indicate that these muscles can, respectively, radially deviate and ulnarly deviate the wrist, as well as flex. However, the flexor carpi radialis muscle does not appear to be effective as a radial deviator of the wrist in an isolated contraction. Its distal attachment on the bases of the second and third metacarpals places it in line with the long axis of the hand. Along with the palmaris longus muscle, the flexor carpi radialis muscle functions as a wrist flexor with little concomitant deviation.[11] The flexor carpi radialis muscle is active during radial deviation, however. The flexor carpi radialis muscle either augments the strong radial deviating force of the **extensor carpi radialis longus (ECRL)** or offsets the extension also produced by the extensor carpi radialis longus muscle. The palmaris longus muscle is a wrist flexor without producing either radial or ulnar deviation. The palmaris longus muscle and tendon are absent unilaterally or bilaterally in approximately 14% of people without any apparent strength or functional deficit.[87] Given its apparent redundancy with other muscles, the palmaris longus tendon (when present) may be “sacrificed” for surgical reconstruction of other structures.[32]

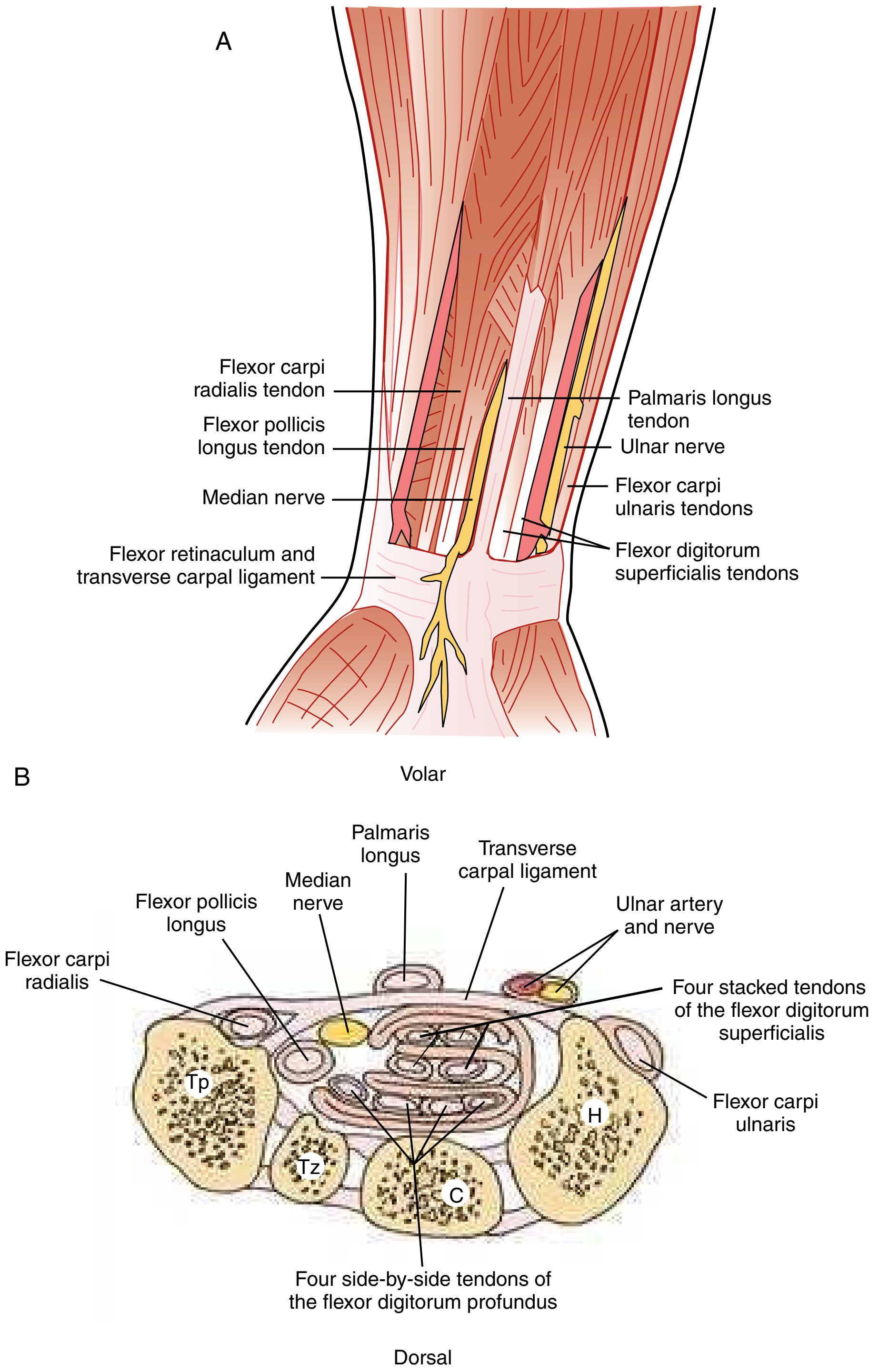

Figure 9–12 **A.** The tendons and nerves of the primary and secondary wrist flexors lie on the volar aspect of the wrist. All but the palmaris longus tendon, the ulnar nerve, and the flexor carpi ulnaris muscle pass beneath the flexor retinaculum. **B.** On cross-section, the relationship of the tendons and nerves to the transverse carpal ligament is more evident. The flexor pollicis longus is encased in its own tendon sheath (or radial bursa), whereas the four deep tendons of the flexor digitorum profundus and the four more superficial stacked tendons of the flexor digitorum superficialis are wrapped by folds in the ulnar bursa.

The flexor carpi ulnaris muscle envelops the pisiform, a sesamoid bone that increases the moment arm of the flexor carpi ulnaris muscle for flexion. The flexor carpi ulnaris muscle can act on the hamate and fifth metacarpal indirectly through the pisiform's ligaments,[35] effectively producing flexion and ulnar deviation of the wrist complex. The flexor carpi ulnaris tendon crosses the wrist at a greater distance from the axis for wrist radial/ulnar deviation than does the flexor carpi radialis muscle, so the flexor carpi ulnaris muscle is more effective in its ulnar deviation function than is the flexor carpi radialis muscle is in its radial deviation function.[4] The flexor carpi ulnaris muscle is able to exert the greatest tension of all the wrist muscles, giving it particular functional relevance, especially with activities requiring high ulnar deviation forces such as chopping wood.[4]

The flexor digitorum superficialis and flexor digitorum profundus muscles are predominantly flexors of the fingers, and the flexor pollicis longus muscle is predominantly the flexor of the thumb. As multijoint muscles, their capacity to produce an effective wrist flexion force depends on synergistic stabilization by the extensor muscles of the more distal joints that these muscles cross to prevent excessive shortening of the muscles over multiple joints. If these muscles attempt to shorten over both the wrist and the more distal joints, the muscles will become actively insufficient. The flexor digitorum superficialis and flexor digitorum profundus muscles show varied activity in wrist radial/ulnar deviation, as might be anticipated from the central location of the tendons. The flexor digitorum superficialis muscle seems to function more consistently as a wrist flexor than does the flexor digitorum profundus muscle.[88] This is logical, because the flexor digitorum profundus muscle is a longer, deeper muscle, crosses more joints, and is therefore more likely to become actively insufficient. The effect of the flexor pollicis longus muscle on the wrist has received relatively little attention. The position of the tendon suggests the ability to contribute to both flexion and radial deviation of the wrist if its more distal joints are stabilized.

Dorsal Wrist Musculature

The dorsum of the wrist complex is crossed by the tendons of nine muscles (Fig. 9–13). Three of the nine muscles are primary wrist muscles: the extensor carpi radialis longus, the **extensor carpi radialis brevis (ECRB),** and the extensor carpi ulnaris. The other six are finger and thumb muscles that may act secondarily on the wrist: the **extensor digitorum communis (EDC),** the **extensor indicis proprius (EIP),** the **extensor digiti minimi (EDM),** the **extensor pollicis longus (EPL),** the **extensor pollicis brevis (EPB),** and the **abductor pollicis longus (APL).** The extensor digitorum communis and the extensor indicis proprius muscles are also known, more simply, as the extensor digitorum and the extensor indicis, respectively. The tendons of all nine muscles pass under the **extensor retinaculum,** which is divided into six distinct tunnels by septa. As the tendons pass deep to the retinaculum, each tendon is encased within its own tendon sheath to prevent friction between the tendons and the retinaculum. The septa of the retinaculum through which the tendons pass are attached to the dorsal carpal ligaments and help maintain stability of the extensor tendons on the dorsum, as well as allowing those muscles to contribute to wrist extension and preventing bowstringing of the tendons with active contraction.[48,89]

The extensor carpi radialis longus and extensor carpi radialis brevis muscles together make up the predominant part of the wrist extensor mass.[90] The extensor carpi radialis brevis muscle is somewhat smaller than the extensor carpi radialis longus muscle but has a more central location, inserting into the third metacarpal, and generally shows more activity during wrist extension activities.[11,91] One study found the extensor carpi radialis brevis muscle to be active during all grasp-and-release hand activities, except those performed in supination.[92] The extensor carpi radialis longus muscle inserts into the more radial second metacarpal and, therefore, has a smaller moment arm for wrist extension than does the extensor carpi radialis brevis muscle.[7] The extensor carpi radialis longus muscle shows increased activity when either radial deviation or

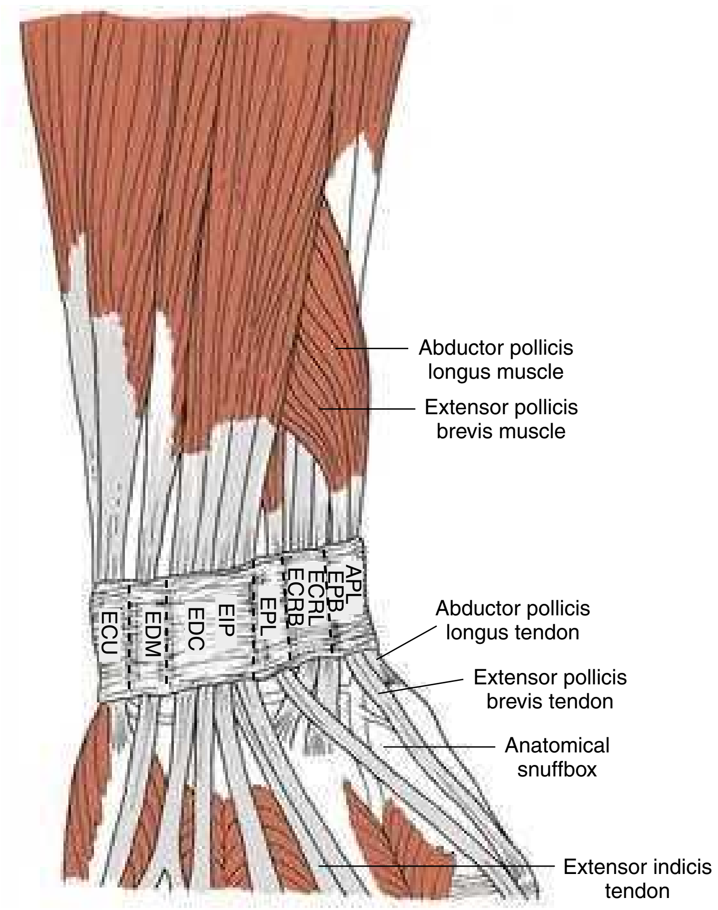

Figure 9–13 The dorsally located extensor tendons pass beneath the extensor retinaculum, where the tendons are compartmentalized. From the radial to the ulnar side, the abductor pollicis longus (APL) and extensor pollicis brevis (EPB) muscles share a compartment; the extensor carpi radialis brevis (ECRB) and the extensor carpi radialis longus (ECRL) muscles share a compartment; the extensor pollicis longus (EPL) muscle has a compartment of its own; the four tendons of the extensor digitorum communis (EDC) muscle share a compartment with the extensor indicis proprius (EIP) muscle; the extensor digiti minimi (EDM) muscle has its own compartment; and the extensor carpi ulnaris (ECU) muscle has its own compartment.

support against ulnar deviation is required or when forceful finger flexion motions are performed.[70,91] The ongoing activity of the extensor carpi radialis brevis muscle makes it vulnerable to overuse and is more likely than the quieter extensor carpi radialis longus muscle to be inflamed in lateral epicondylitis.[93] The literature has also questioned the role of the extensor digitorum communis muscle in development of this pathology.[94,95]

The extensor carpi ulnaris muscle extends and ulnarly deviates the wrist. It is active not only in wrist extension but frequently in wrist flexion as well.[91] Backdahl and Carlsoo hypothesized that the extensor carpi ulnaris muscle activity in wrist flexion adds an additional component of stability to the structurally less stable position of wrist flexion.[88] This is not needed on the radial side of the wrist, which has more developed ligamentous and bony structural checks. The connection of the extensor carpi ulnaris tendon sheath to the triangular fibrocartilage complex also appears to help tether the extensor carpi ulnaris muscle and prevent loss of excursion efficiency that would occur with bowstringing.[20] Tang and colleagues found a 30% increase in excursion of the extensor carpi ulnaris muscle after release of the triangular fibrocartilage complex from the distal ulna.[96] The effectiveness of the extensor carpi ulnaris muscle as a wrist extensor is also affected by forearm position. When the forearm is pronated, the crossing of the radius over the ulna causes a reduction in the moment arm of the extensor carpi ulnaris muscle, making it less effective as a wrist extensor.[4,90,92]

The extensor digiti minimi and the extensor indicis proprius muscles insert into the tendons of the extensor digitorum communis muscle and, therefore, have a common function with the extensor digitorum communis muscle.[97] The extensor indicis proprius and extensor digiti minimi muscles are capable of extending the wrist, but wrist extension is credited more to the extensor digitorum communis muscle. The extensor digitorum communis muscle is a finger extensor muscle but functions also as a wrist extensor (without radial or ulnar deviation). There appears to be some reciprocal synergy of the extensor digitorum communis muscle with the extensor carpi radialis brevis muscle in providing wrist extension, because less extensor carpi radialis brevis muscle activity is seen when the extensor digitorum communis muscle is active.[91]

Three extrinsic thumb muscles cross the wrist. Both the abductor pollicis longus and the extensor pollicis brevis muscles are capable of radially deviating the wrist and may serve a minor role in that function.[87] However, radial deviation of the wrist may detract from their prime action on the thumb. A synergistic contraction of the extensor carpi ulnaris muscle may be required to offset the unwanted wrist motion when the abductor pollicis longus and extensor pollicis brevis muscles act on the thumb. When muscles producing ulnar deviation are absent, the thumb extrinsic muscles may produce a significant radial deviation deformity at the wrist. Little evidence has been found to indicate that the more centrally located extensor pollicis longus muscle has any notable effect on the wrist.

Now that we have examined the wrist complex, let us look at the hand complex that the wrist serves.

THE HAND COMPLEX

The hand consists of five digits: four fingers and a thumb (Fig. 9–14). Each digit has a carpometacarpal joint and a **metacarpophalangeal (MP)** joint. The fingers each have two **interphalangeal (IP) joints,** the **proximal interphalangeal (PIP)** and **distal interphalangeal (DIP),** and the thumb has only one. There are 19 bones and 19 joints distal to the carpals that make up the hand complex. Although the joints of the fingers and the joints of the thumb have structural similarities, function differs significantly enough that the joints of the fingers shall be examined separately from those of the thumb. In examining the joints of the fingers, however, one should be cautious about generalizations that we will make. Ranney pointed out that each digit of the hand is unique and that models proposed for and conclusions drawn about one finger may not be accurate for all.[98]

Carpometacarpal Joints of the Fingers

The carpometacarpal joints of the fingers are composed of the articulations between the distal carpal row and the bases of the second through fifth metacarpal joints (see Fig. 9–1). The distal carpal row, of course, is also part of the midcarpal joint. The proximal portion of the four metacarpals of the fingers articulate with the distal carpals to form the second through fifth carpometacarpal joints (see Fig. 9–14). The

Figure 9–14 Bony anatomy of the thumb and fingers. DIP, distal interphalangeal; PIP, proximal interphalangeal; MP, metacarpophalangeal; CMC, carpometacarpal; M, metacarpal; P1, proximal phalanx; P2, middle phalanx; P3, distal phalanx.

second metacarpal articulates primarily with the trapezoid and secondarily with the trapezium and capitate. The third metacarpal articulates primarily with the capitate, and the fourth metacarpal articulates with the capitate and hamate. Last, the fifth metacarpal articulates with the hamate. Each of the metacarpals also articulates at its base with the contiguous metacarpal or metacarpals, with the exception of the second metacarpal, which articulates at its base with the third but not the first metacarpal. All finger carpometacarpal joints are supported by strong transverse and weaker longitudinal ligaments volarly and dorsally.[99,100]

The **deep transverse metacarpal ligament** spans the heads of the second through fourth metacarpals volarly. The deep transverse metacarpal ligament tethers together the metacarpal heads and effectively prevents the attached metacarpals from any more than minimal abduction at the carpometacarpal joints. Although the transverse metacarpal ligament contributes directly to carpometacarpal stability, it also is structurally part of the metacarpophalangeal joints of the fingers and will be discussed again in that context. The ligamentous structure is primarily responsible for controlling the total ROM available at each carpometacarpal joint, although some differences in articulations also exist.

One attribute of the distal carpals that affects carpometacarpal and hand function but not wrist function is the volar concavity, or **proximal transverse (carpal) arch,** formed by the trapezoid, trapezium, capitate, and hamate (Fig. 9–15). The carpal arch persists even when the hand is fully opened and is created not only by the curved shape of the carpals but also by the ligaments that maintain the concavity. The ligaments that maintain the arch are the transverse carpal ligament and the transversely oriented **intercarpal ligaments.** The transverse carpal ligament is the portion of the flexor retinaculum that attaches to the pisiform and hook of the hamate medially and to the scaphoid and trapezium laterally; the more proximal portion of the flexor retinaculum is continuous with the fascia overlying the forearm muscles. The transverse carpal ligament and intercarpal ligaments that link the four distal carpals maintain the relatively fixed concavity that will contribute to the arches of the palm. These structures also form the **carpal tunnel.** The carpal tunnel contains the median nerve and nine extrinsic flexor tendons of the fingers and thumb (see Fig. 9–12B). A number of intrinsic hand muscles attach to the transverse carpal ligament and bones of the distal carpal row. These may also contribute to maintaining the carpal arch.

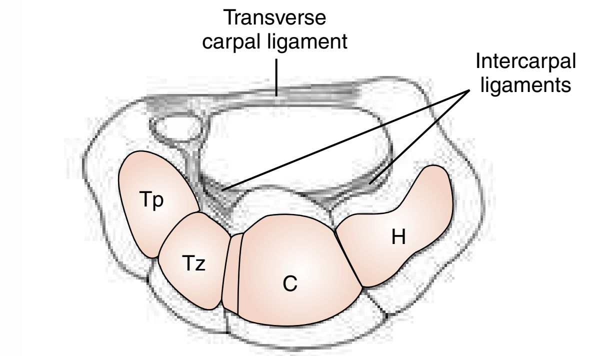

Figure 9–15 The proximal transverse arch, or carpal arch, forms the tunnel through which the median nerve and long finger flexors travel. The transverse carpal ligament and intercarpal ligaments assist in maintaining this concavity. C, capitate; H, hamate; Tp, trapezium; Tz, trapezoid.

9-3 Patient Case *case*

Carpal Tunnel Syndrome

Carl George has been a computer programmer for over 20 years. He spends the majority of his day typing. He reports that he began waking with numbness in his right hand 5 years ago, specifically affecting the thumb as well as the index, middle, and radial half of the ring fingers. He was evaluated by a hand surgeon, who found that tapping on the median nerve over the carpal tunnel reproduced Carl's paresthesias (tingling) in the median nerve distribution (positive Tinel's sign), as did placing Carl's wrist in sustained flexion for 1 minute (positive Phalen's test). The physician prescribed night splinting and patient education regarding proper ergonomics at work.[101] The splint held the wrist in a neutral position at night, which decreased the pressure on the median nerve.[102,103] In the subsequent 6 months, Carl noted progressive difficulty with completing fine motor tasks such as buttoning shirts and handling coins. Carl was referred to a neurologist who performed nerve conduction studies that revealed significant slowing in the median nerve conduction velocity, which was consistent with nerve compression at the wrist level. Also evident was atrophy of median nerve innervated thenar (thumb) muscles, a presentation commonly known as "ape hand" (Fig. 9-16).

Figure 9–16 Long-term median nerve compression can lead to atrophy of the median nerve innervated muscles in the thenar eminence, a presentation known as "ape hand" because of the flattening of the palm and the adducted position of the thumb. (The incision is from a surgical release of the transverse carpal ligament to relieve median nerve compression.)

Continuing Exploration 9-2:

Carpal Tunnel Syndrome

When the median nerve becomes compressed within the carpal tunnel, a neuropathy known as **carpal tunnel syndrome (CTS)** may develop. Cobb and colleagues proposed that the proximal edge of the transverse carpal ligament is the most common site for wrist flexion-induced median nerve compression.[104] The tunnel is narrowest, however, at the level of the hook of the hamate, where median nerve compression is unlikely to be affected by changes in wrist position.[104] When the transverse carpal ligament is cut to release median nerve compression, the carpal arch may widen somewhat, but investigators found that the arch would maintain its dorsovolar stiffness as long as the stronger transverse intercarpal ligaments were intact.[105]

Carpometacarpal Joint Range of Motion

The range of carpometacarpal motion of the second through fifth metacarpals is observable most readily at the metacarpal heads, and shows increasing mobility from the radial to the ulnar side of the hand.[57,106] The second through fourth carpometacarpal joints are plane synovial joints with one degree of freedom: flexion/extension. Although structured to permit flexion/extension, the second and third carpometacarpal joints are essentially immobile and may be considered to have "zero degrees of freedom."[40,98] The fourth carpometacarpal joint has perceptible flexion/extension. The fifth carpometacarpal joint is a saddle joint with two degrees of freedom, including flexion/extension, some abduction/adduction, and a limited amount of opposition.[11,98,107] The immobile second and third metacarpals provide a fixed and stable axis about which the fourth and fifth metacarpals and the very mobile first metacarpal (thumb) can move.[36,98,108] The motion of the fourth and fifth metacarpals facilitates the ability of the ring and little fingers to oppose the thumb.

Palmar Arches

The function of the finger carpometacarpal joints and their segments overall is to contribute (with the thumb) to the **palmar arch system.** The concavity formed by the carpal bones results in the proximal transverse arch of the palm of the hand. The other palmar arches can easily be visualized as occurring transversely across the palm (often considered to be inclusive of the thumb and fourth finger) and longitudinally down the palm (inclusive of the fingers) (Fig. 9–17). The adjustable positions of the first, fourth, and fifth metacarpal heads around the relatively fixed second and third metacarpals form a mobile **distal transverse arch** at the level of the metacarpal heads that augments the fixed proximal transverse arch of the distal carpal row. The **longitudinal arch** traverses the length of the digits from proximal to distal. The deep transverse metacarpal ligament contributes to stability of the mobile arches during grip functions.[109] The palmar arches allow the palm and the digits to conform optimally to the shape of the object being held.[110] This maximizes the amount of surface contact, enhancing stability as well as increasing sensory feedback.

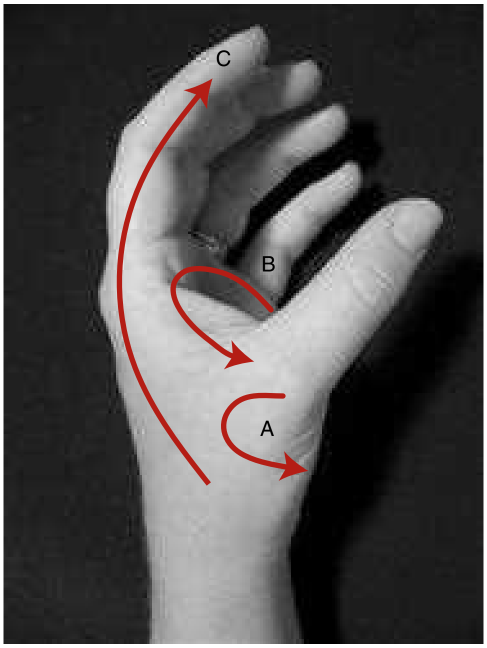

Figure 9–17 The palmar arch system assists with functional grasp. The proximal transverse arch **(A)** is fixed, while the distal transverse arch **(B)** and longitudinal arch **(C)** are mobile.

Muscles that cross the carpometacarpal joints will contribute to palmar cupping (conformation of the palm to an object) by acting on the mobile segments of the palmar arches. Hollowing of the palm accompanies finger flexion, and relative flattening of the palm accompanies finger extension. The fifth carpometacarpal joint is crossed and acted on by the **opponens digiti minimi (ODM)** muscle. This oblique muscle is attached proximally to the hamate and transverse carpal ligament and distally to the ulnar side of the fifth metacarpal. It is optimally positioned, therefore, to flex and rotate the fifth metacarpal about its long axis (Fig. 9–18). No other muscles cross or act on the finger carpometacarpal joints alone. However, increased arching occurs with activity of the flexor carpi ulnaris muscle attached to the pisiform and with activity of the intrinsic hand muscles that insert on the transverse carpal ligament.[10,111] The radial wrist muscles (flexor carpi radialis, extensor carpi radialis longus, and extensor carpi radialis brevis) cross the second and third carpometacarpal joints to insert on the bases of those metacarpals but produce little or no motion at these relatively fixed articulations. The stability of the second and third carpometacarpal joints can be viewed as a functional adaptation that enhances the efficiency of the flexor carpi radialis, extensor carpi radialis longus, and extensor carpi radialis brevis muscles in their wrist functions. If the second and third carpometacarpal joints were mobile, the radial flexor and extensors would act first on the carpometacarpal joints and, consequently, would be less effective at the midcarpal and radiocarpal joints, given the loss in length-tension.

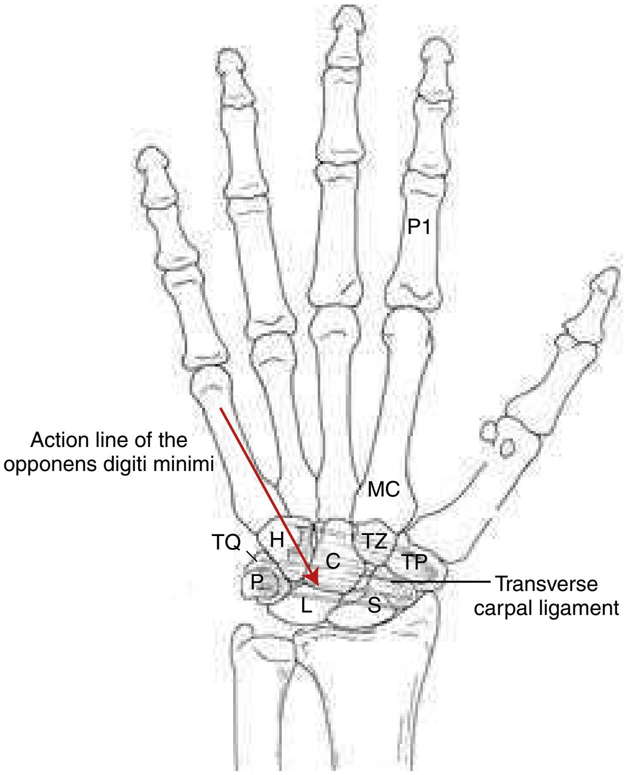

Figure 9–18 The opponens digiti minimi is the only muscle that acts exclusively on a carpometacarpal joint. As indicated by its action line, it is effective at flexion of the fifth metacarpal joint and rotation of the metacarpal joint around its long axis. The opponens digiti minimi muscle's attachment to the transverse carpal ligament may also contribute to supporting the proximal palmar arch.

Metacarpophalangeal Joints of the Fingers

Each of the four metacarpophalangeal joints of the fingers is composed of the convex metacarpal head proximally and the concave base of the first phalanx distally (see Fig. 9–14). The metacarpophalangeal joint is condyloid with two degrees of freedom: flexion/extension and abduction/adduction. The large metacarpal head has 180° of articular surface in the sagittal plane, with the predominant portion lying volarly. This is apposed to approximately 20° of articular surface on the base of the phalanx. In the frontal plane, there is less articular surface than in the sagittal plane, and the articular surfaces are more congruent.

The metacarpophalangeal joint is surrounded by a capsule that is generally considered to be lax in extension. Given the incongruent articular surfaces, capsular laxity in extension allows some passive axial rotation of the proximal phalanx.[98] Two **collateral ligaments** at the volarly located deep transverse metacarpal ligament enhance joint stability. As we noted previously, incongruent joints often have an accessory joint structure to enhance stability. At the metacarpophalangeal joint, this function is served by the **volar plate.**

Volar Plates

The volar plate (or **palmar plate**) at each of the metacarpophalangeal joints is a unique structure that increases joint congruence. It also provides stability to the metacarpophalangeal joint by limiting hyperextension and, therefore, provides indirect support to the longitudinal arch.[57] The volar plate is composed of fibrocartilage and is firmly attached to the base of the proximal phalanx distally but not to the metacarpal proximally.[55] The plate becomes membranous proximally to blend with the volar capsule that then attaches to the metacarpal head just proximal to the articular surface (Fig. 9–19A). The volar plate can also be visualized as a fibrocartilage impregnation of the volar portion of the capsule just superficial to the metacarpal head. The inner surface of the volar plate is effectively a continuation of the articular surface of the base of the proximal phalanx. In metacarpophalangeal extension, the plate adds to the amount of surface in contact with the large metacarpal head. The fibrocartilage composition of the plate is consistent with its ability to resist both tensile stresses in restricting metacarpophalangeal hyperextension and compressive forces needed to protect the volar articular surface of the metacarpal head from objects held in the palm.[112] The flexible attachment of the plate to the phalanx permits the plate to glide proximally down the volar surface of the metacarpal head in flexion without restricting motion, while also preventing pinching of the long flexor tendons in the metacarpophalangeal joint (Fig. 9–19B).

In addition to their connection to their respective proximal phalanges, the four volar plates and their respective capsules of the metacarpophalangeal joints of the fingers also blend with and are interconnected superficially by the deep transverse metacarpal ligament that, as we noted earlier, tethers together the heads of the metacarpals of the four fingers (Fig. 9–20). Dorsal to the deep transverse metacarpal ligament are **sagittal bands** on each side of the metacarpal head that connect each volar plate (via the capsule and deep transverse metacarpal ligament) to the extensor digitorum communis tendon and **extensor expansion** (Fig. 9–21). The sagittal bands help stabilize the volar plates over the four metacarpal heads.[21,98,109]

Collateral Ligaments

The radial and ulnar collateral ligaments of the metacarpophalangeal joint are composed of two parts: the **collateral ligament proper**, which is cordlike, and the **accessory collateral ligament** (see Fig. 9–19). Minami and associates quantified the length changes in the different parts of the collateral ligament at the metacarpophalangeal joint with varying degrees of motion.[113] They found that the more dorsally located collateral ligament proper was lengthened 3 to 4 mm with metacarpophalangeal joint flexion from 0° to 80°, whereas the more volarly located accessory collateral ligament was shortened 1 to 2 mm. Conversely, with metacarpophalangeal joint hyperextension, the accessory portion was lengthened and the proper portion was placed on slack. Tension in the collateral ligaments at full metacarpophalangeal joint flexion (the close-packed position for the metacarpophalangeal joint) is considered to account for the minimal amount of abduction/adduction that can be obtained at the metacarpophalangeal joint in full flexion. Shultz and associates concluded that the collateral ligaments

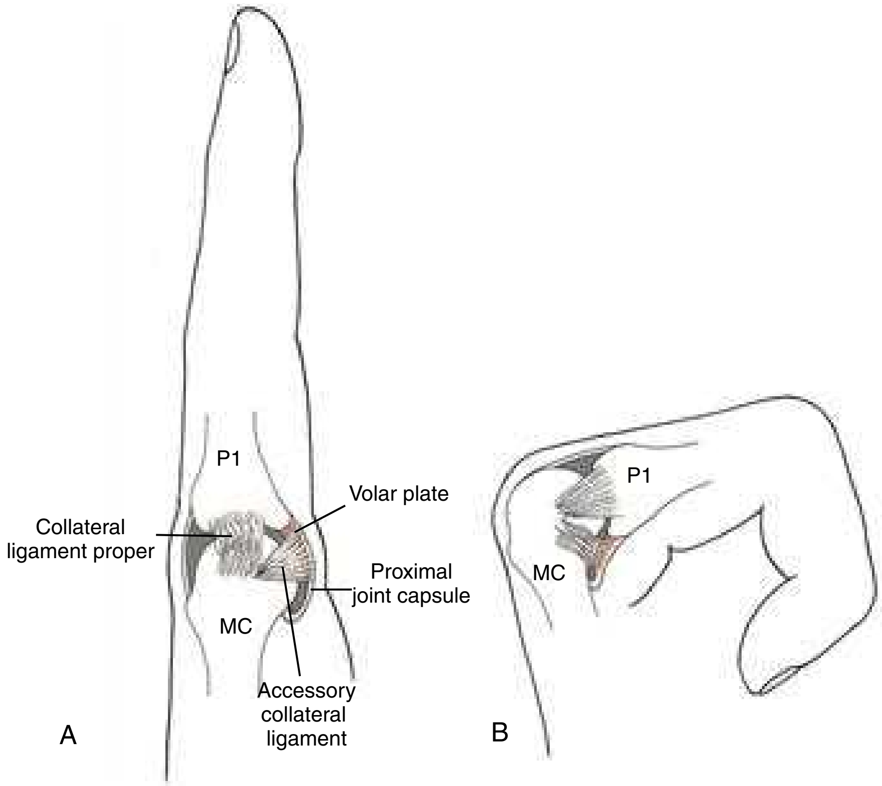

Figure 9-19 **A.** The volar plate at the metacarpophalangeal joint attaches to the base of the proximal phalanx. The plate blends with and lies deep to the metacarpophalangeal joint capsule and the deep transverse metacarpal ligament volarly. **B.** In metacarpophalangeal joint flexion, the flexible attachments of the plate allow the plate to slide proximally on the metacarpal head without impeding motion. The collateral ligament proper is loose in metacarpophalangeal joint extension, whereas the accessory collateral ligament is taut. The reverse occurs in metacarpophalangeal joint flexion.

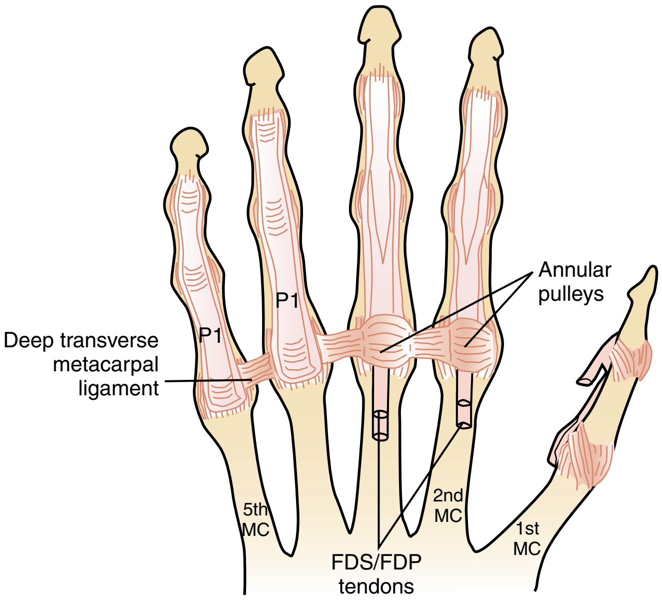

Figure 9–20 The deep transverse metacarpal ligament runs transversely across the heads of the four metacarpophalangeal joints of the fingers. The fibers of the transverse metacarpal ligament blend with each metacarpophalangeal joint capsule and with the deeper volar plates. The superficial aspect of the transverse metacarpal ligament at each metatarsal head is grooved (shown on fourth and fifth metacarpophalangeal joints) for the long finger flexors that pass over the transverse metacarpal ligament and through the annular ligaments (shown on second and third metacarpophalangeal joints).

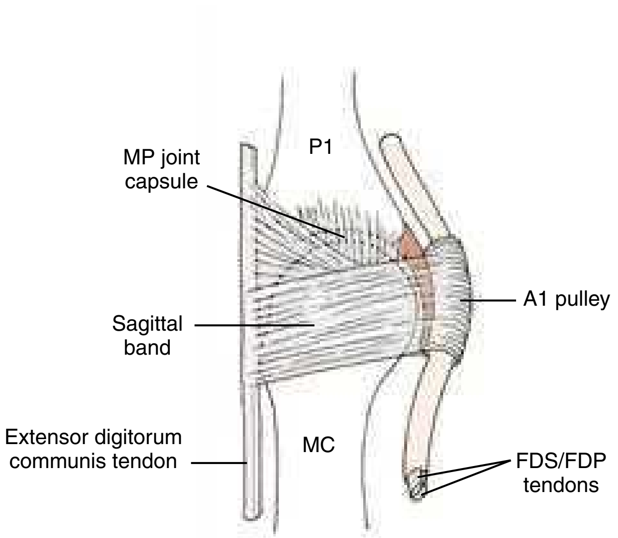

Figure 9–21 The connections of the sagittal bands to each side of the volar plate, the collateral ligaments of the metacarpophalangeal joint (via the capsule), and the extensor digitorum communis muscle via the extensor expansion help stabilize the volar plates on the four metacarpal heads volarly and the extensor digitorum communis tendons over the metacarpophalangeal joints dorsally.

provided stability throughout the metacarpophalangeal joint ROM with parts of the fibers taut at various points in the range.[114] Rather than attributing the limitation of abduction/adduction in metacarpophalangeal flexion to collateral ligamentous tension, they proposed that the bicondylar shape of the volar surface of the metacarpal head resulted in a bony block at about 70° of metacarpophalangeal joint flexion.

Fisher and associates completed a series of dissections of fingers, seeking an explanation for the relatively small incidence of osteoarthritis (OA) in metacarpophalangeal joints in comparison with the fairly common changes seen in the distal interphalangeal joints and, to a lesser extent, in the proximal interphalangeal joints.[115] They found fibrocartilage that projected into the metacarpophalangeal, proximal interphalangeal, and distal interphalangeal joints from the inner surface of the dorsally located **extensor hood,** from the volar plates, and from the collateral ligaments. The fibrocartilage projections were most impressive in the metacarpophalangeal joints and may, like the volar plate itself, increase the surface area on the small base of the phalange for contact with the large metacarpal (and phalangeal) heads.

Range of Motion

The total ROM available at the metacarpophalangeal joint varies with each finger. Flexion/extension increases radially to ulnarly, with the index finger having approximately 90° of metacarpophalangeal joint flexion and the little finger approximately 110°[57] (Fig. 9–22). Hyperextension is fairly consistent between fingers but varies widely among individuals. The range of passive hyperextension has been used as a measure of generalized body flexibility.[10] The range of abduction/adduction is maximal in metacarpophalangeal joint extension. The index and little fingers have more frontal plane mobility than do the middle and ring fingers. As previously noted, abduction/adduction is most restricted in metacarpophalangeal joint flexion.[114] Passive rotation of the metacarpophalangeal joints has been measured, which supports the contention that this mobility allows for adaptation of grasp for different size objects.[116]

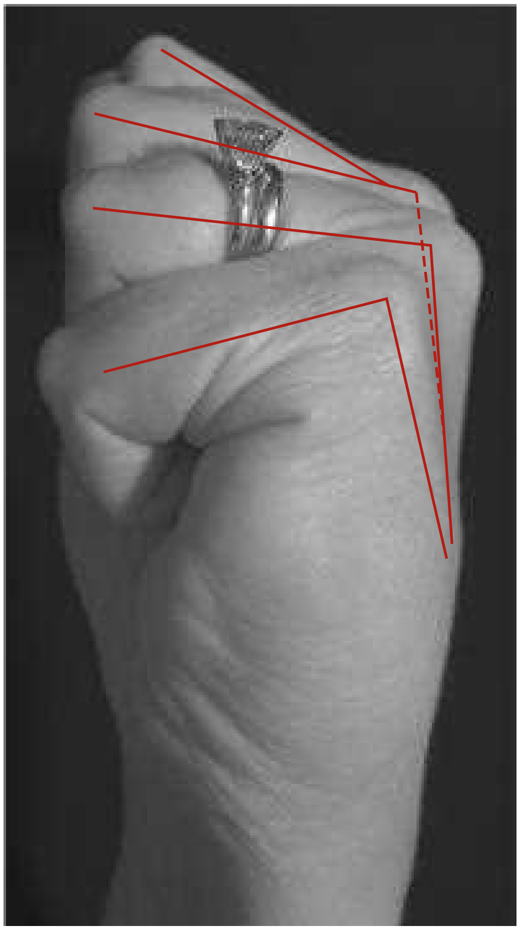

Figure 9–22 The available range of motion at the metacarpophalangeal joints of the fingers increases from the radial to the ulnar side, with the greatest metacarpophalangeal finger range at the fifth metacarpophalangeal joint.

Interphalangeal Joints of the Fingers

Each of the proximal interphalangeal and distal interphalangeal joints of the fingers is composed of the head of a phalanx and the base of the phalanx distal to it. Each interphalangeal joint is a true synovial hinge joint with one degree of freedom (flexion/extension), a joint capsule, a volar plate, and two collateral ligaments (Fig. 9–23). The base of each middle and distal phalanx has two shallow concave facets with a central ridge. The distal phalanx sits on the pulley-shaped head of the phalanx proximal to it. The joint structure is similar to that of the metacarpophalangeal joint in that the proximal articular surface is larger than the distal articular surface. Unlike the metacarpophalangeal joints, there is little posterior articular surface at the proximal or distal interphalangeal joint and, therefore, little hyperextension. The distal interphalangeal joint may have some passive hyperextension, but the proximal interphalangeal joint has essentially none in most individuals.

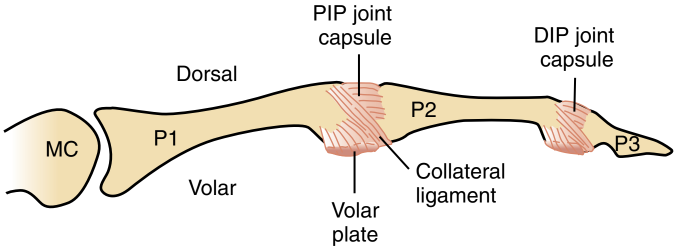

Figure 9–23 The proximal interphalangeal and distal interphalangeal joints, like the metacarpophalangeal joints, have volar plates that blend with the volar capsule portion of the capsule. The orientation of the collateral ligaments at the proximal and distal joints, however, differs from the orientation of the collateral ligaments at the metacarpophalangeal joints.

Volar plates reinforce each of the interphalangeal joint capsules, enhance stability, and limit hyperextension.[117] The plates at the interphalangeal joints are structurally and functionally identical to those at the metacarpophalangeal joint, except that the plates are not connected by a deep transverse ligament.

Fisher and associates found fibrocartilage projections from the **extensor mechanism,** the volar plate, and the collateral ligaments attached to the bases of the phalanges at both the proximal interphalangeal and the distal interphalangeal joints, with the structures more obvious at the proximal interphalangeal joints.[115] The collateral ligaments of the interphalangeal joints are not fully understood but are described to have cord and accessory parts similar to those of the metacarpophalangeal joint.[57] Stability is provided by this collateral ligament complex because some portions remain taut and provide support throughout proximal interphalangeal and distal interphalangeal joint motion.[57,118,119] Injuries to the collateral ligaments of the proximal interphalangeal joint are common, particularly in sports and workplace injuries, with the radial (lateral) collateral twice as likely to be injured as the ulnar (medial) collateral.[120,121] Dzwierzynski and colleagues found the lateral collateral of the index finger to be the strongest of the proximal interphalangeal collateral ligaments, whereas the fifth proximal interphalangeal joint had the weakest collateral ligaments.[120] The relative strengths of the lateral collateral ligaments meet functional expectations because the thumb is most likely to oppose the lateral side of the index (creating a varus stress at the proximal interphalangeal joint) and least likely to do so at the fifth.

The total range of flexion/extension available to the index finger is greater at the proximal interphalangeal joint (100° to 110°) than it is at the distal interphalangeal joint (80°). The ranges for proximal interphalangeal and distal interphalangeal flexion at each finger increase ulnarly, with the fifth proximal interphalangeal and distal interphalangeal joints achieving 135° and 90°, respectively. The pattern of increasing flexion/extension ROM from the radial to the ulnar side of the hand is consistent at the carpometacarpal, metacarpophalangeal, and proximal interphalangeal joints and, to a lesser degree, at the distal interphalangeal joints.[122] The additional range allocated to the more ulnarly located fingers as well as the underlying bony arrangement favors angulation of the fingers toward the scaphoid and facilitates opposition of the fingers with the thumb (Fig. 9–24). The greater available range ulnarly also produces a grip that is tighter, or has greater closure, on the ulnar side of the hand. Many objects are constructed so that the shape is narrower at the ring and small fingers and widens toward the middle and index fingers to fit this ROM pattern.

Continuing Exploration 9-3:

Anti-Deformity Positioning

After trauma to the hand, a custom-fabricated splint is commonly provided to immobilize the injured structures. The purpose of this device is to provide support and protection to the injured region during the healing process, while attempting to minimize the potential problems at the joints created by immobilization (i.e., contractures). Because the collateral ligaments of the metacarpophalangeal joints are slack with extension, immobilization in metacarpophalangeal extension in a splint would place the collateral ligaments at risk for adaptive shortening. Adaptive shortening of the collateral ligaments would limit metacarpophalangeal joint flexion, with concomitant disruption of the longitudinal arch leading to impairments in grasp and functional use. Optimally, an immobilization splint should place the metacarpophalangeal joints in flexion so that the collateral ligaments are on stretch; the interphalangeal joints should be held in extension to reduce the risk of flexion contractures from shortening of the volar plates. The thumb should be placed in some degree of carpometacarpal abduction to prevent a first web space contracture (Fig. 9–25). This position of metacarpophalangeal joint flexion with interphalangeal extension is known as the "anti-deformity position."[123]

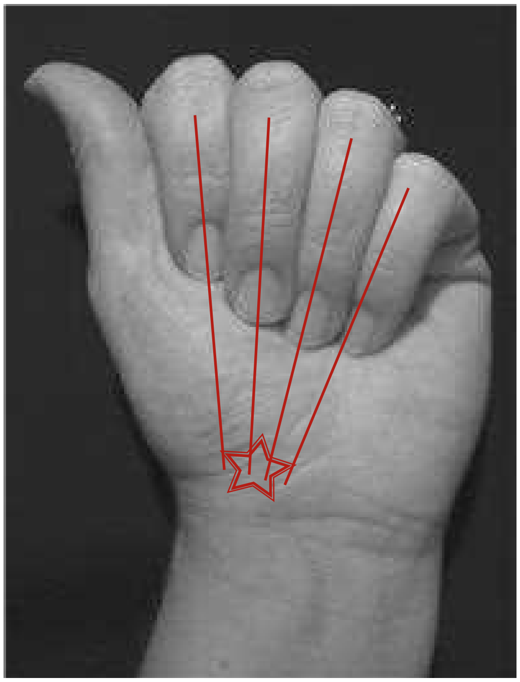

Figure 9–24 With flexion of digits to the palm, there is a convergence toward the scaphoid tubercle (starburst) and toward the thumb. This obliquity is due to the increased flexion mobility of the metacarpophalangeal and proximal interphalangeal joints from the radial to the ulnar side of the hand.

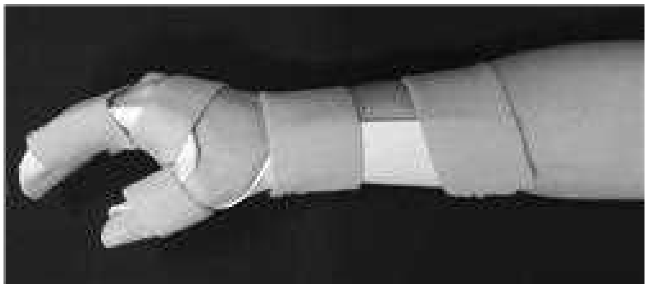

Figure 9–25 Splinting the hand in the "anti-deformity" position minimizes the risk of dysfunctional changes to the immobilized joints.

Extrinsic Finger Flexors

The muscles (also referred to as "motors") of the fingers and thumb that have proximal attachments above (proximal to) the wrist (radiocarpal joint) are extrinsic muscles, whereas those with all attachments distal to the radiocarpal joint are intrinsic muscles. Functionally, the extrinsic muscles are also divided into flexors and extensors. The intrinsic muscles are typically not referred to as flexor or extensor groups because several will flex one joint while extending another. We will first consider the extrinsic muscles of the fingers, then the intrinsic muscles of the fingers, and conclude with the extrinsic and intrinsic muscles of the thumb before discussing coordinated function of all the elements together.

There are two extrinsic muscles that contribute to finger flexion. These are the flexor digitorum superficialis and the flexor digitorum profundus muscles. The flexor digitorum superficialis muscle primarily flexes the proximal interphalangeal joint, but it also contributes to metacarpophalangeal joint flexion. The flexor digitorum profundus muscle can flex the metacarpophalangeal, proximal interphalangeal, and the distal interphalangeal joints and is considered to be the more active of the two muscles.[4] With gentle pinch or grasp, the flexor digitorum profundus muscle alone will be active. As greater flexor force is needed or when finger flexion with wrist flexion is desired, the flexor digitorum superficialis muscle joins the flexor digitorum profundus muscle by increasing its activity.[88,97,124,125]

The flexor digitorum superficialis muscle can produce more torque at the metacarpophalangeal joint than can the flexor digitorum profundus muscle. Not only does the flexor digitorum superficialis muscle cross fewer joints (making it less likely to lose tension as it shortens over multiple joints), but the flexor digitorum superficialis tendon is also superficial to the flexor digitorum profundus tendon at the metacarpophalangeal joint. Consequently, the flexor digitorum superficialis muscle has a greater moment arm for metacarpophalangeal joint flexion.[3] It is often thought that the flexor digitorum superficialis muscle is stronger at proximal interphalangeal flexion because the flexor digitorum superficialis muscle crosses few joints, but this is not the case. In contrast to what is found at the metacarpophalangeal joint, the flexor digitorum superficialis tendon lies deep to the flexor digitorum profundus tendon at the proximal interphalangeal joint and, therefore, has a lesser moment arm at the proximal interphalangeal joint.[98] The switch in position between the flexor digitorum superficialis and flexor digitorum profundus tendons occurs just proximal to the proximal joint, where the flexor digitorum profundus tendon emerges through the split in the flexor digitorum superficialis tendon **(Camper's chiasma)** so the that flexor digitorum superficialis tendon can attach to the base of the middle phalanx deep to the flexor digitorum profundus tendon. Although the moment arm of the flexor digitorum superficialis tendon may not be optimal at the proximal interphalangeal joint, the flexor digitorum superficialis tendon is important for balance at the proximal interphalangeal joint. When the flexor digitorum superficialis tendon is absent, forceful pinch (thumb to fingertip) activity of the flexor digitorum profundus muscle may create proximal interphalangeal *extension* along with distal interphalangeal flexion (Fig. 9–26), rather than flexion at both joints.[126] This phenomenon can be observed in many normal hands because the flexor digitorum superficialis tendon of the little finger is commonly absent or may have anomalous distal attachments.[126,127]

Both the flexor digitorum superficialis and flexor digitorum profundus muscles are dependent on wrist position for an optimal length-tension relationship.[4] If there is no counterbalancing extensor torque at the wrist, the volarly located flexor digitorum superficialis and flexor digitorum profundus muscles will cause wrist flexion to occur. If the finger flexor muscles are permitted to shorten over the wrist, there will be a concomitant loss of tension at the more distal joints. In fact, it is almost impossible to fully flex the fingers actively if the wrist is also flexed.[128] Although the poor length-tension relationship in the flexor digitorum superficialis and flexor digitorum profundus muscles accounts for some of this phenomenon, the inability to complete the flexion ROM is also attributable to the concomitant passive tension in the finger extensors. During active finger flexion (as in grasp activities), the counterbalancing wrist extensor force is usually supplied by an active wrist extensor such as the extensor carpi radialis brevis muscle or, in some instances, the extensor digitorum communis muscle.

Continuing Exploration 9-4:

Finger Flexor Grasp

The greater available range of metacarpophalangeal and interphalangeal joint flexion in the ring and little fingers in comparison with the index or long fingers means that the long flexors of the ring and little fingers must shorten over a greater range, resulting in a loss of tension in the muscles of those fingers. If the object to be held by the fingers is heavy or requires strong grip, the object may be shaped so that it is wider ulnarly than radially, a so-called pistol grip (Fig. 9–27A). The pistol grip limits metacarpophalangeal/interphalangeal joint flexion in the ring and little fingers while the wrist extensors stabilize the wrist against a strong contraction of the finger flexors. The loss of tension in the long finger flexors is not a problem if strong grip is not required (e.g., holding a glass); then the object may be tapered at the ring and little fingers to accommodate the greater ROM (Fig. 9–27B). Notice that the wrist in both forceful and gentle grips tends to assume a position of ulnar deviation that maximizes efficiency of the long finger flexors.[2,83]

Mechanisms of Finger Flexion

Optimal function of the flexor digitorum superficialis and flexor digitorum profundus muscles depends not only on stabilization by the wrist musculature but also on intact flexor gliding mechanisms.[129] The gliding mechanisms consist of the flexor retinacula, **bursae,** and **digital tendon**

Figure 9–26 When the flexor digitorum superficialis muscle is not present (as is occasionally the case in the little finger), forcefully pressing the thumb and finger tip together produces distal interphalangeal joint flexion with proximal interphalangeal joint *extension*, rather than flexion. Without the stabilization of the proximal interphalangeal joint by the flexor digitorum superficialis muscle, the flexor digitorum profundus muscle is not able to flex both joints.

sheaths. The fibrous retinacular structures (proximal flexor retinaculum, transverse carpal ligament, and extensor retinaculum) tether the long flexor tendons to the hand; the bursae and tendon sheaths facilitate friction-free excursion of the tendons on the fibrous retinacula. The retinacula prevent bowstringing of the tendons that would result in loss of excursion and work efficiency in the contracting muscles that pass under them. The tendons must be anchored without interfering with their excursion and without creating frictional forces that would cause degeneration of the tendons over time.

As the tendons of the flexor digitorum superficialis and flexor digitorum profundus muscles cross the wrist to enter the hand, they first pass beneath the proximal flexor retinaculum and through the carpal tunnel under the transverse carpal ligament (see Fig. 9–12A and B). Friction between the tendons themselves and friction of the tendons on the overlying transverse carpal ligament are prevented by the **radial** and **ulnar bursae** that envelop the flexor tendons at this level. All eight tendons of the flexor digitorum profundus and flexor digitorum superficialis muscles are invested together in the ulnar bursa (Fig. 9–28A). The bursa is compartmentalized to prevent friction of tendon on tendon. The flexor pollicis longus muscle that accompanies the flexor digitorum superficialis and flexor digitorum profundus muscles through the carpal tunnel is encased in its own radial bursa (see Figs. 9–12 and 9–28A). The radial and ulnar bursae contain a synovial-like fluid that minimizes frictional forces. The pattern of bursae and tendon sheaths may vary among individuals. The most common representation shows the ulnar bursa to be continuous with the digital tendon sheath for the little finger (see Fig. 9–28A). However, Phillips and colleagues found continuity between the ulnar bursa and tendon sheath of the little finger in only 30% of 60 specimens.[130] The ulnar bursa is typically not continuous with the digital tendon sheaths for the index, middle, and ring fingers. Rather, for these fingers, the ulnar bursa ends just distal to the proximal palmar crease, and the digital tendon sheaths begin at the middle or distal palmar creases.[11] The radial

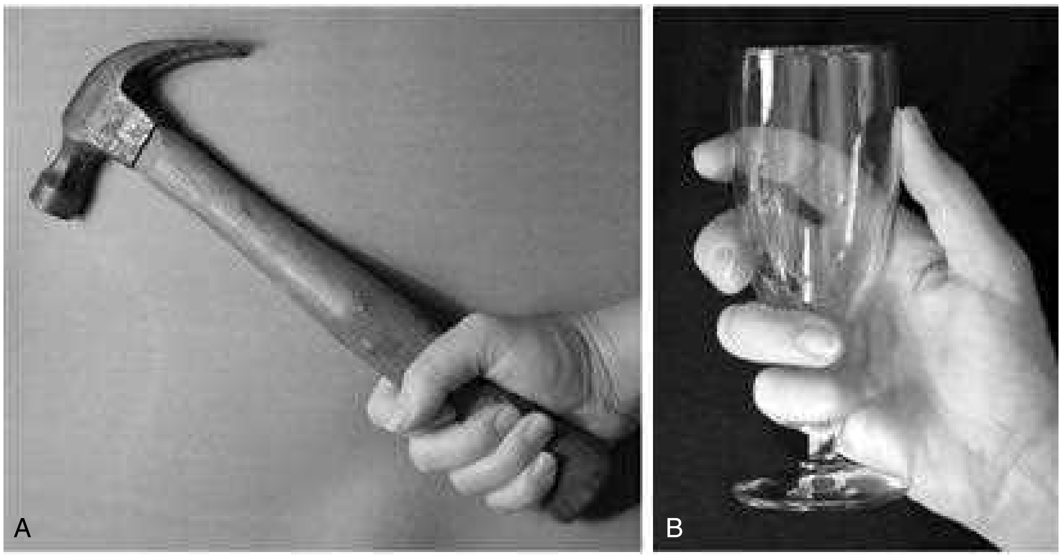

Figure 9–27 **A.** The so-called "pistol grip" of the hammer allows the flexor digitorum superficialis and flexor digitorum profundus muscles to work more forcefully at the ring and little fingers because the range of motion in the more mobile metacarpophalangeal and interphalangeal joints in these fingers is restricted by the shape of the object. The shape also encourages wrist ulnar deviation that further enhances force production in the long finger flexors. **B.** When force is not needed, the shape of an object is often tapered to accommodate to the greater range of the ring and little fingers, allowing the long finger flexors to close the fingers fully around the object.

bursa encases the flexor pollicis longus muscle and is continuous with its digital tendon sheath. The extent and communication of the digital tendon sheaths is functionally relevant because infection within a sheath will travel its full length, producing painful tenosynovitis. If a sheath is continuous with the ulnar or radial bursa, the infection may spread from the sheath into the palm (or vice versa).[32,87] The tendon sheaths for each finger end proximal to the insertion of the flexor digitorum profundus muscle, effectively ending at the distal aspect of the middle phalanx. Consequently, puncture wounds or injuries to the pad (distal phalanx) of the fingers that are a fairly common site of trauma are unlikely to introduce infection into the digital tendon sheaths.

The flexor digitorum superficialis and flexor digitorum profundus tendons of each finger pass through a fibro-osseous tunnel that is comprised of five transversely oriented **annular pulleys** (or **vaginal ligaments**), as well as three obliquely oriented **cruciate pulleys.**[21,131] The first two annular pulleys lie closely together, with one (designated the A1 pulley) at the head of the metacarpal and a second larger one (A2) along the volar midshaft of the proximal phalanx. The floor of the first pulley is formed by the flexor groove in the deep transverse metacarpal ligament, whereas all the other annular pulleys attach directly to bone. The third annular pulley (A3) lies at the distal-most part of the proximal phalanx, and the fourth (A4) lies centrally on the middle phalanx (see Fig. 9–28B). A fifth pulley (A5) may lie at the base of the distal phalanx. The base of each of the pulleys on the bone is longer than the roof superficially, and the roof has a slight concavity volarly. This shape prevents the pulleys from pinching each other at extremes of flexion, forming nearly one continuous tunnel in grasp.[21] The shorter roof of the fibro-osseous tunnel also minimizes the pressure on the tendon when it is under tension, distributing pressure throughout the tunnel during finger flexion rather than just at the edges of the tunnel[131] (see Fig. 9–28B). The three cruciate (crisscrossing) pulleys also tether the long flexor tendons. One is located between the A2 and A3 pulleys and is designated as C1; the next cruciate pulley (C2) lies between the A3 and A4 pulleys; and the last cruciate pulley (C3) lies between the A4 and A5 pulleys. The A4, A5, and C3 structures contain only the flexor digitorum profundus tendon because the flexor digitorum superficialis muscle inserts on the middle phalanx proximal to these structures. The annular pulleys and cruciate ligaments vary among individuals in both number and extent.[131] More recently, an additional annular pulley found proximal to the A1 has also been described and has been named the **palmar aponeurosis (PA) pulley.**[132] The thumb has a distinct pulley system, including two annular and one oblique pulley (see Fig. 9–28A).[133]

Friction of the flexor digitorum superficialis and flexor digitorum profundus tendons on the annular pulleys and

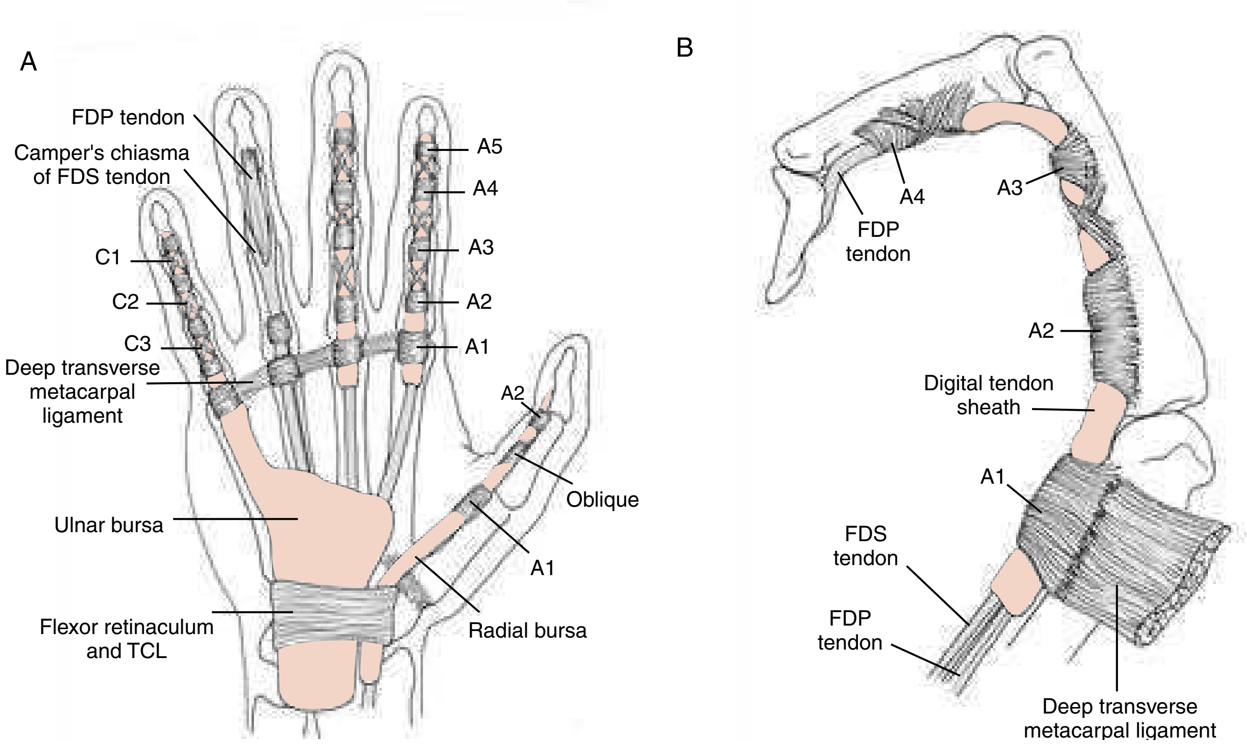

Figure 9–28 **A.** The flexor mechanisms of the fingers and thumb include the fibro-osseous tunnels formed by the flexor retinaculum and transverse carpal ligament at the wrist, the annular pulleys (A1 to A5), and the cruciate pulleys (C1 to C3). The tendons are protected within the tunnels by the radial and ulnar bursae and the digital tendon sheaths. The pulleys and the tendon sheath have been removed from the ring finger to show how the deep flexor digitorum profundus (FDP) tendon emerges through Camper's chiasma in the flexor digitorum superficialis (FDS) tendon to pass on to the distal phalanx, and the split flexor digitorum superficialis tendon rejoins and inserts on the base of the middle phalanx. **B.** The shape of the pulleys allows finger flexion without pinching of the pulleys while more evenly distributing pressure on the tendon and sheath across the roof of the fibro-osseous tunnels.

cruciate ligaments is minimized by the digital tendon sheaths that envelop the tendons from the point at which the tendons pass into the most proximal annular pulley (PA or A1) to the point at which the tendon of the flexor digitorum profundus muscle passes through the most distal cruciate pulley (C3 or A5) (see Fig. 9–28B). The synovial-like fluid contained in each of the digital tendon sheaths permits gliding of the tendons beneath their ligamentous constraints and between each other. This is particularly important over the proximal phalanx, where the flexor digitorum superficialis tendon splits to either side of the flexor digitorum profundus tendon and rejoins *beneath* the flexor digitorum profundus tendon to insert on the middle phalanx. The flexor digitorum profundus tendon, consequently, must pass through Camper's chiasma (see ring finger of Fig. 9–28A). Once the flexor digitorum profundus tendon is distal to the last annular pulley, the tendon sheath ends because lubrication of the tendon is no longer needed. Vascular supply to the gliding mechanism is critical to maintaining synovial fluid and tendon nutrition. Direct vascularization of each tendon occurs through vessels that reach the tendon via the **vincula tendinum.** These are folds of the synovial membrane (usually four in number) that carry blood vessels to the body of the tendon and to the tendinous insertions of the flexor digitorum superficialis and flexor digitorum profundus muscles of each finger.[21,134] The tendons also receive some of their nutrition directly from the synovial fluid within the sheath and, through that mechanism, can withstand at least partial loss of direct vascularization.[134,135]

The function of the annular pulleys is to keep the flexor tendons close to the bone, allowing only a minimum amount of bowstringing and migration volarly from the joint axes.[136,137] This sacrifices the increase in the moment arms that might occur with substantial bowstringing of the tendons but enhances both tendon excursion efficiency and work efficiency of the long flexors.[126,138] Any interruption in either the annular pulleys or the digital tendon sheaths can result in substantial impairment of flexor digitorum superficialis and flexor digitorum profundus muscle functioning or in structural deformity. **Trigger finger** is one example of the disability that can be created when repetitive trauma to a flexor tendon results in the formation of nodules on the tendon and thickening of an annular pulley. During active finger flexion, the nodule gets caught beneath the pulley requiring passive extension to "unlock" the stuck flexed position.[10] Of the potential six annular pulleys (PA, A1 through A5), integrity of pulleys A2 and A4 is credited with being most critical to maintaining flexor digitorum superficialis/flexor digitorum profundus muscle efficiency and they should be salvaged if at all possible during tendon reconstruction procedures.[133,138,139]

Concept Cornerstone **9-3**

Flexor Gliding Mechanism at the Metacarpophalangeal Joint

The flexor gliding mechanism at the metacarpophalangeal joint is particularly complex because of its multilayered structure. From deep to superficial at each of the metacarpophalangeal joints of the fingers, there are (1) the fibrocartilaginous volar plate, which is in contact with the metacarpal head; (2) the fibrous longitudinal fibers of the metacarpophalangeal joint capsule, which blends with the volar aspect of the plate; (3) the fibers of the deep transverse metacarpal ligament (oriented perpendicularly to those of the longitudinal fibers of the capsule), which has grooves on its volar surface for the long flexor tendons of the fingers and form the floor of a fibro-osseous tunnel; (4) the flexor digitorum profundus tendon, which lies in the groove of the transverse metacarpal ligament; (5) the flexor digitorum superficialis tendon, which lies just superficial to the flexor digitorum profundus tendon; (6) the digital tendon sheath that envelops both the flexor digitorum profundus and flexor digitorum superficialis tendons; and (7) the A1 annular pulley that forms the roof of the fibro-osseous tunnel and lies most superficially in this set of interconnected layers.

Extrinsic Finger Extensors

The extrinsic finger extensors are the extensor digitorum communis, the extensor indicis proprius, and the extensor digiti minimi muscles. Each of these muscles passes from the forearm to the hand beneath the extensor retinaculum, which maintains proximity of the tendons to the joints and improves excursion efficiency. Each of these six tendons is contained within a compartment of the extensor retinaculum and is enveloped by an isolated bursa or tendon sheath that generally ends as soon as the tendons emerge distal to the extensor retinaculum (Fig. 9–29). At approximately the level of the metacarpophalangeal joint, the extensor digitorum communis tendon of each finger merges with a broad aponeurosis known interchangeably as the extensor expansion, the **dorsal hood,** or the **extensor hood.** The extensor indicis proprius and extensor digiti minimi tendons insert into the extensor digitorum communis tendons of the index and little fingers, respectively, at or just proximal to the extensor hood. Given the attachments of the extensor indicis proprius and extensor digiti minimi tendons to the extensor digitorum communis structure, the extensor indicis proprius and extensor digiti minimi muscles add independence of action to the index and ring fingers, rather than additional actions.

The tendons of the extensor digitorum communis, extensor indicis proprius, and extensor digiti minimi muscles show a good deal of variability on the dorsum of the hand. Most of the time, the index finger has one extensor digitorum communis tendon leading to the extensor hood and one extensor indicis proprius tendon inserting into the hood on the ulnar side of the extensor digitorum communis tendon.[140–142] At the little finger, the extensor digiti minimi tendon alone may merge with the extensor hood, with no extensor digitorum communis tendon to the little finger in as many as 30% of specimens.[143] The middle and ring fingers do not have their own auxiliary extensor muscles but frequently have two or even three extensor digitorum communis tendons leading to the hood.[142] The extensor digitorum communis tendons of

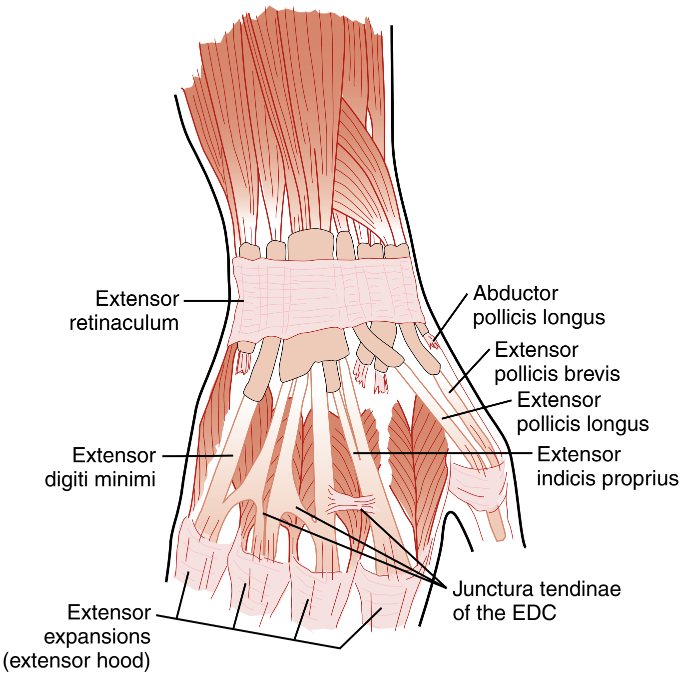

Figure 9–29 Dorsal view of the hand, illustrating the six dorsal compartments of the extensor retinaculum at the wrist, the synovial sheaths, and the finger extensors (extensor digitorum communis, extensor indicis proprius, and extensor digiti minimi muscles) that merge with the extensor expansion at the metacarpophalangeal joint. The juncturae tendinum of the extensor digitorum communis muscle lies just proximal to the metacarpophalangeal joints.

one finger may also be connected to the tendon or tendons of an adjacent finger by **junctura tendinae** (see Fig. 9–29). These fibrous interconnections (frequently visible along with the extensor tendons on the dorsum of the hand) cause active extension of one finger to be accompanied by passive extension of the adjacent finger—with the patterns of interdependence varying with the connections.[89] In general, the extensor digitorum communis, extensor indicis proprius, extensor digiti minimi, and junctura tendinae connections result in the index finger having the most independent extension, with extension of the little, middle, and ring fingers in declining order of independence.[98]

The extensor digitorum communis, extensor indicis proprius, and extensor digiti minimi are the only muscles capable of extending the metacarpophalangeal joints of the fingers. These muscles extend the metacarpophalangeal joint via their connection to the extensor hood and sagittal bands that (as we saw in discussion of the volar plates of the metacarpophalangeal joint) interconnect the volar plates and the extensor digitorum communis tendon or extensor hood.[89] Active tension on the extensor hood from one or more of these muscles will extend the metacarpophalangeal joint even though there are no direct attachments to the proximal phalanx.[144] The extrinsic extensors are also wrist extensors by continued action. Because the extensor indicis proprius and extensor digiti minimi muscles share innervation, insertion, and function with the extensor digitorum communis muscle to which each attaches, discussion of the extensor digitorum communis muscle from this point on should be assumed to include contributions from the extensor indicis proprius or the extensor digiti minimi muscles. For the sake of clarity and brevity, all three muscles will not be named each time.

Distal to the extensor hood (and therefore after the extensor indicis proprius and extensor digiti minimi tendons have joined the extensor digitorum communis tendon), the extensor digitorum communis tendon at each finger splits into three bands: the **central tendon,** which inserts on the base of the middle phalanx, and two **lateral bands,** which rejoin as the **terminal tendon** to insert into the base of the distal phalanx (Fig. 9–30).[89] Although tension on the hood can produce metacarpophalangeal joint extension, the central tendon and terminal tendon distal to the extensor expansion cannot be tightened sufficiently by activity in the extrinsic extensor muscles alone to produce extension at either the proximal or distal joints. In order to produce active interphalangeal extension, the extensor digitorum communis muscle requires the assistance of two intrinsic muscle groups that also have attachments to the extensor hood and the lateral bands. The extensor digitorum communis tendon and all its complicated active and passive interconnections at and distal to the metacarpophalangeal joint are known together as the extensor mechanism.

Extensor Mechanism

The foundation of the extensor mechanism is formed by the tendons of the extensor digitorum communis muscle (with extensor indicis proprius and extensor digiti minimi

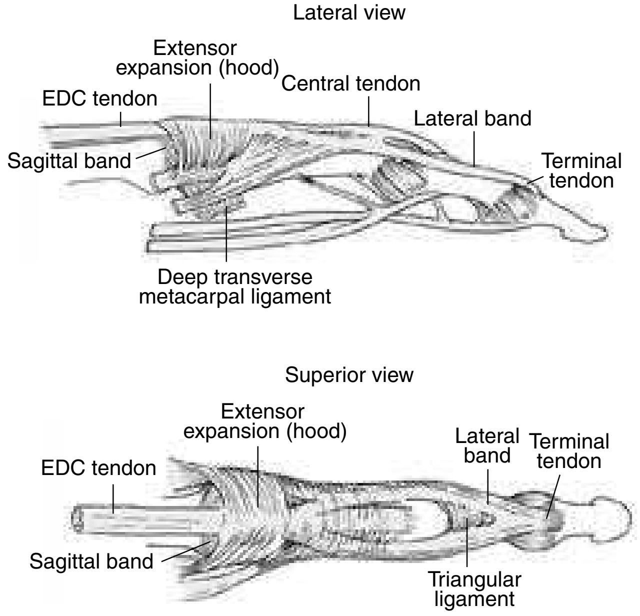

Figure 9–30 The building blocks of the extensor mechanism are the extensor digitorum communis (EDC) tendon, which merges with the extensor hood and sagittal bands and then continues distally to split into a central tendon inserting into the middle phalanx, and two lateral bands that merge into a terminal tendon that inserts into the distal phalanx. The two lateral bands are stabilized dorsally by the triangular ligament.

muscles), the extensor hood, the central tendon, and the lateral bands that merge into the terminal tendon. The first two components that we will add to the extensor mechanism are the passive components of the **triangular ligament** and the sagittal bands (see Fig. 9–30). The lateral bands are interconnected dorsally by a triangular band of superficial fibers known as the triangular, or **dorsal retinacular,** ligament.[89] The triangular ligament helps stabilize the bands on the dorsum of the finger. The sagittal bands connect the volar surface of the hood to the volar plates and deep transverse metacarpal ligament. The sagittal bands aid in stabilization not only of the volar plates but also of the hood at the metacarpophalangeal joint. The sagittal bands help to prevent bowstringing of the extensor mechanism during active metacarpophalangeal joint extension, as well as to transmit force that will extend the proximal phalanx.[89] The sagittal bands are also responsible for centralizing the extensor digitorum communis tendon over the metacarpophalangeal joint, preventing tendon subluxation.[145]

The **dorsal interossei (DI), volar interossei (VI),** and **lumbrical** muscles are the active components of the extensor mechanism (Fig. 9–31). The dorsal interossei and volar interossei muscles arise proximally from the sides of the metacarpal joints. Distally, some muscle fibers go deep to insert directly into the proximal phalanx, whereas others join with and become part of the hood that wraps around the proximal phalanx. The interossei muscles may also contribute fibers to the central tendon and both lateral bands. The lumbrical muscles attach proximally to the flexor digitorum profundus tendons and distally to the lateral band. The lumbrical and interossei muscles are together often referred to as the **intrinsic muscles** of the fingers. With the addition of the **oblique retinacular ligaments (ORLs),** the structure of the extensor mechanism for each finger is complete.

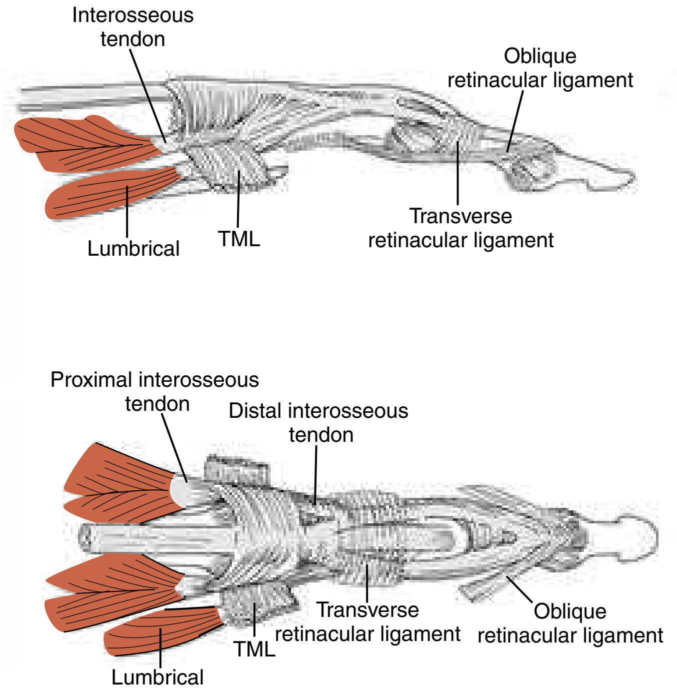

Figure 9–31 The interossei muscles pass dorsal to the transverse metacarpal ligament (TML) and may attach directly to the extensor hood or may have fibers that attach more distally to the central tendon and lateral bands. The lumbrical muscles attach to the flexor digitorum profundus tendon volarly and to the lateral bands. The oblique retinacular ligament is attached to the annular pulley proximally and to the lateral bands distally, lying just deep to the transverse retinacular ligament.

Final passive elements that contribute to the extensor mechanism are the oblique retinacular ligaments. The oblique retinacular ligaments arise from both sides of the proximal phalanx and from the sides of the annular and cruciate pulleys volarly. The oblique retinacular ligaments continue distally as slender bands to insert on the lateral bands distal to the proximal interphalangeal joint and conclude the building of the extensor mechanism (see Fig. 9–31).[146,147] The oblique retinacular ligaments lie volar to the axis of the proximal interphalangeal joint and dorsal to the axis of the distal interphalangeal joint through its attachment to the lateral bands. Function of the extensor mechanism can now be presented by looking in more detail at the active and passive elements that compose it and by referencing the relation of relevant segments to each joint individually.

Extensor Mechanism Influence on Metacarpophalangeal Joint Function

The extensor digitorum communis tendon passes dorsal to the metacarpophalangeal joint axis. An active contraction of the muscle creates tension on the sagittal bands of the extensor mechanism, pulls the bands proximally over the metacarpophalangeal joint, and extends the proximal phalanx. An isolated contraction of the extensor digitorum communis muscle will result in metacarpophalangeal joint hyperextension with interphalangeal flexion.[148–150] The accompanying interphalangeal flexion is produced by passive tension in the flexor digitorum superficialis and flexor digitorum profundus muscles when the metacarpophalangeal joint is extended. This position of the fingers (metacarpophalangeal joint hyperextension with passive interphalangeal flexion) is known as **clawing.** Similar to what we saw at the proximal carpal row of the wrist complex, clawing is the classic zigzag pattern that occurs when a compressive force is exerted across several linked segments, one of which is an unstable “intercalated” segment. In the instance of clawing of the finger, the proximal phalanx hyperextends on the metacarpal below while the middle and distal phalanx flex over it.

In order to simultaneously extend the proximal interphalangeal and distal interphalangeal joints, the extensor digitorum communis muscle requires active assistance. The other active forces that are part of the extensor mechanism are the dorsal interossei, volar interossei, and lumbrical muscles. Each of these muscles passes volar to the metacarpophalangeal joint axis and, through their connections, dorsal to the interphalangeal joints. When the extensor digitorum communis, interossei, and lumbrical muscles all contract simultaneously, the metacarpophalangeal joint will extend (as will the interphalangeal joints) because the torque produced by the extensor digitorum communis muscle at the metacarpophalangeal joint exceeds the metacarpophalangeal joint flexor torque of the intrinsic muscles. “Collapse” (excessive extension) of the proximal phalanx is prevented by active tension in the lumbrical or interossei muscles that pass volar to the metacarpophalangeal joint axis.[151] When the intrinsic muscles are weak or paralyzed (as in a low ulnar nerve injury), the extensor digitorum communis muscle is unopposed, and the fingers claw not only with active metacarpophalangeal joint extension but also at rest (Fig. 9–32). The clawing at rest demonstrates that the passive tension in the intact extensor digitorum communis muscle exceeds the passive tension in the remaining metacarpophalangeal joint flexors. The clawed position is also known as an **intrinsic minus position** because it is attributed to the absence of the finger intrinsic muscles.

Extensor Mechanism Influence on Interphalangeal Joint Function

The proximal interphalangeal and distal interphalangeal joints are joined by active and passive forces in such a way that the distal interphalangeal extension and proximal interphalangeal extension are interdependent. When the proximal interphalangeal joint is actively extended, the distal interphalangeal joint will also extend. Similarly, active distal interphalangeal extension will create proximal interphalangeal extension. The interdependence can be understood by examining structural relationships in the extensor mechanism.

Figure 9–32 In the ulnar nerve-deficient hand (claw hand) at rest, the metacarpophalangeal joints of the ring and little fingers are hyperextended because of relatively unopposed passive tension in the intact extensor digitorum communis muscle resulting from the loss of the interossei and lumbrical muscles; the interphalangeal joints are flexed because of increased passive tension in the long flexors caused by the metacarpophalangeal joint position. The index and middle fingers are less affected because these fingers still have intact lumbrical and flexor digitorum profundus muscles.

Each proximal interphalangeal joint is crossed dorsally by the central tendon and lateral bands of the extensor mechanism (see Fig. 9–30). The extensor digitorum communis, interossei, and lumbrical muscles all have attachments to the hood, central tendon, or lateral bands at or proximal to the proximal interphalangeal joint (see Fig. 9–31). Consequently, the extensor digitorum communis, interossei, and lumbrical muscles are each capable of producing at least some tension in each of the following: the central tendon, the lateral bands, and (via the lateral bands) the terminal tendon, resulting in each contributing to some extensor force at both the proximal interphalangeal and distal interphalangeal joints. An extensor digitorum communis muscle contraction alone will not produce effective interphalangeal extension. An active contraction of a dorsal interossei, volar interossei, or lumbrical muscle alone *is* capable of extending the proximal interphalangeal and distal interphalangeal joints completely because of their more direct attachments to the central tendon and lateral bands. However, if one or more of the intrinsic muscles (dorsal interossei, volar interossei, or lumbrical) contracts without a simultaneous contraction of the extensor digitorum communis muscle of that finger, the metacarpophalangeal joint will flex because each intrinsic muscles passes *volar* to the metacarpophalangeal joint axis. Although it may appear that the intrinsic muscles are independently extending the interphalangeal joints, passive tension in the extensor mechanism may be assisting the active intrinsic muscles.

Stack proposed that the interossei and lumbrical muscles would not be able to generate sufficient tension to cause independent interphalangeal extension if the extensor digitorum communis tendon was completely slack or severed.[152] *Two* sources of tension in the extensor expansion appear to be necessary to fully extend the interphalangeal joints. Source 1 is normally an active contraction of one or more of the intrinsic finger muscles. Source 2 may be either an *active contraction* of the extensor digitorum communis muscle (with active metacarpophalangeal joint extension) or *passive stretch* of the extensor digitorum communis muscle created by metacarpophalangeal joint flexion resulting from an active contraction of the intrinsic muscles.

Continuing Exploration 9-5:

Interphalangeal Joint Extension in the Absence of Intrinsic Muscles

When the finger intrinsic musculature is paralyzed, as in an ulnar nerve injury, the interossei and lumbrical muscles of the ring and little fingers cannot provide the active assistance that the extensor digitorum communis muscle needs to fully extend the interphalangeal joints. The extensor digitorum communis muscle may be able to extend the interphalangeal joints “independently” but *only* if the metacarpophalangeal joint is maintained in at least some flexion by some external force. If some passive tension in the extensor digitorum communis muscle can be attained with metacarpophalangeal joint flexion (source 1), additional tension can then be provided by an active contraction of the extensor digitorum communis muscle (source 2). These two sources (simultaneous active and passive tension in the extensor digitorum communis muscle) may be sufficient to produce full or nearly full proximal interphalangeal and distal interphalangeal joint extension in the absence of intact intrinsic muscles.[149] An external splint or surgical fixation of the metacarpophalangeal joints in a semiflexed position (Fig. 9–33A) is necessary to maintain some metacarpophalangeal joint flexion to stretch the extensor digitorum communis muscle; it also provides adequate resistance to the active metacarpophalangeal joint extensor force of the extensor digitorum communis muscle.[153] This can also be thought of as providing the means for the extensor digitorum communis muscle to strongly contract without the concomitant loss of tension that would happen if permitted to complete the metacarpophalangeal joint extension ROM. The arrangement of the splint shown here restricts metacarpophalangeal joint extension when the hand is actively opened, without restricting the ability of the intact flexor digitorum superficialis muscle (and the intact flexor digitorum profundus muscle, depending on the level of ulnar nerve injury) to actively flex the proximal joints of the ring and little fingers (Fig. 9–33B).

Some of the linkage between proximal interphalangeal and distal interphalangeal joint extension may be attributed to passive tension in the oblique retinacular ligaments. The oblique retinacular ligaments pass just volar to the proximal interphalangeal joint axis and attach distally to the lateral bands (see Fig. 9–31). Tension will increase in the oblique retinacular ligaments as the proximal interphalangeal joint is extended (actively or passively) *if* the lateral bands and their terminal tendon are already tensed by distal interphalangeal flexion. Consequently, proximal interphalangeal extension may make a contribution to initiation of distal interphalangeal extension through passive tension in the oblique retinacular ligaments. The lengths of the oblique retinacular ligaments are such, however, that the contribution of proximal interphalangeal extension to distal interphalangeal extension via the oblique retinacular ligaments may be significant only during the first half of the distal interphalangeal joint's return from flexion (90° to 45° flexion), when the oblique retinacular ligaments are most stretched.[146,152] Overall, the complex structure of the extensor expansion and its contributing active and passive elements result in a relative linking between proximal interphalangeal and distal interphalangeal extension.[10,11,148,150]

Flexion of the distal interphalangeal joint produces flexion of the proximal interphalangeal joint by a similar complex combination of active and passive forces that link proximal interphalangeal and distal interphalangeal joint extension. When the distal interphalangeal joint is flexed by the flexor digitorum profundus muscle, a simultaneous flexor force is applied over both joints crossed by the flexor digitorum profundus muscle, and so simultaneous distal interphalangeal and proximal interphalangeal joint flexion are not surprising. However, the active force of the flexor digitorum profundus muscle on the distal phalanx might *not* be sufficient to produce simultaneous proximal interphalangeal flexion if extensor restraining forces at the proximal interphalangeal joint were not released at the same time.

When distal interphalangeal flexion is initiated by the flexor digitorum profundus muscle, the terminal tendon and its lateral bands are stretched over the dorsal aspect of the

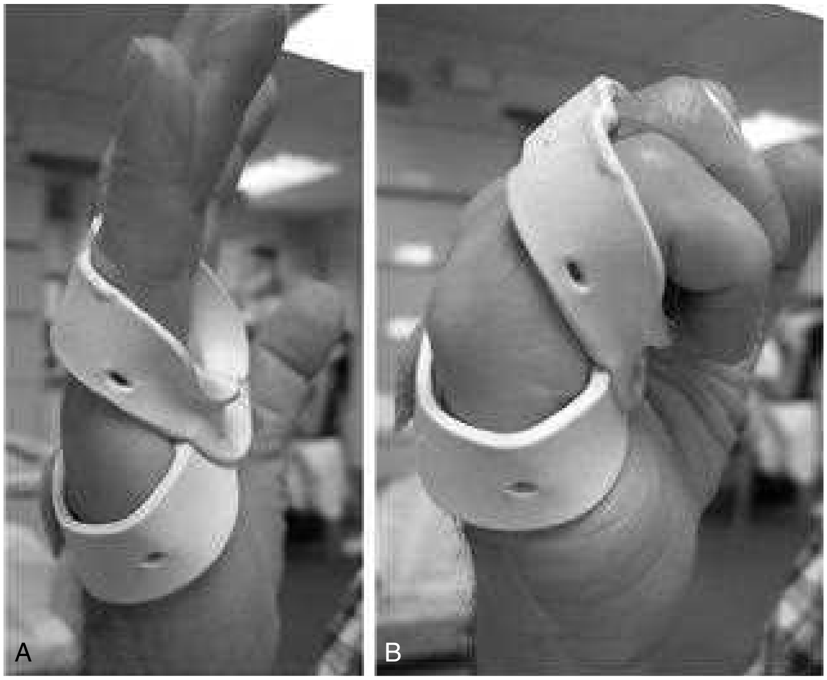

Figure 9–33 **A.** In the ulnar nerve-deficient hand, the extensor digitorum communis muscle alone can extend the interphalangeal joints of the ring and little fingers in the absence of the interossei and lumbrical muscles if full metacarpophalangeal joint extension is prevented by a splint during an active extensor digitorum communis muscle contraction. **B.** The splint is shaped so that the intact long finger flexor or flexors can flex the fingers with minimal interference by the splint.

distal interphalangeal joint. The stretch in the lateral bands pulls the extensor hood (from which the lateral bands arise) distally. The distal migration in the extensor hood causes the central tendon of the extensor expansion to relax, releasing its extensor influence at the proximal interphalangeal joint and facilitating proximal interphalangeal flexion. The simultaneous flexor torque and release of extensor torque still might not be adequate for the flexor digitorum profundus muscle to flex the proximal interphalangeal joint if the lateral bands remained taut on the dorsal aspect of the proximal interphalangeal joint. The bands, however, are permitted to separate somewhat by the elasticity of the interconnecting triangular ligament and are assisted by passive tension in the **transverse retinacular ligament** (see Fig. 9–31). Although the example used here was initiated by an active contraction of the flexor digitorum profundus muscle, the same set of mechanisms will tie passive distal interphalangeal flexion (produced by an external flexor force) to passive proximal interphalangeal flexion.

Continuing Exploration 9-6:

Finger Tricks: Distal Interphalangeal Joint Flexion with Proximal Interphalangeal Joint Extension

The normal coupling of distal interphalangeal joint flexion with proximal interphalangeal joint flexion can be overridden by some individuals; that is, some people can actively flex a distal interphalangeal joint (using the flexor digitorum profundus muscle) while maintaining the proximal interphalangeal joint in extension (Fig. 9–34). This "trick" is due to the influence of the oblique retinacular ligaments and requires some proximal hyperextension of the finger. When the proximal interphalangeal joint can be sufficiently hyperextended, the oblique retinacular ligaments that ordinarily lie *just* volar to the proximal interphalangeal joint axis will migrate *dorsal* to the proximal interphalangeal joint axis. At that point, tension in the oblique retinacular ligaments produced by active distal interphalangeal joint flexion (stretch of the terminal tendon and lateral bands to which the oblique retinacular ligaments attach) will accentuate proximal interphalangeal joint *extension* because the oblique retinacular ligaments function as passive proximal interphalangeal joint extensors. The trick of active distal interphalangeal joint flexion and proximal interphalangeal joint extension serves no functional purpose and can be accomplished only in fingers in which proximal interphalangeal jont hyperextension is available. The "trick" does, however, highlight the necessity of releasing extensor tension at the proximal interphalangeal joint before the flexor digitorum profundus muscle can effectively flex that joint.

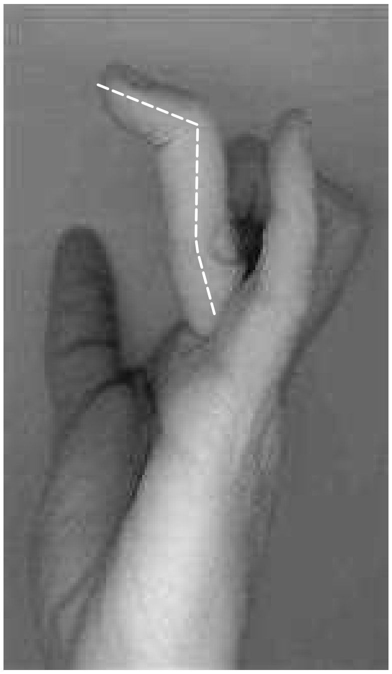

Figure 9–34 Some individuals can actively flex the distal interphalangeal joint in the presence of proximal interphalangeal joint extension. This generally requires that the individual have proximal interphalangeal joint hyperextension, so that in the extended finger, the oblique retinacular ligaments migrate dorsal to the proximal interphalangeal joint axis. With initiation of distal interphalangeal joint flexion, tension in the terminal tendon and lateral bands caused by the distal interphalangeal joint flexion tenses the oblique retinacular ligaments that serve as proximal interphalangeal joint extensors rather than flexors.

The functional coupling of proximal/distal interphalangeal joint action can be demonstrated by one other proximal/distal interphalangeal joint relation.[57] When the proximal interphalangeal joint is fully flexed actively (by the flexor digitorum superficialis muscle) or passively (by an external force), the distal interphalangeal joint cannot be actively extended. When the proximal interphalangeal joint is flexing, the dorsally located central tendon is becoming stretched. The increasing tension in the central tendon pulls the extensor hood (from which the central tendon arises) distally. This distal migration of the hood releases some of the tension in the lateral bands. The tension in the lateral bands is further released as the lateral bands separate slightly at the flexing proximal interphalangeal joint. Releasing tension in the lateral bands releases tension in the terminal tendon on the distal phalanx. As 90° of proximal interphalangeal flexion is reached, loss of tension in the terminal tendon completely eliminates any extensor force at the distal interphalangeal joint, including any potential contribution from the oblique retinacular ligaments that have also been released by proximal interphalangeal flexion.[154] Although the distal interphalangeal joint can be actively flexed by the flexor digitorum profundus muscle when the proximal phalanx is already flexed, the distal phalanx cannot be actively re-extended as long as the proximal interphalangeal joint remains flexed.

Concept Cornerstone **9-4**

Summary of Coupled Actions of the Proximal and Distal Interphalangeal Joints

- Active extension of the proximal interphalangeal joint will normally be accompanied by extension of the distal interphalangeal joint.
- Active or passive flexion of the distal interphalangeal joint will normally initiate flexion of the proximal interphalangeal joint.
- Full flexion of the proximal interphalangeal joint (actively or passively) will prevent the distal interphalangeal joint from being actively extended.

Intrinsic Finger Musculature

Dorsal and Volar Interossei Muscles

The dorsal interossei and volar interossei muscles, as already noted, arise from between the metacarpals and are an important part of the extensor mechanism. There are four dorsal interossei muscles (one to each finger) and three to four volar interossei muscles. Many (but not all) anatomy texts describe the thumb as having the first volar interossei muscle. Mardel and Underwood suggested that the discrepancy may be in whether the controversial muscle is considered a separate volar interossei muscle or as part of the **flexor pollicis brevis (FPB).**[155] Although we will consider the thumb as having the first volar interossei muscle, at this time we will consider only the action of the volar interossei and dorsal interossei muscles of the fingers. Because the dorsal interossei and volar interossei muscles are alike in location and in some of their actions, these two muscle groups are often characterized by their ability to produce metacarpophalangeal joint abduction and adduction, respectively. Additional detail on the variable attachments of these muscles, as well as studies revealing multiple muscle heads, has increased our understanding of their contribution to hand function.[156] We will now look at how the attachments of the interossei muscles affect their role as metacarpophalangeal joint flexors or stabilizers and as interphalangeal extensors.

The interossei muscle fibers join the extensor expansions in two locations. Some fibers attach *proximally* to the proximal phalanx and to the extensor hood; some fibers attach more *distally* to the lateral bands and central tendon (see Fig. 9–31). Although individual variations in muscle attachments exist, studies have found some consistency in the point of attachment of the different interossei muscles.[147,152,157] The first dorsal interossei muscle has the most consistent attachment of its group, inserting entirely into the bony base of the proximal phalanx and the extensor hood. The dorsal interossei muscles of the middle and ring fingers (with the middle finger having a dorsal interossei muscle on each side) each have both proximal and distal attachments (to the proximal phalanx/hood and to the lateral bands/central tendon). The little finger does not have a dorsal interossei muscle. The **abductor digiti minimi (ADM)** muscle is, in effect, a dorsal interossei muscle and typically has only a proximal attachment (proximal phalanx/hood).[98] The three volar interossei muscles of the fingers consistently appear to have distal attachments only (attachments to the lateral bands/central tendon). Conceptually, then, we can establish a frame of reference in which we can summarize the dorsal interossei and volar interossei attachments as follows: The first dorsal interossei muscle has only a proximal attachment; the second, third, and fourth dorsal interossei muscles have both proximal and distal attachments; the “fifth dorsal interossei” muscle (the abductor digiti minimi) has only a proximal attachment; and the three volar interossei muscles of the fingers have only distal attachments.

Given the particular proximal or distal attachment patterns of the dorsal interossei, volar interossei, and abductor digiti minimi muscles, these muscles can be characterized not only as abductors or adductors of the metacarpophalangeal joint but also as proximal or distal interossei according to the pattern of attachment. Proximal interossei will have their predominant effect at the metacarpophalangeal joint alone, whereas the distal interossei will produce their predominant action at the interphalangeal joints, with some effect by continued action at the metacarpophalangeal joint.

All of the dorsal interossei and volar interossei muscles (regardless of their designation as proximal or distal) pass *dorsal* to the transverse metacarpal ligament but just volar to the axis for metacarpophalangeal joint flexion/extension. All the interossei muscles, therefore, are potentially flexors of the metacarpophalangeal joint. The ability of the interossei muscles to flex the metacarpophalangeal joint, however, will vary somewhat with metacarpophalangeal joint position.

Role of the Interossei Muscles at the Metacarpophalangeal Joint in Metacarpophalangeal Joint Extension

When the metacarpophalangeal joint is in extension, the moment arm (and rotary component) of all the interossei muscles for metacarpophalangeal joint flexion is so small that little flexion torque is produced. Given that the action lines of the interossei muscles pass almost directly through the coronal axis when the metacarpophalangeal joint is in extension, the interossei muscles are not very effective flexors when the metacarpophalangeal joint is extended. In spite of their poor flexor torque with the metacarpophalangeal joint extended, the interossei muscles can be effective stabilizers (joint compressors) and appear to be important in helping to prevent clawing (metacarpophalangeal joint hyperextension) of the fingers.[98,148]

There is typically no EMG activity recorded in the interossei muscles when the hand is at rest, when there is isolated extensor digitorum communis muscle activity, or when there is combined extensor digitorum communis/flexor digitorum profundus muscle activity. However, when these activities are performed in a hand with long-standing ulnar nerve paralysis (therefore, no interossei muscles), an exaggerated metacarpophalangeal joint extension or hyperextension (clawing) results. In a low ulnar nerve injury, the index and middle fingers retain a lumbrical muscle as well as both the flexor digitorum superficialis and flexor digitorum profundus muscles. The loss of the interossei muscles is reflected by a resting metacarpophalangeal joint posture of neutral flexion/extension, rather than slight flexion, as is usually observed. The ring and little fingers, missing both interossei and lumbrical muscles in a hand with ulnar nerve deficit, will assume a metacarpophalangeal joint hyperextended and interphalangeal flexed posture at rest (clawing) even in the presence of intact flexor digitorum superficialis and flexor digitorum profundus muscles.[10,11,86,148] Clawing is not evident at rest in the ulnar nerve-deficient hand until the viscoelastic tension in the interossei muscles has been lost through atrophy and the volar plates have stretched out. Once such atrophy occurs, the predominance of extensor digitorum communis muscle tension even in relaxation is evidenced by the metacarpophalangeal joint posture assumed by each finger of the hand at rest (see Fig. 9–32). The role of the interossei muscles in balancing passive tension in the extrinsic extensors at the metacarpophalangeal joints at rest appears, therefore, to be provided by *passive* viscoelastic tension in the muscle.

Continuing Exploration 9-7:

Wartenberg's Sign

In addition to the clawing evident in the ring and little fingers in the ulnar nerve-deficient hand, the little finger may also assume a metacarpophalangeal joint abducted position with loss of the intrinsic muscles. Abduction of the little finger (Wartenberg's sign) may be the result of the unbalanced pull of the extensor digiti minimi muscle among those individuals having a direct connection of the extensor digiti minimi muscle to the abductor tubercle of the proximal phalanx—the only one of the extensor tendons that has an insertion directly on to the proximal phalanx in any substantial number of people.[140]

When the metacarpophalangeal joint is extended, the interossei (and abductor digiti minimi) muscles lie at a relatively large distance from the A-P axis for metacarpophalangeal joint abduction/adduction. Consequently, in the metacarpophalangeal joint extended position, the interossei (and abductor digiti minimi) muscles are effective abductors or adductors of the metacarpophalangeal joint without the loss of tension that would occur if the muscles were simultaneously producing metacarpophalangeal joint flexion. The interossei muscles that insert proximally (on the proximal phalanx/hood) are better as metacarpophalangeal joint abductors/adductors, whereas the interossei muscles with more distal insertions (lateral bands/central tendon) are less effective at the metacarpophalangeal joint because they must act on the metacarpophalangeal joint by continued action. In our conceptual framework, all the dorsal interossei muscles (metacarpophalangeal joint abductors) have proximal insertions, and the volar interossei muscles (metacarpophalangeal joint adductors) have only distal insertions. Therefore, it makes sense that metacarpophalangeal joint abduction is stronger than metacarpophalangeal joint adduction. The dorsal interossei muscles also have twice the muscle mass of the volar interossei muscles. In a progressive ulnar nerve paralysis, the relatively ineffective metacarpophalangeal joint adduction component of the volar interossei muscles is the first to show weakness.

Role of the Interossei Muscles at the Metacarpophalangeal Joint in Metacarpophalangeal Joint Flexion. As the metacarpophalangeal joint flexes from the extended position, the tendons and action lines of the interossei muscles migrate volarly away from the metacarpophalangeal joint's coronal axis, increasing the moment arm for metacarpophalangeal flexion. In fact, in full metacarpophalangeal joint flexion, the action lines of the interossei muscles are nearly perpendicular to the moving segment (proximal phalanx) (Fig. 9–35). Consequently, the ability of the interossei muscles to create a metacarpophalangeal joint flexion torque increases as the metacarpophalangeal joint moves toward full flexion. As the metacarpophalangeal joint approaches full flexion, the volar migration of the interossei muscles is restricted by deep transverse metacarpal ligament that lies volar to the tendons.

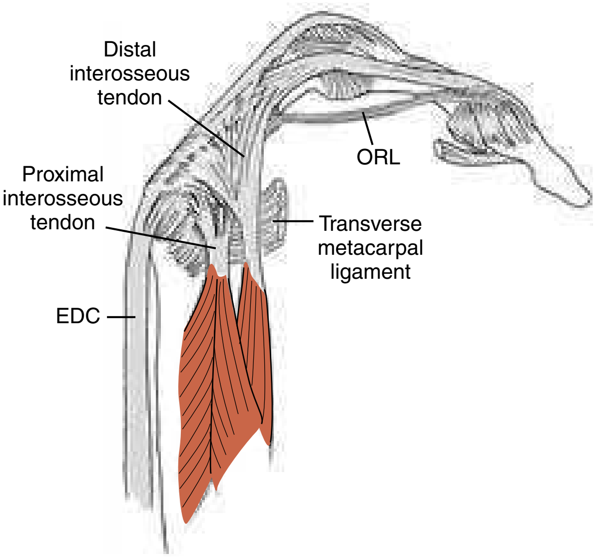

Figure 9–35 When the metacarpophalangeal joint is flexed, the interossei muscles (those with both proximal and distal attachments) migrate volarly away from the metacarpophalangeal joint axis for flexion/extension, which results in a relatively large moment arm and a line of pull that is nearly perpendicular to the proximal phalanx. The volar migration of the interossei muscles is limited by the deep transverse metacarpal ligament, which prevents loss of tension and serves as an anatomical pulley. EDC, extensor digitorum communis; ORL, oblique retinacular ligament.

Although the deep transverse metacarpal ligament limits the moment arm of the interossei muscles, the ligament also both prevents the loss of active tension that would occur with bowstringing and serves as an anatomical pulley. With increased metacarpophalangeal joint flexion, the collateral ligaments of the metacarpophalangeal joint also become increasingly taut. The increasing tension in the collateral ligaments helps prevent the loss of metacarpophalangeal joint flexor force that would occur if the interossei muscles concomitantly produced metacarpophalangeal joint abduction/adduction. In full metacarpophalangeal joint flexion, metacarpophalangeal joint abduction and adduction are completely restricted by tight collateral ligaments, by the shape of the condyles of the metacarpal head, and by active insufficiency of the fully shortened interossei muscles. The net effect of these combined mechanisms is that the ability of the interossei muscles to produce a metacarpophalangeal joint flexion torque (in the metacarpophalangeal flexed position) makes them powerful metacarpophalangeal joint flexor muscles[158] that contribute to grip when a strong pinch or grip is required.[125,159]

Role of the Interossei Muscles at the Interphalangeal Joint in Interphalangeal Joint Extension. The ability of the interossei muscles to produce interphalangeal joint extension is influenced by their attachments. To create sufficient tension in the extensor mechanism to contribute effectively to interphalangeal extension, the muscles must attach to the central tendon or lateral bands. All the interossei muscles have distal attachments except the two "outside" abductors on the first and fourth fingers (first dorsal interossei and abductor digiti minimi muscles).

When the metacarpophalangeal joint is extended, the action lines of the distal interossei are ineffective in producing metacarpophalangeal joint flexion (because of the poor moment arm) but capable of extending the interphalangeal joints because the distal interossei attach directly to the central tendon and lateral bands. The interphalangeal extension produced by the distal interossei is stronger than the metacarpophalangeal joint abduction/adduction action because the abduction/adduction is produced by continued action. We have already noted that when the metacarpophalangeal joint flexes, the tendons of the interossei muscles migrate volarly at the metacarpophalangeal joint but are restricted in their volar excursion by the deep transverse metacarpal ligament. The deep transverse metacarpal ligament prevents the interosseous tendons from becoming slack through volar migration and has a pulley effect on the distal tendons. The anatomical pulley effect of the deep transverse metacarpal ligament may enhance the function of the distal interossei muscles, because interphalangeal extension appears to be more effective in metacarpophalangeal joint flexion than in metacarpophalangeal joint extension.

The index and little fingers each have only one interosseous muscle with a distal insertion (second and fourth volar interossei muscles, respectively). The middle and ring fingers each have two distal tendons (second and third dorsal interossei muscles for the middle finger, and fourth volar interossei and fourth dorsal interossei muscles for the ring finger). The index and little fingers, therefore, are weaker in interphalangeal extension than are the middle and ring fingers because they have fewer distal interossei muscles.[157]

Overall, in approaching or holding the position of metacarpophalangeal flexion and interphalangeal extension, both proximal and distal insertions of the interossei muscles contribute to the metacarpophalangeal joint flexion torque. The proximal components are effective metacarpophalangeal joint flexors, and the distal components are effective as both metacarpophalangeal joint flexors and interphalangeal extensors. The most consistent activity of the interossei muscles appears to occur when the metacarpophalangeal joints are being flexed and the interphalangeal joints are simultaneously extended,[148,160] a position that takes advantage of optimal biomechanics for both the proximal and the distal interossei muscles.

Lumbrical Muscles

The lumbrical muscles are the only muscles in the body that attach at both ends to tendons of other muscles. Each muscle arises from a tendon of the flexor digitorum profundus muscle in the palm, passes volar to the deep transverse metacarpal ligament, and attaches to the lateral band of the extensor mechanism on the radial side (see Fig. 9–31).[161] Like the interossei muscles, the lumbrical muscles cross the metacarpophalangeal joint volarly and the interphalangeal joints dorsally. Differences in function in the two intrinsic muscle groups can be attributed to the more distal insertion of the lumbrical muscles on the lateral band as compared to the distal interossei, to the lumbrical's flexor digitorum profundus tendon origin, and to the lumbrical's great contractile range.

The insertion of the lumbrical muscles on the lateral bands of the extensor mechanism is distal to the attachment of the distal interossei muscles, making the lumbricals consistently effective interphalangeal extensors regardless of metacarpophalangeal joint position. Studies have found the lumbrical muscles to be more frequently active as interphalangeal extensors in the metacarpophalangeal joint extended position than are the interossei muscles.[148,162] The deep transverse metacarpal ligament prevents the volarly located lumbrical muscle from migrating dorsally and losing tension as the metacarpophalangeal and interphalangeal joints extend. When a lumbrical muscle contracts, it pulls not only on its distal attachment (the lateral band) but also on its proximal attachment (the flexor digitorum profundus tendon). Because the proximal attachment of the lumbrical muscle is on a somewhat movable tendon, shortening of the lumbrical muscle not only increases tension in the lateral bands to extend the interphalangeal joints but also pulls the flexor digitorum profundus tendon distally in the palm. The distal migration of the flexor digitorum profundus tendon releases much of the passive flexor force of the inactive flexor digitorum profundus muscle at the metacarpophalangeal and interphalangeal joints (Fig. 9–36). Ranney and Wells confirmed this, finding that the lumbrical muscles did not begin to extend the interphalangeal joints until the tension within the lumbrical muscle equaled the tension in the flexor digitorum profundus tendon (produced by the

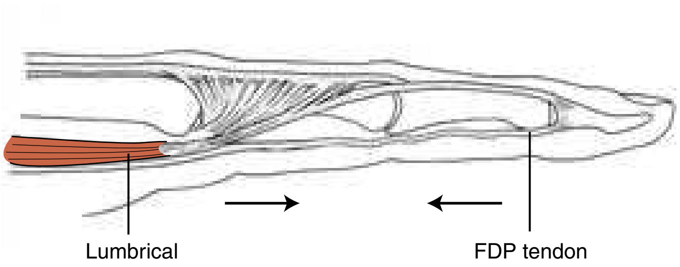

Figure 9–36 The lumbrical muscle attaches to the flexor digitorum profundus (FDP) tendon proximally and to the lateral band of the extensor expansion distally. A contraction of the lumbrical muscle will create tension in the lateral band, leading to proximal/ distal interphalangeal joint extension, while concomitantly pulling the flexor digitorum profundus tendon distally and releasing the passive flexor tension that could impede interphalangeal extension.

lumbrical muscle's distal pull on the flexor digitorum profundus tendon).[151] Given these circumstances, the lumbrical muscles might be considered to be both agonists and synergists for interphalangeal extension. Tension in the lumbrical muscles on the lateral bands produces interphalangeal extension, while the lumbrical muscle simultaneously releases antagonistic tension in the flexor digitorum profundus tendon.[163] The distal insertion of the interossei muscles can extend the interphalangeal joints. However, they are less effective as interphalangeal extensors in the absence of the lumbrical muscles, because the interossei muscles do not have the same ability to release the passive resistance of the flexor digitorum profundus tendon to interphalangeal extension.

The complexity of the interconnections of the intrinsic muscles with the extensor mechanism can be highlighted not only by the interrelationships of the interossei muscles but also by the lumbrical muscle's interdependence with the flexor digitorum profundus and extensor muscle expansion to produce interphalangeal extension. Although active interphalangeal extension is facilitated by the active lumbrical muscle's effective release of passive flexor digitorum profundus tendon tension, the lumbrical muscle is also dependent on flexor digitorum profundus tendon tension; that is, *some* tension in the flexor digitorum profundus tendon is critical to lumbrical function. If passive tension were not present in the tendon of the inactive flexor digitorum profundus muscle (e.g., if the flexor digitorum profundus tendon were cut), an active lumbrical contraction would pull the flexor digitorum profundus tendon so far distally that the lumbrical would become actively insufficient and ineffective as an interphalangeal extensor. Similarly, active or passive tension in the extensor digitorum communis tendon and extensor expansion are necessary as one source of tension before the second source of tension, the active lumbrical muscle, can be effective in fully extending both interphalangeal joints.[152] The lumbrical muscles may also assist the flexor digitorum profundus muscle indirectly with hand closure. When the flexor digitorum profundus muscle contracts, the flexor digitorum profundus tendon moves proximally, carrying its associated (presumably passive) lumbrical muscle along with it. This creates a passive pull of the lumbrical muscle on the lateral band during hand closure that may assist the flexor digitorum profundus muscle in flexing the metacarpophalangeal joint *before* the interphalangeal joints, which avoids the problem of catching the fingertips in the palm during grasp as occurs in the intrinsic minus hand.[158]

The lumbrical muscles' role as a metacarpophalangeal joint flexor is relatively minimal. The lumbrical muscles actually have a greater moment arm for metacarpophalangeal joint flexion than do the interossei muscles because the lumbrical muscles lie volar to the interossei muscles. Functionally, however, metacarpophalgeal flexion is weaker in the lumbrical muscles than in the interossei muscles.[148,157,158,162,164] This relative weakness may be attributed to the small cross-section of the lumbrical muscles in comparison with the interossei muscles. However, it may also have to do with the moving attachment of the lumbrical muscle on the flexor digitorum profundus tendon. A contraction of a lumbrical muscle causes the associated flexor digitorum profundus tendon to migrate distally and carries the lumbrical muscle along with it. The distal migration of the flexor digitorum profundus tendon and lumbrical muscle has the effect both of releasing passive tension in the inactive flexor digitorum profundus tendon that might contribute to metacarpophalangeal joint flexion and of minimizing the active force of the lumbrical muscle at the metacarpophalangeal joint. Although the active lumbrical muscles may lose tension at the metacarpophalangeal joint in metacarpophalangeal flexion, metacarpophalangeal flexion does not appear to weaken the lumbricals' effectiveness as interphalangeal extensors. The unusually large contractile range of the lumbrical muscles seems to prevent the lumbrical muscles from becoming actively insufficient when shortening both over the metacarpophalangeal joints and at the interphalangeal joints.

Concept Cornerstone 9-5

Intrinsic Muscles Summary

The complex functions of the interossei muscles are summarized in Table 9–1. The function of the lumbrical muscles is simpler than that of the interossei muscles. The lumbrical muscles are strong extensors of the interphalangeal joints, regardless of metacarpophalangeal joint position. The lumbrical muscles are also relatively weak metacarpophalangeal joint flexors, regardless of metacarpophalangeal joint position. The ability of the lumbrical muscles to extend the interphalangeal joints appears to depend only on intact tension in the extensor mechanism and in the flexor digitorum profundus tendons. When the lumbrical and interossei muscles contract together without any extrinsic finger muscle activity, these muscles produce flexion and interphalangeal extension, the so-called **intrinsic plus** position of the hand (Fig. 9–37A). When the extrinsic finger flexors and extensors are active without any concomitant activity of the intrinsic muscles, the hand assumes an intrinsic minus position (Fig. 9–37B).

Table 9–1 Summary of Interossei Muscle Action

MUSCLE	ATTACHMENTS	ACTION	
		MP Extended	*MP Flexed*
		First Finger	
DI	Proximal only	MP abduction	MP flexion
VI	Distal only	IP extension and MP adduction*	IP extension and MP flexion*
		Second Finger	
DI	Proximal and distal	MP abduction and IP extension	MP flexion and IP extension
DI	Proximal and distal	MP abduction and IP extension	MP flexion and IP extension
		Third Finger	
DI	Proximal and distal	MP abduction and IP extension	MP flexion and IP extension
VI	Distal only	IP extension and MP adduction*	IP extension and MP flexion*
		Fourth Finger	
DI	Proximal only	MP abduction	MP flexion
VI	Distal only	IP extension and MP adduction*	IP extension and MP flexion*

*Occurs indirectly by continued action.
DI, dorsal interossei; IP, interphalangeal; MP, metacarpophalangeal; VI, volar interossei.

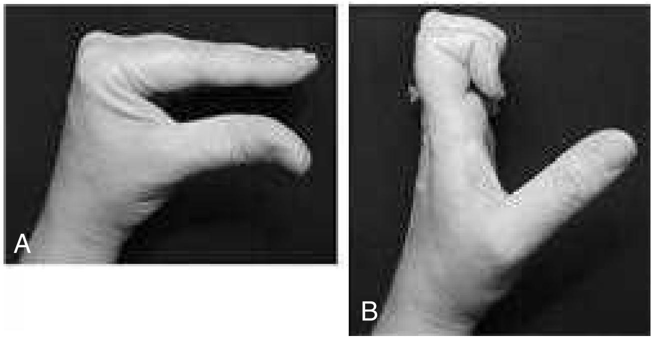

Figure 9–37 **A.** Activity of the lumbrical and interossei muscles without any extrinsic finger flexors or extensors produces the "intrinsic plus" position of the hand. **B.** Activity of the extrinsic finger flexors and extensors without any activity of intrinsic finger muscles produces the "intrinsic minus" position of the hand.

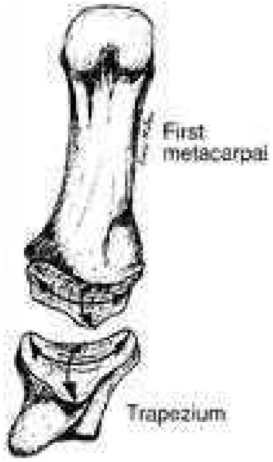

Figure 9–38 The saddle-shaped portion of the trapezium is concave in the sagittal plane (abduction/adduction) and convex in the frontal plane (flexion/extension). The spherical portion found near the anterior radial tubercle is convex in all directions. The base of the first metacarpal joint has a shape reciprocal to that of the trapezium.

Structure of the Thumb

Carpometacarpal Joint of the Thumb

The carpometacarpal (or **trapeziometacarpal [TM]**) joint of the thumb is the articulation between the trapezium and the base of the first metacarpal. Unlike the carpometacarpal joints of the fingers, the first carpometacarpal joint is a saddle joint with two degrees of freedom: flexion/extension and abduction/adduction (Fig. 9–38).[165] The joint also permits some axial rotation, which occurs concurrently with the other motions. The net effect at this joint is a circumduction motion commonly termed **opposition.** Opposition permits the tip of the thumb to oppose the tips of the fingers.

First Carpometacarpal Joint Structure

Zancolli and associates proposed that the first carpometacarpal joint surfaces consist not only of the traditionally described saddle-shaped surfaces but also of a spherical portion located near the anterior radial tubercle of the trapezium.[166] The saddle-shaped portion of the trapezium is concave in the sagittal plane (abduction/adduction) and convex in the frontal plane (flexion/extension). The spherical portion is convex in all directions. The base of the first metacarpal has a reciprocal shape to that of the trapezium (see Fig. 9–38). Flexion/extension and abduction/adduction are proposed to occur on the saddle-shaped surfaces, whereas the axial rotation of the metacarpal that accompanies opposition is proposed to occur on the spherical surfaces.[166] Flexion/extension of the joint occurs around a somewhat oblique A-P axis, whereas abduction/adduction occurs around an oblique coronal axis. This is a reversal of what is found at most other joints, with flexion/extension usually occurring around a coronal axis and abduction/adduction around an A-P axis. The change in the carpometacarpal joint motions occurs because of the orientation of the trapezium, which effectively rotates the volar surface of the thumb medially. As a consequence, flexion/extension occurs nearly parallel to the palm, with abduction/adduction occurring nearly perpendicular to the palm. Cooney and associates measured the first carpometacarpal joint ROM as an average of 53° of flexion/extension, 42° of abduction/adduction, and 17° of rotation.[167]

The capsule of the carpometacarpal joint is relatively lax but is reinforced by radial, ulnar, volar, and dorsal ligaments. There is also an **intermetacarpal ligament** that helps tether the bases of the first and second metacarpals, preventing extremes of radial and dorsal displacement of the base of the first metacarpal joint.[166,168] The **dorsoradial** and **anterior oblique ligaments** are reported to be key stabilizers of the carpometacarpal joint.[169,170] Although some investigators hold that the axial rotation seen in the metacarpal during opposition is a function of incongruence and joint laxity,[11,171] Zancolli and associates theorized that it is a result of the congruence of the spherical surfaces and resultant tensions encountered in the supporting ligaments.[166] It seems, however, that some incongruence must exist at the joint. Osteoarthritic (OA) changes with aging are common at the first carpometacarpal joint and may be attributable to the cartilage thinning in high-load areas imposed on this joint by pinch and grasp across incongruent surfaces.[172] Ateshian and colleagues found gender differences in the fit of the trapezium with the metacarpal, with the trapezium of women showing more incongruence than that of men in a group of older individuals.[173] This matches an increased incidence of OA of the first carpometacarpal joint among older women, but it does not address whether the incongruence of the trapezium is a cause or effect of degenerative changes. The first carpometacarpal joint is close-packed both in extremes of abduction and adduction, with maximal motion available in neutral position.[107]

First Carpometacarpal Joint Function

It is the unique range and direction of motion at the first carpometacarpal joint that produces opposition of the thumb. Opposition is, sequentially, abduction, flexion, and adduction of the first metacarpal, with simultaneous rotation.[174] These movements change the orientation of the metacarpal, bringing the thumb out of the palm and positioning the thumb for contact with the fingers. The functional significance of the carpometacarpal joint of the thumb and of the movement of opposition can be appreciated when one realizes that use of the thumb against a finger occurs in almost all forms of **prehension** (grasp and dexterity activities). When the first carpometacarpal joint is fused in extension and adduction, opposition cannot occur. The importance of opposition is such that fusion of the first carpometacarpal joint may be followed over time by an adaptation of the **trapezioscaphoid joint** that develops a more saddle-shaped configuration to restore some of the lost opposition.[111] This amazing shift in joint function is an excellent example of the body's ability to replace essential functions whenever possible.

9-4 **Patient Case** case

Carpometacarpal Osteoarthritis

Marilyn Ferrier is a 78-year-old woman referred to a hand surgeon by her primary care physician after she reported progressive onset of thumb pain. Her symptoms of aching and tenderness were exacerbated by activities such as turning keys, opening jars, and writing. Manual longitudinal compression of the first metacarpal joint into the trapezium produced pain and crepitation (positive Grind test), indicative of carpometacarpal osteoarthritis. Osteoarthritic changes and subluxation of the metacarpal were evident on radiograph (Fig. 9–39). A custom thumb splint was fabricated, and the patient was instructed in activity modifications along with general joint protection principles, including avoiding forceful, repetitive, and sustained pinching, along with utilizing pens and kitchen utensils with larger handles.

Figure 9–39 Degenerative changes between the trapezium and first metacarpal joint (first carpometacarpal joint) cause painful opposition.

Metacarpophalangeal and Interphalangeal Joints of the Thumb

The metacarpophalangeal joint of the thumb is the articulation between the head of the first metacarpal and the base of its proximal phalanx. It is considered to be a condyloid joint with two degrees of freedom: flexion/extension and abduction/adduction.[98] There is an insignificant amount of passive rotation.[111] The metacarpal head is not covered with cartilage dorsally or laterally, and it more closely resembles the head of the proximal phalanx, without the central groove of the proximal phalanx. The joint capsule, the reinforcing volar plate, and the collateral ligaments are similar to those of the other metacarpophalangeal joints. The main functional contribution of the first metacarpophalangeal joint is to provide additional flexion range to the thumb in opposition and to allow the thumb to grasp and contour to objects. Despite the structural similarities between the metacarpophalangeal joints, the first metacarpophalangeal joint is far more restricted in motion than those of the fingers. Although the available range varies significantly among individuals, the first metacarpophalangeal joint rarely has more than half the flexion available at the fingers and little if any hyperextension. Abduction/adduction and rotation are extremely limited. This limitation to motion is probably attributable to the major structural difference between the metacarpophalangeal joints of the thumb and fingers. The first metacarpophalangeal joint is reinforced extracapsularly on its volar surface by two sesamoid bones (Fig. 9–40). These are maintained in position by fibers from the collateral ligaments and by an **intersesamoid ligament.** Goldberg and Nathan proposed that the sesamoid bones are the result of friction and pressure on the tendons in which the sesamoid bones are embedded.[175] They support this by noting that the sesamoid bones of the first metacarpophalangeal joint do not appear until around 12 years of age and that sesamoid bones in some investigations have also been found in as many as 70% of fifth metacarpophalangeal joints and 50% of second metacarpophalangeal joints.

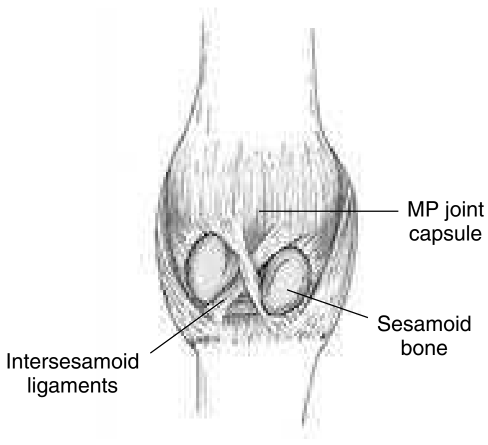

Figure 9–40 The metacarpophalangeal joint of the thumb has two sesamoid bones secured to the volar aspect of the joint capsule by intersesamoid ligaments.

The interphalangeal joint of the thumb is the articulation between the head of the proximal phalanx and the base of the distal phalanx. It is structurally and functionally identical to the interphalangeal joints of the fingers.

Thumb Musculature

The muscles of the thumb have been compared to guy wires supporting a flagpole, in which there must be a continuous effective pull in every direction to maintain stability. The metacarpal joint and the proximal and distal phalanges form an articulated shaft that sits on the trapezium. As in the flagpole, tension from the muscular guy wires must be provided in every direction for stability to be maintained. Because the stability comes from the muscles more so than it does from articular constraints (at least at the carpometacarpal joint), the majority of muscles that attach to the thumb tend to be active during most thumb motions. There is also substantial individual variability in motor strategies among normal subjects.[176] Consequently, exploration of muscle function in the thumb (and, to a somewhat lesser extent, function through the hand) is largely an issue not of when a muscle is active but of when the preponderance of muscle activity might be expected with shifting tasks. The role of the extrinsic and intrinsic thumb muscles will be presented as generalizations (conceptual frameworks), as will the final section of this chapter, on hand prehension.

Extrinsic Thumb Muscles

There are four extrinsic thumb muscles: the flexor pollicis longus (FPL), extensor pollicis brevis (EPB), extensor pollicis longus (EPL), and abductor pollicis longus (APL) muscles. The flexor pollicis longus muscle is located volarly (see Fig. 9–12A). The flexor pollicis longus muscle inserts on the distal phalanx and is the correlate of the flexor digitorum profundus muscles of the fingers. The flexor pollicis longus tendon at the wrist is invested by the radial bursa, which is continuous with its digital tendon sheath (see Fig. 9–28A). The flexor pollicis longus muscle is unique in that it functions independently of other muscles and is the only muscle responsible for flexion of the thumb interphalangeal joint.[6] The flexor pollicis longus tendon sits between the sesamoid bones and appears to derive some protection from those bones.

Three of the thumb extrinsic muscles are located dorsoradially. The extensor pollicis brevis and abductor pollicis longus muscles run a common course from the dorsal forearm, traversing through the first dorsal compartment and crossing the wrist on its radial aspect (see Fig. 9–29) to their insertion. The abductor pollicis longus muscle inserts on the base of the metacarpal joint, whereas the extensor pollicis brevis muscle inserts on the base of the proximal phalanx. Both muscles abduct the carpometacarpal joint. The extensor pollicis brevis muscle also extends the metacarpophalangeal joint. The abductor pollicis longus and extensor pollicis brevis muscles also radially deviate the wrist slightly.

The extensor pollicis longus muscle originates in the forearm by the abductor pollicis longus and extensor pollicis brevis muscle but crosses the wrist closer to the dorsal midline before using the **dorsal radial (Lister's) tubercle** as an anatomical pulley to turn toward the thumb; the extensor pollicis longus muscle inserts on the base of the distal phalanx. At the level of the proximal phalanx, the extensor pollicis longus tendon is joined by expansions from the **abductor pollicis brevis (APB)** muscle, the first volar interossei muscle, and the **adductor pollicis (AdP)** muscle.[21] There is no further elaboration of the extensor expansion at the metacarpophalangeal joint of the thumb (compared to what we see in the fingers), but we see the same balance of metacarpophalangeal joint abductors and adductors contributing to resting tension in the metacarpophalangeal joint and to stabilization of the long extensor tendon. The more volarly located abductor pollicis brevis and adductor pollicis muscles that attach to the extensor pollicis longus tendon can extend the thumb interphalangeal joint to neutral but cannot complete the range into hyperextension in individuals who have that range available. The extensor pollicis longus is the only muscle that can complete the full range of hyperextension at the interphalangeal joint, as well as applying an extensor force at the metacarpophalangeal joint along with the extensor pollicis brevis muscle.[89] The extensor pollicis longus muscle can also extend and adduct the carpometacarpal joint of the thumb. Maximal glide of the extensor pollicis longus tendon is achieved with the wrist in an extended position.[177] In contrast to the fingers, there is a separate extensor tendon for each joint of the thumb. The abductor pollicis longus muscle attaches to the base of the metacarpal, the extensor pollicis brevis muscle to the base of the proximal phalanx, and the extensor pollicis longus muscle to the base of the distal phalanx.

Because the extrinsic thumb muscles span multiple joints, they must coordinate together to influence the positioning and functioning of the thumb as a whole.[178] As is true for other extrinsic hand muscles, wrist positioning is an essential factor in providing an optimal length-tension relationship for the extrinsic muscles of the thumb.[4] The flexor pollicis longus muscle is less effective as an interphalangeal flexor in wrist flexion. The extensor pollicis longus muscle cannot complete interphalangeal extension when the wrist, carpometacarpal, and metacarpophalangeal joints are simultaneously extended. The abductor pollicis longus and extensor pollicis brevis muscles require the synergy of an ulnar deviator of the wrist to prevent the muscles from creating wrist radial deviation, which thus affects their ability to generate tension over the joints of the thumb.

Intrinsic Thumb Muscles

There are five **thenar** (intrinsic thumb) muscles that originate primarily from the carpal bones and the flexor retinaculum (or transverse carpal ligament). The **opponens pollicis (OP)** is the only intrinsic thumb muscle to have its distal attachment on the first metacarpal. Its action line is nearly perpendicular to the long axis of the metacarpal joint and is applied to the lateral side of the bone. The opponens pollicis muscle, therefore, is very effective in positioning the metacarpal in an abducted, flexed, and rotated posture. The abductor pollicis brevis, flexor pollicis brevis, adductor pollicis, and first volar interossei muscles all insert on the proximal phalanx. The flexor pollicis brevis muscle has two heads of insertion. Its larger lateral head attaches distally with the abductor pollicis brevis muscle and also applies some abductor force. The flexor pollicis brevis muscle crosses the sesamoid bones at the metacarpophalangeal joint, increasing the moment arm of the flexor pollicis brevis muscle for metacarpophalangeal joint flexion. The medial head of the flexor pollicis brevis muscle attaches distally with the adductor pollicis muscle and assists in thumb adduction. The first volar interossei muscle arises from the first metacarpal and attaches to the ulnar sesamoid bone and then attaches distally to the proximal phalanx.

Although not generally considered a thenar muscle, the first dorsal interossei muscle may make a contribution to thumb function, along with its contribution to metacarpophalangeal flexion and interphalangeal extension of the index finger. The first dorsal interossei muscle is a bipennate muscle arising from both the first and second metacarpals and from the intercarpal ligament that joins the metacarpal bases. Brand and Hollister[158] proposed that the first dorsal interossei muscle is a carpometacarpal joint distractor, rather than, as is typically found, a joint compressor, because it pulls the first metacarpal distally toward the first dorsal interossei muscle's insertion on the base of the index proximal phalanx (Fig. 9–41). These investigators also argued that the thumb attachment of the first dorsal interossei muscle has little or no ability to move the thumb but it is important in offsetting the compressive and dorsoradially directed forces that the flexor/adductor muscles create across the carpometacarpal joint in lateral pinch and power grip. When

Figure 9–41 Brand and Hollister[149] proposed that the first dorsal interossei muscle is a distractor of the first carpometacarpal joint, which helps offset the strong compressive forces that occur across the first carpometacarpal joint.

these forces were created in laboratory specimens without tension in the first dorsal interossei muscle, the carpometacarpal joint subluxed.[158] Belanger and Noel suggested that the first dorsal interossei muscle can assist with thumb adduction.[179]

The thenar muscles are active in most grasping activities, regardless of the precise position of the thumb as it participates. The opponens pollicis muscle works together most frequently with the abductor pollicis brevis and the flexor pollicis brevis muscles, although the intensity of the relation varies. When the thumb is *gently* brought into contact with any of the fingers, activity of the opponens pollicis muscle predominates in the thumb, and abductor pollicis brevis activity exceeds that of the flexor pollicis brevis muscle. When opposition to the index finger or middle finger is performed *firmly*, activity of the flexor pollicis brevis muscle exceeds that of the opponens pollicis muscle. With *firm* opposition to the ring and little fingers, however, the relation changes; opponens pollicis activity increases with firm opposition to the ring finger, equaling activity of the flexor pollicis brevis muscle with firm opposition to the little finger.[70] The change in balance of muscle activity with firm opposition and with increasingly ulnar opposition can be accounted for by the increased need for abduction and metacarpal rotation. Increased pressure in opposition additionally appears to bring in activity of the adductor pollicis muscle. The adductor pollicis muscle stabilizes the thumb against the opposed finger. In *firm* opposition to the index and middle fingers, adductor pollicis activity exceeds the very minimal activity of the abductor pollicis brevis muscle. With a more ulnarly located position, the increased need for abduction results in simultaneous activity of the abductor and adductor.[70] Activity of the extrinsic thumb musculature in grasp appears to be partially a function of helping to position the metacarpophalangeal and interphalangeal joints. The main function of the extrinsic muscles, however, is in returning the thumb to extension from its position in the palm. Although release of an object is essentially an extrinsic function, some opponens pollicis and abductor brevis activity have been identified.[70] This muscular activity would assist in maintaining the thumb in abduction and in maintaining the metacarpal rotation that facilitates the next move of the thumb back into opposition.

The joint structure and musculature of the wrist complex, the fingers, and the thumb have each been examined. Some instances of specific muscle activity have been presented to clarify the potential function of the muscle. A summary of wrist and hand function, however, can best be presented through the assessment of purposeful hand activity. Because the entire upper limb is geared toward execution of movement of the hand, it is appropriate to complete the description of the upper limb by looking at an overview of the wrist and hand in prehension activities.

PREHENSION

Prehension activities of the hand involve the grasping or taking hold of an object between any two surfaces in the hand; the thumb participates in most but not all prehension tasks. There are numerous ways that objects of varying sizes and shapes may be grasped, with strategies also varying among individuals. Consequently, the nomenclature related to these functional patterns also varies.[180] In spite of nomenclature differences, a broad classification system for grasp has evolved that will permit general observations about the coordinated muscular function necessary to produce or maintain common forms of grasp.

Prehension can be categorized as either **power grip** (full hand prehension) (Fig. 9–42A) or **precision handling** (finger-thumb prehension) (Fig. 9–42B).[181] Each of these

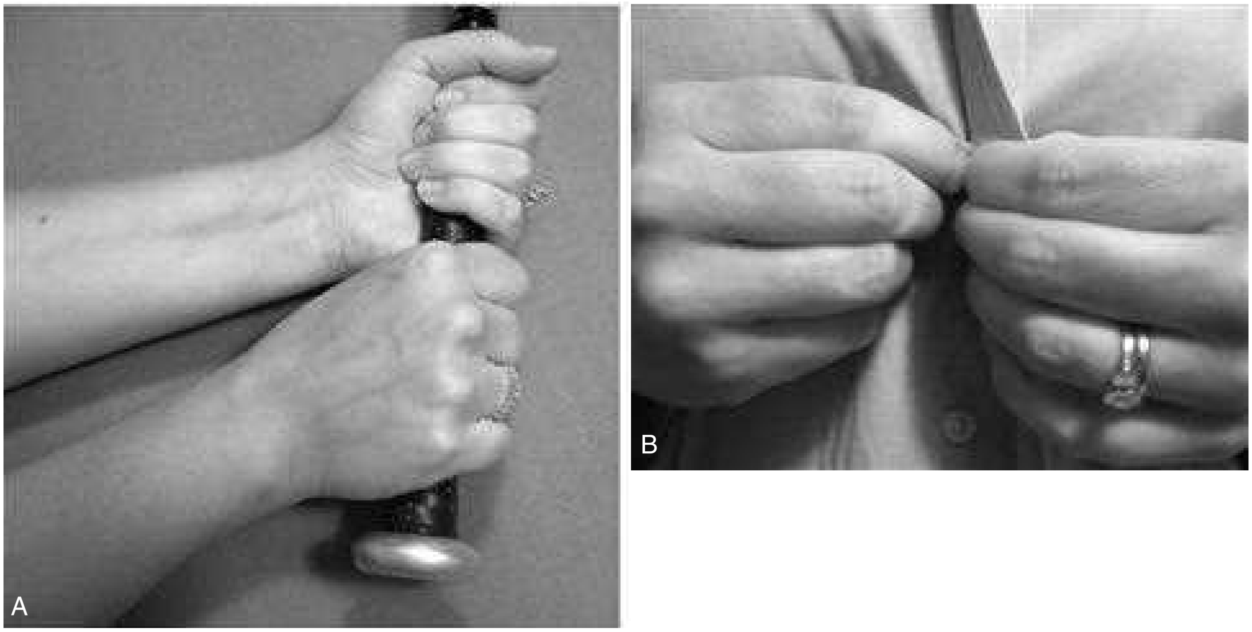

Figure 9–42 Prehension generally consists of either **(A)** power grip, in which the object makes full contact with the palm and is moved through space, or **(B)** precision handling, in which the thumb and fingers dynamically manipulate the object.

two categories has subgroups that further define the grasp. Power grip is generally a forceful act resulting in flexion at all finger joints. When the thumb is used, it acts as a stabilizer to the object held between the fingers and, most commonly, the palm. Precision handling, in contrast, is the skillful placement of an object between fingers or between finger and thumb.[182] The palm is not involved. Landsmeer suggested that power grip and precision handling can be differentiated on the basis of the dynamic and static phases.[183] Power grip is the result of a sequence of (1) opening the hand, (2) positioning the fingers, (3) bringing the fingers to the object, and (4) maintaining a static phase that actually constitutes the grip. This is in contrast to precision handling, which shares the first three steps of the sequence but does not contain a static phase at all. In power grip, the object is grasped so that the object can be moved through space by the more proximal joints; in precision handling, the fingers and thumb grasp the object for the purpose of manipulating it within the hand.

In assessment of the muscular function during each type of grasp, synergy of the hand muscles results in almost constant activity of all intrinsic and extrinsic muscles.[159,184,185] The task becomes more one of identifying when muscles are *not* working or when the balance of activity between muscles might change. It should also be emphasized that the muscular activity documented by EMG studies is specific to the activity as performed in a given study. Even in studies using similar forms of prehension, variables such as size of object, firmness of grip, timing, and instructions to the subject can cause substantial changes in reported muscle activity. However, as indications of general muscular activity patterns, the studies are useful in the development of a conceptual framework within which hand function can be understood.

Power Grip

The fingers in power grip usually function in concert to clamp on and hold an object into the palm. The fingers assume a position of sustained flexion that varies in degree with the size, shape, and weight of the object. The palm is likely to contour to the object as the palmar arches form around it. The thumb may serve as an additional surface to the finger-palm vise by adducting against the object, or it may be removed from the object. When the thumb is involved, it generally is adducted to clamp the object to the palm. This is in contrast to precision handling, in which the thumb is more likely to assume a position of abduction.[186] Four varieties of power grip studied by Long and associates[124] exemplify the similarities and differences seen in power grip. These are **cylindrical grip, spherical grip, hook grip,** and **lateral prehension.**

Cylindrical Grip

Cylindrical grip (Fig. 9–43A) almost exclusively involves use of the flexors to position the fingers around and maintain grasp on an object. The function in the fingers is performed largely by the flexor digitorum profundus muscle, especially in the dynamic closing action of the fingers. In the static phase, the flexor digitorum superficialis muscle assists when the intensity of the grip requires greater force. Although power grip traditionally has been thought of as an extrinsic muscle activity, studies have indicated considerable interosseous (intrinsic) muscle activity. The interossei muscles are considered to be functioning primarily as metacarpophalangeal joint flexors and abductors/adductors. In strong grip, the magnitude of torque production of the interossei muscles for metacarpal flexion was found to nearly equal that of the extrinsic flexors.[159,164,182] Because both the metacarpophalangeal and the interphalangeal joints are being flexed during cylindrical grip, the metacarpophalangeal joint flexion task most likely falls to the proximal (dorsal) interossei muscles because their attachments to the proximal phalanx and hood do not have a significant (and antagonistic) interphalangeal extension influence. The interossei muscles may also ulnarly deviate the metacarpophalangeal joint to direct the distal phalanges of the fingers toward the thumb. The combination of metacarpophalangeal joint flexion and ulnar deviation (adduction for the index finger and abduction for the middle, ring, and little fingers) (Fig. 9–43B) points the fingers toward the thumb but also tends to produce ulnar subluxation forces on the metacarpophalangeal joints and on the tendons of the long flexors at the metacarpophalangeal joint. The subluxing forces are ordinarily counteracted by the radial collateral ligaments, by the annular pulleys that anchor the flexor long tendons in place, and by the sagittal bands that connect the volar structures to the extensor mechanism. Active or passive tension in the extensor digitorum communis muscle can further stabilize the restraining mechanisms, as well as increase joint compression and enhance overall joint stability during power grip.[182] Although the location of the lumbrical muscles indicates a possible contribution to metacarpophalangeal joint flexion in power grip, their lack of EMG activity, regardless of strength grip, is consistent with their role as interphalangeal extensors.[124]

Thumb position in cylindrical grip is the most variable of the digits. The thumb usually comes around the object, then flexes and adducts to close the vise. The flexor pollicis longus and thenar muscles are all active. The activity of the thenar muscles will vary with the width of the web space, with the carpometacarpal rotation required, and with increased pressure or resistance. A distinguishing characteristic of power grip over precision handling is, in general, the greater magnitude of activity in the adductor pollicis muscle during power grip. The extensor pollicis longus muscle may be variably active as a metacarpophalangeal joint stabilizer or adductor.

Muscles of the **hypothenar eminence** usually are active in cylindrical grip. The abductor digiti minimi functions as a proximal interosseous muscle to flex and abduct (ulnarly deviate) the fifth metacarpophalangeal joint. The opponens digiti minimi and the **flexor digiti minimi (FDM)** muscles are more variable but frequently are active in direct proportion to the amount of abduction and rotation of the first metacarpal. In fact, increased activity of the opponens pollicis muscle automatically results in increased activity of the opponens digiti minimi and flexor digiti minimi muscles.

Cylindrical grip is typically performed with the wrist in neutral flexion/extension and slight ulnar deviation. Ulnar

Figure 9–43 Cylindrical grip may orient the finger tips toward the thumb **(A).** This is accomplished by ulnarly deviating the metacarpophalangeal joints using the interossei muscles **(B).**

deviation also puts the thumb in line with the long axis of the forearm (see Fig. 9–43A); this alignment better positions the object in the hand to be turned by pronation/supination of the forearm[186] as, for example, in turning a door knob. Ulnar deviation of the wrist is the position that optimizes force of the long finger flexors. The least flexion force is generated at these joints in wrist flexion.[2] The heavier an object is, the more likely it is that the wrist will ulnarly deviate. In addition, a strong contraction of the flexor carpi ulnaris muscle at the wrist will increase tension on the transverse carpal ligament. This provides a more stable base for the active hypothenar muscles that originate from that ligament. It is interesting to note that regardless of wrist position, the percentage of total interphalangeal flexor force allocated to each finger is relatively constant. The ring and little fingers can generate only 70% of the flexor force of the index and middle fingers.[2] The ring and little fingers seem to serve as weaker but more mobile assists to the more stable and stronger index and middle fingers. The contribution of the ring and little finger to grip can be improved if full flexion of the joints in those fingers (and concomitant loss of tension) is prevented by an object that is wider ulnarly than radially (the pistol-grip shape).

Spherical Grip

Spherical grip (Fig. 9–44A) is similar in most respects to cylindrical grip. The extrinsic finger and thumb flexors and the thenar muscles follow similar patterns of activity and variability. The main distinction can be made by the greater spread of the fingers to encompass the object. This evokes more interosseous activity than is seen in other forms of power grip.[124] The metacarpophalangeal joints do not deviate in the same direction (e.g., ulnarly) but tend to abduct. The phalanges are no longer parallel to each other, as they commonly are in cylindrical grip. The metacarpophalangeal joint abductors must be joined by the adductors to stabilize the joints that are in the loose-packed position of semiflexion. Although flexor activity predominates in the digits as it does in all forms of power grip, the extensors do have a role. The extensors not only provide a balancing force for the flexors but also are essential for smooth and controlled opening of the hand and release of the object. Opening the hand during object approach and object release is primarily an extensor function, calling in activity of the lumbrical, extensor digitorum communis, and thumb extrinsic muscles.

Hook Grip

Hook grip (Fig. 9–44B) is actually a specialized form of prehension. It is included in power grip because it has more characteristics of power grip than of precision handling. It is a function primarily of the fingers. It may include the palm but never includes the thumb. It can be sustained for prolonged periods of time, as anyone can attest who has carried a briefcase or books at his side or hung onto a commuter strap on a bus or train. The major muscular activity is provided by the flexor digitorum profundus and flexor digitorum superficialis muscles. The load may be sustained completely by one muscle or the other or by both muscles in concert. This depends on the position of the load in relation to the phalanges. If the load is carried more distally so that distal flexion is mandatory, the flexor digitorum profundus muscle must participate. If the load is carried more in the middle of the fingers, the flexor digitorum superficialis muscle may be sufficient. Some interosseous muscle activity has been demonstrated on EMG, but its purpose is not fully understood. It may help prevent clawing in the metacarpophalangeal joints, although the activity is not evident in every finger.[124] In hook grip, the thumb is held in moderate to full extension by thumb extrinsic muscles.

Lateral Prehension

Lateral prehension (Fig. 9–44C) is a unique form of grasp. Contact occurs between two adjacent fingers. The metacarpophalangeal and interphalangeal joints are usually maintained in extension as the contiguous metacarpophalangeal joints simultaneously abduct and adduct. This is the only form of prehension in which the extensor musculature predominates in the maintenance of the posture. The extensor digitorum communis and the lumbrical muscles are active to extend the metacarpophalangeal and interphalangeal joints, and metacarpophalangeal joint abduction and adduction are performed by the interossei muscles. Lateral prehension is included here as a form of power grip because lateral grip involves the static holding of an object that is then moved by the more proximal joints of the upper extremity. Although not a "powerful" grip, neither is lateral prehension used to manipulate objects in the hand. It is generally typified by the holding of a cigarette.

Precision Handling

The positions and muscular requirements of precision handling are somewhat more variable than those of power grip, require much finer motor control, and are more dependent on intact sensation. The thumb serves as one "jaw" of what has been termed a "two-jaw chuck"; the thumb is generally abducted and rotated from the palm. The second and opposing "jaw" is formed by the distal tip, the pad, or the side of a finger. When two fingers oppose the thumb, it is called a "three-jaw chuck." The three varieties of precision handling that exemplify this mode of prehension are **pad-to-pad prehension, tip-to-tip prehension,** and **pad-to-side prehension.** Each tends to be a dynamic function with relatively little static holding.

Pad-to-Pad Prehension

Pad-to-pad prehension involves opposition of the pad, or pulp, of the thumb to the pad, or pulp, of the finger (Fig. 9–45A). The pad of the distal phalanx of each digit has the greatest concentration of tactile corpuscles found

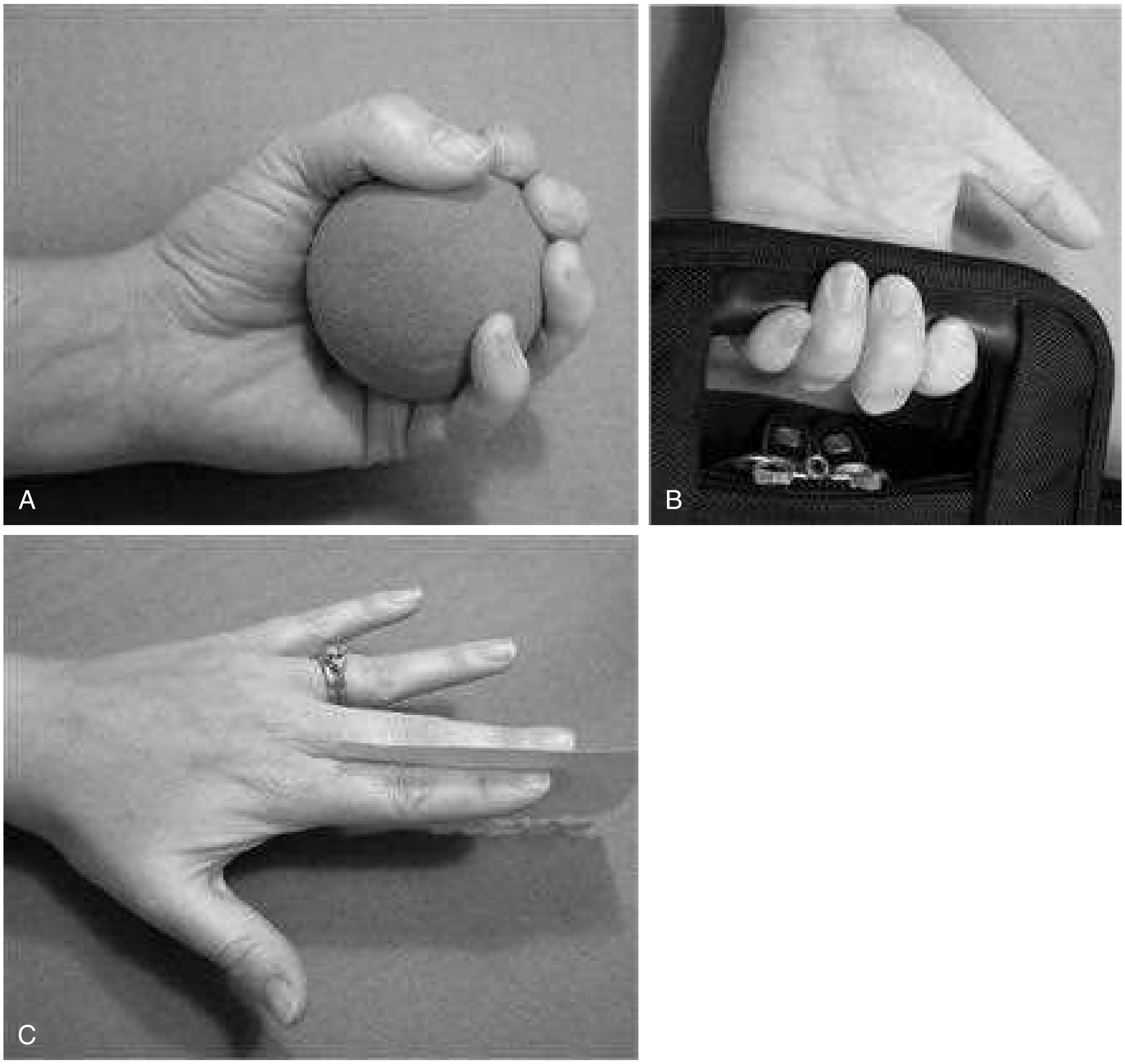

Figure 9–44 **A.** Spherical grip. **B.** Hook grip. **C.** Lateral prehension.

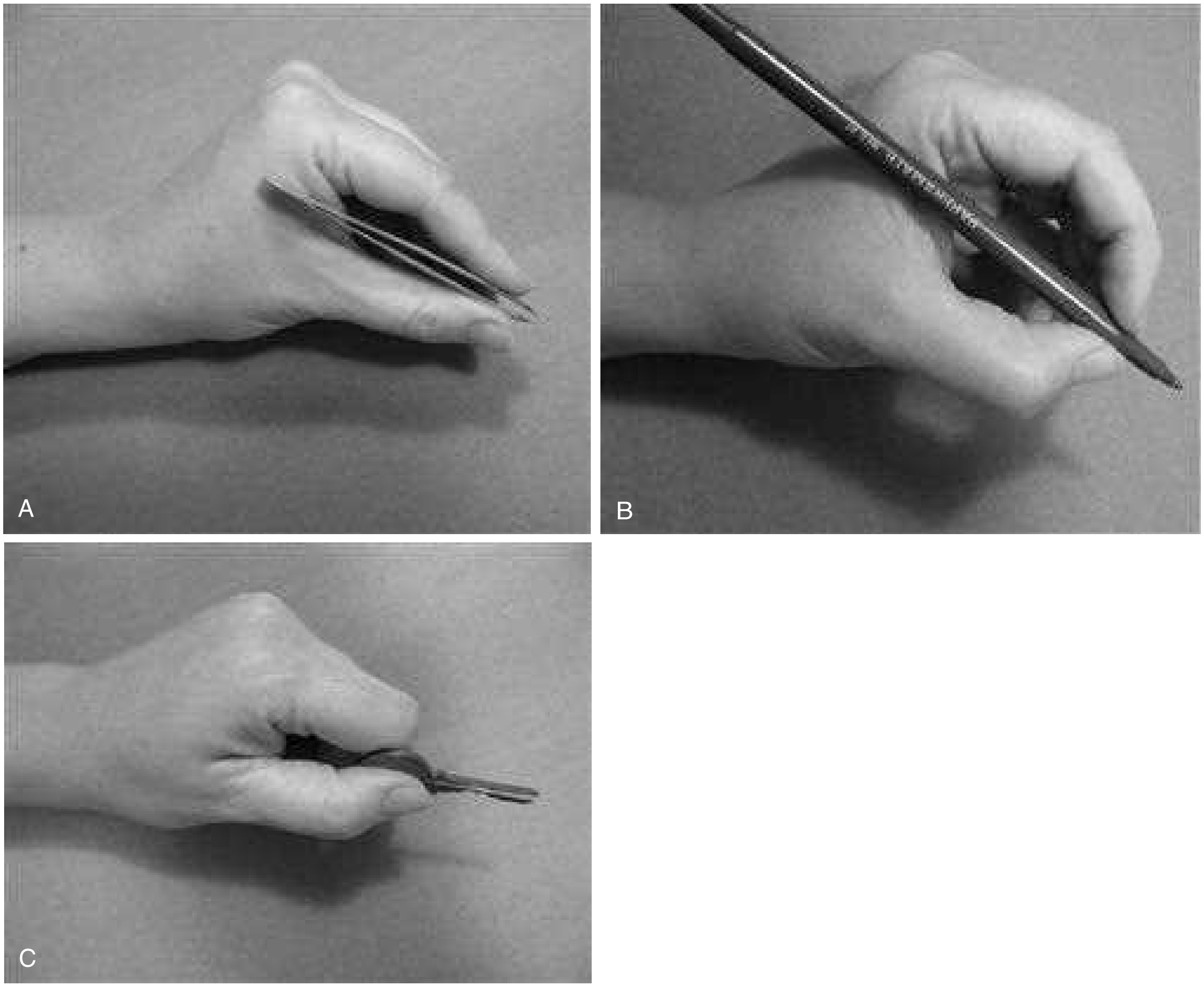

Figure 9–45 Three varieties of precision handling: **(A)** pad-to-pad prehension, **(B)** tip-to-tip prehension, and **(C)** pad-to-side prehension.

in the body. Of all forms of precision handling, 80% are considered to fall into the category of pad to pad.[1] The finger used in two-jaw chuck is usually the index; in three-jaw chuck, the middle finger is added. The metacarpophalangeal and proximal interphalangeal joints of the fingers are partially flexed, with the degree of flexion being dependent on the size of the object being held. The distal interphalangeal joint may be fully extended or in slight flexion. When the distal interphalangeal joint is extended, the flexor digitorum superficialis muscle can perform the function alone, without the assistance of the flexor digitorum profundus muscle. Extension of the distal interphalangeal joint in this instance is caused by flexion of the middle phalanx (flexor digitorum superficialis muscle) against the upward force of the object or thumb on the distal phalanx in what is effectively a closed chain. When partial flexion of the distal phalanx is required by the pad-to-pad task, the flexor digitorum profundus muscle must be active. Interosseous activity is often present both to supplement metacarpophalangeal joint flexor force and to provide the metacarpophalangeal joint abduction or adduction required in object manipulation. In dynamic manipulation, the volar interossei and dorsal interossei muscles tend to work reciprocally, rather than in the synergistic co-contraction pattern observed during power grip. In a static but firm pad-to-pad pinch, the interossei muscles may again co-contract.[124]

The thumb in pad-to-pad prehension is held in carpometacarpal flexion, abduction, and rotation (opposition). The first metacarpophalangeal and interphalangeal joints may be partially flexed or fully extended. The thenar muscle control is provided by the opponens pollicis, flexor pollicis brevis, and abductor pollicis brevis muscles, each of which is innervated by the median nerve. The adductor pollicis activity (ulnar nerve innervated) increases with increased pressure of pinch. In ulnar nerve paralysis, loss of adductor pollicis function (as well as loss of function of the first dorsal interossei and first volar interossei muscles) makes the thumb less stable and affects the precision of the grasp activity.

Fine adjustments in the flexion angle of the distal interphalangeal joint of the finger and the interphalangeal joint of the thumb control the points of contact on the pads of the digits. In full-finger distal interphalangeal and thumb interphalangeal joint extension, contact occurs on the more proximal portion of the distal phalanx (see Fig. 9–45A). As flexion of the finger distal interphalangeal and thumb interphalangeal joints increases, the contact moves distally toward the nails. Flexion of the distal phalanx, when required, is provided by the flexor digitorum profundus muscle for the finger and by the flexor pollicis longus muscle for the thumb. Distal interphalangeal flexion in the finger is accompanied by a proportional flexion in the proximal interphalangeal joint.

As is found in power grip, the extensor musculature is used for opening the hand to grasp, for release, and for stabilization when necessary. In the thumb, the extensor pollicis longus muscle may be used to maintain the interphalangeal joint in extension when contact is light and on the proximal pad. Synergistic wrist activity must also occur to balance the forces created by the flexor digitorum superficialis and flexor digitorum profundus muscles. The wrist is more typically held in neutral radial/ulnar deviation and slight extension.[184]

Tip-to-Tip Prehension

Although the muscular activity found in tip-to-tip prehension (see Fig. 9–45B) is nearly identical to that of pad-to-pad prehension,[124] there are some key differences. In tip-to-tip prehension, the interphalangeal joints of the finger and thumb must have the range and available muscle force to create nearly full joint flexion. The metacarpophalangeal joint of the opposing finger must also be ulnarly deviated (with fingertip pointed radially) to present the tip of the finger to the thumb. In the first finger, the ulnar deviation occurs as metacarpophalangeal joint adduction. In the remaining fingers, metacarpophalangeal abduction produces ulnar deviation. If the flexion range for the distal phalanx in either the opposing finger or the thumb is not available, or if the active force for interphalangeal flexion and metacarpophalangeal joint ulnar deviation cannot be provided, tip-to-tip prehension cannot be performed effectively. As the most precise form of grasp, it is also the most easily disturbed. Tip-to-tip prehension has all the same muscular requirements as pad-to-pad prehension in both fingers and thumb. In addition, however, activity of the flexor digitorum profundus, flexor pollicis longus, and interossei muscles is a necessity in tip-to-tip prehension, whereas they are not a necessity in pad-to-pad prehension.

Pad-to-Side Prehension

Pad-to-side prehension is also known as **key grip** (or **lateral pinch**) because a key is held between the pad of the thumb and side of the index finger (see Fig. 9–45C). Pad-to-side prehension differs from the other forms of precision handling only in that the thumb is more adducted and less rotated. The activity level of the flexor pollicis brevis muscle increases and that of the opponens pollicis muscle decreases, in comparison with tip-to-tip prehension. Activity of the adductor pollicis muscle also increases over that seen in either tip-to-tip or pad-to-pad prehension.[14] Slight flexion of the distal phalanx of the thumb is required. If the pad-to-side prehension is being used for something like turning a key, the wrist will again assume neutral flexion/extension and drop into slight ulnar deviation to put the key in line with the forearm so that pronation or supination can be used to turn the key.

Pad-to-side prehension is the least precise of the forms of precision handling; it can actually be performed by a person with paralysis of all hand muscles. If the hand muscles are paralyzed as they would be in a person with a spinal cord injury above the C7 level, active wrist extensors (assuming they are present) can create pad-to-side prehension. Wrist extension provided by the intact and active extensor carpi ulnaris, extensor carpi radialis longus, and extensor carpi radialis brevis muscles create the force needed to flex the metacarpophalangeal and interphalangeal joints of the fingers and thumb by generating passive tension in the extrinsic finger flexor tendons (flexor digitorum superficialis and flexor digitorum profundus) as the tendons are stretched over the extending wrist. The grip may be released by relaxing the wrist extensor muscles and allowing gravity to flex the wrist. As the wrist flexes, the tendons of the flexor digitorum superficialis and flexor digitorum profundus muscles become slack, and the tendons of the extensor digitorum communis (with the related extensor indicis proprius and extensor digiti minimi tendons) and extensor pollicis longus tendons become stretched. The passive tension in the long finger extensors in a dropped (flexed) wrist is adequate for partially extending both metacarpophalangeal and interphalangeal joints. The phenomenon of using active wrist extension to passively close the fingers and passive wrist flexion to passively open the fingers is known as **tenodesis.** The same tenodesis action can achieve a cylindrical grip if the proper balance of tension exists in the extrinsic flexors. The flexors must be loose enough to permit the partially flexed fingers of the "open" hand to surround the object in wrist flexion while still being tight enough to hold onto the object when the wrist is extended. Active control of at least one wrist extensor muscle is the minimal requirement for functional use of tenodesis in a person without any active control of finger or thumb musculature. Tenodesis was described in Chapter 3 when we first discussed passive insufficiency. As was noted then, tenodesis can and does also occur in the fully intact hand, although the presence of balancing muscles permits us to override it.

FUNCTIONAL POSITION OF THE WRIST AND HAND

Although it is difficult to isolate any one joint or function as being singularly important among all those examined, grasp would have to take precedence. There can be little doubt that the hand cannot function either as a manipulator or as a sensory organ unless an object can enter the palmar surface and unless moderate finger flexion and thumb opposition are available to allow sustained contact. Application of either an active muscular or passive tendinous flexor force to the digits requires the wrist to be stabilized in moderate extension and ulnar deviation. Delineation of the so-called functional position of the wrist and hand takes into account these needs and is the position from which optimal function is most likely to occur. It is *not necessarily* the position in which a hand should be immobilized. Position for immobilization depends on the disability.

The functional position is (1) wrist complex in slight extension (20°) and slight ulnar deviation (10°) and (2) fingers moderately flexed at the metacarpophalangeal joints (45°) and proximal interphalangeal joints (30°) and slightly flexed at the distal interphalangeal joints (Fig. 9–46).[1] The wrist position optimizes the power of the finger flexors so that hand closure can be accomplished with the least possible effort. It is also the position in which all wrist muscles are under equal tension. With similar considerations for the position of the joints of the digits, the functional position provides the best opportunity for the disabled hand to interact with the brain that controls it.

Figure 9–46 Functional position of the hand: wrist extension and ulnar deviation with moderate flexion of the metacarpophalangeal and interphalangeal joints of the finger and thumb.

SUMMARY

Despite the many articulations that make up the hand and wrist complex, the bony and ligamentous components of these joints have less potential for problems than the musculotendinous structures that cross and act on other joints. The motor control of and sensory feedback from the wrist and hand alone occupy more space topographically on the primary motor and sensory cortices of the brain than does the entire lower extremity. As we proceed to examine the joints of the lower extremity, an analogy to the corresponding joints of the upper extremity can and should be made. However, the primary weight-bearing function of the lower extremities does not require the complexity and delicate balance of muscular control that can so profoundly affect functional performance in the hand.

Acknowledgment

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STUDY QUESTIONS

1. Name the bones of the wrist complex; describe the articulations that occur between these bones and the functional joints that are formed.
2. Describe the components and role of the triangular fibrocartilage complex in wrist function.
3. What is the total ROM normally available at the wrist complex? How are the motions distributed between the radiocarpal and midcarpal joints of the complex?
4. Describe the sequence of carpal motion occurring from full wrist flexion to full extension and radial to ulnar deviation, emphasizing the role of the scaphoid.
5. What effect does release of the scapholunate stabilizers or of the lunotriquetral stabilizers have on bony positions?
6. Identify the muscles that can extend the wrist; include the joints crossed, actions produced, and activity levels of each.
7. Describe the transverse carpal ligament, its attachments, and its role in wrist and hand function.
8. What is the function of the carpometacarpal joints of the fingers? How do the variations in ROM among the four carpometacarpal joints of the fingers contribute to function?
9. What role does the transverse metacarpal ligament play at the carpometacarpal joint? What role at the metacarpophalangeal joint?
10. Describe the locations and functions of the volar (palmar) fibrocartilage plates.
11. What metacarpophalangeal joint position is most prone to injury and why?
12. Compare the joint structure of the metacarpophalangeal joints with that of the interphalangeal joints of the fingers. Identify both similarities and differences.
13. Describe the mechanisms, joint motions, and muscles that are necessary for the fingers to gently close into the palm without friction or loss of the length-tension relationship.
14. How does the “pistol-grip” design of most tools (larger ulnarly) relate to the metacarpophalangeal joint ROM and muscular function of the four fingers?

STUDY QUESTIONS—cont'd

15. When is the flexor digitorum superficialis muscle active as the primary finger flexor? When does it back up the flexor digitorum profundus muscle?
16. What muscles are active in gentle closure of the normal hand? What role, if any, do the intrinsic muscles play in this activity?
17. What wrist position is assumed when a person needs to optimize finger flexion strength? Which wrist position is least effective for grasp?
18. What are annular pulleys and cruciate ligaments in the digits? Where are they found, and what functions do they serve?
19. Identify the bursae of the hand. What are their functions and how are they most typically related to the digital tendon sheaths?
20. Describe the active and passive elements that make up the extensor mechanism.
21. What role do the extensor digitorum communis, extensor indicis proprius, and extensor digiti minimi muscles play in active extension of the proximal interphalangeal and distal interphalangeal joints of the hand?
22. How do the proximal and distal attachments of the interossei muscles affect function at the metacarpophalangeal and interphalangeal joints?
23. Describe the attachments of the lumbrical muscles to the extensor mechanism. How do these muscles contribute to interphalangeal extension? What is their role at the metacarpophalangeal joint?
24. Why is active distal interphalangeal joint flexion normally accompanied by proximal interphalangeal joint flexion at the same time?
25. Explain why the distal interphalangeal joint cannot be actively extended if the proximal interphalangeal joint is fully flexed.
26. Why will an isolated contraction of the extensor digitorum communis muscle produce flexion of the proximal interphalangeal and distal interphalangeal joints? What is this finger position called?
27. How are the extrinsic flexors and extensors stabilized at the metacarpophalangeal joints?
28. Why is finger extension weaker in the index and little fingers?
29. Why does metacarpophalangeal joint adduction weaken more quickly than abduction in a progressive ulnar nerve problem?
30. Which are stronger flexors of the metacarpophalangeal joint, the lumbrical or the interossei muscles?
31. Compare and contrast the metacarpophalangeal joint structure of the thumb with the metacarpophalangeal joint structure of the fingers.
32. What does the motion of thumb opposition require in terms of joint function and musculature?
33. What are the primary muscles of release in the wrist and hand?
34. In general, what is the difference between power grip and precision handling at the wrist, in the fingers, and in the thumb? What do these two forms of prehension have in common?
35. Cylindrical grip is generally referred to as an extrinsic hand function. Why is this true?
36. What requirement does spherical grip have that differentiates it from cylindrical grip?
37. Which form of prehension requires only intrinsic musculature?
38. Which forms of prehension do not require the thumb?
39. What roles do interossei muscles play in precision handling?
40. What requirements does tip-to-tip prehension have that are not necessary for pad-to-pad prehension?
41. What is the finest (most precise) form of prehension that can be accomplished by someone without intact hand musculature, assuming availability of an active wrist extensor?
42. What is the functional position of the wrist and hand? Why is this the optimal resting position when there is no specific hand problem?
43. Why is an ulnar nerve injury called "claw hand"? What deficiency causes the clawing, and in which fingers does it occur?

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Section 4

Hip Joint

Chapter 10 **The Hip Complex**
Chapter 11 **The Knee**
Chapter 12 **The Ankle and Foot Complex**

Chapter 10

The Hip Complex

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Introduction

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INTRODUCTION

The hip joint, or **coxofemoral joint,** is the articulation of the acetabulum of the pelvis and the head of the femur (Fig. 10–1). These two segments form a diarthrodial ball-and-socket joint with three degrees of freedom: flexion/extension in the sagittal plane, abduction/adduction in the frontal plane, and medial/lateral rotation in the transverse plane. Although the hip joint and the shoulder complex have a number of common features, the functional and structural adaptations of each to its respective roles have been so extensive that such comparisons are more of general interest than of functional relevance. The role of the shoulder complex is to provide a stable base on which a wide range of mobility for the hand can be superimposed. Shoulder complex structure gives precedence to open-chain function. The primary function of the hip joint is to support the weight of the head, arms, and trunk **(HAT)** both in static erect posture and in dynamic postures such as ambulation, running, and stair climbing. The hip joint, like the other joints of the lower extremity that we will examine, is structured primarily to serve its weight-bearing functions. Although we examine hip joint structure and function as if the joint were designed to move the foot through space in an open chain, hip joint structure is more influenced by the demands placed on the joint when the limb is bearing weight. As we shall see later in this chapter, weight-bearing function of the hip joint and its related weight-bearing responses are basic to understanding the hip joint and the interactions that occur between the hip joint and the other joints of the spine and lower extremities.

10-1 **Patient Case** *case*

Gabriella Martinez is a 32-year-old woman currently teaching kindergarten, a position she has held for the past 10 years. Gabriella has been bothered with recurrent left hip pain that has been intermittent throughout her young adult life. She recalls several instances of hip pain that interrupted her participation in gymnastics and dance as a teenager. Although Gabriella no longer participates in dance or gymnastics, she remained physically active until recently by running as well as taking Pilates and yoga classes.

Over the past few months, though, she has experienced increasing problems, predominantly on her left side, that interfere with climbing the stairs in her two-story home, performing activities with her kindergarten class, and caring for her 3-year-old daughter. In addition to pain complaints, she also reports feeling as though her hip may “pop out of the socket” when she is doing twisting poses in yoga and Pilates and has an occasional painful “click” in the hip. Therefore, it has become too painful for her to run and she has suspended participation in her exercise class. She remembers being told by a physician when she was in her teens that she would probably have problems with her hips when she got older because they were “shallow.”

Gabriella localizes her primary pain to her left groin, although the lateral hip area can also be painful. On clinical examination, Gabriella is observed to walk with reduced hip extension on the left. Pain occurs during midstance to late stance phase and swing phase on the left. She also has a slight left lateral lean during left stance. During examination, the physician finds that Gabriella has passive hip flexion with medial rotation painful on the left. In the supine position, there is an increase in medial rotation and decrease in lateral rotation at the left hip.

STRUCTURE OF THE HIP JOINT

Proximal Articular Surface

The cuplike concave socket of the hip joint is called the **acetabulum** and is located on the lateral aspect of the pelvic bone (**innominate** or **os coxa**). Three bones form the pelvis: the ilium, the ischium, and the pubis. Each of the three bones contributes to the structure of the acetabulum (Fig. 10–2A). The pubis forms one fifth of the acetabulum, the ischium forms two fifths, and the ilium forms the remainder. Until full ossification of the pelvis occurs between 20 and 25 years of age, the separate segments of the acetabulum may remain visible on radiograph[1] (Fig. 10–2B).

The periphery of the acetabulum **(lunate surface)** is covered with hyaline cartilage. This horseshoe-shaped area of cartilage articulates with the head of the femur (see Fig. 10–2A) and allows for contact stress to be uniformly distributed.[2] The inferior aspect of the lunate surface (the base of the horseshoe) is interrupted by a deep notch called the **acetabular notch.** The acetabular notch is spanned by a fibrous band, the **transverse acetabular ligament,** that connects the two ends of the horseshoe. As the transverse acetabular ligament spans the acetabular notch, it creates a fibro-osseous tunnel through which blood vessels pass into the central or deepest portion

Figure 10–1 The hip joint is formed by the head of the femur and the acetabulum of the innominate bone (one half) of the pelvis.

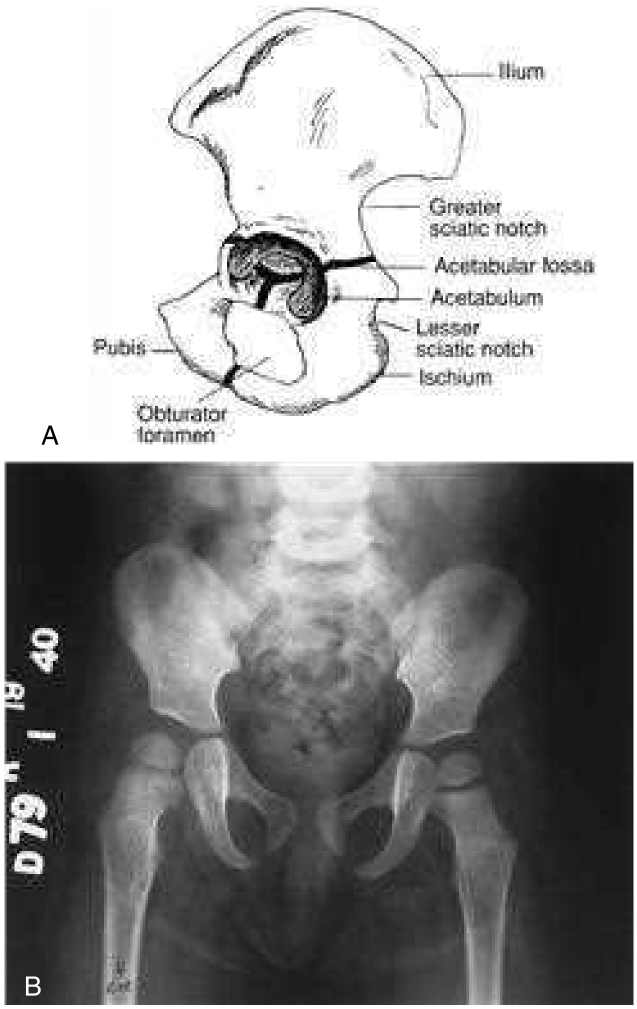

Figure 10–2 **A.** The acetabulum is formed by the union of the three bones of the pelvis, with only the upper horseshoe-shaped area being articular. **B.** In this radiograph of a 2-year-old without impairments, the cartilaginous rather than bony union of the acetabulum is clearly evident.

of the acetabulum, called the **acetabular fossa.** The acetabulum is deepened by the fibrocartilaginous **acetabular labrum,** which surrounds the periphery of the acetabulum. The acetabular fossa is nonarticular; the femoral head does not contact this surface (Fig. 10–3). The acetabular fossa contains fibroelastic fat covered with synovial membrane.

Acetabulum

Although appearing to be spherical, only the upper margin of the acetabulum has a true circular contour.[3] The acetabulum is positioned laterally with an inferior and anterior tilt. The opening of the acetabulum is approximately laterally inclined 50°; anteriorly rotated (anteversion) 20°; and anteriorly tilted 20° in the frontal, transverse, and sagittal planes, respectively.[4–7] The articular surface is smaller in women than it is in men.[6] Normal functioning of the hip requires optimal femoral head coverage by the acetabulum. Femoral head coverage is largely determined by acetabular depth. **Acetabular dysplasia, coxa profunda, acetabular protrusio, anteversion,** and **retroversion** are terms used to describe acetabular abnormalities that can potentially lead to pathology, including excessive cartilage wear and osteoarthritis.[8]

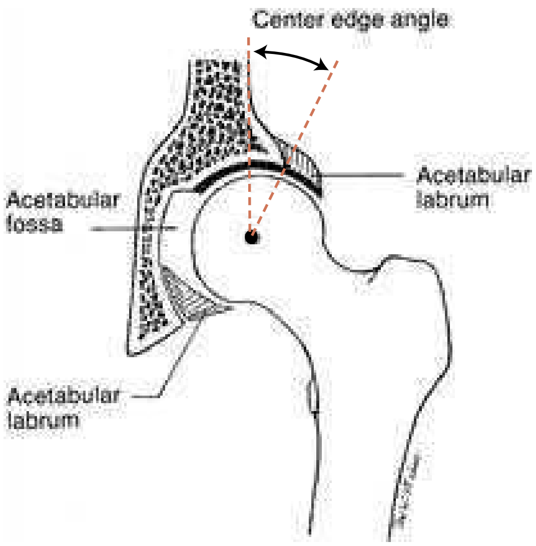

Figure 10–3 The center edge angle of the acetabulum is formed between a vertical line through the center of the femoral head and a line connecting the center of the femoral head and the bony edge of the acetabulum. The acetabular labrum deepens the acetabulum.

Acetabular dysplasia is an abnormally shallow acetabulum that results in a lack of femoral head coverage.[9] *Dysplasia* is the basic mechanical abnormality for instability and disproportionate loading of the superior acetabular rim. *Coxa profunda* and *acetabular protrusio* are terms used to describe conditions in which the acetabulum excessively covers the femoral head. Acetabular overcoverage can lead to limited range of motion (ROM) and internal impingement between the femoral head-neck junction and acetabulum.[9] Acetabular depth can be measured as the **center edge angle of Wiberg**[10] (see Fig. 10–3). The center edge angle is formed by a line connecting the lateral rim of the acetabulum and the center of the femoral head and a vertical line from the center of the femoral head. Center edge angles are classified as follows: definite dysplasia less than 16°, possible dysplasia 16° to 25°, and normal greater than 25°.[10] Center edge angles greater than normal, which would relate to abnormal overcoverage, have not been well defined. However, center edge angles greater than 40° may indicate excessive acetabular overcoverage.[9]

In addition, abnormalities in acetabular depth, inclination, and version (abnormal positioning in the transverse plane) can also affect femoral head coverage. Anteversion of the acetabulum exists when the acetabulum is positioned too far anteriorly in the transverse plane; retroversion exists when the acetabulum is positioned too far posteriorly in the transverse plane. An acetabulum that is positioned with less inclination and/or more anteversion can lead to instability. Likewise, an acetabulum that is positioned with more inclination and/or retroversion can lead to overcoverage and impingement between the acetabulum femoral-head neck junction.[9]

Acetabular Labrum

The entire periphery of the acetabulum is rimmed by a ring of wedge-shaped fibrocartilage called the acetabular labrum (see labrum cross-section in Fig. 10–3). The labrum of the hip, to a large extent, is analogous to the meniscus of the knee and the labrum of the glenohumeral joint. Given the need for stability at the hip joint, it is not surprising to find an accessory joint structure. The labrum is attached to the periphery of the acetabulum by a zone of calcified cartilage with a well-defined tidemark.[11] The acetabular labrum not only deepens the socket but also increases the concavity of the acetabulum through its triangular shape, grasping the head of the femur to maintain contact with the acetabulum. It enhances joint stability not only by deepening the acetabulum but also by acting as a seal to maintain negative intra-articular pressure.[12] The labrum also decreases force transmitted to the articular cartilage and provides proprioceptive feedback.[12–15] Nerve endings within the labrum not only provide proprioceptive feedback but can also be a source of pain.[14] Although the normal hip is intrinsically stable because of the deeply recessed acetabulum, an abnormally shallow acetabulum will increase stress on the surrounding capsule and labrum.

The transverse acetabular ligament is considered to be part of the acetabular labrum although, unlike the labrum, it contains no cartilage cells.[16] Although it is positioned to protect the blood vessels traveling beneath it to reach the head of the femur, experimental data do not support the notion of the transverse acetabular ligament as a load-bearing structure.[17] Konrath and colleagues[17] supported the hypothesis of others that the ligament served as a tension band between the anteroinferior and posteroinferior aspects of the acetabulum (the "feet" of the horseshoe-shaped articular surface) but were not able to corroborate this from their data.

CASE APPLICATION

Labral Tear

case 10–1

Gabriella's left groin pain is provoked by passive hip flexion and medial rotation.[18] This clinical finding may be indicative of damage to the anteriosuperior labrum and possibly a site of osteoarthritic changes.[19,20] Due to her age, history of hip pain, and clinical findings her physician ordered a magnetic resonance arthrogram (MRA) to identity the pathoanatomical source of her pain. After reviewing her MRA, Gabriella's physician confirmed a superior anterior labral tear and early stage osteoarthritis. Acetabular labral tears are increasingly recognized as a source of hip pain and as a starting point for degenerative changes at the acetabular rim.[11,19,21–24] Damage to the labrum was evident in 96% of postmortem and cadaveric hip joints in persons 61 to 98 years of age, with 74% showing damage in the anteriosuperior quadrant.[11] Isolated labral tears are uncommon and usually occur in conjunction with bony abnormalities of the acetabulum and/or femoral-neck junction.[25] Although a labral tear is not always symptomatic,[26] a torn labrum can potentially cause anterior groin pain, clicking, locking, catching, instability, giving way, and/or stiffness.[18,27]

Distal Articular Surface

The head of the femur is a fairly rounded hyaline cartilage-covered surface. The articular area of the femoral head forms approximately two thirds of a sphere and is more circular than the acetabulum.[3,28] Femoral shape and dimensions were found to be variable even in bones of the same overall size.[28] The radius of curvature of the femoral head is smaller in women than in men in comparison with the dimensions of the pelvis.[28,29] Just inferior to the most medial point on the femoral head is a small, roughened pit called the **fovea** or **fovea capitis** (Fig. 10–4). The fovea is not covered with articular cartilage and is the point at which the **ligament of the head** of the femur **(ligamentum teres)** is attached.

The femoral head is attached to the femoral neck; the femoral neck is attached to the shaft of the femur between the greater and lesser trochanters. The femoral neck is, in general, only about 5 cm long.[16] The femoral neck is angulated so that the femoral head faces medially, superiorly, and anteriorly with respect to the femoral shaft and distal femoral condyles.[30]

Angulation of the Femur

There are two angulations made by the head and neck of the femur in relation to the shaft. One angulation **(angle of inclination)** occurs in the frontal plane between an axis through the femoral head and neck and the longitudinal axis of the femoral shaft. The other angulation **(angle of torsion)** occurs in the transverse plane between an axis through the femoral head and neck and an axis through the distal femoral condyles. The origin and variability of these angulations can be understood in the context of the embryonic development of the lower limb. In the early stages of fetal development, both upper extremity and lower extremity limb buds project laterally from the body as if in full abduction. During the seventh and eighth weeks of gestational age and before full definition of the

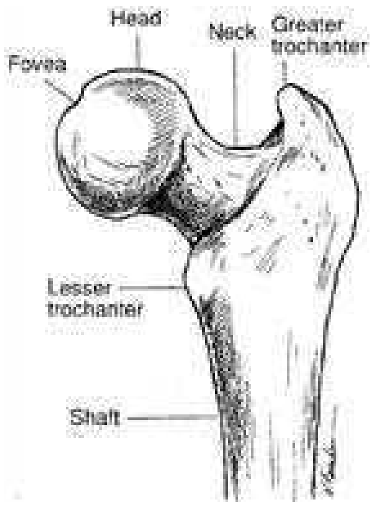

Figure 10–4 Posterior view of the proximal portion of the right femur shows the relationship between the head, neck, trochanters, and femoral shaft.

joints, adduction of the buds begins. At the end of the eighth week, the "fetal position" has been achieved, but the upper and lower limbs are no longer positioned similarly. Although the upper limb buds have undergone torsion somewhat laterally (so that the ventral surface of the limb bud faces anteriorly), the lower limb buds have undergone torsion medially, so that the ventral surface faces posteriorly.[16] The result for the lower limb is critical to understanding function. The knee flexes in the opposite direction from the elbow, and the extensor (dorsal) surface of the lower limb is located anteriorly rather than posteriorly. Although the head and neck of the femur retain the original position of the limb bud, the femoral shaft is inclined medially and undergoes medial torsion with regard to the head and neck. The magnitude of medial inclination and torsion of the distal femur (with regard to the head and neck) is dependent on embryonic growth and, presumably, fetal positioning during the remaining months of uterine life. The development of the angulations of the femur appears to continue after birth and through the early years of development. It should be recognized that both normal and abnormal angles of inclination and torsion of the femur are *properties of the femur alone*. However, abnormalities in the angulations of the femur can cause compensatory changes that can substantially alter hip joint stability, weight-bearing biomechanics of the hip joint, and muscle biomechanics.

Angle of Inclination of the Femur

The angle of inclination of the femur approximates 125° (referencing the medial angle formed by the axes of the head/neck and the shaft).[31] This value can have a normal range from 110° to 144° in the unimpaired adult[30,32–34] (Fig. 10–5). As with the angle of inclination of the humerus, there are variations not only among individuals but also from side to side. In women, the angle of inclination is somewhat smaller than it is in men, owing to the greater width of the female pelvis.[16] With a normal angle of inclination, the greater trochanter lies at the level of the center of the femoral head.[35] The angle of inclination of the femur changes across the life span; it approximates 150° at birth (see Fig. 10–2B) and gradually declines to about 125° at skeletal maturity.[31] This angle can continue to decline again in the elderly.[34] A pathological increase in the medial angulation between the neck and shaft is called **coxa valga** (Fig. 10–6A), and a pathological decrease is called **coxa vara** (Fig. 10–6B).

Figure 10–5 The axis of the femoral head and neck forms an angle with the axis of the femoral shaft called the *angle of inclination*. In this adult subject without impairments, the angles are slightly less than 130°, with a couple of degrees of variation from side to side.

Both coxa vara and coxa valga can lead to abnormal lower extremity biomechanics altered muscle function, and gait abnormalities[36] that contribute to pathologies such as hip and knee osteoarthritis and slipped capital femoral epiphysis.

In coxa valga (see Fig. 10–6A), the angle of inclination in the femur is greater than the normal adult angle of 125°. The increased angle brings the vertical weight-bearing line closer to the shaft of the femur, diminishing the shear, or bending, force across the femoral neck. The reduction in force is actually reflected in a reduction in density of the lateral trabecular system.[45] However, the decreased distance between the femoral head and the greater trochanter also decreases the length of the moment arm of the hip abductor muscles. The decreased muscular moment arm results in an increased demand for muscular force generation to maintain sufficient abduction torque to counterbalance the gravitational adduction moment acting around the supporting hip joint during single-limb support. Either the additional muscular force requirement will increase the total joint reaction force within the hip joint or the abductor muscles will be unable to meet the increased demand and will be functionally weakened. Although the abductors may be otherwise normal, the reduction in biomechanical effectiveness may produce the compensations typical of primary abductor muscle weakness. Coxa valga also decreases the amount of femoral articular surface in contact with the dome of the acetabulum. As the femoral head points more superiorly, there is a decreasing amount of coverage from the acetabulum superiorly. Consequently, coxa valga decreases the stability of the hip and predisposes the hip to dislocation.[9,23,46] This may further lead to femoroacetabular impingement and the progression of labral tearing, loss of joint stability, and eventual arthrosis.[39]

Coxa vara is considered to give the advantage of improved hip joint stability (if angle reduction is not too extreme). The apparent improvement in congruence occurs because the decreased angle between the neck and shaft of the femur will turn the femoral head deeper into the acetabulum, decreasing the amount of articular surface exposed superiorly and increasing coverage from the acetabulum. A varus femur, if not caused by trauma, may also increase the length of the moment arm of the hip abductor muscles by increasing the distance between the femoral head and the greater trochanter.[22] The increased moment arm decreases the amount of force that must be generated by the abductor muscles in single-limb support and reduces the joint reaction force. However, coxa vara

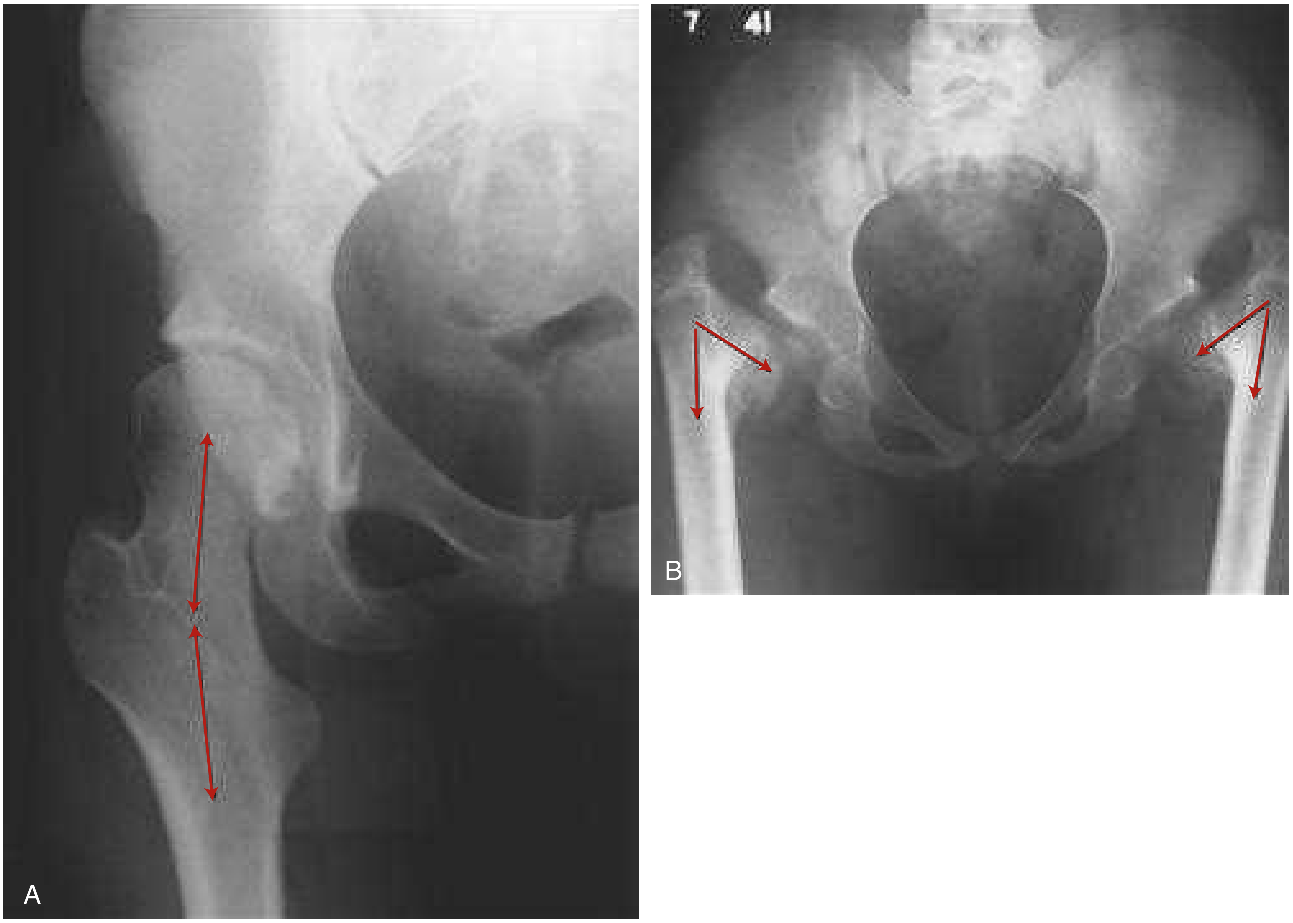

Figure 10–6 Abnormal angles of inclination found in two young adults with developmental hip dysplasia. **A.** A pathological increase in the angle of inclination is called *coxa valga.* **B.** A pathological decrease in the angle is called *coxa vara.*

has the disadvantage of increasing the bending moment along the femoral head and neck. This increase in bending force can actually be seen by the increased density of trabeculae laterally in the femur, caused by the increase in tensile stresses.[45] The increased shear force along the femoral neck will increase the predisposition toward femoral neck fracture.[23,46]

Coxa vara may increase the likelihood in the adolescent that the femoral head will slide on the cartilaginous epiphysis of the head of the femur. In childhood, the epiphysis is fairly horizontal.[7] Consequently, the superimposed weight merely compresses the head into the epiphyseal plate. In adolescence, growth of the bone results in a more oblique orientation of the epiphyseal plate. The epiphyseal obliquity makes the plate more vulnerable to shear forces at a time when the plate is already weakened by the rapid growth that occurs during this period of life.[91] Weight-bearing forces may slide the femoral head inferiorly, resulting in a **slipped capital femoral epiphysis.** Trigui and colleagues[40] reported the outcomes of surgical interventions to correct coxa vara deformities in children. Despite correction of the deformity, a common complication and resultant failure of the procedure was related to the unstable femoral epiphysis. Thus it was concluded that optimal results need to address the stability of the proximal femoral epiphysis and exemplifies the vulnerability of the growth plate of the femur in patients with a preexisting coxa vara deformity. As is true for a hip fracture, the altered biomechanics and at-risk blood supply necessitate restoration of normal alignment before secondary degenerative changes can occur.

Continuing Exploration 10-1:

Slipped Capital Femoral Epiphysis

Slipped capital femoral epiphysis (SCFE) is the most common adolescent hip disorder occurring when the femoral head displaces posteriorly on the femoral neck at the level of the growth plate (physis). A decrease in femoral neck-shaft angle (coxa vara)[41] and high body mass index increase the shear force across the physis.[42] Slipped capital femoral epiphysis can cause widening of the femoral head-neck junction and lead to internal impingement with the acetabulum.[42]

Angle of Torsion of the Femur

The angle of torsion of the femur can best be viewed by looking down the length of the femur from top to bottom. An axis through the femoral head and neck in the transverse plane will lie at an angle to an axis through the femoral condyles, with the head and neck torsioned anteriorly (laterally) with regard to an angle through the femoral condyles (Fig. 10–7). This angulation reflects the medial rotational migration of the lower limb bud that occurred during fetal development. The apparent contradiction between medial torsion of the embryonic limb bud and lateral torsion of the femoral head and neck simply reflects a shift in reference. In medial torsion of the limb bud, the proximal end is fixed and the distal end migrates medially. When torsion of the femur is assessed in a child or adult, the reference is an axis through the femoral condyles (the knee joint axis) that is generally presumed to lie in the frontal plane. If the axis through the femoral condyles lies in the frontal plane (as it functionally should), then the head and neck of the femur are torsioned anteriorly, relatively speaking, on the femoral condyles (Fig. 10–8A, B). The angle of anterior torsion decreases with age. In the newborn, femoral neck anteversion has been estimated to approximate 30° to 40° on average.[43] This angle decreases of approximately 1.5° per year until skeletal maturity and is usually symmetrical in the right and left sides.[43–45] In the adult, the normal angle of torsion is considered to be 10° to 20°, 15° for males and 18° for females.[43,46,47]

Femoral anteversion is considered to exist when angle of anterior torsion is greater than 15° to 20°. A reversal of anterior torsion, known as **femoral retroversion,** occurs when angles are less than 15° to 20° (Fig. 10–8C). There may not be one angulation at which pathological femoral torsion may be diagnosed, given the substantial normal variability.[48] Variations in the degree of anteversion or retroversion may also be dependent on the method used to assess it, as computed tomography (CT scan), radiograph, and ultrasound have each been used to measure the angle of femoral torsion.[48] Clinical measures of anteversion have correlated with imaging measurements. Femoral anteversion is associated with increased medial rotation ROM and concurrent decreased lateral rotation so that the total excursion of hip rotation motion remains the same.[49]

Figure 10–7 A line parallel to the posterior femoral condyles and a line through the head and neck of the femur normally make an angle with each other that averages 10° to 20° in the adult without impairments. The femoral head and neck are in torsion anteriorly (medially) with respect to the femoral condyles.

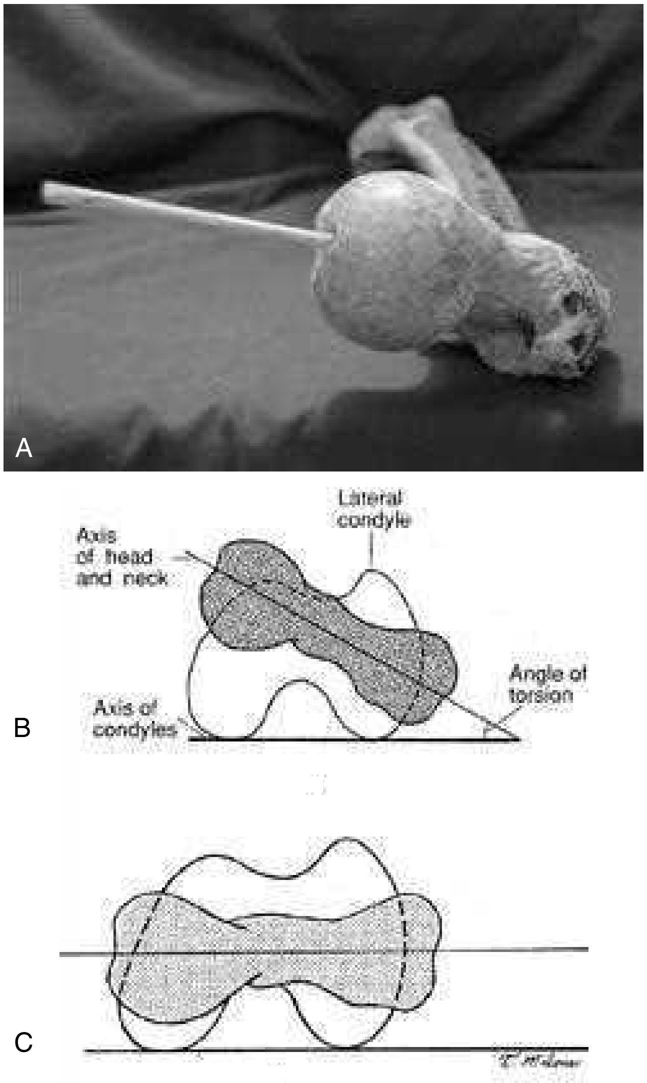

Figure 10–8 Angles of torsion in a right femur. **A.** An angle of torsion within normal limits. **B.** A pathological increase in the angle of torsion is called anteversion. **C.** A pathological decrease in the normal angle of torsion is called retroversion.

Although some structural deviations such as femoral anteversion and coxa valga are commonly found together, each may occur independently of the other. Each structural deviation warrants careful consideration as to the impact on hip joint function *and* function of the joints both proximal and distal to the hip joint. As shall be evident when the knee and foot are discussed in subsequent chapters, femoral anteversion is often implicated in dysfunction at both the knee and at the foot. The other pathological angulations of the femur (retroversion, coxa vara, and coxa valga) similarly affect the hip joint and other joints proximally and distally.

Variations in the angle of torsion also affect hip biomechanics and function. Anteversion of the femoral head reduces hip joint stability because the femoral articular surface is more exposed anteriorly (Fig. 10–9). The line of the hip abductors may fall more posterior to the joint, reducing the

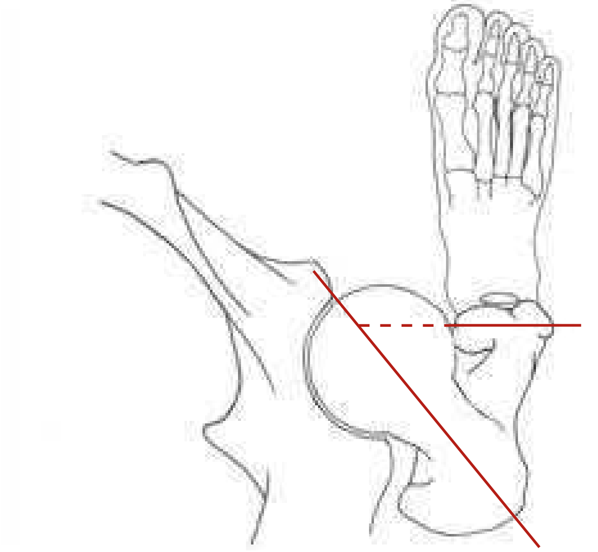

Figure 10–9 In the supine position with the femoral condyles parallel to the supporting surface, the anteverted femoral head is exposed anteriorly. Lateral rotation will be limited, but medial rotation is relatively excessive.

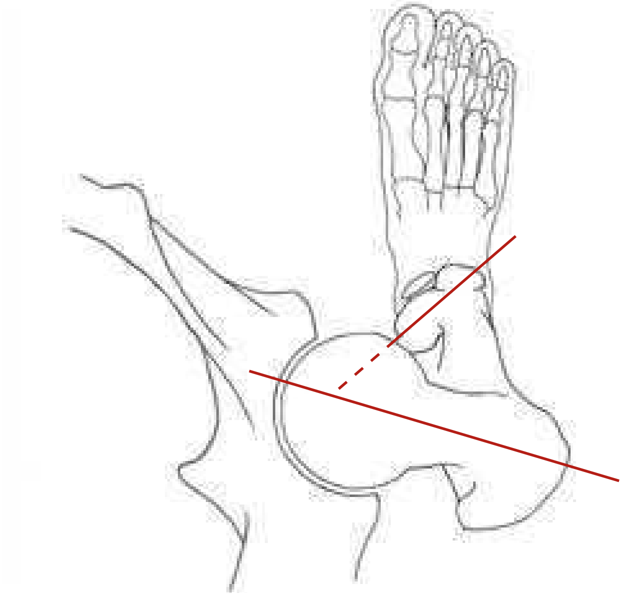

Figure 10–10 In standing, the anteverted femur tends to medially rotate within the acetabulum, resulting in medial rotation of the femoral condyles in relation to the plane of progression. The torsional deformity of the femur, when assessed in standing, is referred to as medial femoral torsion. If there is an accompanying lateral tibial torsion, the expected toe-in may be minimized or reversed.

moment arm for abduction.[50] As is true for coxa valga, the resulting need for additional abductor muscle force may predispose the joint to arthrosis or may functionally weaken the joint, producing energy-consuming and wearing gait deviations. The effect of femoral anteversion may also be seen at the knee joint. When the femoral head is anteverted, pressure from the anterior capsuloligamentous structures and the anterior musculature may push the femoral head back into the acetabulum, causing the entire femur to rotate medially (Fig. 10–10). Although the medial rotation of the femur improves the congruence in the acetabulum, the knee joint axis through the femoral condyles is now turned medially. Medial rotation of the femoral condyles alters the plane of knee flexion/extension and results, at least initially, in a toe-in gait. However, the toe-in position of the foot may appear to diminish over time because it is not uncommon to see a compensatory lateral tibial torsion develop (see Fig. 10–10). Although the foot placement looks better, the underlying hip problem generally remains (with some developmental reduction). The abnormal position of the knee joint axis is commonly labeled **medial femoral torsion.** Medial femoral torsion and femoral anteversion are the same abnormal condition of the femur. The label designates whether the exaggerated twist in the femur is altering the mechanics at the hip joint (femoral anteversion) or at the knee joint (medial femoral torsion). As shall be seen in the next two chapters, an anteverted femur will also affect the biomechanics of the patellofemoral joint at the knee and of the subtalar joint in the foot.

Femoral retroversion is the opposite of anteversion and creates problems opposite those of femoral anteversion. Interestingly, in a study by Ito and colleagues[24] that examined proximal femoral characteristics in patients with and without symptomatic labral tears, the group with labral tears had significantly reduced femoral anterior torsion compared to the healthy controls. This may explain the importance of femoral anterior torsion as a protective measure against impingement and subsequent labral pathology. Thus the relative femoral retroversion compared to the normal variation of femoral torsion may be a factor in progressive hip joint disease.

Articular Congruence

The specific shape of the human hip joint helps to decrease the peak contact stress.[2] The hip joint is considered to be a congruent joint. However, there is substantially more articular surface on the head of the femur than there is on the acetabulum. In the neutral or standing position, the articular surface of the femoral head remains exposed anteriorly and somewhat superiorly (Fig. 10–11A). The acetabulum does not fully cover the head superiorly, and the anterior torsion of the femoral head exposes a substantial amount of the femoral head's articular surface anteriorly. Articular contact between the femur and the acetabulum can be increased in the normal non-weight-bearing hip joint by a combination of flexion, abduction, and slight lateral rotation[51] (Fig. 10–11B). This position (also known as the frog-leg position) corresponds to that assumed by the hip joint in a quadruped position and, according to Kapandji,[51] is the true physiological position of the hip joint.

Konrath and colleagues[17] concluded both from their work and from evidence in the literature that the hip joint actually functions as an incongruent joint in non-weightbearing, given the larger femoral head. In weight-bearing, the elastic deformation of the acetabulum increases contact with the femoral head, with primary contact at the anterior, superior, and posterior articular surfaces of the acetabulum.[17] An additional contribution to articular congruence and

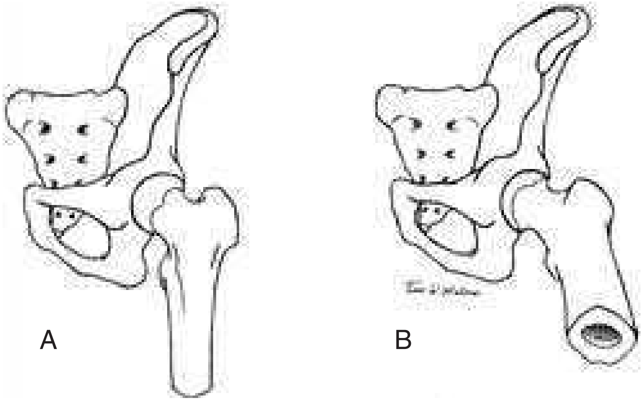

Figure 10–11 In the neutral hip joint, articular cartilage from the head of the femur is exposed anteriorly and, to a lesser extent, superiorly. Maximum articular contact of the head of the femur with the acetabulum is obtained when the femur is flexed, abducted, and laterally rotated slightly.

coaptation of joint surfaces may be made by the nonarticular and non-weightbearing acetabular fossa. The acetabular fossa may be important in setting up a partial vacuum in the joint so that atmospheric pressure contributes to stability by helping maintain contact between the femoral head and the acetabulum. Wingstrand and colleagues[52] concluded that atmospheric pressure in hip flexion activities played a stronger role in stabilization than capsuloligamentous structures. It is also true that the head and acetabulum will remain together in an anesthetized patient even after the joint capsule has been opened. The pressure within the joint must be broken before the hip can be dislocated.[51] The labrum enhances joint stability not only by deepening the acetabulum but also by acting as a seal to maintain negative intra-articular pressure.[12] When a labral tear is present, the labrum's ability to act as a buttress to prevent excessive movement and to act as a seal to maintain negative intra-articular pressure may be compromised, placing increased stress on the surrounding joint capsule.[53,54]

CASE APPLICATION

Articular Contact in the Dysplastic Hip

case 10–2

Gabriella's history may be consistent with her history of hip dysplasia. New radiographs and magnetic resonance imaging (MRI) showed a valgus anteverted femur and a shallow acetabulum of her left hip. Gabriella's structural deviations of femoral anteversion and coxa valga and a shallow acetabulum (decreased center edge angle) can result in increased articular exposure of the femoral head, less congruence, and reduced stability of the hip joint in the neutral weight-bearing position.

If Gabriella's hip dysplasia had been diagnosed in infancy, frog-leg positioning might have been maintained using a Frejka pillow or Pavlik harness[55] (Fig. 10–12). These devices may decrease the deformities by increasing the contact between the femoral head and acetabulum. The position of combined flexion, abduction, and rotation is commonly used for immobilization of the hip joint when the goal is to improve articular contact and joint congruence in conditions such as congenital dislocation of the hip and in Legg-Calvé-Perthes disease.[56] Whether this was done or not, Gabriella's deformities of the femur and acetabulum persisted. Considering the reduction in center edge angle alone, investigators have demonstrated that the stress distribution within the joint is concentrated in a smaller weight-bearing area throughout the gait cycle.[57] That increase in stress is likely to lead to degenerative changes over time. Recently, Jessel[58] identified that center edge angle was among the significant factors associated with osteoarthritis in addition to age and the presence of a labral tear. It appears that Gabriella's history of hip dysplasia may be predisposing her to labral pathology and subsequent osteoarthritis of the hip.

Figure 10–12 An infant can easily be maintained the hip joint position of flexion, abduction, and external rotation (frog-leg position) by using a positioning device.

Hip Joint Capsule and Ligaments

Hip Joint Capsule

Unlike the relatively weak articular capsule of the shoulder, the hip joint capsule is a substantial contributor to joint stability. The articular capsule of the hip joint is an irregular, dense fibrous structure with longitudinal and oblique fibers and with three thickened regions that constitute the capsular ligaments.[16,59] The capsule is attached proximally to the entire periphery of the acetabulum beyond the acetabular labrum.[16] Fibers near the proximal attachment are aligned in a somewhat circumferential manner and form a tight ring just below the femoral head.[16,59,60] This area is known as the **zona orbicularis.** It is believed the primary role of the zona orbicularis is to provide stability of the hip joint during distractive forces.[60] The capsule itself is thickened anterosuperiorly, where the predominant stresses occur; it is relatively thin and loosely attached posteroinferiorly,[16] with some areas of the capsule thin enough to be nearly translucent.[59] The capsule covers the femoral head and neck like a cylindrical sleeve and attaches to the base of the femoral neck. The femoral neck is intracapsular, whereas both the greater and lesser trochanters are extracapsular. The synovial membrane lines the inside of the capsule. Anteriorly, there are longitudinal retinacular fibers deep in the capsule that travel along the neck toward the femoral head.[16] The retinacular fibers carry blood vessels that are the major source of nutrition to the femoral head and neck.[1] The retinacular blood vessels arise from a vascular ring located at the base of the neck and formed by the medial and lateral circumflex arteries (branches of the deep femoral artery). Kalhor and colleagues[61] recently examined the arterial supply of the joint capsule and contributing blood vessels feeding the femoral head. The superior and inferior gluteal arteries supplied the hip capsule more proximally, whereas the medial and lateral circumflex arteries supplied the capsule more distally. Gluteal contributions dominated the posterior portions of the joint capsule, whereas the circumflex branches supply the anterior capsule. In the majority of specimens, the femoral head was supplied through the medial femoral circumflex artery.

As with the other joints already described, there are numerous bursae associated with the hip joint. Although as many as 20 bursae have been described, there are commonly recognized to be three primary or important bursae.[22,62,63] Because the bursae are more strongly associated with the hip joint muscles rather than its capsule, the bursae will be described with their corresponding musculature.

Hip Joint Ligaments

The ligamentum teres is an intra-articular but extrasynovial accessory joint structure. The ligament is a triangular band that attaches to the peripheral edge of the acetabular notch. The ligament then passes under the transverse acetabular ligament (with which it blends) to attach at its other end to the fovea of the femur; thus, it is also called the ligament of the head of the femur (Fig. 10–13). The ligamentum teres is encased in a flattened sleeve of synovial membrane so that it does not communicate with the synovial cavity of the joint. The material properties of the ligament of the head are similar to those of other ligaments.[64] Traditionally it has been believed that the sole purpose of the ligamentum teres was to serve as a conduit for blood supply to the femoral head and that it did not appear to play a significant role in joint stabilization regardless of joint position.[65] However, recent information suggests the ligamentum teres has a role in stabilizing the hip and when injured can contribute to symptoms.[66,67] Individuals with acquired anterior hip instability and abnormally increased lateral rotation ROM of the hip may put excessive strain on and potentially cause tearing of the ligamentum teres. Under arthroscopic assessment, the ligament was found to tighten with lateral rotation when the hip was flexed 10°.[68] Because of its laxity, pinching of the ligamentum teres between the femoral head and acetabulum can occur, causing complaints of pain and clicking. A lesion of the ligamentum teres was found to be the third most common finding in athletes undergoing arthroscopic surgery, occurring in 18% of individuals.[69]

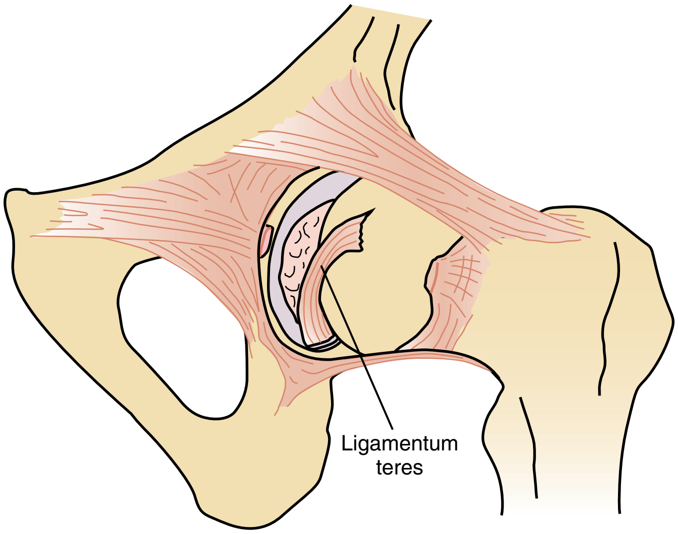

Figure 10–13 Anterior view of a right hip shows the centrally located ligamentum teres arising from the fovea on the femoral head. The joint capsule and other structures have been removed.

Continuing Exploration 10-2:

Blood Supply to the Femoral Head

The importance of the secondary blood supply carried by the ligamentum teres will vary across the life span, with a greater contribution to be made in childhood. While a child is still growing, the primary retinacular vessels (the medial and lateral circumflex arteries) cannot travel through the avascular cartilaginous epiphysis but must travel across the surface, where the vessels are more vulnerable to disruption. Crock[70] proposed that the femoral head was supplied predominantly by the blood vessels of the ligamentum teres until bony maturation and epiphyseal closure. However, Tan and Wong[71] found the ligament absent in 10% of their examined specimens. The vessels of the ligament of the head are commonly sclerosed in elderly persons.[1] Therefore, the secondary blood supply cannot be counted on to back up the primary retinacular supply when that supply is disrupted by such problems as femoral neck fracture.[71] The absence of a secondary blood supply to the head increases the risk of avascular necrosis of the femoral head with femoral neck trauma.

Continuing Exploration 10-3:

Legg-Calvé-Perthes Disease

Legg-Calvé-Perthes disease is noted by collapse of the femoral head, resulting in flattening of the head from loss of blood supply. This poorly understood childhood disorder is usually a self-limiting condition with a variable natural history. Impaired growth and abnormal development can result in deformity, joint incongruence, and the early onset of osteoarthritis over the long term if not properly treated.[72,73]

The hip joint capsule is typically considered to have three reinforcing capsular ligaments (two anteriorly and one posteriorly), although some investigators have further divided or otherwise renamed the ligaments.[51,65] For purposes of understanding hip joint function, the following three traditional descriptions appear to suffice. The two anterior ligaments are the **iliofemoral ligament** and the **pubofemoral ligament.** The iliofemoral ligament is a fan-shaped ligament that resembles an inverted letter *Y* (Fig. 10–14). It often is referred to as the **Y ligament of Bigelow.** The apex of the ligament is attached to the anterior inferior iliac spine, and the two arms of the Y fan out to attach along the intertrochanteric line of the femur. The superior band of the iliofemoral ligament is the strongest and thickest of the hip joint ligaments.[51,74] The pubofemoral ligament (see Fig. 10–14) is also anteriorly located, arising from the anterior aspect of the pubic ramus and passing to the anterior surface of the intertrochanteric fossa. The bands of the iliofemoral and the pubofemoral ligaments form a Z on the anterior capsule, similar to that of the glenohumeral ligaments. The **ischiofemoral ligament** is the posterior capsular ligament (Fig. 10–15). The ischiofemoral ligament attaches to the posterior surface of the acetabular rim and the acetabulum labrum. Some of its fibers spiral around the femoral neck and blend with the fibers of the circumferential fibers of the capsule. Other fibers are arranged horizontally and attach to the inner surface of the greater trochanter.

There is at the hip joint, as at other joints, some disagreement as to the roles of the joint ligaments. Fuss and Bacher[65] provided an excellent summary of the similarities and discrepancies to be found among several investigators. It may be sufficient to conclude, however, that each of the hip joint motions will be checked by at least one portion of one of the hip joint ligaments[65] and that the forces transmitted by the ligaments (and capsule) are dependent on orientation of the femur in relation to the acetabulum.[59,75] There is consensus that the hip joint capsule and the majority of its ligaments are quite strong and that each tightens with full hip extension (hyperextension). However, there is also evidence that the anterior ligaments are stronger (stiffer and withstanding greater force at failure) than the ischiofemoral ligament[74] and each ligamentous structure has a purpose related to maintaining the integrity of hip stability. Martin and colleagues[75] suggested that the posteriorly located ischiofemoral ligament is the primary restraint to medial rotation of the hip regardless of hip position in flexion or extension. On the anterior side of the hip joint, the pubofemoral ligament controls lateral rotation in an extended position. However, the primary stabilizing component of the anterior

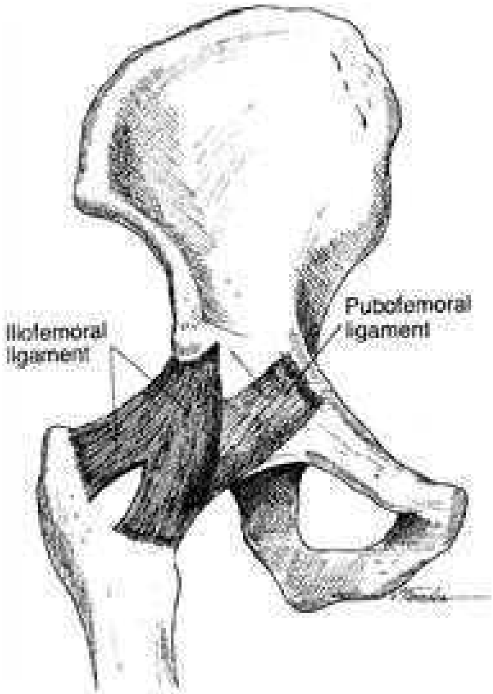

Figure 10–14 Anterior view of the right hip joint shows the two bands of the iliofemoral (Y) ligament and the more inferiorly located pubofemoral ligament.

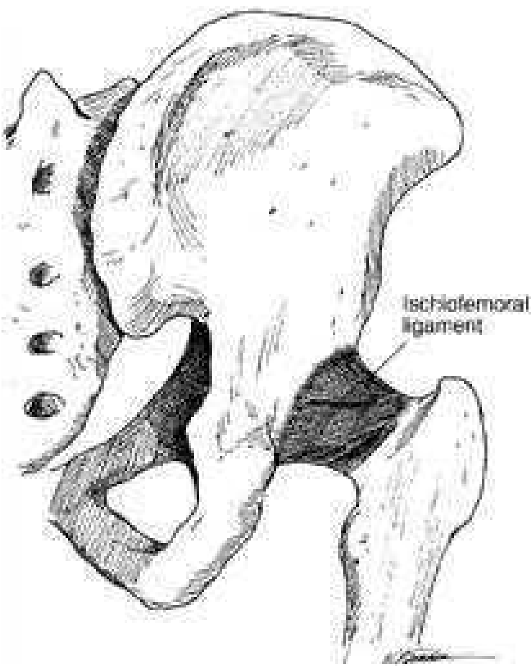

Figure 10–15 Posterior view of a right hip joint shows that the spiral fibers of the ischiofemoral ligament are tightened during hyperextension and therefore limit hyperextension.

hip joint is the iliofemoral ligament. This ligament is capable of providing resistance to excessive motion in both medial and lateral rotation. Martin and colleagues[75] demonstrated that the lateral branch of the iliofemoral limited medial rotation, especially when the hip was in extension. It also offered the most significant restraint to lateral rotation when the hip was in neutral position and when the hip was flexed.[75]

The capsule and ligaments permit little or no joint distraction even under strong traction forces. When a dysplastic hip is completely dislocated, the capsule and ligaments are strong enough to support the femoral head in weight-bearing. In these unusual conditions, the stresses on the capsule imposed by the femoral head may lead to impregnation of the capsule with cartilage cells that contribute to a sliding surface for the head.[76]

Under normal circumstances, the hip joint, its capsule, and ligaments routinely support two thirds of the body weight (the weight of head, arms, and trunk, or HAT). In bilateral stance, the hip joint is typically in neutral position or slight extension. In this position, the capsule and ligaments are under some tension.[51] The normal line of gravity in bilateral stance falls behind the hip joint axis, creating a gravitational extension moment. Further hip joint extension creates additional passive tension in the capsuloligamentous complex that is sufficient to offset the gravitation extension moment. As long as the line of gravity falls behind the hip joint axis, the capsuloligamentous structures are adequate to support the superimposed body weight in symmetrical bilateral stance without active assistance from the muscles crossing the hip.

Capsuloligamentous Tension

Hip joint extension, with slight abduction and medial rotation, is the close-packed position for the hip joint.[16] With increased extension, the ligaments twist around the femoral head and neck, drawing the head into the acetabulum. In contrast to most other joints in the body, the close-packed and stable position for the hip joint is not the position of optimal articular contact (congruence). As already noted, optimal articular contact occurs with combined flexion, abduction, and lateral rotation. Under circumstances in which the joint surfaces are neither maximally congruent nor close packed, the hip joint is at greatest risk for traumatic dislocation. A position of particular vulnerability occurs when the hip joint is flexed and adducted (as it is when sitting with the thighs crossed). In this position, a strong force up the femoral shaft toward the hip joint (as when the knee hits the dashboard in a car accident) may push the femoral head out of the acetabulum.[51] Macrotraumatic injuries associated with high impact are known to cause capsular laxity. Recent information suggests microtrauma associated with sports that involve repeated and forceful end-range movements may also be a cause of laxity.[77]

The capsuloligamentous tension at the hip joint is least when the hip is in moderate flexion, slight abduction, and midrotation. In this position, the normal intra-articular pressure is minimized, and the capacity of the synovial capsule to accommodate abnormal amounts of fluid is greatest.[52] This is the position assumed by the hip when there is pain arising from capsuloligamentous problems or from excessive intra-articular pressure caused by extra fluid (blood or synovial fluid) in the joint. Extra fluid in the joint may be a result of such conditions as **synovitis** of the hip joint or bleeding in the joint from tearing of blood vessels with femoral neck fracture. Wingstrand and colleagues[52] proposed that minimizing intra-articular pressure not only decreases pain in the joint but also prevents the excessive pressure from compressing the intra-articular blood vessels and interfering with the blood supply to the femoral head.

Structural Adaptations to Weight-Bearing

The internal architecture of the pelvis and femur reveals the remarkable interaction between mechanical stresses and structural adaptation created by the transmission of forces between the femur and the pelvis. The trabeculae (calcified plates of tissue within the cancellous bone) line up along lines of stress and form systems that normally adapt to stress requirements. The trabeculae are quite evident on bony cross-section, as seen in Figure 10–16, along with some of the other structural elements of the hip joint.

In Chapter 4, we followed the line of weight-bearing through the vertebrae of the spinal column to the sacral promontory and on through the sacroiliac joints. Most of the weight-bearing stresses in the pelvis pass from the sacroiliac joints to the acetabulum.[16] In standing or upright weight-bearing activities, at least half the weight of the HAT (the gravitational force) passes down through the pelvis to the femoral head, whereas the ground reaction force (GRF) travels up the shaft. These two forces, nearly parallel and in

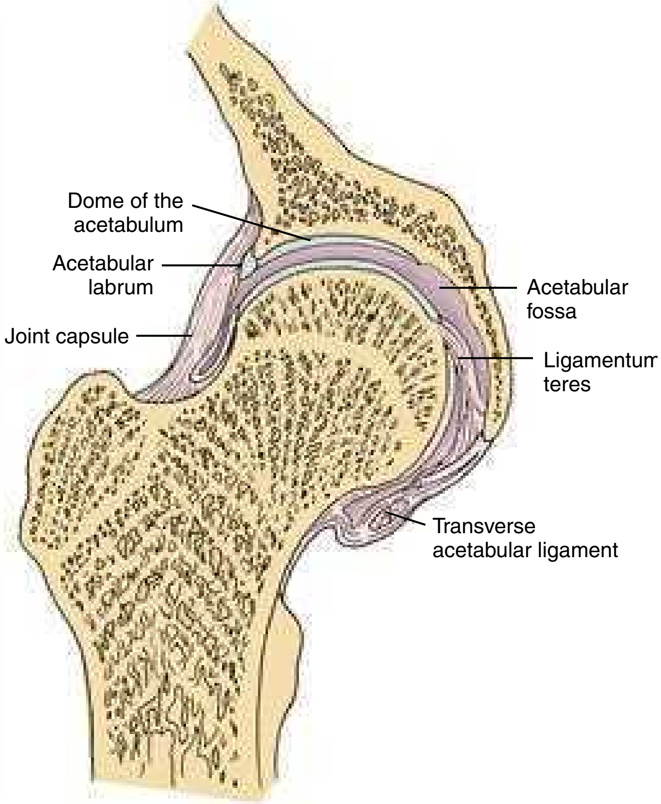

Figure 10–16 The trabeculae of the femur line up along lines of stress in the cancellous bone and can be seen on cross-section.

opposite directions, create a force couple with a moment arm (MA) equal to the distance between the superimposed body weight on the femoral head and the GRF up the shaft. These forces create a bending moment (or set of shear forces) across the femoral neck (Fig. 10–17).[78] The bending stress creates a tensile force on the superior aspect of the femoral neck and a compressive stress on the inferior aspect. A complex set of forces prevents the rotation and resists the shear forces that the force couple causes; among these forces are the structural resistance of two major and three minor **trabecular systems** (Fig. 10–18).

The medial (or principal compressive) trabecular system arises from the medial cortex of the upper femoral shaft and radiates through the cancellous bone to the cortical bone of the superior aspect of the femoral head. The medial system of trabeculae is oriented along the vertical compressive forces passing through the hip joint.[51] The lateral (or principal tensile) trabecular system of the femur arises from the lateral cortex of the upper femoral shaft and, after crossing the medial system, terminates in the cortical bone on the inferior aspect of the head of the femur. The lateral trabecular system is oblique and may develop in response to parallel (shear) forces of the weight of HAT and the ground reaction force.[51] There are two accessory (or secondary) trabecular systems, of which one is considered compressive and the other is considered tensile.[79] Another secondary trabecular system is confined to the trochanteric area of the femur.[51,79] Heller and colleagues[80] used data from instrumented in vivo hip prostheses and mathematical modeling to conclude that the loading environment in the femur during activity was largely compressive, with relatively small shear forces.

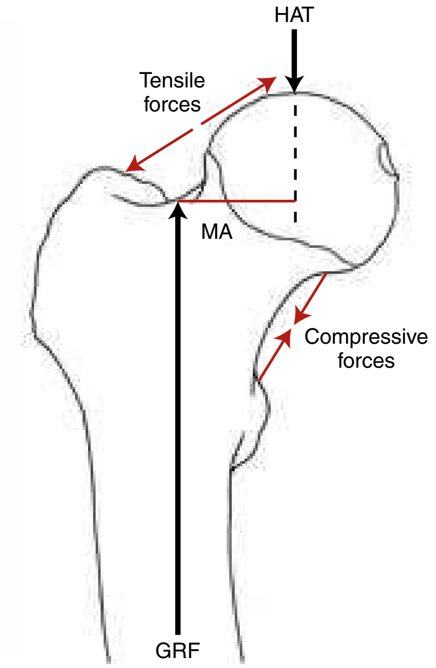

Figure 10–17 The weight-bearing line of the head, arms, and trunk (HAT) loads the head of the femur, whereas the ground reaction force (GRF) comes up the shaft of the femur, resulting in a force couple that creates a bending moment, with a moment arm (MA) that is dependent on the length and angle of the neck of the femur. The bending moment creates tensile stress on the superior aspect of the femoral neck and compressive stress on the inferior aspect.

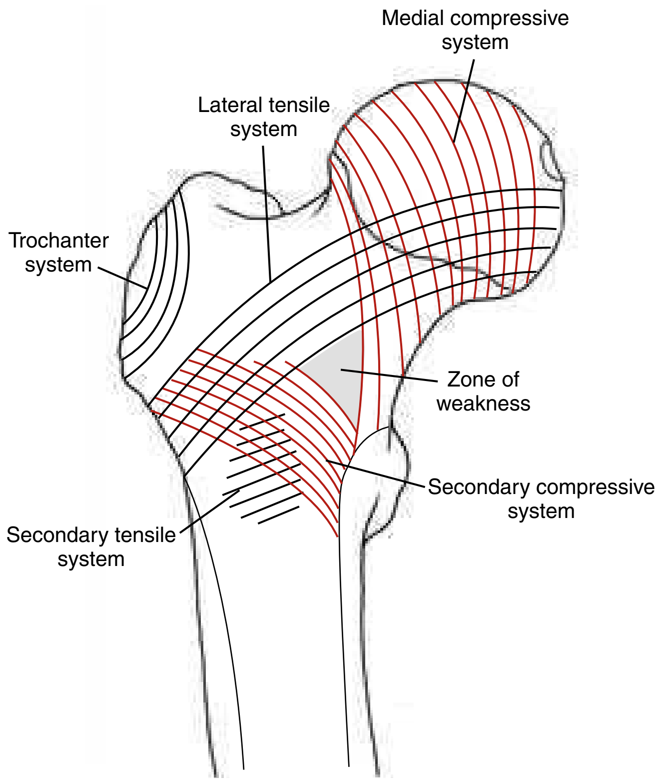

Figure 10–18 Two major (the medial compressive and lateral tensile) trabecular systems show the primary transmission of forces. Additional lines of stress are evident at the secondary compressive and tensile systems and at the trochanteric system.

The areas in which the trabecular systems cross each other at right angles are areas that offer the greatest resistance to stress and strain. There is an area in the femoral neck in which the trabeculae are relatively thin and do not cross each other. This **zone of weakness** (see Fig. 10–18) has less reinforcement and thus more potential for failure. The zone of weakness of the femoral neck is particularly susceptible to the bending forces across the area and can fracture either when forces are excessive or when compromised bony composition reduces the tissue's ability to resist typical forces.

Continuing Exploration 10-4:

Femoral Neck Stresses

Although the zone of weakness in the cancellous bone has received a great deal of attention as a factor in hip fracture, Crabtree and colleagues[81] used data from patients and cadavers with a hip fracture to conclude that the cortical bone in the femoral neck supports at least 50% of the load placed on the proximal femur. They suggested that compromise of cortical bone may be more of a factor in fracture than is diminished cancellous bone. A more detailed description of the problems of hip fracture will be presented later in the chapter.

The primary weight-bearing surface of the acetabulum, or **dome** of the acetabulum, is located on the superior portion of the lunate surface[82,83] (see Fig. 10–16). In the normal hip, the dome lies directly over the center of rotation of the femoral head.[83] Genda and colleagues,[29] using radiographs and modeling, found peak contact pressures during unilateral stance to be located near the dome. Stress distribution can be altered with a lack of femoral head coverage by the acetabulum[2,29] (decreased center edge angle) as well as excessive acetabular anterversion.[2] Contact area was found to be significantly smaller in women than in men with associated higher peak contact forces.[29] The dome shows the greatest prevalence of degenerative changes in the acetabulum which corresponds to the area of greatest pressure.[82,84,85] The primary weight-bearing area of the femoral head is, correspondingly, its superior portion.[82,86] Degenerative changes in the femoral head include loss of the regular "ball" shape with flattening of the superior portion. Degenerative changes are also consistently noted near the attachment of the ligamentum teres.[28]

Athanasiou and colleagues[82] proposed that the variations in material properties, creep characteristics, and thickness may explain the differences in response of articular cartilage in the acetabular and femoral primary weight-bearing areas. Full loading of the hip joint is presumably necessary to achieve congruence and optimize load distribution between the larger femoral head and the acetabulum.[17] Persisting incongruence in the dome of the acetabulum in the moderately loaded hip (especially in young adults) could result in incomplete compression of the dome cartilage and, therefore, inadequate fluid exchange to maintain cartilage nutrition.[82] The superior femoral head receives compression not only from the dome in standing but also from the posterior acetabulum in sitting and the anterior acetabulum in extension. More frequent and complete compression of the cartilage of the superior femoral head, according to this premise, leads to better nutrition within the cartilage. It must be remembered, however, that avascular articular cartilage is dependent on both *compression* and *release* to move nutrients through the tissue; both too little compression and excessive compression can lead to compromise of the cartilage structure.

The forces of HAT and the ground reaction force that act on the articular surfaces of the hip joint and on the femoral head and neck also act on the femoral shaft. The shaft of the femur is not vertical but lies at an angle that varies considerably among individuals. However, the vertical loading on the oblique femur results in bending stresses in the shaft.[51] The medial cortical bone in the shaft (diaphysis) must resist compressive stresses, whereas the lateral cortical bone must resist tensile stresses (Fig. 10–19).

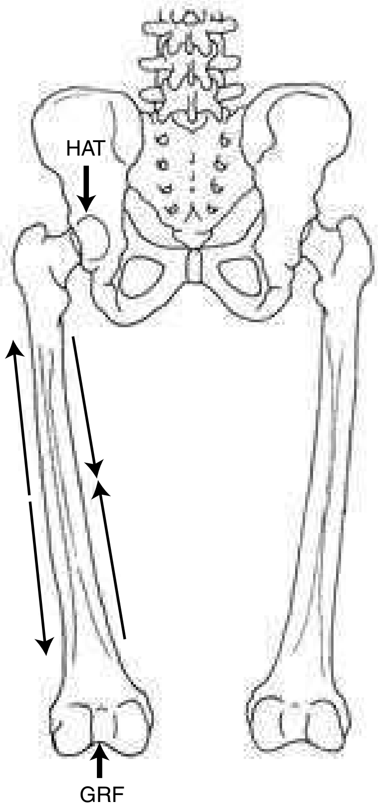

Figure 10–19 The weight-bearing line (HAT) from the center of rotation of the femoral head and the ground reaction force (GRF) causes a bending force on the shaft of the femur that results in compressive forces medially and tensile forces laterally.

FUNCTION OF THE HIP JOINT

Motion of the Femur on the Acetabulum

The motions of the hip joint are easiest to visualize as movement of the convex femoral head within the concavity of the acetabulum as the femur moves through its three degrees of freedom: flexion/extension, abduction/adduction, and medial/lateral rotation. The femoral head will glide within the acetabulum in a direction opposite to the motion of the distal end of the femur. Flexion and extension of the femur occur from a neutral position as an almost pure spin of the femoral head around a coronal axis through the head and neck of the femur. The head spins posteriorly in flexion and anteriorly in extension. However, flexion and extension from other positions (e.g., in abduction or medial rotation) must include both spinning and gliding of the articular surfaces, depending on the combination of motions. The motions of abduction/adduction and medial/lateral rotation must include both spinning and gliding of the femoral head within the acetabulum, but the intra-articular motion again occurs in a direction opposite to motion of the distal end of the femur.

As is true at most joints, the hip joint's ROM is influenced by structural elements, as well as by whether the motion is performed actively or passively and whether passive tension in two-joint muscles is encountered or avoided. The following ranges of passive joint motion are typical of the hip joint.[87] Flexion of the hip is generally about 90° with the knee extended and 120° when the knee is flexed and passive tension in the two-joint **hamstrings** muscle group is released. Hip extension is considered to have a range of 10° to 30°. Hip extension ROM appears to diminish somewhat with age, whereas flexion remains relatively unchanged.[88] When hip extension is combined with knee flexion, passive tension in the two-joint **rectus femoris** muscle may limit the movement. The femur can be abducted 45° to 50° and adducted 20° to 30°. Abduction can be limited by the two-joint **gracilis** muscle and adduction limited by the **tensor fascia lata (TFL)** muscle and its associated **iliotibial (IT) band.** Medial and lateral rotations of the hip are usually measured with the hip joint in 90° of flexion; the typical range is 42° to 50°.

Normal gait on level ground requires at least the following hip joint ranges: 30° flexion, 10° hyperextension, 5° of both abduction and adduction, and 5° of both medial and lateral rotation.[52,80] Walking on uneven terrain or stairs will increase the need for joint range beyond that required for level ground, as will activities such as sitting in a chair or sitting cross-legged.

CASE APPLICATION

Range of Motion in the Anteverted Dysplastic Hip

case 10–3

Gabriella's passive range of motion (ROM) findings and asymmetries in her gait correspond to femoral anteversion on the left. In the supine position on the examination table (hip extended), she has more hip joint medial than lateral rotation (although medial rotation with hip flexion is limited by pain). In her case, her shallow acetabulum increases the tendency for joint subluxation with lateral rotation of the hip. This would partially explain her complaints that her hip feels like it is "popping out of socket." When she walks, Gabriella's left foot is toed out less than her right because her involved side is in greater hip medial rotation compared to her uninvolved side. This may be the body's way of maximizing congruence of the anteverted femur on the weight-bearing joint, or it may be to minimize stretch on the proprioceptor-rich anterior capsule. The consequence for increased hip joint congruence is that the torsional deformity now appears as medial femoral torsion, resulting in a deviant positioning of the patella and femoral condyles.[89] Gabriella's foot would be more toed-in than she demonstrates, but it appears that Gabriella, like many persons with femoral anteversion (medial femoral torsion), has also developed an excessive lateral tibial torsion. It is evident in Gabriella's case that altered biomechanical alignment of the hips relates directly changes throughout the entire kinetic chain, specifically the knee, foot, and ankle.

Motion of the Pelvis on the Femur

Whenever the hip joint is weight-bearing, the femur is relatively fixed, and, in fact, motion of the hip joint is produced by movement of the pelvis on the femur. At all joints, the motion between articular surfaces is the same whether the distal lever moves or the proximal lever moves. However, the proximal lever and distal lever move in opposite directions to produce the same articular motion. For example, elbow flexion can be a rotation of the distal forearm upward or, conversely, a rotation of the proximal humerus downward. In examinations of the upper extremity joint complexes thus far, motion of the distal lever tended to predominate functionally, and so this apparent reversal of motions was not a point of discussion. At the hip joint, this reversal of motion of the lever is further complicated by the horizontal orientation and shape of the pelvis (the "levers" of the hip are not in line but lie essentially perpendicular to each other). In contrast to other joints, there is also a new set of terms to identify joint motion when the pelvis (rather than femur) is the moving segment. The terms for pelvic motions are used with weight-bearing hip motion because the motions of the pelvis are more apparent to the eye of the examiner and are, in fact, key to what occurs at the joints above and below the pelvis. It must be emphasized, however, that the motion of the pelvis presented in the next sections are *not new motions of the hip joint* but are simply how the same three degrees of freedom for the hip joint are accomplished by the pelvis rather than by the femur.

Anterior and Posterior Pelvic Tilt

Anterior and **posterior pelvic tilts** are motions of the entire pelvic ring in the sagittal plane around a coronal axis. In the normally aligned pelvis, the anterior superior iliac spines (ASISs) of the pelvis lie on a horizontal line with the posterior superior iliac spines and on a vertical line with the symphysis pubis[90] (Fig. 10–20A). Anterior and posterior tilting of the pelvis on the fixed femur produce hip flexion and extension, respectively. Hip joint extension through posterior tilting of the pelvis brings the symphysis pubis up and the sacrum of the pelvis closer to the femur, rather than moving the femur posteriorly on the pelvis (Fig. 10–20B). Hip flexion through anterior tilting of the pelvis moves the anterior superior iliac spines anteriorly and inferiorly; the inferior sacrum moves farther from the femur, rather than moving the femur away from the sacrum (Fig. 10–20C). Anterior and posterior tilting will result in flexion and extension of both hip joints simultaneously in bilateral stance or can occur at the stance hip joint alone if the opposite limb is non-weightbearing.

Concept Cornerstone 10-1

Anterior/Posterior Pelvic Tilt Versus Pelvic Torsion

Clinicians who evaluate and treat sacroiliac joint dysfunction may attempt to diagnose someone as having asymmetry in the sagittal plane between the two halves of the pelvis (ilia or innominate bones). There are a number of terms used to label this imbalance that are beyond this discussion. However, when the imbalance is referred to as anterior or posterior torsion—or, more confusingly, anterior or posterior tilt—the potential for confusion for the novice is great. When the terms *anterior/posterior torsion* or *anterior/posterior tilt* are used in reference to the *sacroiliac joint* and *sacroiliac joint dysfunction*, these terms generally need to be distinguished as different from anterior/posterior tilt of the entire pelvis, in which the pelvis is considered to, in effect, move as a single fixed unit. In this chapter and through this text, **anterior/posterior tilt** of the pelvis will be used exclusively to refer to the pelvis as a fixed unit.

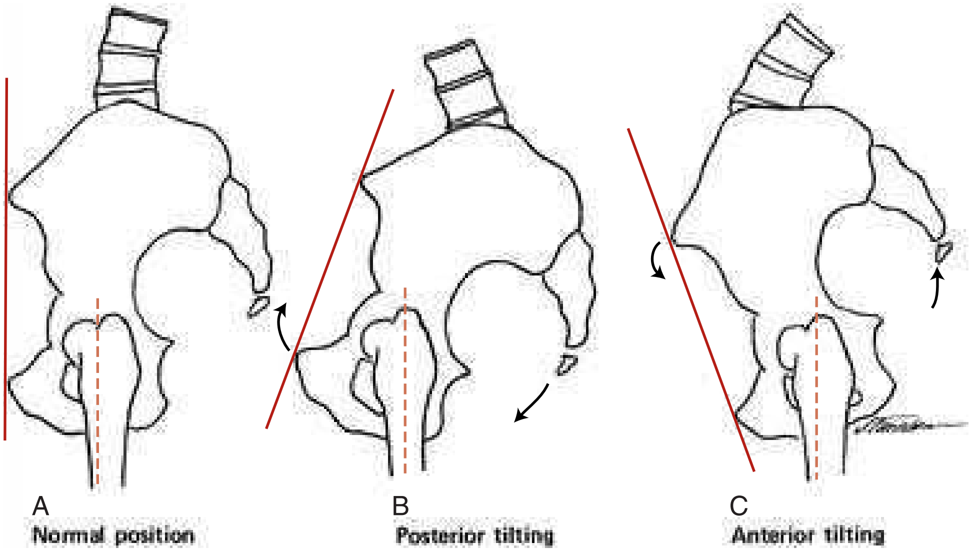

Figure 10–20 Flexion and extension of the hip occurring as tilting of the pelvis in the sagittal plane. **A.** The pelvis is shown in its normal position in erect stance. **B.** Posterior tilting of the pelvis moves the symphysis pubis superiorly on the fixed femur. The hip joint extends. **C.** In anterior tilting, the anterior superior iliac spines move inferiorly on the fixed femur. The hip joint flexes.

Lateral Pelvic Tilt

Lateral pelvic tilt is a frontal plane motion of the entire pelvis around an anteroposterior axis. In the normally aligned pelvis, a line through the anterior superior iliac spines is horizontal. In lateral tilt of the pelvis in unilateral stance, one hip joint (e.g., the left hip joint) is the pivot point or axis for motion of the opposite side of the pelvis (e.g., the right side) as that side of the pelvis elevates **(pelvic hike)** or drops **(pelvic drop).** If a person stands on the left limb and hikes the pelvis, the left hip joint is being abducted because the medial angle between the femur and a line through the anterior superior iliac spines increases (Fig. 10–21A). If a person stands on the left leg and drops the pelvis, the left hip joint will adduct because the medial angle formed by the femur and a line through the anterior superior iliac spines will decrease (Fig. 10–21B).

In descriptions of the hip joint motions that occur in unilateral stance, the hip joint of the non-weightbearing limb is in an open chain and has no *imposed* motions on it. However, the non-weightbearing leg typically hangs straight down as the pelvis moves. If the non-weightbearing limb continues to hang vertically, the non-weightbearing hip will adduct and abduct with hike and drop of the pelvis, respectively, around the weight-bearing hip joint (see Fig. 10–21A, B).

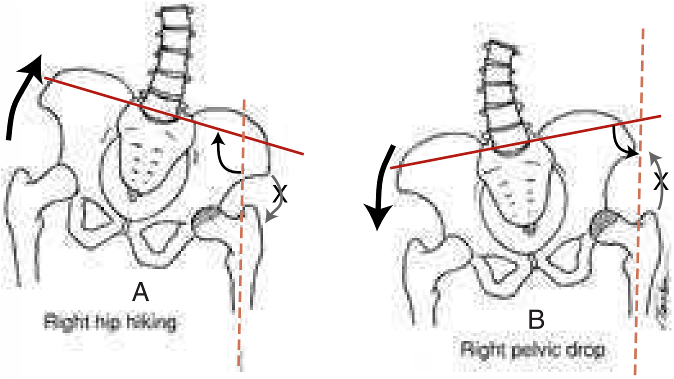

Figure 10–21 Lateral tilting of the pelvis around the left can occur either as hip hiking (elevation of the opposite side of the pelvis) or as pelvic drop (drop of the opposite side of the pelvis). **A.** Hiking of the pelvis around the left hip joint results in left hip abduction. **B.** Dropping of the pelvis around the left hip joint results in left hip joint adduction. Although it is visually tempting to name the direction of lateral tilt by the motion of the side of the pelvis *nearest* the hip (gray arrows that are "crossed out"), this is incorrect.

Concept Cornerstone **10-2**

Pelvic Hike and Pelvic Drop

Identifying the motions of pelvic hike or pelvic drop in lateral pelvic tilt often confuses the examiner because the eye tends to follow the iliac crest on the same side as the supporting hip joint rather than the opposite side. Because the hip joint is not at the end of the pelvic lever but is offset quite a bit (more medially located), the eye can see motion of both the iliac crest on the side of the weight-bearing hip joint and the iliac crest on the side opposite to the weight-bearing hip joint. In Figure 10–21A and B, the gray arrows indicate the side of the pelvis that the eye might be tempted to follow, but the arrows are also "crossed" out to indicate that the wrong side of the pelvis is being referenced. Although it may not have been necessary to specify this previously, it should also be kept in mind that, in naming motions of levers, the motion of the end of the lever farthest from the joint axis is always referenced. Lateral pelvic tilt is named (and should be observed) by what is happening to the side of the pelvis *opposite* to the weight-bearing hip in unilateral stance. The weight-bearing hip joint in unilateral stance will always be the axis of rotation, and the opposite side of the pelvis will always the reference side for naming the movement. If a woman is standing on her right leg and hikes her pelvis, it should not be necessary to specify that the left side of the pelvis is the one that is rising. Because the right hip joint is the axis, the motion is defined by movement of the left side of the pelvis.

Lateral Shift of the Pelvis

Lateral pelvic tilt can also occur in bilateral stance. If both feet are on the ground and the hip and knee of one limb are flexed, the opposite limb is largely the weight-bearing limb and the terminology is the same as for unilateral stance. However, if both limbs are weight-bearing, lateral tilt of the pelvis will cause the pelvis to shift to one side or the other. With **pelvic shift,** the pelvis cannot hike; it can only drop. Because there is a closed chain between the two weight-bearing feet and the pelvis, both hip joints will move in the frontal plane in a predictable way as the pelvic tilt (or pelvic shift) occurs. If the pelvis is shifted to the right in bilateral stance, the left side of the pelvis will drop, the right hip joint will be adducted, and the left hip joint will be abducted (Fig. 10–22).

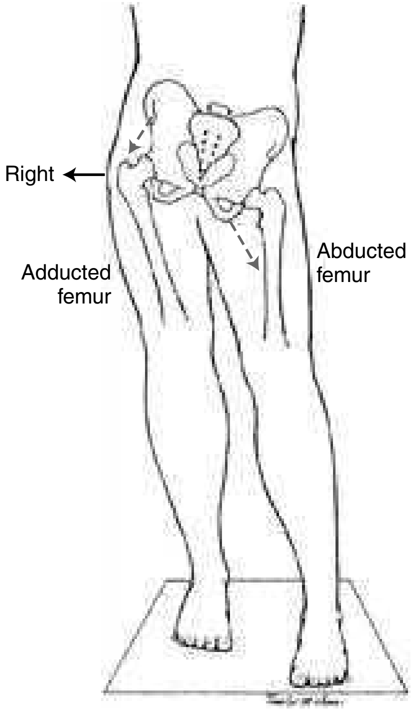

Figure 10–22 When the pelvis is shifted to the right in bilateral stance, the right hip joint will be adducted and the left hip joint will be abducted. To return to neutral position while continuing to bear weight on both feet, the right abductor and left adductor muscles work synergistically to shorten and shift the weight back to center.

Forward and Backward Pelvic Rotation

Pelvic rotation is motion of the entire pelvic ring in the transverse plane around a vertical axis. Although rotation can occur around a vertical axis through the middle of the pelvis in bilateral stance, it most commonly and more importantly occurs in single-limb support around the axis of the supporting or weight-bearing hip joint. **Forward** (anterior) rotation of the pelvis occurs in unilateral stance when the side of the pelvis opposite to the weight-bearing hip joint moves anteriorly (Fig. 10–23A) from the neutral position (Fig. 10–23B). Forward rotation of the pelvis produces medial rotation of the weight-bearing hip joint. **Backward** (posterior) **rotation** of the pelvis occurs when the side of the pelvis opposite the weight-bearing hip moves posteriorly (Fig. 10–23C). Backward rotation of the pelvis produces lateral rotation of the supporting hip joint.

Pelvic rotation can occur in bilateral stance as well as unilateral stance, as is true for lateral pelvic tilt. If both feet are bearing weight and the axis of motion occurs around a vertical axis through the *center* of the pelvis, the terms *forward rotation* and *backward rotation* must be used by referencing a side (e.g., forward rotation on the right and backward rotation on the left).

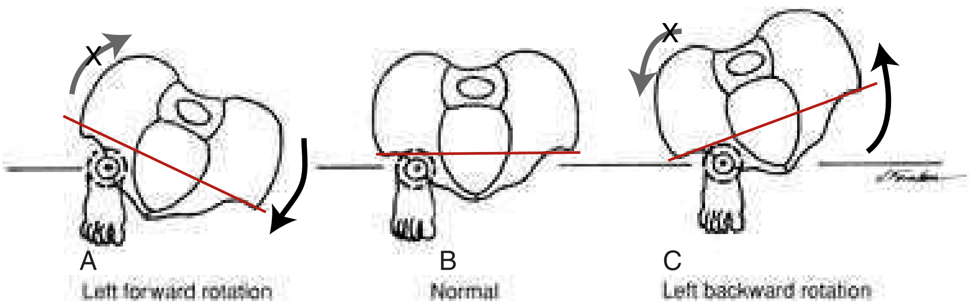

Figure 10–23 A superior view of rotation of the pelvis in the transverse plane. **A.** Forward rotation of the pelvis around the right hip joint results in medial rotation of the right hip joint. **B.** Neutral position of the pelvis and the right hip joint. **C.** Backward rotation of the pelvis around the right hip joint results in lateral rotation of the right hip joint. The reference for forward and backward rotation is the side *opposite* the supporting hip, although the eye often erroneously catches the opposite motion of the pelvis on the same side (gray crossed-out arrows).

Concept Cornerstone **10-3**

Pelvic Rotation and Hip Joint Rotation

In referencing forward and backward rotation, we must again make sure that the reference is the side of the pelvis opposite to the rotating (weight-bearing) hip joint. In Figure 10–23, the gray arrows again indicate the side the eye often erroneously follows (and so are "crossed out"). If it is known which leg a person is standing on in unilateral stance, forward or backward rotation of the pelvis always references the side of the pelvis opposite to the weight-bearing hip joint.

The relative rotation of the hip that occurs with forward or backward rotation of the pelvis in unilateral stance is often difficult for the novice to identify. The rotation of the hip joint that occurs during rotation of the pelvis can best be appreciated by performing the motion yourself. Standing on one leg and rotating the pelvis and trunk forward as much as possible will give a clear "feeling" of the relative medial rotation of the supporting limb. Similarly, rotating the pelvis backward as far as possible will give the feeling of the relative lateral rotation of the stance hip joint.

Continuing Exploration 10-5:

Pelvic Rotation in Gait

One exception to the convention of naming pelvic rotation by the side of the pelvis opposite the supporting hip may occur in observational gait analysis. In normal gait, the pelvis forwardly rotates around the weight-bearing hip while the other limb prepares for or is in swing.[91] Because this happens first on one leg and then on the other, it appears to the eye as if the pelvis is forwardly rotating and then backwardly rotating (Fig. 10–24). Because gait is often observed for one side (the referent side) of the body at a time, the pelvis may be identified as forwardly rotating during normal swing of the referent side and backwardly rotating during normal stance of the referent limb.[92] This terminology, although useful during observation, is misleading and misrepresents both the pelvic and hip joint motions during normal gait.

Coordinated Motions of the Femur, Pelvis, and Lumbar Spine

When the pelvis moves on a relatively fixed femur, there are two possible outcomes to consider. Either the head and trunk will follow the motion of the pelvis (moving the head through space) or the head will continue to remain relatively upright and vertical despite the pelvic motions. These are open- and closed-chain responses, respectively. Each of these two situations produces very different reactions from the joints and segments proximal and distal to the hip joints and pelvis and must be examined separately.

Pelvifemoral Motion

When the femur, pelvis, and spine move in a coordinated manner to produce a larger ROM than is available to one segment alone, the hip joint is participating in what will predominantly (but not exclusively) be an open-chain motion termed **pelvifemoral motion.** Pelvifemoral motion can be considered analogous to scapulohumeral motion because the combination of motions at several joints serves to increase the range available to the distal segment. In the case of scapulohumeral motion, the joints are serving the hand. In the case of pelvifemoral motion, the joints may serve either end of the chain: the foot or head.

Example 10-1

Moving the Head and Arms Through Space

If the goal is to bend forward to bring the hands (and head) toward the floor, isolated flexion at the hip joints (anteriorly tilting the pelvis on the femurs) is generally insufficient to reach the ground. *If* the knees remain extended, the hips will typically flex no more than 90° (and often less, depending on extensibility of the hamstrings). The addition of flexion of the lumbar spine (and, perhaps, flexion of the thoracic spine) will add to the total ROM (Fig. 10–25). The combination of hip and trunk flexion is generally sufficient for the hands to reach the ground—as long as the hamstrings and lumbar extensors allow sufficient lengthening. The

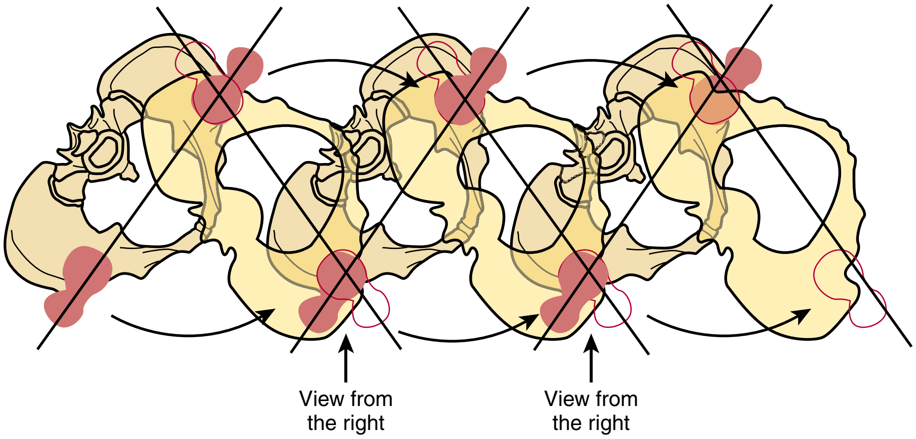

Figure 10–24 This schematic representation of a superior view of the pelvis is shown forwardly rotating sequentially around the left and right hips during gait (the rotation is exaggerated to make the point more clearly). Observation of the right side of the pelvis alone will give the illusion of the pelvis forwardly and backwardly rotating sequentially.

combination of hip motion and lumbar motion to achieve a greater ROM for the hands and head is an example of a largely open-chain response in the hips and trunk.

Sidebar: Please note that this is *not* an example of how to reach the floor to pick up an object!

The open-chain response (the ability of each joint in the chain to move independently) is somewhat constrained (largely at the ankles) by the need to keep the line of gravity within the base of support.

Example 10-2

Moving the Foot Through Space

When a person is lying on the right side, the left foot may be moved through an arc of motion approaching 90° (Fig. 10–26). This is clearly not all from the left hip joint, which can typically abduct only to 45°; motion of the foot through space also includes lateral tilting of the pelvis (hiking around the right hip joint) and lateral flexion of the lumbar spine to the left. The abducting limb is in an open chain; the lumbar spine (and thoracic spine) is constrained by the body weight and contact with the ground.

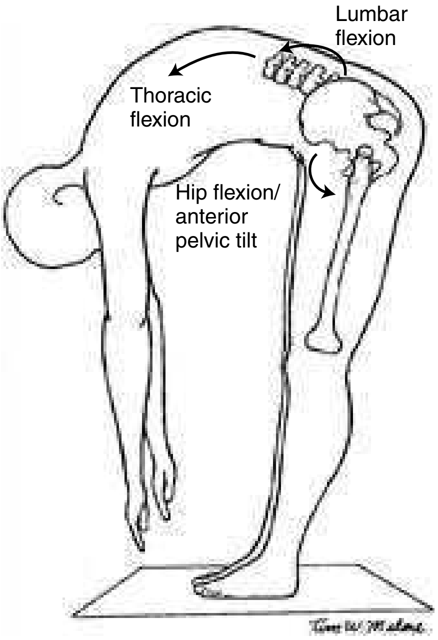

Figure 10–25 Pelvifemoral motion can increase the range of forward flexion of the head and arms by combining hip flexion, anterior pelvic tilt, and flexion of the lumbar spine. This combination permits the hands to maximize the reach toward the ground.

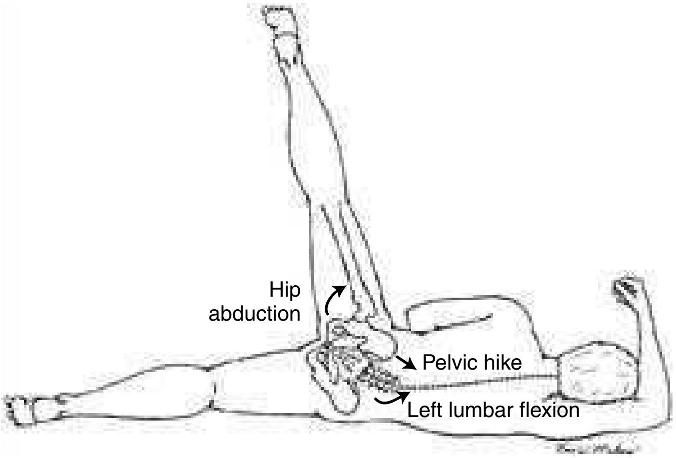

Figure 10–26 Pelvifemoral motion increases the range through which the foot can be moved in space by combining left hip abduction, lateral pelvic tilt (left hike), and flexion of the lumbar spine to the left.

Pelvifemoral motion has also been referred to as **pelvifemoral "rhythm,"** which implies a continuous relationship between the two segments that is arguable because the relative contributions can vary among individuals and in different activities. Bohannon and colleagues determined that pelvic tilt contributed between 30% and 46% of the total range of a passive straight leg raise.[93] During active maximal hip flexion (knee flexed) in standing (e.g., marching in place), Murray and colleagues[94] found that pelvic tilt contributed between 8% and 32% of the total motion, with an even greater variability among individuals (9% to 53%) when a 4.53-kg ankle weight was added.[94] The link between hip, pelvis, and lumbar motion is the basis of using provocation of pain with active straight-leg raising as a test for severity of dysfunction in persons with low back pain.[95,96]

Closed-Chain Hip Joint Function

The joints of the right and left lower limbs are part of a true closed chain when both lower limbs are weight-bearing and the chain is defined as all the segments between the right foot, up through the pelvis, and down through the left foot. A true closed chain is formed because both ends of the chain (both feet in this example) are "fixed" and movement at any one joint in the chain invariably involves movement at one or more other links in the chain. It is important not to confuse the idea of weight-bearing and closed chain as interchangeable terms. In Example 10-1, pelvic motion on the fixed femur results in moving the head and trunk through space. This is an open chain movement despite the feet remaining securely to the floor in a weight-bearing position.

For the hips (and other lower limb joints) to be in a closed chain in standing, both ends of the chain (the head and the feet) must be fixed. The feet are, in fact, fixed by weight-bearing. The head, however, is often (but not necessarily) functionally "fixed." Although the head is certainly free to move in space, the head most often remains upright and vertically oriented during upright activities. The drive to keep the head upright is due, in part, to the influence of the tonic labyrinthine and optical righting reflexes that are normally evident almost immediately at birth and continue to operate throughout life.[97] The drive to keep the head upright and over the sacrum will effectively *fix the head in relative space* even though this is not structurally the case; that is, the head is functionally rather than structurally fixed. When the head (one end of the chain) is held upright and over the feet (the other end of the chain), all the segments in the axial skeleton and lower limbs function as part of a closed chain; movement at one joint will create movement in at least one other linkage in the chain. Consequently, in our functional closed-chain premise, hip flexion does not occur independently (which would move the head forward in space) but is accompanied by mandatory motion in one or more interposed segments to ensure that the head remains upright over the base of support and that the body does not become unstable. Table 10–1 presents the compensatory motions of the lumbar spine that accompany given motions of the pelvis and hip joint in a functional closed chain.

Example 10-3

Closed-Chain Hip Joint Function

A common example of closed-chain (versus open-chain) function is seen when the hip flexor musculature is tight and the hip joint is maintained in flexion. A person standing with fixed hip flexion is shown in Figure 10–27A (an open-chain response) and B (a closed-chain response). A true open-chain response to isolated hip flexion would displace the head and trunk forward, with the line of gravity falling in front of the supporting feet. More commonly, hip flexion in stance is not isolated to the hips but is accompanied by compensatory movements of the vertebral column (including extension or lordosis of the lumbar spine) that maintain the head in the upright position and keep the line of gravity within the base of support. In a functional closed chain, motion at the hip (one link in the chain) is accompanied by an essentially mandatory lumbar *extension* to maintain the head over the sacrum (see Fig. 10–27B). In contrast, hip flexion in open-chain pelvifemoral motion is accompanied by *lumbar flexion* because the goal is to achieve more range for the head in space (see Fig. 10–25).

CASE APPLICATION

The Hip Joint and Leg Length Discrepancy

case 10–4

Skeletal shortening of the limb with a developmental hip dysplasia is not unusual. As Gabriella stands with her weight evenly distributed between her feet, her pelvis will be laterally tilted (down on the left) as a result of a measured 1-in. shortening of her left leg. To keep the line of gravity within the center of her base of support in bilateral stance, Gabriella's lumbar spine will be slightly laterally flexed to the right (away from the side of shortening). This is the opposite lumbar motion to what we saw in Example 10-2 because Gabriella's goal is to keep her head upright, not to gain ROM. The lateral flexion of the spine with asymmetrical leg lengths puts a person at risk for low back pain, although this is not one of Gabriella's presenting complaints. Interestingly, the relative abduction of the left leg that occurs in stance with shortening of Gabriella's dysplastic limb may reduce stress on the hip. The abducted position of the hip has the potential to increase congruence slightly, diminishing the peak pressure at the hip joint by distributing the forces over a larger contact area.

Hip Joint Musculature

There have been numerous studies of the muscles of the hip joint. Most confirm underlying principles of muscle physiology seen at the other joints we have examined so far. That is, hip joint muscles work best in the middle of their contractile range or on a slight stretch (at so-called optimal length-tension); two-joint muscles generate greatest force when not required to shorten over both joints simultaneously; and tension generation is optimal with eccentric contractions, followed by isometric and then concentric contractions.

The muscles of the hip joint make their most important contributions to function during weight-bearing. In weight-bearing, the muscles are called on to move or support the HAT (approximately two thirds of body weight) rather than the weight of one lower limb (approximately one sixth of body weight). Consequently, the hip joint muscles adapt

Table 10–1 Relationship of Pelvis, Hip Joint, and Lumbar Spine During Right Lower Extremity Weight-Bearing and Upright Posture

PELVIC MOTION	ACCOMPANYING HIP JOINT MOTION	COMPENSATORY LUMBAR SPINE MOTION
Anterior pelvic tilt	Hip flexion	Lumbar extension
Posterior pelvic tilt	Hip extension	Lumbar flexion
Lateral pelvic tilt (pelvic drop)	Right hip adduction	Right lateral flexion
Lateral pelvic tilt (pelvic hike)	Right hip abduction	Left lateral flexion
Forward rotation	Right hip medial rotation	Rotation to the left
Backward rotation	Right hip lateral rotation	Rotation to the right

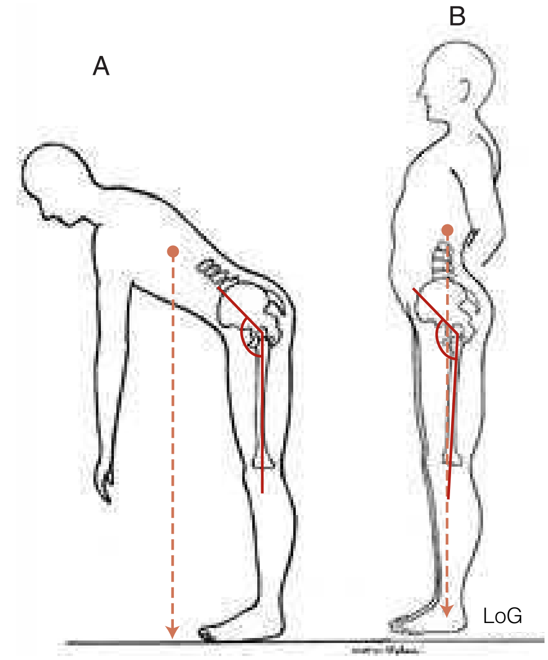

Figure 10–27 **A.** In an open-chain response to tight hip flexors that is isolated to the hip joints, the trunk will be inclined forward. The line of gravity (LoG) will fall outside the base of support if no other adjustments are made. **B.** In a functional closed-chain response to tight hip flexors, the head seeks to maintain a vertical position; the lumbar spine will extend (become lordotic) to return the head to a position over the sacrum and maintain the LoG within the base of support.

their structure to the required function, as can be seen in their large areas of attachment, their length, and their large cross-section. The alignment of the hip joint muscles and the large ROM available at the hip joint result in muscle functions that are strongly influenced by hip joint position. For example, the adductor muscles may be hip flexors in the neutral hip joint but will be hip extensors when the hip joint is already flexed.[98] Delp and colleagues[99] used computer modeling to determine that the torque-generating capability of the medial rotators increased with increased hip flexion, whereas the torque-generating capacity of the lateral rotators decreased with increasing hip flexion. They similarly determined that the **piriformis muscle** was a lateral rotator at 0° of hip flexion but a medial rotator at 90° of hip flexion. Such inversions of function are found in a few muscles at the shoulder (the clavicular portion of the pectoralis major, for example), but are fairly common in the hip joint. As a consequence, results of various studies may appear to be contradictory, but, in fact, testing conditions explain differing results. Some gender-related differences also have been found that explain differential findings.[100]

It is best to examine muscle action at the hip joint in the context of specific functions such as single-limb support, posture, and gait. The next section will briefly review muscle function, but we will leave more detailed analyses for later in this and other chapters. Although the traditional action of each muscle on the distal femoral segment is described for the most part, it must be emphasized that any of the muscles are as likely (or more likely) to produce joint action by moving the proximal pelvic segment instead.

Flexors

The flexors of the hip joint function primarily as mobility muscles in open-chain function; that is, they function primarily to bring the swinging limb forward during ambulation or in various sports-specific movements. The flexors may function secondarily to resist strong hip extension forces that occur as the body passes over the weight-bearing foot. Nine muscles have action lines crossing the anterior aspect of the hip joint. Of these, the primary muscles of hip flexion are the **iliopsoas,** rectus femoris, tensor fascia lata, and **sartorius.** The iliopsoas muscle is considered to be the most important of the primary hip flexors. It consists of two separate muscles, the **iliacus** muscle and the **psoas major** muscle, both of which attach to the femur by a common tendon. The two components of the iliopsoas muscle have many points of origin, including the iliac fossa and the disks, bodies, and transverse processes of the lumbar vertebrae. Given the attachments of the psoas major muscle to the anterior vertebrae and the iliacus muscle to the iliac fossa, activity of or passive tension in these muscles would anteriorly tilt the pelvis (iliacus muscle) and, apparently, pull the lumbar vertebrae anteriorly into flexion (psoas major muscle). In closed-chain function (head vertical), however, these muscles seem to create a paradoxical lumbar lordosis (lumbar extension) that results from the body's attempt to keep the head over the sacrum with anterior pelvic tilt and lower lumbar flexion. The role of the iliopsoas muscle in hip flexion may be particularly critical when active hip flexion from a sitting position is required. Smith and colleagues[101] proposed that the hip cannot be flexed beyond 90° when the iliopsoas muscle is paralyzed, because the other hip flexor muscles are effectively actively insufficient in that position. Basmajian and DeLuca[98] summarized the often contradictory evidence of many investigations by concluding that both segments of the iliopsoas muscle are active in various stages of hip flexion. The moment arm of the iliopsoas muscle for medial or lateral rotation is very small and probably not functionally relevant.[98,99]

The rectus femoris muscle is the only portion of the quadriceps muscle that crosses both the hip joint and knee joint. It originates on the anterior inferior iliac spine and inserts by way of a common tendon into the tibial tuberosity. The rectus femoris muscle flexes the hip joint and extends the knee joint. Because it is a two-joint hip flexor, the position of the knee during hip flexion will affect its ability to generate force at the hip. Simultaneous hip flexion and knee extension considerably shorten this muscle and increase the likelihood of active insufficiency. Consequently, the rectus femoris muscle makes its best contribution to hip flexion when the knee is maintained in flexion.

The sartorius muscle is a strap-like muscle originating on the anterior superior iliac spines. It crosses the anterior aspect of the femur to insert into the upper portion of the medial aspect of the tibia. The sartorius muscle is considered to be a flexor, abductor, and lateral rotator of the hip, as well as a flexor and medial rotator of the knee. Wheatley and Jahnke[102] proposed that the sartorius muscle, although a two-joint muscle, should be relatively unaffected by the position of the knee, given the relatively small proportional change in length with increased knee flexion. Its function is probably most important when the knee and hip need to be flexed simultaneously (as in climbing stairs), but its small cross-section argues against a unique or critical role at the hip joint.[16]

The tensor fascia lata muscle originates more laterally than the sartorius muscle, on the anterolateral lip of the iliac crest. The muscle fibers insert into the iliotibial band about one fourth of the way down the lateral aspect of the thigh. The iliotibial band or iliotibial tract is the thickened lateral portion of the **fascia lata** of the hip and thigh. The iliotibial band attaches proximally to the iliac crest lateral to the tensor fascia lata and **gluteus maximus** muscle.[103] The iliotibial band continues distally on the lateral thigh to insert into the lateral condyle of the tibia. The tensor fascia lata muscle is considered to flex, abduct, and medially rotate the femur at the hip,[98] although the tensor fascia lata's contribution to hip abduction may be dependent on simultaneous hip flexion.[104] The most important contribution of the tensor fascia lata muscle may be in maintaining tension in the iliotibial band, in combination with the gluteus maximus. The iliotibial band assists in relieving the femur of some of the tensile stresses imposed on the shaft by weight-bearing forces.[104,105] Because bone more effectively resists compressive than tensile stresses, reduction of tensile stresses is important in maintaining integrity of the bone.

Although the importance of the tensor fascia lata muscle and iliotibial band is controversial, evidence suggests they contribute to stability of the hip joint.[103] Excessive tension in the iliotibial band may also contribute to reduced hip adduction ROM when the hip is extended. Gajdosik and colleagues[106] performed the **Ober test,** presumed to test tension in the iliotibial band, on men and women without impairments. They found an average passive hip adduction of 9° for men and 4° for women when both the hip and knee were extended. When the knee was flexed during the maneuver, the hip remained in 4° of abduction for men and 6° of abduction for women, which implied that there was greater tension in the lateral hip joint structures (potentially with the iliotibial band as a key factor) when the knee was flexed.[106] The Ober test presumably moves the iliotibial band from its position anterior to the greater trochanter to a position posterior to the greater trochanter by extending the hip. Movement of the iliotibial band anteriorly and posteriorly over the greater trochanter during functional activities has been implicated in **"snapping hip" syndrome** and in inflammation of the **trochanteric bursae.**[107]

The secondary hip flexors are the **pectineus, adductor longus, adductor magnus,** and the gracilis muscles. These muscles are described in the next section because they are predominantly adductors of the hip. Each, however, is capable of contributing to hip joint flexion, but that contribution is dependent on hip joint position. Kapandji[51] noted that these muscles contribute to flexion only up to 40° to 50° of hip flexion. Once the femur is superior to the point of origin of a muscle, the muscle will become an extensor of the hip joint. The gracilis, a two-joint muscle, is active as a hip flexor when the knee is extended but not when the knee is flexed.[102]

Adductors

The hip adductor muscle group is generally considered to include the pectineus, **adductor brevis,** adductor longus, adductor magnus, and the gracilis muscles. The adductors are located anteromedially. The adductors longus, brevis, and magnus muscles arise in a group from the body and inferior ramus of the pubis to insert along the linea aspera of the femur. The gracilis muscle is the only two-joint adductor. It originates on the symphysis pubis and pubic arch and inserts on the medial surface of the shaft of the tibia.

The contribution of the adductor muscles to hip joint function has been debated for many years. One of the reasons for debate is a question concerning the degree to which the flexed, adducted, and medially rotated posture assumed by many individuals with cerebral palsy is attributable to adductor spasticity. Arnold and Delp[46] (using kinematic data from children with cerebral palsy and excessive medial rotation of the hip, and a "deformable femur" model) concluded that, in the normal hip in standing, the adductor brevis, adductor longus, pectineus, and posterior adductor magnus muscles had only small moment arms for medial rotation, whereas the gracilis and anterior adductor magnus muscles had small moment arms for lateral rotation. With excessive femoral anteversion, the moment arms of the adductor brevis, pectineus and the middle adductor magnus muscles switched from medial rotational to lateral rotational lines of pull. After examining the changes in moment arms with femoral anteversion or combined hip medial rotation and knee flexion, the investigators concluded that the adductors were unlikely to have a strong influence on the medially rotated hip position during the gait cycle.[46]

Basmajian and DeLuca[98] believed that the adductors function not as prime movers but by reflex response to gait activities. As shall be seen in our discussion of muscle function in bilateral stance, the adductors may be synergists to the abductor muscles when both feet are on the ground, enhancing side-to-side stabilization of the pelvis. Although the role of the adductor muscles may be less clear than that of other hip muscle groups, the relative importance of the adductors should not be underestimated. The adductors as a group contribute 22.5% to the total muscle mass of the lower extremity, in comparison with only 18.4% for the flexors and 14.9% for the abductors.[108] The adductors are relevant in sports-related injuries because of the high frequency of adductor longus strains, particular in sports such as hockey.[109]

Extensors

The one-joint gluteus maximus muscle and the two-joint hamstrings muscle group are the primary hip joint extensors. These muscles may receive assistance from the posterior fibers of the **gluteus medius,** from the posterior adductor magnus muscle, and from the piriformis muscle. The gluteus maximus is a large, quadrangular muscle that originates from the posterior sacrum, dorsal sacroiliac ligaments, sacrotuberous ligament, and a small portion of the ilium. The gluteus maximus crosses the sacroiliac joint before its most superior fibers insert into the iliotibial band (as do the fibers of the tensor fascia lata muscle) and its inferior fibers insert into the gluteal tuberosity. The gluteus maximus is the largest of the lower extremity muscles; this muscle alone constitutes 12.8% of the total muscle mass of the lower extremity.[108] The maximus is a strong hip extensor that appears to be active primarily against a resistance greater than the weight of the limb. Its moment arm for hip extension is considerably longer than that of either the hamstrings or the adductor magnus muscles and is maximal in the neutral hip joint position.[100] A favorable length-tension relationship, however, allows it to exert its peak extensor moment at 70° of hip flexion.[110] The segments of the maximus have a substantial capacity to laterally rotate the femur, although the moment arms for lateral rotation diminish with increased hip flexion.[99]

The three two-joint extensors are the long head of the **biceps femoris,** the **semitendinosus,** and the **semimembranosus** muscles, known collectively as the hamstrings. Each of these three muscles originates on the ischial tuberosity. The biceps femoris crosses the posterior femur to insert into the head of the fibula and lateral aspect of the lateral tibial condyle. The other two hamstrings insert on the medial aspect of the tibia. All three muscles extend the hip with or without resistance, as well as serving as important knee flexors. The hamstrings increase their moment arm for hip extension as the hip flexes to 35° and decrease it thereafter. This is somewhat in contrast to the moment arm of the gluteus maximus that is maximal at neutral position and decreases with any hip flexion thereafter.[100] Regardless of these changes in moment arm with joint position, the moment arm of the combined hamstrings for hip extension is smaller than that of the gluteus maximus at all points in the hip flexion/extension ROM. As two-joint muscles, the role of the hamstrings in hip extension is also strongly influenced by knee position. Chleboun and colleagues[111] (using ultrasonography) determined that the moment arm for the long head of the biceps femoris was greater for hip extension than for knee flexion, with hip position affecting its excursion capability more than did knee position. Although these investigators reported only on the long head of the biceps femoris, the anatomy of the medial hamstrings (semimembranosus and semitendinosus) makes it likely that these muscles have similar attributes. If the hip is extended and the knee is flexed to 90° or more, the hamstrings may not be able to contribute much to hip extension force because of active insufficiency or approaching active insufficiency. Extension forces in the hip increase by 30% if the knee is extended during hip extension.[110] The optimal length-tension relationship for the long head of the biceps is estimated to be at 90° of hip flexion and 90° of knee flexion,[111] and it is likely that the medial hamstrings show similar behavior. The medial hamstrings have a small moment arm for medial rotation in the neutral hip but appear to switch to lateral rotators with hip or knee flexion.[46] The biceps femoris appears to contribute to lateral rotation of the hip.[98]

Abductors

The abductors have been likened to the "rotator cuff of the hip," with the gluteus medius and **gluteus minimus** being analogous to the supraspinatus and subscapularis muscles, respectively.[112–114] The medius tendon inserts on the lateral and posterior superior portion of the greater trochanter, resulting in a moment arm similar in direction and force to the supraspinatus.[115] Active abduction of the hip is brought about predominantly by the gluteus medius and the gluteus minimus muscles. The superior fibers of the gluteus maximus and the sartorius muscles may assist when the hip is abducted against strong resistance. The tensor fascia lata muscle is given variable credit for its contribution and may be effective as an abductor only during simultaneous hip flexion. The gluteus medius originates on the lateral surface of the wing of the ilium and inserts into the greater trochanter, beneath the gluteus maximus. The gluteus medius has anterior, middle, and posterior parts that function asynchronously during movement at the hip.[116] Analogous to the deltoid muscle of the glenohumeral joint, the anterior fibers of the gluteus medius are active in hip flexion, whereas the posterior fibers function during extension. In the neutral hip, the posterior portion of the medius will produce a lateral rotational moment, whereas the middle and anterior portions have small medial rotational moments. In hip flexion, all portions medially rotate the hip.[99] All portions of the muscle abduct, regardless of hip joint position.

The gluteus minimus muscle lies deep to the gluteus medius, arising from the outer surface of the ilium with its fibers converging on an aponeurosis that ends in a tendon on the greater trochanter. The minimus is consistently an abductor and flexor of the hip, with its rotator function dependent on hip position. However, the minimus is a medial rotator in hip flexion.[117] There appears to be consensus that the gluteus minimus commonly has a tendinous insertion into the joint capsule as it passes to the greater trochanter. It is hypothesized that this attachment retracts the capsule during hip abduction to prevent entrapment,[118] or tightens the capsule to add to the gluteus minimus's primary function of stabilizing the femoral head in the acetabulum.[117] The minimus inserts on the anterior portion of the greater trochanter and can exert several different moments, including flexion, abduction, medial rotation, or lateral rotation, depending on the position of the femur. Based on these observations, it follows that one of the primary functions of the minimus is to act as a femoral head stabilizer.[119]

The gluteus minimus and medius muscles function together either to abduct the femur (distal level free) or, more important, to stabilize the pelvis (and superimposed HAT) in unilateral stance against the effects of gravity. As will be presented later, the gluteus medius and minimus muscles will offset the gravitation adduction torque on the pelvis (pelvis drop) around the weight-bearing hip in unilateral stance. The abductors are physiologically designed to work most effectively in a neutral or slightly adducted hip (slightly lengthened abductors).[120,121] Isometric abduction torque in the neutral hip position is 82% greater than abduction torque when the hip is in 25° of abduction (shortened abductors).[122] Because the gluteus medius is an important pelvic stabilizer, its weakness has been implicated in lower extremity overuse injuries such as bursitis and hip osteoarthritis, as well as disorders at the knee.[123–125]

Continuing Exploration 10-6:

Trochanteric Bursae

The greater trochanter has become the focus of increased interest as lateral hip pain syndromes among both the elderly and athletes are diagnosed more often.[22,62,63] Although a number of possible pathologies of both intra-articular and extra-articular origin are probably involved, there is consensus that the bursae around the greater trochanter are commonly implicated. There does not appear to be consensus on how many discrete bursae there are or how to name them. Pfirrmann and colleagues[62] used MRI, bursography, and conventional radiography to study the greater trochanter and its bursae in cadavers and asymptomatic volunteers. They concluded that the greater trochanter consisted of four facets. The gluteus minimus attached to the anterior facet, with the subgluteus minimus bursa beneath the tendon; the gluteus medius attached to the posterosuperior and lateral facets, with the subgluteus medius bursa beneath the tendon at the lateral facet; and the large trochanteric bursa covered the posterior facet, which was free of tendinous attachments (Fig. 10–28). Presumably, the trochanteric bursa serves to reduce friction between the posterior facet and the overlying gluteus maximus, as well as between the iliotibial band and the trochanter.

CASE APPLICATION

Lateral Hip Pain

case 10–5

In addition to her other problems, Gabriella was complaining of lateral hip pain that was tender to palpation. Although other explanations for her pain exist, greater trochanter pain syndrome is a common complaint in middle aged women (with a 4:1 ratio of women to men) and in those diagnosed with osteoarthritis.[22,63,126] The altered biomechanics associated with her leg length discrepancy may further predispose her to pathology of the lateral hip. Trochanteric bursitis and lesions of the abductor (gluteus medius and gluteus minimus) tendons commonly coexist and have been analogized to rotator cuff tears and bursitis in the shoulder.[22,62,63,127] As we continue to explore the role of the hip abductors in standing and the possible effects of hip dysplasia on the hip abductors, it will become clear that Gabriella's hip abductors and associated bursae are likely at risk for overuse injury and perhaps the start of degenerative changes.

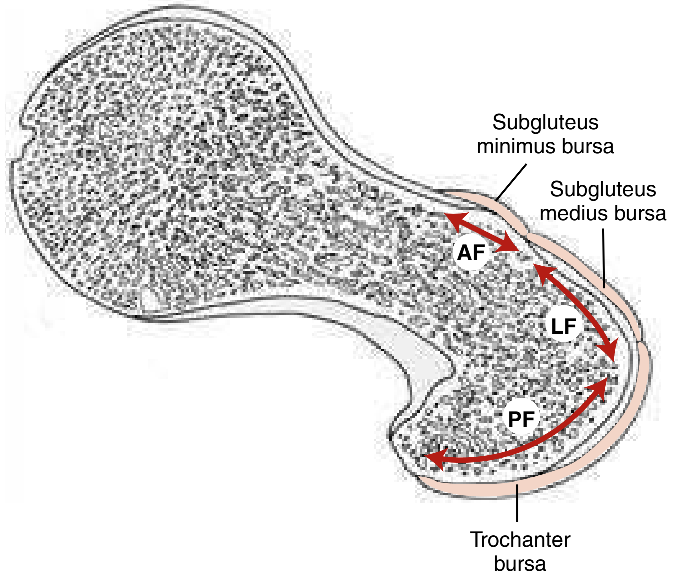

Figure 10–28 The greater trochanter has four facets, three of which can be seen in this horizontal cross-section: the anterior facet, the lateral facet, and the posterior facet. The posterosuperior facet is not seen. Also seen on cross-section are three bursae. *(From Pfirrmann C, Chung C, Theumann B, et al: Greater trochanter of the hip: Attachment of the abductor mechanism and a complex of three bursae—MR imaging and MR bursography in cadavers and MR imaging in asymptomatic volunteers. Radiology 221:469, 2001.)*

Lateral Rotators

Six short muscles have lateral rotation as a primary function. These muscles are the **obturator internus** and **externus,** the **gemellus superior** and **inferior,** the **quadratus femoris,** and the piriformis muscles. Other muscles that have fibers posterior to the axis of motion at the hip (the posterior fibers of the gluteus medius and minimus and the gluteus maximus) may produce lateral rotation combined with the primary action of the muscle (although it has already been noted that the lateral rotary function of these muscles decreases or becomes medial with increased hip flexion).[99] Of the primary lateral rotators, each inserts either on or in the vicinity of the greater trochanter (Fig. 10–29). The obturator internus muscle originates from the inside (posterior aspect) of the obturator foramen and emerges through the lesser sciatic foramen to insert on the medial aspect (inside) of the greater trochanter. The gemellus superior and gemellus inferior muscles arise from the ischium of the pelvis, just above and just below the point at which the obturator internus passes through the lesser sciatic notch. Both gemelli follow and blend with the obturator internus tendon to insert with the internus tendon into the greater trochanter.

The obturator externus muscle is sometimes considered to be an anteromedial muscle of the thigh because it originates on the external (anterior) surface of the obturator foramen. However, it crosses the posterior aspect of the hip joint and inserts on the medial aspect of the greater trochanter in the trochanteric fossa. The quadratus femoris muscle is a small quadrangular muscle that originates on the ischial tuberosity and inserts on the posterior femur

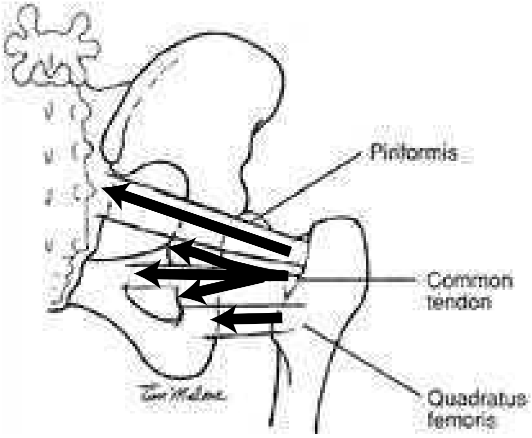

Figure 10–29 The lateral rotators of the hip joint have action lines that lie nearly perpendicular to the femoral shaft (making them excellent rotators) and parallel to the head and neck of the femur (making them excellent compressors). The common tendon is the shared insertion of the gemellus superior, gemellus inferior, and the obturator internus muscles. The obturator externus muscle is not shown.

between the greater and lesser trochanters. The piriformis muscle originates largely on the anterior surface of the sacrum, passes through the greater sciatic notch, and follows the inferior border of the posterior gluteus medius to insert above the other lateral rotators into the medial aspect of the greater trochanter. The piriformis and gluteus maximus are the only two muscles that cross the sacroiliac joint. The sciatic nerve, the largest nerve in the body, enters the gluteal region just inferior to the piriformis muscle as both structures pass through the greater sciatic notch.

The lateral rotator muscles are positioned to perform their rotary function effectively, given the nearly perpendicular orientation to the shaft of the femur (see Fig. 10–29). However, exploration of function of these muscles has been restricted because of the relatively limited access to electromyography (EMG) surface or wire electrodes. Like their rotator cuff counterpart at the glenohumeral joint, these muscles would certainly appear to be effective joint compressors because their combined action line parallels the head and neck of the femur. Using modeling, Delp and colleagues[99] determined that the obturator internus, like the gluteal muscles, decreased its moment arm for lateral rotation with increased hip flexion. The piriformis was estimated to have a large moment arm for lateral rotation with the hip joint at 0° but switched to a medial rotator with half the moment arm when the hip reached 90° of flexion. The obturator externus and quadratus femoris were the only lateral rotators that did not diminish their moment arm for lateral rotation with increased hip joint flexion.[99] Hypothetically, the lines of pull of the deep one-joint lateral rotators should make them ideal tonic stabilizers of the joint during most weight-bearing and non-weightbearing hip joint activities. Although their ability to perform lateral rotation may decrease with hip flexion in some instances, the action lines of these muscles should remain largely compressive (parallel to the femoral neck) throughout the hip joint ROM.

Medial Rotators

There are no muscles with a *primary* function of producing medial rotation of the hip joint. The more consistent medial rotators are the anterior portion of the gluteus medius, gluteus minimus, and the tensor fascia lata muscles. Although the role of the adductors in hip joint rotation is controversial, the weight of evidence appears to support the adductor muscles as medial rotators of the joint,[98,101] with the possible exception of the gracilis muscle.[101] The ability of hip joint muscles to shift function with changing position of the hip joint is evident when medial rotation of the hip is examined. There is a trend toward increased medial rotation torques (or decreased lateral rotation torques) with increased hip flexion among many of the hip joint muscles,[46,99] with three times more medial rotation torque in the flexed hip than in the extended hip.[101] Delp and colleagues,[99] although clear as to the limitations of their modeling, suggested that the medial rotation that accompanies a "crouched" gait seen in many individuals with cerebral palsy may be attributable more to hip flexion than to adductor spasticity.

Continuing Exploration 10-7:

Rotator Cuff of the Hip

The shoulder rotator cuff can be used to help understand the function of muscles around the hip. External rotation of the shoulder is performed by the infraspinatus, teres minor, and suprasinatus and is compared to the piriformis, obturator externus and internus, superior and inferior gemelli, as well as the posterior gluteus medius at the hip. Internal rotation of the shoulder is performed by the subscapularis compared to the anterior fibers of the gluteus medius and gluteus minimus. Abduction of the shoulder is performed by the supraspinatus and is compared to the gluteus medius of the hip.[128]

HIP JOINT FORCES AND MUSCLE FUNCTION IN STANCE

Bilateral Stance

In erect bilateral stance, both hips are in neutral or slight hyperextension, and weight is evenly distributed between both legs. The line of gravity falls just posterior to the axis for flexion/extension of the hip joint. The posterior location of the line of gravity creates an extension moment of force around the hip that tends to tilt the pelvis posteriorly on the femoral heads. The gravitational extension moment is largely checked by passive tension in the hip joint capsuloligamentous structures, although slight or intermittent activity in the iliopsoas muscles in relaxed standing may assist the passive structures.[98]

In the frontal plane during bilateral stance, the superincumbent body weight is transmitted through the sacroiliac joints and pelvis to the right and left femoral heads. Hypothetically, the weight of the HAT (two thirds of body weight) should be distributed so that each femoral head receives approximately half of the superincumbent weight.[129] As shown in Figure 10–30, the joint axis of each hip lies at an equal distance from the line of gravity of HAT; that is, the gravitational moment arms for the right hip (DR) and the left hip (DL) are equal. Because the body weight (W) on each femoral head is the same (WR = WL), the magnitude of the gravitational torques around each hip must be identical (WR × DR = WL × DL). The gravitational torques on the right and left hips, however, occur in opposite directions. The weight of the body acting around the right hip tends to drop the pelvis down on the left (right adduction moment), whereas the weight acting around the left hip tends to drop the pelvis down on the right (left adduction moment). These two opposing gravitational moments of equal magnitude balance each other, and the pelvis is maintained in equilibrium in the frontal plane without the assistance of active muscles. Assuming that muscular forces are not required to maintain either sagittal or frontal plane stability at the hip joint in bilateral stance, the compression across each hip joint in bilateral stance should simply be half the superimposed body weight (or one third of HAT to each hip).

Hip joint compression can be altered by different means. This includes leaning over the lower extremity during single supported activity and the use of a cane either ipsilaterally or contralaterally. The magnitude of hip joint compression as it relates to bilateral stance, unilateral stance, compensatory leaning, ipsilateral cane use, and contralateral cane use is presented in Examples 10-4, 10-5, 10-6, 10-7, and 10-8, respectively. A summary of the calculations is presented after the examples in Table 10–2 for direct comparison.

Example 10-4

Calculating Hip Joint Compression in Bilateral Stance

Using a hypothetical case of someone weighing 825 N (~185 lb), the weight of HAT (2/3 body weight) will be 550 N (~124 lb). Of that 550 N, half will presumably be distributed through each hip. Because we are assuming no additional compressive force produced by hip muscle activity, the total hip joint compression at each hip in bilateral stance is estimated to be 225 N (~50 lb); that is, total hip joint compression through each hip in bilateral stance is one third of body weight.

The rationale presented in Example 10-4 for assuming that each hip receives one third of body weight in bilateral stance is reasonable. However, Bergmann and colleagues[130] showed in several subjects with an instrumented pressure-sensitive hip prostheses that the joint compression across each hip in bilateral stance was 80% to 100% of body weight, rather than one third (33%) of body weight, as commonly proposed. When they added a symmetrically distributed load to the subject's trunk, the forces at both hip joints increased by the full weight of the load, rather than by half of the superimposed load as might be expected. Although the mechanics of someone standing who has a prosthetic hip may not fully represent normal hip joint forces, the findings of Bergmann and colleagues call into question the simplistic view of hip joint forces in bilateral stance. The slight activity in the iliopsoas muscle may account for more joint compression than previously thought. Alternatively, capsuloligamentous tension may contribute to joint compression.

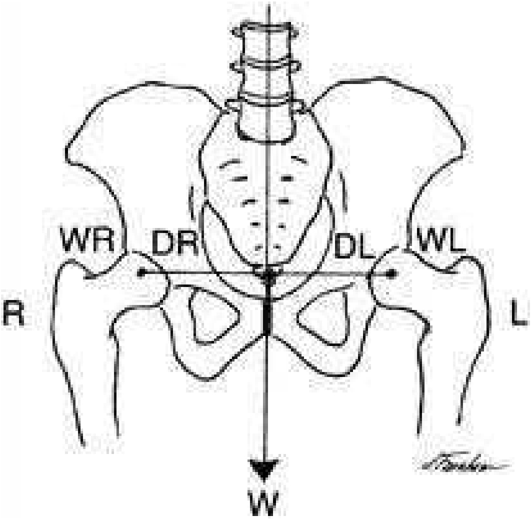

Figure 10–30 An anterior view of the pelvis in normal erect bilateral stance. The weight acting on the right hip joint (WR) multiplied by the distance from the right hip joint axis to the body's center of gravity (DR) is equal to the weight acting at the left hip joint (WL) multiplied by the distance from the left hip to the body's center of gravity (DL). Therefore, WR × DR = WL × DL.

When bilateral stance is not symmetrical, frontal plane muscle activity will be necessary either to control the side-to-side motion or to return the hips to symmetrical stance. If the pelvis is shifted to the right (see Fig. 10–22), there is a relative adduction of the right hip and abduction of the left hip. To return to neutral position, an active contraction of the right hip abductors would be expected. However, a contraction of the left hip adductors would accomplish the same goal. Thus, in bilateral stance when both lower limbs bear at least some of the superimposed weight, the contralateral abductors and adductors may function as synergists to control the frontal plane motion of the pelvis. In unilateral stance, activity of the adductors either in the weight-bearing or non-weightbearing hip *cannot* contribute to stability of the stance limb. Hip joint stability in unilateral stance is the sole domain of the hip joint abductors. In the absence of adequate hip abductor function, the adductors can contribute to stability—but only in bilateral stance.

Unilateral Stance

In Figure 10–31, the left leg has been lifted from the ground and the full superimposed body weight (HAT) is being supported by the right hip joint. Rather than sharing the compressive force of the superimposed body weight with the left limb, the right hip joint must now carry the full burden. In addition, the weight of the non-weightbearing left limb that is hanging on the left side of the pelvis must

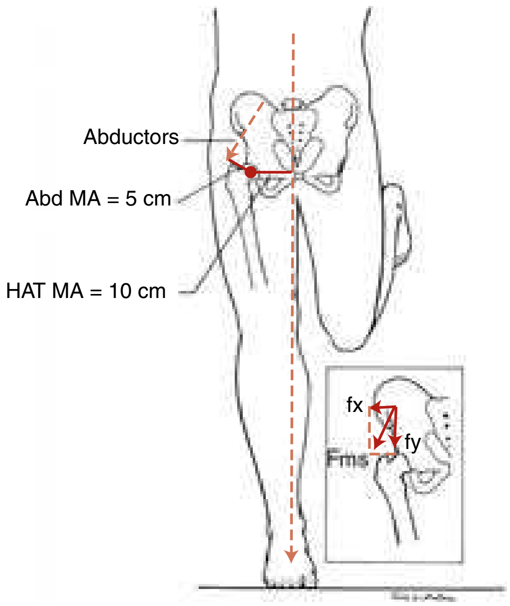

Figure 10–31 In right unilateral stance, the weight of HAT acts 10 cm from the right hip joint. The 10-cm moment arm slightly underestimates the location of the LoG because it does not account for the weight of the hanging left limb. The hip abductors have a moment arm of approximately 5 cm. Inset. The pull of the abductors (F_{ms}) on the horizontally oriented pelvis will resolve into a parallel component (Fx) that will pull the acetabulum into the center of the femoral head and a perpendicular component (Fy) that will pull the pelvis down on the superior aspect of the femoral head, as well as pull the ilium closer to the femur, producing a hip abduction torque.

be supported along with the weight of HAT. Of the one third of the portion of body weight found in the lower extremities, the non-weightbearing limb must account for half of that, or one sixth of the full body weight.[131] The magnitude of body weight (W) compressing the right hip joint in right unilateral stance, therefore, is

$$\text{Right hip joint compression}_{\text{body weight}} = [2/3 \times W] + [1/6 \times W]$$
$$\text{Right hip joint compression}_{\text{body weight}} = 5/6 \times W$$

In our hypothetical subject from Example 10-4 who weighs 825 N, HAT accounts for 550 N. One lower extremity weighs one sixth of body weight, or 137.5 N. Therefore, when this individual lifts one leg off the ground, the supporting hip joint will undergo 687.5 N (or five sixths of body weight)[6] of compression *from body weight alone.*

Although we have accounted for the increase in hip joint compression from body weight as a person moves from double-limb support (bilateral stance) to single-limb support, the problem is more complex. Not only is the hip joint in unilateral stance being compressed by body weight (gravity), but also that body weight is concomitantly creating a torque around the hip joint.

The force of gravity acting on HAT and the non-weightbearing left lower limb (HATLL) will create an adduction torque around the weight-bearing hip joint; that is, gravity will attempt to drop the pelvis around the right weight-bearing hip joint axis. The abduction countertorque will have to be supplied by the hip abductor musculature. The result will be joint compression or a joint reaction force that is a combination of both body weight and abductor muscular compression. The total joint compression can be calculated for our hypothetical 825-N subject. The line of gravity of HATLL can be estimated to lie 10 cm (0.1 m, or ~4 in.) from the right hip joint axis. That is, the moment arm (MA) = 0.1 m, although the actual distance will vary among individuals.[132] The 10-cm estimate of the gravitational moment arm is for symmetrical stance. The actual moment arm is likely to be slightly greater because the weight of the hanging left leg will pull the center of gravity of the superimposed weight slightly to the left, although the line of gravity will simultaneously be shifted slightly right to get the line of gravity within the single foot base of support.

Example 10-5

Calculating Hip Joint Compression in Unilateral Stance

For simplicity, the possible increase in the moment arm of HATLL from the non-weightbearing limb will be ignored, so our torque calculation here is likely to *underestimate* the actual gravitational torque. In our simplified hypothetical example, the magnitude of the gravitational adduction torque at the right hip will be as follows:

$$\text{HATLL torque}_{\text{adduction}}: 687.5\ \text{N} \times 0.1\ \text{m} = 68.75\ \text{Nm}$$

To maintain the single-limb support position, there must be a countertorque (abduction moment) of equivalent magnitude. The countertorque must be produced by the force of the hip abductors (gluteus medius, minimus, and tensor fascia lata muscles) acting on the pelvis. Assuming that the abductor muscles act through a typical moment arm of 5 cm (0.05 m, or 2 in.)[78] and knowing that the muscles must generate an abduction torque equivalent to the adduction torque of gravity (68.75 Nm), we can solve for the magnitude of muscle contraction (F_{ms}) needed to maintain equilibrium in our hypothetical example.

$$\text{Torque}_{\text{abduction}}: 68.75\ \text{Nm} = F_{ms} \times 0.05\ \text{m}$$
$$F_{ms} = 68.75\ \text{Nm} \div 0.05\ \text{m} = 1{,}375\ \text{N}$$

Assuming that all the abductor muscular force is transmitted through the acetabulum to the femoral head, the 1,375-N abductor muscular compressive force is now added to the 687.5 N of compression caused by body weight passing through the supporting hip. Thus, the total hip joint compression, or joint reaction force, at the stance hip joint in unilateral support can be estimated for our hypothetical subject at:

$$\begin{array}{r} 1{,}375\ \text{N abductor joint compression} \\ +\ 687.5\ \text{N body weight (HATLL) compression} \\ \hline 2{,}062.5\ \text{N total joint compression} \end{array}$$

The hypothetical figures used in the examples oversimplify and underestimate the forces involved in hip joint stresses as already noted. Total hip joint compression or joint reaction forces are generally found to be two to three times body weight in unilateral stance.[110,129,133] Some investigators have found peak forces are higher than typically calculated for unilateral stance and can achieve four times body weight depending on measurement technique and individual characteristics.[134,135] Individual differences in hip structure, (i.e., angulation of the femur) may substantially alter forces and pressures across the hip joint.[57,80,136]

If total joint compression in unilateral stance is approximately three times body weight, a loss of 1 N (~4.5 lb) of body weight will reduce the joint reaction force by 3 N (13.5 lb). For painful hip joints, however, the reductions in compression generally required are greater than can be realistically achieved through weight loss. The solution must be in a reduction of abductor muscle force requirements. If less muscular countertorque is needed to offset the effects of gravity, there will be a decrease in the amount of *muscular compression* across the joint, although the body weight compression will remain unchanged. Several options are available when there is a need to decrease abductor muscle force requirements. Some compression reduction strategies occur automatically, but at a cost of extra energy expenditure and structural stress. Other strategies require intervention such as assistive devices but minimize the energy cost.

Compensatory Lateral Lean of the Trunk

Gravitational torque at the pelvis is the product of body weight and the distance that the line of gravity lies from the hip joint axis (its moment arm). If there is a need to reduce the torque of gravity in unilateral stance and if body weight cannot be reduced, the moment arm of the gravitational force can be reduced by laterally leaning the trunk over the pelvis toward the side of pain or weakness when in unilateral stance on the painful limb. Although leaning toward the side of pain might appear counterintuitive, the compensatory lateral lean of the trunk toward the painful stance limb will swing the line of gravity closer to the hip joint, thereby reducing the gravitational moment arm. Because the weight of HATLL must pass through the weight-bearing hip joint regardless of trunk position, leaning toward the painful or weak supporting hip does not increase the joint compression caused by body weight. However, it does reduce the gravitational torque. If there is a smaller gravitational adduction torque, there will be a proportional reduction in the need for an abductor countertorque. Although it is theoretically possible to laterally lean the trunk enough to bring the line of gravity through the weight-bearing hip (reducing the torque to zero) or to the opposite side of the supporting hip (reversing the direction of the gravitational torque), these are relatively extreme motions that require high energy expenditure and would result in excessive wear and tear on the lumbar spine. More energy efficient and less structurally stressful compensations can still yield dramatic reductions in the hip abductor force.

Example 10-6

Calculating Hip Joint Compression With Lateral Lean

Returning to our hypothetical subject weighing 825 N, let us assume that he can laterally lean to the right enough to bring the line of gravity within 2.5 cm (0.025 m) of the right hip joint axis (Fig. 10–32). The gravitational adduction torque would now be

$$\text{HATLL torque}_{\text{adduction}} = [5/6\ (825\ \text{N})] \times 0.25\ \text{m}$$
$$\text{HATLL torque}_{\text{adduction}} = 17.2\ \text{Nm}$$

If only 17.2 Nm of adduction torque were produced by the superimposed weight, the abductor force needed would be as follows:

$$\text{Torque}_{\text{abduction}}\text{: } 17.2\ \text{Nm} = F_{ms} \times 0.05\ \text{m}$$
$$F_{ms}\text{: } 17.2\ \text{Nm} \div 0.05\ \text{m} = 343.75\ \text{N}$$

If only ~344 N (~77 lb) of abductor force were required, the total hip joint compression in unilateral stance using the compensatory lateral lean would now be

$$\begin{array}{r} 343.75\ \text{N abductor joint compression} \\ +\ 687.5\ \text{N body weight (HATLL) compression} \\ \hline 1{,}031.25\ \text{N total joint compression} \end{array}$$

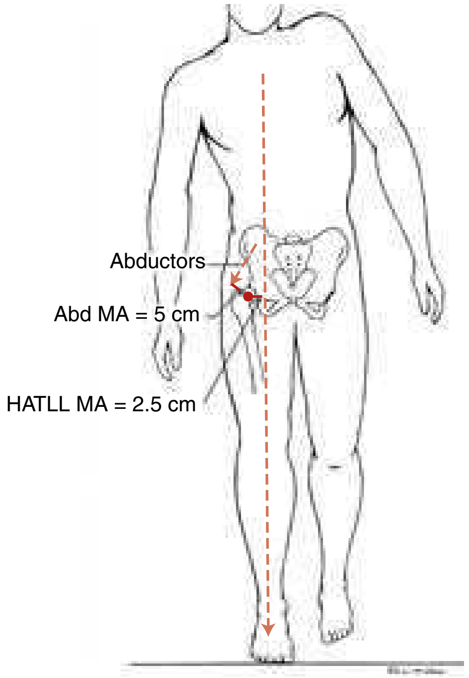

Figure 10–32 When the trunk is laterally flexed toward the stance limb, the moment arm of HATLL is substantially reduced (e.g., 2.5 cm, in comparison with 10 cm with the neutral trunk), whereas that of the abductors remains unchanged (e.g., 5 cm). The result is a substantially diminished torque from HATLL and, consequently, a substantially decreased need for hip abductor force to generate a countertorque.

The 1,031.25-N joint reaction force estimated in Example 10-6 is half the 2,062.5 N of hip joint compression previously calculated for our hypothetical subject in single-limb support (see Table 10–2). This 50% reduction in joint compression is enough to relieve some of the pain symptoms experienced by a person with arthritic changes in the hip joint or to provide some relief to a weak or painful set of abductors. The compensatory lean is instinctive and commonly seen in people with hip joint disability.

Continuing Exploration 10-8:

Pathological Gaits

When a lateral trunk lean is seen during gait and is due to hip abductor muscle weakness, it is known as a **gluteus medius gait.** If the same compensation is due to hip joint pain, it is known as an **antalgic gait.** In some instances, the 344-N abductor force we calculated as necessary to stabilize the pelvis in Example 10-6 is still beyond the work capacity of very weak or completely paralyzed hip abductors. In such cases of extreme abductor muscle weakness, the pelvis will drop to the unsupported side even in the presence of a lateral trunk lean to the supported side. If lateral lean and pelvic drop occur during walking, the gait deviation is commonly referred to as a **Trendelenburg gait.** The lateral lean that accompanies the drop of the pelvis must be sufficient to keep the line of gravity within the supporting foot.

Whether a lateral trunk lean is due to muscular weakness or pain, a lateral lean of the trunk during walking still uses more energy than required for normal single-limb support and may result in stress changes within the lumbar spine if used over an extended time period. Use of a cane or some other assistive device offers a realistic alternative to the person with hip pain or weakness.

Use of a Cane Ipsilaterally

Pushing downward on a cane held in the hand on the side of pain or weakness should reduce the superimposed body weight by the amount of downward thrust; that is, some of the weight of HATLL would follow the arm to the cane, rather than arriving on the sacrum and the weight-bearing hip joint. Inman and colleagues[110] suggested that it is realistic to expect that someone can push down on a cane with approximately 15% of his body weight. The proportion of body weight that passes through the cane will not pass through the hip joint and will not create an adduction torque around the supporting hip joint.

Total hip joint compression of 1,691.35 N calculated in Example 10-7 when a cane is used ipsilaterally provides some relief over the total hip joint compression of 2,062.5 N ordinarily experienced in unilateral stance. The total hip joint compression when the cane is used ipsilaterally is still greater, however, than the total joint compression of 1,031.25 N found with a compensatory lateral trunk lean (see Table 10–2). Although a cane used ipsilaterally provides some benefits in energy expenditure and structural stress reduction, it is not as effective in reducing hip joint compression as the undesirable lateral lean of the trunk. *Moving the cane to the opposite hand produces substantially different and better results.*

Example 10-7

Calculating Hip Joint Compression With a Cane Ipsilaterally

If our 825-N subject can push down on the cane with 15% of his body weight, 123.75 N of body weight (825 N × 0.15) will pass through the cane. The magnitude of HATLL is thereby reduced to 563.75 N (687.5 N − 123.75 N). If the gravitational force of HATLL works through our estimated "normal" moment arm of 10 cm or 0.10 m (remember, the cane is intended to prevent the trunk lean), the torque of gravity is reduced to 56.38 Nm (563.75 N × 0.10 m). With a gravitational adduction torque of 56.38 Nm, the required force of the abductors acting through the usual 5 cm (0.05 m) moment arm is reduced to 1,127.6 N (56.38 Nm ÷ 0.05 m). The new hip joint reaction force using a cane *ipsilaterally* would then be:

1,127.6 N abductor joint compression
+ 563.75 N body weight (HATLL-cane) compression
1,691.35 N total joint compression

Use of a Cane Contralaterally

When the cane is moved to the side opposite the painful or weak hip joint, the reduction in the magnitude of HATLL is the same as it is when the cane is used on the same side as the painful hip joint; that is, the superimposed body weight passing through the weight-bearing hip joint is reduced by approximately 15% of body weight. However, the cane is now substantially farther from the painful supporting hip joint (Fig. 10–33) than it would be if the cane is used on the same side; that is, in addition to relieving some of the superimposed body weight, the cane is now in a position to assist the abductor muscles in providing a countertorque to the torque of gravity.[137] A classic description of the benefit of using a cane in the hand opposite to the hip impairment presumes that the downward force on the cane acts through the full distance between the hand and the weight-bearing (impaired) hip joint.[137] We will first look at an example using the classic analysis and then determine how this analysis might be misleading.

According to the classic analysis of the value of a cane in the opposite hand in Example 10-8, the hip joint reaction force would be due exclusively to body weight (563.75 N). This is, of course, an improvement over our calculated total hip compression with a lateral lean (1,031.25 N) and an even greater improvement over joint compression in normal unilateral stance (2,062.5 N) for a person weighing 825 N (see Table 10–2).

Unfortunately, the classic treatment of biomechanics of cane use appears to overestimate substantially the effects of the cane. Krebs and colleagues[138] (monitoring the patient with an instrumented hip prosthesis) found reductions in peak pressure magnitudes of 28% to 40% during cane-assisted gait. Although they reported pressures rather than forces, these values do not match the nearly 75% reduction in force that the classic calculation would indicate. Furthermore, Krebs and colleagues found a 45% reduction in gluteus medius EMG, not an elimination of activity.[138] The discrepancy in the classic analysis and laboratory and modeling data can be resolved by examining *how* the force applied to the cane by a person provides a countertorque to gravity. Although this is conjectural, we propose that the force of the downward thrust on the cane arrives on the pelvis through an equivalent contraction of the latissimus dorsi muscle (123.75 N or 15% of body weight). The latissimus dorsi muscle has an attachment to the pelvis on the posterior iliac crest, lateral to the erector spinae.[16] Given this attachment site, the line of pull of the muscle can be approximated to have a point of application on the pelvis above the ipsilateral acetabulum (20 cm or 0.20 m from the right hip joint axis). If the adduction torque around the right weight-bearing hip continued but was of diminished magnitude because of the countertorque provided by the latissimus dorsi (123.75 × 0.20m, or 24.75 Nm), contraction of the right hip abductors would still be needed to offset the remaining 31.63 Nm of adduction torque. Acting through their 0.05-M moment arm, the adbuctors would need to contract with a force of 632.6 N. The total joint compression would be estimated as 1,193.35 N (see Table 10–2). The hypothesized action of

Example 10-8

Classic Calculation of Hip Joint Compression With a Cane Contralaterally

Our sample 825-N patient has a superimposed body weight (HATLL) of 687.5 N, of which 123.75 N (W × 0.15) passes through the cane. Consequently, 563.75 N of body weight will pass through the right stance hip joint and the gravitation adduction torque will be:

HATLL torque$_{\text{adduction}}$: 563.75 N × 0.10 m = 56.38 Nm.

The downward force on the cane of 123.75 N acts through an estimated moment arm of 50 cm (0.5 m) between the cane in the left hand and the right weight-bearing hip joint (see Fig. 10–33). The cane, therefore, would generate an opposing abduction torque as follows:

Cane torque$_{\text{abduction}}$: 123.75 N × 0.5 m = 61.88 Nm

The torque around the right stance hip produced by a cane in the left hand (61.88 Nm) exceeds the torque produced by the remaining weight of HATLL (56.38 Nm). Because the gravitational torque (HATLL) may be underestimated, let us assume that the gravitational adduction torque and the countertorque provided by the cane offset each other. If the cane completely offset the adduction effect of gravity, there would be no need for hip abductor muscle force. The total hip joint compression in unilateral stance when a cane is used in the opposite hand would then be

$$\begin{array}{c} \text{0 N abductor joint compression} \\ \underline{+\ \text{563.75 N body weight (HATLL-cane) compression}} \\ \text{563.75 N total joint compression} \end{array}$$

Table 10–2 Summary of Calculations of Hip Joint Compression

Example: BW = 825 N HAT MA = 0.1 m Abductor MA = 0.05 m	UNILATERAL STANCE	LATERAL LEAN TOWARD STANCE LIMB	CANE IPSILATERAL	PROPOSED ANALYSIS OF A CANE CONTRALATERALLY
		Lean decreases HAT MA	Pushing down with cane reduces BW	Pushing down with cane reduces BW Cane counter torque
BW compression = 5/6 × BW	687.50 N	687.50 N	563.75 N (BW ↓ 15%)	563.75 N (BW ↓ 15%)
HATLL torque$_{\text{Adduction}}$ = BW$_{\text{compression}}$ × HAT MA	68.75 Nm	17.2 Nm (HAT MA ↓ to 0.25m)	56.38 Nm	31.63 Nm (Latissimus dorsi pull offsets 24.75 Nm of adduction torque)
Force contraction $_{\text{Abductor}}$ = HATLL torque$_{\text{Adduction}}$ ÷ Abductor MA	1,375.00 N	343.75 N	1,127.60 N	632.6 N
Total joint compression = Force contraction$_{\text{Abductor}}$ + BW compression	2,062.50 N	1,031.25 N	1,692.35 N	1,193.35 N

BW, body weight; HAT, head, arms, and trunk; HATLL= head, arms, and trunk non-weightbearing left lower limb; MA, moment arm.

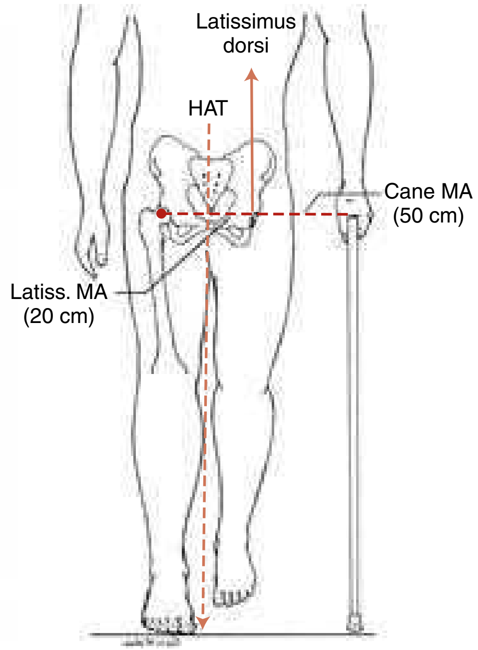

Figure 10–33 When a cane is placed in the hand opposite the painful supporting hip, the weight passing through the right hip is reduced, and activation of the left latissimus dorsi provides a countertorque to that of HATLL and diminishes the need for a contraction of the right hip abductors. The moment arm (MA) of the cane is erroneously estimated to be 50 cm, whereas the moment arm of the more relevant latissimus dorsi is estimated to be 20 cm.

the latissimus dorsi on the side of the cane could account for the difference in compressive forces between classic calculations with the cane used on the contralateral side and actual force measured in vivo.

CASE APPLICATION

Strategies to Reduce Hip Pain

case 10–6

Gabriella has osteoarthritic changes in her left hip that, if sufficient, can be a source of her pain. Furthermore, she has evidence of a labral tear, lateral hip pain, and a lean during left stance that may be at least partially attributable to degenerative changes in the gluteus medius/minimus tendons and trochanteric bursa inflammation. There is little doubt that Gabriella can benefit from reducing the demands on her left hip abductor muscles. Reduction in demand may both minimize aggravation of any tears or inflammation and reduce hip joint compressive force that, with her hip dysplasia, are likely to be carried over a reduced area of articular contact. Using a cane in her right hand would be appropriate. Although a lateral lean actually reduces the demands on the hip abductors a bit more, the energy expenditure and structural stress on the spine must be considered.

HIP JOINT PATHOLOGY

The very large active and passive forces crossing the hip joint make the joint's structures susceptible to wear and tear of normal components and to failure of weakened components. Small changes in the biomechanics of the femur or the acetabulum can result in increases in passive forces above normal levels or in weakness of the dynamic joint stabilizers. Some of the more common problems and the underlying mechanisms are discussed in this section.

Femoroacetabular Impingement

Femoroacetabular impingement (FAI) is described as the dysfunctional abutment of the proximal femur and the acetabulum. The result of such impingement causes pain and can lead to progressive degenerative changes in the hip joint, specifically the labrum. The causes of femoroacetabular impingement are thought to be multifactorial and the structure and deviant anatomy of the proximal hip joint are thought to be the primary causes of impingement. Femoroacetabular impingement has been classified as having one of two origins, each with its own etiologic and pathoanatomical characteristics. **Cam impingement** is thought to originate from what is described as a pistol-grip deformity of the femoral neck.[139] The deformity is likened to a pistol-grip in that the femoral neck fails to taper as the femoral head merges laterally into the femoral neck (Fig. 10–34A). The origin of the deformity is largely unknown, but researchers have suggested that it may evolve from a subtle and asymptomatic slippage and resultant calcification of the proximal femoral epiphysis.[140] Others have suggested that it is the body's natural and protective response to strengthen a vulnerability to stresses across the femoral neck. Radiographs taken from a lateral frog-leg angle may identify a cam deformity in which the junction of the femoral head and neck becomes indistinguishable. This thickening leads to poor clearance of the femur, specifically the femoral head-neck junction as the hip joint flexes or abducts (Fig. 10–34B). The abnormal junction or cam causes undersurface wear and tear to the anteriosuperior region of the labrum and the adjacent articular cartilage of the acetabulum.[139] Histological observation is distinguished by a lack of inflammatory cell markers and by the appearance of a chronic degenerative process. Cystic changes, disorganization, and fraying of the cartilaginous labral structures as well as fibrillation, malacia, and balding of the articular cartilage of the anteriosuperior acetabulum was consistently observed among those diagnosed with cam impingement.[141]

Pincer impingement in femoroacetabular impingement (in contrast to cam impingement) is caused by aberrations of the acetabulum. Patients presenting with pincer-type impingement may demonstrate greater coverage, or overhang, of the acetabulum on the femoral head due to excessive retroversion of the acetabulum, and a deeper acetabular fossa, referred to as *coxa profunda*[25,140] (Fig. 10–35A). The excessive coverage of the femoral head causes a compression of the superior labrum between the acetabular rim and the femoral head/neck in abduction[141,142] (Fig. 10–35B). The

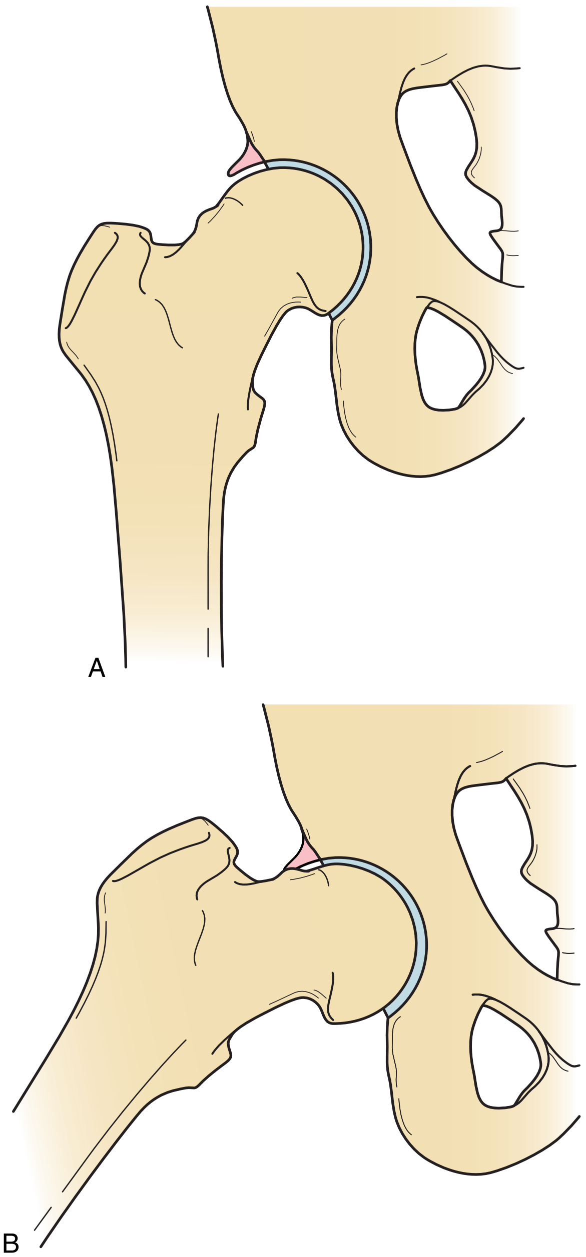

Figure 10–34 Cam impingement occurs as a result of an abnormal widening of the femoral neck **(A)** that leads to the abutment of the anterior superior labrum and the articular cartilage of the acetabulum during flexion or abduction of the hip **(B).**

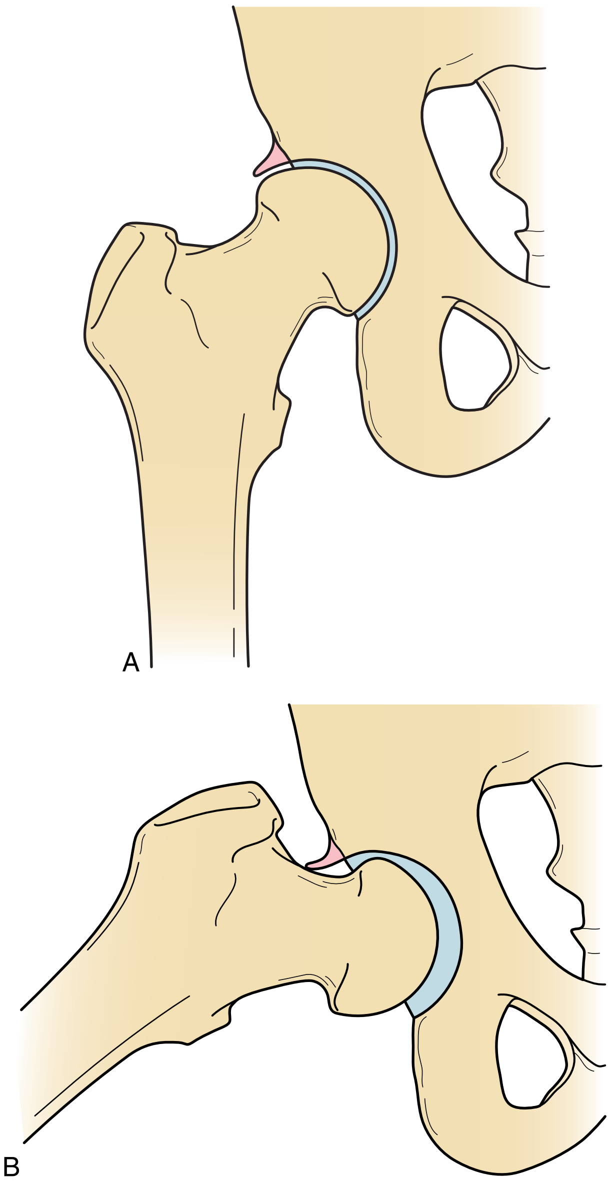

Figure 10–35 Pincer impingement occurs as a result of an overcoverage of the acetabulum on the femoral head **(A)**, causing compression of the superior labrum between the acetabular rim and the femoral head-neck junction with flexion or abduction **(B)**.

condition is analogous to primary subacromial impingement of the shoulder, in which a hooked acromion invades the subacromial space and creates a mechanical impingement. Similarly, in pincer-type impingement of the hip, the overcoverage of the femoral head by the acetabulum leads to a mechanical impingement of the superior labral-acetabular complex.[139] Repetitive impingement of the labrum may result in ossification of the labrum that may further worsen the overcoverage of the femoral head.[142] This calcification may further lead to articular lesions to the femoral head and acetabulum. Researchers have suggested that the mechanical blocking of the natural arthrokinematics of hip flexion leads to abnormal posterior displacement of the femoral head as the hip is forced into greater flexion angles.[139] This would explain articular erosion to the posterior-inferior acetabulum and the posterior-medial surface of the femoral head associated with pincer-type impingement.

Although femoroacetabular impingement is typically considered to result from either cam or pincer deformities, current research indicates that it may be difficult and erroneous to classify femoroacetabular impingement into one category versus the other.[140] Authors studied surgically dislocated hips with radiological evidence of either a pistol-grip deformity (cam impingement) or coxa profunda abnormality (pincer impingement). Although the imaging results indicated classification into one of the two categories, the surgical observation revealed that the majority of the studied hips had a combination of both abnormal findings. This suggests that the classification scheme is more complex and that many patients may in fact have varying degrees of both types of impingement.[139]

It should also be noted that aberrant anatomical findings may not always present in those presenting with symptoms of femoroacetabular impingement. Ito and colleagues studied subjects with inguinal pain related to femoroacetabular impingement who had significant loss of ROM into flexion, abduction, medial rotation, and lateral rotation without findings of dysplasia.[141] This suggests that other factors related to hip ROM deficits, including capsuloligamentous or musculotendinous restriction and arthritic changes may exist as etiologic factors of femoroacetabular impingement and should be considered in the evaluation and treatment of the condition.

Labral Pathology

Labral pathology is often discussed synonymously with femoroacetabular impingement, as the two conditions may often coexist. As previously discussed, the mechanisms of labral pathology are associated with femoroacetabular impingement. Burnett found 95% of patients with labral tears confirmed by hip arthroscopy had clinical signs of femoroacetabular impingement.[27] Femoroacetabular impingement, however, is not the only bony abnormality associated with a torn labrum. Guerva and colleagues reported that in comparison to the unaffected side, hips with symptomatic labral tears displayed significant differences in several radiologic measures, including decreased center edge angle, acetabular retroversion, and coxa vara compared to the unaffected side.[25] Collectively, these abnormalities lead to a lateral and superior displacement of the femoral head and predispose to an anterior superior impingement of the acetabulum and femoral neck.[25,141]

The labrum is a legitimate source of pain. Kim and Azuma[14] identified that the labrum is innervated tissue with the capability of causing and referring pain in patients with labral pathology. The onset of labral pathology may be caused either by a single traumatic event or more commonly by the cumulative effects of microtrauma. Traumatic labral injury may be the result of a rapid and forceful rotation of the hip. Although this may occur in open chain lower limb function, such trauma is more often a result of long axis rotation in a closed chain position, such as seen commonly in athletics that may require a rapid change of direction with planting and pivoting on a fixed leg. Another mechanism for traumatic labral injury may be the result of a dislocating force of the hip. Although the hip is an inherently stable joint, a blunt force to a flexed hip that may be seen in falls on the knee may lead to posterior dislocation and subsequent injury to the posterior labrum.

Cumulative microtrauma appears to be the more common mechanism of injury to the labrum. As evidenced by the absence of inflammatory markers in symptomatic labral tears, it appears that the labrum is vulnerable to a progressive degenerative process that leads to pathology.[25,141] As discussed earlier, the repetitive mechanical stress induced by femoroacetabular impingement contributes to nontraumatic labral injury. Thus, the morphologic factors that characterize femoroacetabular impingement are also common in those patients with labral pathology. Collectively, these factors may ultimately lead to either an undercoverage or an overcoverage of the femoral head. Undercoverage may also be the result of a shallow acetabulum or dysplastic changes to the femoral head neck junction as described in cam impingement. This leads to disproportionate loading of the acetabular rim and labral structures. Undercoverage may also be produced functionally through laxity or an acquired instability of the hip joint.

The role of the labrum as a key stabilizing component of the hip joint also helps explain the vulnerability to injury. Tension through the capsuloligamentous structures creates extreme strain through the anterior labrum. This is accentuated when the hip is moving into positions of abduction and external rotation. Further tension may be exhibited on the capsulolabral structures while these movements are performed with combined extension of the hip joint.[143] These positions may be common in athletic movements. Thus throwing and swinging movements as well as quick planting and explosive changes in direction may lead to cumulative damage to the anterior capsulolabral complex. Athletes participating in baseball, golf, gymnastics, dance, and martial arts are particularly vulnerable to capsular and ligamentous redundancy (laxity) and labral injury. Crawford and colleagues[143] recreated capsular redundancy and a simulated labral tear in cadaver specimens and found a 60% reduction of force needed to distract the hip joint.[143] Researchers suggest that a labral tear with capsular redundancy causes a loss in intra-articular pressure of the joint that ultimately compromises joint stability.

Arthrosis

Our understandings of more recently discovered pathologies associated with hip joint dysfunction are also helping us to understand the mechanisms and the interrelationships to other pathologies of the hip. Femoroacetabular impingement has been implicated as a potential etiologic factor in the presence of **osteoarthritis (OA)** of the hip joint. Beck[139] described the process of articular deterioration as it relates to findings of cam impingement. The presence of a labral tear has also been identified as an important risk factor in the development of OA of the hip.[58,144] Chronic synovial irritation, joint fluid leakage, and the relationship to microinstability of the hip joint may contribute to abnormal joint loading and deterioration of the articular surfaces of the joint.

Regardless of the etiology, osteoarthritis is the most common painful condition of the hip. It is defined as deterioration of the articular cartilage and to subsequent related changes in articular tissues.[132] OA of the hip occurs among 7% and 25% of individuals,[145] with an incidence among females slightly greater than that among males.[146] Juhakoski and colleagues[147] conducted 22-year prospective study that attempted to identify the key risk factors associated with the onset of hip OA symptoms. Many variables were analyzed, including body mass index, work history, injury history, participation in exercise programs, and history of smoking and alcohol use. Of the aforementioned factors, previous history of musculoskeletal injury and work history including extensive physicality and manual labor were the only two factors predictive of OA. One study noted that more than 90% of hip osteoarthritis was attributed to anatomical abnormalities.[148] Changes may be due to subtle deviations present from birth, to tissue changes inherent in aging, to the repetitive mechanical stress of loading the body weight on the hip joint over a prolonged period, to impingement between the femur and labrum or adjacent acetabulum, or to interactions between these factors.[19,22,149] The factors most closely associated with idiopathic hip joint arthrosis are increased age and increased weight/height ratio.[150] Contrary to popular belief, repetitive weight-bearing exercise, specifically running, was found to have no association between OA changes and running status among older subjects.[151]

The mechanism for cartilaginous degeneration in the hip joint is not clearcut. In the absence of preexisting trauma or biomechanical abnormality, degenerative changes may be the result of inadequate joint forces, rather than excessive forces at the hip joint. Articular cartilage is nourished through joint movement and compression. When a person is inactive, nourishment to the articular cartilage is diminished and more vulnerable to injury and deterioration. This would explain why there is little or no association between increased activity level with sports or recreational activities and hip arthrosis.[151,152] In fact, there is evidence to suggest that lower activity levels are associated with hip OA. Hirata and colleagues[153] found that 40% of women diagnosed with hip OA were classified as physically inactive and spent less than 6 minutes per day in what would be considered moderately intense physical activity. Rosemann and colleagues[154] suggested that moderate activity can actually be preventive in the progression of OA symptoms. Forces in excess of half body weight are needed to fully compress the femoral head into congruent contact with the dome of the acetabulum.[155] Using a number of other studies as a base, Bullough and associates[155] hypothesized that we typically spend no more than 5% to 25% of our time in unilateral lower extremity weight-bearing activities in which the load may be sufficient to compress the articular cartilage of the dome of the acetabulum. Lower loads and infrequent high joint forces may be inadequate to maintain flow of nutrients and wastes through the avascular cartilage. The theory of inadequate compression as a contributing factor to hip joint degeneration is supported by the fact that the more common degenerative changes in the femur are at the periphery of the head and the perifoveal area, rather than at the superior primary weight-bearing area.[82,155] The periphery of the head receives only about one third the compressive force of the superior portion of the head.[129] Conversely, the superior portion of the head is compressed not only in standing but is also in contact with the posterior acetabulum during sitting activities.[82] Yet, the area of the femoral head around the fovea is most commonly in the non-weightbearing acetabular notch and would undergo compression relatively infrequently. Wingstrand and colleagues[52] proposed that excessive intra-articular fluid from relatively benign synovitis or trauma may reduce articular congruence and the stabilizing effect of atmospheric pressure, resulting in microinstability and unfavorable cartilage loads. This theory has been substantiated by evidence by Tarasevicius and colleagues,[156] who demonstrated lower intra-capsular pressures to be related to the severity of OA of the hip.

Fracture

To maintain integrity of the hip joint, the bony components must be of sufficient strength to withstand the forces that are acting around and through the hip joint. As noted in the section on the weight-bearing structure of the hip joint, the vertical weight-bearing forces that pass down through the superior margin of the acetabulum in both unilateral and bilateral stance act at some distance from ground reaction force up the shaft of the femur. The result is a bending force across the femoral neck (see Fig. 10–15). Normally the trabecular systems are capable of resisting the bending forces, but abnormal increases in the magnitude of the force or weakening of the bone can lead to bony failure. The site of failure is likely to be in areas of thinner trabecular distribution such as the zone of weakness (see Fig. 10–16). Thomas and colleagues[157] found the superior, lateral, and posterior portions of the femoral neck suffered the most significant amount of trabecular bone loss. It was estimated that the women they studied experienced a 42% decrease in the trabecular density to this region over the age span of 50 years of adult life.[157] Crabtree and colleagues[81] studied both patients and cadavers with a fracture to conclude that a loss in cortical bone, not cancellous bone (trabeculae), may be the source of the problem. Although cancellous bone mass was similar for cases and controls in similar age groups, there was a 25% reduction in cortical bone mass in the fracture cases.[81] Thomas and colleagues[157] reported similar findings in that there was cortical thinning of 37% to 49% in cases where fractures occurred versus controls, but trabecular density was similar between groups. The authors acknowledged, however, the importance of the trabecular infrastructure in resisting the deforming forces that cause a fracture, especially during a fall. Although cortical thinning is a more significant finding in cases of proximal hip fracture, the use of therapies and treatment directed toward increasing trabecular density may have a substantial impact on reducing the risk of fracture.[157]

Bony failure in the femoral neck is uncommon in the child or young adult, even with large applied loads. However, femoral neck fractures occur at the rate of about 98/100,000 people in the United States, with the average age at occurrence being in the 70s. It is estimated that three out of four hip fractures occur in an elderly population. Adults over the age of 75 are over 5 times more likely to sustain a hip fracture than those aged 65 to 74 and over 30 times more common than those aged 45 to 64.[158] There is a predominance of fractures in women, although this is certainly influenced by their greater longevity. Of middle-aged people, women actually suffer fewer hip fractures than do men, although the fractures in this age group are usually attributable to substantial trauma.[159]

In 87% of cases of hip fracture among the elderly population, the precipitating factor appears to be moderate trauma such as that caused by a fall from standing, from a chair, or from a bed. There is consensus that hip fracture is associated with, but not exclusively due to, diminished bone density.[160] Bone density decreases about 2% per year after age 50 and trabeculae clearly thin and disappear with aging.[161] Cummings and Nevitt[161] believed the exponential increase in hip fractures with age could not be accounted for by decreased bone density alone and proposed that the slowed gait characteristic of the elderly may play an important part. They contended that the slowing of gait makes it less likely that momentum will carry the body forward in a fall (generally onto an outstretched hand) and more likely that the fall will occur backward on to the hip area weakened by bone loss and no longer padded by the fat and muscle bulk of youth. Gait speed and characteristics have been shown to have a direction relationship to falls and subsequently may be meaningful in assessing the risk for a hip fracture. Lindsey and colleagues[162] reported that subjects with higher gait speed and longer step length show greater bone mineral density and thus have less risk for femur fracture. In a similar study, Dargent-Molina[163] identified clinical predictors that categorized females into a high- or low-risk group for proximal femur fracture. Among the key clinical factors was gait speed as well as a tandem walk test that tests dynamic balance. These factors proved to be significant in distinguishing the high-risk group and low-risk group for sustaining a hip fracture.

Hip fracture will continue to receive considerable attention because of the high health-care costs of both conservative and operative treatment. Of all fall-related fractures, hip fractures cause the greatest number of deaths and lead to the most severe health problems and reduced quality of life. Hip fractures alone account for nearly half of fractures leading to mortality and 4% of all injury related deaths.[158] Mortality within the 1 year following a femoral neck fracture has been estimated between 21% to 24%.[164] Although mortality rates among the elderly are high, younger patients are more vulnerable to complications after a fracture. This may include nonunion of the fracture and femoral head osteonecrosis, which occurs in 23% of cases.[165] Not only are these conditions painful, but malunion of the fracture can also lead to joint instability or cartilaginous deterioration (or both) as a result of poorly aligned bony segments. Although the femoral head may receive some blood supply via the ligament of the femoral head, an absent or diminished supply through the ligament of the head (as occurs with aging) means reliance on anastomoses from the circumflex arteries. This circumflex arterial supply may be disrupted by femoral neck fracture, which leaves the femoral head susceptible to avascular necrosis and necessitates replacement of the head of the femur with an artificial implant. Due to high complication and mortality rates, careful consideration in the management of patients with hip fracture is necessary. Several factors, including age, level of functional activity, and comorbidities, must be considered in the effective management of these conditions.[166]

CASE APPLICATION

Hip Dysplasia *case 10–7*

Although we have probably covered the potential sources of Gabriella's problems fairly well at this point, it is worth taking note of the most likely underlying causes. Gabriella's persisting coxa valga and femoral anteversion are the likely sources of many, if not all, of her problems. With a valgus and anteverted femur, the articular contact within Gabriella's hip joint is substantially reduced, increasing the contact pressures and locating them atypically within the acetabulum and on the head of the femur. This is consistent with her complaints of instability and may also have contributed to her labral tear. Wenger and colleagues[39] found that over half of the hips studied that had an identifiable labral tear also had a coxa valga deformity of the femur. Both her hip dysplasia and her history of dance, gymnastics, and years of working at floor level with young children have exacerbated the likelihood of anterior impingement of the labrum, resulting in probable labral tear and chondral lesions. The mechanical disadvantage to her hip abductors is likely to contribute to overuse and degenerative lesions of the abductor mechanism, further affecting abductor function and resulting in what might be referred to either as a gluteus medius or antalgic gait. It appears that Gabriella's hip symptoms have been the result of an ongoing and progressive phenomenon that is leading to the early stages of joint degeneration. It likely began during repetitive movements and activities that required excessive and extremes of ROM. These repetitive movements, combined with altered hip mechanics due to her hip dysplasia, resulted in chronic and repeated trauma that damaged her anterior capsule and labrum. The degradation and ultimate failure of her capsule and labrum likely contributed to her feeling of instability of the hip. A labral tear causes a leakage of joint fluid and disrupts the "seal" of the hip joint that creates intra-articular pressure.[143] This pressure helps stabilize the hip joint. Combined, all of these factors make one more susceptible to osteoarthritis. If she continues down this path it is likely that her pain will worsen and her joint may continue to deteriorate. This may lead to an eventual total joint replacement. However, Gabriella is not without treatment options for her current condition. Medications and injections as well as physical therapy remain options for the conservative management of Gabriella's symptoms.[167] The emergence of hip arthroscopy has resulted in new treatment options for patients with similar symptoms, providing more advanced osteoarthritis is not present as well. Labral pathology and tears to the abductor mechanism have shown to be managed successfully through minimally invasive techniques of arthroscopy. Philippon and colleagues[168] reported excellent results that included a 24% improvement in self-reported functional outcomes and a median satisfaction score of 9 out of a possible 10 for patients 2 years after hip arthroscopy. Addressing her dysplastic hip findings may also suggest a surgical option. However, the procedures to correct her acetabular and femoral abnormalities would require much more aggressive and invasive surgery with a likely protracted

Continued

recovery period. Although there has been no concrete evidence to suggest that any of these surgical approaches and techniques prevent the progression of osteoarthritis of the hip, surgery may improve the patient's associated biomechanical faults or abnormities and may eliminate the diseased source of pain.

It used to be thought that patients like Gabriella would likely continue to progress in the process of degeneration of the hip joint until the pain and functional limitations would eventually necessitate a need for a joint replacement surgery. Our understanding of this process is helping to discover more effective, conservative approaches to the management of such conditions. When conservative measures have failed, current surgical approaches and the focus of future treatment options in the bioengineering and restorative techniques for the articular surfaces of joints are encouraging that viable and effective treatment options exist and will evolve into even better treatment options for patients like Gabriella.[169]

SUMMARY

The normal hip joint is well designed to withstand the forces that act through and around it, assisted by the trabecular systems, cartilaginous coverings, muscles, and ligaments. Alterations in the direction or magnitude of forces acting around the hip create abnormal concentrations of stress that predispose the joint structures to injury and degenerative changes. The degenerative changes, in turn, can create additional alterations in function that not only affect the hip joint's ability to support the body weight in standing, in locomotor activities, and in other activities of daily living but may also result in adaptive changes at more proximal and distal joints. Consequently, the reader must understand both the dysfunction that might occur at the hip *and* the associated dysfunctions that may result in or from dysfunction elsewhere in the lower extremity and spine. The remaining chapters of this text will focus not only on primary dysfunction at a joint complex but also on associated dysfunction related to proximal and distal joint problems.

STUDY QUESTIONS

1. Which side of the femoral neck and which side of the femoral shaft are subjected to compressive stresses during weight-bearing? How does the bone respond to these stresses?
2. What is the primary weight-bearing area of the femoral head? Of the acetabulum? Where are degenerative changes most commonly found in the femoral head and acetabulum?
3. Describe why using a cane on the side opposite hip joint pain or weakness is more effective than using the cane on the same side.
4. Demonstrate how variations in the angle of inclination affect the moment arm of the hip abductors by drawing the following: a normal angle of inclination at the hip, the angle in coxa vara, and the angle in coxa valga. Please include the action line and the moment arm of the hip abductors in the diagram.
5. Describe what would happen to the pelvis in left unilateral stance when the left hip abductors are paralyzed. How is equilibrium maintained in this situation?
6. Describe motion at the right and left hip joints and at the lumbar spine during hiking of the pelvis in right limb stance, assuming that the person is to remain upright.
7. Contrast the close-packed versus maximally congruent position for the hip joint.
8. Calculate the minimum joint reaction force (total hip joint compression) at the right hip joint that would occur for a 200-lb person standing symmetrically on both legs versus one leg (assuming a gravitational moment arm of 4 in. and the abductor muscle moment arm of 2 in.).
9. Under what circumstances does the hip joint participate as part of an open chain? As part of a closed chain?
10. What bony abnormality or abnormalities of the femur or pelvis predispose the hip joint to the possibility of dislocation? Why?
11. Which structures at the hip joint, given their location, appear likely to limit the extremes of motion in flexion? In extension? In lateral rotation? In medial rotation? In abduction and adduction?
12. Which muscles of the hip joint are affected by knee joint position? Which position of the knee makes these muscles less effective at the hip joint?
13. If someone were in a unilateral stance on the left limb, what hip joint motion would result from forward rotation of the pelvis?
14. If a person has a painful right hip, in which direction should she lean her trunk to reduce the forces on the right hip during right unilateral support? Explain the reasons for your answer.
15. What position does the hip joint tend to assume when there is joint pain? Why is this?
16. Identify several factors that might predispose someone to a femoral neck fracture.
17. How does the femoral head receive its blood supply? What problems might jeopardize that supply?
18. Relate femoral anteversion to medial femoral torsion.
19. Under what circumstances might the hip adductors work synergistically with the hip abductors?
20. Why is the acetabular notch nonarticular? In what ways does this serve hip joint function?

STUDY QUESTIONS—cont'd

21. Explain the differences between cam and pincer-type impingement.
22. Describe how the presence of femoroacetabular impingement may lead to osteoarthritis of the hip.
23. What factors contribute to stability of the hip joint?
24. How are abnormalities at the femur and acetabulum related to femoroacetabular impingement? Labral tears? Fractures?

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Chapter 11

The Knee

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INTRODUCTION

The knee complex is one of the most often injured joints in the human body. The myriad of ligamentous attachments, along with numerous muscles crossing the joint, provide insight into the joint's complexity. This anatomical complexity is necessary to allow for the elaborate interplay between the joint's mobility and stability roles. The knee joint works in conjunction with the hip and ankle joints to support the body's weight during static erect posture. Dynamically, the knee complex is responsible for moving and supporting the body during a variety of both routine and difficult activities. The fact that the knee must fulfill major stability as well as major mobility roles is reflected in its structure and function.

The knee complex is composed of two distinct articulations located within a single joint capsule: the tibiofemoral joint and the patellofemoral joint. The **tibiofemoral joint** is the articulation between the distal femur and the proximal tibia. The **patellofemoral joint** is the articulation between the posterior patella and the femur. Although the patella enhances the tibiofemoral mechanism, the characteristics, responses, and problems of the patellofemoral joint are distinct enough from the tibiofemoral joint to warrant separate attention. Despite its adjacent location, the superior tibiofibular joint is not considered to be a part of the knee complex because it is not contained within the knee joint capsule and is functionally related to the ankle joint; it will therefore be discussed in Chapter 12.

11-1 Patient Case

case

Tina Mongelli is a 43-year-old female patient who presents to your clinic with complaints of knee pain with increased activity. Tina's chief complaint is pain in and around her right knee (pain at best: 2/10) that worsens when she plays tennis and during stair ascents and descents (pain at worst: 8/10). She has palpable pain along the medial aspect of her tibiofemoral joint line. She schematically indicates pain around her patella that she subjectively describes as being behind her patella. She is currently unable to jog, play tennis, and walk for more than 1 mile without discomfort. Tina's past medical history includes tearing her anterior cruciate ligament, medial collateral ligament, and medial meniscus on the right side when she was 24 years old. After 4 months of exercise in an attempt to delay surgery, she subsequently underwent surgical reconstruction of the anterior cruciate ligament with a patellar tendon autograft and a partial medial meniscectomy. The medial collateral ligament was left to heal on its own. Clinical testing of her right knee revealed increased laxity with a valgus stress test with no pain at the end of range, full tibiofemoral range of motion (ROM), a hypomobile medial patellar glide, and 80% quadriceps strength compared to her left side. Diagnostic images were obtained of Tina's lower extremities. Weight-bearing radiographs of the right limb revealed genu varum with moderate joint space narrowing in the patellofemoral and medial tibiofemoral compartments. What structural abnormalities do you think are contributing to the atypical function at her knee, and could be contributing to her pain?

TIBIOFEMORAL JOINT STRUCTURE

The tibiofemoral, or knee, joint is a double condyloid joint with three degrees of freedom of angular (rotary) motion. Flexion and extension occur in the sagittal plane around a coronal axis through the epicondyles of the distal femur,[1] medial/lateral (internal/external) rotation occur in the transverse plane about a longitudinal axis through the lateral side of the medial tibial condyle,[2] and abduction and adduction occur in the frontal plane around an anteroposterior axis.[3] The double condyloid knee joint is defined by its medial and lateral articular surfaces, also referred to as the **medial** and **lateral compartments** of the knee. Careful examination of the articular surfaces and understanding of the relationship of the surfaces to each other are necessary for a full understanding of the knee joint's movements and of both the functions and dysfunctions common to the joint.

Femur

The proximal articular surface of the knee joint is composed of the large medial and lateral condyles of the distal femur. The medial condyle is larger, with a greater radius of curvature and projects further than does the lateral condyle.[4] Because of the medial obliquity of the shaft of the femur, the femoral condyles do not lie immediately below the femoral head but are slightly medial to it (Fig. 11–1A). As a result, the lateral condyle lies more directly in line with the shaft than does the medial condyle. Since the medial condyle extends further distally, the distal end of the femur remains essentially horizontal despite the angulation of the femur's shaft.

In the sagittal plane, the condyles have a convex shape, with a smaller radius of curvature posteriorly (Fig. 11–1B).[2,5] Although the distal femur as a whole has very little curvature in the frontal plane, both the medial and lateral condyles individually exhibit a slight convexity in the frontal plane.

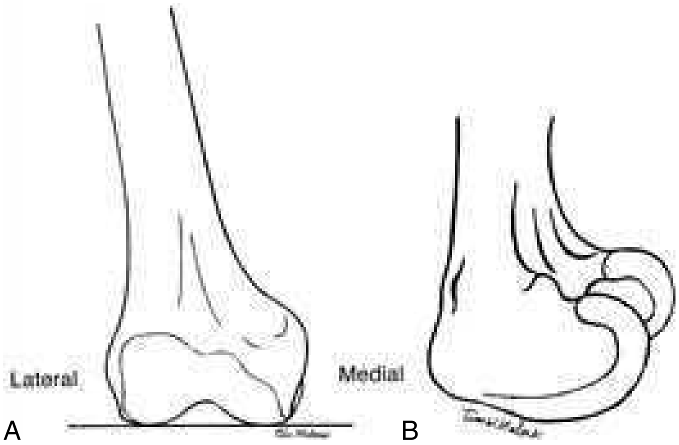


Figure 11–1 **A.** Because of the obliquity of the shaft of the femur, the lateral femoral condyle lies more directly in line with the shaft than does the medial condyle. The medial condyle is more prominent, however, which results in a horizontal distal femoral surface despite the oblique shaft. **B.** The anteroposterior convexity of the condyles is not consistently spherical, having a smaller radius of curvature posteriorly.

The lateral femoral condyle is shifted anteriorly in relation to the medial femoral condyle.[6] In addition, the tibial articular surface of the lateral femoral condyle is shorter than the tibial articular surface of the medial femoral condyle.[5] When the femur is examined from an inferior view (Fig. 11–2), the lateral femoral condyle appears at first glance to be longer. However, when the patellofemoral surface is excluded, we can observe that the lateral tibial surface extends less anteriorly than the medial condyle. The two condyles are separated inferiorly by the **intercondylar notch** through most of their length but are joined anteriorly by an asymmetrical, shallow groove called the **femoral sulcus, patellar groove,** or **patellar surface** that engages the patella during early flexion.

Tibia

The large convex femoral condyles sit on the relatively flat tibial condyles. The asymmetrical medial and lateral tibial condyles or plateaus constitute the distal articular surface of the knee joint (Fig. 11–3A). The medial tibial plateau is longer in the anteroposterior direction than the lateral tibial plateau;[5] however, the lateral tibial articular cartilage is thicker than the articular cartilage on the medial side.[7] The proximal tibia is larger than the tibial shaft and, consequently, overhangs the tibial shaft posteriorly (Fig. 11–3B). Accompanying this posterior overhang, the tibial plateau slopes posteriorly approximately 7° to 10°,[5,6] which is conducive for flexing the tibiofemoral joint. The medial and lateral tibial condyles are separated by a roughened area and two bony spines called the **intercondylar tubercles** (Fig. 11–4). These tubercles become lodged in the intercondylar notch of the femur during knee extension. The tibial plateaus are predominantly flat, with a slight convexity at the anterior and posterior margins,[2] which suggests that the combined bony architecture of the somewhat convex tibial plateaus and convex femoral condyles does not bode well for joint stability. Because of this lack of bony

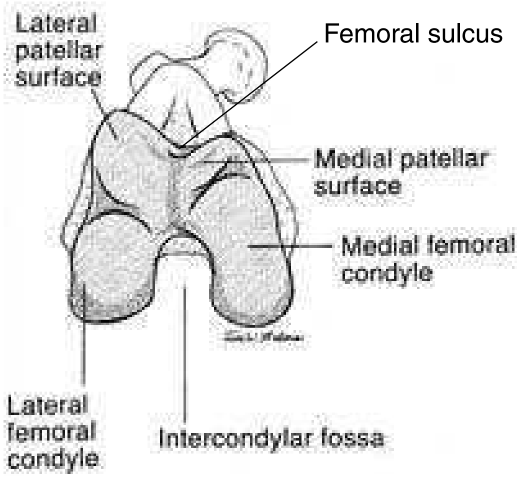


Figure 11–2 The patellar surface (shaded light pink) is separated from the femur's tibial articular surface (shaded darker pink) by two slight grooves that run obliquely across the condyles. The medial femoral condyle is longer than the lateral femoral condyle; the lateral lip of the patellar surface is larger than the medial lip of the patellar surface.

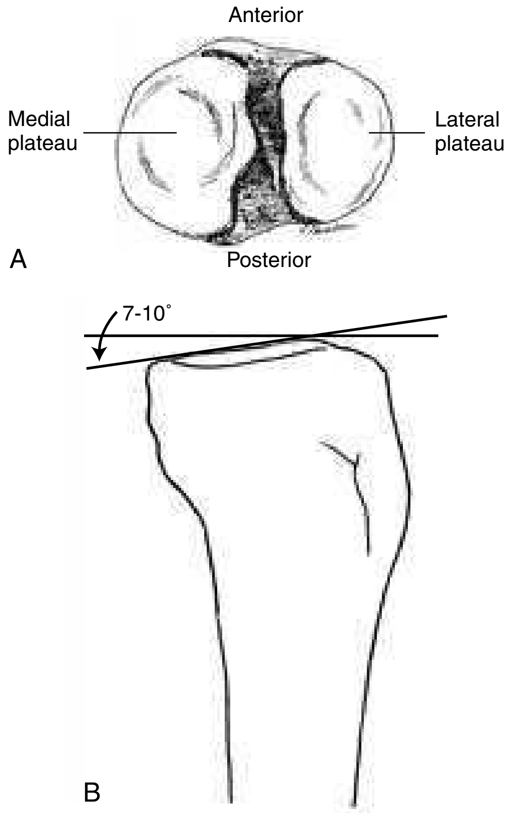


Figure 11–3 **A.** A superior view of the articulating surfaces on the tibia illustrates differences in size and configuration between the medial and lateral tibial plateaus. **B.** The tibial plateau overhangs the shaft of the tibia posteriorly and is inclined posteriorly 7° to 10°.

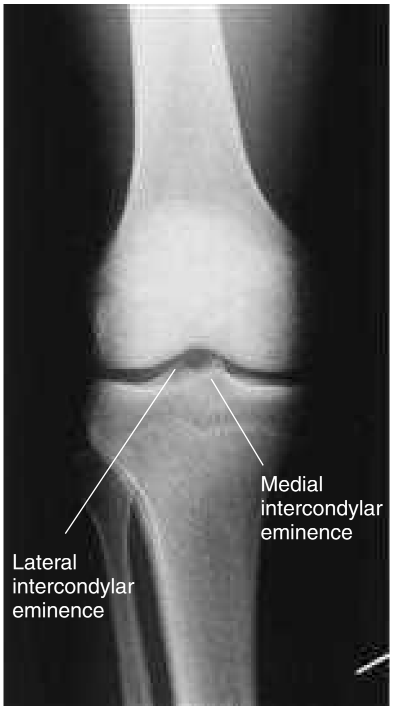


Figure 11–4 A radiograph of the right knee shows how the medial and lateral intercondylar eminences of the tibia lodge in the intercondylar notch of the femur in the extended knee.

stability, accessory joint structures **(menisci)** are necessary to improve joint congruency.

Tibiofemoral Alignment and Weight-Bearing Forces

The **anatomical (longitudinal) axis** of the femur, as already noted, is oblique, directed inferiorly and medially from its proximal to distal end. The anatomical axis of the tibia is directed almost vertically. Consequently, the femoral and tibial longitudinal axes normally form an angle medially at the knee joint of 180° to 185°; that is, the femur is angled up to 5° off vertical, creating a slight physiological (normal) valgus angle at the knee (Fig. 11–5). If the medial tibiofemoral angle is greater than 185°, an abnormal condition called **genu valgum** ("knock knees") exists. If the medial tibiofemoral angle is 175° or less, the resulting abnormality is called **genu varum** ("bow legs"). Each condition alters the compressive and tensile stresses on the medial and lateral compartments of the knee joint.

An alternative method of measuring tibiofemoral alignment is performed by drawing a line on a radiograph from the center of the femoral head to the center of the head of the talus (see Fig. 11–5). This line represents the **mechanical axis,** or **weight-bearing line,** of the lower extremity, and in a normally aligned knee, it will pass through the center of the joint between the intercondylar tubercles.[8] The weight-bearing line can be used as a simplification of the ground reaction force as it travels up the lower extremity. In bilateral stance, the weight-bearing stresses on the knee joint are, therefore, equally distributed between the medial and lateral condyles (or medial and lateral compartments).[8] However, once unilateral stance is adopted or dynamic forces are applied to the joint, compartmental loading is altered. In the case of unilateral stance (e.g., during the stance phase of gait), the weight-bearing line shifts toward the medial compartment to account for the now smaller base of support below the center of mass (Fig. 11–6A). This shift increases the compressive forces on the medial compartment (Fig. 11–6B).[9]

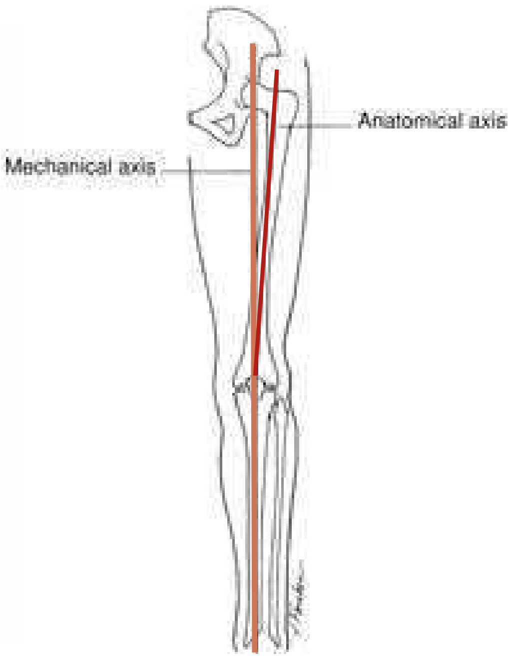


Figure 11–5 The anatomical axes of the femur and tibia result in a normal physiological valgus angulation of approximately 185°. The mechanical axis (weight-bearing line) of the lower extremity passes from the center of the hip to the center of the ankle joint and, in a neutrally aligned limb, results in weight-bearing forces that are distributed about equally between the medial and lateral condyles of the knee joint.

Advances in technology allow the mechanical loading across the medial and lateral compartments to be measured both directly and indirectly. In vivo knee loading characteristics have been measured by instrumenting the prostheses used for total knee arthroplasty. Such direct measurements indicate that most activities of daily living place a greater load on the medial compartment compared to the lateral

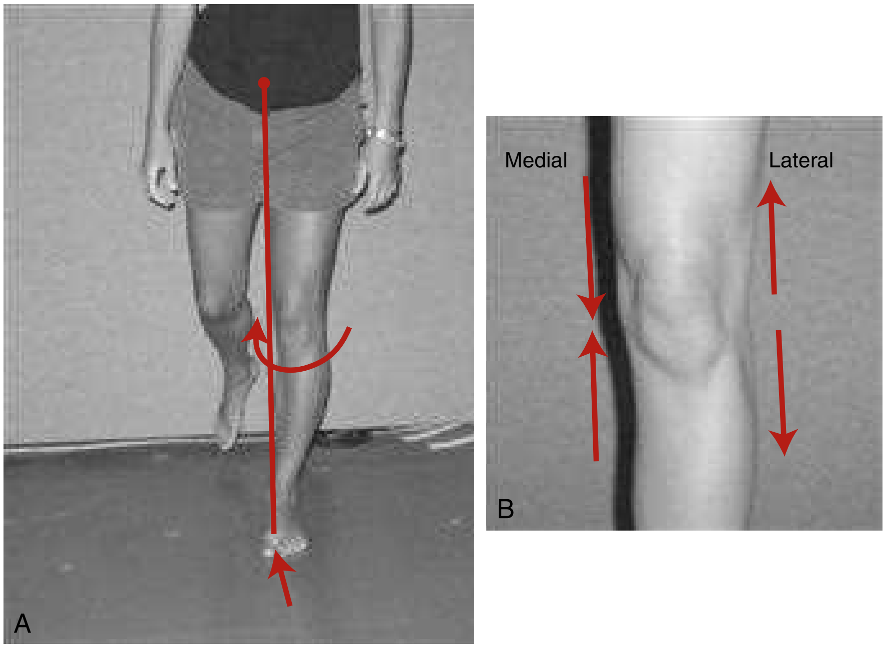


Figure 11–6 **A.** During dynamic activities, such as gait, the line of force shifts medially to the knee joint center. **B.** This medial shift increases the compressive stresses medially and increases the tensile stresses laterally.

compartment.[10] Less invasive measures can be used to estimate the compressive loads within the knee joint. Specifically, external forces through the leg can be measured from a force plate embedded in a walkway, allowing researchers to measure ground reaction forces during activities of daily living (e.g., walking, running). These external forces can be used as surrogates for internal forces incurred by the knee joint and may be used to calculate torques (moments of force) about the joint. For example, if the force from the floor extends medial to the knee joint center, then an adduction moment is created around the knee joint, which acts to rotate the knee into relatively greater varus (adduction). Thus, the magnitude of the knee adduction moment can be used as a surrogate measure for medial compartment loading during gait and other activities of daily living. Abnormally high knee adduction moments are associated with the development of medial knee osteoarthritis (OA).[11]

The association between knee malalignment and progression of knee OA[12–15] has implications for patients who present with abnormal anatomical alignment. Genu valgum, for instance, shifts the weight-bearing line onto the lateral compartment, relatively increasing the lateral compressive force (Fig. 11–7A). In the case of genu varum, the weight-bearing line is shifted medially, further increasing the compressive force on the medial condyle (Fig. 11–7B). The presence of genu valgum or genu varum creates a constant overload of the lateral or medial articular cartilage, respectively, which may result in damage to the cartilage[16] and the development of frontal plane knee laxity. Genu varum, for instance, contributes to the progression of medial compartment knee osteoarthritis[17] and corresponding medial joint laxity as the medial capsular ligament's attachment sites are gradually approximated through the erosion of the medial compartment's articular cartilage.

Continuing Exploration 11-1:

Effects and Corrections of Malalignment

In the presence of severe frontal plane malalignment and osteoarthritis, some orthopedic surgeons will perform a realignment procedure at the knee, called a high tibial osteotomy. This procedure realigns the limb to lessen the compressive force on the damaged painful tibiofemoral compartment. In the case of either significant genu varum or genu valgum, the surgery creates a surgical fracture in the tibia (or sometimes in the femur) in order to realign the limb to a more neutral position. Other less invasive methods of attempting to diminish compartmental loads in the presence of malalignment include lateral/medial heel wedges or a knee brace that shifts weight-bearing to the uninvolved compartment (so-called unloading braces).

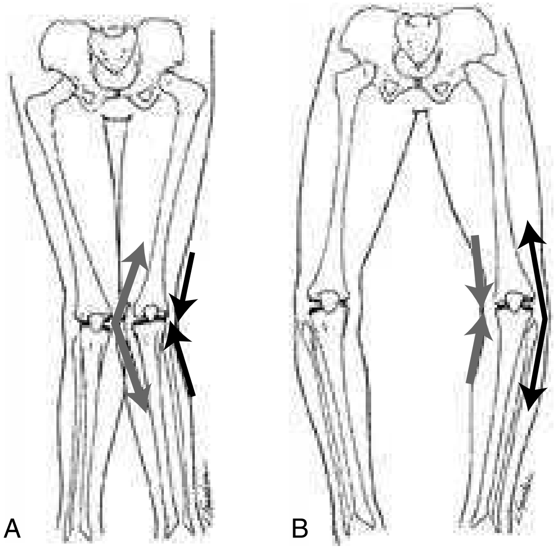


Figure 11–7 **A.** An increase in the normal tibiofemoral angle results in genu valgum, or "knock knees." Arrows on the lateral aspect of the left tibiofemoral joint indicate the presence of compression forces, whereas the arrows on the medial aspect indicate the presence of distraction (tensile) forces. **B.** A decrease in the normal tibiofemoral angle results in genu varum, or "bow legs." Arrows on the lateral aspect of the left tibiofemoral joint indicate the presence of distraction (tensile) forces, whereas arrows on the medial aspect of the joint indicate the presence of compression forces.

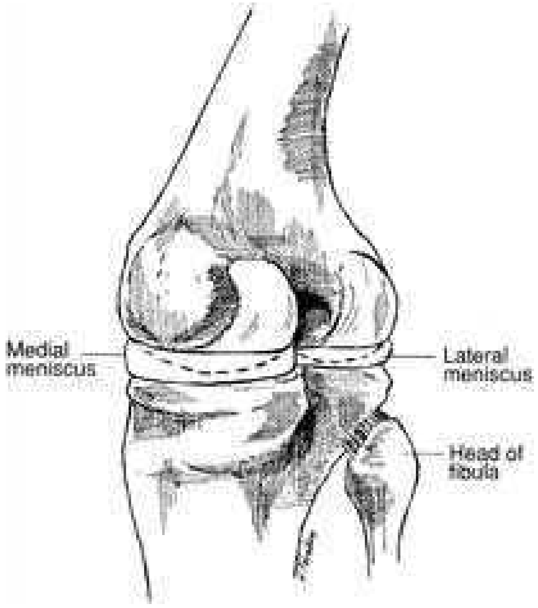


Figure 11–8 A posteromedial view of an extended right tibiofemoral joint, showing the menisci tightly interposed between the femur and the tibia. The dotted lines indicate the wedge shape of the menisci and show how the menisci deepen and contour the tibial articulating surface to accommodate the femoral condyles.

Menisci

The relative tibiofemoral incongruence is improved by the addition of **medial** and **lateral menisci,** which act to convert the convex tibial plateau into concavities for the femoral condyles (Fig. 11–8). In addition to enhancing joint congruence, these accessory joint structures play an important role in distributing weight-bearing forces, in reducing friction between the tibia and the femur, and in serving as shock absorbers.[18,19] The arrangement of mensical fibers allows axial loads to be dispersed in a radial direction, thereby reducing wear on the hyaline articular cartilage.[20–22] The menisci are fibrocartilaginous discs with a semicircular shape. The medial meniscus is C-shaped, whereas the lateral meniscus forms four fifths of a circle.[18] Lying within the tibiofemoral joint, the menisci are located on top of the tibial condyles, covering one half to two thirds of the articular surface of the tibial plateau (Fig. 11–9).[19] Both menisci are open toward the intercondylar tubercles, thick peripherally and thin centrally. The lateral meniscus covers a greater percentage of the smaller lateral tibial surface than the surface covered by the medial meniscus.[23] As a result of its larger exposed surface, the medial condyle is more susceptible to injury from the relatively greater compressive loads that pass through the medial condyle during routine daily activities. Compressive forces in the knee may reach one to two times body weight during gait and stair climbing[24,25] and three to four times body weight during running,[26] with the menisci assuming 50% to 70% of this imposed load.[18] These loads, however, can be influenced by the presence of frontal plane malalignment. The greater the degree of genu varum, for instance, the greater is the compression on the medial meniscus.

Meniscal Attachments

Meniscal motion on the tibia is limited by multiple attachments to surrounding structures, some common to both menisci and some unique to each. The medial meniscus has greater ligamentous and capsular restraints, limiting translation to a greater extent than is true for the lateral meniscus. The relative lack of mobility of the medial meniscus may contribute to its greater incidence of injury.[23,27]

The open anterior and posterior ends of the menisci are called the **anterior** and **posterior horns,** each of which is firmly attached to the tibia below.[18,28–30] Anteriorly, the menisci are connected to each other by the **transverse ligament.**[19,23] Both menisci are also attached directly or indirectly to the patella via the **patellomeniscal ligaments,** which are anterior capsular thickenings.[31] At the periphery, the menisci are connected to the tibial condyle by the **coronary ligaments,** which are composed of fibers from the knee joint capsule. Some of these connections can be seen in Figures 11–9 and 11–10. The medial meniscus is firmly attached to the joint capsule through medial thickening that extends distally from the femur to the tibia. This capsular thickening, referred to as the deep portion of the **medial collateral ligament (MCL),** contributes to the restricted motion of the medial meniscus.[23] The anterior and posterior horns of the medial meniscus are attached to the **anterior cruciate ligament (ACL)** and **posterior cruciate ligament (PCL),** respectively. Through capsular connections, the **semimembranosus muscle** connects to the medial meniscus.[32]

The anterior horn of the lateral meniscus and the anterior cruciate ligament share a tibial insertion site.[33] Posteriorly, the lateral meniscus attaches to the posterior cruciate ligament and the medial femoral condyle through the **meniscofemoral ligaments**[19,34,35] (see Fig. 11–9). In much the same way that the semimembranosus tendon is attached to the medial meniscus, the tendon of the **popliteus muscle** attaches to the lateral meniscus.[19,28] The attachment to the popliteus tendon helps restrain or control the motion of the lateral meniscus.[29]

Role of the Menisci

The strong attachments of the menisci prevent them from being squeezed out during compression of the tibiofemoral joint, allowing for greater contact area between the menisci and the femur. If the femoral condyles sat directly on the relatively flat tibial plateau, there

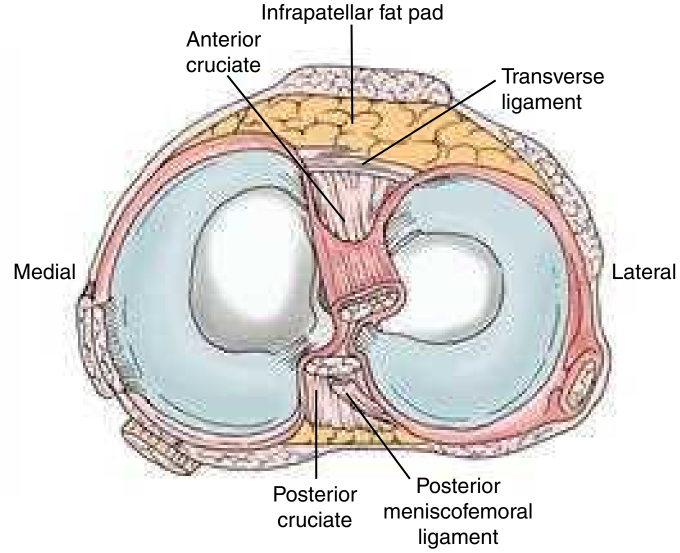


Figure 11–9 Structure of the menisci. A superior view of the menisci illustrates differences in size and configuration between the medial and lateral menisci. The medial meniscus is C-shaped, whereas the lateral meniscus is shaped like a nearly complete ring or circle. The location of the attachments of the anterior cruciate ligament and posterior cruciate ligament on the tibial plateau are also shown.

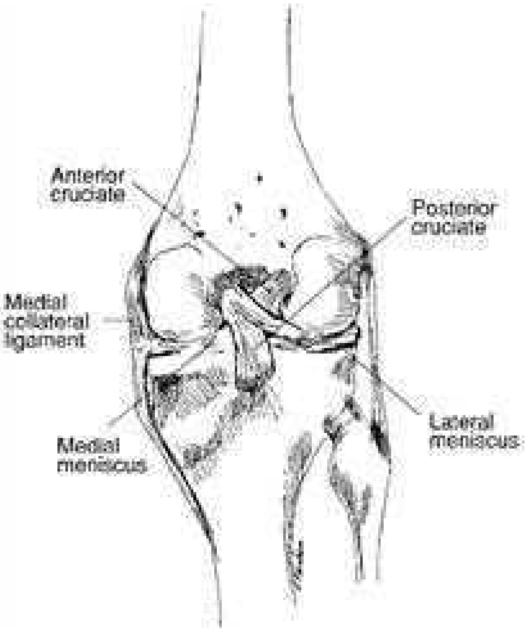


Figure 11–10 The medial meniscus is attached to the medial collateral, anterior cruciate, and posterior cruciate ligaments. The lateral meniscus is also attached to the anterior and posterior cruciate ligament (the joint capsule has been removed for visualization).

would be a small contact area between the bony surfaces. With the addition of the menisci, the contact area at the tibiofemoral joint is increased and joint stress (force per unit area) is reduced (Fig. 11–11).[18] After the removal of a meniscus, the contact area in the tibiofemoral joint is decreased, which increases joint stress and may lead to damage of the articular cartilage. Removal of the menisci nearly doubles the articular cartilage stress on the femur and multiplies the forces by six or seven times on the tibial plateau.[36] The increase in joint stress may contribute to degenerative changes within the tibiofemoral joint. For this reason, total meniscectomies are rarely performed after a meniscal tear; instead, care is taken to preserve as much of the meniscus as possible, either through débridement (removal of damaged tissue) or repair.[37] Meniscal transplants, although still considered investigational procedures, have shown promise in short-term results of improved function and relief of pain; however, long-term clinical outcomes are variable.[38–40]

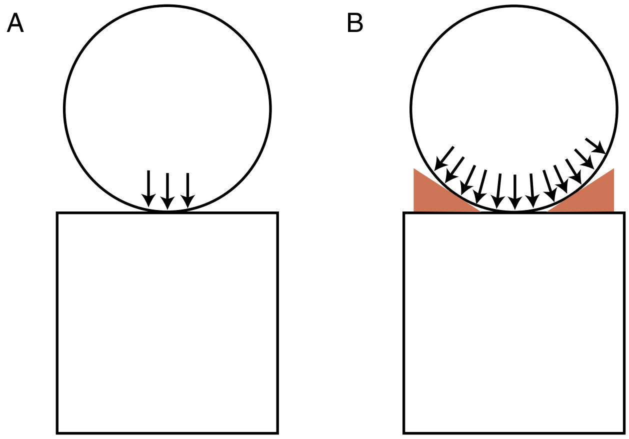


Figure 11–11 **A.** If the round block (the femoral condyles) sits on the flat block (the tibial plateau), the stress (force per unit area) is high because of the limited contact. **B.** With the addition of the soft chocks or wedges (menisci), the contact area is increased, and the stress between the blocks (bony surfaces) is reduced.

CASE APPLICATION

Alignment case 11–1

Our patient Tina's prior history of anterior cruciate ligament rupture, medial meniscal damage, medial collateral ligament healing with scar tissue, partial meniscectomy, anterior cruciate ligament reconstruction, and joint space narrowing all predispose her knee to static malalignment.[14] Malalignment in the presence of her existing knee osteoarthritis can accelerate the degenerative process.[13,15] Tina's genu varum, according to measurements taken from her radiographs, may alter the distribution of compartmental joint forces during activities of daily living, with possible subsequent cartilage degradation. The presence of Tina's frontal plane malalignment could be increasing the magnitude of the compressive force through the medial tibiofemoral joint, promoting the breakdown of the medial compartment's articular cartilage. This could have resulted in the joint space narrowing evident on her radiograph. In the presence of the greater medial compartment compressive force due to genu varum, the partial removal of the medial meniscus in her initial surgery becomes more detrimental. The partial removal of Tina's medial meniscus diminishes the contact area within the medial tibiofemoral compartment. A greater force is now being focused through a smaller contact area, creating significantly higher joint stress and raising the potential for degenerative articular cartilage changes within the joint. Furthermore Tina's quadriceps weakness and knee joint laxity may be influenced by her knee malalignment.[41] A history of compensatory strategies could lead to medial compartment irritation and may explain at least some of her medial knee pain.

Meniscal Nutrition and Innervation

The location of a meniscal lesion, along with the age and health of the patient, influences the options available after injury because of the capacity of the meniscus to heal. During the first year of life, blood vessels are contained throughout the meniscal body. Once weight-bearing is initiated, vascularity begins to diminish until only the outer 25% to 33% is vascularized by capillaries from the joint capsule and the synovial membrane.[42] After 50 years of age, only the periphery of the meniscal body is vascularized.[42] Therefore, the peripheral portion obtains its nutrition through blood vessels, but the central portion must rely on the diffusion of synovial fluid.[27] The process of fluid diffusion to support nutrition requires intermittent loading of the meniscus by either weight-bearing or muscular contractions.[42] Subsequently, during prolonged periods of immobilization or conditions of non-weightbearing, the meniscus may not receive appropriate nutrition. Furthermore, the avascular nature of the central portion of the meniscus reduces the potential for healing after an injury.[43] In adults, only the peripheral vascularized region of the meniscus is capable of inflammation, repair, and remodeling after a tearing injury.

The horns of the menisci and the peripheral vascularized portion of the meniscal bodies are well innervated with free nerve endings (nociceptors) and three different mechanoreceptors (Ruffini corpuscles, pacinian corpuscles, and Golgi tendon organs).[42,44,45] The presence of nociceptors in the meniscus could explain some of the pain felt by patients after a meniscal tear, at least for tears located in the periphery.[44] Proprioceptive deficits may potentially occur after meniscal injury as a result of injury to the mechanoreceptors within the meniscus.

Joint Capsule

Given the bony incongruence of the knee joint, even with the improvements provided by the menisci, joint stability is heavily dependent on the surrounding joint structures. The delicate balance between stability and mobility varies as the knee is flexed from full extension toward flexion. Bony congruence and overall ligament tautness are maximal in full extension, representing the close-packed position of the knee joint. In knee flexion, the periarticular passive structures tend to be lax, and the relative bony incongruence of the joint permits greater anterior and posterior translations, as well as rotation of the tibia beneath the femur.[46]

The joint capsule that encloses the tibiofemoral and patellofemoral joints is large and lax. It is grossly composed of a superficial fibrous layer and a thinner deep synovial membrane. In general, the outer fibrous portion of the capsule is firmly attached to the inferior aspect of the femur and the superior portion of the tibia.[47] Posteriorly, the capsule is attached proximally to the posterior margins of the femoral condyles and intercondylar notch and distally to the posterior tibial condyle.[48] The patella, the tendon of the quadriceps muscles superiorly, and the **patellar tendon** inferiorly complete the anterior portion of the joint capsule. The anteromedial and anterolateral portions of the capsule, as we shall see, are often separately identified as the **medial** and **lateral patellar retinaculae** or together as the **extensor retinaculum.**[49] The joint capsule is reinforced medially, laterally, and posteriorly by capsular ligaments.

The knee joint capsule and its associated ligaments are critical in restricting excessive joint motions to maintain joint integrity and normal function. Although muscles clearly play a dominant role in stabilization (as we shall examine more closely later in the chapter), it is difficult to stabilize the knee with active muscular forces alone in the presence of substantial disruption of passive restraining mechanisms of the capsule and ligaments. The joint capsule plays a role beyond that of a simple passive structure, however. The joint capsule is strongly innervated by both nociceptors as well as pacinian and Ruffini corpuscles. These mechanoreceptors may contribute to muscular stabilization of the knee joint by initiating reflex-mediated muscular responses. In addition, the joint capsule is responsible for providing a tight seal for keeping the lubricating synovial fluid within the joint space.[47]

Synovial Layer of the Joint Capsule

The synovial membrane forms the inner lining in much of the knee joint capsule.[50,51] The roles of the synovial tissue are to secrete and absorb synovial fluid into the joint for lubrication and to provide nutrition to avascular structures, such as the menisci. The synovial lining of the joint capsule is quite complex and is among the most extensive and involved in the body (Fig. 11–12). Posteriorly, the synovium breaks away from the inner wall of the fibrous joint capsule and invaginates anteriorly between the femoral condyles. The invaginated synovium adheres to the anterior aspect and sides of the anterior cruciate ligament and the posterior cruciate ligament.[51–53] Therefore, both the anterior cruciate ligament and the posterior cruciate ligament are contained within the fibrous capsule (intracapsular) but lie outside of the synovial sheath (extrasynovial).[51–53] Posterolaterally, the synovial lining delves between the popliteus muscle and lateral femoral condyle, whereas posteromedially it may invaginate between the semimembranosus tendon, the medial head of the **gastrocnemius** muscle, and the medial femoral condyle. The intricate folds of the synovium exclude several fat pads that lie within the fibrous capsule, making them intracapsular but extrasynovial, like the cruciate ligaments.[54] The anterior and posterior suprapatellar fat pads lie posterior to the **quadriceps tendon** and anterior to the distal femoral epiphysis, respectively. The **infrapatellar (Hoffa's) fat pad** lies deep to the patellar tendon (see Fig. 11–9).[54]

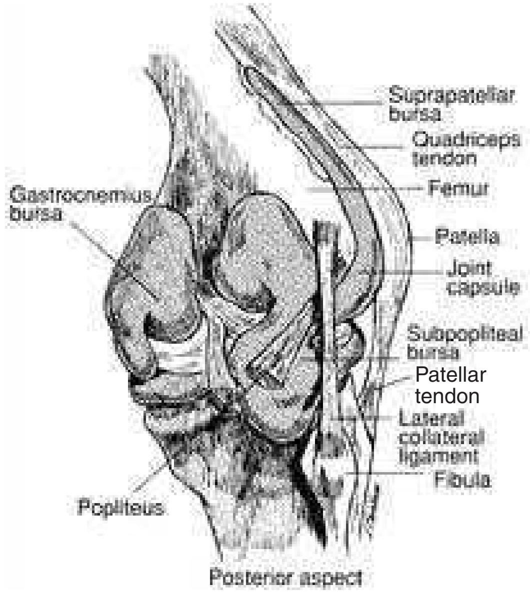


Figure 11–12 This view of the posterolateral aspect of the knee complex (with the fibrous outer layer of the capsule removed) shows the complex course of the synovial layer of the knee joint capsule, including the related bursae.

Patellar Plicae

Formation of the knee joint's synovial membrane occurs in early embryonic development.[55] Initially, the synovial membrane may separate the medial and lateral articular surfaces into separate joint cavities. By 12 weeks of gestation, the synovial septae are resorbed to some degree, which results in a single joint cavity but with retention of the posterior invagination of the synovium that forms some separation of the condyles.[51] The failure of the synovial membrane to become fully resorbed results in persistent folds in specific regions of the membrane. These folds are called **patellar plicae.**[55,56] There are four potential locations where patellar plicae may be found. Because size, shape, and frequency of these plicae vary among individuals, descriptions also vary among authors. The most frequent locations for the plicae, in descending order of incidence, are inferior (infrapatellar plica), superior (suprapatellar plica), and medial (mediopatellar plica) (Fig. 11–13).[56] There is also the potential for a lateral plica, although they are relatively rare.[55] Synovial plicae, when they exist, are generally composed of loose, pliant, and elastic fibrous connective tissue that easily passes back and forth over the femoral condyles as the knee flexes and extends.[55] On occasion, a plica may become irritated and inflamed, which leads to pain, effusion, and changes in joint structure and function, called **plica syndrome.**[55]

Continuing Exploration 11-2:

Patellar Plicae

The locations of the most commonly found patellar plicae are shown in Figure 11-13. The inferior plica, also called the **ligamentum mucosum,** is located anterior to the anterior cruciate ligament in the intercondylar area, passes through the infrapatellar fat pad (of Hoffa), and attaches to the inferior pole of the patella.[54,55,57] A superior plica is generally located superior to the patella, between the **suprapatellar bursa** and the superior portion of the patella. It connects the posterior aspect of the quadriceps tendon above to the synovial pouch at the anterior aspect of the distal femoral shaft. Despite its location above the patella, the superior plica rarely gets impinged between the patella and the femur.[55] In contrast, the medial plica is found less frequently (in only 25% to 30% of knees) than either the superior or inferior plica (found in 50% to 65% of knees), but is more often implicated as a source of pain. The medial plica arises from the medial wall of the pouch of the extensor retinaculum and runs parallel to the medial edge of the patella to attach to the infrapatellar fat pad and synovium of the inferior plicae.[55] The plica syndrome generally arises not from the most common infrapatellar plica but from either the medial or superior plica.[55,58] Discomfort can arise as the medial plica is impinged between the medial aspect of the patella and the medial femoral condyle. The great deal of pain that occurs from the irritated synovial membrane can be attributed to its rich supply of pacinian corpuscles and free nerve endings.[50]

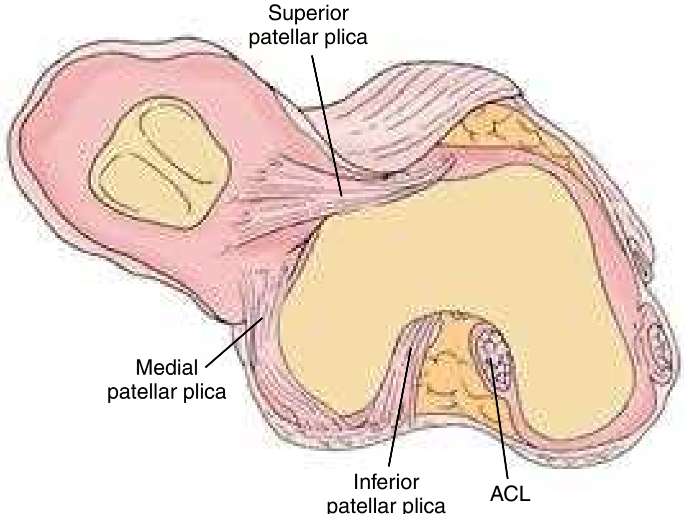


Figure 11–13 The knee joint may contain an inferior, superior, or medial patellar plica or a combination of these. These plicae are folds within the synovial layer of the joint capsule and can become irritated through repetitive trauma.

Fibrous Layer of the Joint Capsule

The fibrous joint capsule lies superficial to the synovial lining of the knee joint and provides passive support for the joint. The fibrous joint capsule itself is composed of two or three layers, depending on location. Additional structural support to the incongruent knee joint is provided by several capsular thickenings (or capsular ligaments), as well as both intracapsular and extracapsular ligaments.

The anterior portion of the knee joint capsule is called the extensor retinaculum. A fascial layer covers the distal **quadriceps muscles** and extends inferiorly. Deep to this layer, the medial and lateral retinacula are composed of a series of transverse and longitudinal fibrous bands connecting the patella to the surrounding structures (Fig. 11–14). Medially, the thickest and clinically most important band within the medial retinaculum is the **medial patellofemoral ligament (MPFL).**[59,60] Its fibers, oriented in a transverse manner, course anteriorly from the adductor tubercle of the femur to blend with the distal fibers of the **vastus medialis** and eventually insert onto the superomedial border of the patella.[60,61] The transversely oriented fibers within the lateral retinaculum, called the **lateral patellofemoral ligament,** travel from the **iliotibial (IT) band** to the lateral border of the patella.[28,49] The remainder of the retinacular bands include the obliquely oriented **medial patellomeniscal ligament,** the **lateral patellomeniscal ligament,** and the longitudinally positioned **medial** and **lateral patellotibial ligaments** (see Fig. 11–14).[28,49,59,62]

The medial portion of the joint capsule is composed of the deep and superficial portions of the medial collateral ligament. The most superficial layer of the joint capsule on the medial side of the knee joint is a fascial layer

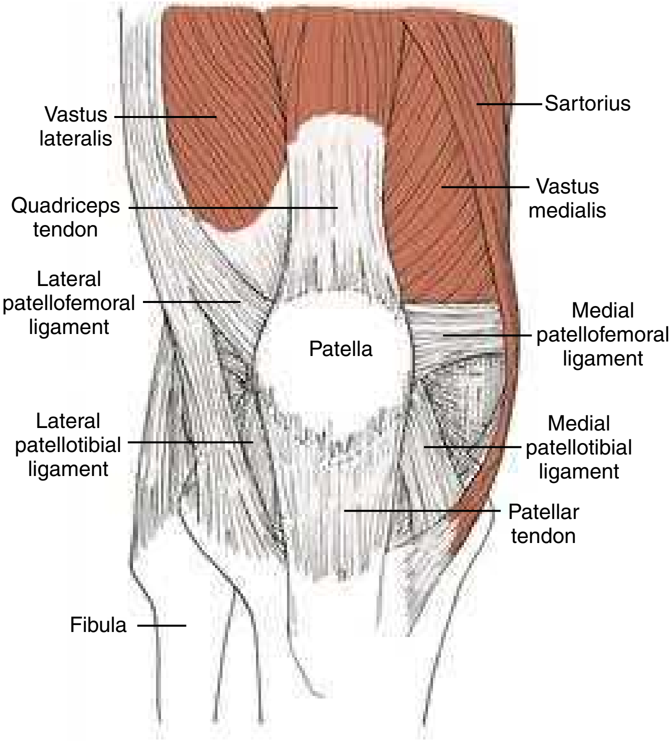


Figure 11–14 The extensor retinaculum is reinforced medially by the transversely oriented medial patellofemoral ligament and the longitudinally oriented medial patellotibial ligament. Laterally, the lateral patellofemoral ligament and lateral patellotibial ligament help resist an excessive medial glide of the patella.

that covers the vastus medialis muscle anteriorly and the **sartorius** muscle posteriorly.[63] Laterally, the joint capsule is composed superficially of the iliotibial band and its thick **fascia lata.**[28] The capsule is reinforced posterolaterally by the **arcuate ligament**[64,65] and posteromedially by the **posterior oblique ligament (POL).**[63]

Ligaments

The roles of the various ligaments of the knee have received extensive attention, which reflects their importance for knee joint stability and the frequency with which function is disrupted through injury. Given the lack of bony restraint to virtually any of the knee motions, the knee joint ligaments are variously credited with resisting or controlling the following:

1. Excessive knee extension
2. Varus and valgus stresses at the knee (attempted adduction or abduction of the tibia, respectively)
3. Anterior or posterior displacement of the tibia beneath the femur
4. Medial or lateral rotation of the tibia beneath the femur
5. Combinations of anteroposterior displacements and rotations of the tibia, together known as **rotary stabilization** of the tibia

Concept Cornerstone **11-1**

Weight-Bearing/Non-Weightbearing Versus Open/Closed Chain

Although the ligamentous checks just described were defined by the tibia's motions, it is also possible that stresses may occur on the femur while the tibia is fixed (as in weight-bearing). Such weight-bearing activities (often called "closed-chain" activities) involve motion of the femur moving on a relatively fixed tibia. In contrast, non-weightbearing activities (often termed "open-chain" activities) involve a moving tibia on a relatively fixed femur. As noted in earlier chapters, weight-bearing activities result in true closed-chain effects only when the position of the head (or trunk) is relatively fixed in space, generally to maintain the line of gravity within the base of support. Consequently, we will refer to activities as weight-bearing or non-weightbearing, rather than as "closed chain" or "open chain." The difference between weight-bearing and non-weightbearing motions is important because the displacements and rotations of the tibia and the femur are expressed differently for each condition. For example, anterior displacement of the tibia on the femur (during non-weightbearing) is equivalent to posterior displacement of the femur on the tibia (during weight-bearing) and so forth.

The large body of literature available on ligamentous function of the knee joint can be confusing and appears contradictory. This may be due to some confusion in terms as to whether the tibia or the femur is being referenced, but it is more likely due to complex and variable functioning of the knee ligaments and to dissimilar testing conditions. It is clear that ligamentous function can change, depending on the position of the knee joint, on how the stresses are applied, and on what active or passive structures are concomitantly intact. Our knowledge of the stresses and strains of ligamentous tissues has been significantly advanced using cadaveric experiments. Varying approaches have been used to reveal the behavior of the knee ligaments. One approach is to measure the amount that the knee will displace in response to an applied load. A ligament is then cut and the joint displacement is measured again with the same applied load. The relative difference in displacement provides evidence for the ligament's role in restricting motion against the magnitude and direction of the applied force. An alternative approach uses a predetermined joint displacement and measures the load necessary to achieve that displacement. A ligament is then cut, and the same preestablished displacement is applied while force values are measured. The change in the force required to displace the joint provides evidence of that ligament's role in resisting a given joint displacement.

Medial Collateral Ligament

The medial collateral ligament can be divided into a superficial portion and a deep portion that are separated by a bursa. The superficial portion of the medial collateral ligament arises proximally from the medial femoral epicondyle and travels distally to insert into the medial aspect of the proximal tibia distal to the **pes anserinus** (Fig. 11–15). The superficial portion of the medial collateral ligament attaches anterior to the origin of the posterior oblique ligament, and just distal to the femoral insertion of the medial patellofemoral ligament.[66] The deep portion of the medial collateral ligament is continuous with the joint capsule, originates from the inferior aspect of the medial femoral condyle, and inserts on the proximal aspect of

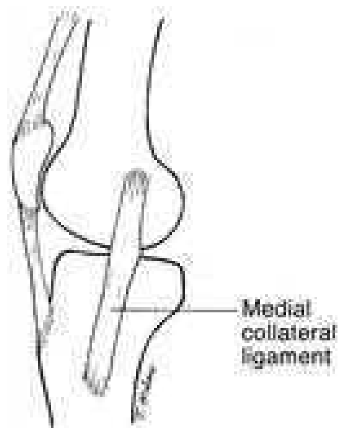


Figure 11–15 The superficial portion of the medial collateral ligament runs inferiorly from the medial femoral condyle to the anteromedial tibial condyle.

the medial tibial plateau. Throughout its course of travel, the deep portion of the medial collateral ligament is rigidly affixed to the medial border of the medial meniscus (see Fig. 11–10).[67]

The medial collateral ligament, specifically the superficial portion, is the primary restraint to excessive valgus (abduction) and lateral tibial rotation stresses at the knee.[67,68] The knee joint is better able to resist a valgus stress at full extension compared to a position of knee flexion because the medial collateral ligament is taut in extension (e.g., close-packed position). As joint flexion is increased, the medial collateral ligament becomes more lax and greater joint space opening is possible (medially gapping).[68] With the knee flexed, the medial collateral ligament plays a more critical role in resisting valgus stress despite the permitted joint gapping. Grood and colleagues determined that at close to full extension, the medial collateral ligament accounted for 57% of the restraining force against valgus opening, but at 25° of knee flexion, the medial collateral ligament accounted for 78% of the load.[69] This difference is likely due to the greater bony congruence and inclusion of other soft tissue structures (e.g., posteromedial capsule, anterior cruciate ligament) that can more effectively assist with checking a valgus stress at full extension.

The medial collateral ligament also plays a supportive role in resisting anterior translation of the tibia on the femur in the absence of the primary restraints against anterior tibial translation.[70] Because of its secondary role as a restraint to anterior tibial translation, both partial and complete medial collateral ligament tears will significantly increase the load on the anterior cruciate ligament.[71] Individuals with a medial collateral ligament injury may need protection from valgus and lateral rotation forces, especially when concomitant anterior cruciate ligament tears or newly reconstructed anterior cruciate ligament grafts are present. Importantly, the medial collateral ligament has a rich blood supply providing it with the capacity to heal when ruptured or damaged. Surgical stabilization of an isolated medial collateral ligament injury is rarely necessary, as the ligament will often heal on its own. Nevertheless, the remodeling process can take up to a year.[72,73]

Lateral Collateral Ligament

The **lateral collateral ligament (LCL)** is located on the lateral side of the tibiofemoral joint. Attaching proximally from the lateral femoral condyle, the lateral collateral ligament then travels distally to the fibular head (Fig. 11–16), where it joins with the tendon of the **biceps femoris muscle** to form the conjoined tendon.[28,74] Unlike the medial collateral ligament, the lateral collateral ligament is not a thickening of the capsule but is separate throughout much of its length and is therefore considered to be an extracapsular ligament. The lateral collateral ligament is primarily responsible for resisting varus stresses, and like the medial collateral ligament, limits frontal plane motion most successfully at full extension.[68,69] Grood and colleagues reported that at 5° of knee flexion, the lateral collateral ligament accounted for 55% of the restraining force against varus stress.[69] This capacity increased to 69% with the knee flexed to 25°. Although the lateral collateral ligament's primary role is to resist varus stresses, its orientation enables the lateral collateral ligament to limit excessive lateral rotation of the tibia as well.

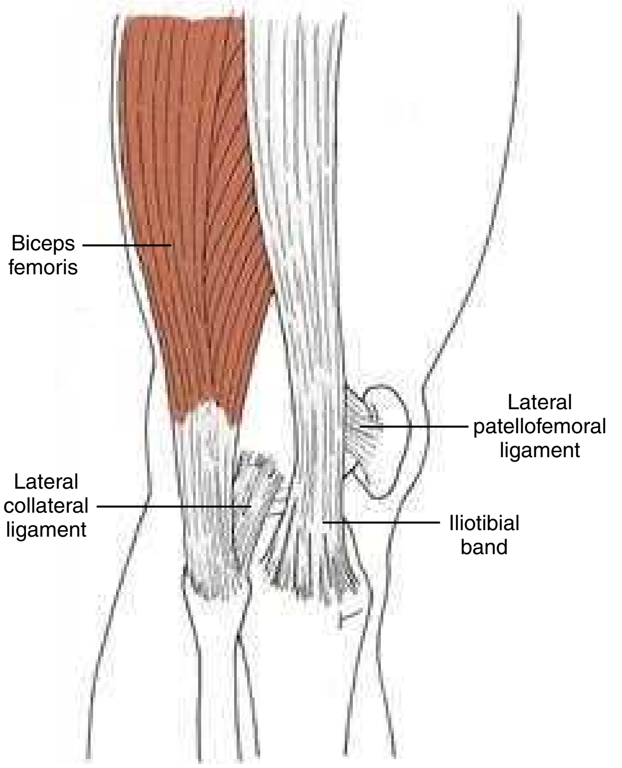


Figure 11–16 Lateral collateral ligament joins the biceps femoris muscle in a common attachment to the fibular head, whereas the iliotibial band is attached distally to the anterolateral tibia.

Anterior Cruciate Ligament

The relatively high anterior cruciate ligament injury rate among athletes and other active individuals has resulted in a substantial amount of research related to the anterior cruciate ligament's structure and function. The anterior cruciate ligament is attached distally to the tibia on the lateral and anterior aspect of the medial intercondylar tibial spine (see Fig. 11–9). It extends superiorly, laterally, and posteriorly to attach to the posteromedial aspect of the lateral femoral condyle (Fig. 11–17).[52,75] In addition, the anterior cruciate ligament twists inwardly (medially) as it travels proximally.[52] The anterior cruciate ligament may also be considered to consist of two separate bundles that wrap around each other as the knee flexes.[75] The **anteriomedial bundle (AMB)** and **posterolateral bundle (PLB)** are named for their tibial insertions[52] and have slightly different functions. The major blood supply to the anterior cruciate ligament arises primarily from the middle genicular artery.[52]

The anterior cruciate ligament functions as the primary restraint against anterior translation (anterior shear) of the

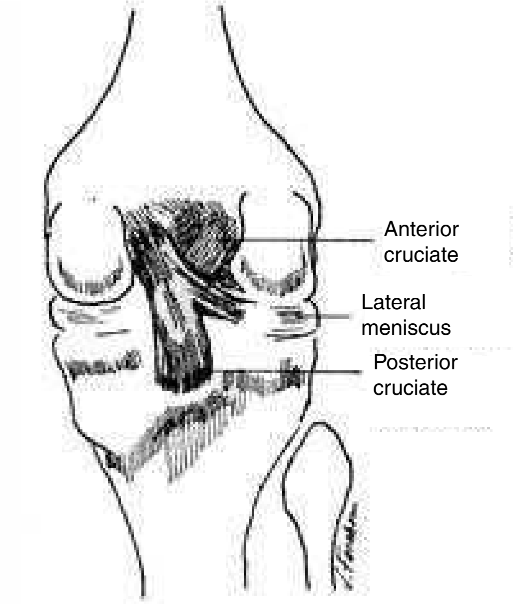


Figure 11–17 A posterior view of the knee joint shows the femoral condyles to which the anterior cruciate ligament and posterior cruciate ligament each attach.

tibia on the femur.[53] This role, however, is attributed to either the anteromedial bundle or the posterolateral bundle, depending on the knee flexion angle. With the knee close to full extension, the posterolateral bundle is taut; as the knee flexes, however, the posterolateral bundle loosens and the anteromedial bundle becomes tight, as demonstrated by the data plotted in Figure 11–18.[52,76] This shift in tension between the bundles allows some portion of the anterior cruciate ligament to remain tight at all times. In the intact joint, forces producing an anterior translation of the tibia on the femur will result in maximal tibial excursion at about 30° of flexion[77] when neither of the anterior cruciate ligament bundles are particularly tensed. In contrast, there appears to be essentially no anterior translation of the tibia possible in full extension when many of the supporting passive structures of the knee are taut (including the posterolateral bundle of the anterior cruciate ligament). In this manner, the anterior cruciate ligament is also responsible for resisting hyperextension of the knee.[78]

In addition to its primary restraint against anterior shear forces on the tibia, the anterior cruciate ligament provides rotary stability of the knee during medial/lateral rotation, varus/valgus angulations, and combinations thereof.[78–82] Rotational stability is thought to be provided to a greater extent by the posterolateral bundle than by the anteromedial bundle.[75] With valgus loading, the lengths of both bundles of the anterior cruciate ligament increase as knee flexion increases. After injury to the medial collateral ligament, a valgus moment will increase the strain on the anterior cruciate ligament throughout the flexion range. Although the anterior cruciate ligament may not make an important contribution to limiting medial rotation of the tibia, medial rotation of the tibia on the femur increases the strain on the anteromedial bundle of the anterior cruciate ligament, with the peak strain occurring between 10° and 15° of knee flexion.[79,83,84] This is most likely due to the orientation of the anterior cruciate ligament, inasmuch as it winds its way medially around the posterior cruciate ligament, becoming tighter with medial rotation. Furthermore, Li and colleagues reported that quadriceps loading of an anterior cruciate ligament deficient knee results in increased medial tibial translation.[81] The medial translation might shift the contact point of joint loading, contributing to the development of degenerative changes in the joint.

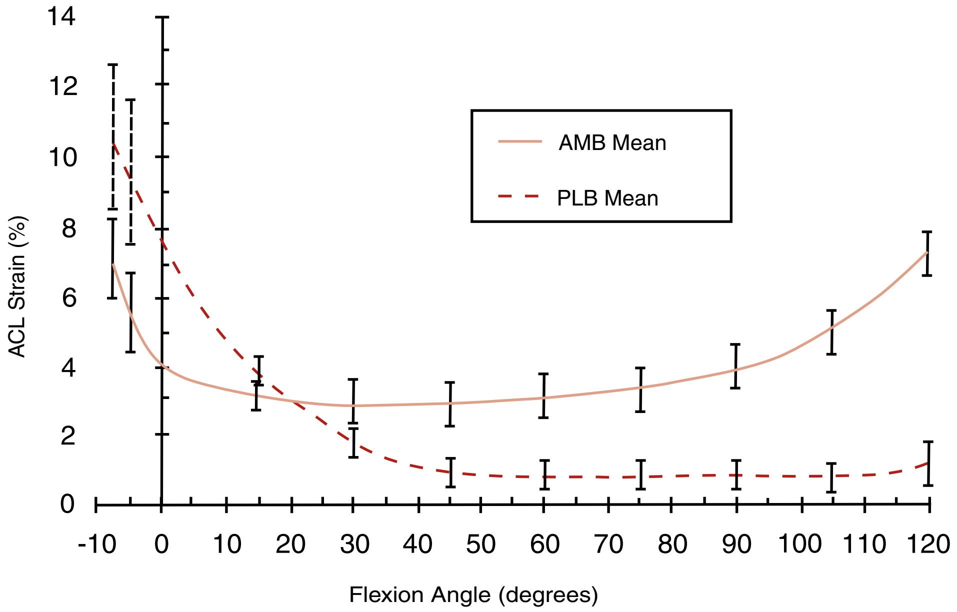


Figure 11–18 Although the anteromedial bundle (AMB) of the anterior cruciate ligament (ACL) is slack in extension and the posterolateral bundle (PLB) is slack in flexion, there is a continuum between the two, so that some portion of the anterior cruciate ligament remains fairly taut throughout the range of motion.

Continuing Exploration 11-3:

Loading the Anterior Cruciate Ligament Through Combined Motions

Markolf and colleagues demonstrated that the translational and rotational movements that individually stress the anterior cruciate ligament can in combination generate even greater stress on the ligament.[83] They determined that a combination of either varus or valgus forces with anterior translation of the tibia increases the strain on the anterior cruciate ligament, as did the combination of a valgus force and medial rotation. A combination of medial rotation and anterior translation increased the force on the anterior cruciate ligament beyond that of isolated anterior translation during a knee ROM from 5° of hyperextension to 10° of flexion. The inclusion of tibial lateral rotation with anterior tibial translation reduced the force on the anterior cruciate ligament at all knee flexion angles greater than 10°.[83]

Regardless of the rotational effect on the anterior cruciate ligament's loading pattern, injury to the anterior cruciate ligament appears to occur most commonly when the knee is slightly flexed and the tibia is rotated in either direction in weight-bearing. In flexion and medial rotation, the anterior cruciate ligament is tensed as it winds around the posterior cruciate ligament. In flexion and lateral rotation, the anterior cruciate ligament is tensed as it is stretched over the lateral femoral condyle.[85]

The muscles surrounding the knee joint are capable of either inducing or minimizing strain in the anterior cruciate ligament. With the tibiofemoral joint near full extension, an isolated quadriceps muscle contraction is capable of generating an anterior shear force on the tibia,[79,86,87] thereby increasing strain in the anterior cruciate ligament. Fleming and colleagues reported that the gastrocnemius muscle similarly has the potential to translate the tibia anteriorly and strain the anterior cruciate ligament because the proximal tendon of the gastrocnemius wraps around the posterior tibia,[88] effectively pushing the tibia forward when the muscle becomes tense through active contraction or passive stretch. The **hamstring muscles** are capable of inducing a posterior shear force on the tibia throughout the range of knee flexion,[89,90] becoming more effective in this role at greater knee flexion angles.[91] The hamstrings, therefore, have the potential to relieve the anterior cruciate ligament of some of the stress of checking anterior shear of the tibia on the femur. With the foot on the ground, the soleus muscle may also have the ability to posteriorly translate the tibia and assist the anterior cruciate ligament in restraining anterior tibial translation (Fig. 11–19).[92]

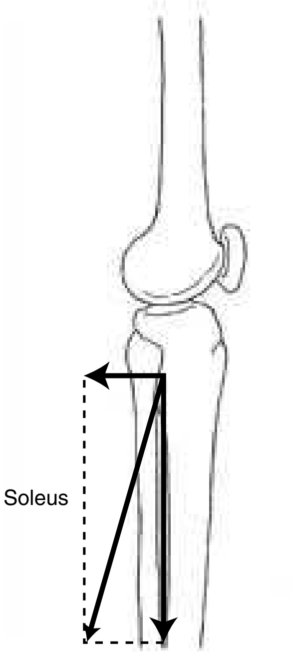


Figure 11–19 A contraction of the soleus muscle acting on the tibia in weight-bearing has a component that will produce posterior tibial translation at the knee.

Given the potential of individual muscles to either increase or decrease loads on the anterior cruciate ligament, it is not surprising that co-contraction of multiple muscles across the knee can influence the strain on the anterior cruciate ligament. For example, co-contraction of the hamstrings and quadriceps muscles may allow the hamstrings to counter the anterior translation imposed by the quadriceps to reduce the strain on the anterior cruciate ligament. In contrast, activation of both the gastrocnemius and the quadriceps muscles may result in greater strain on the anterior cruciate ligament than either muscle alone would produce,[88] unless the hamstrings are also co-contracted to mitigate the anterior translation imposed by the quadriceps and gastrocnemius.[88] Although muscular co-contraction will limit the strain imposed on the ligaments of the knee, it comes at a price. Co-contraction will reduce the anterior shear force on the tibia, but it increases joint compressive loads.[89]

CASE APPLICATION

Anterior Cruciate Ligament Injury and Tibial Shear

case 11–2

The anterior cruciate ligament tear that our patient, Tina, experienced resulted in excessive anterior laxity of the tibia on the femur. Patients will often experience episodes of giving way, including buckling, shifting, or slipping of the knee with weight-bearing, inasmuch as there appears to be an anterior shift of the tibia as the knee is loaded.[93,94] Because of the greater shearing forces across the tibiofemoral joint, there is the potential for creating more damage to the joint with each episode of instability. To avoid these episodes of giving way, early surgical stabilization is typically recommended. However, Tina chose to delay surgery and was called on to resist excessive anterior tibial translation through active muscular co-contractions. At the time of Tina's anterior cruciate

Continued

ligament reconstruction, her anterior cruciate ligament was completely ruptured. However, her preoperative therapeutic exercise program consisted of a regime to protect her healing medial collateral ligament and reduce strain on the anterior cruciate ligament on the chance that some fibers were intact. She was given an exercise program that included hamstrings-dominant exercises avoiding full flexion to protect the medial collateral ligament. Quadriceps exercises were performed with the knee flexed beyond 60° to minimize anterior cruciate ligament strain, as data from Beynnon and Fleming identified the amount of anterior cruciate ligament strain that occurred with various exercises (Fig. 11–20).[95]

Continuing Exploration 11-4:

Coping With Anterior Cruciate Ligament Deficiency

Certain individuals are capable of compensating successfully with a dynamic knee stabilization strategy in the presence of a complete anterior cruciate ligament rupture.[96–99] A percentage of athletes may even return to high-level sports that require cutting and pivoting without reconstructing the passive restraint to anterior tibial translation.[97,100] Furthermore, evidence indicates that patients who chose a nonoperative approach are at no greater risk for developing knee OA compared to those who underwent anterior cruciate ligament reconstructions.[101,102] Quadriceps strength training and neuromuscular re-education may help facilitate proper dynamic control and normal knee motion after anterior cruciate ligament injury.[103–106] However not all patients return to pre-injury activity levels after sustaining an anterior cruciate ligament rupture regardless of operative and rehabilitative interventions.[97,98,107–111]

Posterior Cruciate Ligament

The posterior cruciate ligament attaches distally to the posterior tibial surface, between the posterior horns of the two menisci[112] (see Fig. 11–9) and travels superiorly and somewhat anteriorly to attach to the lateral aspect of the medial femoral condyle (see Fig. 11–17). Like the anterior cruciate ligament, the posterior cruciate ligament is intracapsular but extrasynovial.[53] The posterior cruciate ligament is a shorter and less oblique structure than the anterior cruciate ligament, with a cross-sectional area 120% to 150% greater than that of the anterior cruciate ligament.[113] The extensive femoral attachment, size and shape of the posterior cruciate ligament possibly account for its ability to resist greater loads than the anterior cruciate ligament.[112] The posterior cruciate ligament blends with the posterior capsule and periosteum as it crosses to its tibial attachment.[114] The posterior cruciate ligament, like the anterior cruciate ligament, is typically divided into bands that are named for their tibial origins: **posteromedial bundle (PMB)** and the **anterolateral bundle (ALB).**[115] When the knee is close to full extension, the larger and stronger anterolateral bundle is lax, whereas the posteromedial bundle becomes taut. At 80° to 90° of flexion, the anterolateral bundle is maximally taut and the posteromedial bundle is relaxed.[116] In deep knee flexion, the femoral attachment of the posteromedial bundle moves away from its tibial attachment. This translation creates greater tension in the posteromedial bundle, increasing its ability to resist posterior displacement of the tibia. Concurrently, the orientation of the anterolateral bundle decreases its ability to resist tibial displacement posteriorly in full knee flexion.[112]

The posterior cruciate ligament serves as the primary restraint to posterior displacement, or posterior shear, of the tibia beneath the femur.[117] The posterior cruciate ligament restrains approximately 90% of the posterior load directed along the tibia across the majority of the knee flexion range of motion (ROM).[118] In the fully extended knee, the posterior cruciate ligament will absorb 93% of a posteriorly directed load applied to the tibia, yielding only minimal posterior displacements.[119] Unlike the anterior cruciate ligament, which resists force better at full extension, the posterior cruciate ligament is more adept at restraining posterior tibial motion with the knee flexed.[78] Although maximal posterior displacement of the tibia occurs at 75° to 90° of flexion, the secondary restraints against posterior translation become ineffective in full flexion, enhancing the role of the posterior cruciate ligament. Sectioning of the posterior cruciate ligament, therefore, increases posterior translation at all angles of knee flexion.[120] Like the anterior cruciate ligament, the posterior cruciate ligament has a role in restraining varus and valgus stresses at the knee[119] and appears to play a role in both restraining and guiding rotation of the tibia. The orientation of the posterior cruciate ligament may result in a concomitant lateral rotation of the tibia when a posterior translational force is applied to the tibia. The posterior cruciate ligament resists tibial medial rotation at 90° but less so in full extension.[78] The posterior cruciate ligament does not resist lateral rotation very well.[78]

In the absence of the posterior cruciate ligament, muscles must be recruited to actively stabilize against excessive posterior tibial translation. The popliteus muscle shares the role of the posterior cruciate ligament in resisting posteriorly directed forces on the tibia and can contribute to knee stability when the posterior cruciate ligament is absent.[113] In contrast, an isolated hamstring contraction might destabilize the knee joint in the absence of the posterior cruciate ligament because of its posterior shear on the tibia in the flexed knee. Contraction of the gastrocnemius muscle also significantly strains the posterior cruciate ligament at flexion angles greater than 40°, whereas quadriceps contraction reduces the strain in the posterior cruciate ligament at knee flexion angles between 20° and 60°.[86]

Ligaments of the Posterior Capsule

Several structures reinforce the “corners” of the posterior knee joint capsule (Fig. 11–21). The posteromedial corner of the capsule is reinforced by the semimembranosus muscle, by its tendinous expansion called the **oblique**

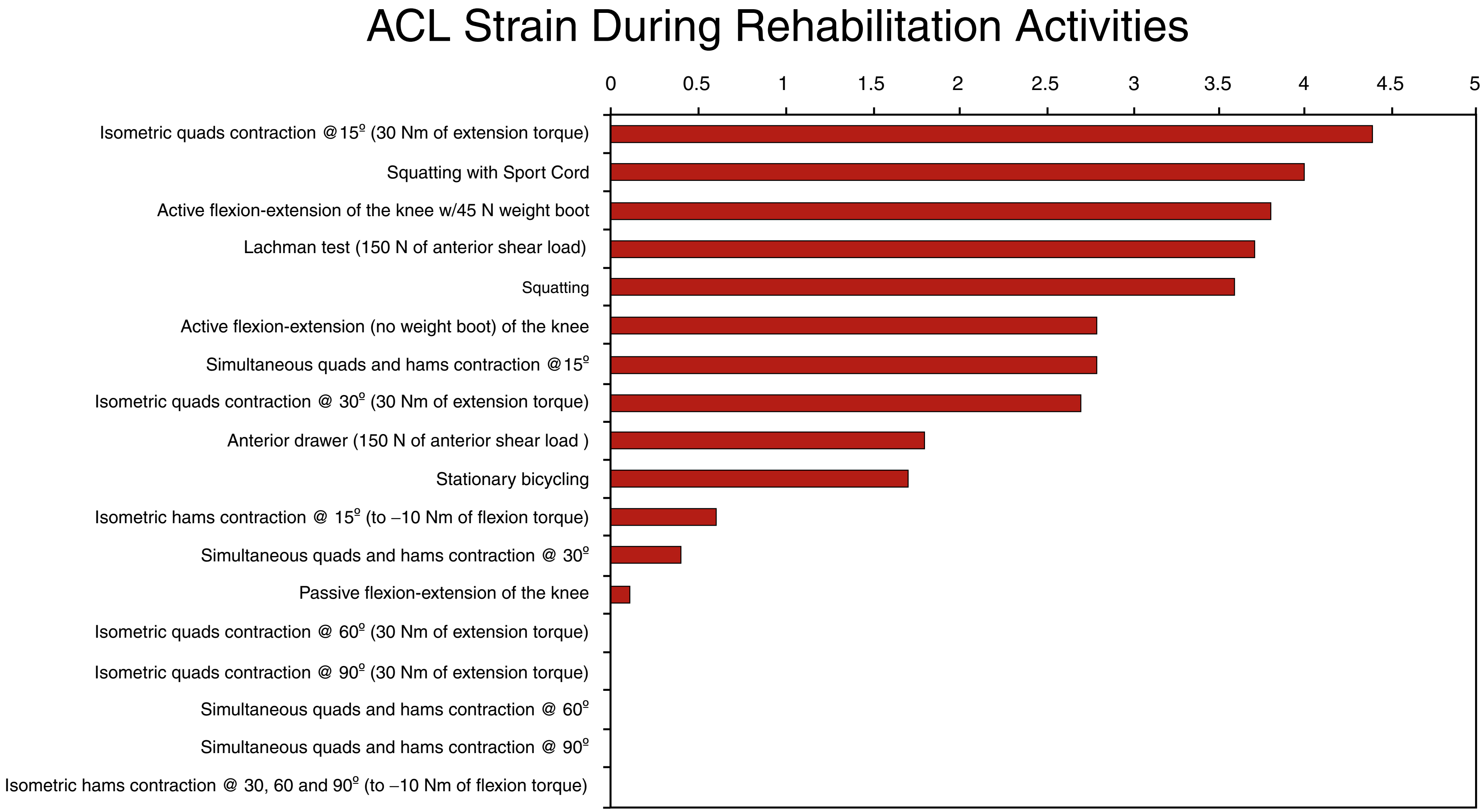


Figure 11–20 Various activities are routinely prescribed to improve muscle strength and joint function after anterior cruciate ligament tear or reconstruction. This graph provides information on the magnitude of strain on the anterior cruciate ligament during various activities. It should be noted, however that it is currently unclear as to how much strain can be detrimental to an already damaged anterior cruciate ligament or a healing graft.

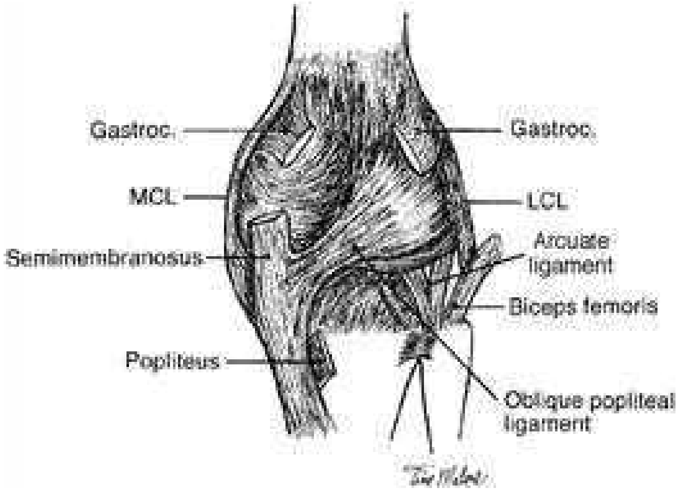


Figure 11–21 A view of the posterior capsule of the knee joint shows the reinforcing oblique popliteal ligament. Also seen are the collateral ligaments (medial collateral ligament [MCL] and lateral collateral ligament [LCL]), the arcuate ligament, and some of the reinforcing posterior musculature (semimembranosus, biceps femoris, medial and lateral heads of the gastrocnemius, and the upper and lower sections of the popliteus muscles). The medially located posterior oblique ligament is not shown because it lies superficial to the other medial capsular structures.

popliteal ligament, and by the stronger and more superficial posterior oblique ligament.[121] The posterior oblique ligament attaches posterior to the adductor tubercle and anterior to the gastrocmenius tubercle on the femur.[122] Distally, the posterior oblique ligament attaches to the tibia, distally and posterior to the distal attachment of the superficial medial collateral ligament.[123] The posterolateral corner of the capsule is reinforced by the arcuate ligament, the lateral collateral ligament, the iliotibial band, the posterolateral part of the joint capsule, and the popliteus complex (popliteus muscle, tendon, and popliteofibular ligament). The arcuate ligament is a Y-shaped capsular thickening found in nearly 70% of knees.[64] (Attachments of these ligaments are given in Table 11–1.) Both the posterior oblique ligament and the arcuate ligaments are taut in full extension and assist in checking hyperextension of the knee; the posterior oblique ligament and arcuate ligaments also check valgus and varus forces, respectively.[124] The orientation of the lateral branch of the arcuate ligament allows it to become tight in tibial lateral rotation.[125,126] After trauma involving the posterior cruciate ligament and medial collateral ligament, the posterior oblique ligament and posteromedial capsule become important stabilizers against posterior tibial translation.[127]

Continuing Exploration 11-5:

Meniscofemoral Ligaments

There are two potential portions of the meniscofemoral ligaments that have a variable presence in the human knee. The meniscofemoral ligaments are not true ligaments because they attach bone to meniscus, rather than bone to bone. When present, however, both originate from the posterior horn of the lateral meniscus and insert on the lateral aspect of the medial femoral condyle either anterior to the posterior cruciate ligament **(ligament of Humphry)** or posterior to the posterior cruciate ligament **(ligament of Wrisberg).**[18,34,35] In a review of the literature, Gupte and colleagues reported that at least one of the meniscofemoral ligaments are present in 91% of knees, with approximately 30% of knees having both of the meniscofemoral ligaments. The incidence of the posterior meniscofemoral ligament is greater than the occurrence of the anterior ligament.[34] Although the cross-sectional area of the meniscofemoral ligaments is only about 14% of that of the posterior cruciate ligament, they may assist the posterior cruciate ligament in restraining posterior translation of the tibia on the femur.[35] The meniscofemoral ligaments can also assist the popliteus muscle by checking tibial lateral rotation.

Table 11–1 Ligaments of the Posterior Knee Joint Capsule

LIGAMENT	PROXIMAL ATTACHMENT	DISTAL ATTACHMENT	FUNCTION
Oblique popliteal ligament [63]	The central part of the posterior aspect of the joint capsule	Posterior medial tibial condyle	Reinforces the posteromedial knee joint capsule obliquely on a lateral-to-medial diagonal from proximal to distal
Posterior oblique ligament [124]	Near the proximal origin of the MCL and adductor tubercle	Posteromedial tibia, posterior capsule and posteromedial aspect of the medial meniscus	Reinforces the posteromedial knee joint capsule obliquely on a medial-to-lateral diagonal from proximal to distal
Arcuate ligament: lateral branch [48, 64, 65, 125, 126]	The tendon of the popliteus muscle and the posterior capsule	The posterior aspect of the head of the fibula	Reinforces the posterolateral knee joint capsule obliquely on a medial to lateral diagonal from proximal to distal
Arcuate ligament: medial branch [48, 65]		The medical branch inserts into the oblique popliteal ligament on the medial side of the joint	

Iliotibial Band

The iliotibial (IT) band or tract is formed proximally from the fascia investing the tensor fascia lata, the gluteus maximus, and the gluteus medius muscles. The iliotibial band continues distally to attach to the lateral intermuscular septum and inserts into the anterolateral tibia (Gerdy's tubercle), reinforcing the anterolateral aspect of the knee joint (see Fig. 11–16).[74,125] The iliotibial band can be viewed as (1) a tendinous portion consisting of the proximal band to the lateral femoral epicondyle attachment, and (2) a ligamentous portion between the lateral femoral epicondyle and Gerdy's tubercle on the tibia.

Despite the muscular attachments to the proximal end of the iliotibial band, it remains an essentially passive structure at the knee joint. For example, a contraction of the tensor fascia lata (TFL) or the gluteus maximus muscles that attach to the proximal end of the iliotibial band produce only minimal longitudinal excursion of the band distally. Despite limited longitudinal movements, the iliotibial band is thought to move anterior to the knee joint axis as the knee is extended and posterior as the knee is flexed[125,126] (Fig. 11–22). Nevertheless, cadaveric studies have challenged this concept that the fibrous band moves or "rolls" over the lateral femoral epicondyle during knee flexion/extension because fibrous connections have been observed to firmly attach the iliotibial band to the femoral epicondyle.[128] Thus, the iliotibial band remains consistently taut, regardless of the position of the hip or knee. Despite its lateral location, the iliotibial band alone provides only minimal resistance to lateral joint space opening.[69]

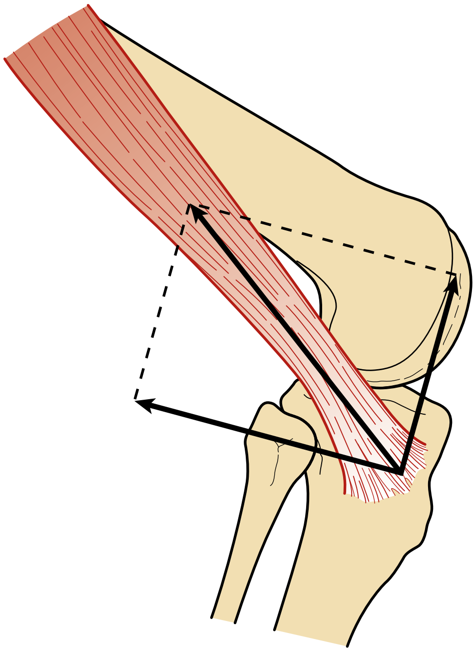


Figure 11–22 The iliotibial band provides lateral support to the knee joint. In the flexed knee, the iliotibial band tends to migrate posteriorly, increasingly its ability to restrict excessive anterior translation of the tibia under the femur.

Additional fibrous connections from the iliotibial band to the biceps femoris and **vastus lateralis** muscles form a sling behind the lateral femoral condyle, which assist the anterior cruciate ligament in restraining posterior femoral (or anterior tibial) translation with the knee near full extension.[74,129] With the knee flexed, however, the combination of the iliotibial band, the lateral collateral ligament, and the popliteal tendon can provide even greater assistance in resisting anterior displacement of the tibia on the femur (see Fig. 11–22) as well as increase the stability of the lateral side of the joint.[125] The iliotibial band also attaches to the patella via the lateral patellofemoral ligament of the lateral retinaculum. This portion of the iliotibial band become tense as the knee moves in to a flexed position.[128] As we shall see, this attachment of the iliotibial band to the lateral border of the patella may affect patellofemoral function.

Cadaveric and diagnostic studies have failed to identify a bursa between the iliotibial band and the epicondyle of the femur,[125,128,130] although adipose tissue is present between the iliotibial band and bony interface.[128] As the knee moves from full extension to 30° of flexion, compression of the highly vascularized and richly innervated adipose tissue increases between the iliotibial band and the lateral epicondyle. The adipose tissue is less compressed in full extension,[128] which may account for the complaints of pain at the distal insertion of the iliotibial band during 30° of flexion and not at full extension in patients with iliotibial band symptoms.[131]

Bursae

The extensive array of ligaments and muscles crossing the tibiofemoral joint, in combination with the large excursions of bony segments, sets up the potential for substantial frictional forces among muscular, ligamentous, and bony structures. Numerous bursae, however, prevent or limit such degenerative forces. Three of the knee joint's bursae, the suprapatellar bursa, the **subpopliteal bursa,** and the **gastrocnemius bursa,** are not separate entities but are either extensions of the capsule's synovium or communicate with the synovial lining of the joint capsule through small openings (see Fig. 11–12). The anteriorly located suprapatellar bursa lies between the quadriceps tendon and the anterior femur, superior to the patella. The posteriorly located subpopliteal bursa lies between the tendon of the popliteus muscle and the lateral femoral condyle. The gastrocnemius bursa lies between the tendon of the medial head of the gastrocnemius muscle and the medial femoral condyle. The gastrocnemius bursa continues beneath the tendon of the semimembranosus muscle to protect it from the medial femoral condyle.

The three bursae that are connected to the synovial lining of the joint capsule allow the lubricating synovial fluid to move from recess to recess during flexion and extension of the knee. In extension, the posterior capsule and ligaments are taut, and the gastrocnemius and subpopliteal bursae are compressed. This shifts the synovial fluid anteriorly (Fig. 11–23A).[132] In flexion, the suprapatellar bursa anteriorly is compressed and the fluid is forced posteriorly (Fig. 11–23B). When the knee joint is

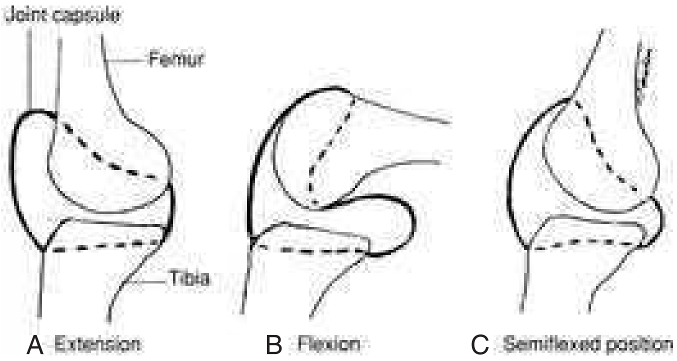


Figure 11–23 **A.** The synovial fluid is forced anteriorly during extension. **B.** In flexion, the synovial fluid is forced posteriorly. **C.** In the semiflexed position, the capsule is under the least amount of tension, and therefore this is the most comfortable position when joint effusion is present.

in the semiflexed position (e.g., loose-packed position), the synovial fluid is under the least amount of pressure (Fig. 11–23C). Clinically, when there is excess fluid within the joint cavity as a result of injury or disease (termed **joint effusion**), the semiflexed knee position helps to relieve tension in the capsule and, therefore, minimizes discomfort.

Besides the bursae that communicate with the synovial capsule, there are several other bursae associated with the knee joint (Fig. 11–24). The **prepatellar bursa,** located between the skin and the anterior surface of the patella, allows free movement of the skin over the patella during flexion and extension. The **infrapatellar bursa** lies inferior to the patella, between the patellar tendon and the overlying skin. Both the infrapatellar bursa and the prepatellar bursa may become inflamed as a result of direct trauma to the front of the knee or through activities such as prolonged kneeling. The **deep infrapatellar bursa,** located between the patellar tendon and the tibial tuberosity, helps to reduce friction between the patellar tendon and the tibial tuberosity.[133] This bursa is separated from the synovial cavity of the joint by the infrapatellar (Hoffa's) fat pad.[133] If the infrapatellar fat pad is removed, patellofemoral contact area decreases,[134] which localizes loads and increases the potential for contact surface irritation. There are also several small bursae that are associated with the ligaments of the knee joint. There is commonly a bursa between the lateral collateral ligament and the biceps femoris tendon.[135] On the medial side of the joint, small bursae can be found both superficial and deep to the superficial portion of the medial collateral ligament to protect it from the deep portion of the medial collateral ligament and the tendons of the **semitendinosus** and **gracilis** muscles, respectively.[63]

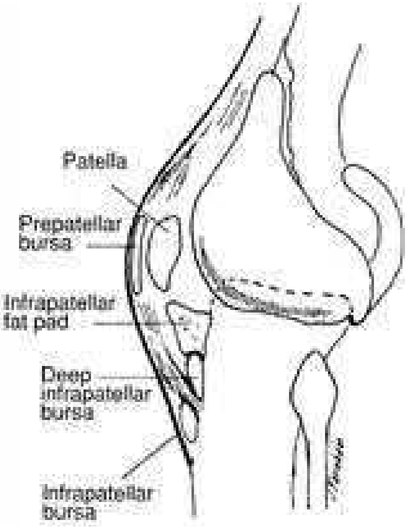


Figure 11–24 The prepatellar bursa, deep infrapatellar bursa, and infrapatellar bursa are separate from the knee joint cavity.

TIBIOFEMORAL JOINT FUNCTION

Joint Kinematics

The primary angular (or rotary) motion of the tibiofemoral joint is flexion/extension, although both medial/lateral (internal/external) rotation and varus/valgus (adduction/abduction) motions occur to a lesser extent. Each of these motions occurs about changing but definable axes. The complex bony architecture between the femoral condyles and the tibial plateau results in a constantly changing center of rotation at the knee joint. Current advances in motion analysis tracking systems have permitted accurate representation of three-dimensional descriptions of motion, with six degrees of freedom at the tibiofemoral joint.[4] Understanding the three primary axes of motion about which the knee rotates will provide the necessary framework to comprehend normal knee kinematics and to diagnose and treat abnormal motion. In addition to angular motions, translation of the tibia on the femur in an anteroposterior direction is common in both the medial and lateral compartments; to a lesser extent, medial and lateral translations can occur in response to varus and valgus forces. The small amounts of anteroposterior and medial-lateral displacements that occur in the normal knee are the result of joint incongruence and variations in ligamentous elasticity. Although these translations may be seen as undesirable, they are necessary for normal joint motions to occur. Excessive translational motions, however, should be considered abnormal and generally indicate some degree of ligamentous incompetence. We will focus here on normal knee joint motions, including both osteokinematics and arthrokinematics.

Flexion/Extension

The axis for tibiofemoral flexion and extension can be simplified as a horizontal line passing through the femoral epicondyles.[1] Although this transepicondylar axis represents an accurate estimate of the axis for flexion and extension, it should be appreciated that this axis is not truly fixed but rather shifts throughout the ROM. Much of the shift in the axis can be attributed to the incongruence of the joint surfaces.

The large articular surface of the femur and the relatively small tibial condyles create a potential problem as the femur begins to flex on the fixed tibia (e.g., weight-bearing activity). If the convex femoral condyles were permitted to roll posteriorly on the concave tibial plateau, the femur would run out of tibia and limit flexion (Fig. 11–25). For the femoral condyles to continue to roll posteriorly as flexion increases, the femoral condyles must simultaneously glide anteriorly (Fig. 11–26A). The initiation of knee flexion (0° to 25°), therefore, occurs primarily as posterior rolling of the femoral condyles on the tibia that moves the contact of the femoral condyles posteriorly on the tibial plateau. As flexion continues, the rolling of the femoral condyles is accompanied by a simultaneous anterior glide that is just sufficient to create a nearly pure spin of the femur on the posterior tibia with little linear displacement of the femoral condyles after 25° of flexion. Extension of the knee from flexion is essentially a reversal of this motion. Tibiofemoral extension occurs initially as an anterior rolling of the femoral condyles on the tibial plateau, displacing the femoral condyles back to a neutral position on the tibial plateau. After the initial forward rolling, the femoral condyles glide posteriorly just enough to continue extension of the femur as an almost pure spin of the femoral condyles on the tibial plateau (Fig. 11–26B). These interdependent osteokinematic and arthrokinematic motions describe how the femur moves on a fixed tibia (e.g., during a squat). The tibia, of course, is also capable of moving on a fixed femur (e.g., during a seated knee extension or the swing phase of gait). In this case, the movements would be somewhat different. When the tibia is flexing on a fixed femur, the tibia both rolls and glides posteriorly on the relatively fixed femoral condyles. Extension of the tibia on a fixed femur incorporates an anterior roll and glide of the tibial plateau on the fixed femur.

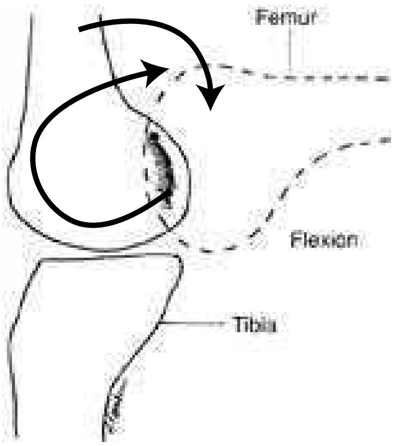


Figure 11–25 Schematic illustration of pure rolling of the femoral condyles on a fixed tibia shows the femur rolling off the tibia.

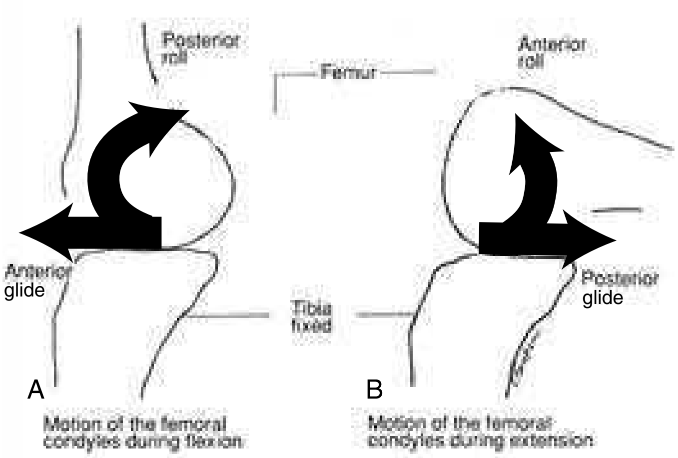


Figure 11–26 **A.** A schematic representation of rolling and gliding of the femoral condyles on a fixed tibia. The femoral condyles roll posteriorly while simultaneously gliding anteriorly. **B.** Motion of the femoral condyles during extension. The femoral condyles roll anteriorly while simultaneously gliding posteriorly.

Continuing Exploration 11-6:

Tibiofemoral Contact Points

Advancements in diagnostic imaging techniques and biomedical modeling techniques have improved the ability to accurately predict the contact points of cartilage. Li and colleagues used advanced diagnostic imaging techniques to model surface contacts points between the articular cartilage of the femur and tibia.[136] Three-dimensional motions were assessed to measure the contact points of the cartilage within the medial and lateral compartments of the knee. This investigation concluded that the contact points of the lateral tibial cartilage translated posteriorly with increased flexion, whereas the contact points of the medial tibia did not translate significantly during knee flexion. These results suggest that a rolling motion dominates the motion between the lateral femoral condyle and the lateral tibial plateau while a sliding motion occurs to a greater extent between the medial femoral condyle and the medial tibial plateau.[136]

Role of the Cruciate Ligaments and Menisci in Flexion/Extension

The arthrokinematics associated with tibiofemoral flexion and extension are somewhat dictated by the presence of the cruciate ligaments. If the cruciate ligaments are assumed to be rigid segments with a constant length,[137] posterior rolling of the femur during weight-bearing knee flexion would cause the “rigid” anterior cruciate ligament to tighten (or serve as a check rein). Continued rolling of the femur would result in the taut anterior cruciate ligament’s simultaneously creating an anterior translational force on the femoral condyles (Fig. 11–27A). During weight-bearing knee extension, the femoral condyles roll anteriorly on the tibial plateau until the “rigid” posterior cruciate ligament checks further anterior progression of the femur, creating a posterior translational force on the femoral condyles (Fig. 11–27B).

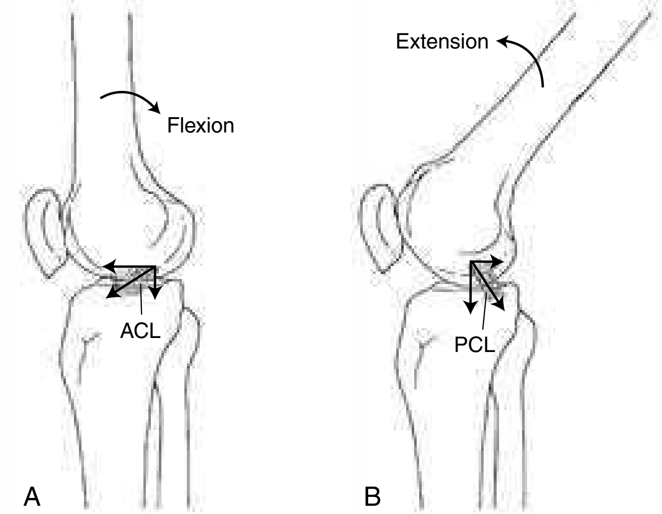


Figure 11–27 **A.** In flexion of the femur, posterior rolling of the femoral condyles creates tension in the "rigid" anterior cruciate ligament (ACL) that results in an anterior translational force imposed by the anterior cruciate ligament on the femur. **B.** In extension of the femur, anterior rolling of the femoral condyles creates tension in the "rigid" posterior cruciate ligament (PCL) that results in a posterior translational force imposed by the posterior cruciate ligament on the femur.

The anterior glide of the femur during weight-bearing flexion may be further facilitated by the shape of the menisci. Because the menisci create a circular "wedge" around the tibial plateaus, as the femoral condyles roll posteriorly on the tibial plateau, they must roll "uphill" with weight-bearing knee flexion. The oblique contact force of the menisci on the femur helps guide the femur anteriorly during flexion, while the reaction force of the femur on the menisci deforms the menisci posteriorly on the tibial plateau (Fig. 11–28).[138] Posterior deformation of the menisci occurs because the rigid attachments at the meniscal horns limit the ability of the menisci to move in its entirety.[138] Posterior deformation also allows the menisci to remain beneath the rounded femoral condyles as the condyles move on the relatively flat tibial plateau. As the knee joint begins to return to extension from full flexion, the posterior margins of the menisci return to their neutral position. As extension continues, the anterior margins of the menisci are deformed anteriorly by the femoral condyles.

The motion (or distortion) of the menisci is an important component of tibiofemoral flexion and extension. Given the need of the menisci to reduce friction and absorb the forces of the femoral condyles that are imposed on the relatively small tibial plateau, the menisci must remain beneath the femoral condyles to continue their function. The posterior deformation of the menisci during flexion is assisted by muscular mechanisms to ensure that appropriate meniscal motion occurs. During knee flexion, for example, the semimembranosus muscle exerts a posterior pull on the medial meniscus (Fig. 11–29),[32] whereas the popliteus muscle assists with posterior deformation of the lateral meniscus.[29]

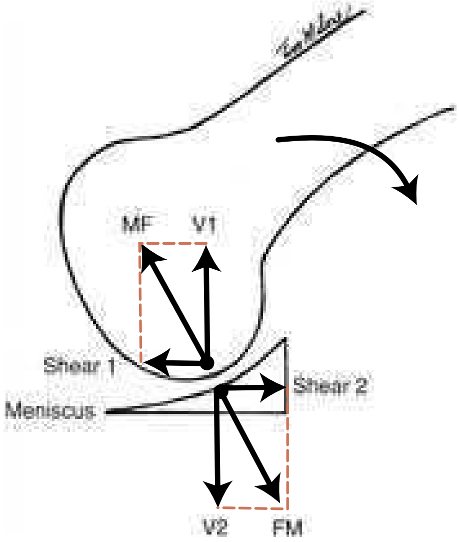


Figure 11–28 Schematically represented, the oblique contact of the femur with the wedge-shaped meniscus results in the forces of **meniscus-on-femur (MF)** and **femur-on-meniscus (FM).** These can be resolved into vertical (V1 and V2) and shear (shear 1 and shear 2) components. Shear 1 assists the femur in its forward glide during flexion, and shear 2 assists in the posterior migration of the menisci that occurs with knee flexion.

CASE APPLICATION

Meniscal Entrapment

case 11–3

Failure of the menisci to distort in the proper direction can result in limitations of joint motion and/or damage to the menisci. If the femur literally rolls up the wedge-shaped menisci in flexion (without either the anterior glide of the femur or the posterior distortion of the menisci), the increasing thickness of the menisci and the threat of rolling off the posterior margin will cause flexion to be limited. Alternatively, the stress on the meniscus (especially the less mobile medial meniscus) may cause the meniscus to tear. Similarly, failures of the menisci to distort anteriorly during extension causes the thick anterior margins to become wedged between the femur and tibia as the segments are drawn together in the final stages of extension, thus limiting extension. The failure of the meniscus or femoral condyles to move appropriately on each other may be part of the explanation for Tina's original injury to her medial meniscus, although it is likely that additional stresses to the meniscus contributed.

Flexion/Extension Range of Motion

Passive range of knee flexion is generally considered to be 130° to 140°.[139] During an activity such as deep knee squatting, knee flexion may reach as much as 160° as the hip and knee are both flexed and the body weight is superimposed on the joint. Normal gait on level ground requires approximately 60° to 70° of knee flexion, whereas ascending stairs requires about 80°, and sitting down into and

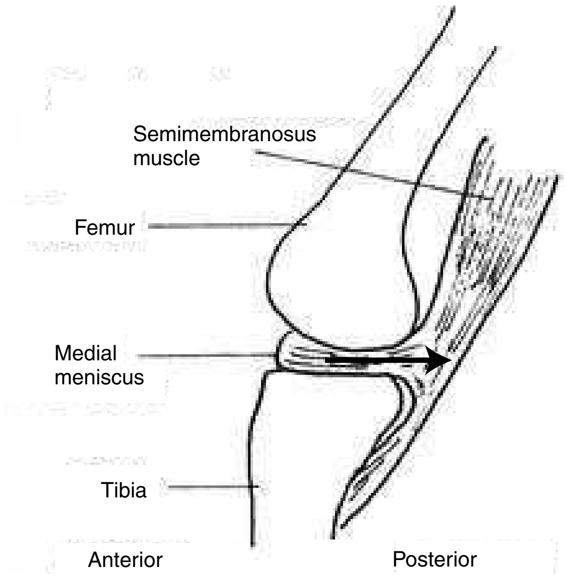


Figure 11–29 A schematic representation of the semimembranosus muscle and its attachment to the medial meniscus is shown. The arrow represents the direction of pull of the muscle on the medial meniscus during flexion.

arising from a chair requires 90° of flexion or more.[139] Knee joint extension (or hyperextension) up to 5° is considered within normal limits. Excessive knee hyperextension (e.g., beyond 5° of hyperextension) is termed **genu recurvatum.**[140]

Many of the muscles acting at the knee are two-joint muscles crossing not only the knee but also the hip or ankle. Therefore, the hip and/or ankle joint position can influence the knee joint's ROM. Passive insufficiency of the **rectus femoris** could limit knee flexion to 120° or less if the hip joint is simultaneously extended. When the lower extremity is in weight-bearing, ROM limitations at other joints such as the ankle may cause restrictions in knee flexion or extension.

Example 11-1

Ankle-Knee Interaction in Weight-Bearing

Ski boots generally hold the ankle in dorsiflexion, preventing full knee extension when the foot is on the ground (Fig. 11–30A). The choice is either to walk with flexed knees or to walk on the heels. The same problem may be created by a fixed dorsiflexion deformity in the ankle-foot complex. The opposite situation happens with a limitation to dorsiflexion. A limitation to ankle dorsiflexion (e.g., caused by tight plantarflexors) may limit the amount of knee flexion that can be performed without lifting the heel off the ground. If there is a fixed plantarflexion deformity at the ankle, the inability to bring the tibia forward in weight-bearing may result in a hyperextension deformity (genu recurvatum) at the knee (Fig. 11–30B). The relationship between ankle and knee motions when the foot is on the ground can be exploited by intentionally altering ankle joint motion (e.g., through a heel lift or an ankle-foot orthosis) to prevent or control knee motions.

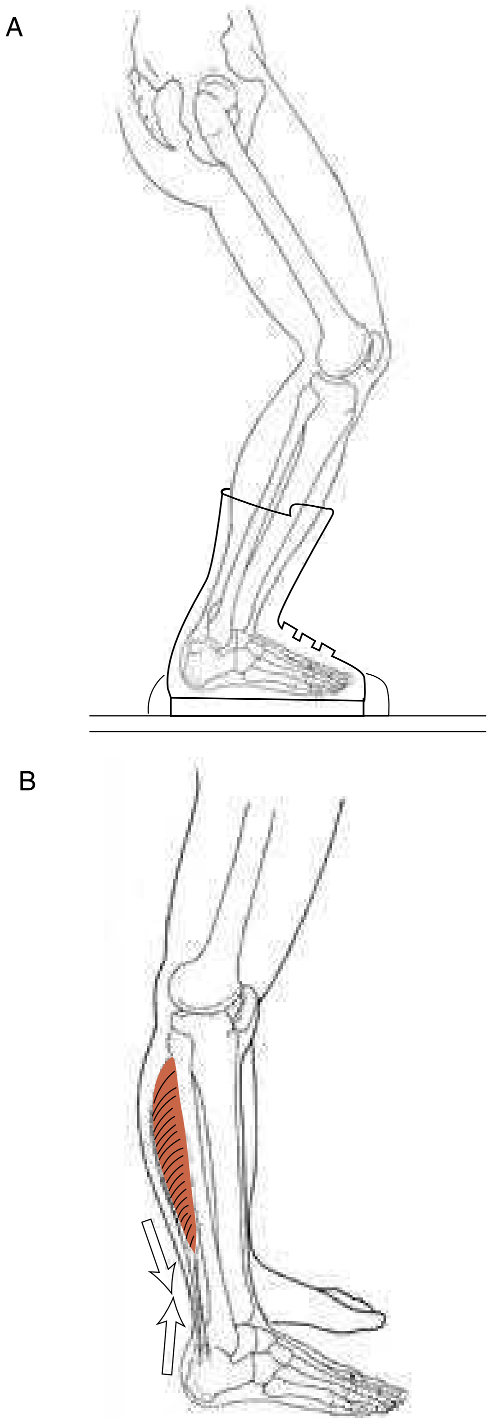


Figure 11–30 **A.** With the ankle fixed in dorsiflexion by the ski boot, the knee cannot be fully extended without the forefoot's being lifted from the ground. **B.** With a fixed plantarflexion deformity of the ankle/foot, the knee is forced into hyperextension when the foot is flat on the ground.

Medial/Lateral Rotation

Medial and lateral **(axial)** rotation of the knee joint are angular motions that describe the motion (or relative motion) of the tibia on the femur. These rotations of the knee joint occur about a longitudinal axis that runs through or close to the medial tibial intercondylar tubercle.[2,141] Consequently, the medial condyle acts as the pivot point while the lateral condyles move through a greater arc of motion, regardless of the direction of rotation (Fig. 11–31). As the tibia laterally rotates on the femur, the medial tibial condyle moves only slightly on the relatively fixed medial femoral condyle, whereas the lateral tibial condyle moves a larger distance posteriorly on the relatively fixed lateral femoral condyle. During tibial medial rotation, the medial tibial condyle again rotates only slightly, whereas the lateral condyle moves anteriorly through a larger arc of motion. With the center of rotation located within the medial tibial plateau, the contact forces are focused on a smaller area on the medial condyle whereas the contact forces are distributed over a larger surface area on the lateral tibial condyle. During both medial and lateral rotation, the knee joint's menisci will distort in the direction of movement of the corresponding femoral condyle and, therefore, maintain their relationship to the femoral condyles just as they did in flexion and extension. For example, as the tibia medially rotates (femur laterally rotates on the tibia), the medial meniscus will distort anteriorly on the tibial condyle to remain beneath the relatively anteriorly moving medial femoral condyle, and the lateral meniscus will distort posteriorly to remain beneath the posteriorly moving lateral femoral condyle. In this way, the menisci continue to reduce friction and distribute forces without restricting motion of the femur, as more solid or rigidly attached meniscal structures would do.

Axial rotation is permitted by articular incongruence and ligamentous laxity. Therefore, the range of knee joint rotation depends on the flexion/extension position of the knee. When the knee is in full extension, the ligaments are taut, the tibial tubercles are lodged in the intercondylar notch, and the menisci are tightly interposed between the articulating surfaces; consequently, very little axial rotation is possible. As the knee flexes toward 90°, capsular and ligamentous laxity increase, the tibial tubercles are no longer in the intercondylar notch, and the condyles of the tibia and femur are free to move on each other. The maximum range of axial rotation is available at 90° of knee flexion. The magnitude of axial rotation diminishes as the knee approaches both full extension and full flexion. At 90°, the total medial/lateral rotation available is approximately 35°, with the range for lateral rotation being slightly greater (0° to 20°) than the range for medial rotation (0° to 15°).[142]

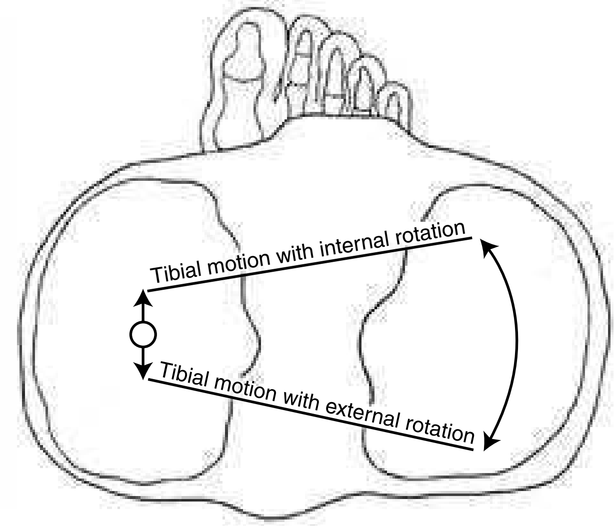


Figure 11–31 With medial/lateral rotation of the tibia, there is more motion of the lateral tibial condyle than of the medial tibial condyle in both directions; that is, the longitudinal axis for medial/lateral rotation appears to be located on the medial tibial plateau.

Valgus (Abduction)/Varus (Adduction)

Frontal plane motion at the knee, although minimal, does exist and can contribute to normal functioning of the tibiofemoral joint. Frontal plane ROM is typically only 8° at full extension, and 13° with 20° of knee flexion.[46,143] Excessive frontal plane motion could indicate ligamentous insufficiency. There is evidence that the muscles that cross the knee joint have the ability both to generate and control substantial valgus and varus torques.[144,145] When there is ligamentous laxity, the excessive varus/valgus motion or increased dynamic activity of muscles attempting to control this excessive motion could precipitate greater peak stresses across the joint.[9]

Coupled Motions

Typical tibiofemoral motions are, unfortunately, not as straightforward as we have described. In fact, biplanar intra-articular motions can occur because of the oblique orientation of the axes of motion with respect to the bony levers. The true flexion/extension axis is not perpendicular to the shafts of the femur and tibia.[146] Therefore, flexion and extension do not occur as pure sagittal plane motions but include frontal plane components termed "coupled motions" (similar to coupling that occurs with lateral flexion and rotation in the vertebral column). As already noted, the medial femoral condyle lies slightly distal to the lateral femoral condyle, which results in a physiological valgus angle in the extended knee that is similar to the physiological valgus angle that exists at the elbow. With knee flexion around the obliquely oriented axis, the tibia moves from a position oriented slightly lateral to the femur to a position slightly medial to the femur in full flexion; that is, the foot approaches the midline of the body with knee flexion just as the hand may approach the midline of the body with elbow flexion. Flexion is, therefore, considered to be coupled to a varus motion, while extension is coupled with valgus motion.

Automatic or Locking Mechanism of the Knee

There is an obligatory lateral rotation of the tibia that accompanies the final stages of knee extension that is not voluntary or produced by muscular forces. This coupled motion (lateral rotation with extension) is referred to as **automatic** or **terminal rotation.** We have already noted that the medial articular surface of the knee is longer (has more articular surface) than does the lateral articular surface (see Fig. 11–3). Consequently, during the last 30° of non-weightbearing knee extension (30° to 0°), the shorter lateral tibial plateau/femoral condyle pair completes its rolling-gliding motion before the longer medial articular surfaces do. As extension continues, the longer medial plateau continues to roll and to glide anteriorly after the lateral side of the plateau has halted. This continued anterior motion of the medial tibial condyle results in lateral rotation of the tibia on the femur, with the motion most evident in the final 5° of extension. Increasing tension in the knee joint ligaments as the knee approaches full extension may also contribute to the obligatory rotational motion, bringing the knee joint into its close-packed or locked position. The tibial tubercles have now become lodged in the intercondylar notch, the menisci are tightly interposed between the tibial and femoral condyles, and the ligaments are taut. Consequently, automatic rotation is also known as the **locking** or **screw home mechanism** of the knee. To initiate knee flexion from full extension, the knee must first be "unlocked"; that is, the laterally rotated tibia cannot simply flex but must medially rotate concomitantly as flexion is initiated. A flexion force will automatically result in medial rotation of the tibia because the longer medial side will move before the shorter lateral compartment. If there is a lateral restraint to unlocking or derotation of the femur, the joint surfaces, ligaments, and menisci can become damaged as the tibia or femur is forced into flexion. This automatic rotation or locking of the knee occurs in both weight-bearing and non-weightbearing knee joint function. In weight-bearing, the freely moving femur medially rotates on the relatively fixed tibia during the last 30° of extension. Unlocking, consequently, is brought about by lateral rotation of the femur on the tibia before flexion can proceed.

The motions of the knee joint, exclusive of automatic rotation, are produced to a great extent by the muscles that cross the joint. We will complete our examination of the tibiofemoral joint by first examining the individual contribution of the muscles, emphasizing their role in producing and controlling knee joint motion. We will then reexamine both the passive knee joint structures and the muscles in their combined role as stabilizers of this very complicated joint.

Muscles

The muscles that cross the knee are typically thought of as either flexors or extensors, because flexion and extension are the primary motions occurring at the tibiofemoral joint. Each of the muscles that flex and extend the knee has a moment arm that is capable of generating both frontal and transverse plane motions, although the moment arms for these latter motions are generally small. Therefore, each of the muscles, although grouped as flexors and extensors, will also be discussed with regard to its role in controlling frontal and transverse plane motions.

Knee Flexor Group

There are seven muscles that cross the knee joint posteriorly, and thus have the ability to flex the knee. These are the semimembranosus, semitendinosus, biceps femoris (long and short heads), sartorius, gracilis, popliteus, and gastrocnemius muscles. The **plantaris muscle** may be considered an eighth knee flexor, but it is commonly absent. With the exception of the short head of the biceps femoris and the popliteus, all of the knee flexors are two-joint muscles. As two-joint muscles, the ability to produce effective force at the knee is influenced by the relative position of the other joint that muscle crosses. Five of the flexors (the popliteus, gracilis, sartorius, semimembranosus, and semitendinosus muscles) have the potential to medially rotate the tibia on a fixed femur, whereas the biceps femoris has a moment arm capable of laterally rotating the tibia.[147] The lateral muscles (biceps femoris, lateral head of the gastrocnemius, and the popliteus) are capable of producing valgus moments at the knee, whereas those on the medial side of the joint (semimembranosus, semitendinosus, medial head of the gastrocnemius, sartorius, and gracilis) can generate varus moments.[145]

The semitendinosus, semimembranosus, and the long and short heads of the biceps femoris muscles are collectively known as the hamstrings. These muscles each attach proximally to the ischial tuberosity of the pelvis, except the short head of the biceps, which has a proximal attachment on the posterior femur. The semitendinosus muscle attaches distally to the anteromedial aspect of the tibia by way of a common tendon with the sartorius and the gracilis muscles. The common tendon is called the pes anserinus because of its shape (pes anserinus means "goose's foot") (Fig. 11–32). The semimembranosus muscle inserts posteromedially on the tibia (and, as noted earlier, has fibers that attach to the medial meniscus that can facilitate posterior distortion of the medial meniscus during knee flexion[32]). Both heads of the biceps femoris muscle attach distally to the head of the fibula, with a slip to the lateral tibia. The short head of the biceps femoris muscle does not cross the hip joint and, therefore, acts uniquely at the knee joint. The rest of the hamstring muscles cross both the hip (as extensors) and the knee (as flexors); therefore, their force producing capability at the knee is dictated by the angle of the hip joint. Greater hamstring force is produced with the hip flexed because the hamstrings are lengthened across the hip.[148] When the two-joint hamstrings are required to contract with the hip extended and the knee flexed to 90° or more, the hamstrings are shortened across both the hip and the knee. The hamstrings produce less force as knee flexion approaches the hamstring's maximal shortened position (active insufficiency) and the hamstrings must overcome the increasing tension in the lengthened rectus femoris muscle (passive insufficiency).[148] In non-weightbearing activities, the hamstrings generate a posterior shearing force of the tibia on the femur that increases as knee flexion increases,[149] peaking between 75° and 90° of knee flexion. This posterior shear or posterior translational force can reduce strain on the anterior cruciate ligament, although conceivably it increases the strain on the posterior cruciate ligament.

The gastrocnemius muscle originates by two heads from the posterior aspects of the medial and lateral condyles of the femur and attaches distally to the calcaneal (or Achilles) tendon. Except for the small and often absent plantaris muscle, the gastrocnemius muscle is the only muscle that crosses both the knee joint and the ankle joint. Much like the hamstrings' interaction with the hip joint, the gastrocnemius

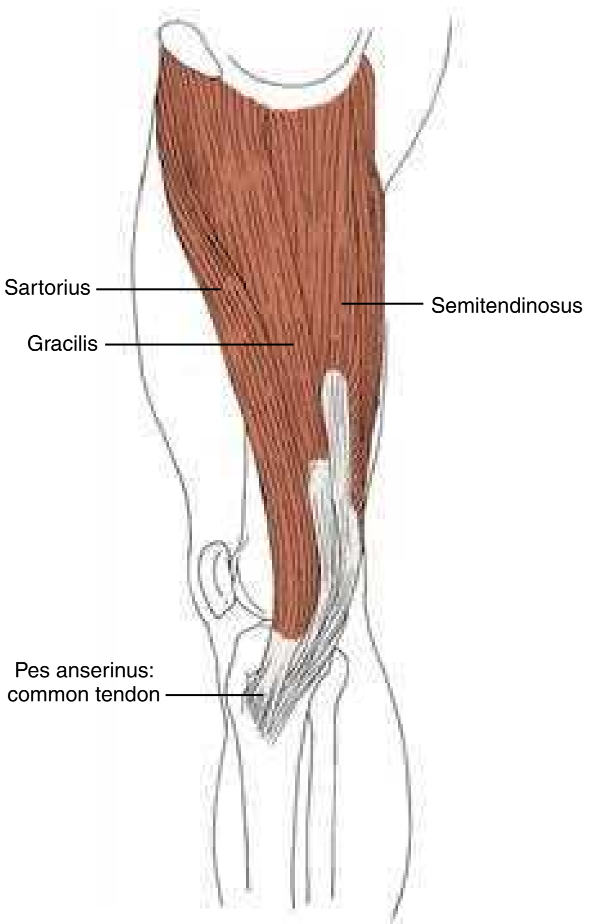


Figure 11–32 The semitendinosus, sartorius, and gracilis muscles form a common tendon (the pes anserinus) that inserts into the anteromedial tibia.

muscle quickly weakens as a knee flexor as the ankle is plantarflexed. The gastrocnemius muscle (capable of generating a large plantarflexor torque at the ankle) makes a relatively small contribution to knee flexion, producing the greatest knee flexion torque when the knee is in full extension.[150] As the knee is flexed, the ability of the gastrocnemius muscle to produce a knee flexion torque is significantly diminished.[150] The gastrocnemius muscle does, however, work synergistically with the quadriceps[93] and, during gait, may be capable of increasing the stiffness of the knee joint.[92] At the knee, therefore, the gastrocnemius muscle appears to be less of a mobility muscle than it is a dynamic stabilizer.

The sartorius muscle arises anteriorly from the anterior superior iliac spine (ASIS) and crosses the femur to insert into the anteromedial surface of the tibial shaft (most often as part of the common pes anserinus tendon). Variations in the distal attachment of the sartorius muscle are not uncommon and may be functionally relevant. When attached just anterior to its typical location, the sartorius muscle may fall anterior to the knee joint axis, serving as a mild knee joint extensor rather than as a knee flexor. Typically, however, the sartorius muscle functions as a flexor and medial rotator of the tibia. Despite its potential actions at the knee, activity in the sartorius muscle is more common with hip motion rather than with knee motion. During gait, the sartorius muscle is typically active only during the swing phase.[100]

The gracilis muscle arises from the symphysis pubis and attaches distally to the common pes anserinus tendon. The gracilis muscle functions primarily as a hip joint flexor and adductor, as well as having the capability to flex the knee joint and produce slight medial rotation of the tibia. The three muscles of the pes anserinus appear to function effectively as a group to resist valgus forces and provide dynamic stability to the anteromedial aspect of the knee joint.

The popliteus muscle is a relatively small single-joint muscle that attaches to the posterolateral aspect of the lateral femoral condyle[64] and courses inferiorly and medially to attach to the posteromedial surface of the proximal tibia.[151] The primary function of the popliteus muscle is as a medial rotator of the tibia on the femur.[147] Because medial rotation of the tibia is required to unlock the knee, the role of unlocking the knee has been attributed to the popliteus muscle. However, it should be noted that unlocking is part of automatic rotation and is due in part to the obliquity of the joint axis and the anatomy of the articular surfaces. The obligatory medial rotation of the knee joint during early flexion is a coupled motion that would likely occur even with paralysis of the popliteus muscle. The popliteus muscle does, however, play a role in deforming the lateral meniscus posteriorly[19] during active knee flexion, given its attachment to the lateral meniscus. This activity of both the semimembranosus and the popliteus muscles will generate a flexion torque at the knee, as well as contribute to the posterior movement and deformation of their respective menisci on the tibial plateau. Nevertheless, the menisci will move posteriorly on the tibial condyle even during passive flexion. Active assistance of the semimembranosus and popliteus muscles ensures that tibiofemoral congruence is maximized throughout the range of knee flexion as the menisci remain beneath the femoral condyles. This actively assisted motion of the menisci minimizes the chance that the menisci will become entrapped, which might limit knee flexion and increase the risk of meniscal injury.

The soleus and gluteus maximus muscles do not cross the knee joint. However, we would be remiss if we did not mention their function at the knee during weight-bearing activities. The soleus muscle attaches proximally to the proximal posterior aspect of the tibia and fibula and attaches distally to the calcaneal tendon. With the foot fixed on the ground during weight-bearing, a soleus muscle contraction can assist with knee extension by pulling the tibia posteriorly (Fig. 11–33). As noted earlier, the posterior pull of the soleus on the weight-bearing leg can also assist the hamstrings in restraining excessive anterior displacement of the tibia.[92] The gluteus maximus muscle, like the soleus muscle, is capable of assisting with knee extension in a weight-bearing position. It is well known that the large muscle mass of the gluteus maximus functions well as a hip extensor. With the foot flat on the ground and the knee bent, a contraction of the gluteus maximus will influence each of the joints below it. In this case, the contraction generates knee extension and ankle plantarflexion (see Fig. 11–33). The gluteus maximus, however, would produce, if anything, a posterior shear of the femur on the tibia

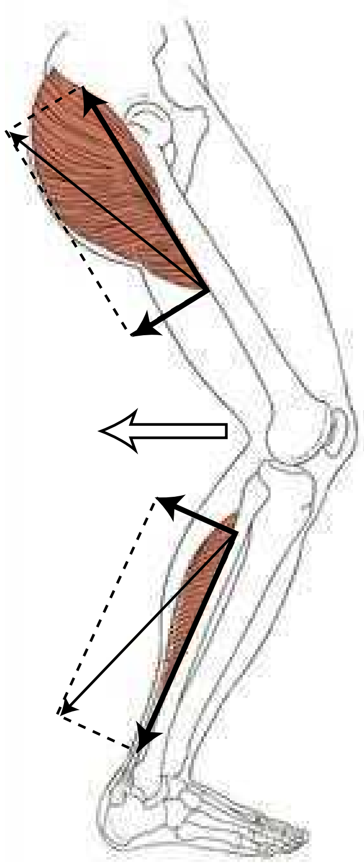


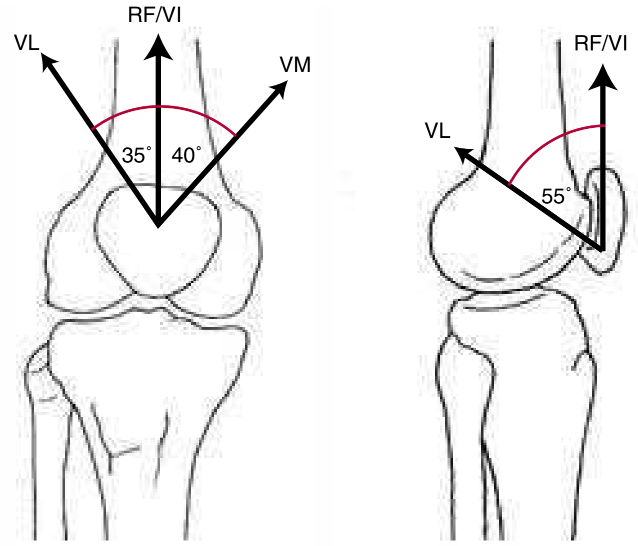
Figure 11–33 The actions of the gluteus maximus and soleus muscles can influence knee motion in weight-bearing. Although they do not cross the knee joint, these muscles are capable of assisting with knee extension.

(or a relative anterior shear of the tibia on the femur) that would increase tension in the anterior cruciate ligament unless there was offsetting co-contraction of other muscles.

Knee Extensor Group

The four extensors of the knee (rectus femoris, vastus lateralis, vastus medialis, **vastus intermedius**) are known collectively as the quadriceps femoris muscle. The only portion of the quadriceps that crosses two joints is the rectus femoris muscle, which crosses the hip and knee from its attachment on the anterior inferior iliac spine. The vastus intermedius, vastus lateralis, and vastus medialis muscles originate on the femur and merge with the rectus femoris muscle into a common tendon, called the quadriceps tendon. The quadriceps tendon inserts into the proximal aspect of the patella and then continues distally past the patella, where it is known as the patellar tendon (or **patellar ligament**). The patellar tendon runs from the apex of the patella into the proximal portion of the tibial tuberosity. The vastus medialis and vastus lateralis also insert directly into the medial and lateral aspects of the patella by way of the retinacular fibers of the joint capsule (see Fig. 11–14).

Together, the four components of the quadriceps femoris muscle function to extend the knee. In 1968, Lieb and Perry examined the direction of pull of each of the components of the quadriceps.[152] The pull of the vastus lateralis muscle alone was found to be 12° to 15° lateral to the long axis of the femur, with the distal fibers the most angled. The pull of the vastus intermedius muscle was parallel to the shaft of the femur, making it the purest knee extensor of the group. The angulation of the pull of the vastus medialis muscle depended on which segment of the muscle was assessed. The upper fibers were angled 15° to 18° medially to the femoral shaft, whereas the distal fibers were angled as much as 50° to 55° medially.[152,153] Powers and colleagues, using more current technology, reported that the resultant pull of the vastus lateralis muscle was 35° laterally, whereas the resultant pull of the vastus medialis muscle was 40° medially (Fig. 11–34A).[154] Because of the drastically different orientation of the upper and lower fibers of the vastus medialis muscle, the upper fibers are commonly referred to as the **vastus medialis longus (VML),** and the lower fibers are referred to as the **vastus medialis oblique (VMO).** The obliquity of the distal portion of the vastus medialis muscle has become the focus of attention in patients with patellofemoral pain as clinicians and researchers have attempted to try to preferentially recruit the vastus medialis oblique to maximize its medial pull on the patella. It should be noted, however, that despite the different orientation of the fibers of the vastus medialis oblique and the vastus medialis longus, these fibers are simply portions of the same muscle.[153,155] Lieb and Perry found the resultant pull of the four portions of the quadriceps muscle to be 7° to 10° in the lateral direction and 3° to 5° anteriorly in relation to the long axis of the femur.[152] Powers and colleagues, however, used a multiplane analysis and noted that the relatively large



A Frontal view

B Lateral view

Figure 11–34 With the data from Powers and colleagues,[104] the orientation of the four components of the quadriceps muscle are shown, including the vastus lateralis (VL), vastus intermedius (VI), rectus femoris (RF), and vastus medialis (VM) **(A).** The posteriorly directed vectors of the VL and VM (VL shown) were found to result in net compression of the patella against the tibia even in full extension **(B).**

vastus lateralis and vastus medialis muscles have a posterior attachment site, which results in a net posterior or compressive force that averages 55° in the extended knee (Fig 11–34B).[154] The compressive force from these muscles is present throughout the ROM but is minimized at full extension and increases as knee flexion continues.

Patellar Influence on Quadriceps Muscle Function

Function of the quadriceps muscle is strongly influenced by the patella (which, in turn, is strongly influenced by the quadriceps, as we shall see shortly). From the perspective of mechanical efficiency, the patella lengthens the moment arm of the quadriceps by increasing the distance of the quadriceps tendon and patellar tendon from the axis of the knee joint. The patella, as an anatomical pulley, deflects the action line of the quadriceps femoris muscle away from the joint center, increasing the angle of pull on the tibia to enhance the ability of the quadriceps to generate extension torque. The patella does not, however, function as a simple pulley, because in a simple pulley the tension is equal on either side of the pulley. In contrast, the tension in the patellar tendon on the inferior aspect of the patella is less than the tension in the quadriceps tendon at the superior aspect of the patella.[156]

The knee joint's geometry and the patella together dictate the quadriceps angle of pull on the tibia as the knee flexes and extends. During early flexion, the patella plays a primary role in increasing the quadriceps angle of pull. In full knee flexion, however, the patella is fixed firmly inside the intercondylar notch of the femur, which significantly reduces the patella's function as a pulley. Despite this, the quadriceps maintains a fairly large moment arm because the rounded contour of the femoral condyles deflects the muscle's action line and because the axis of rotation has shifted posteriorly into the femoral condyle. Consequently, the quadriceps maintains a reasonable ability to produce torque in full knee flexion, although the patella contributes little to its moment arm. During knee extension from full flexion, the moment arm of the quadriceps muscle lengthens as the patella leaves the intercondylar notch and begins to travel up and over the rounded femoral condyles. At about 50° of knee flexion, the femoral condyles have pushed the patella as far as it will go from the axis of rotation. The influence of the changing moment arm on quadriceps torque production is readily apparent when knee extension strength is measured throughout the ROM. Peak torques are often observed at approximately 45° to 60° of knee flexion, a region in which both the moment arm and the length-tension relationship of the muscle are maximized.[157] Finally, with continued extension, the moment arm will once again diminish.[158]

Although the patella's effect on the quadriceps' moment arm is diminished in the final stages of knee extension, the small improvement in joint torque provided by the patella may be most important here. Near end range extension, the quadriceps is in a shortened position, which reduces its ability to generate active tension. The decreased ability of the quadriceps to produce active force makes the relative size of the moment arm critical to torque production in the last 15° of knee extension.

Continuing Exploration 11-7:

Quadriceps Lag

If there is substantial quadriceps weakness or if the patella has been removed because of trauma (a procedure known as a patellectomy), the quadriceps may not be able to produce adequate torque to complete the last 15° of nonweightbearing knee extension. This can be seen clinically in a patient who demonstrates a "quad lag" or "extension lag." For example, the patient may have difficulty maintaining full knee extension while performing a straight leg raise (Fig. 11–35). With the tibiofemoral joint in greater flexion, removal of the patella or quadriceps weakness will have less effect on the ability of the quadriceps to generate extension torque because the femoral condyles also serve as a pulley, and the total muscle tension of the quadriceps will be greater than in the muscle's shortened state. The patient will not have a "quad lag" in weightbearing because the soleus and gluteus maximus muscles can assist the quadriceps with knee extension once the foot is fixed.

Figure 11–35 Severe quadriceps weakness can result in a quadriceps lag ("quad lag") during a straight-leg–raise exercise. Near full extension, the patella increases the moment arm only slightly, and the decreased length-tension relationship of the already weakened quadriceps renders it incapable of generating sufficient torque to complete the range of motion.

The patella's role in increasing the quadriceps' angle of pull on the tibia enhances torque production, but at a cost. Increasing the quadriceps' moment arm, by definition, increases the rotary component of the pull of the quadriceps on the tibia. The quadriceps, however, not only produces an extension torque but also creates an anterior shear of the tibia on the femur (Fig. 11–36A). This anterior translational force must be resisted by active or passive forces capable of either producing a posterior tibial translation or passively resisting the anterior tibial translation imposed by the quadriceps. The anterior cruciate ligament represents the most prominent passive restraint to the imposed anterior

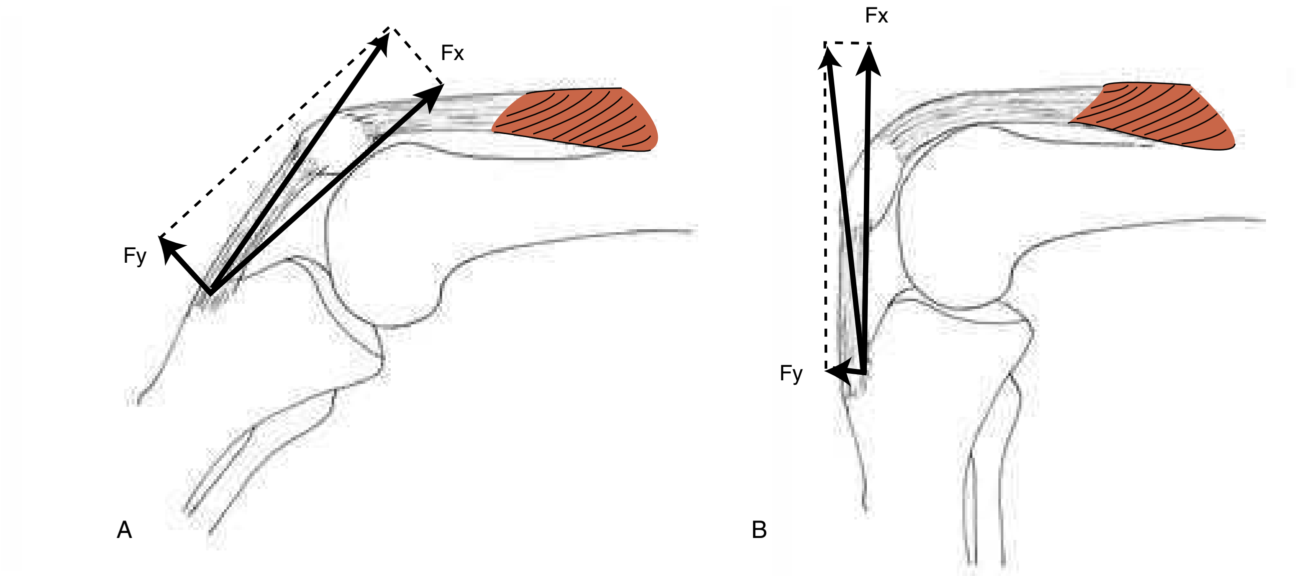


Figure 11–36 **A.** With the knee close to full extension, a forceful quadriceps contraction is capable of inducing an anterior tibial translation. **B.** Once the knee is flexed to greater than 60°, little to no anterior translation occurs.

tibial translation of the quadriceps. Increases and decreases in the angle of pull of the quadriceps are accompanied by concomitant increases and decreases in stress in the anterior cruciate ligament. The strain on both bundles of the anterior cruciate ligament ordinarily increases as the knee joint approaches full extension. In the absence of passive stabilizers such as the anterior cruciate ligament, a quadriceps contraction near full extension has the potential to generate a large anterior tibial translation,[87] which the patient may describe as the knee "giving way." The strain on the anterior cruciate ligament evoked by a quadriceps contraction is substantially diminished as the knee is flexed beyond 60° because the shear component of the quadriceps diminishes from its maximum value (Fig. 11–36B).

CASE APPLICATION

Consequences of Anterior Cruciate Ligament Injury

case 11–4

Although Tina's anterior cruciate ligament has been reconstructed, she had to go for some time without an anterior cruciate ligament. During that time, she had to restrain excessive anterior tibial translation caused by a forceful quadriceps contraction or ground reaction forces with muscles, such as the hamstrings, gastrocnemius, and soleus muscles. All these muscles can help restrain the tibia or, along with the gluteus maximus, stiffen the knee to minimize movement. Although this can be helpful for maintaining knee joint stability, there may be detrimental consequences. Large amounts of co-contraction in muscles crossing the knee joint can increase tibiofemoral compression, because these muscles produce substantially larger joint compression than rotation or shear components. In addition, tendinitis may develop in muscles that are overworked from trying actively to maintain joint stability. The moderate joint space narrowing in the medial tibiofemoral compartment evident in Tina's weight-bearing radiographs may be associated (along with other factors) with excessive shear and compressive forces during her 4-month period without an anterior cruciate ligament. Furthermore, tibiofemoral motion is not restored to "normal" three-dimensional kinematics after anterior cruciate ligament reconstruction,[159,160] likely contributing to the increased prevalence of osteoarthritis after anterior cruciate ligament injury.[101,102]

During weight-bearing activities, the quadriceps' activity in knee extension is influenced by a number of other factors. Muscles such as the soleus and gluteus maximus muscles are capable of assisting with knee joint extension. When an erect posture is attained, activity of the quadriceps is minimal because the line of gravity passes just anterior to the knee axis for flexion/extension, which results in a gravitational extension torque that maintains the joint in extension. The posterior joint capsule, ligaments, and largely passive posterior muscles maintain equilibrium by offsetting the gravitational torque and preventing hyperextension. In weight-bearing with the knee somewhat flexed, as during a squat or when someone cannot fully extend the knee (as in the case of a flexion contraction), the line of gravity will pass posterior to the knee joint axis. The gravitational torque will now tend to promote knee flexion, and activity of the quadriceps is necessary to counterbalance the gravitational torque and maintain the knee joint in equilibrium. Because the quadriceps femoris muscle has the responsibility of supporting the body weight and resisting the force of gravity, it is about twice as strong as the hamstring muscles. Although the hamstrings perform a similar function in supporting the body weight when there is a gravitational flexion moment at the hip, the hamstrings are assisted in this function by the large gluteus maximus. The quadriceps, however, are the primary knee joint extensors.

Clearly, the quadriceps muscle functions differently, depending on the activity or the exercise condition. During non-weightbearing knee extension, the moment arm of the resistance (e.g., weight of the leg plus external resistance) is minimal when the knee is flexed to 90° but increases as knee extension progresses (Fig. 11–37). Therefore, greater quadriceps force is required as the knee approaches full extension. The opposite happens during weight-bearing activities. In a standing squat, the moment arm of the resistance (e.g., the superimposed body weight) is minimal when the knee is extended and increases with increasing knee flexion (Fig. 11–38). Therefore, during weight-bearing activities such as a squat, the quadriceps muscle must produce greater force with increased knee flexion.[161]

Continuing Exploration 11-8:

Quadriceps Strengthening: Weight-Bearing Versus Non-Weightbearing

Weight-bearing exercises are often prescribed after anterior cruciate ligament, posterior cruciate ligament, or patellofemoral injury on the premise that they are less stressful, more like functional movements, and safer than non-weightbearing exercises. Wilk and colleagues computationally estimated anteroposterior shear force, compression force, and extensor torque at the knee.[162] These investigators compared the forces during weight-bearing and non-weightbearing exercises that are commonly used for quadriceps muscle strengthening. Findings concluded that the weight-bearing quadriceps exercises of a squat and leg press resulted in a posterior tibial shear force at the knee throughout the entire ROM, peaking between 83° and 105° of knee flexion.[162,163] The posterior shear would presumably stress the posterior cruciate ligament. In contrast, there was an anterior shear force in a non-weightbearing knee extension exercise when the quadriceps actively extended the knee from 40° to 10°, with the maximal anterior shear occurring between 20° and 10°. A posterior shear force was also found during non-weightbearing exercise, but this force was present only between 60° and 101° of flexion. This study demonstrated that the stress on the posterior cruciate ligament that is present during some types of weight-bearing exercises may actually be detrimental to the healing process if this ligament is damaged.[162,163]

Beynnon and colleagues measured anterior cruciate ligament strain values in vivo to compare strain values during commonly performed therapeutic exercises.[95,164] Similar anterior cruciate ligament strain values were found regardless of whether the therapeutic exercises were performed during weight-bearing or non-weightbearing activities.[95,164] For example, one-legged sit to stands, lunges, step ups and step downs (weight-bearing) produced anterior cruciate ligament strain values similar to active knee extension exercises (non-weightbearing). Furthermore, higher anterior cruciate ligament strains occurred when the quadriceps contracted between 50° to 0° of knee flexion regardless of whether the exercise was closed or open chain.[95,164]

The results of the computationally derived forces and experimentally measured strains question the rationale of basing rehabilitation exercises on the premise of that weight-bearing or non-weightbearing protocols are inherently better, safer or more effective.

Stabilizers of the Knee

Since the beginning of this chapter, we have identified the role of both passive (capsuloligamentous) and active (muscular) forces in contributing to stability of the tibiofemoral joint. The contributions of both muscles and capsuloligamentous structures to maintaining appropriate joint stability are dependent on the position not only of the knee joint but also of the surrounding joints, the magnitude and direction of the applied force, and the availability of secondary restraints. There can also be considerable variation among individuals (as well as between knees in the same individual) that contributes to the diversity of findings observed by both clinicians and researchers. Although admittedly an oversimplification, Table 11–2 summarizes the potential

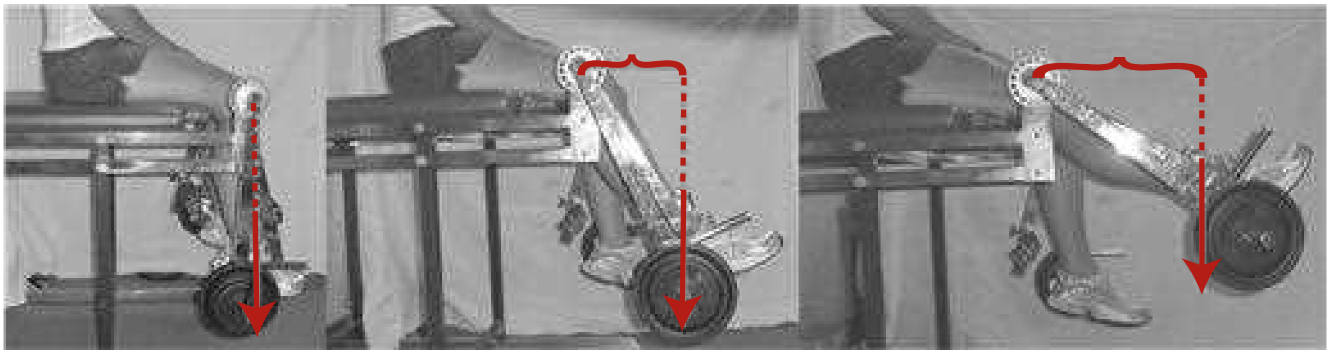


Figure 11–37 During non-weightbearing exercises, the quadriceps muscle must generate more torque (and more force) as the knee approaches full extension to overcome the increasing moment arm (and torque) of the resistance.

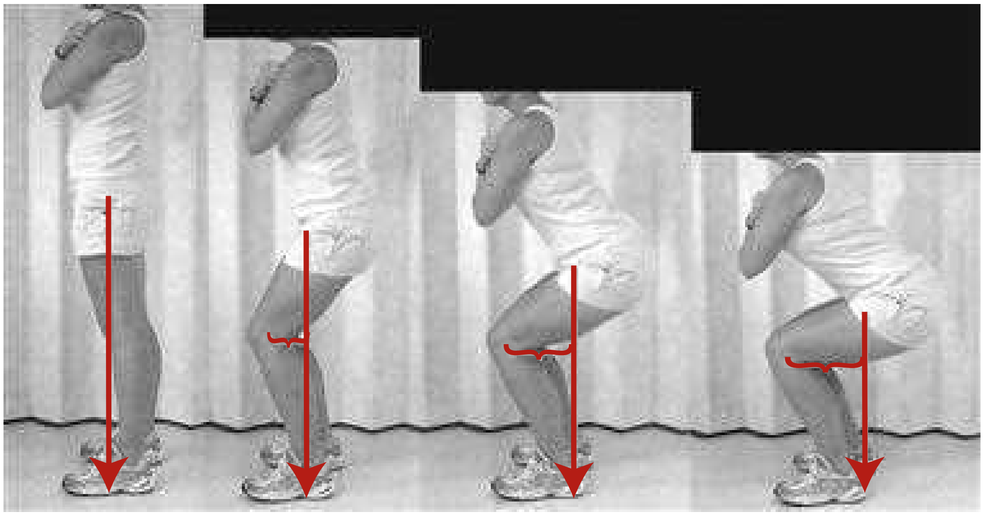


Figure 11–38 In a weight-bearing exercise, the quadriceps must generate more torque (and more force) as knee flexion increases to control the increasing moment arm (and torque) of the superimposed body weight at the knee joint.

Table 11–2 **Summary of Knee Joint Stabilizers***

	STRUCTURES	FUNCTION
A-P/hyperextension stabilizers	Anterior cruciate ligament Iliotibial band Hamstring muscles Soleus muscle (in weight-bearing) Gluteus maximus muscle (in weight-bearing)	Limit anterior tibial (or posterior femoral) translation
	Posterior cruciate ligament Meniscofemoral ligaments [35] Quadriceps muscle Popliteus muscle Medial and lateral heads of gastrocnemius [88]	Limit posterior tibial (or anterior femoral) translation
Varus/valgus stabilizers	Medial collateral ligament Anterior cruciate ligament Posterior cruciate ligament Arcuate ligament Posterior oblique ligament Sartorius muscle Gracilis muscle } Pes anserinus Semitendinosus muscle Semimembranosus muscle Medial head of gastrocnemius muscle	Limit valgus of tibia
	Lateral collateral ligament Iliotibial band Anterior cruciate ligament Posterior cruciate ligament Arcuate ligament Posterior oblique ligament Biceps femoris muscle Lateral head of gastrocnemius muscle	Limit varus of tibia
Medial/lateral rotational stabilizers	Anterior cruciate ligament Posterior cruciate ligament Posterior medial capsule Meniscofemoral ligament Biceps femoris	Limit medial rotation of tibia

Continued

Table 11–2 Summary of Knee Joint Stabilizers*—cont'd

	STRUCTURES	FUNCTION
Medial/lateral rotational stabilizers (cont'd)	Posterolateral capsule [124] Popliteus muscle Sartorius muscle Gracilis muscle } Pes anserinus Semitendinosus muscle Semimembranosus muscle Medial collateral ligament Lateral collateral ligament	Limit lateral rotation of tibia

*The contribution of both muscles and capsuloligamentous structures depends on the position of both the knee and contiguous joints, the magnitude and direction of the applied force, and the availability of secondary restraints. Findings vary among investigators, given the testing conditions.
A-P, anteroposterior.

contribution of the different structures that limit anteroposterior translation or knee joint hyperextension, varus/valgus rotation, and medial/lateral rotation of the knee joint.

Table 11–2 describes stability in terms of straight plane movements. In reality, more complicated motions are possible, requiring coupled stability, or rotary stability (a combination of uniplanar motions) (Table 11–3). For example, injury to the posterolateral corner (e.g., posterolateral joint capsule, popliteus muscle, arcuate ligament) can yield posterior instability and excessive lateral tibial rotation. This is termed **posterolateral instability.** In contrast, damage to the posterior oblique ligament, medial hamstrings, medial collateral ligament, and posteromedial joint capsule contribute to **posteromedial instability.**

Table 11–3 Components to Rotary Stability

	MEDIAL	LATERAL
	*Anteromedial Stability**	*Anterolateral Stability†*
Anterior	Medial collateral ligament Posterior oblique ligament Posteromedial capsule Anterior cruciate ligament	Anterior cruciate ligament Lateral collateral ligament Posterolateral capsule Arcuate complex/popliteus Iliotibial band
	Posteromedial Stability	*Posterolateral Stability*
Posterior	Posterior cruciate ligament Posterior oblique ligament Medial collateral ligament Semimembranosis Posteromedial capsule Anterior cruciate ligament	Posterior cruciate ligament Arcuate complex/popliteus Lateral collateral ligament Biceps femoris Posterolateral capsule

*Indicates that the following active and passive stabilizers are capable of resisting one or more of the following: anterior translation, valgus, or external rotation of the tibia.
†Indicates that the following active and passive stabilizers are capable of resisting one or more of the following: anterior translation, varus, or internal rotation of the tibia.

The extensor retinaculum, which is composed of fibers from the quadriceps femoris muscle, fuses with fibers of the joint capsule to provide dynamic support for the anteromedial and anterolateral aspects of the knee.

PATELLOFEMORAL JOINT

Embedded within the quadriceps muscle, the flat, triangularly shaped patella is the largest sesamoid bone in the body. The patella is an inverted triangle with its apex directed inferiorly. The posterior surface is divided by a vertical ridge and covered by articular cartilage (Fig. 11–39). This ridge is situated approximately in the center of the patella, dividing the articular surface into approximately equally sized medial and lateral facets. Both the medial and lateral facets are flat to slightly convex side to side and top to bottom. Most patellae also have a second vertical ridge toward the medial border that separates the medial facet from an extreme medial edge, known as the **odd facet** of the patella (see Fig. 11–39).[165] The posterior surface of the patella in the extended knee sits on the femoral sulcus (or patellar surface) of the anterior aspect of the distal femur (Fig. 11–40). The femoral sulcus has a groove that corresponds to the ridge on the posterior patella and divides the sulcus into medial and lateral facets. The lateral facet of the femoral sulcus is slightly more convex than the medial facet and has a more highly developed lip than does the medial surface (see Fig. 11–2). The patella is attached to the tibial tuberosity by the patellar tendon. Given the shape of the articular

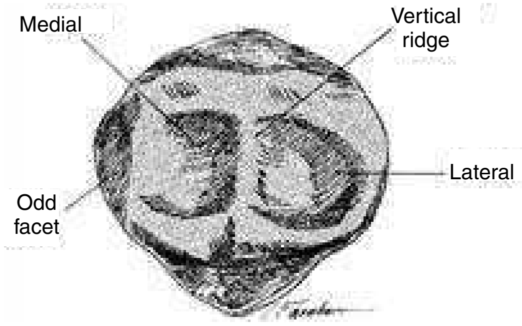


Figure 11–39 Articulating surfaces on the posterior aspect of the patella.

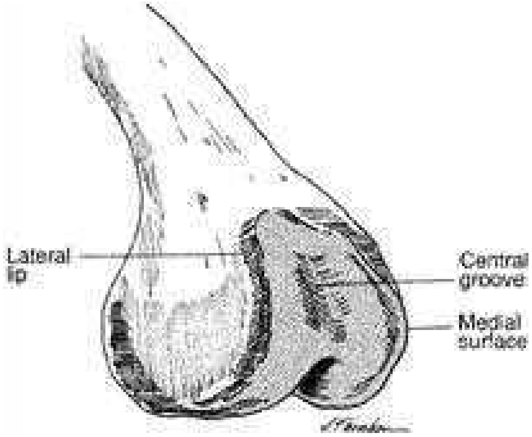


Figure 11–40 Articulating surfaces on the femoral sulcus. Note the well-developed lateral lip on the lateral aspect of the articulating surface.

surfaces and the fact that the patella has a much smaller articular surface area than its femoral counterpart, the patellofemoral joint is one of the most incongruent joints in the body.

The patella functions primarily as an anatomical pulley for the quadriceps muscle. Interposing the patella between the quadriceps tendon and the femoral condyles also reduces friction as the femoral condyles contact the hyaline cartilage–covered posterior surface of the patella rather than the quadriceps tendon. The ability of the patella to perform its functions without restricting knee motion depends on its mobility. Because of the incongruence of the patellofemoral joint, however, the patella is dependent on static and dynamic structures for its stability. We must closely examine the oddly shaped patella and the uneven surface on which it sits in order to understand the normal motions of the patella that accompany knee joint motion and the tremendous forces to which the patella and patellofemoral surfaces are susceptible. The goal of such examination is to understand the many potential problems encountered by the patella in performing what appears to be a relatively simple function. A comprehension of the structures and forces that influence patellofemoral function leads readily to an understanding of the common clinical problems found at the patellofemoral joint as it attempts to meet its contradictory demands for both mobility and stability.

Patellofemoral Articular Surfaces and Joint Congruence

In the fully extended knee, the patella lies on the femoral sulcus. Because the patella has not yet entered the intercondylar groove, joint congruency in this position is minimal, which suggests that there is greater potential for patellar instability with the knee near full extension. Stability of the patella is affected by the vertical position of the patella in the femoral sulcus, because the superior aspect of the femoral sulcus is shallower than the inferior aspect. The vertical position of the patella, in turn, is related to the length of the patellar tendon. Ordinarily, the ratio of the length of the patellar tendon to the length of the patella is approximately 1:1 and is referred to as the **Insall-Salvati index.**[166] A markedly long tendon produces an abnormally high position of the patella on the femoral sulcus known as **patella alta,** which increases the risk for patellar instability. The interaction of the height of the lateral lip of the femoral sulcus with patella alta may also be a factor in patellar instability. In this condition, the lateral lip is not necessarily underdeveloped (although it may be), but the relatively more superior position of the patella places the patella proximal to the high lateral femoral wall, rendering the patella less stable and easier to sublux. In patients with patella alta, the tibiofemoral joint must be flexed more before the patella translates inferiorly enough to engage the intercondylar groove. This leaves a larger knee ROM within which the patella is relatively unstable.

Given the incongruence of the patella, the contact between the patella and the femur changes throughout the knee ROM (Fig. 11–41). When the patella sits in the femoral sulcus in the extended knee, only the inferior pole of the patella is making contact with the femur.[167] As the knee begins to flex, the patella slides down the femur, increasing the surface contact area. In this manner, the first consistent contact between the patella and the femur occurs along the inferior margin of both the medial and lateral facets of the patella at 10° to 20° of knee flexion. As tibiofemoral flexion progresses, the contact area increases

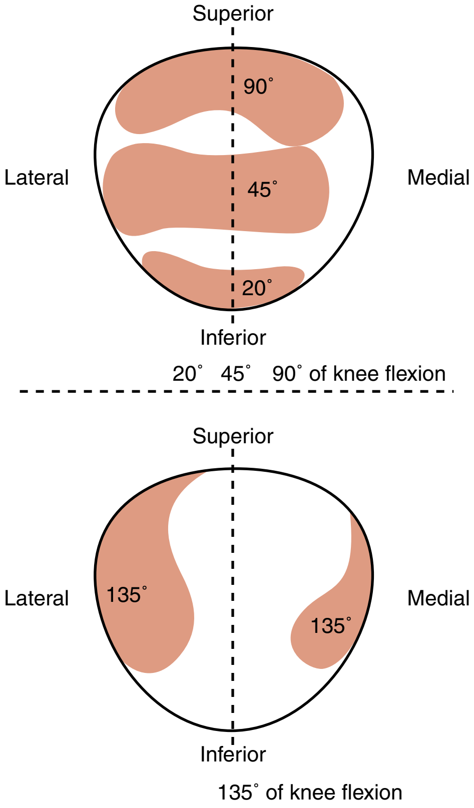


Figure 11–41 Near full extension, only the inferior pole of the patella makes contact with the femur. As flexion continues, the contact area moves superiorly and then laterally along the patella. By full flexion, only the lateral and odd facets are making contact with the femur.

and shifts from the initial inferior location on the patella to a more superior position. As the contact area shifts superiorly along the posterior aspect of the patella, it also spreads outward to cover the medial and lateral facet. By 90° of knee flexion, all portions of the patella have experienced some (although inconsistent) contact, with the exception of the odd facet. As flexion continues beyond 90°, the area of contact begins to migrate inferiorly once again as the smaller odd facet makes contact with the medial femoral condyle for the first time. At full flexion, the patella is lodged in the intercondylar groove, and contact is on the lateral and odd facets, with the medial facet completely out of contact.[168,169]

Motions of the Patella

The relative movement between femoral, tibial, and patellar surfaces during knee motion is more intricate than what a simple hinge joint represents. As the contact between the patella and the femur changes with knee joint motion, the patella simultaneously translates and rotates on the femoral condyles. These movements are influenced by and reflect the patella's relationship to both the femur and the tibia. When the femur is fixed while the knee is flexing, the patella (fixed to the tibial tuberosity via the patellar tendon) is pulled down, gliding inferiorly and rotating on the femoral condyles with the distal apex of the patella moving posteriorly. This sagittal plane rotation of the patella as the patella travels (or "tracks") down the intercondylar groove of the femur is termed **patellar flexion.** Knee extension brings the patella back to its original position in the femoral sulcus, with the apex of the patella pointing inferiorly at the end of the normal ROM. This patellar motion of gliding superiorly while rotating up and around the femoral condyles is referred to as **patellar extension.**

In addition to patellar flexion and extension, the patella tilts around a longitudinal axis (proximal to distal through the patella), shifts medially and laterally in the frontal plane, and spins or rotates around an anteroposterior axis (perpendicular to the patella). Tilting about the longitudinal axis is termed **lateral/medial patellar tilt** and is named for the direction in which the anterior surface of the patella is moving relative to femoral condyles. Lateral patella tilt occurs when the lateral edge of the patella approximates the surface of the lateral femoral condyle and medial patella tilt occurs when the medial edge of the patella moves toward the medial femoral condyle (Fig. 11–42).[170] Patellar tilt is also dictated somewhat by the asymmetrical nature of the femoral condyles. For instance, the more anteriorly protruding lateral femoral condyle forces the anterior surface of the patella to tilt medially during much of knee flexion.[171–173]

Lateral and **medial patella shifts** are translations (gliding) that occur in a plane of motion, rather than rotation around an axis. A lateral patella shift is defined as the patella moving toward the lateral femoral condyle in the frontal plane along the medial-lateral axis.[170] A lateral patella shift is commonly used during the patella apprehension test to evaluate the stability of the patella within the femoral groove.[174]

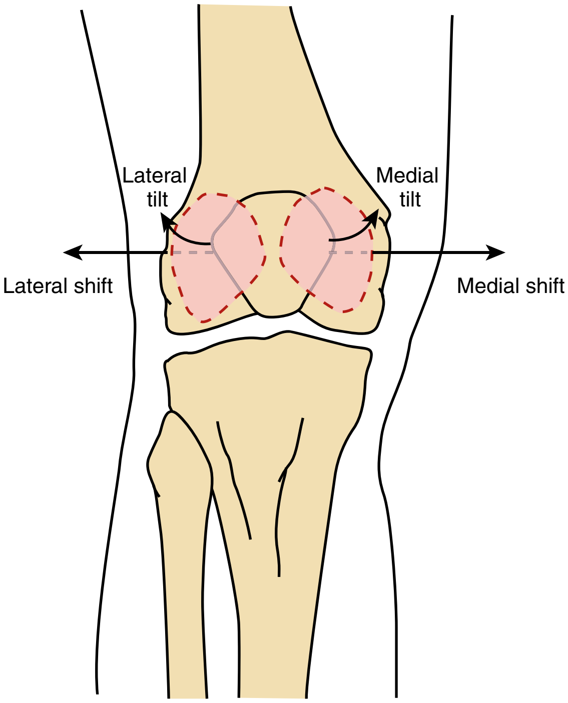


Figure 11–42 Patellar motions with respect to the femur. Medial/lateral shift is named on the basis of the direction in which the patella is moving toward the medial and lateral femoral condyles, respectively; medial/lateral tilt is named for the direction toward which the anterior surface of the patella is tipping, with the medial surface approximating the medial femoral condyle described as a medial tilt and vice versa.

Rotation of the patella about an anteroposterior axis (termed **medial/lateral rotation** of the patella) is referenced by the movement of the distal apex of the patella. Medial rotation occurs when the patella spins around this perpendicular axis with the apex of the patella pointing toward the medial femoral condyle and the base of the patella moving closer to the lateral femoral condyle.[170] Patella rotation, like patellar tilt, is necessary in order for the patella to remain seated between the femoral condyles as the femur undergoes axial rotation on the tibia. Because the inferior aspect of the patella is "tied" to the tibia via the patellar tendon, the inferior patella continually points toward the tibial tuberosity while moving with the femur (Fig. 11–43).[175] Therefore, when the knee is in some flexion and there is medial rotation of the tibia on the fixed femur, the inferior pole of the patella will point medially as the patella medially rotates. Similarly, as the tibia rotates laterally, the patella will rotate laterally to keep the apex aligned with the tibial tuberosity. During knee flexion, the patella medially rotates as the tibia medially rotates to "unlock" the knee. The patella then laterally rotates approximately 5° as the knee flexes from 20° to 90°,[172] given the asymmetrical configuration of the femoral condyles.

The patella, although firmly attached to soft tissue stabilizers (e.g., the extensor retinaculum), undergoes translational motions that are dependent on the point in the tibiofemoral ROM. The patella translates superiorly and inferiorly with knee extension and flexion, respectively.

Understanding these motions is important because they can influence knee joint function. During active knee extension, for example, if superior gliding of the patella is restricted, quadriceps function can be compromised, and knee extension may be lost. During active tibiofemoral flexion, the patella glides inferiorly. A restricted inferior glide could therefore limit knee flexion. There is a simultaneous medial-lateral shift of the patella that accompanies the superior-inferior glide (see Fig. 11–42).[172] The patella is typically situated slightly laterally in the femoral sulcus with the knee in full extension. As knee flexion is initiated, the patella is pushed medially by the large lateral femoral condyle. As knee flexion proceeds past 30°, the patella may shift slightly laterally or remain fairly stable, inasmuch as the patella is now firmly engaged within the femoral condyles (Fig. 11–44). Consequently, the patella shifts as the knee moves from full extension into flexion. Failure of the patella to glide, tilt, rotate, or shift appropriately can lead to restrictions in knee joint ROM, maltracking of the patellofemoral joint, or pain caused by compression of the patellofemoral articular surfaces. Therefore, passive and active mobility of the patella should be assessed clinically to determine the presence of hypermobility or hypomobility with respect to the femur.

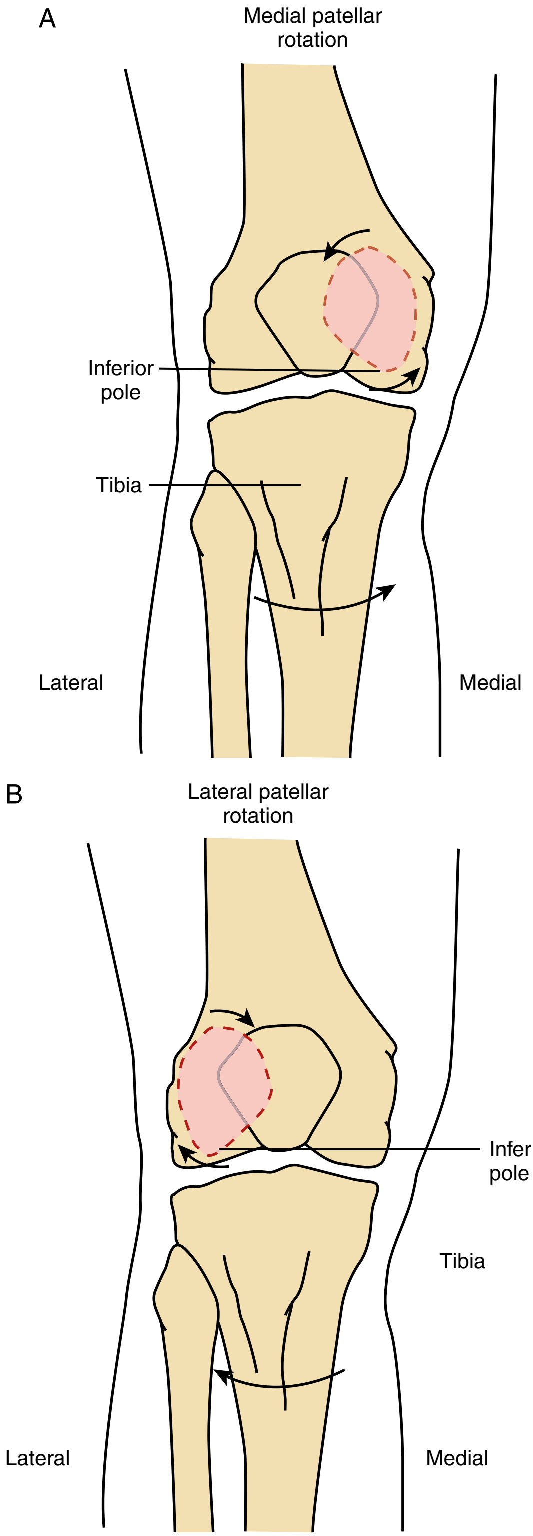


Figure 11–43 **A.** Medial rotation of the patella. The inferior pole of the patella follows the tibial tuberosity during medial rotation of the tibia. **B.** Lateral rotation of the patella. The inferior pole of the patella follows the tibial tuberosity during lateral rotation of the tibia.

Patellofemoral Joint Stress

The patellofemoral joint can undergo very high stresses during typical activities of daily living.[156,176] Joint stress (force per unit area) can be influenced by altering either joint forces and/or joint surface contact areas. The patellofemoral joint reaction (contact) force is influenced by both the force generated by the quadriceps and the knee angle. As the knee flexes and extends, the patella is pulled superiorly by the quadriceps tendon, with the pull resisted inferiorly by the patella tendon. The combination of these pulls produces a posterior compressive force of the patella

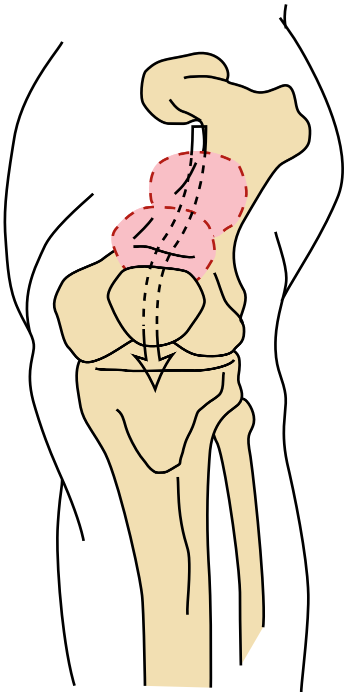


Figure 11–44 The patella shifts medially during early flexion and then either remains there or shifts slightly laterally with deeper flexion.

on the femur that varies with the amount of knee flexion. At full extension, the quadriceps posterior compressive force on the patella is minimized and due exclusively to the origin of the vastus medialis and vastus lateralis muscles on the posterior femur.[154] Despite the small contact area that the patella has with the femur in full extension, the minimal posterior compressive vector of the vastus lateralis and vastus medialis muscles maintains low joint stress at full extension. This is the rationale for the use of straight-leg–raising exercises as a way of improving quadriceps muscle strength without creating or exacerbating patellofemoral pain.

As knee flexion progresses from full extension, the angle of pull between the quadriceps tendon and the patellar tendon decreases, which increases the joint reaction force and in turn produces greater patellofemoral joint compression (Fig. 11–45). This increased compression occurs whether the muscle is active or passive. If the quadriceps muscle is inactive, then elastic tension alone increases with increased knee joint flexion. If the quadriceps muscle is active, then both the active tension and passive elastic tension will contribute to increasing the joint compressive force. The total joint reaction force is therefore influenced by the magnitude of active and passive pull of the quadriceps, as well as by the angle of knee flexion.

Patellofemoral joint reaction forces can become very high during routine daily activities. During the stance phase of walking, when peak knee flexion is only approximately 20°, the patellofemoral compressive force is approximately 25% to 50% of body weight.[121] With greater knee flexion and greater quadriceps activity, as during running, patellofemoral compressive forces have been estimated to reach between five and six times body weight.[177] Deep knee flexion exercises that require large magnitudes of quadriceps activity can increase this compressive force further. Although reaction forces at other lower extremity joints may reach these same magnitudes, they do so over much more congruent joints; that is, the compressive forces are distributed over larger areas.

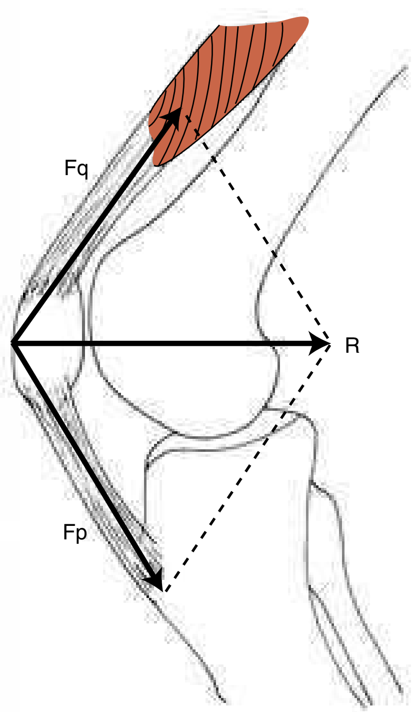


Figure 11–45 Patellofemoral joint reaction forces are partially explained by the knee flexion angle. As the knee is flexed further, the patellofemoral compressive load is increased.

At the normal patellofemoral joint, the medial facet bears the majority of the compressive force. Several mechanisms help minimize or dissipate the patellofemoral joint stress on the patella in general and on the medial facet in particular. In full extension, there is minimal compressive force on the patella; therefore, no compensatory mechanisms are necessary. As knee joint flexion proceeds, the area of patellar contact gradually increases, dispersing the increased compressive force.[154] This simultaneous increase in contact area and compressive force helps to minimize patellofemoral joint stress until approximately 90° of flexion. Specifically, from 30° to 70° of flexion, the magnitude of contact force is higher at the thick cartilage of the medial facet near the central ridge. This articular cartilage is among the thickest hyaline cartilage in the human body. The presence of this thick cartilage is better able to withstand the substantial compressive forces transmitted across the medial facet of the patella. Within this same ROM, the patella has its greatest effect as a pulley, maximizing the moment arm of the quadriceps. With a larger moment arm, less quadriceps muscle force is needed to produce the same torque, minimizing patellofemoral joint compression. As flexion proceeds, the moment arm diminishes, which necessitates an increase in force production by the quadriceps. As knee flexion continues beyond 90°, patellofemoral stress will increase as the patellar contact area once again diminishes. The quadriceps tendon now contacts the femoral condyles, however, which helps to dissipate some of the increasing compressive force on the patella.[178]

The vertical position of the patella can also significantly influence patellofemoral stress. Singerman and colleagues demonstrated that in the presence of patella alta, the onset of contact between the quadriceps tendon and femoral condyles is delayed.[178] As flexion increases, patellofemoral compressive forces will therefore continue to rise. In contrast to patella alta, the patella can also sit lower than normal. If the patella is positioned more inferiorly, it is termed **patella baja** and may be due to a shortened patellar tendon. With patella baja, the contact between the quadriceps tendon and the femoral condyles occurs earlier in the range, resulting in a concomitant reduction in the magnitude of the patellofemoral contact force.[178–180]

Frontal Plane Patellofemoral Joint Stability

The patellofemoral joint exhibits the potential for frontal plane instability near full knee extension, as well as for degenerative changes resulting from increased patellofemoral joint stresses (in flexion). This problem makes understanding the control of the patella's frontal plane motion particularly important. In the extended knee, instability can arise because the patella sits on the shallow aspect of the superior femoral sulcus where bony stability and patellofemoral compression from the quadriceps are reduced. Because of the physiological valgus that normally exists between the tibia and femur, the action lines of the quadriceps and the patellar tendon do not coincide. Instead, the patella is pulled slightly laterally by the two forces (Fig. 11–46). The presence of a resultant lateral pull on the patella in extension, where bony stability is reduced, suggests that soft tissue stabilizers must assume more responsibility for medial-lateral stability. Once knee flexion is initiated and the patella begins to slide down into the femoral sulcus (at about 20° of flexion), medial-lateral stability is increased by the addition of the bony stability. However, the concomitant increased compression of the patella against the femoral condyles can lead to joint pain. Whether the patella is at risk for instability or for increased compression, the position, mobility, and control of the patella in the frontal plane are of utmost concern. These factors are determined by the relative tension in both the transverse and longitudinal stabilizers of the patella.

The longitudinal stabilizers of the patella consist of the patellar tendon inferiorly and the quadriceps tendon superiorly. The patellotibial ligaments, which are part of the extensor retinaculum and reinforce the capsule, also are considered longitudinal stabilizers (see Fig. 11–14).[28,49,59] The longitudinal stabilizers provide passive increases in patellofemoral compression (see Fig. 11–45), which in turn helps to stabilize the patella in the medial-lateral direction. In the extended knee, where patellofemoral compression is minimal, the patella is in a relatively unstable position. When extension is exaggerated, as in genu recurvatum, the pull of the quadriceps muscle and patellar tendon may actually distract the patella somewhat from the femoral sulcus, further increasing the potential for frontal plane patellar instability.

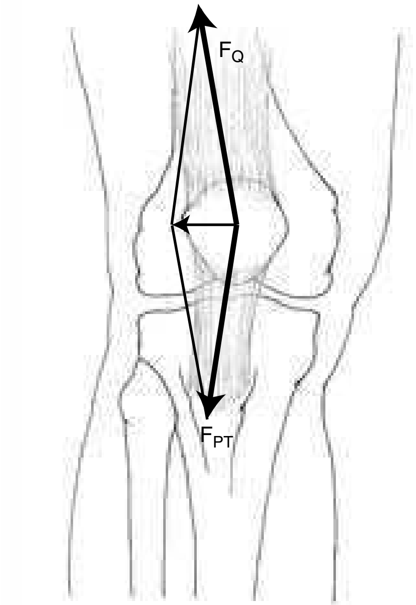


Figure 11–46 Because of the obliquity (physiological valgus angle) between the long axis of the femur and the tibia, the pull of the quadriceps (F_Q) and the pull of the patellar tendon (F_{PT}) lie at a slight angle to each other, producing a slight lateral force on the patella.

The transverse stabilizers of the patellofemoral joint are composed of the superficial portion of the extensor retinaculum. This retinaculum connects the vastus medialis and vastus lateralis muscles directly to the patella for improved active stabilization. In addition, passive stabilizers such as the medial and lateral patellofemoral ligaments firmly attach the patella to the adductor tubercle medially[59,60] and the iliotibial band laterally.[28,49] The role of the medial patellofemoral ligament in assisting normal patellar tracking should not be underestimated. As the thickest portion of the medial retinaculum, the medial patellofemoral ligament alone provides approximately 60% of the passive restraining force against lateral translation (lateral shift) of the patella.[61] An additional important passive stabilizer is the anteriorly protruding lateral lip of the femoral sulcus (see Fig. 11–2). This steep lateral femoral condyle acts as a buttress to excessive lateral patellar shift. Therefore, even large lateral forces will not sublux or dislocate the patella, provided that the lateral lip of the femoral sulcus is of sufficient height. In the case of trochlear dysplasia, however, even relatively small lateral forces imposed on the patella can cause the patella to sublux or fully dislocate. Both the transverse and the longitudinal structures can thus influence the medial-lateral positioning of the patella within the femoral sulcus, as well as influence patellar tracking as the patella slides down the femoral condyles and into the intercondylar groove.

The presence of hypermobility may result in patellar subluxations or dislocations, whereas hypomobility may yield greater patellofemoral stresses. Passive mobility of the patella is maximal when the knee is fully extended and the musculature is relaxed. An imbalance in the passive tension or a change in the line of pull of the dynamic structures will substantially influence the orientation of the patella. This is predominantly true when the knee joint is in extension and the patella sits on the relatively shallow superior femoral sulcus. Abnormal forces, however, may influence the excursion of the patella even in its more secure location within the intercondylar groove with the knee in flexion.

As already noted, tension in the active and/or stretched quadriceps muscle creates compression between the patella and the femur to increase patellofemoral stability. The force on the patella is determined by the resultant pull of the four muscles that constitute the quadriceps and by the pull of the patellar tendon. Each of the segments of the quadriceps can contribute to frontal plane mobility and stability. As noted earlier, the pull of the vastus lateralis muscle is normally 35° lateral to the long axis of the femur,[154] whereas the pull of the proximal portion of the vastus medialis muscle (vastus medialis longus) is approximately 15° to 18° medial to the femoral shaft with the distal fibers (vastus medialis oblique) oriented 50° to 55° medially (see Fig. 11–34).[152]

Because the vastus medialis and vastus lateralis muscles not only pull on the quadriceps tendon but also exert a pull on the patella through their retinacular connections, complementary function is critical. Relative weakness of the vastus medialis muscle may substantially increase the resultant lateral forces on the patella. The individual pulls of each respective portion of the quadriceps are impossible to measure in vivo. Anatomical variations may contribute to asymmetrical pulls on the patella. In general, the vastus medialis oblique inserts into the superomedial aspect of the patella about one third to one half of the way down on the medial border. In instances of patellar malalignment, the vastus medialis oblique insertion site may be located less than a fourth of the way down on the patella's medial aspect, and as a result, the vastus medialis muscle cannot effectively counteract the lateral motion of the patella.[156]

Although individual components of the quadriceps may not be differentially influenced by pain, the quadriceps muscle as a whole does appear to be susceptible to the inhibitory effects of acute joint effusions caused by injury.[181] This inhibition of the quadriceps can result in hypotonia and atrophy, minimizing the compressive role of the quadriceps and altering the resultant pull on the patella.

CASE APPLICATION

Quadriceps Inhibition

case 11–5

Tina underwent an anterior cruciate ligament reconstruction in which a graft from the central third of her patellar tendon was used. This disruption of her extensor tendon could influence how she uses her quadriceps and may contribute to her knee extensor weakness. With each quadriceps contraction, she is pulling on the patellar tendon, which, if sore, could cause her to reduce quadriceps activation to avoid pain. The result is a weak, atrophied quadriceps muscle. The quadriceps weakness that Tina exhibits may contribute to the pain and dysfunction around her patella. Because her quadriceps have become weaker, Tina is now unable to provide adequate dynamic control of the patella. The distal portion of the vastus medialis is thought to be partially responsible for frontal plane control of patellar motions. With quadriceps weakness, including weakness of the vastus medialis oblique, this role is diminished, and patellofemoral forces can increase laterally. Poor control of the patella can also lead to a hypomobile patella with limited medial-lateral glide.

Continuing Exploration 11-9:

Selective Strengthening of the Vastus Medialis Oblique

For years, therapists have assumed that a weak vastus medialis oblique contributed to diminished medial glide of the patella. Therefore, numerous investigators have attempted to devise exercise regimens to selectively strengthen the vastus medialis oblique. It is sometimes incorrectly assumed that the vastus medialis oblique can be selectively recruited in order to preferentially strengthen that particular component of the vastus medialis muscle. Both portions of the vastus medialis muscle, like the other components of the quadriceps, are innervated by the femoral nerve, making preferential recruitment quite difficult.[153] In the absence of evidence supporting differential recruitment of the vastus medialis oblique, strengthening of the vastus medialis oblique portion of the vastus medialis muscle should be accomplished through whole quadriceps strengthening, with techniques such as biofeedback or neuromuscular electrical stimulation if activation deficits exist. In this way, it can be ensured that the quadriceps, and specifically the vastus medialis oblique, is being sufficiently overloaded to promote muscle hypertrophy.

Asymmetry of Patellofemoral Stabilization

The orientation of the quadriceps' resultant pull with respect to the pull of the patellar tendon provides information about the net force on the patella in the frontal plane. The net effect of the pull of the quadriceps and the patellar ligament can be assessed clinically using a measurement called the **Q-angle** (quadriceps angle). The Q-angle is the angle formed between a line connecting the anterior superior iliac spine to the midpoint of the patella (representing the direction of pull of the quadriceps) and a line connecting the tibial tuberosity and the midpoint of the patella (Fig. 11–47). A Q-angle of 10° to 15° measured with the knee either in full extension or slightly flexed is considered normal.[182] Any alteration in alignment that increases the Q-angle is thought to increase the lateral force on the patella. An increase in this lateral force may increase the compression of the lateral patellar facet against the lateral lip of the femoral sulcus. In the presence of a large enough lateral force, the patella may actually sublux or dislocate over the femoral sulcus when the quadriceps muscle is activated with an extended knee. The Q-angle is usually measured with the knee at or near full extension because lateral forces on the patella may be more problematic in this position. With the knee flexed, the patella is set within the

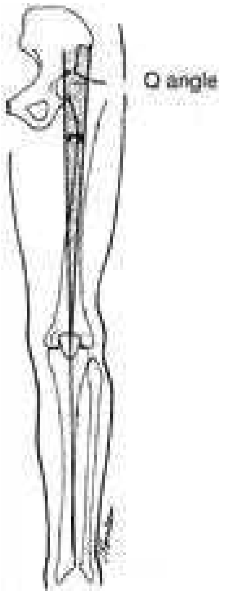


Figure 11–47 The Q-angle is the angle between a line connecting the anterior superior iliac spine to the midpoint of the patella and the extension of a line connecting the tibial tubercle and the midpoint of the patella.

femoral sulcus, and even a very large lateral force on the patella is unlikely to result in dislocation. Furthermore, the Q-angle will diminish with knee flexion as the tibia rotates medially in relation to the femur.[183]

It has been postulated that women have a slightly greater Q-angle than do men because of the presence of a wider pelvis, increased femoral anteversion, and a relative knee valgus angle. However, other authors have disputed this, and the presence of a gender difference in Q-angle is still a matter of debate.[182,184] Although an excessively large Q-angle of 20° or more is usually an indicator of some structural malalignment, an apparently normal Q-angle will not necessarily ensure the absence of problems. Large Q-angles are thought to create excessive lateral forces on the patella that may predispose the patella to pathological changes. One problem with using the Q-angle as a measure of the lateral pull on the patella is that the line between the anterior superior iliac spine and the midpatella is only an estimate of the line of pull of the quadriceps and does not necessarily reflect the actual line of pull in the patient being examined. If a substantial imbalance exists between the vastus medialis and vastus lateralis muscles in a patient, the Q-angle may lead to an incorrect estimate of the lateral force on the patella because the actual pull of the quadriceps muscle is no longer along the estimated line. Furthermore, a patella that sits in an abnormal lateral position in the femoral sulcus, because of imbalanced forces, will yield a smaller Q-angle because the patella lies more in line with the anterior superior iliac spine and tibial tuberosity.

There are several abnormalities that can yield increased lateral forces on the patella. There is a potential for imbalance between the vastus lateralis and vastus medialis muscles, although, as identified earlier, this imbalance is difficult to measure. In vivo investigations indicate that the contribution of the vastus medialis to knee extension torque in patients with patellofemoral pain syndrome with lateral patellar tilt malalignment was less than healthy controls.[185] In healthy controls, the torque contribution from the vastus medialis and the vastus lateralis muscles was not different, suggesting synergistic activation of the quadriceps during knee extension while potentially maintaining proper patellar alignment in the femoral groove.[185] Furthermore, Van Tiggelen and colleagues reported that altered vastus medialis firing contributed to the risk of developing patellofemoral pain.[186] Healthy subjects who demonstrated a delayed onset of vastus medialus oblique muscle activity relative to the vastus lateralis prior to basic military training were more likely to develop patellar femoral pain after basic military training.[186] However, other investigators support the contention that muscle activity of the two portions of the vastus medialis and the vastus lateralis muscles are not selectively altered in patients with patellofemoral pain.[187–189] At this time, the interactions between decreased vastus medialis activity, knee pain, and patellar malalignment and its effect on patellofemoral dysfunction remain controversial.

The presence of a tight iliotibial band could limit the patella's ability to shift medially during flexion, contributing to increased stress under the lateral facet of the patella.[126] With knee flexion, the iliotibial band exerts an even greater lateral pull on the patella that results in a progressive lateral tilting.[126] The increased lateral tilt could focus the patellofemoral load on the lateral facet, increasing joint stress. The tibiofemoral frontal plane deviation of genu valgum increases the obliquity of the femur (see Fig. 11–7A) and, concomitantly, the lateral obliquity of the pull of the quadriceps. In contrast, individuals with genu varum exhibit less obliquity of the femur (see Fig. 11–7B), and therefore should have a diminished lateral quadriceps pull. The transverse plane deviation of medial femoral torsion (or femoral anteversion) generally results in the femoral condyles being turned in (medially rotated). The medially oriented femoral sulcus carries the patella medially and increases the Q-angle by increasing the obliquity of the pull of the quadriceps on the patella. Likewise, in lateral tibial torsion there is an increased Q-angle due to the increased obliquity of the patellar tendon. When medial femoral torsion and lateral tibial torsion coexist, the Q-angle will increase substantially, resulting in a substantial lateral force on the patella (Fig. 11–48). As we will see in Chapter 12, the presence of excessive or prolonged pronation in the foot can contribute to excessive or prolonged medial rotation of the lower extremity that moves the patella medially, increasing the Q-angle and promoting a greater lateral force on the patella in a way similar to that

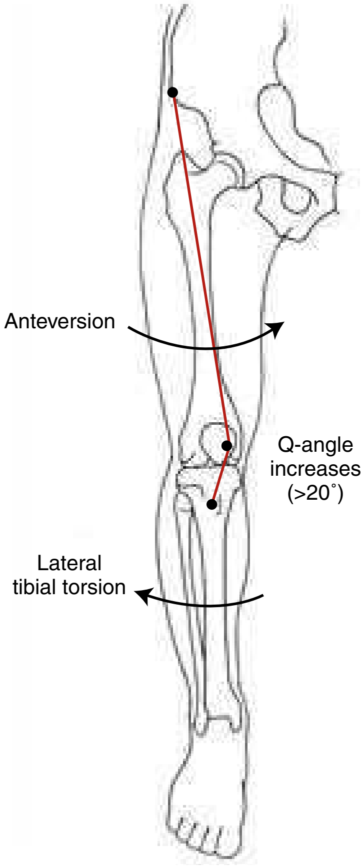


Figure 11–48 Increased medial femoral torsion (femoral anteversion) and tibial lateral torsion will result in a larger Q-angle and an increased lateral force on the patella.

of medial femoral torsion. Each of these conditions can predispose the patella to excessive pressure laterally or to lateral subluxation or dislocation.

Forces other than the alignment and balance of the quadriceps muscle components may influence patellar positioning. Either laxity of the medial retinaculum or excessive tension in or adaptive shortening of the lateral retinaculum may contribute to a laterally tilted patella in the femoral sulcus (Fig. 11–49). It is currently unknown whether such changes in the passive structures are primary or are secondary to abnormalities in the dynamic stabilizers.

Weight-Bearing Versus Non-Weightbearing Exercises With Patellofemoral Pain

Both weight-bearing and non-weightbearing exercises are often prescribed for patients with patellofemoral pain. Each mode of exercise influences the patellofemoral joint differently on the basis of the knee's position within the ROM. Effective quadriceps strengthening in a patient with pain must be performed in a pain-free range. We already noted that in non-weightbearing extension exercises, such as the seated knee extension, the quadriceps must produce more torque as extension progresses (quadriceps force increases with decreasing knee flexion angle) (see Fig. 11–37). The increased torque by the quadriceps near knee extension is necessary to compensate for the increased moment arm of the resistance. However, the greater compressive force generated by the increased quadriceps contraction can be detrimental for an individual with patellofemoral pain, especially if the degeneration is located on the inferior aspect of the patella that is in contact with the femur near extension. In contrast, a weight-bearing exercise requires greater quadriceps activity with greater knee flexion (e.g., at the bottom of a squat) as the resistance moment arm increases (see Fig. 11–38). During weight-bearing exercises, greater knee flexion will therefore increase the compressive force across the patellofemoral joint both because of increased force demands on the quadriceps muscle and because of the increased patellofemoral compression that occurs even with passive knee flexion. The substantial patellofemoral compression may aggravate patellofemoral pain. Exercise recommendations for patients with patellofemoral pain can be based on changing patellofemoral joint stress with weight-bearing and non-weightbearing exercises and knee flexion angle (Fig. 11–50). It has been recommended that those with patellofemoral pain avoid deep flexion while doing weight-bearing extension exercises and avoid the final 30° of extension during non-weightbearing knee extension exercises.[190]

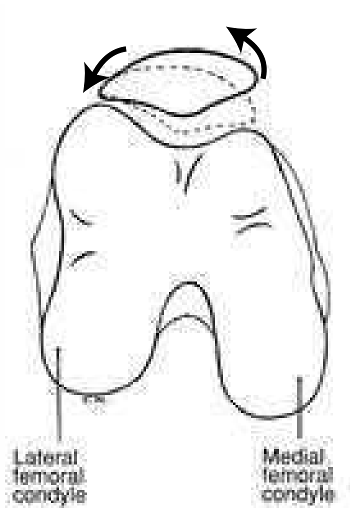


Figure 11–49 Laxity in the medial extensor retinaculum or adaptive shortening of the lateral retinaculum may result in maintaining the patella in a laterally tilted position on the femoral sulcus (shown in a view up the femur from its distal end).

Continuing Exploration 11-10:

Patellofemoral Pain: Weight-Bearing Versus Non-Weightbearing Exercises

The use of weight-bearing exercises has occasionally been promoted as safer and "more functional" than non-weightbearing exercises.[190] There are, however, numerous activities performed throughout the day in a non-weightbearing position. Although it is true that other muscles are forced to work together in a weight-bearing situation in order to control other joints, this is not always the best strategy for strengthening. The use of non-weightbearing exercise isolates the target muscle, which may make strengthening more effective.[191,192] For example, non-weightbearing quadriceps exercises produce large anterior shear forces near full extension, which diminishes with increased flexion, and produce larger patellofemoral joint stress closer to full extension. In contrast, during weight-bearing extension exercises, patellofemoral joint stress is minimal near full extension but increases with increasing flexion, whereas anterior shearing forces are similar to those produced during non-weightbearing exercises throughout the ROM. Furthermore, subjective and functional outcomes were not different between patients who performed therapeutic exercises using either open-chain or closed-chain exercises in isolation.[193] Witvrouw and colleagues determined that both the open-chain and closed-chain groups maintained good subjective and functional outcomes with no reported differences in outcomes between groups over a 5-year follow-up period.[193] With regard to safety (e.g., patellofemoral joint stress, anterior cruciate ligament strain), an understanding of how joint stress and tissue strain change throughout the knee ROM and performing the exercises within a pain-free ROM will assist with patient safety.

EFFECTS OF INJURY AND DISEASE

The joints of the knee complex, like other joints in the body, are subject to developmental defects, injury, and disease processes. A number of factors, however, make the knee joint unique in its development of various pathologies. The

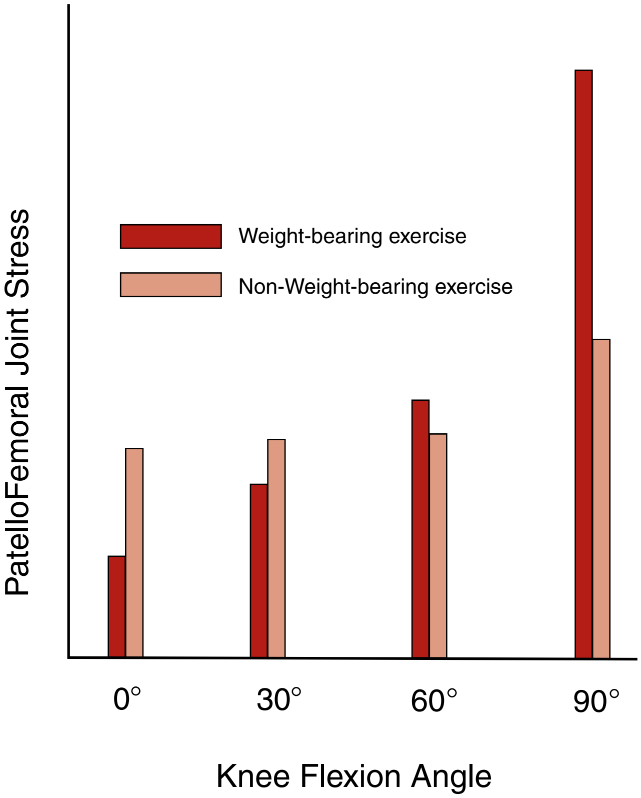


Figure 11–50 Simulations showed patellofemoral joint stress to be greater during loaded non-weightbearing exercises than weight-bearing exercises when the knee was closer to knee extension. Patellofemoral joint stress was higher, however, during weight-bearing exercises when knee flexion exceeds approximately 50°. *(Data from Cohen ZA, Roglic H, Grelsamer RP, et al: Patellofemoral stresses during open and closed kinetic chain exercises. An analysis using computer simulation. Am J Sports Med 29:483, 2001.)*

knee, unlike the shoulder, elbow, and wrist, must support the body weight and at the same time provide considerable mobility. Although the hip and ankle joints similarly support the body's weight, the knee is a more complex structure than either the hip or ankle. The anatomical complexity is necessary to dissipate the enormous forces applied through the joint as two of the longest levers in the body meet at the knee complex.

Tibiofemoral Joint Injury

The tremendous forces applied through the knee have the potential to contribute to numerous injuries and degenerative damage. In addition, participation in physical fitness and sports activities that involve jumping, pivoting, cutting, or repetitive cyclic loading among all age groups and both sexes can increase the risk of injury to the menisci, ligaments, bones, bursae, or the musculotendinous structures.

Meniscal injuries are common and usually occur as a result of sudden rotation of the femur on the fixed tibia when the knee is in flexion. The pivot point during axial rotation in the flexed knee occurs through the medial meniscus. Therefore, the more rigidly attached medial meniscus may tear under the sudden load. Ligamentous injuries may occur as a result of a force that causes the joint to exceed its normal ROM. Although excessive forces may cause ligamentous tears, lower-level forces may similarly cause disruption in ligaments weakened by aging, disease, immobilization, steroids, or vascular insufficiency. Cyclic loading (whether short term and intense or over a prolonged period) may also affect viscoelasticity and stiffness. A weakened ligament may take 10 months or more to return to normal stiffness once the underlying problem has been resolved. After a ligament injury or reconstruction, the new or damaged tissue must be protected to minimize excessive stress through the healing tissue. Absence of tissue stress, however, is also detrimental, because the new tissue will not adapt and become stronger under unloaded conditions. Rehabilitation of a repaired or reconstructed ligament, therefore, is a balance in avoiding either too much or too little applied stress.

The bony and cartilaginous structures of the tibiofemoral joint may be injured either by the application of a large direct force, such as during a twist or fall, or by forces exerted by abnormal ligamentous and muscular forces. Knee osteoarthritis is often seen in older adults and is particularly common in women. This progressive erosion of articular cartilage may be initiated by a previous traumatic joint injury, obesity, malalignment, instability, or quadriceps muscle weakness, to name just a few of the many suspected contributors to the development of osteoarthritis. Tibial plateau fractures can occur when large magnitudes of force are applied through the joint. Knee joint instability, as frequently seen in the knee after anterior cruciate ligament injury, can lead to progressive changes in the articular cartilage, in the menisci, and in the other ligaments attempting to restrain the increased joint mobility. The presence of ligamentous instability induces abnormal forces through the joint, inasmuch as excessive shearing can often occur. In addition, this excessive laxity must be controlled in order to avoid episodes of giving way. Because the knee has poor bony congruency, the muscles must provide greater control of all fine movements of the tibiofemoral joint in the absence of one or more ligaments. Increased muscular co-contraction, however, may generate greater compressive forces through the joint, contributing to articular cartilage degeneration. An improved method of providing dynamic stability to a lax joint, therefore, is to generate isolated muscle contractions as needed, rather than a massive co-contraction to stiffen the joint.

The numerous bursae and tendons at the knee are also subject to injury. The cause of injuries to these structures may be either a direct blow or prolonged compressive or tensile stresses. Bursitis is common after either blunt trauma or repetitive low-level compressions, which can irritate the tissue. The prepatellar bursa, the superficial infrapatellar bursa (known as **housemaid's knee** when it is inflamed), and the bursa beneath the pes anserinus are common locations for injury. Tendinitis results from repetitive low-level stresses to the tissues of the tendon. Frequently this is caused by overuse of the affected muscle and can occur in response to a previous ligamentous injury. Another potential source of pain and dysfunction in the knee joint is the irritation of a patellar plica. Classic symptoms include pain with prolonged sitting, with stair climbing, and during resisted extension exercises. In flexion, the medial patellar plica is drawn over the medial femoral condyle and can become compressed beneath the patella causing inflammation. If the inflamed plica becomes fibrotic, it may create a secondary synovitis around the femoral condyle, and deterioration of the condylar cartilage may occur. A thickened or inflamed superior plica may erode the superior aspect of the medial facet of the patella.

Patellofemoral Joint Injury

We have presented the pathomechanics that may predispose the knee to patellofemoral dysfunction. Any one problem in isolation or various combinations of problems may lead to excessive pressure on the lateral facets of the patella, to lateral subluxation, or to lateral dislocation. Both patellar instability and increased patellofemoral compression are commonly associated with knee pain, poor tolerance of sustained passive knee flexion (as in sitting for long periods), "giving way" of the knee, and exacerbation of symptoms by repeated use of the quadriceps on a flexed knee. Often this results in diminished use of the quadriceps, leading to atrophy and subsequently a further deterioration of patellar control. As muscle function declines, patellofemoral dysfunction may progress, necessitating a reversal of muscle function under a series of controlled situations to generate hypertrophy of the quadriceps, while minimizing discomfort.

Among the causes of increased lateral patellar compression include a tight iliotibial band, large Q-angle (e.g., as in genu valgum or femoral anteversion with lateral tibial torsion), relative vastus medialis muscle weakness, or patellar hypomobility. With patellar hypermobility, lax medial structures, and a short lateral femoral condyle, the risk of lateral patellar subluxation or dislocation is increased. After a lateral patellar subluxation or dislocation, the medial retinaculum is stretched as the patella deviates toward or slips over the lateral lip of the femoral sulcus or condyle. The return of the laterally dislocated patella into the femoral sulcus may affect the medial patella (occasionally causing an osteochondral fracture). There are a host of other pathologies that can occur around the patellofemoral joint, including pain from the lateral patellofemoral ligament, inflammation of the medial patellar plica (discussed previously), and pain from the quadriceps tendon above or the patellar tendon below. Patellofemoral pain is most often observed in adolescents and may resolve spontaneously. In addition, patellar subluxation is more often seen in younger patients, who may have a less developed patella and lateral condyle that less effectively resist an excessive lateral force on the patella.

Cartilaginous changes seen on the lateral patellar facet were once considered to be diagnostic of patellofemoral dysfunction, and the term **chondromalacia patella** (softening of the cartilage) was assigned. With the knowledge that similar cartilaginous changes can be found in asymptomatic knees and that the medial patellar facet can show greater change without symptoms or progressive cartilage deterioration, more general diagnoses have been used, including **patellofemoral arthralgia** or **patellofemoral pain syndrome.** The use of this more general terminology suggests that the damage extends beyond the articular cartilage. Cartilage is aneural and therefore cannot be the cause of pain.[194] Instead, patients with patellofemoral pain can experience discomfort from damage to subchondral bone, the synovial membrane, and ligamentous or musculotendinous structures. Advances in diagnostic imaging technologies now allow for patella tracking during dynamic tasks. The effects that femoral rotation and tibial rotation have on patella position have warranted interventions that control the hip, pelvis, and ankle motion when treating patients with patellofemoral joint injury and pain.

CASE APPLICATION

Case Summary

case 11–6

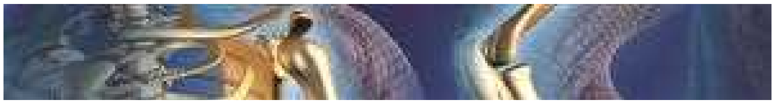
Tina's initial injury (in which she tore her anterior cruciate ligament, medial collateral ligament, and medial meniscus) has likely led to the development of the problems that we are now observing. Tina went for 4 months without important passive tibiofemoral stabilizers (anterior cruciate ligament and medial collateral ligament). During that time, Tina was unable to develop a successful strategy to dynamically stabilize her knee during activities of daily living. Patients who present with knee instability often use a knee stiffening strategy that consists of excessive muscle co-contraction and reduced knee joint excursions in an attempt to control the knee. Tina's unsuccessful knee strategy in combination with her medial meniscal tear likely resulted in greater compressive forces absorbed through a smaller surface area within the medial compartment. The increased force across a smaller surface area increased joint stress and likely led to the gradual erosion of Tina's medial compartment articular cartilage, leading in turn to the development of genu varum. The joint space narrowing observed on the weight-bearing radiographs is indicative of medial compartment osteoarthritis; however, her medial joint pain may be attributed to other causes. For instance, Tina may have overworked the medial muscles trying to control excessive laxity, which can result in tendinitis. It is also possible that Tina has further damaged the already injured medial meniscus, which will contribute to medial joint pain.

After her anterior cruciate ligament reconstruction, the discomfort that she felt around the patellar tendon donor site led to disuse atrophy of her quadriceps muscle. As the muscle atrophied, she became less adept at controlling the tracking of her patella, which is likely responsible for increased lateral patellar compression. The repetitive compression of the lateral patellar facet against the lateral femoral condyle has gradually resulted in patellofemoral degenerative changes on the lateral facet. This lateral patellofemoral joint space narrowing was observed on her radiographs and likely was influenced by her long-standing quadriceps weakness, as well as by potential structural abnormalities. The presence of a tight iliotibial band, excessive Q-angle, excessive or prolonged foot pronation or hip medial rotation, or patella baja are but a few of the contributing factors that could have contributed to Tina's excessive lateral patellofemoral compression and must be screened for to determine the cause of her patellar pain. A complete understanding of the structures and relevant functioning of the tibiofemoral and the patellofemoral joints of the knee complex, as well as the other joints of the lower extremity, allow for appropriate diagnosis and treatment of the knee.

SUMMARY

Given the range of possible problems that can occur in the knee joint, an exhaustive discussion is beyond the scope of this text. A thorough knowledge of normal structure and function, however, can be used to predict or understand the immediate impact of a specific injury and the secondary effects on intact structures. The variety of forces transmitted through the knee complex arises from gravity (weight-bearing forces), muscles, ligaments, and other passive soft tissue structures. Any alteration of the knee's anatomy can substantially influence these forces and can have a dramatic impact on the function of the knee joint. Damage to the tibiofemoral joint or the patellofemoral joint can result from either a large rapid load or the accumulation of smaller repetitive loads. An understanding of both the primary and secondary effects of injury is important in order to gain a full appreciation for the pathogenesis of knee disorders.

STUDY QUESTIONS



1. Describe the congruency of the tibiofemoral joint. What factors add to or detract from stability?
2. Describe the menisci of the knee, including their function, shape, and attachments.
3. Describe the intra-articular movement of the femur on the tibia, as the femur moves from full extension into flexion.
4. Describe the automatic axial rotation mechanism of the knee, including the structure or structures responsible.
5. What happens to the menisci during motions of the knee? How do their attachments contribute to the movement?
6. Identify the major bursae of the knee joint. Which of these are generally separate from and which are parts of the capsule?
7. Which knee joint ligaments contribute to anteroposterior stability of the knee joint?
8. Which ligaments contribute to medial-lateral stability of the knee joint?
9. What are the dynamic stabilizers of the knee, and in what plane or planes do they contribute to stability?
10. At which point in the knee's ROM is axial rotation greatest? Which muscles produce active medial rotation? Lateral rotation?
11. What is the patella plica, and what implications does it have for knee joint dysfunction?
12. Describe the patellofemoral articulation, including the number and shape of the surfaces.
13. What function or functions does the patella serve at the knee joint?
14. How does the patella move in relation to the femur in normal motions? How would function be affected if the patella could not slide on the femur?
15. Describe the contact of the patella with the femur at rest in full extension. Describe the contact as knee flexion proceeds.
16. Is the patella equally effective as an anatomical pulley at all points in the knee ROM? At which point or points is it most effective? Least effective?
17. Which facet of the patella is most likely to undergo excessive degenerative changes when there is malalignment? Describe the malalignment and the condition or conditions that may predispose these changes.
18. What is the Q-angle of the knee joint? How is it measured, and what implications does it have for patellofemoral problems?
19. What changes will the condition of genu recurvatum produce at the patellofemoral joint?
20. Why is ascending stairs commonly cited as producing knee pain? Relate this to patellofemoral joint compression.

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Chapter 12

The Ankle and Foot Complex

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Introduction

Definitions of Motions

Ankle Joint
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  - Proximal Articular Surfaces
  - Distal Articular Surface
  - Capsule and Ligaments
  - Axis
- Ankle Joint Function

The Subtalar Joint
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  - Ligaments
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  - The Subtalar Axis
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  - Weight-Bearing Subtalar Joint Motion
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Transverse Tarsal Joint
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Muscles of the Ankle and Foot
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- Intrinsic Musculature

Deviations From Normal Structure and Function

INTRODUCTION

The ankle/foot complex is structurally analogous to the wrist/hand complex of the upper extremity but has a number of distinct differences to optimize its primary role to bear weight. The complementing structures of the ankle/foot complex permit both stability and mobility depending on conditions acting on it. The foot is able to sustain large weight-bearing stresses while accommodating to a variety of surfaces and activities. The foot must be stable to provide an adequate base of support and function as a rigid lever for pushing-off when walking, running, or jumping. In contrast, the foot must also be mobile to adapt to uneven terrain, absorb shock as the foot hits the ground, and dampen rotations imposed by the more proximal joints of the lower extremity. The ankle/foot complex meets these diverse requirements through the integrated movements of its 28 bones that form 25 component joints. These joints include the proximal and distal tibiofibular joints, talocrural (ankle) joint, talocalcaneal (subtalar) joint, talonavicular and calcaneocuboid joints (transverse tarsal joints), five tarsometatarsal joints, metatarsophalangeal joints, and nine interphalangeal joints.

To facilitate description and understanding of the ankle/foot complex, the bones of the foot are traditionally divided into three functional segments. These are the **hindfoot** (posterior segment), composed of the talus and calcaneus; the **midfoot** (middle segment), composed of the navicular, cuboid, and three cuneiform bones; and the **forefoot** (anterior segment), composed of the metatarsals and the phalanges (Fig. 12–1). These terms are commonly used in descriptions of ankle/foot dysfunction or deformity and are similarly useful in understanding normal ankle and foot function.

Ankle or foot pathology can be related to the integrated multifunctional structures of the ankle/foot complex combined with the demands of stability and mobility placed upon these structures. Additionally, the ankle and foot complex must not only respond to forces from the ground but also imposed forces from the spine, pelvis, hip, and knee. Structural abnormalities can lead to altered movements between joints and contribute to excessive stresses that result in injury to the ankle/foot complex.[1]

12-1 **Patient Case**

Arnold Benson is a 63-year-old man seeking intervention for right foot pain. Three weeks ago, at the suggestion of his physician, Mr. Benson (who is quite overweight) started a walking program. Mr. Benson reports that after about a week, he had pain at his right heel that was greatest when he first got out of bed in the morning. He reports that this pain eases after a few steps but increases again when he walks more than two blocks. He says he has “flat feet,” which seem to have worsened over the past few years. Despite having a sedentary job, Mr. Benson identifies that his feet often ache at the end of the day. He also reports that occasionally the “bunion” on his right foot will flare up with associated pain in the region of his big toe.

Mr. Benson stands with his hips positioned in medial rotation so that both knees are pointing slightly medially. He has a low arch and a valgus positioning of his calcaneus when viewed from behind (more noticeable on the right than on the left). During non-weightbearing examination his calcaneus could be passively moved into a neutral position. He reports that he starts to feel pain in a line behind his right medial malleolus that increases when he points his foot down and in (plantarflexion and inversion). He also reports heel pain when his toes, particularly the first toe, are extended. Finally, Mr. Benson feels a strong pulling behind his knee and into the calf when he sits, extends his knee, and pull his toes up (dorsiflexion). This discomfort is greater on the right side than left.

DEFINITIONS OF MOTIONS

A unique set of terms is used to refer to motion of the foot and ankle. The same terms are used at most of the joints of the ankle and foot, and, consequently, it is useful to describe them at the outset. As we have seen at other joint complexes,

Figure 12–1 Functional segments and bones of the foot.

few if any of the joint axes lies in the cardinal planes; more commonly, the joint axes are oblique and cut across all three planes of motion. The obliquity of the axes and implications for motion and function will be described in detail as we present individual joints.

The three motions of the ankle/foot complex that *approximate* cardinal planes and axes are **dorsiflexion/plantarflexion, inversion/eversion,** and **abduction/adduction** (Fig. 12–2). Dorsiflexion and plantarflexion are motions that occur approximately in the sagittal plane around a coronal axis. Dorsiflexion decreases the angle between the leg and the dorsum of the foot, whereas plantarflexion increases this angle. At the toes, motion around a similar axis is termed extension (bringing the toes up), whereas the opposite motion is flexion (bringing the toes down or curling them). Inversion and eversion occur approximately in the frontal plane around a longitudinal (anteroposterior [A-P]) axis that runs through the length of the foot. Inversion occurs when the plantar surface of the segment is brought toward the midline; eversion is the opposite. Abduction and adduction occur approximately in the transverse plane around a vertical axis. Abduction occurs when the distal aspect of a segment moves away from the midline of the body (or away from the midline of the foot in the case of the toes); adduction is the opposite.

Pronation/supination in the foot are motions that occur around an axis that lies at an angle to each of the axes for "cardinal" motions of dorsiflexion/plantarflexion, inversion/eversion, and abduction/adduction. Consequently, *pronation* and *supination* are terms used to describe "composite" motions that have components of, or are coupled to, each of the cardinal motions. In non-weightbearing, pronation is motion about an axis that results in coupled motions of dorsiflexion, eversion, and abduction. Likewise, supination is a motion about an axis that results in coupled motions of plantarflexion, inversion, and adduction. The proportional contribution that each of the coupled motions makes to pronation/supination is dependent on and varies with the angle of the pronation/supination joint axis.

Valgus and **varus** are terms that may be used for the ankle/foot complex in several ways, depending on the context. The definitions that we used throughout discussion of other joints in other chapters will not change. That is, *valgus* refers to an increase in the medial angle between two bones (or movement of the distal segment away from the midline); *varus* refers to the opposite. However, *valgus* and *varus* are sometimes used to refer to *fixed deformities* in the ankle/foot complex, whereas at other times the terms are used to describe or as synonyms for other normal motions. An example of common usage is to describe the fixed or weightbearing position of the posterior calcaneus in relation to the posterior midline of the leg, with an increase in the medial angle between the two reference lines being valgus of the calcaneus (or calcaneovalgus) and a decrease being varus of the calcaneus (or calcaneovarus) (Fig. 12–3). We will define use and context as we encounter these terms in descriptions of ankle/foot structure and function.

Concept Cornerstone **12-1**

Ankle/Foot Terminology

As we have seen at other joints, terminology used to describe motions around a joint or of a segment is often not consistent among investigators. This is very much the case for the ankle/foot complex. Because the anterior surface of the leg and the top of the foot are embryologically dorsal surfaces,[2] *dorsiflexion* may also be referred to as *extension*, and *plantarflexion* may be referred to as *flexion*. Although the flexion/extension terminology is commonly used for the toes, it may also be applied to the ankle. Some resources reverse the terminology applied to the "composite" movement of pronation/supination and the coupled (component) movement of inversion/eversion; that is, *inversion/eversion* may be used to refer to the composite motion, and *pronation/supination* would be used correspondingly to refer to the component (coupled) motion. As we proceed to describe the joints and their motions, we will see that some of these terminology differences are not really as problematic as they might initially seem.

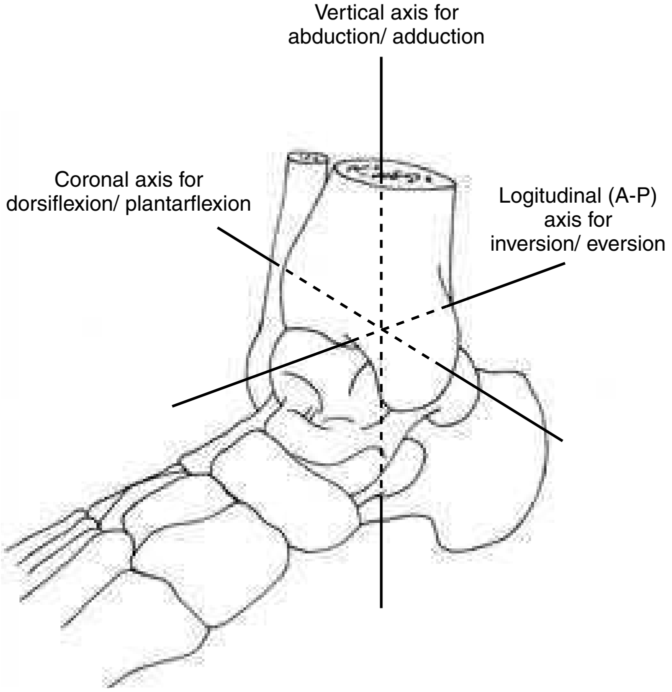

Figure 12–2 "Cardinal" axes for the motions of the ankle/foot complex.

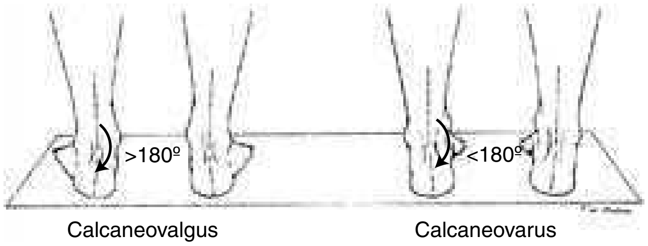

Figure 12–3 The term *valgus* (or *calcaneovalgus*) refers to an increase in the medial angle between the calcaneus and posterior leg. The term *varus* (or *calcaneovarus*) refers to a decrease in the medial angle between the calcaneus and posterior leg.

ANKLE JOINT

The term **ankle** refers specifically to the **talocrural joint,** that is, the articulation between the distal tibia and fibula proximally and the body of the talus distally (Fig. 12–4). The ankle is a synovial hinge joint with a joint capsule and associated ligaments. It is generally considered to have a single oblique axis with one degree of freedom around which the motions of dorsiflexion/plantarflexion occur.

Ankle Joint Structure

Proximal Articular Surfaces

The proximal segment of the ankle is composed of the concave surface of the distal tibia and of the tibial and fibular malleoli. These three facets form an almost continuous concave joint surface that extends more distally on the fibular (lateral) side than on the tibial (medial) side (see Fig. 12–4) and more distally on the posterior margin of the tibia than on the anterior margin. The structure of the distal tibia and two malleoli resembles and is referred to as a **mortise.** A common example of a mortise is the gripping part of a wrench. Either the wrench can be fixed (fitting a bolt of only one size) or it can be adjustable (permitting use of the wrench on a variety of bolt sizes). The adjustable mortise is more complex than a fixed mortise because it combines mobility and stability functions. The mortise of the ankle is adjustable, relying on the **proximal** and **distal tibiofibular joints** to both permit and control the changes in the mortise.

The proximal and distal tibiofibular joints (Fig. 12–5) are anatomically distinct from the ankle joint, but functionally linked exclusively to the ankle. Unlike their upper extremity counterparts, the proximal and distal radioulnar joints, the tibiofibular joints do not add any degrees of freedom to the more distal ankle and foot. However, fusion of the radioulnar joints would have little effect on wrist range of motion (ROM), whereas fusion of the tibiofibular joints may impair normal ankle function.

Proximal Tibiofibular Joint

The proximal tibiofibular joint is a plane synovial joint formed by the articulation of the head of the fibula with the posterolateral aspect of the tibia. The facets of the proximal tibiofibular joint are fairly flat and can vary in configuration between individuals. Generally, a convex tibial facet and concave fibular facet is the most common articulation pattern.[3] The proximal tibiofibular joint is surrounded by a joint capsule that is reinforced by **anterior** and **posterior tibiofibular ligaments.** Although the proximal tibiofibular joint is generally thought to be anatomically separate from the knee joint, communication between these joints can occur.[4] Studies looking at proximal tibiofibular joint movement have shown variations with respect to fibular translation and rotation that occur with ankle dorsiflexion.[5] The motion at this joint is consistently small; however, symptoms of instability can occur following trauma to this area.[6] The relevance of motion at the proximal and distal tibiofibular joints will be seen when ankle joint motion is discussed.

Distal Tibiofibular Joint

The distal tibiofibular joint is a syndesmosis, or fibrous union, between the concave facet of the tibia and the convex facet of the fibula. The distal tibia and fibula do not actually come into contact with each other but are separated by fibroadipose tissue. Although there is no joint capsule, there are several associated ligaments. Because the proximal and distal joints are linked (the tibia, fibula, and tibiofibular

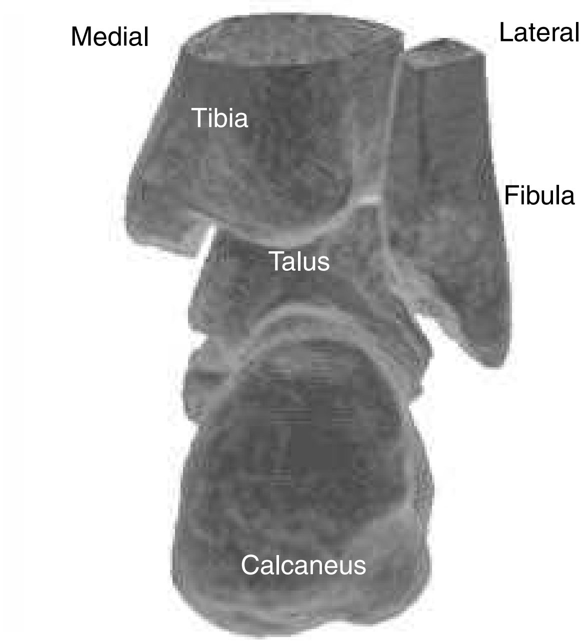

Figure 12–4 The ankle joint is formed by the tibia and fibular (mortise) proximally and by the talus distally.

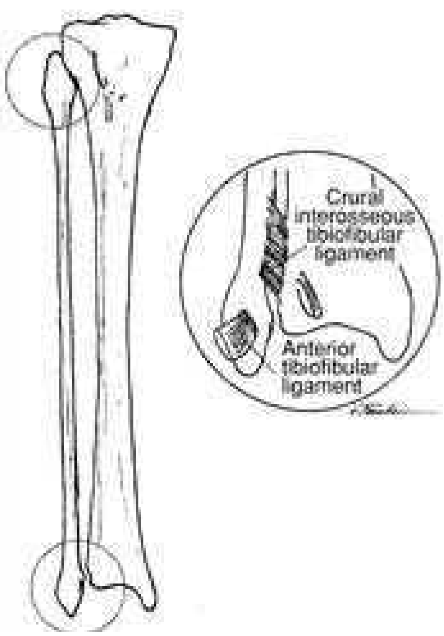

Figure 12–5 Proximal and distal tibiofibular joints.

joints are part of a closed chain), all the ligaments that lie between the tibia and fibula contribute to stability at both the proximal and distal tibiofibular joints.

The ligaments of the distal tibiofibular joint are primarily responsible for maintaining a stable mortise. The **anterior** and **posterior tibiofibular ligaments** and **interosseous membrane** provide support to the distal tibiofibular joint.[7] The interosseous membrane directly supports both proximal and distal tibiofibular articulations. Although the distal tibiofibular joint is an extremely strong articulation, injuries can occur the talus is forcible laterally rotated within the ankle mortise.[8] These injuries cause the fibula and tibia to separate and are diagnosed as high or syndesmotic ankle sprains. If the force were to continue, fracture of the fibula proximal to the distal tibiofibular ligaments could result.[9]

The function of the ankle (talocrural) joint is dependent on stability of the tibiofibular mortise. The ankle mortise would be unable to grasp and hold on to the talus if the tibia and fibula were permitted to separate or if one side of the mortise were missing. This would be similar to a wrench that could not perform its function of grasping a bolt if the two pincer segments moved apart every time a force was applied to the wrench. Conversely, the ankle mortise must have some mobility function or a single fused arch would better serve ankle joint function. The mobility role of the mortise belongs primarily to the fibula. The fibula is generally thought to have little weight-bearing function. One study found the fibular transmitted no more than 10% of weight-bearing forces.[10] However, the amount of axial load through the fibula may be related to positioning, as greater weight-bearing forces were transmitted through the fibula when the ankle was in a position of eversion.[11] The synovial proximal tibiofibular joint appears to be dependent on joint motion (rather than weight-bearing) to maintain nutrition of the cartilage. That is, the proximal tibiofibular joint must be mobile; if the proximal tibiofibular joint is mobile, so too must the distal tibiofibular joint be, because the two joints are mechanically linked.

Distal Articular Surface

The body of the talus (Fig. 12–6) forms the distal articulation of the ankle joint. The **body** of the talus has three articular surfaces: a large lateral (fibular) facet, a smaller medial (tibial) facet, and a **trochlear** (superior) **facet.** The large, convex trochlear surface has a central groove that runs at a slight angle to the **head** and **neck** of the talus. The body of the talus also appears wider anteriorly than posteriorly, which gives it a wedge shape. The articular cartilage covering the trochlea is continuous with the cartilage covering the more extensive lateral facet and the smaller medial facet.

The structural integrity of the ankle joint is maintained throughout the ROM of the joint by a number of supportive structures.

Capsule and Ligaments

The capsule of the ankle joint is fairly thin and especially weak anteriorly and posteriorly. Therefore, the stability of the ankle depends on an intact ligamentous structure. The ligaments that support the proximal and distal tibiofibular joints (**the crural tibiofibular interosseous ligament** (see Fig. 12–5 inset), the anterior and posterior tibiofibular ligaments, and the tibiofibular interosseous membrane) are important for stability of the mortise and, therefore, for stability of the ankle. Two other major ligament complexes maintain contact and congruence of the mortise and talus and control medial-lateral joint stability. These are the **medial collateral ligament (MCL)** and the **lateral collateral ligament (LCL).** Portions of these ligaments also provide support for the **subtalar** (or **talocalcaneal**) **joint** that they also cross.[12] The function of the collateral ligaments at the ankle joint, therefore, is difficult to separate from the function at the subtalar joint.

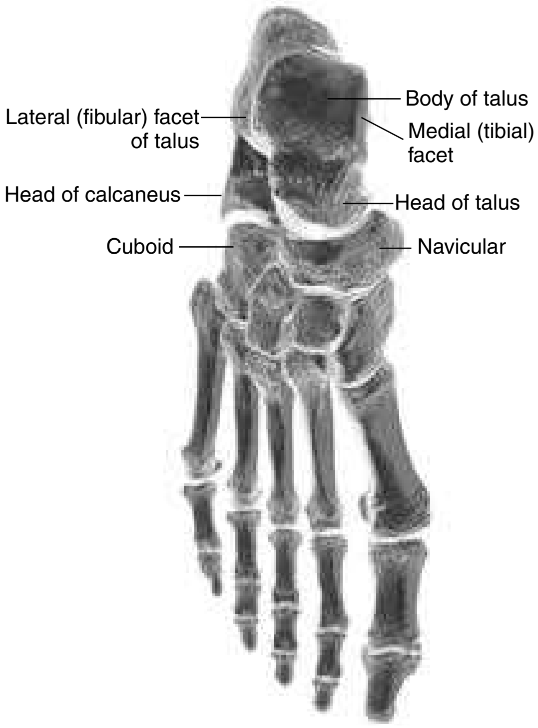

Figure 12–6 The body of the talus with its trochlear (superior) surface, medial (tibial) facet, and lateral (fibular) facet form the distal aspect of the ankle joint.

The medial collateral ligament is most commonly called the **deltoid ligament.** As its name implies, the deltoid ligament is a fan shaped. It has superficial and deep fibers that arise from the borders of the tibial malleolus and insert in a continuous line on the navicular bone anteriorly and on the talus and calcaneus distally and posteriorly (Fig. 12–7).[13] The deltoid ligament as a whole is extremely strong, which partially accounts for the relative low frequency of injury to this ligament. Eversion and/or pronation of the ankle and talus (i.e., outward rotation of the foot combined with inward rotation of the tibia) can injure the deltoid ligament.[14,15] However, these forces may actually fracture and displace (avulse) the tibial malleolus before the deltoid ligament tears.

The lateral collateral ligament is composed of three distinct bands that are commonly referred to as separate ligaments. These are the **anterior** and **posterior talofibular ligaments** and the **calcaneofibular ligament** (Fig. 12–8). The anterior

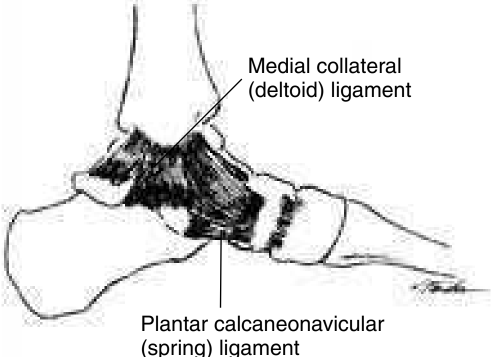

Figure 12–7 Medial ligaments of the posterior ankle/foot complex.

and posterior ligaments run in a fairly horizontal position, whereas the longer calcaneofibular ligament is nearly vertical.[16] In contrast to the medial collateral ligament, the lateral collateral ligament helps control inversion and/or supination of the ankle and talus. (i.e., inward rotation of the foot combined with outward rotation of the tibia).[14,17] In general, the components of the lateral collateral ligament are weaker and more susceptible to injury than are those of the medial collateral ligament.

Concept Cornerstone **12-2**

Summary of Studies Investigating Stresses Applied to the Lateral Collateral Ligament

1. In comparison to the other two ligaments that make up the lateral collateral ligament, the anterior talofibular ligament is the weakest and most commonly injured.[18] This ligament is stressed when the ankle is moved into greater degrees of plantar flexion, medial rotation, and inversion.[19] Rupture of the anterior talofibular ligament often results in anterolateral rotary instability of the ankle.[20]
2. The calcaneofibular ligament is stressed when the ankle is dorsiflexed and inverted while the posterior talofibular ligament is stressed when the ankle is dorsiflexed and externally rotated.[19] The posterior talofibular ligament is rarely torn in isolation but can be injured in more severe dislocations.
3. The contribution of the various segments of the lateral collateral ligament in checking motion of the talus in the mortise depends on the position of the ankle joint.[14,17] Therefore, to assess these ligaments, testing needs to be done with the ankle in multiple positions and planes. The **anterior drawer** and **talar tilt tests** are clinical ligament stress tests used to assess the anterior talofibular and calcaneofibular ligaments, respectively. Although these tests are commonly used, there appears to be poor correlation with clinical test results in either identifying the degree of ligamentous disruption or the particular ligaments involved.[21]

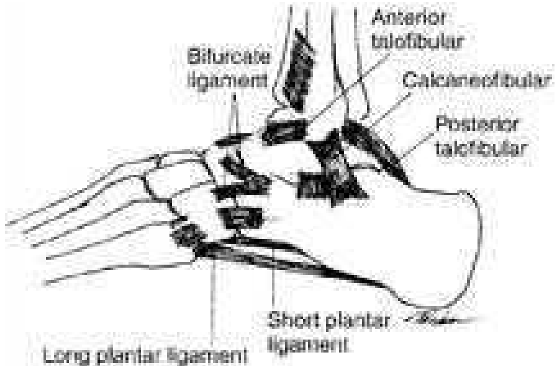

Figure 12–8 Lateral ligaments of the posterior ankle/foot complex.

In addition to the medial collateral and lateral collateral ligaments, portions of the extensor and peroneal retinacula of the ankle are also credited with contributing to stability at the ankle joint.[22,23] The inferior band of the **superior peroneal retinaculum** (Fig. 12–9), which lies close and parallel to the calcaneofibular ligament, appears to reinforce that ligament.[23] Because the ankle collateral ligaments and retinacula also contribute to stability of the subtalar joint they will be discussed again in that context.

Axis

In a neutral ankle position, the joint axis passes approximately through the fibular malleolus and the body of the talus and through or just below the tibial malleolus.[24] The fibular malleolus and its associated fibular facet on the talus are located more distally (Fig. 12–10A) and posteriorly (Fig. 12–10B) than the tibial malleolus and its associated tibial facet. The more posterior position of the fibular malleolus is due to the normal torsion or twist that exists in the distal tibia in relation to the tibia's proximal plateau. This **tibial torsion** (or tibiofibular torsion because both the tibia and fibula are involved with the rotation in the transverse plane)[25] accounts for the toe-out position of the foot in normal standing. The torsion in the tibia is similar to the torsion found in the shaft of the femur, although normally reversed in direction. The amount of outward tibial torsion

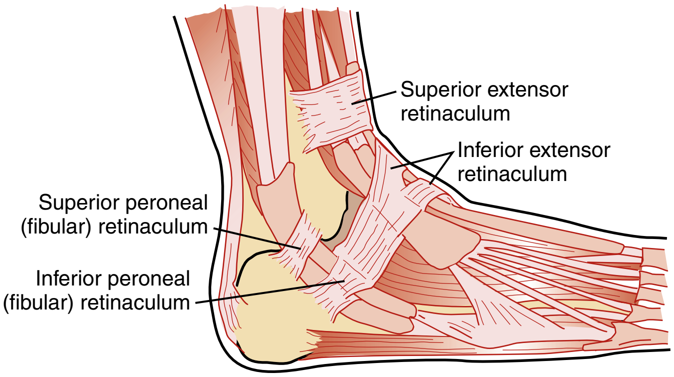

Figure 12–9 The superior and inferior extensor retinacula; the superior and inferior peroneal retinacula.

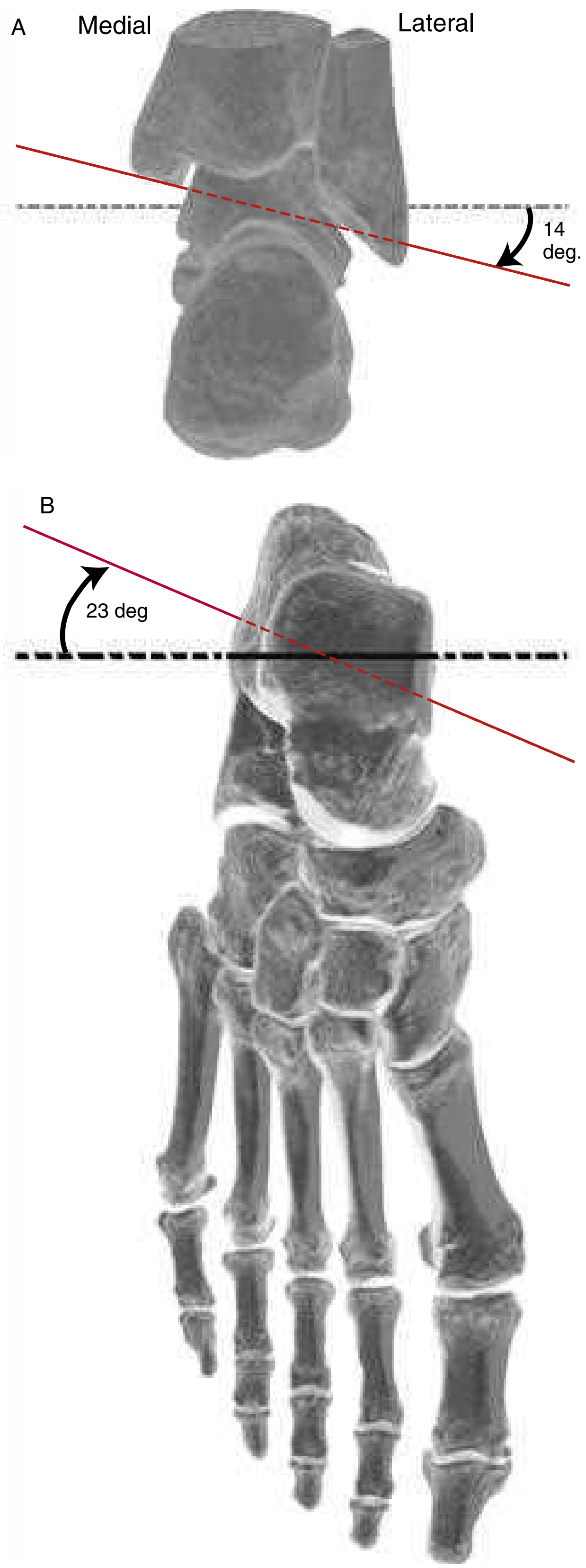

Figure 12–10 The axis of the ankle joint. **A**. Posterior view showing the mortise around the body of the talus and the average 14° inclination of the ankle axis from the transverse plane. **B**. Superior view showing the ankle axis rotated, on average, 23° from the frontal plane.

increases from birth until 10 years of age.[26] As tibial torsion increases, the axis of the ankle joint is positioned more laterally in the transverse plane. Although variation exists, clinical measures have reported values that approximate 19º of lateral torsion for both adult males and females.[27,28] It should be noted larger values for tibial torsion have been reported in studies that used computerized tomography.[26]

The ankle joint classically is considered to have one degree of freedom, with dorsiflexion/plantarflexion occurring between the talus and the mortise. At the ankle, *dorsiflexion* refers to a motion of the head of the talus (see Fig. 12–6) dorsally (or upward) while the body of the talus moves posteriorly in the mortise. *Plantarflexion* is the opposite motion of the head and body of the talus. Because of the lower position of the fibular malleolus, the axis of the ankle is inclined down on the lateral side. A classic anatomical study found this downward inclination to average 14°.[29] However, the amount of inclination may be highly variable between individuals. Stiehl used a simple hinged model with a level indicator to demonstrate how an axis inclined more distally and more posteriorly on the lateral side will create a motion across three planes (triplanar motion) while still around a single fixed axis.[30] He showed that dorsiflexion of the foot around a typically inclined ankle axis will not only bring the foot up but will also simultaneously bring it slightly lateral to the leg and appear to turn the foot longitudinally away from the midline. Conversely, plantarflexion around the same single oblique ankle axis will result in the foot's going down, moving medial to the leg, and appearing to turn the foot longitudinally toward the midline. When the foot is weight-bearing, the same relative pattern of motion exists when the tibia and fibula move on the foot. In weight-bearing ankle dorsiflexion, the leg (tibia and fibula) will move toward and medial to the foot, as well as appear to rotate medially in the transverse plane. The opposite occurs during weight-bearing ankle plantarflexion.

It should be noted that, while a pure hinge model helps to explain ankle motion, anatomical studies have demonstrated that the instantaneous axis of rotation for the ankle joint changes.[24,31–33] Talar rotation within the mortise, in both the transverse plane around a vertical axis (**talar rotation** or **talar abduction/adduction**) and in the frontal plane around an A-P axis (**talar tilt** or **talar inversion/eversion**), must occur to maintain tibiotalar congruency.[34] These talar motions are relatively small in comparison with sagittal plane dorsiflexion and plantarflexion motion. The literature has reported that 7° of medial and 10° of lateral talar rotation occur in the transverse plane. Talar tilt in the frontal plane was found to average 5° or less.[35–38]

Continuing Exploration 12-1:

Talocrural Joint Arthroplasty

Ankle replacement (total ankle arthroplasty) is being used more commonly to treat ankle arthritis. First introduced in the 1970s, the long-term results of total ankle arthroplasty demonstrated unacceptable failure rates. The use of a hinge type of prosthesis with a constrained design and fixed ankle axis was thought to contribute to loosening of some of these first-generation prostheses. In the 1990s second-generation total ankle replacements were developed. These newer prostheses were designed to better represent normal ankle anatomy and joint kinematics. This included allowing rotation of the talus to occur in multiple planes.[39,40]

Ankle Joint Function

The primary motions allowed at the ankle joint are dorsiflexion and plantarflexion. A commonly referenced source noted normal ankle joint ROM is 20° for dorsiflexion and 50° for plantarflexion.[41] Large variations in ankle motion can exist between individuals, depending on measurement technique and subject characteristics.[42] Differences in technique include the amount of force used to determine when end range of motion occurs and whether talocrural joint motion is isolated.[43] Ten degrees of ankle dorsiflexion is considered the minimal amount needed to ambulate without deviation.[44]

During ankle joint dorsiflexion/plantarflexion, the shape of the body of the talus facilitates joint stability. The trochlear (superior) surface of the talus is wider anteriorly than posteriorly (see Fig. 12–10B). When the foot is weight-bearing, dorsiflexion occurs as the tibia rotates over the talus. As the tibia rotates over the talus, the concave tibiofibular segment slides forward on the trochlear surface of the talus. The wider anterior portion of the talus will "wedge" into the mortise, separating the tibia and fibula to enhance stability of the ankle joint. The loose-packed position of the ankle joint is in plantarflexion when the narrower posterior body of the talus is within with the mortise. The ankle being less stable in plantarflexion is consistent with the pattern of injury to the lateral ankle ligaments.[14] The ability of the ankle to distribute weight-bearing forces combined with the mechanical properties of the tibiotalar articular cartilage reduces the risk of osteoarthritis at the talocrural joint.[39] In fact, post-traumatic arthritis is the most common type of arthritis occurring at the ankle.[45] Trauma may lead to ankle arthritis not only because of direct cartilage damage but also structural changes that lead to biomechanical abnormalities.

The asymmetry in size and orientation of the lateral and medial facets of the ankle joint contribute to changes in the ankle mortise that occur during ankle dorsiflexion. The lateral (fibular) facet is substantially larger than the medial (tibial) facet, and its surface is oriented slightly obliquely to that of the medial facet (see Fig. 12–10). Inman and Mann proposed that the body of the talus can be thought of as a segment of a cone lying on its side with its base directed laterally.[46] The cone should be visualized as "truncated" or cut off on either end at slightly different angles[25] (Fig. 12–11). The asymmetry in size and orientation of the facets means that the distal fibula moving on the larger lateral facet of the talus must undergo a greater displacement (in a slightly different plane) than the tibial malleolus as the tibia and fibula move together during dorsiflexion. The greater arc of motion for the fibular malleolus than for the tibial malleolus results in superior/inferior motion and medial/lateral rotation of the fibula that requires mobility of the fibula at both the proximal and the distal tibiofibular joints.

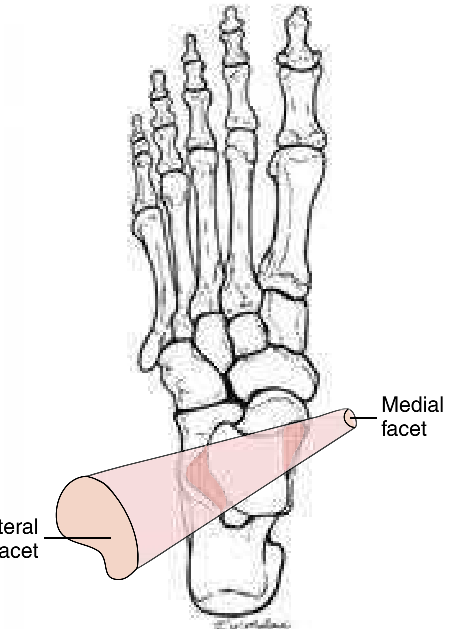

Figure 12–11 The three articular surfaces of the talus (the trochlea, smaller medial facet, and larger lateral facet) can be pictured as part of a cone-shaped surface, with ends of the cone cut off (the larger end of the cone facing laterally).

Although fibula motion can occur in transverse, sagittal, and frontal planes with ankle plantarflexion and dorsiflexion, the amount of motion seems to be highly variable between individuals.[47] Differences in fibular motion may be related to orientation of the proximal tibiofibular facet, with more mobility available in the facets that are more vertical. Effectively, however, mobility of the fibula at the tibiofibular joints should be considered a component of normal ankle motion. One might expect the magnitude of proximal tibiofibular joint motion should exceed that of the distal tibiofibular joint, given that small motion at the distal fibula would be magnified at the opposite (proximal) end. This presumably accounts for the proximal joint's being synovial, whereas the distal joint is a comparatively less mobile syndesmosis joint.

Continuing Exploration 12-2:

Tibiofibular and Ankle Joint Linkage

Some mobility of the fibula appears to be required at the proximal and distal tibiofibular joints to allow the talus to posteriorly rotate fully into the ankle mortise during ankle dorsiflexion. Injury that disrupts the ligaments supporting the distal tibiofibular joint can cause increased motion of the fibula.[48] However, the role of fibular movement in allowing full ankle dorsiflexion remains unclear. Clinically, a limitation in dorsiflexion can be seen after long periods of

Continued

immobilization or when the distal tibiofibular joint is fixated with open reduction internal fixation (ORIF). Many times this lack of motion is attributed to reduced fibular movement. However, this is not consistent with cadaveric studies that found surgical fixation of the distal tibiofibular joint did not affect dorsiflexion range of motion.[49] The functional implications for restricted movement of the fibula (or mortise) are unclear.

Ankle dorsiflexion and plantarflexion movements are limited primarily by soft tissue restrictions. Active or passive tension in the triceps surae (**gastrocnemius** and **soleus muscles**) is the primary limitation to dorsiflexion. Typically, less ankle dorsiflexion range of motion is seen with the knee extended compared to a knee in a flexed position (as demonstrated in Patient Case 12-1). This can be attributed to the gastrocnemius muscle lengthening across two joints when the knee is extended.[50] Ankle dorsiflexion is limited by the soleus and posterior talocrural joint capsule when the knee is flexed. Tension in the **tibialis anterior, extensor hallucis longus,** and **extensor digitorum longus** muscles is the primary limit to plantarflexion. The medial and lateral collateral ankle ligaments are under constant tension during dorsiflexion and plantarflexion and therefore help to guide the sagittal plane motion.[51] The more important function of the collateral ligaments appears to be in minimizing side-to-side movement or rotation of the mortise on the talus. The ligaments are assisted in that function by the muscles that pass on either side of the ankle. The **tibialis posterior, flexor hallucis longus,** and **flexor digitorum longus** muscles help protect the medial aspect of the ankle; the **peroneus longus** and **peroneus brevis** muscles protect the lateral aspect. Bony checks of any of the potential ankle motions are rarely encountered unless there is extreme hypermobility (as may be found among gymnasts or dancers) or a failure of one or more of the other restraint systems. A more complete analysis of the function of the muscles crossing the ankle will be presented later, as all muscles of the ankle cross at least two and generally three or more joints of the ankle and foot.

THE SUBTALAR JOINT

The talocalcaneal, or subtalar, joint is a composite joint formed by three separate plane articulations between the talus superiorly and the calcaneus inferiorly. Together, the three surfaces provide a triplanar movement around a single joint axis. Functioning of the subtalar joint in weight-bearing is critical for dampening proximal rotational forces while maintaining contact of the foot with the ground.

Subtalar Joint Structure

The subtalar joint articulating surfaces are highly variable, but the posterior articulation is consistently the largest of the three articulations found between the talus and calcaneus. The posterior articulation is formed by a concave facet on the undersurface of the body of the talus and a convex facet on the body of the calcaneus; the smaller anterior and medial talocalcaneal articulations are formed by two convex facets on the inferior body and neck of the talus and two concave facets on the calcaneus (Fig. 12–12). The anterior and medial articulations, therefore, have an intra-articular configuration that is the reverse of that found at the posterior facet. Between the posterior articulation and the anterior and medial articulations, there is a bony tunnel formed by a **sulcus** (concave groove) in the inferior talus and superior calcaneus. This funnel-shaped tunnel, known as the **tarsal canal,** runs obliquely across the foot. Its large end (the **sinus tarsi**) lies just anterior to the fibular malleolus (Fig. 12–13); its small end lies below the tibial malleolus and above a bony outcropping on the calcaneus called the **sustentaculum tali** (see Fig. 12–12). The tarsal canal and ligaments running the length of the tarsal canal divide the posterior articulation and the anterior and medial articulations of the subtalar joint into two separate noncommunicating joint cavities. The posterior articulation has its own capsule; the anterior and medial articulations share a capsule with the **talonavicular joint.**[52]

Wang and colleagues found that the subtalar articular surfaces, although smaller than those of the ankle joint surfaces, showed a similar proportion of contact across surfaces under similar conditions.[53] These investigators found that

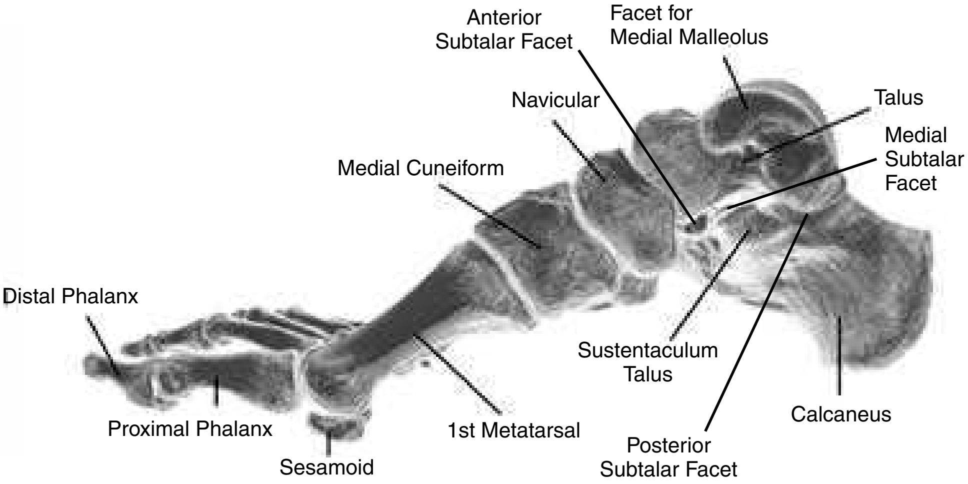

Figure 12–12 Medial view of the foot, showing the talus sitting on the calcaneus (the subtalar joint).

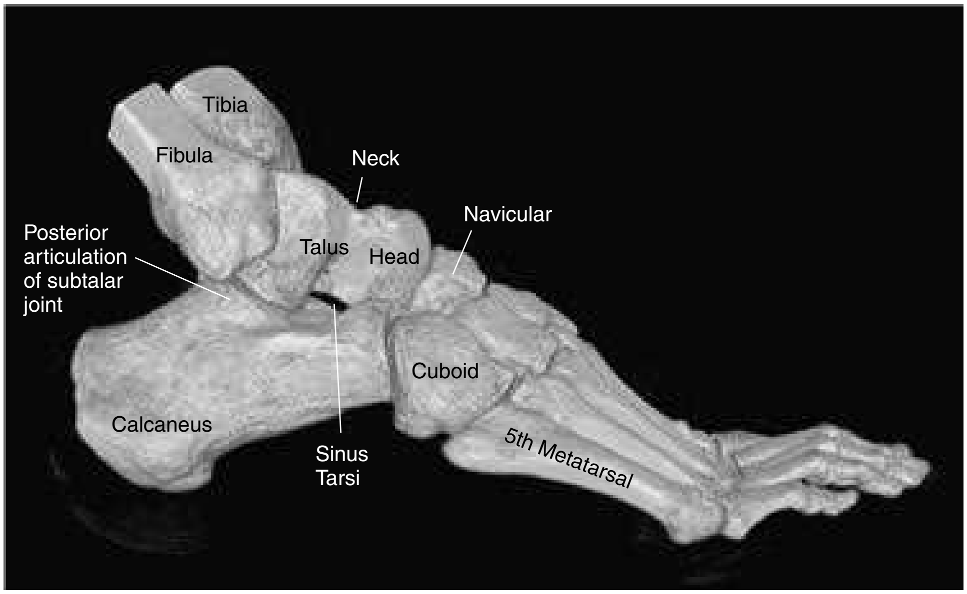

Figure 12–13 Lateral view of the foot, showing the talus sitting on the calcaneus (the subtalar joint). The sinus tarsi is the lateral opening of the tarsal canal.

the posterior facet received 75% of the force transmitted through the subtalar joint. They also determined that the pressure in the posterior facet was similar to that at the medial and anterior facets, given the larger contact area of the posterior facet. Like the ankle joint, the subtalar joint rarely undergoes degenerative change unless damaged by high stresses (e.g., fracture).

Ligaments

The subtalar joint is a stable joint that is rarely dislocated. It has a congruent osseous anatomy as well as strong ligamentous support. The subtalar joint receives support from the ligamentous structures that support the ankle, as well as from ligamentous structures that only cross the subtalar joint.[52] Through anatomical studies, Harper described a number of structures contributing to the lateral support of the subtalar joint.[54] These included, from superficial to deep, the calcaneofibular, lateral talocalcaneal, **cervical,** and **interosseous talocalcaneal ligaments.** The cervical ligament (Fig. 12–14) is the strongest of the talocalcaneal structures.[54,55] It lies in the anterior sinus tarsi and joins the neck of the talus to the neck of the calcaneus (hence its name). The interosseous talocalcaneal ligament lies more medially within the tarsal canal and follows an oblique path from the talus to the calcaneus[56] (see Fig. 12–14). Divisions of the **inferior extensor retinaculum** also provide stability to the subtalar joint superficially as well as through connections within the tarsal canal.[56] Although there is consensus that the subtalar joint is very stable, controversy exists as to the hierarchy of support between the bony configuration, calcaneofibular ligament, cervical ligament, and interosseous ligament.[57]

Subtalar Joint Function

Although the subtalar joint is composed of three articulations, the alternating convex-concave facets limit the potential mobility of the joint. When the talus moves on the posterior facet of the calcaneus, the articular surface of the talus should, theoretically, slide in the *same* direction as the bone moves—a concave surface moving on a stable convex surface. However, at the medial and anterior joints, the talar surfaces (again, theoretically) should glide in a direction opposite to movement of the bone—a convex surface moving on a stable concave surface. Motion of the talus on the calcaneus, therefore, is a complex twisting or screwlike motion that can proceed only as long as the facets can accommodate simultaneous and opposite motions across the surfaces. The result is a triplanar motion of the talus around a single oblique joint axis, producing the motion of supination/pronation.

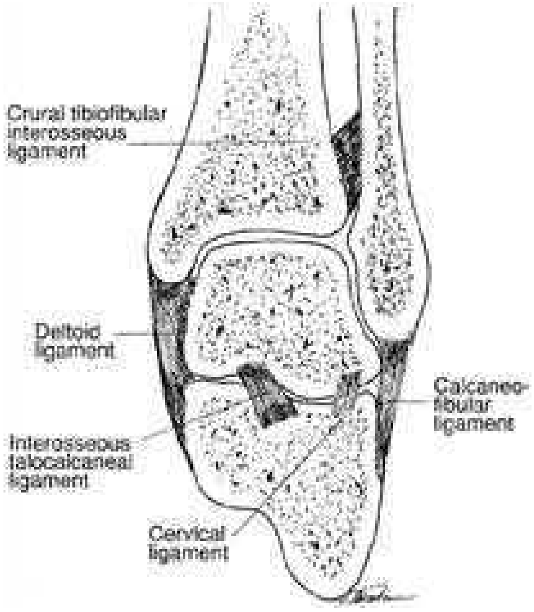

Figure 12–14 The ligaments of the subtalar joint (in a posterior cross-sectional view).

The Subtalar Axis

The axis for subtalar supination/pronation has been the subject of many investigations that indicate substantial variability, even among healthy individuals without impairments. Manter[58] reported that the average subtalar axis was (1) inclined 42° upward and anteriorly from the transverse plane (with a broad interindividual range of 29° to 47°) (Fig. 12–15A), and (2) inclined medially 16° from the sagittal plane (with a broad interindividual range of 8° to 24°) (Fig. 12–15B). Clearly, motion about this oblique axis will cross all three planes. Supination/pronation, like the triplanar ankle joint motion, can be modeled by a single oblique hinge joint.[59] Although the triplanar motions of pronation/supination can be described by its three component (cardinal) motions, these subtalar component motions are coupled and cannot occur independently. The coupled motions must occur simultaneously as the calcaneus (or talus) twists across the subtalar joint's three articular surfaces.

To understand the components of subtalar pronation/supination, we can consider how the subtalar axis varies from the cardinal axes shown in Figure 12–2. If the subtalar joint axis were vertical, the motion around that axis would be as purely abduction/adduction; if the subtalar axis were longitudinal, the motion would be purely inversion/eversion; and if the subtalar axis were coronal, the motion would be purely plantarflexion/dorsiflexion. In reality, the subtalar axis lies about halfway between being longitudinal and being vertical. Consequently, pronation/supination includes about equal magnitudes of eversion/inversion and abduction/adduction. The subtalar axis is inclined only very slightly toward being a coronal axis (~16°) and therefore has only a small component of dorsiflexion/plantarflexion. The contribution of each of the coupled movements to supination or pronation will depend greatly on individual differences in inclination of the subtalar axis. As one example, if the subtalar axis is inclined upwardly only 30° (rather than the average of 42°), the relative amount of inversion/eversion will be much greater than the relative amount of adduction/abduction because the axis is closer to being longitudinal. We will now examine how subtalar joint component motions are coupled to constitute the complex motions of pronation/supination in both non-weightbearing and weight-bearing positions.

Non-Weightbearing Subtalar Joint Motion

In non-weightbearing supination and pronation, subtalar motion is described by motion of its distal segment (the calcaneus) on the stationary talus and lower leg, where the reference point on the calcaneus is its anteriorly located head (see Fig. 12–6). Non-weightbearing supination is composed of the coupled calcaneal motions of adduction, inversion, and plantarflexion. Pronation of the non-weightbearing calcaneus on the fixed talus and lower leg is composed of the coupled motions of abduction, eversion, and dorsiflexion (Table 12–1). The most readily observable

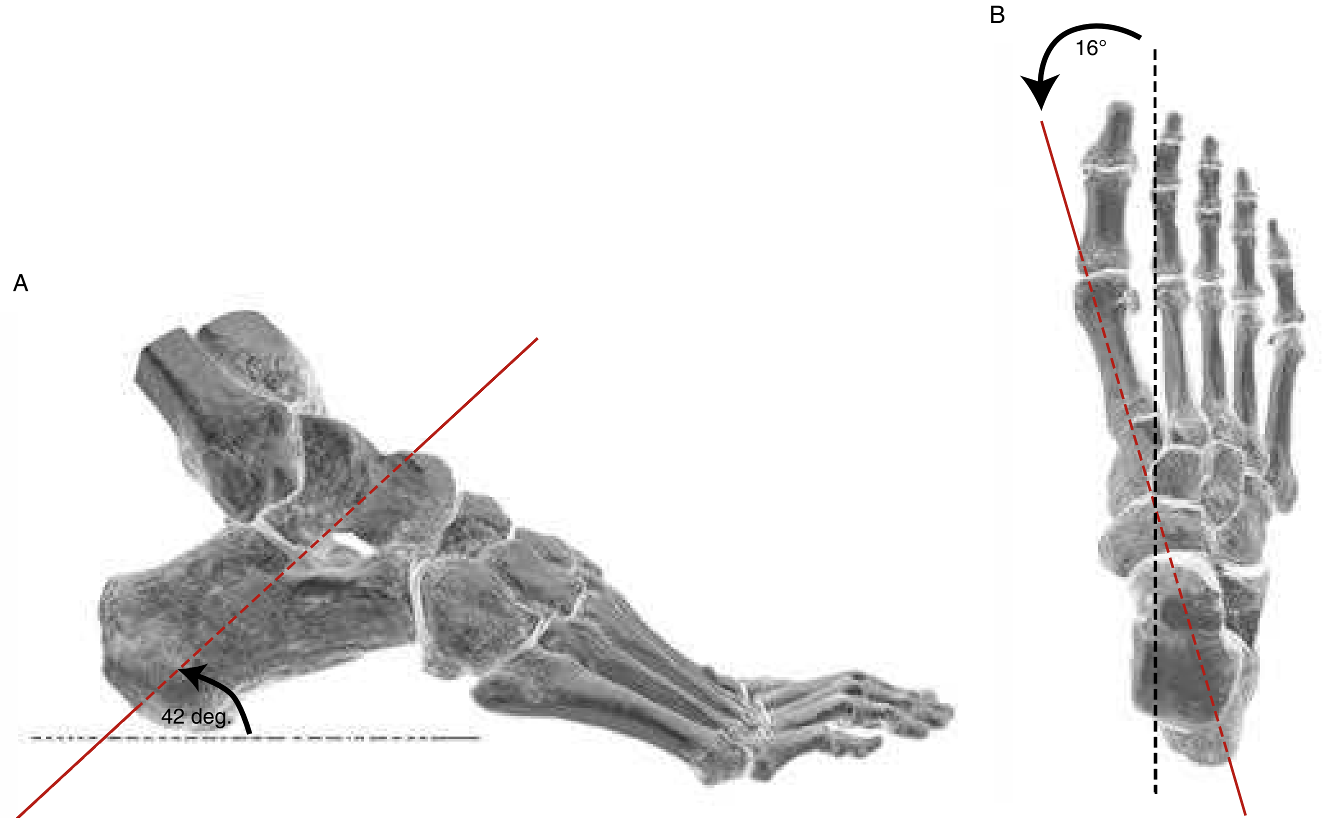

Figure 12–15 Axis of the subtalar joint **(A)** inclined up from the transverse plane approximately 42° and **(B)** inclined medially from an A-P axis approximately 16°.

Table 12–1 Summary of Coupled Subtalar Motions: Coupled Movements of Subtalar Pronation/Supination

	NON-WEIGHTBEARING	WEIGHT-BEARING
Supination	Calcaneal inversion (or varus) Calcaneal adduction Calcaneal plantarflexion	Calcaneal inversion (or varus) Talar abduction (or lateral rotation) Talar dorsiflexion Tibiofibular lateral rotation
Pronation	Calcaneal eversion (or valgus) Calcaneal abduction Calcaneal dorsiflexion	Calcaneal eversion (or valgus) Talar adduction (or medial rotation) Talar plantarflexion Tibiofibular medial rotation

of the coupled motions of the calcaneus during pronation and supination are eversion and inversion, respectively. These motions of the calcaneus are often observed at the posterior calcaneus with the subject prone and the foot and lower leg over the end of the plinth. Eversion (Fig. 12–16A) may also be referred to as valgus movement of the calcaneus. Inversion (Fig. 12–16B) may also be referred to as varus movement of the calcaneus. The eversion and inversion components of pronation/supination appear as if they are occurring in isolation. However, the coupled (but less visible) components of calcaneal abduction and dorsiflexion must simultaneously accompany eversion, and the coupled components of calcaneal adduction and plantarflexion must simultaneously accompany inversion.

Concept Cornerstone **12-3**

Terminology Revisited

Although the apparent interchangeable use of the terms *pronation/supination* and *eversion/inversion* to describe the composite uniaxial motions of the subtalar joint appears contradictory, the disagreement in terminology is not as discrepant as it first appears. When the composite term *pronation* is used to describe subtalar motion, the coupled calcaneal component of *eversion* will always be part of the motion. If the composite term *eversion* is used to describe subtalar motion, then the coupled calcaneal component of *pronation* will always be part of the motion. Consequently, *pronation* and *eversion* are invariably linked, regardless of the terminology frame of reference. The same is true of *supination* and *inversion*; whether used as a composite term or a component term, *supination* and *inversion* are invariably linked. It would certainly be less troublesome if a universal definitional framework were accepted. However, the wary reader who understands the association between *supination/pronation* and *inversion/eversion* may be able to infer definitions when they are not overtly offered by authors.

Weight-Bearing Subtalar Joint Motion

When an individual is weight-bearing, the calcaneus is on the ground and generally free to move around a longitudinal axis (inversion/eversion motion) but limited in its ability to move around a coronal axis (plantarflexion/dorsiflexion motion) and vertical axis (adduction/abduction motion) because of the superimposed body weight. Consequently, the coupled motions that contribute to pronation/supination cannot be accomplished exclusively by the calcaneus in weight-bearing. Although the weight-bearing calcaneus will continue to contribute the inversion/eversion component of subtalar motion, the other two coupled components of the subtalar motion (abduction/adduction and dorsiflexion/plantarflexion) will be accomplished by movement of the talus (whereby the head of the talus is used as the reference) on the more fixed calcaneus rather than by movement of the calcaneus on the relatively fixed talus. The motion accomplished at any joint around a given axis remains unchanged whether the distal segment of the joint moves or whether the proximal segment moves. When the proximal segment moves on the distal segment, however, the motion of the proximal segment will be the opposite of what was described as occurring to the distal segment. In weight-bearing subtalar motion, the direction of the component movement contributed by the talus is the opposite of what the calcaneus would contribute, although the same relative motion occurs between the segments.

In weight-bearing supination, the calcaneus continues to contribute the component of inversion. However, the calcaneus cannot adduct and plantarflex in weight-bearing, and so the remaining coupled components of subtalar supination are accomplished by abduction and dorsiflexion of the head of the *talus*. Weight-bearing subtalar supination (see Table 12–1), therefore, is observable as inversion (or varus movement) of the calcaneus, whereas the less visible dorsiflexion and abduction of the head of the talus are reflected in elevation of the medial longitudinal arch and a convexity on the dorsal lateral midfoot. Although subtalar joint supination is a normal foot motion, a foot that appears fixed in this position often is called a "supinated" or **cavus foot.**

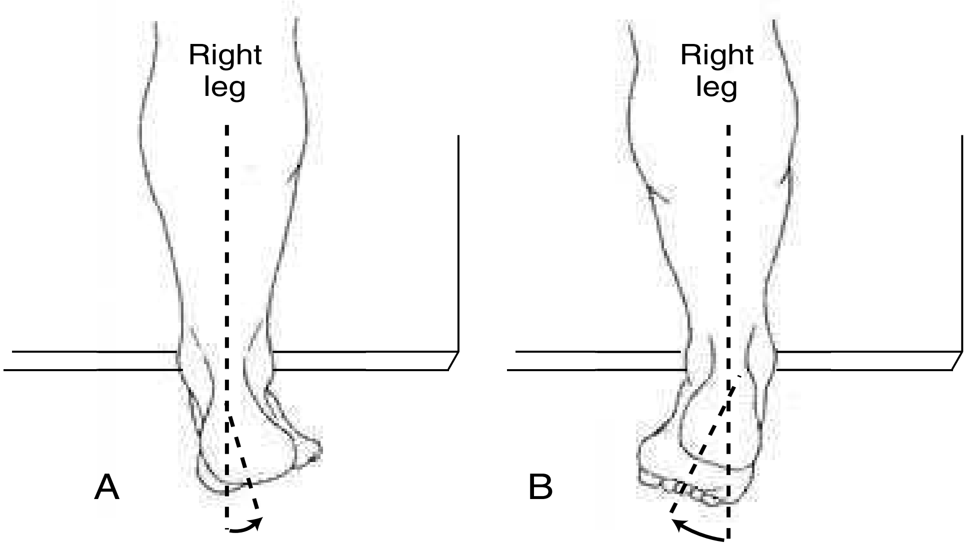

Figure 12–16 Non-weightbearing motion at the right subtalar joint. **A.** Pronation of the subtalar joint is observable as eversion (valgus movement) of the calcaneus, although the coupled motions of dorsiflexion and abduction of the calcaneus must also be occurring. **B.** Supination of the subtalar joint is observable as inversion (varus movement) of the calcaneus, although the coupled motions of plantarflexion and adduction of the calcaneus must also be occurring.

Weight-bearing subtalar pronation is accomplished by the coupled component movements of eversion of the calcaneus and plantarflexion and adduction of the head of the talus (see Table 12–1). In standing, the calcaneus can be observed to move into eversion (or valgus movement), whereas talar adduction and plantarflexion are reflected in a lowering of the medial longitudinal arch and a bulging or convexity in the plantar medial midfoot. Although subtalar joint pronation is a normal foot motion, a foot that appears fixed in this position often is called "pronated," **pes planus,** or **flat foot.**

The most critical functions of the foot occur in weight-bearing. When the foot is weight-bearing and the head remains relatively positioned over one or both feet, the joints of the lower extremity effectively form a closed chain. Consequently, the kinematics and kinetics of the subtalar joint will affect and be affected by more proximal and distal joints. An important consequence of closed-chain subtalar function can be seen in its interdependence with lower extremity or leg rotation.

Weight-Bearing Subtalar Joint Motion and Its Effect on the Leg

During weight-bearing subtalar supination/pronation, the coupled component motions of dorsiflexion/plantarflexion and abduction/adduction of the talar head require that the body of the talus move as well. The body of the talus is, of course, lodged within the superimposed mortise. Dorsiflexion of the head of the talus requires the body of the talus to slide posteriorly within the mortise (Fig. 12–17A), whereas plantarflexion of the head of the talus requires the body of the talus to move anteriorly within the mortise. The tibia (leg) remains unaffected by the talar dorsiflexion/plantarflexion as long as the ankle joint is free to move. However, the ankle joint cannot absorb the coupled component motions of talar abduction/adduction without affecting the leg.

When the head of the talus abducts in weight-bearing subtalar supination, the body of the talus must rotate laterally in the transverse plane (Fig. 12–17B). When the head of the talus adducts in weight-bearing subtalar pronation, the body of the talus must rotate medially in the transverse plane. Because the body of the talus can rotate only minimally at most *within* the mortise, rotation of the body of the talus can occur in weight-bearing only if the superimposed mortise moves *with* the talus. When the subtalar joint supinates in a weight-bearing position, the coupled component of talar abduction carries the mortise (the tibia and fibula) laterally, producing lateral rotation of the leg. Correspondingly, weight-bearing subtalar joint pronation causes talar adduction, with the body of the talus rotating medially and carrying the superimposed tibia and fibula into medial rotation.

Through the component movements of abduction and adduction of the talus, weight-bearing subtalar joint motion directly influences the segments and joints superior to it. A weight-bearing subtalar joint maintained in a pronated position (e.g., a flat foot) can create a medial rotation force on the leg that may influence the knee and hip joints. Just as subtalar pronation and supination may impose rotary forces on the leg in weight-bearing, so too may rotation of the leg influence the subtalar joint. When a lateral rotary force is imposed on the weight-bearing leg (as when you rotate to the right around a planted right foot), the lateral motion of the leg carries the mortise and its mated body of the talus laterally. Lateral rotation of the body of the talus (adduction of the head of the talus) cannot occur without its coupled components of talar dorsiflexion and calcaneal inversion, which produce supination of the subtalar joint. A medial rotary force imposed on the weight-bearing leg will necessarily result in subtalar pronation as the talus is medially rotated (adducted) by the rotating tibiofibular mortise and carries with it the coupled components of

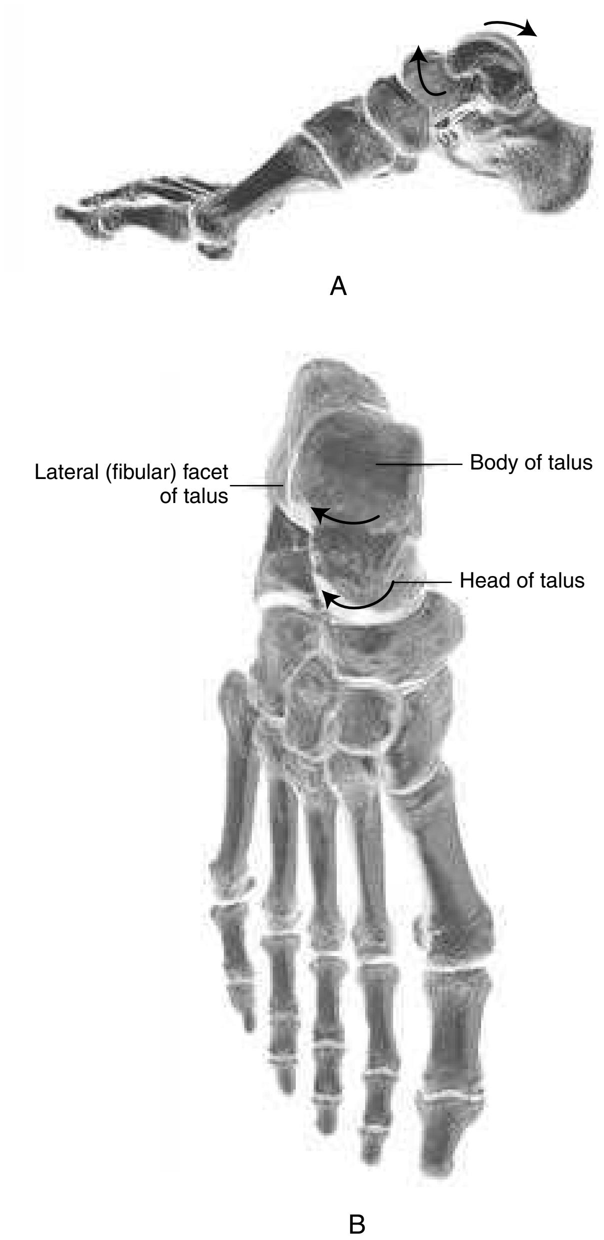

Figure 12–17 **A.** Dorsiflexion of the head of the talus during weight-bearing subtalar supination slides the body of the talus posteriorly within the tibiofibular mortise. **B.** Abduction of the head of the talus during weight-bearing subtalar supination rotates the body of the talus laterally, potentially taking the tibiofibular mortise along with it.

talar plantarflexion and calcaneal eversion. The interdependence of the leg and talus were mechanically represented by Inman and Mann, who used the concept of the subtalar joint as a mitered hinge.[46] This mitered hinge concept (Fig. 12–18) presents a good visualization of the concept of the interdependence of the leg and foot through the oblique subtalar axis.

This kinematic coupling is thought to play an important role in lower extremity injuries when abnormalities exist.[60] While this hinge model helps to understand movement between the subtalar joint and leg, in vivo studies have found great deal of individual variability in movement coupling.[61–63]

CASE APPLICATION

Lower Extremity Rotation and Subtalar Joint Position

case 12–1

When the foot is maintained in a more pronated position during weight-bearing, as is true for Mr. Benson, there can be a sustained medial rotation force on the lower extremity. That force may cause medial rotation at the knee and hip. However, it may be difficult to determine if subtalar pronation is causing the medial rotation of extremity or if the pronation is a result of top down proximal forces from the hip and/or knee.

Range of Subtalar Motion and Subtalar Neutral

The range of subtalar supination and pronation is difficult to determine objectively because of the triplanar nature of the movement and because the component contributions vary with the inclination of the subtalar axis. The calcaneal inversion/eversion (varus/valgus) component of subtalar motion can be measured in both weight-bearing and non-weightbearing positions by using the posterior calcaneus and posterior midline of the leg as reference points. This technique assumes that neutral position (0°) is when the two posterior lines align to form a straight line (see Fig. 12–16). For individuals without impairments, 5° to 10° of calcaneal eversion (valgus) and 20° to 30° of calcaneal inversion (varus) have been reported for a total range of 25° to 40°.[64–66] This measurement technique has been found to be problematic, as isolating subtalar motion from talocrural motion is clinically difficult.[67,68] Additionally, this measurement technique may have problems related to reliability.[69–73] Although it is acknowledged that the ranges of calcaneal inversion/

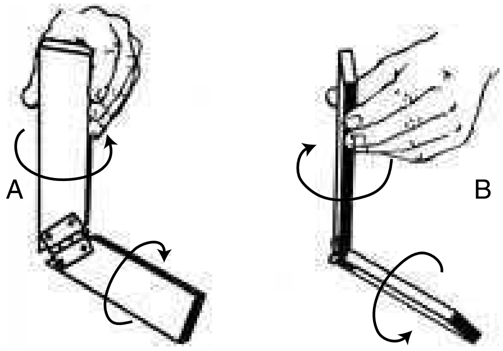

Figure 12–18 The subtalar joint can be visualized as a mitered hinge between the leg and the foot. **A.** Medial rotation of the weight-bearing leg imposes pronation on the distally located subtalar joint. **B.** Lateral rotation of the leg proximally imposes supination on the distally located subtalar joint. *(From Mann RA: Biomechanics of running. In Mann RA [ed]: Surgery of the Foot [ed. 5], p 19. St. Louis, MO, CV Mosby, 1986, with permission.)*

eversion are not equivalent in magnitude to those of subtalar supination/pronation, the ranges should be directly proportional.

The variability in the inclination of the subtalar axis described by Manter[58] directly affects the range of the coupled components of subtalar motion. If the axis is inclined upward less than the average of 42° (see Fig. 12–15A), the subtalar axis will more closely approximate a longitudinal axis. The proportion of inversion/eversion of the calcaneus that is part of subtalar motion will concomitantly increase, whereas the proportion of coupled abduction/adduction of the calcaneus (or talus) will decrease. Because the change in inclination of the subtalar axis will affect both the foot and leg position in weight-bearing, a considerable amount of attention has been given to determining how an individual's subtalar axis might differ from the average (or "standard") axis. This has led to an attempt to define an individual's **subtalar neutral** position of the subtalar joint and use it as a reference for a "normal" or "ideal" foot position. The presumption that an individual's neutral subtalar joint position may deviate from the point at which the midlines of the posterior calcaneus and the posterior leg coincide, with a medial increase in that angle referred to as valgus and an decrease referred to as varus (see Fig. 12–3).

The subtalar neutral position has been defined differently by various investigators, with some issues raised as to the appropriateness of the concept or the measurement techniques. Root and colleagues defined subtalar neutral position as the point from which the calcaneus will invert twice as many degrees as it will evert.[65] Bailey and colleagues used radiographic evidence to demonstrate that the neutral position of the subtalar joint was not always found two thirds of the way from maximum supination, although the average neutral subtalar position for their subjects was close to this value.[74] Elveru and colleagues proposed palpating the medial head and neck of the talus while supinating and pronating the subtalar joint, with subtalar neutral as the point where the talus is equally positioned between the fingers.[69] This technique is fairly subjective, and the inter-rater reliability is generally poor.[69,75] Only one study has noted acceptable reliability when standardized methods and practice were employed as part of the methodology.[76] This study however has not been replicated.

The work of Cornwall and McPoil[77] can be used to highlight some of the controversy that exists around the definition and application of the term *subtalar neutral.* Cornwall and McPoil described calcaneal motion during walking in 153 subjects between 18 and 41 years old with no history of foot impairments. They reported that the calcaneus is inverted 3.0° (±2.7°) at heel strike relative to the tibia and then everts 2.2° (±62.4°) by 55% of stance phase. After a period of eversion, the motion is reversed to 5.5° (±3.2°) of inversion just before the foot leaves the ground.[77] When looking at the overall results of the study, it was apparent the subtalar joint does not function about a neutral position during walking but actually functions more in more of an everted position.[77] This finding was in agreement with their previous work.[78–80] They concluded that the "neutral" position of the rear foot during the walking cycle was better represented by the resting position of the calcaneus with respect to the lower leg when weight-bearing as opposed to the palpated subtalar joint neutral position. The resting position of the calcaneus in relaxed bilateral stance averaged approximately 3.5° of calcaneal valgus angulation (eversion) in a group of their subjects.[78] These findings appear to support the conclusion of Åstrom and Arvidson who noted that the normal weight-bearing foot is more pronated than previously thought and that reliance on the palpated subtalar neutral position could lead to over diagnosis of excessive subtalar pronation.[66] It should also be acknowledged that very few individuals may actually have an "ideal" foot position[81] and clinical examination may not be able to predict dynamic foot function.[82,83]

The calcaneal inversion/eversion components of subtalar motion have received a large amount of attention because of the availability of measurement strategies. The talar dorsiflexion/plantarflexion and abduction/adduction components of weight-bearing subtalar motion are more difficult to measure. Estimates of talar abduction/adduction have been made by measuring the tibial rotation that accompanies abduction/adduction of the talus in weight-bearing.[77] In vivo measures of subtalar motion using bone-anchored markers have been made. A recent study found total talocalcaneal joint motion averaged 6.8º, 9.8º, and 7.5º in the sagittal, frontal, and transverse planes, respectively.[62] It should be noted this study also found a large variation in motion between subjects.[62]

Concept Cornerstone 12-4

Subtalar Joint Neutral Summary

Morton Root, a podiatrist, is credited with describing the theoretical management approach to foot and ankle problems that focused on the subtalar joint neutral position.[65] A basic premise of the approach is that the subtalar joint should be in a neutral position during midstance. The approach to intervention for several foot deformities includes use of an orthotic device to "balance the foot" and achieve a defined subtalar joint neutral position during midstance. However, as outlined, there are a number of problems with this approach. These problems include the subtalar joint not actually being in neutral at midstance, poor reliability of measures, and inability to predict dynamic function for static measure. Additionally, although the subtalar joint position may contribute to our understanding of foot structure and function, the influence of subtalar position must be considered with other interdependent factors, including boney abnormalities (i.e., femoral or tibial rotation); extrinsic factors such as footwear, running surface, and activity level (magnitude and change); and physiological factors such as obesity or disease.[1,84]

When the subtalar joint is non-weightbearing (open chain), the motions of the subtalar joint and the leg are independent and do not influence each other. When the foot is weight-bearing, a primary function of the subtalar joint is to absorb the imposed lower extremity transverse plane rotations that occur during walking and other weight-bearing activities. Such rotations would otherwise spin the foot on the ground or disrupt the ankle joint by rotating the talus within the mortise. In supination, ligamentous tension draws the subtalar joint surfaces together, which results in locking (close packing) of the articular surfaces. Conversely, the adduction and plantarflexion of the talus that occur in weight-bearing pronation cause a splaying (spreading) of the adjacent tarsal bones that permits some intertarsal mobility. The role of the ligaments in contributing to mobility or stability at the subtalar joint, however, is somewhat controversial.[57] Sarrafian believed that the position of the cervical and interosseous talocalcaneal ligaments are along the subtalar axis, which causes these structures to remain tight in both pronation and supination.[64] According to this premise, individual shifts in location of the axis or of the ligaments could account for discrepant findings of other investigators.

The subtalar joint is strategically located between the ankle joint proximally and the **transverse tarsal joint** distally. We have already discussed how motions at the subtalar joint are associated with motions of the leg and ankle joint in weight-bearing. We will now focus our attention on motion between the talus and the navicular bone and between the calcaneus and cuboid bones. These articulations are grouped differently according to various authors, but the approach of this chapter will focus on the transverse tarsal joint as the primary functional unit accounting for motion in the midfoot. We will also see how motion at the subtalar joint influences motion at the transverse tarsal joint.

TRANSVERSE TARSAL JOINT

The transverse tarsal joint, also called the **midtarsal** or **Chopart joint,** is a compound joint formed by the talonavicular and **calcaneocuboid joints** (Fig. 12–19). The two joints together present an S-shaped joint line that transects the foot horizontally, dividing the hindfoot from the midfoot and forefoot. The navicular and the cuboid bones are considered, in essence, immobile in the weight-bearing foot. Transverse tarsal joint motion, therefore, is often considered to be motion of the talus and of the calcaneus on the relatively fixed naviculocuboid unit. Motion at the compound transverse tarsal joint, however, is more complex than the relatively simple joint line might suggest and occurs synchronously with motion at the subtalar joint.[85]

Transverse Tarsal Joint Structure

Talonavicular Joint

The proximal portion of the talonavicular articulation is formed by the anterior portion of the head of the talus, and the distal portion of the articulation by the concave posterior aspect of the navicular bone. We also noted earlier that the talar head articulates inferiorly with the anterior and medial facets of the calcaneus as the anterior part of the subtalar joint. A single joint capsule encompasses the talonavicular joint facets and the anterior and medial facets of the subtalar joint. The inferior aspect of this joint capsule is formed by the **plantar calcaneonavicular ligament (spring ligament)** that spans the gap between the calcaneus and navicular immediately below the talar head. The capsule is reinforced medially by the deltoid and laterally by the **bifurcate ligaments.** Given these structural relationships, the large convexity of the head of the talus can be considered the "ball" that is received by a large "socket" formed anteriorly by the concavity of the navicular bone, inferiorly by the concavities of the anterior and medial calcaneal facets and by the plantar calcaneonavicular ligament, medially by the deltoid ligament, and laterally by the bifurcate ligament (Fig. 12–20).

Figure 12–19 The talonavicular joint and calcaneocuboid joint form a compound joint known as the transverse tarsal joint line that transects the foot.

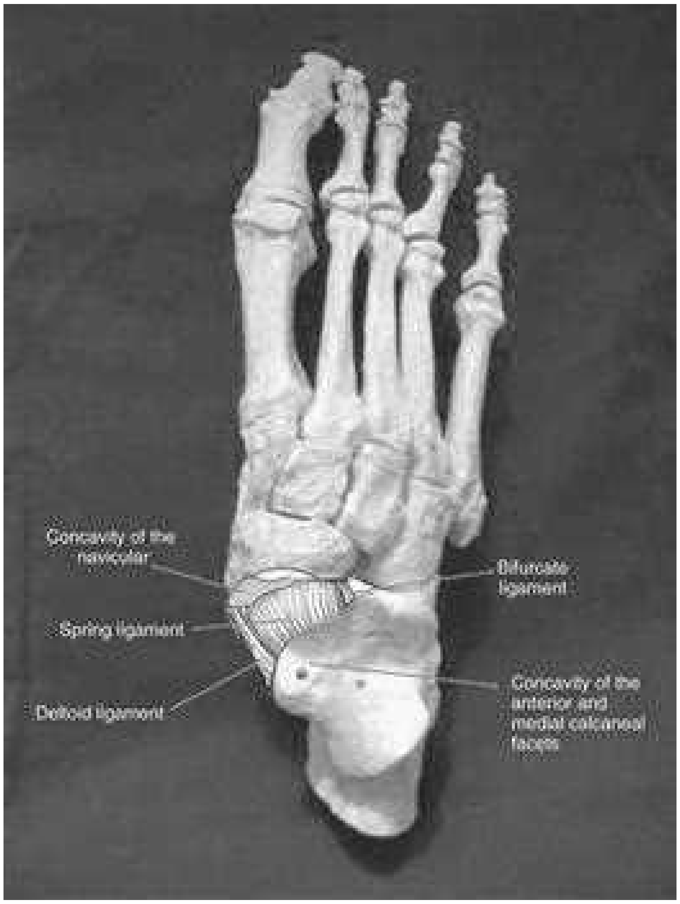

Figure 12–20 With the talus removed, this superior view shows the concavity ("socket") formed by the navicular bone anteriorly, the deltoid ligament medially, the medial band of the bifurcate ligament laterally, and the spring (plantar calcaneonavicular) ligament inferiorly.

The spring (plantar calcaneonavicular) ligament (see Figs. 12–7 and 12–20) is a triangular sheet of ligamentous connective tissue arising from the sustentaculum tali of the calcaneus and inserting on the inferior navicular bone. The spring ligament is continuous medially with a portion of the deltoid ligament of the ankle and joins laterally with the medial band of the bifurcate ligament.[86] Recent evidence suggests the spring ligament is composed of three components: superomedial, medioplantar oblique, and inferoplantar longitudinal bands.[87–89] The spring ligament plays an important role in supporting the head of the talus and talonavicular joint, as well as functioning as one of the main static or passive stabilizers of the medial longitudinal arch.[86,90]

We already noted that the talonavicular facets and the anteriorly located talocalcaneal facets share a joint capsule. The large posterior facet of the subtalar joint is contained within its own capsule and is physically separated from the capsule containing the talonavicular joint by the tarsal canal and the ligaments within the canal. However, the talonavicular joint and the subtalar joint are linked in the weight-bearing foot. Weight-bearing dorsiflexion/plantarflexion and abduction/adduction of the talus on the calcaneus during subtalar supination/pronation necessarily involve simultaneous movement of the head of the talus on the relatively fixed navicular bone. In weight-bearing, therefore, the talonavicular joint and subtalar joint are both anatomically and functionally related.[85]

Concept Cornerstone **12-5**

Talar Linkages

Because of the anatomical and functional linkage of the talus to the structures below and anterior to it, the subtalar joint and talonavicular joint have been referred to by the compound term **talocalcaneonavicular joint.**[2] However, it might be argued that even this compound term is incomplete. The talus in weight-bearing also can be considered to act as a ball-bearing between three joints: (1) the tibiofibular mortise (the ankle joint) superiorly, (2) the calcaneus (the subtalar joint) inferiorly, and (3) the navicular bone (the talonavicular joint) anteriorly. In weight-bearing supination/pronation, the talus dorsiflexes and plantarflexes within the mortise, as well as on the calcaneus and navicular bone. Abduction/adduction of the talus during supination/pronation not only occurs on the calcaneus and the navicular bone but also affects the position of the mortise as the body of the talus laterally and medially rotates.

The ligaments of the talonavicular joint include, of course, the ligaments that help compose it: the spring and bifurcate ligaments. The talonavicular articulation is also supported by the **dorsal talonavicular ligament** and receives support from the ligaments of the subtalar joint—including the medial collateral and lateral collateral ligaments, the inferior extensor retinacular structures, and the cervical and interosseous talocalcaneal ligaments. Additional support is also received from the ligaments that reinforce the adjacent calcaneocuboid joint, which forms the remainder of the transverse tarsal joint and to which the talonavicular joint is linked functionally.

Calcaneocuboid Joint

The calcaneocuboid joint is formed proximally by the anterior calcaneus and distally by the posterior cuboid bone (see Fig. 12–19). The articular surfaces of both the calcaneus and the cuboid bone are complex, being reciprocally concave/convex both side to side and top to bottom. The reciprocal shape makes available motion at the calcaneocuboid joint more restricted than that of the ball-and-socket–shaped talonavicular joint. The calcaneocuboid joint, like the talonavicular joint, is linked in weight-bearing to the subtalar joint.[85] In weight-bearing subtalar supination/pronation, the inversion/eversion of the calcaneus on the talus causes the calcaneus to move simultaneously on the relatively fixed cuboid bone. As the calcaneus moves at the subtalar joint during weight-bearing activities, it must meet the conflicting intra-articular demands of the opposing saddle-shaped surfaces, which results in a twisting motion.

The calcaneocuboid articulation has its own capsule that is reinforced by several important ligaments. The capsule is reinforced laterally by the lateral band of the bifurcate ligament (also known as the **calcaneocuboid ligament**), dorsally by the **dorsal calcaneocuboid ligament,** and inferiorly by the **plantar calcaneocuboid (short plantar)** and the **long plantar ligaments** (see Fig. 12–8). The long plantar ligament is the most important of these ligaments, because the inferiorly located long plantar ligament spans the calcaneus and the cuboid bone and then continues on distally to the bases of the second, third, and fourth metatarsals. Therefore, the long plantar ligament provides support to the transverse tarsal joint as well as the lateral longitudinal arch of the foot.[91] The extrinsic muscles of the foot also provide important support for the transverse tarsal joint as they pass medial, lateral, and inferior to the joint.

Transverse Tarsal Joint Axes

Movements at the transverse tarsal joint are more difficult to study than movement at the ankle or subtalar joint because multiple segments and axes are involved. Although the talonavicular and calcaneocuboid joints have some independent movement, motion at one is generally accompanied by at least some motion of the other because of their functional, bony, and ligamentous connections. We continue to rely on the classic works of Elftman,[92] Manter,[58] and Hicks,[29] who proposed longitudinal and oblique axes around which the talus and calcaneus move on the relatively fixed naviculocuboid unit. The longitudinal axis is nearly horizontal, being inclined 15° upward from the transverse plane (Fig. 12–21A) and angled 9° medially from the sagittal plane[48] (Fig. 12–21B). Motion around this axis is triplanar, producing supination/pronation with components similar in coupling (but not magnitude) to those seen at the subtalar joint but now simultaneously including both the talus and calcaneus segments moving on the navicular and cuboid segments. Unlike the axis of the subtalar joint, the longitudinal axis of the transverse tarsal joint approaches a true A-P axis, and so the inversion/eversion components of the transverse tarsal movement predominate.

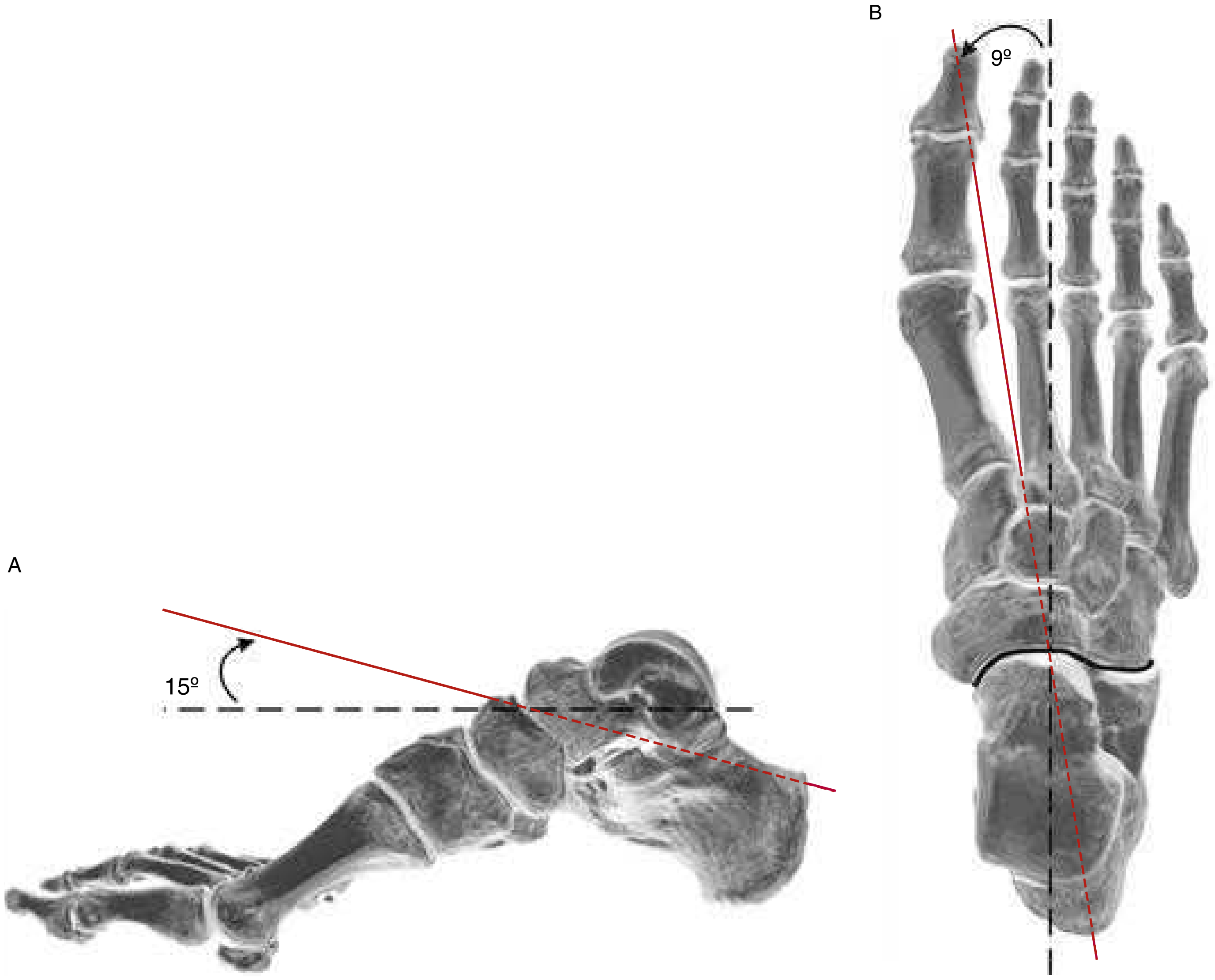

Figure 12–21 The longitudinal axis of the transverse tarsal joint is **(A)** inclined 15° superiorly from the transverse plane and **(B)** inclined 9° medially from the sagittal plane.

The oblique (transverse) axis of the transverse tarsal joint is positioned approximately 57° medial to the sagittal plane (Fig. 12–22A) and 52° superior to the transverse plane (Fig. 12–22B).[58] This triplanar axis also provides supination/pronation with coupled component movements of the talus and calcaneus segments moving together on the navicular and cuboid bones, but dorsiflexion/plantarflexion and abduction/adduction components predominate over inversion/eversion motions. Motions about the longitudinal and oblique axes are difficult to separate and to quantify. The longitudinal and oblique axes together provide a total range of supination/pronation of the talus and calcaneus that is about one third to one half of the range available at the subtalar joint.[29]

Transverse Tarsal Joint Function

The proposed longitudinal and oblique axes for the transverse tarsal joint indicate a function similar to that of the subtalar joint. In fact, as already noted, the subtalar and the transverse tarsal joints are linked mechanically so that any weight-bearing subtalar motion causes the talonavicular and calcaneocuboid joint to move simultaneously as well. As the subtalar joint supinates, its linkage to the transverse tarsal joint causes both the talonavicular joint and the calcaneocuboid joint to begin to supinate. When the subtalar joint is fully supinated and locked (bony surfaces are drawn together), the transverse tarsal joint is also carried into full supination, and its bony surfaces are similarly drawn together into a locked position. When the subtalar joint is pronated and loose packed, the transverse tarsal joint is also mobile and loose packed.

The transverse tarsal joint is the transitional link between the hindfoot and the forefoot, serving to (1) add to the supination/pronation range of the subtalar joint and (2) compensate the forefoot for hindfoot position. Compensation in this context refers to the ability of the forefoot to remain flat on the ground (relatively immobile) while the hindfoot (talus and calcaneus) pronates or supinates in response to the terrain or the rotations imposed by the leg. The first of

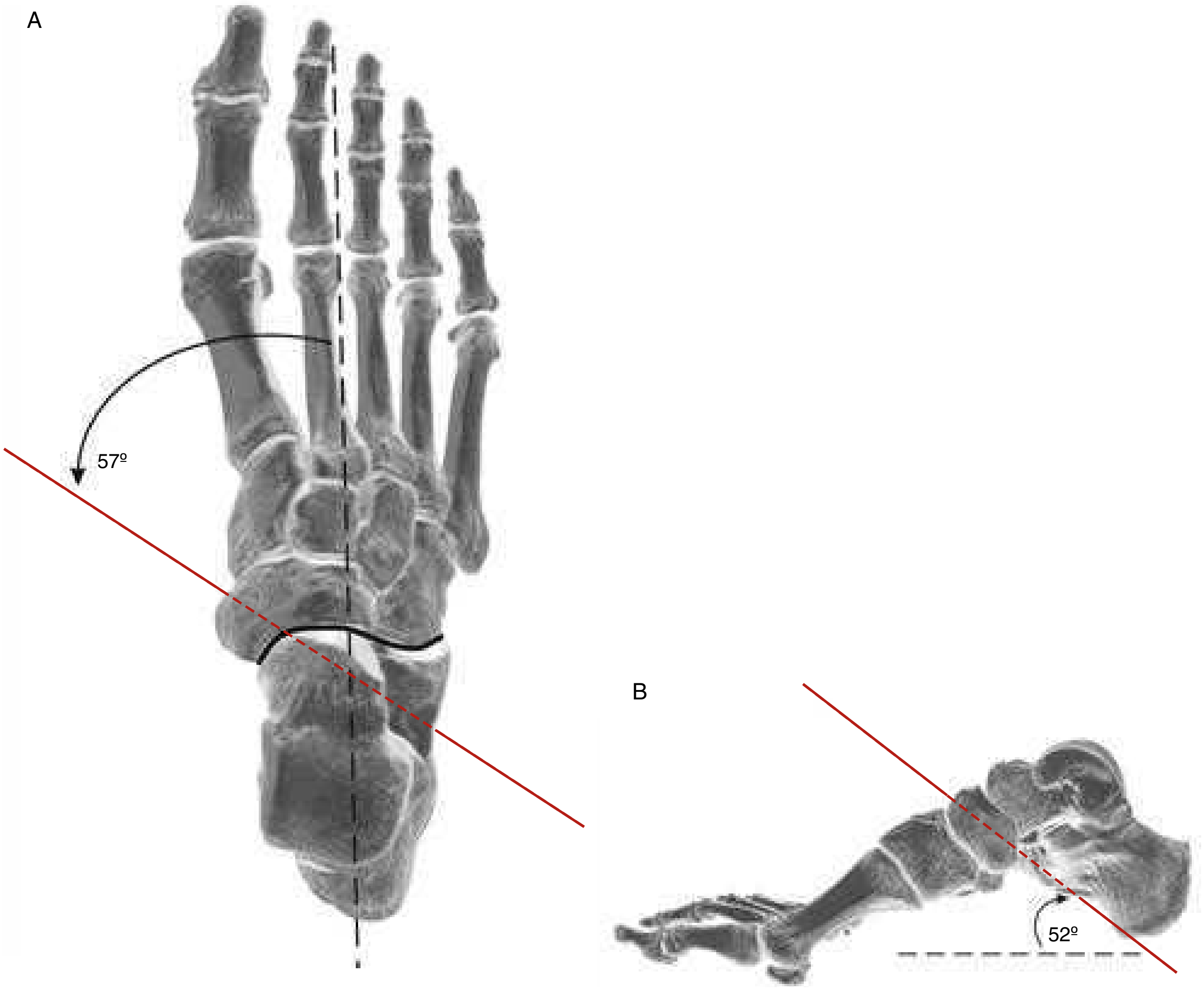

Figure 12–22 The oblique axis of the transverse tarsal joint is **(A)** inclined 57° from the sagittal plane and **(B)** inclined 52° superiorly from the transverse plane.

the transverse tarsal joint functions (adding range to supination/pronation) can occur either in the weight-bearing foot or in the non-weightbearing foot. The second function requires closer analysis.

Weight-Bearing Hindfoot Pronation and Transverse Tarsal Joint Motion

In the weight-bearing position, medial rotation of the tibia (as occurs, for example, if someone pivots on a fixed foot) imposes pronation on the subtalar joint. If the pronation force continued distally through the foot, the lateral border of the foot would tend to lift from the ground, diminishing the stability of the base of support, resulting in unequal weight-bearing, and imposing stress at multiple joints. This undesirable effect of weight-bearing subtalar joint pronation may be avoided if the forefoot remains flat on the ground. This can occur if the transverse tarsal joint is mobile and can effectively "absorb" the hindfoot pronation (allowing the hindfoot to move without passing the movement on to the forefoot). When the talus and calcaneus move on an essentially fixed naviculocuboid unit, there is a relative supination of the bony segments distal to the transverse tarsal joint, with the result that the forefoot remains relatively flat on the ground. The transverse tarsal joint maintains normal weight-bearing forces on the forefoot while allowing the hindfoot (subtalar joint) to absorb the rotation of the lower limb. Inman and Mann's[46] mechanical model (Fig. 12–23A) nicely represents how a medial rotary force imposed on the leg acts through the oblique axis of the "subtalar joint" and through the "transverse tarsal joint" to maintain the forefoot in a relatively fixed position. Note that the fixed "forefoot" has effectively moved in a direction opposite to that of the "hindfoot" segment.

Inman and Mann's[46] model indicates that when the weight-bearing hindfoot (subtalar joint) is pronated, the transverse tarsal joint will supinate (move in a direction opposite to the hindfoot). However, in reality the transverse tarsal joint is relatively free to move either into pronation or supination (depending on the demands of the terrain) because both the subtalar and the transverse tarsal joints are loose packed. In a bilateral standing position on level ground, both the subtalar joint and the transverse tarsal joints pronate slightly (Fig. 12–23B), presumably to allow the foot to absorb the body's weight. As a result of the pronation, there will be a slight medial rotary force on the leg. As a person moves into single-limb support and begins to walk, the subtalar joint will continue to pronate, whereas the transverse tarsal joint will move in the direction of supination approximately an equal amount to maintain proper weight-bearing in the forefoot. During walking on uneven terrain, as long as the hindfoot is in pronation, the forefoot can move either toward supination or pronation, depending on the demands of the terrain. If, for example, there is a rock under the medial forefoot during walking, the transverse tarsal joint may move into greater supination to maintain appropriate contact of the forefoot with the ground (Fig. 12–23C). If the supination range is not available at the transverse tarsal joint, the rock may also force the hindfoot into a supinated position (putting the lateral collateral ligament at risk for injury). With other surface demands, such as standing sideways on a steep hill, the uphill foot must pronate substantially to maintain contact with the ground. Therefore, pronation may be required at both the subtalar and the transverse tarsal joints. As long as the subtalar joint is in some degree of pronation, both the subtalar joint and the transverse tarsal joint are relatively mobile and free to make compensatory changes (within the limits of the joints' ROM) to maintain contact of the foot with the ground.

Weight-Bearing Hindfoot Supination and Transverse Tarsal Joint Motion

As shown in the mechanical model of Inman and Mann,[46] a lateral rotary force on the leg will create subtalar supination in the weight-bearing subtalar joint with a relative pronation of the transverse tarsal joint (opposite motion of the

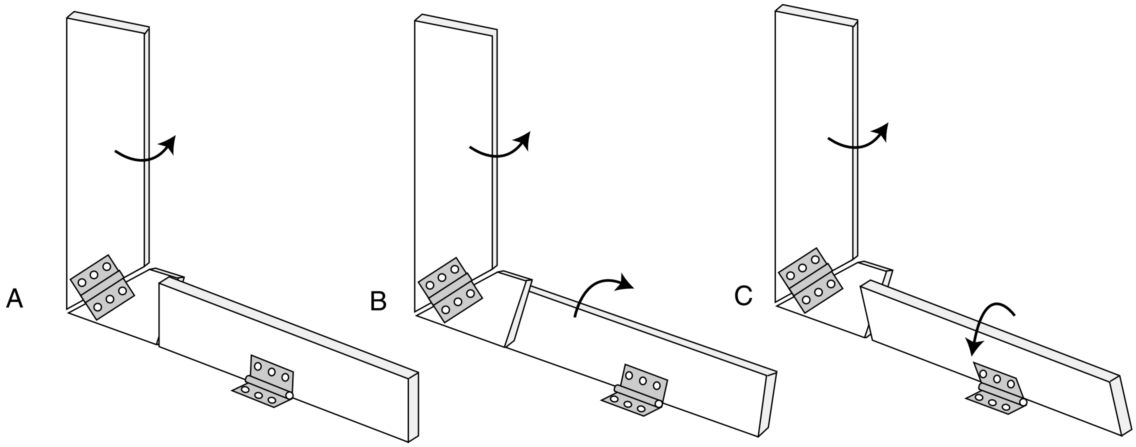

Figure 12–23 With pronation occurring at the subtalar joint through medial rotation of the leg, the transverse tarsal joint is free to **(A)** supinate slightly to maintain the relatively fixed position of the forefoot segment; **(B)** pronate slightly as occurs in normal standing; or **(C)** supinate substantially to maintain appropriate weight-bearing of the forefoot segment on uneven terrain. *(Adapted from Mann RA: Biomechanics of running. In Mann RA [ed]: Surgery of the Foot [ed. 5], p 15. St. Louis, MO, CV Mosby, 1986.)*

forefoot segment) to maintain appropriate weight-bearing on a level surface (Fig. 12–24A). Supination of the subtalar joint, however, can proceed only to a certain point before the transverse tarsal joint also begins to supinate. As bony and ligamentous structures of the subtalar joint draw the talus and calcaneus closer together (become increasingly close packed), the navicular and cuboid bones are also drawn toward the talus and calcaneus; that is, transverse tarsal joint mobility is increasingly limited as the subtalar joint moves toward full supination. With increasing supination of the subtalar joint (caused either by the terrain or by an increased lateral rotary force on the leg), the transverse tarsal joint cannot absorb the additional rotation but begins to move toward supination as well (Fig. 12–24B).

In full subtalar joint supination, such as when the tibia is maximally laterally rotated on the weight-bearing foot, supination locks not only the subtalar joint but also the transverse tarsal joint (Fig. 12–24C). The fully supinated subtalar joint and transverse tarsal joint will tend to shift the weight-bearing in the forefoot fully to the lateral border of the foot. Barring other compensatory mechanisms or when the demands of the terrain exceed the foot's ability to compensate, the entire medial border of the foot may lift and, unless the muscles on the lateral side of the foot and ankle are active, a supination sprain of the lateral ligaments may occur. When the locked subtalar and transverse tarsal joints are unable to absorb the rotation superimposed by the weight-bearing limb or by uneven ground, the forces must be dissipated at the ankle, and excessive stresses may result in injury to the ankle joint structures. The subtalar joint of a high-arched (pes cavus) foot tends to be set in a supinated position with limited pronation motion. This supinated position also limits the ability of the transverse tarsal joint to compensate. Therefore, a high-arched foot is relatively more rigid[93] and can be more susceptible to impact-type injuries, especially on the lateral side of the foot.[94]

CASE APPLICATION

Flat Feet

case 12–2

Mr. Benson, according to report and observation, has "flat feet" that have progressively worsening. Mr. Bensen is likely to have *adult-acquired flat foot,* which is defined as a progressive loss of dynamic and static medial longitudinal arch support.[95] In a flat foot (pes planus or pes valgus) deformity, the foot typically remains in a position of excessive pronation at the subtalar joint during weight-bearing. Tibialis posterior tendon dysfunction is usually associated with adult-acquired flat foot. For Mr. Benson, this could explain why he has pain at his medial malleolus with active plantarflexion and inversion. When the function of the tibialis posterior muscle is reduced, increased stress is placed on the other supporting structures of the medial longitudinal arch. This results in progressive flattening of the foot with decreased medial arch height, plantarflexion and medial rotation of the talus, hindfoot valgus, and forefoot abduction. The lateral malleolus can be positioned more anteriorly than normal, as a result of medial rotation of the lower extremity. There is also an increase in loading on the first metatarsal and increased stress on the ligaments supporting the joints of the medial arch.[96–99] The deformities associated with adult-acquired flat foot were found to affect gait; stride length, cadence, and walking speed were all diminished, while stance duration was prolonged.[100] A classification scheme and treatment guidelines for adult-acquired flat foot are available based on the severity of the deformity,[101] including whether the deformities are flexible or fixed. Mr. Benson's deformities were able to be passively corrected to neutral and are, therefore, defined as flexible. A number of risk factors for developing adult-acquired flat foot have been identified. Of these, obesity seems to be the most significant factor.[102] Orthotics and exercises can be used to effectively treat early stages when the deformities are flexible.[103] However, surgical intervention may be indicated when the "flat-feet" become rigid and the deformities cannot be passively corrected to neutral.[104]

TARSOMETATARSAL JOINTS

Tarsometatarsal Joint Structure

The **tarsometatarsal (TMT) joints** are plane synovial joints formed by the distal row of tarsal bones (posteriorly) and the bases of the metatarsals (Fig. 12–25). The first (medial) tarsometatarsal joint is composed of the articulation between the base of the first metatarsal and the medial cuneiform bone and has its own articular capsule. The second tarsometatarsal joint is composed of the articulation of the base of the second metatarsal with a mortise formed by the middle cuneiform bone and the sides of the medial and lateral cuneiform bones. This joint is set more posteriorly than the other tarsometatarsal joints; it is stronger and its motion is more restricted. The third tarsometatarsal joint, formed by the third metatarsal and the lateral cuneiform, shares a capsule with the second tarsometatarsal joint. The bases of the fourth and fifth metatarsals, with the distal surface of the cuboid bone, form the fourth and fifth tarsometatarsal joints. These two joints also share a common joint capsule. Small plane articulations exist between the bases of the metatarsals to permit motion of one metatarsal on the next. Numerous dorsal, plantar, and interosseous ligaments reinforce each tarsometatarsal joint. In addition, there is a **deep transverse metatarsal ligament** that spans the heads of the metatarsals on the plantar surface[105,106] and is similar to that found in the hand. Just as the deep transverse metacarpal ligament contributed to stability of the more proximally located carpometacarpal (CMC) joints, the deep transverse metatarsal ligament contributes to stability of the proximally located tarsometatarsal joints by preventing excessive motion and splaying of the metatarsal heads.[107]

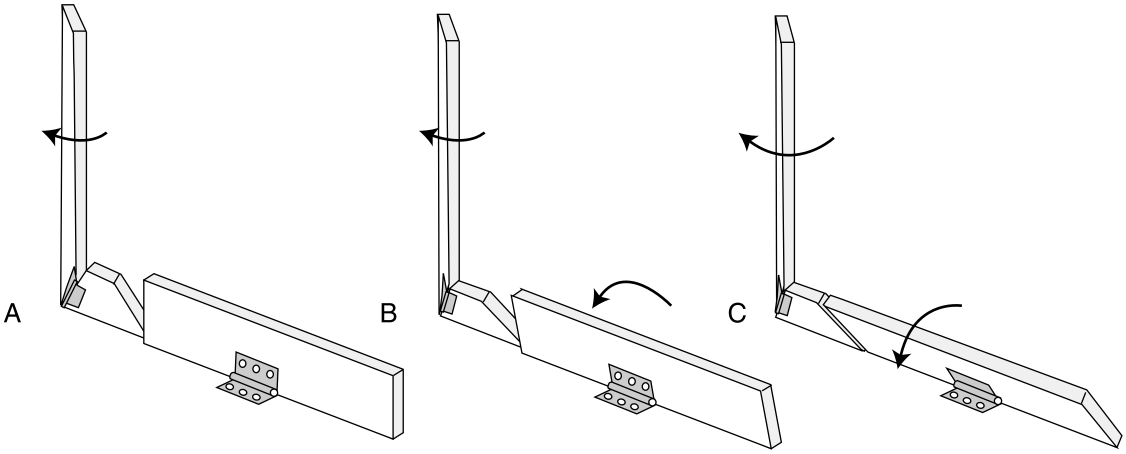

Figure 12–24 With supination occurring at the subtalar joint through lateral rotation of the leg, the transverse tarsal joint has limited ability to pronate to maintain the relatively fixed position of the forefoot segment **(A);** will begin to supinate with a greater range of subtalar supination and lateral rotation of the leg **(B);** or will fully supinate along with a fully supinated subtalar joint and maximal lateral rotation of the superimposed leg **(C)**.

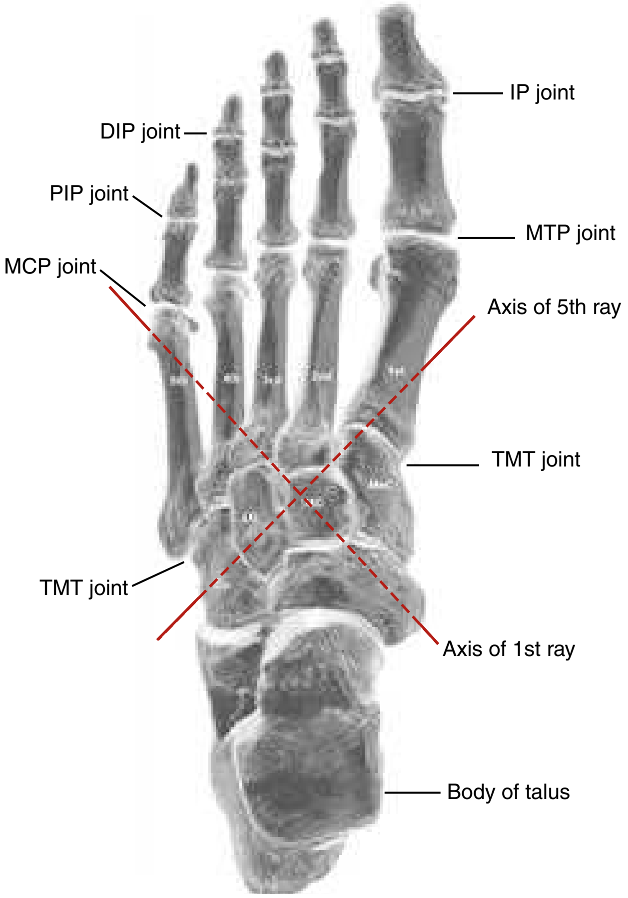

Figure 12–25 Tarsometatarsal (TMT), metatarsophalangeal (MTP), and interphalangeal (PIP/DIP) joints of the foot, showing the axes of the first and fifth tarsometatarsal joints. CU, cuboid; LC, lateral cuneiform; MC, middle cuneiform; MeC, medial cuneiform.

Axes

Each tarsometatarsal joint is considered to have a unique, although not fully independent, axis of motion. Hicks examined the axes for the five rays.[29] A **ray** is defined as a functional unit formed by a metatarsal and (for the first through third rays) its associated cuneiform bone. The cuneiform bones are included as parts of the movement units of the tarsometatarsal rays because of the small and relatively insignificant amount of motion occurring at the cuneonavicular joints. The cuneonavicular motion, therefore, becomes functionally part of the available tarsometatarsal motions. The fourth and fifth rays are formed by the metatarsal alone because these metatarsals both articulate with the cuboid bone.

According to Hicks, most motion at the tarsometatarsal joints occurs at the first and fifth rays.[29] The axes for the first and fifth rays are shown in Figure 12–25. Each axis is oblique and, therefore, triplanar. Of the tarsometatarsal joints, the first has the largest ROM. The axis of the first ray is inclined in such as way that dorsiflexion of the first ray also includes inversion and adduction, whereas plantarflexion is accompanied by eversion and abduction. The abduction/adduction components normally are minimal. Movements of the fifth ray around its axis are more restricted and occur with the opposite arrangement of components: Dorsiflexion is accompanied by eversion and abduction, and plantarflexion is accompanied by inversion and adduction.

The axis for the third ray nearly coincides with a coronal axis; the predominant motion, therefore, is dorsiflexion/plantarflexion. The axes for the second and fourth rays were not determined by Hicks[29] but were considered to be intermediate between the adjacent axes for the first and fifth rays, respectively. The second ray moves around an axis that is inclined toward, but is not as oblique as, the first axis. The fourth ray moves around an axis that is similar to, but not as steep as, the fifth axis. The second ray is considered to be the least mobile of the five.

Tarsometatarsal Joint Function

The motions of the tarsometatarsal joints are interdependent, as are the motions of the carpometacarpal joints in the hand. Like the carpometacarpal joints of the hand, the tarsometatarsal joints contribute to hollowing and flattening of the plantar surface of the foot. In contrast to the hand, however, the greatest relevance of tarsometatarsal joint motions is found during weight-bearing. In weight-bearing, the tarsometatarsal joints function primarily to augment the function of the transverse tarsal joint. That is, the tarsometatarsal joints attempt to regulate position of the metatarsals and phalanges (the forefoot) in relation to the weight-bearing surface.[108] As long as transverse tarsal joint motion is adequate to compensate for the hindfoot position, substantive tarsometatarsal joint motion is not required. However, when the hindfoot position is at an end point in its available ROM or the transverse tarsal joint is inadequate to provide full compensation, the tarsometatarsal joints may rotate to provide further adjustment of forefoot position.[65]

Supination Twist

When the hindfoot pronates substantially in weight-bearing, the transverse tarsal joint generally will supinate to some degree to counterrotate the forefoot and keep the plantar aspect of the foot in contact with the ground. If the range of transverse tarsal supination is not sufficient to meet the demands of the pronating hindfoot (or if the transverse tarsal joint is prevented from effectively serving this function), the medial forefoot will press into the ground, and the lateral forefoot will tend to lift. The first and second rays will be pushed into dorsiflexion by the ground reaction force, and the muscles controlling the fourth and fifth rays will plantarflex those tarsometatarsal joints in an attempt to maintain contact with the ground. Both dorsiflexion of the first and second rays and plantarflexion of the fourth and fifth rays include the component motion of inversion of the ray. Consequently, the entire forefoot (each ray and its associated toe) undergoes an inversion rotation around a hypothetical axis at the second ray. This rotation is referred to as **supination twist** of the tarsometatarsal joints.[29]

As an example of supination twist of the forefoot, Figure 12–26 shows the response of the segments of the foot to a strong pronation torque across the subtalar joint that may be caused either by a strong medial rotary force from the leg or by inadequate support of the arch. The calcaneus everts, and the talus plantarflexes and adducts. With sufficient pronation, the navicular bone is pushed downward with the motion of the head of the talus, limiting the ability of the transverse tarsal joint to supinate adequately. The first and second rays will dorsiflex and invert, whereas the fourth and fifth rays will plantarflex and invert, which results in a supination (inversion) twist of the tarsometatarsal joints to attempt to adequately adjust the forefoot to the ground.

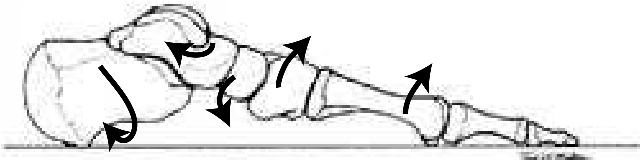

Figure 12–26 Extreme pronation at the subtalar joint is accompanied by adduction and plantarflexion of the head of the talus, eversion of the calcaneus, and (in some instances) pronation at the transverse tarsal joint as a result of the navicular bone's being forced down by the talus. If the forefoot is to remain on the ground, the tarsometatarsal joints must undergo a counteracting supination twist.

Because the five tarsometatarsal joints have some independence, the configuration of the forefoot in a supination twist can vary according to the weight-bearing needs of the foot and the terrain.

Pronation Twist

When both the hindfoot and the transverse tarsal joints are locked in supination, the adjustment of forefoot position must be left entirely to the tarsometatarsal joints. With hindfoot supination, the forefoot tends to lift off the ground on its medial side and press into the ground on its lateral side. The muscles controlling the first and second rays will plantarflex those rays in order to maintain contact with the ground, whereas the fourth and fifth rays are forced into dorsiflexion by the ground reaction force. Because eversion accompanies both plantarflexion of the first and second rays and dorsiflexion of the fourth and fifth rays, the forefoot as a whole undergoes a **pronation twist.**[29]

Pronation twist, like supination twist, can vary in configuration. Although the pronation twist may provide adequate counterrotation for moderate hindfoot supination, it may be inadequate to maintain forefoot stability in extreme supination. In Figure 12–27, subtalar supination results in calcaneal inversion, with dorsiflexion and abduction of the talus. The transverse tarsal joint will have little if any ability to pronate, inasmuch as the navicular and cuboid bones are carried along with the hindfoot motion. The first and second rays will plantarflex and evert, whereas the fourth and fifth rays will dorsiflex and evert. These motions result in a pronation (eversion) twist of the tarsometatarsal joints in an attempt to adjust the forefoot adequately.

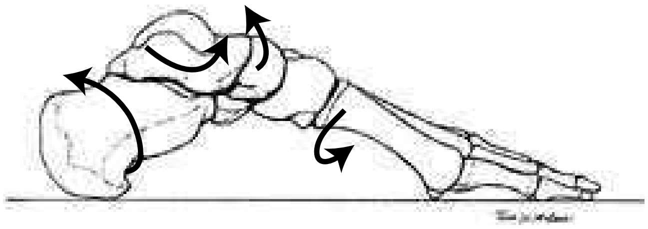

Figure 12–27 Extreme supination at the subtalar joint is accompanied by abduction and dorsiflexion of the head of the talus, inversion of the calcaneus, and forced supination of the transverse tarsal joint. If the forefoot is to remain on the ground, the tarsometatarsal joints must undergo a counteracting pronation twist.

Pronation twist and supination twist of the tarsometatarsal joints occur only when the transverse tarsal joint function is inadequate, that is, when the transverse tarsal joint is unable to counterrotate or when the transverse tarsal joint range is insufficient to compensate fully for hindfoot position.

Continuing Exploration 12-3:

Forefoot Varus

Excessive pronation of the hindfoot has been associated with a forefoot varus deformity. With hindfoot pronation in weight-bearing, the forefoot must supinate at the tarsometatarsal joints to maintain appropriate weight distribution across the metatarsal heads. If adaptive tissue changes result in a sustained tarsometatarsal supination twist, the deformity is known as a **forefoot varus** (effectively the same as a fixed supination twist). In chronically pronated feet, it would be wise to look for adaptive changes in the forefoot. Forefoot varus can be identified by assessing the position of the forefoot in the frontal plane *in relation to* the subtalar neutral position of the hindfoot (typically in a non-weightbearing position). A forefoot varus deformity is considered present if the forefoot is inverted in relation to the frontal plane when the subtalar joint (calcaneus) is manually held in its neutral position (Fig. 12–28). Identifying forefoot varus can be challenging, given the problems with ascertaining subtalar neutral position in the pronated foot,[69,75,109] and may best be identified visually as simply present or not present.

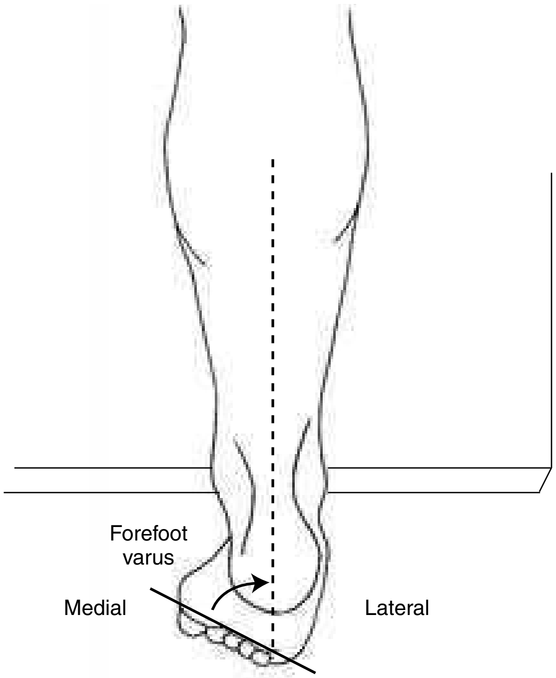

Figure 12–28 A forefoot varus deformity is identified by manually placing the non-weightbearing calcaneus in subtalar neutral position (manipulating hand not shown) and determining whether the forefoot is deviated in the frontal plane from a line bisecting the calcaneus.

METATARSOPHALANGEAL JOINTS

The five **metatarsophalangeal (MTP) joints** are condyloid synovial joints with two degrees of freedom: extension/flexion (or dorsiflexion/plantarflexion) and abduction/adduction. Although both degrees of freedom might be useful to the metatarsophalangeal joints in the rare instances when the foot participates in grasping-like activities, flexion and extension are the predominant functional movements at these joints. During the late stance phase of walking, toe extension at the metatarsophalangeal joints permits the foot to pass over the toes, whereas the metatarsal heads and toes help balance the superimposed body weight through activity of the intrinsic and extrinsic toe flexor muscles.

Metatarsophalangeal Joint Structure

The metatarsophalangeal joints are formed proximally by the convex heads of the metatarsals and distally by the concave bases of the proximal phalanges (see Fig. 12–25).

Continuing Exploration 12-4:

Metatarsal Length

The lengths of the five metatarsals may vary among individuals and are classified into three types. **Index plus** is characterized by the first metatarsal being longer than the second with the other remaining three progressively decreasing in length. **Index plus minus** is characterized by the first and second metatarsals being the same length, with the other remaining three progressively decreasing in length. **Index minus** or **Morton's foot** is classified by the second metatarsal being longest followed progressively by the first, third, fourth, and fifth.[110] A Morton's foot has been hypothesized to cause an increased force through the second metatarsal, predisposing it to injury such as a stress fracture. The relationship between metatarsal length and injury pattern, however, is unclear as the literature has not supported this hypothesis in athletes.[110,111]

The structure of the metatarsophalangeal joints is analogous to the structure of the metacarpophalangeal (MCP) joints of the hands, with a few exceptions. Unlike the metacarpophalangeal joints, the range of metatarsophalangeal extension exceeds the range of metatarsophalangeal flexion. All metatarsal heads bear weight in stance. Consequently, the articular cartilage must remain clear of the weight-bearing surface on the plantar aspect of the metatarsal head. This structural requirement restricts the available range of metatarsophalangeal flexion. Also in contrast to the hand, there is no opposition available at the first tarsometatarsal joint; the first toe **(hallux)** moves exclusively in the same planes as the other four digits.

The first metatarsophalangeal joint has two sesamoid bones associated with it that are located on the plantar aspect of the first metatarsal head (Fig. 12–29). These are

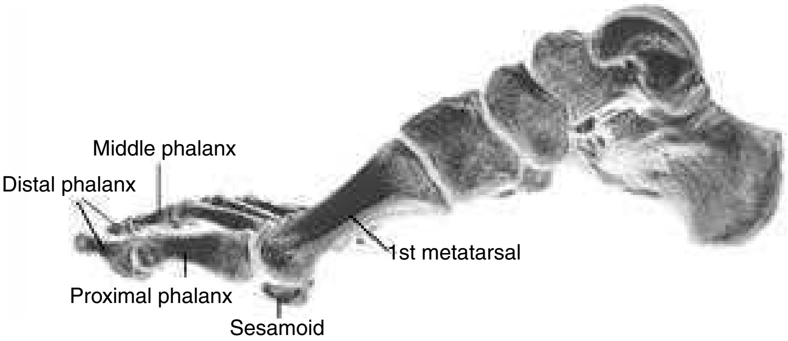

Figure 12–29 In this radiograph, the two sesamoid bones can easily be seen sitting on the head of the first metatarsal.

analogous to the sesamoid bones on the volar surface of the metacarpophalangeal joint. In the neutral position of the first metatarsophalangeal joint, the sesamoid bones lie in two grooves, separated by the intersesamoid ridge on the metatarsal head.[112,113] The ligaments associated with the sesamoid bones form a triangular mass that stabilize the sesamoid bones within their grooves.[112,114] The sesamoid bones serve as anatomic pulleys for the **flexor hallucis brevis** muscle and protect the tendon of the flexor hallucis longus muscle from weight-bearing trauma. The flexor hallucis longus is protected as it passes through a tunnel formed by the sesamoid bones and the intersesamoidal ligament that connects the sesamoid bones across their plantar surfaces.[112,115] Unlike the sesamoid bones of the thumb, the sesamoid bones of the first metatarsal absorb weight-bearing stress, along with the relatively large quadrilaterally shaped first metatarsal head.[114–116] With toe extension greater than 10°, the sesamoid bones no longer lie in their grooves and may become unstable. Chronic lateral instability of the sesamoid bones may lead to metatarsophalangeal deformity.[116]

Continuing Exploration 12-5:

Sesamoiditis

The sesamoid bones and their supporting structures can become traumatized with excessive loading, leading to acute fracture, stress fracture, osteonecrosis, and/or chondromalacia. Inflammatory conditions of the sesamoids, which result in pain under the first metatarsal head, are generally known as **sesamoiditis.**[117] Active individuals who participate in prolonged running, jumping, or gymnastics may be more susceptible, particularly if deformities such as pes cavus exist.[117–119] Conservative treatment focuses on protecting the region from continued stresses by modifying activity, shoe wear accommodation, and orthotic devices. If a fracture is identified or if the symptoms do not improve, surgical excision of part or all of the sesamoid bones may be necessary.[115,118,120]

Stability of the metatarsophalangeal joints is provided by a joint capsule, **plantar plates, collateral ligaments,** and the deep transverse metatarsal ligament. The plantar plates are structurally similar to the volar plates in the hand. These fibrocartilaginous structures in the four lesser toes are each connected to the base of the proximal phalange distally and blend with the joint capsule proximally. The plates of the four lesser toes are interconnected by the deep transverse metatarsal ligament and **plantar aponeurosis.**[121,122] The collateral ligaments of the metatarsophalangeal joints, like those at the metacarpophalangeal joint, have two components: a phalangeal portion that parallels the metatarsal and phalange, and an accessory component that runs obliquely from the metatarsal head to the plantar plate.[121–123] The plantar plates protect the weight-bearing surface of the metatarsal heads and, with the collateral ligaments, contribute to stability of the metatarsophalangeal joints. Each plantar plate, therefore serves as a central stabilizing structure with a fibrocartilaginous composition that allows it to withstand compressive loads.[121,122] At the first metatarsophalangeal joint, the sesamoid bones and thick plantar capsule are in place of the plantar plates found at the other toes.[114]

Metatarsophalangeal Joint Function

The metatarsophalangeal joints have two degrees of freedom, but flexion/extension motion is much greater than abduction/adduction motion, and extension exceeds flexion. Although metatarsophalangeal motions can occur in weight-bearing or non-weightbearing, the metatarsophalangeal joints serve primarily to allow the weight-bearing foot to rotate over the toes through metatarsophalangeal extension (known as the **metatarsal break**) when rising on the toes or during walking.

Metatarsophalangeal Extension and the Metatarsal Break

The metatarsal break derives its name from the hinge or “break” that occurs at the metatarsophalangeal joints as the heel rises and the metatarsal heads and toes remain weight-bearing. The metatarsal break occurs as metatarsophalangeal extension around a single oblique axis that lies through the second to fifth metatarsal heads (Fig. 12–30). The inclination of the axis is produced by the diminishing lengths of the metatarsals from the second through the fifth toes and varies among individuals. The angle of the axis

Figure 12–30 The metatarsal break occurs around an oblique axis that passes through the heads of the four lesser toes, at an angle to the long axis of the foot that varies widely among individuals from 54° to 73°.

around which the metatarsal break occurs may range from 54° to 73° with respect to the long axis of the foot.[124] The range of metatarsophalangeal extension will vary, depending on the amount of dorsiflexion/plantarflexion motion at the tarsometatarsal joints, the age of the individual,[125] and whether the motion is assessed in weight-bearing or nonweightbearing.[126,127] One study reported that the first metatarsophalangeal joint extension averaged 81° in a younger population (mean age of 21 years) compared to 56° in an older population (mean age of 80 years).[126] Gait studies have reported averages ranging between 36° and 65° of metatarsophalangeal extension during walking.[126–128] Limited extension ROM at the first metatarsophalangeal joint will interfere with the metatarsal break and is known as **hallux rigidus.**

For the heel to rise during weight-bearing, there must be an active contraction of ankle plantarflexor musculature. Most of the plantarflexion muscles also contribute to supination of the subtalar and transverse tarsal joints. The plantarflexion musculature normally cannot lift the heel completely unless the joints of the hindfoot and midfoot are supinated and locked so that the foot can become a rigid lever from the calcaneus through the metatarsals. This rigid lever will then rotate ("break") around the metatarsophalangeal axis. As metatarsophalangeal joint extension occurs, the metatarsal heads glide in a posterior and plantar direction on the plantar plates and the phalanges that are stabilized by the supporting surface. The metatarsal heads and toes become the base of support, and the body's line of gravity must move within this new and more limited base to remain stable. The obliquity of the axis for the metatarsal break allows weight to be distributed across the metatarsal heads and toes more evenly than would occur if the axis were truly coronal. If the body weight passed forward through the foot and the metatarsal break occurred around a true coronal metatarsophalangeal axis, an excessive amount of weight would be placed on the first and second metatarsal heads. These two toes would also require a disproportionately large extension range. The obliquity of the axis of the metatarsal break shifts the weight laterally, minimizing the large load on the first two digits.

Continuing Exploration 12-6:

Hammer Toe Deformity

Excessive extension at the metatarsophalangeal joint in a resting position is called a **hammer toe** deformity. In one group of 20 healthy subjects averaging 56 (± 11) years of age without foot problems, the resting metatarsophalangeal joint angle was 11° (± 5°) of extension for the first metatarsophalangeal joint and 23° to 42° for the second through fifth metatarsophalangeal joints.[129] This metatarsophalangeal joint angle generally is higher in patients with diabetes and peripheral neuropathy (Fig. 12–31), possibly because of weakness in the intrinsic foot muscles that stabilize the metatarsophalangeal joint.[130] Presumably because the toes cannot participate properly in weight-bearing, hammer toe deformity has been associated with increased pressures under the metatarsal heads that can result in pain or in skin breakdown in vulnerable individuals.[129]

Metatarsophalangeal Flexion, Abduction, and Adduction

Flexion ROM at the metatarsophalangeal joints can occur to a limited degree from neutral position but has relatively little purpose in the weight-bearing foot other than when the supporting terrain drops away distal to the metatarsal heads. Most metatarsophalangeal flexion occurs as a return to neutral position from extension. However, toe flexor musculature is quite important and should be distinguished from the functionally less relevant metatarsophalangeal flexion ROM. Abduction and adduction of the metatarsophalangeal joint appear to be helpful in absorbing some of the force that would be imposed on the toes by the metatarsals as they move in a pronation or supination twist. The first toe normally is adducted on the first metatarsal about 15° to 19°.[116,129] An increase in this normal valgus angulation of the first metatarsophalangeal joint is referred to as **hallux valgus** and may be associated

Figure 12–31 Radiographic image of a foot from a healthy subject **(A)** and a foot from a subject with diabetes and peripheral neuropathy **(B).** The diabetic foot shows a hammer toe deformity (hyperextension at the metatarsophalangeal joint and flexion at the interphalangeal joint).

with a varus angulation of the first metatarsal at the tarsometatarsal joint, known as **metatarsus varus** (Fig. 12–32).

CASE APPLICATION

Hallux Valgus case 12–3

Mr. Benson reported that he has a **bunion** that bothers him occasionally with periodic pain around his great toe. By inspection, he has an evident hallux valgus deformity. Hallux valgus can result in or may be associated with a reduction in first metatarsophalangeal joint ROM, gradual lateral subluxation of the toe flexor tendons crossing the first metatarsophalangeal joint, reduced weight-bearing on the great toe, and increased weight-bearing on the first metatarsal head.[131] These structural changes can lead to pain and difficulty during walking. People with a pronated foot may push off during walking with a greater than normal adductor moment on the great toe that pushes the toe into a valgus (metatarsophalangeal joint adducted) position. Localized swelling and pain at the medial or dorsal aspect of the first metatarsophalangeal joint may be related to an inflamed medial bursa and is commonly called a bunion. The etiology of hallux valgus deformities is likely multifactorial and related to repetitive forces applied to the first metatarsophalangeal joint.[131] The person with a flat foot and excessive pronation, like Mr. Benson, may have instability and excessive mobility of the first ray, contributing to hallux valgus deformity.[132] Although familial disposition, narrow shoes, pes planus, and hypermobile first tarsometatarsal joint have been proposed as related factors, there is insufficient evidence to prove or disprove their association to hallux valgus.[131]

Continuing Exploration 12-7:

Varus/Valgus Terminology

Varus and *valgus* are consistently used to refer to a decrease or increase, respectively, in the medial angle. However, the reference line and the location of the angles keep changing. In the hindfoot, we already noted that *varus/valgus* either can be synonyms for *inversion/eversion* of the calcaneus (see Fig. 12–16) or may refer to fixed positioning of the subtalar joint in excessive supination or pronation (see Fig. 12–3). In both cases, the reference line was the posterior leg and a line bisecting the calcaneus. Now we refer to *metatarsus varus* as a deformity identified as a decrease in the medial angle between the long axis of the metatarsal and the long axis of the foot (or adduction of the metatarsal); *hallux valgus* is a deformity identified by an increase in the medial angle between the long axes of the metatarsal and proximal phalanx (adduction of the proximal phalanx) (see Fig. 12–32). We also defined *forefoot varus* as a fixed supination twist of the forefoot in relation to a neutral subtalar joint. In this instance, the medial angle is formed by a line through the metatarsal heads and a line bisecting the "neutral" subtalar joint (see Fig. 12–28). Unfortunately, here the terminology may create problems.

To someone who most often assesses deviations in an otherwise normal foot (as we have with Mr. Benson), a *forefoot varus* refers to a supinated forefoot. To someone who most often assesses paralytic or congenital deformities in the foot, the term *forefoot varus* may indicate a metatarsus varus of all the metatarsals and toes. The plane of the deviation differs in these two usages. Until the terminology is standardized, the context of the presentation will have to give the reader clues as to which usage is being applied.

INTERPHALANGEAL JOINTS

The **interphalangeal (IP) joints** of the toes are synovial hinge joints with one degree of freedom: flexion/extension. The great toe has only one interphalangeal joint connecting two phalanges, whereas the four lesser toes have two interphalangeal joints (proximal and distal interphalangeal joints) connecting three phalanges (see Fig. 12–29). Each phalanx is virtually identical in structure to its counterpart in the hand, although substantially shorter in length. Consequently, the reader is referred to Chapter 9 for details on interphalangeal joint structure of the thumb and fingers to understand the structure of the interphalangeal joints of the toes. The toes function to smooth the weight shift to the opposite foot in gait and help maintain stability by pressing against the ground in standing. Injuries to the interphalangeal joints are relatively uncommon but may be involved in dislocations and fractures.

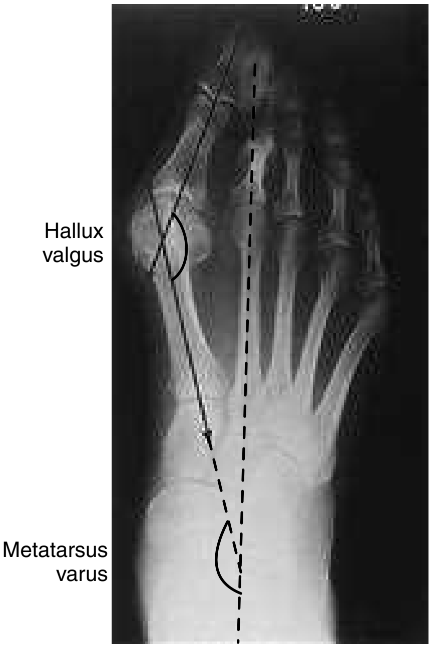

Figure 12–32 A radiograph showing both a hallux valgus at the first metatarsophalangeal joint, and a metatarsus varus at the first tarsometatarsal joint. Excessive bony growth at the head of the first metatarsal is due to abnormal pressures from the malalignment.

PLANTAR ARCHES

Although we have examined the function of the joints of the foot individually and discussed the effect of each joint on contiguous joints, combined function is best investigated by looking at the behavior of the archlike structures of the foot. The foot typically is characterized as having three arches: medial and lateral longitudinal arches and a transverse arch. The medial longitudinal arch is the largest and most commonly discussed. Although we may think of and refer to the three arches as if they were separate, the arches are fully integrated with one another (being more analogous to a segmented continuous vault) to enhance the dynamic function of the foot. The arches are usually not present at birth but evolve with the progression of weight-bearing. Flattened longitudinal arches are seen in younger children less 5 years of age. When gait parameters become similar to that of an adult (around age 5), the majority of children will develop an normal arch.[133] One study found the prevalence of flat foot in those 3 years of age was 54% but decreased to 25% by the age of 6.[134] In addition to age, the prevalence of flat foot was found to be related weight and gender.[134,135]

Structure of the Arches

The longitudinal arches are anchored posteriorly at the calcaneus and anteriorly at the metatarsal heads. The longitudinal arch is continuous both laterally (Fig. 12–33A) and medially (Fig. 12–33B) through the foot. However, because the medial arch is higher than its lower lateral counterpart, the medial side usually is the side of reference. The talus rests at the top of the vault of the foot and is considered to be the "keystone" of the arch. All weight transferred from the body to the heel or the forefoot must pass through the talus.

The transverse arch, like the longitudinal arch, is a continuous structure. It is easiest to visualize in the midfoot at the level of the tarsometatarsal joints. At the anterior tarsals (Fig. 12–34A), the middle cuneiform bone forms the keystone of the arch. The transverse arch still can be visualized at the distal metatarsals but with less curvature (Fig. 12–34B). The second metatarsal, recessed into its mortise, is at the apex of this part of the arch. The transverse arch is completely reduced at the level of the metatarsal heads, with all metatarsal heads parallel to the weight-bearing surface.

The shape and arrangement of the bones are partially responsible for stability of the plantar arches. As illustrated in Figure 12–34A, the wedge-shaped midtarsal bones provide

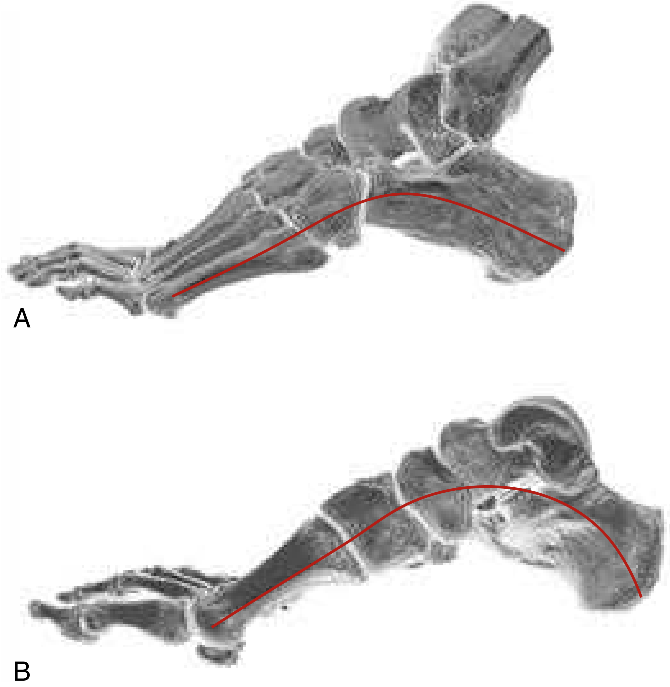

Figure 12–33 The longitudinal arch viewed from **(A)** the lateral side of the foot is low in comparison with the view from **(B)** the medial side of the foot.

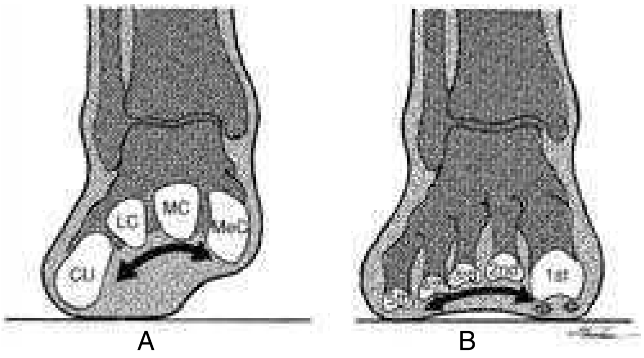

Figure 12–34 The transverse arch. **A.** At the level of the anterior tarsals. **B.** At the level of the middle of the metatarsals. CU, cuboid; LC, lateral cuneiform; MC, middle cuneiform; MeC, medial cuneiform.

an inherent stability to the transverse arch. The inclination of the calcaneus and first metatarsal contribute to stability of the medial longitudinal arch, particularly in standing (see Fig. 12–33B). Although the structure of the tarsal bones provides a certain inherent stability to the arches, the arches would collapse without additional support from ligaments and muscles.

Because the three arches can be thought of as a segmented vault or one continuous set of interdependent linkages, support at one point in the system contributes to support throughout the system. The plantar calcaneonavicular (spring) ligament, the interosseous talocalcaneal ligament, deltoid ligament, and the plantar aponeurosis have been credited with providing key passive support to the medial longitudinal arch (Fig. 12–35).[136,137] The "articular" (superomedial portion) portion of the spring ligament directly supports the head of the talus[85] and is the most important static stabilizer.[98,136] The laterally located long and short plantar ligaments support the lateral longitudinal arch and may also contribute, although indirectly, in supporting the medial longitudinal arch.

Function of the Arches

Although the archlike structures of the foot are similar to the palmar arches of the hand, the purpose served by each of these systems is quite different. The arches of the hand are structured predominantly to facilitate grasping and manipulation but must also assist the hand in occasional weight-bearing functions. In contrast, the foot in most individuals is rarely called on to perform any grasping activities. The plantar arches are adapted uniquely to serve two contrasting mobility and stability weight-bearing functions.[138] First, the foot must accept weight during early stance phase and adapt to various surface shapes. To accomplish this weight-bearing mobility function, the plantar arches must be flexible enough to allow the foot to (1) dampen the impact of weight-bearing forces, (2) dampen superimposed rotational motions, and (3) adapt to changes in the supporting surface. To accomplish weight-bearing stability functions, the arches must allow (1) distribution of weight through the foot for proper weight-bearing and (2) conversion of the flexible foot to a rigid lever. The mobility-stability functions of the arches of the weight-bearing foot may be examined by looking at the role of the plantar aponeurosis and by looking at the distribution of weight through the foot in different activities.

Plantar Aponeurosis

The plantar aponeurosis **(plantar fascia)** is a dense fascia that runs nearly the entire length of the foot. It begins posteriorly on the medial tubercle of the calcaneus and continues anteriorly to attach by digitations to the plantar

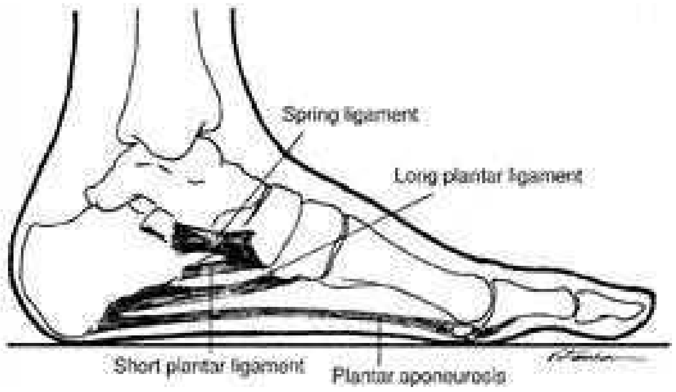

Figure 12–35 The medial longitudinal arch with its associated ligamentous support, including the plantar aponeurosis. The more laterally located short plantar ligament would not ordinarily be seen in a medial view but is shown as if projected "through" the foot.

plates and then, via the plates, to the proximal phalanx of each toe[107,123,139] (see Fig. 12–35). From the beginning to the end of the stance phase of gait, tension on the plantar aponeurosis increases, with maximum tension averaging 96% of body weight as force is transmitted from the Achilles tendon to the forefoot.[140] In vivo experiments using radiographic fluoroscopy have shown that the plantar aponeurosis deforms, or stretches, 9% to 12% during the stance phase of gait.[141] For this reason, the function of the aponeurosis in supporting the arches has been compared to the function of a tie-rod on a truss in classic descriptions.[142] The truss and the tie-rod form a triangle (Fig. 12–36); the two struts of the truss form the sides of the triangle and the tie-rod is the bottom. The talus and calcaneus form the posterior strut, and the remaining tarsal and metatarsals form the anterior strut. The plantar aponeurosis, as the tie-rod, holds together the anterior and posterior struts when the body weight is loaded on the triangle. This structural design is efficient for the weight-bearing foot because the struts (bones) are subjected to compression forces, whereas the tie-rod (aponeurosis) is subjected to tension forces. Bending moments to the bone that can cause injury are minimized. It was found that when the foot is loaded, considerable tension develops in the plantar aponeurosis while strain in the spring and long plantar ligament is less.[143] The fibrocartilaginous plantar plates of the metatarsophalangeal joints are organized not only to resist compressive forces from weight-bearing on the metatarsal heads but also to resist tensile stresses presumably applied through the tensed plantar aponeurosis that connects to them.[123] Therefore, each biological structure is positioned to maximize its optimal loading pattern and minimize the opportunity for injury.

The plantar aponeurosis and its role in arch support are linked to the relationship between the plantar aponeurosis and the metatarsophalangeal joint. When the toes are extended at the metatarsophalangeal joints (regardless of whether the motion is active or passive, weight-bearing or non-weightbearing), the plantar aponeurosis is pulled increasingly tight as the proximal phalanges glide dorsally in relation to the metatarsals or as the metatarsal heads glide in a relatively plantar direction on the fixed toes. The metatarsal heads act as pulleys around which the plantar aponeurosis is pulled and tightened (Fig. 12–37). As the plantar aponeurosis wraps around the metatarsal heads and is tensed with metatarsophalangeal extension, the heel and metatarsophalangeal joint are drawn toward each other as the plantar portion of the tie-rod is shortened, raising the arch and contributing to supination of the foot. This phenomenon, which has been referred to as the “windlass mechanism,”[144] allows the plantar aponeurosis to increase its role in supporting the arches as the heel rises and the foot rotates around the metatarsophalangeal joints in weight-bearing (during the metatarsal break).

The tension in the plantar aponeurosis (the tie-rod) in the loaded foot is evident if active or passive metatarsophalangeal extension is attempted while the triangle is flattened (that is, when the subtalar and transverse tarsal joint are pronated). The range of metatarsophalangeal extension will be limited because further lengthening of the aponeurosis is limited. Alternatively, raising the height of the triangle by acting directly on the struts can unload the tie-rod. For example, when the tibia is subjected to a lateral rotary force, the hindfoot will supinate, the posterior strut will become more oblique, the height of the medial longitudinal arch will increase, and the plantar aponeurosis (the tie-rod) will be relatively unloaded. The reduction in tension in the plantar aponeurosis will allow an increase in the range of metatarsophalangeal extension.

Through the pulley affect of the metatarsophalangeal joints on the plantar aponeurosis, the plantar aponeurosis acts interdependently with the joints of the hindfoot to contribute to increasing the longitudinal arch (supination of the foot) as the heel rises during the metatarsal break, thus contributing to converting the foot to a rigid lever for effective push-off. The tightened plantar aponeurosis also increases the passive flexor force at the metatarsophalangeal joints, preventing *excessive* toe extension that might stress the metatarsophalangeal joint or allow the line of gravity to move anterior to the toes. Finally, the passive flexor force of the tensed plantar aponeurosis also assists the active toe flexor musculature in pressing the toes into the ground to support the body weight on its limited base of support.

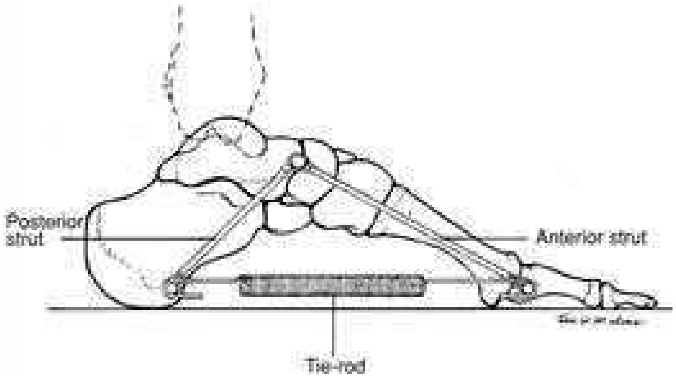

Figure 12–36 The foot can be considered to function as a truss and tie-rod, with the calcaneus and talus serving as the posterior strut, the remainder of the tarsals and the metatarsals serving as the anterior strut, and the plantar aponeurosis serving as a tensed tie-rod. Weighting the foot will compress the struts and create additional tension in the tie-rod.

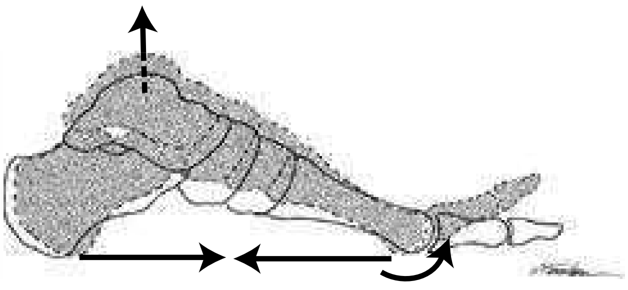

Figure 12–37 Elevation of the arch with toe extension occurs as the plantar aponeurosis winds around the metatarsal heads and draws the two ends of the aponeurosis toward each other.

Concept Cornerstone **12-6**

Summary of Tie-Rod and Truss Relations

- Tension in the plantar aponeurosis (the tie-rod) caused by metatarsophalangeal joint extension can draw the hindfoot and forefoot (the struts) together to raise the longitudinal arch (supinate the foot).
- Supination of the weight-bearing foot through lateral rotation of the leg or by applying a varus force to the calcaneus will decrease the angle between the struts (raise the apex of the triangle) and release tension in the tie-rod (plantar aponeurosis).
- Flattening of the triangle (pronation of the foot) in weight-bearing will increase tension in the plantar aponeurosis (the tie-rod) and limit metatarsophalangeal joint extension.

CASE APPLICATION

Plantar Fasciitis

case 12–4

Mr. Benson reported heel pain that was greatest in the morning when he first got out of bed. The pain decreased after several steps but increased again with prolonged walking. These signs are classic indicators of **plantar fasciitis** (inflammation of the plantar aponeurosis).[145] The pain typically is localized at the medial calcaneal tubercle where the plantar aponeurosis inserts. Pain can spread distally down the fascia toward the toes in more chronic conditions. Toe extension may also increase pain because extending the toes places additional tension on the fascia (aponeurosis). The risk factors for plantar fasciitis have been identified and include limited ankle dorsiflexion range of motion and a high body mass index.[145] Given Mr. Benson's overweight status, a discussion that includes weight loss would be appropriate. Arch taping or orthotic devices (either prefabricated or custom foot orthotics) can be used to support the medial longitudinal arch and decrease stress off the plantar aponeurosis (plantar fascia).[145]

Weight Distribution

Because the foot is a flexible rather than fixed arch, the distribution of body weight through the foot depends on many factors, including the shape of the arch and the location of the line of gravity at any given moment. Distribution of superimposed body weight begins with the talus, because the body of the talus receives all the weight that passes down through the leg. In bilateral stance, each talus receives 50% of the body weight. In unilateral stance, the weight-bearing talus receives 100% of the superimposed body weight. In standing, at least 50% of the weight received by the talus passes through the large posterior subtalar articulation to the calcaneus, and 50% or less passes anteriorly through the talonavicular and calcaneocuboid joints to the forefoot. The pattern of weight distribution through the foot can be seen by looking at the trabeculae in the bones of the foot (Fig. 12–38). Because of the more medial location of the talar head, about twice as much weight passes through the talonavicular joint as through the calcaneocuboid joint. The somewhat lesser roles of the more laterally located long and short plantar ligaments in supporting the longitudinal arch may be attributable to the reduced weight-bearing compression through the calcaneocuboid joint in comparison with the more medially located talonavicular joint.[146]

In static standing, the distribution of weight-bearing can be variable and largely depend on foot type, whether pes planus, pes cavus, or without impairment. Also, structural deformities will potentially affect the weight-bearing pattern. Generally, during quiet standing the rearfoot and forefoot bear a majority of the force. Birtane and Tuna[147] found a larger load under the rearfoot, with peak pressures there almost twice as much as under the forefoot.

Plantar pressures are much greater during walking than during standing, with the highest pressures typically under the metatarsal heads and occurring during the push-off phase of walking (~80% of stance), when only the forefoot is in contact with the ground and the forefoot is pushing to accelerate the body forward.[148] Plantar pressure under the foot during walking is related to a complex interaction of multiple factors.[149] These factors include physical characteristics, such as age, body weight, and height,[149–151] as well as foot structure, such as arch height, hallux length, and joint motion.[149,152,153] Gait style and muscle action are also factors that will influence plantar pressures.[149] As walking progresses to running, plantar force and peak pressures increase. Maximum force increased from 1.11 to 2.14 times body weight when walking was compared to running.[154] Excessive plantar pressures can contribute to pain and injury in otherwise healthy people or contribute to skin breakdown in patients with

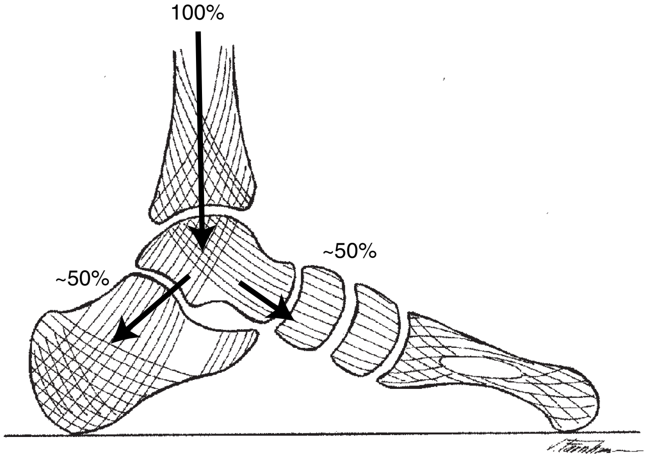

Figure 12–38 Trabeculae of the bones on the medial aspect of the foot illustrating transfer of 100% of the force through the talus, with 50% passing posteriorly to the calcaneus and 50% anteriorly to the forefoot through the talonavicular and calcaneocuboid joints.

diabetes and peripheral neuropathy. Structural and functional factors such as hammer toe deformity, soft tissue thickness, hallux valgus, foot type, and walking speed have been shown to be important predictors of forefoot plantar pressures during walking in people without impairments and in people with diabetes.[129]

Muscular Contribution to the Arches

The role of muscle activity in maintaining and supporting the arches of the foot has not been well defined. Extrinsic muscles that include the tibialis posterior,[136,155] tibialis anterior,[156] peroneus longus,[136,156] flexor digitorum longus,[136] and **peroneus tertius**[156] have all been found to play a role in dynamic arch support. The tibialis posterior has been shown to have the most consistent function in medial longitudinal arch support.[136] The plantar intrinsic muscles, which include the **abductor hallucis,** flexor hallucis brevis, **flexor digitorum brevis, abductor digiti minimi,** and **dorsal interossei,** have been shown to be active during walking.[157,158] Historically, muscle activity was thought to contribute little to arch support in the normal *static* foot. However recent evidence suggests that these intrinsic muscles do provide substantial support to the arch in static standing.[159,160]

MUSCLES OF THE ANKLE AND FOOT

As discussed throughout this chapter, muscle activity is critical for the dynamic stability and integration of movement at multiple joints of the foot. There are no muscles in the ankle or foot that cross and act on one joint in isolation; all these muscles act on at least two joints or joint complexes. Muscle function is dependent on the muscle's structure and, of course, where the muscle passes in relation to each joint axis the muscle crosses. The position of the ankle/foot muscles with respect to the talocrural joint axis and subtalar joint axis is represented in Figure 12–39. As illustrated in this figure, all muscles that pass anterior to the talocrural (ankle) joint will cause dorsiflexion torques or moments, whereas those that pass posterior to the axis will cause plantarflexion moments. Muscles that pass medial to the subtalar axis will create supination moments at the subtalar joint, whereas those that pass lateral to the subtalar axis will create pronation moments. A muscle, of course, can (and will) create both an ankle joint and a subtalar joint moment simultaneously. For example, the tibialis anterior muscle passes anterior to the talocrural axis *and* medial to the subtalar joint axis, and so it will create a simultaneous dorsiflexion moment at the ankle and a supination moment at the subtalar joint. An understanding of the position of the muscle with respect to the axis is critical for understanding its function.

A brief overview of muscle function is presented in this chapter with a more comprehensive description of the muscles described in Chapters 13 and 14. Extrinsic ankle/foot muscles are those that arise proximal to the ankle and insert onto the foot. Intrinsic foot muscles arise from within the foot (do not cross the ankle) and insert on the foot. Extrinsic muscles will be divided further into the three compartments of the lower leg: the posterior, lateral, and anterior compartments.

Extrinsic Musculature

Posterior Compartment Muscles

The posterior compartment muscles all pass posterior to the talocrural joint axis and, therefore, are all plantarflexors. The muscles in the posterior compartment are the gastrocnemius, soleus, tibialis posterior, flexor digitorum longus, and flexor hallucis longus muscles. The gastrocnemius muscle arises from two heads of origin on the condyles of the femur and inserts via the Achilles tendon into the most posterior aspect of the calcaneus. The soleus muscle is deep to the gastrocnemius, originating on the tibia and fibula and inserting with the gastrocnemius into the posterior calcaneus via the Achilles tendon. The two heads of the gastrocnemius and soleus muscles together are known as the **triceps surae** and are the strongest plantarflexors of the ankle. The large volume of the triceps surae is strongly associated with its ability to generate torque ($r^2 = 0.69$).[161] The Achilles tendon inserts perpendicularly on the calcaneus relatively far from the ankle joint axis (see Fig. 12–39). This efficient attachment provides a large moment arm to generate plantarflexion torque. The Achilles tendon has been referred to as a supinator; however, recent evidence suggests that the function of the triceps surae in the frontal plane may be variable and dependent on supination or pronation of the subtalar joint.[162,163] When the subtalar joint was pronated, the triceps surae produced an supination moment and when the subtalar joint was supinated, it produced a pronation moment.[162] Because the foot is pronated near midstance, activity of the gastrocnemius and soleus on the weight-bearing foot helps to supinate and lock the foot into a rigid lever both through direct supination of the subtalar joint and through indirect supination of the transverse tarsal joint. Continued plantarflexion force will raise the heel and cause elevation of the arch (potentially assisted by the increased tension in the plantar aponeurosis as the metatarsophalangeal joints extend). Elevation of the arch by the triceps surae when the heel is lifted off the ground is observable in most people when they actively plantarflex the weight-bearing foot (Fig. 12–40).

The soleus and the gastrocnemius together eccentrically control dorsiflexion of the ankle while also supinating the subtalar joint after the foot is loaded in stance. These muscles provide supination torque that contributes to making the foot a rigid lever for push-off and continue to provide plantarflexion torque throughout heel rise and plantarflexion of the ankle as the ground reaction force moves to the metatarsal heads and toes.

Continuing Exploration 12-8:

Shortening of the Gastrocnemius and Soleus Muscles

Because the gastrocnemius and soleus muscles pass behind the ankle joint, a limitation in the length of the muscles results in limited dorsiflexion ROM. Furthermore, the gastrocnemius also passes behind the knee joint, and so shortness in the gastrocnemius may further limit dorsiflexion

Continued

ROM when the knee is extended. Mr. Benson had limited dorsiflexion ROM as a result of a short gastrocnemius muscle, as evidenced by the reported pulling sensation behind his knee when he sat and simultaneously extended his knee and dorsiflexed his ankle. Tight hamstring muscles also may contribute to this type of pulling sensation behind the knee and could be distinguished from a short gastrocnemius muscle by extending the hip (which relieves tension on the hamstrings but not the gastrocnemius muscle). Limited dorsiflexion ROM as a result of a short triceps surae muscle group is thought to contribute to excessive pronation at the subtalar joint and is associated with midfoot and forefoot pain.[44]

The other ankle plantarflexion muscles are the **plantaris,** the tibialis posterior, the flexor hallucis longus, the flexor digitorum longus, the peroneus longus, and the peroneus brevis muscles. Although each of these muscles passes posterior to the ankle axis, the moment arm for plantarflexion for these muscles is so small that they provide only 5% of the total plantarflexor force at the ankle.[164] The plantaris muscle is so small that its function can essentially be disregarded.

The tendon of tibialis posterior muscle passes just behind the medial malleolus, medial to the subtalar joint (see Fig. 12–39), to insert into the navicular bone and plantar medial arch. The tibialis posterior muscle is the largest extrinsic foot muscle after the triceps surae and has a relatively large moment arm for supination of both the subtalar joint

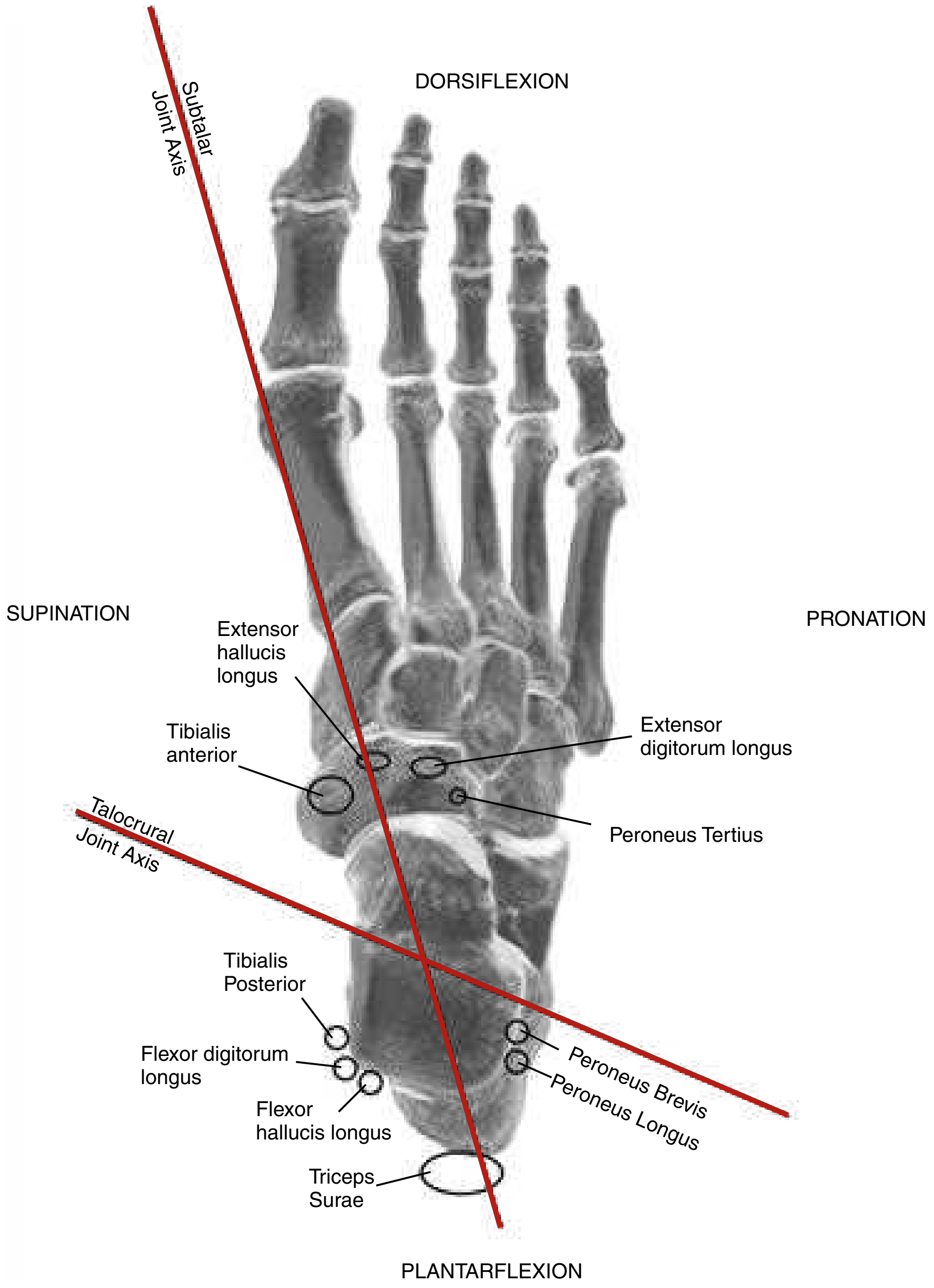

Figure 12–39 Location of muscle insertions in relation to ankle (talocrural) and subtalar joint axes. Muscles that insert anterior to the ankle joint axis will cause dorsiflexion torques at the ankle joint, whereas those that insert posterior to the axis will cause plantarflexion torques. Muscles that insert medial to subtalar joint axis will cause supination torques, whereas those that insert laterally will cause pronation torques.

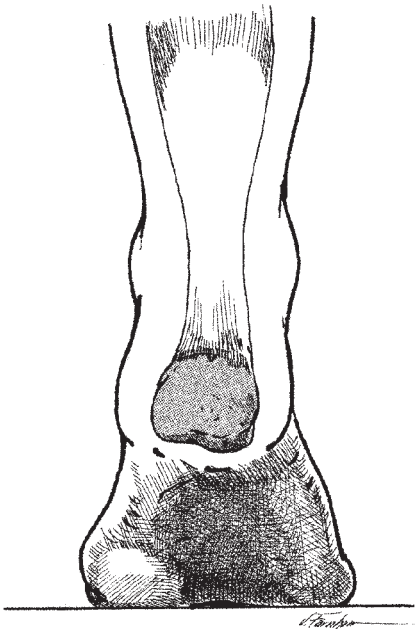

Figure 12–40 Activity of the triceps surae muscles on the fixed foot will cause ankle plantarflexion, subtalar supination, and elevation of the longitudinal arch.

and transverse tarsal joint.[162] The tibialis posterior muscle is an important dynamic contributor to arch support and has a significant role in controlling and reversing pronation of the foot that occurs during gait.[136,155,162] When the foot is being loaded early in the stance phase of walking, the tibialis posterior muscle contracts eccentrically to control subtalar and transverse tarsal pronation. Tibialis posterior muscle activity continues to work concentrically as the foot moves toward supination and plantarflexion. Because of its insertion along the plantar medial longitudinal arch, tibialis posterior dysfunction is a key problem associated with acquired pes planus, or flat foot.[136,155,162]

The flexor hallucis longus and the flexor digitorum longus muscles pass posterior to the tibialis posterior muscles and the medial malleolus, spanning the medial longitudinal arch and helping support the arch during gait. Because the tendons pass medial to the subtalar joint, the extrinsic toe flexors also assist in subtalar joint supination. These muscles attach to the distal phalanges of each digit and, through their actions, cause the toes to flex. The flexor digitorum longus tendon courses around the medial malleolus before splitting and passing to the distal phalanx of each of the four lesser toes. The line of pull of the tendon is oblique and, without assistance, would cause the toes simultaneously to flex and deviate toward the medial aspect of the foot. The **quadratus plantae** muscle is an intrinsic muscle arising from either side of the inferior calcaneus that inserts into the lateral border and plantar surface of the flexor digitorum longus tendons. The quadratus plantae muscle and the long toe flexors together form a concurrent force system with a resultant line of pull that flexes the four lesser toes with minimal deviation.

Although there is relatively little need for the toes to actually go into flexion, the toe flexors play an important role in balance when the line of gravity moves toward the metatarsal heads and toes. The toe flexors actively reinforce the passive role of the plantar aponeurosis during gait by eccentrically controlling the metatarsophalangeal extension (metatarsal break) at the end of stance phase, preventing the line of gravity from passing too far forward in the foot. In more static activities, the toes effectively lengthen the base of support for postural sway and during activities such as leaning forward to reach or pick up objects as long as the toe flexor muscles are strong enough to resist metatarsophalangeal extension and press firmly into the ground.

Flexion of the interphalangeal joint of the hallux by the flexor hallucis longus muscles produces a press of the toe against the ground (Fig. 12–41A). Flexion of the distal and proximal interphalangeal joints of the four lesser toes by the flexor digitorum longus causes clawing (metatarsophalangeal extension with interphalangeal flexion) similar to what occurs in the fingers when the proximal phalanx is not stabilized by intrinsic musculature (Fig. 12–41B). As is true in the hand, activity of the interossei muscles can stabilize the metatarsophalangeal joint and prevent metatarsophalangeal hyperextension. Pathologies (such as peripheral neuropathy) that cause weakness of the interossei muscles can contribute to destabilization of the metatarsophalangeal joint, hammer toe deformity (hyperextension at the metatarsophalangeal joint), and excessive stresses under the metatarsal heads.[130] These excessive stresses can contribute to pain under the metatarsal heads (i.e., **metatarsalgia**) or skin breakdown in persons who lack protective sensation (i.e., those with peripheral neuropathy).

Lateral Compartment Muscles

The peroneus longus and brevis muscles pass lateral to the subtalar joint and, because of their significant moment arms, are the primary pronators of the subtalar joint.[162] Their tendons pass posterior but close to the ankle axis and thus are weak plantar flexors. The tendon of the peroneus longus muscle passes around the lateral malleolus, under the cuboid bone, and across the transverse arch and inserts into the medial cuneiform bone and base of the first metatarsal (Fig. 12–42). Muscle contraction during late stance phase of

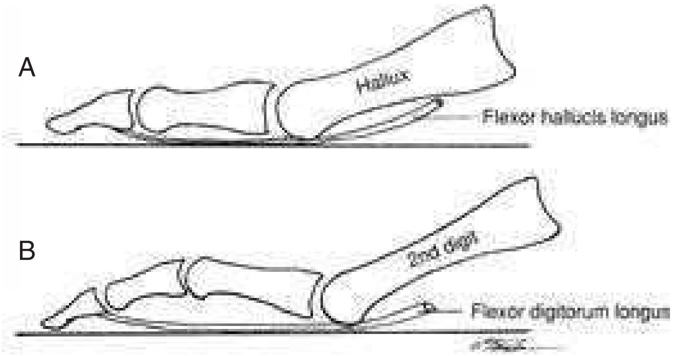

Figure 12–41 **A.** Action of the flexor hallucis longus causes the distal phalanx of the hallux to press against the ground. **B.** Activity of the flexor digitorum longus causes the four lesser toes to grip the ground.

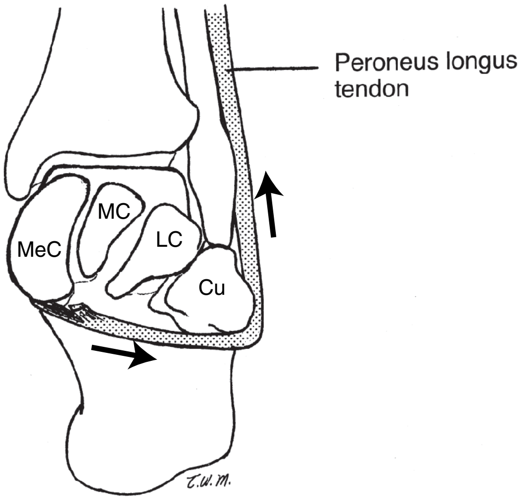

Figure 12–42 The tendon of the peroneus longus passes transversely beneath the foot to insert into the base of the first metatarsal. An active contraction of the muscle can support the transverse arch and the first ray of the foot.

gait facilitates transfer of weight from the lateral to the medial side of the foot and stabilizes the first ray as the ground reaction force attempts to dorsiflex it.[102] The peroneus longus, therefore, actively facilitates pronation twist of the tarsometatarsal joints while the hindfoot moves into increased supination. Because of its path across the arches, the peroneus longus tendon is credited with support of the transverse and lateral longitudinal arches as it causes pronation after heel contact, and stabilizes the forefoot after heel rise.[165]

The stability of each of the peroneal tendons at the lateral malleolus depends on the integrity of the superior and **inferior peroneal retinacula** located just superior and inferior to the ankle joint, respectively (see Fig. 12–9). Sprains of the lateral ankle structures may affect the peroneal retinacula that contribute to lateral ankle and subtalar support. Laxity of the superior retinaculum can lead to acute or chronic subluxation of the peroneal tendons causing lateral ankle pain and possibly a sense of instability.[166]

Anterior Compartment Muscles

The muscles of the anterior compartment of the leg are the tibialis anterior, the extensor hallucis longus, the extensor digitorum longus, and the peroneus tertius muscles. All muscles in the anterior compartment of the lower leg pass under the extensor retinaculum (see Fig. 12–9) and insert well anterior to the talocrural joint axis (see Fig. 12–39); these muscles are strong ankle dorsiflexors. Besides being a strong dorsiflexor muscle at the ankle joint, the tibialis anterior muscle passes medial to the subtalar axis and is a key supinator of the subtalar and transverse tarsal joints. The tibialis anterior was found to control rearfoot plantarflexion from heel contact to 10% stance, and pronation between 10% stance and footflat.[165] The tendon of the extensor hallucis longus muscle inserts near the subtalar joint axis and is, at best, a weak supinator of the foot. However, the extensor hallucis longus muscle is active in gait when the heel first contacts the ground to control the strong plantarflexion moment at the ankle created by the ground reaction force. Both the tibialis anterior and extensor hallucis longus muscles are active in dorsiflexing the ankle as the foot leaves the ground and in holding the foot up against the plantarflexion torque of gravity when the foot is off the ground. The extensor hallucis longus muscle also prevents the toes from dragging by extending (or preventing flexion) of the metatarsophalangeal joints of the hallux.

The tendons of the extensor digitorum longus and the peroneus tertius muscles pass beneath the extensor retinaculum and insert anterior to the ankle joint axis and lateral to the subtalar joint axis; consequently, these muscles are dorsiflexors of the ankle and pronators of the hindfoot. The extensor digitorum longus muscle also extends the metatarsophalangeal joints of the lesser toes, working with the extensor hallucis longus muscle to hold the toes up when the foot is off the ground. The structure and function of the extensor digitorum longus muscle at the metatarsophalangeal and interphalangeal joints are identical to those of the extensor digitorum communis of the hand.

The extrinsic musculature producing supination of the foot is stronger than that producing pronation. This phenomenon is likely to be attributable to the fact that the line of gravity in weight-bearing most often falls medial to the subtalar joint, creating a strong pronation torque that must be controlled.[167] Similarly, the plantarflexors are stronger than the dorsiflexors because the line of gravity in weight-bearing is most often anterior to the ankle joint axis.

Intrinsic Musculature

The most important functions of the intrinsic muscles of the foot are their roles as (1) stabilizers of the toes and (2) dynamic supporters of the transverse and longitudinal arches during gait. The intrinsic muscles of the hallux attach either directly or indirectly to the sesamoid bones and contribute to the stabilization of these weight-bearing bones.[114] The extensor mechanism of the toes is similar to that of the fingers. The extensor digitorum longus and brevis muscles are metatarsophalangeal extensors. Activity in the **lumbricals** and the dorsal and plantar interossei muscles flex metatarsophalangeal joints while producing interphalangeal extension. This helps to stabilize the metatarsophalangeal joints during walking to allow the toes to remain weight-bearing and reduce loading on the metatarsal heads. The metatarsophalangeal flexors act together to resist toe extension during the stance phase of locomotion, while the abductors affect the medial-lateral pressure by positioning the forefoot.[168] Furthermore, many of the intrinsic muscles arise on the posterior strut (calcaneus) and insert on the anterior strut (metatarsals) of the longitudinal arch, thereby serving to actively augment the tie-rod function of the plantar aponeurosis.[158] Periodic contraction during standing and consistent contraction during the stance phase of walking dynamically help relieve stress on the passive connective tissue structures supporting the longitudinal arch.

CASE APPLICATION

Achy, Flat Feet

case 12–5

Mr. Benson has flat feet, and he complained that his feet often ache at the end of the day. Although there can be several reasons for this type of generalized foot pain, a primary reason may be that the intrinsic muscles have to work harder and longer to stabilize the arches of the flat foot in comparison with a normal or high-arched foot. The high-arched foot in particular receives substantial passive support from bony alignment and ligaments, whereas the flat foot may rely more on the active contraction of the intrinsic muscles, which results in overuse, fatigue, and an "achy feeling" at the end of the day that could lead to an inflammatory response over time.

The specific function of the intrinsic muscles of the foot can be understood and appreciated by comparing each foot muscle with its corresponding hand muscle. Although most people are not able to use the muscles of the foot with the ability of those in the hand, the potential for similar function is limited only by the unopposable hallux and the length of the digits. Table 12–2 summarizes the specific functions of the intrinsic muscles of the foot.

DEVIATIONS FROM NORMAL STRUCTURE AND FUNCTION

The complex interdependency of the foot and ankle joints makes it almost impossible to have dysfunction or abnormality in only one joint or structure. Once present, deviations from neutral positions will affect both proximal and distal joints. The large number of congenital and acquired ankle/foot problems cannot each be described, although several have already been referenced in the chapter. The key is the "domino effect" that an ankle or foot problem has on the joints proximal and distal to the problem.

Example 12-1

Supinated Foot (Pes Cavus)

Pes cavus or **cavovarus** is described as a high arched foot, both in the weight-bearing and non-weightbearing positions. Reports have noted approximately 60% of individuals have an arch height considered normal, 20% have a low arch (pes planus), and 20% have a high arch (pes cavus).[169,170] In pes cavus, the high medial longitudinal arch is associated with a calcaneus that is noticeably inverted. This inverted calcaneus allows the medial heel pad to be observed when the patient stands with the foot straight ahead and has been referred to as the **"peek-a-boo" sign.**[171] The subtalar and transverse tarsal joints are excessively supinated, which prohibits these joints from participating in shock absorption or in adapting to uneven terrain. Hindfoot supination often is associated with a lateral rotation stress on the leg and lateral weight shift. Recurrent ankle sprains, peroneal tendon pathology, metatarsalgia, and fifth metatarsal stress fractures have been attributed to this lateral weight shift.[172] Although the pes cavus deformity can be the result of Charcot-Marie-Tooth disease, it can also be idiopathic in nature.

Soft tissue and muscle imbalances are thought to be the driving forces behind the pes cavus deformity. Weakness of the intrinsic musculature (lumbricals and interossei) can contribute to cavus deformity. Because the intrinsic muscles cause metatarsophalangeal flexion and interphalangeal extension, weakness of the intrinsic muscles allows the extrinsic flexors and extensors to have a dominant action. The long flexors cause flexion at the proximal interphalangeal joint and distal interphalangeal joint, while the long extensors cause hyperextension at the metatarsophalangeal joint. Prolonged unopposed flexion at the proximal interphalangeal and distal interphalangeal joints will result in a retrograde buckling of the metatarsophalangeal joint and further flexion of the metatarsal bones. Ultimately, this deformity can lead to contractures, increased pressure under the metatarsal head, and the pain associated with metatarsalgia.[173]

Imbalance in the agonist/antagonist relationship between two groups of muscles, (1) the anterior tibialis/peroneal longus and (2) the peroneus brevis/posterior tibialis, can also contribute to the deformities associated with pes cavus. Weakness of the anterior tibialis will give a mechanical advantage to the peroneus longus, resulting in a plantarflexion force to the first metatarsal. Plantarflexion of the first ray causes a forefoot valgus and a compensatory hindfoot varus deformity. Weakness of the peroneus brevis will give a mechanical advantage to the posterior tibialis, resulting in hindfoot varus. The compensation to hindfoot varus is a forefoot valgus deformity.

These deformities initially will be flexible during the early stages of the condition but, over time, become progressively become more rigid.[173]

CASE APPLICATION

Case Summary

case 12–6

We have established the following regarding Mr. Benson: (1) the medially rotated position of his lower extremity may be the result of a pronated foot; (2) he is likely to have flexible adult-acquired flat foot with involvement of the posterior tibialis; (3) his hallux valgus or bunion has caused localized pain at the medial/dorsal aspect of his first metatarsophalangeal joint; (4) he has classic signs and symptoms of plantar fasciitis (inflammation of the plantar aponeurosis); (5) the reported pulling sensation behind his

Continued

knee is likely a result of a short gastrocnemius muscle; and (6) his generalized plantar foot pain may be a result of intrinsic muscle fatigue.

In Mr. Benson's case, his overweight status is likely placing excessive stress on his ankle and foot complex. This includes his posterior tibialis muscle and tendon, plantar fascia, and intrinsic musculature. The excessive stress may also be contributing to his first metatarsophalangeal joint pain. His walking program has added an additional level of stress to already susceptible structures, forcing tissues past their threshold for injury.[1] As the posterior tibialis muscle becomes less effective, a progressive flattening of the foot with plantarflexion and medial rotation, hindfoot valgus, increased loading on the first metatarsal, and medial rotation of the lower extremity occur. As the medial longitudinal arch flattens, there is an increased stress on the passive support structures of the arch. The plantar calcaneonavicular (spring) ligament, the interosseous talocalcaneal ligament, deltoid ligament, and the plantar aponeurosis can all become involved as they provide passive support to the medial longitudinal arch. This case exemplifies how biomechanics and pathology can potentially be interrelated.

An important component of Mr. Benson's intervention needs to include a discussion and plan for weight loss. It has been documented that for every 1 pound of weight loss, there was a fourfold reduction in weight-bearing force.[174] Although this research was done at the knee, it would make sense that a similar reduction in force would benefit the ankle and foot complex. Orthotics to help address the excessive pronation and support the medial longitudinal arch would also be included in the intervention plan. Orthotics could potentially decrease stress on the posterior tibialis, passive support structures for the medial longitudinal arch, first metatarsophalangeal joint, and intrinsic muscles while helping to correct the medially rotated position of the lower extremity. Eccentric plantarflexion and inversion exercises should be included.[103] Given that Mr. Benson has a flexible flat foot (his deformity could be passively corrected to a neutral position), he is a good candidate for a conservative program directed to posterior tibialis tendon dysfunction.[103] A stretching program that includes dorsiflexion with the knee extended for his gastrocnemius should also be done. Night splints to hold the ankle and foot in neutral position may be added to allow the plantar aponeurosis to heal in a lengthened position and thereby decrease the pain first thing in the morning.[145] If Mr. Benson is compliant with this evidence-based program, then resolution of some, if not all, of his symptoms could be expected.

Table 12–2 **Intrinsic Muscles of the Foot**

MUSCLE	FUNCTION	ANALOG IN HAND
Extensor digitorum brevis	Extends the MTP joints	None
Abductor hallucis	Abducts and flexes MTP of hallux	Abductor pollicis brevis
Flexor digitorum brevis	Flexes PIP of four lesser toes	Flexor digitorum superficialis*
Abductor digiti minimi	Abducts and flexes small toe	Abductor digiti minimi
Quadratus plantae	Adjusts oblique pull of flexor digitorum longus into line with long axes of digits	None
Lumbricals	Flex MTPs, extend IPs of four lesser toes	Lumbricals
Flexor hallucis brevis	Flexes MTP of hallux	Flexor pollicis brevis
Adductor hallucis	Oblique head: adducts and flexes MTP of hallux Transverse head: adducts metatarsal heads transversely	Adductor pollicis
Flexor digiti minimi	Flexes MTP of small toe	Flexor digiti minimi
Plantar interossei	Adduct MTPs of 3rd–5th toes, flex MTPs, extend IPs of four lesser toes	Volar interossei
Dorsal interossei	Abduct MTPs of 2nd toe (either way), abduct MTPs, 3rd and 4th toes, flex MTPs, extend IPs of four lesser toes	Dorsal interossei

*The flexor digitorum superficialis is an extrinsic muscle, whereas the flexor digitorum brevis is an intrinsic foot muscle.
IP, interphalangeal; MTP, metatarsophalangeal; PIP, proximal interphalangeal.

SUMMARY

The foot and ankle consist of a complex arrangement of structures and joints that allow the foot to be flexible and accommodating during early and middle stance phase and relatively rigid during late stance phase. The complexity of the interrelationships makes it easy to disrupt normal function or exceed the limitations of the active and passive tissues that make up the ankle/foot complex. Studies that investigated the relationship between various foot types or alignments and lower extremity injury often show little or no correlation. This should not be surprising, given the many factors that may contribute to health or injury of a given tissue.[1] An important point of this chapter, however, has been to consider how various structures and structural deviations can affect movement and stresses on adjacent structures and tissues. Although group studies may minimize the apparent influence of a given structural problem in dysfunction, the problem should not be ruled out as a factor in, or even primary cause of, pain or dysfunction in an individual case. The kinesiology impact of a given set of alignments and apparent forces in producing maladaptive stresses should be placed in the full context of the individual person, his or her health status, and his or her activity level and activity goals.

STUDY QUESTIONS

1. Identify the proximal and distal articular surfaces that constitute the ankle (talocrural) joint. What is the joint classification?
2. Describe the proximal and distal tibiofibular joints, including classification and their composite function.
3. Identify the ligaments that support the tibiofibular joints.
4. Describe the ligaments that support the ankle joint, including the names of components when relevant.
5. Why is ankle joint motion considered triplanar?
6. Why does the fibula move during dorsiflexion/plantarflexion of the ankle?
7. What are the primary checks of ankle joint motion?
8. Which muscles crossing the ankle are single-joint muscles?
9. Describe the three articular surfaces of the subtalar joint, including the capsular arrangement.
10. Which ligaments support the subtalar joint?
11. Describe the axis for subtalar motion. What movements take place around that axis, and how are these motions defined?
12. When the foot is weight-bearing, the calcaneus (the distal segment) of the subtalar joint is not free to move in all directions. Describe the movements that take place during weight-bearing subtalar supination/pronation.
13. What is the close-packed position for the subtalar joint? Which motion of the tibia will lock the weight-bearing subtalar joint?
14. Describe the relationship between the subtalar and the talonavicular joint with regard to articular surfaces, axes, and available motion.
15. Describe the articulations of the transverse tarsal joint.
16. What is the general function of the transverse tarsal joint in relation to the subtalar joint?
17. What are the tarsometatarsal rays? Describe the axis for the first and fifth rays and the movements that occur around each axis.
18. What is the function of the tarsometatarsal joints in relation to the subtalar and the transverse tarsal joints?
19. How does pronation twist of the tarsometatarsal joints relate to supination of the subtalar joint?
20. What ligaments contribute to support of the medial longitudinal arch of the foot?
21. What is the weight distribution through the various joints from the ankle through the metatarsal heads in unilateral stance?
22. How does extension of the metatarsophalangeal joints contribute to stability of the foot?
23. In terms of structure, compare the metatarsophalangeal joints of the foot with the metacarpophalangeal joints of the fingers.
24. What is the metatarsal break? When does this occur, and how is it related to support of the longitudinal arch?
25. What is the role of the triceps surae muscle group at each joint it crosses?
26. What is the non-weightbearing posture of the subtalar and transverse tarsal joints?
27. What other muscles besides the triceps surae exert a plantarflexion influence at the ankle? What is the primary function of each of these muscles?
28. Which muscles may contribute to support of the arches of the foot?
29. What is the function of the quadratus plantae? What is the analog of this muscle in the hand?
30. Drawing an analogy between the foot and the hand, describe the function of each of the intrinsic and extrinsic foot muscles.
31. If a person has a pes planus, describe two possible causes for this condition.
32. Identify at least three possible effects of pes planus.
33. Identify three primary signs to identify an excessively supinated or pronated foot.

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Section 5

Integrated Function

Chapter 13 **Posture**
Chapter 14 **Gait**

Chapter 13

Posture

Cynthia C. Norkin, PT, EdD

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INTRODUCTION

In this chapter, the focus is on how the various body structures are integrated into a system that enables the body as a whole to maintain a particular posture. We will use our knowledge of individual joint and muscle structure and function as the basis for determining how each structure contributes to the equilibrium and stability of the body in the optimal standing posture We will consider the internal and external forces acting on the body in relation to standing, sitting, and lying postures, and we will explore how these forces will affect the patient in Patient Case 13-1. Throughout the chapter, we will include discussions, in the form of Case Applications, related to our patient's ability to function without many of the normal postural control mechanisms and the internal lower extremity forces necessary to maintain his body in the standing posture, as well as potential problems that he might have in the sitting and lying postures.

13-1 **Patient Case** case

Dave Nguyen, a 19-year-old college varsity ice hockey player, was injured during a game when two members of the opposing team checked him against the boards. The impact of the collision knocked all three players down onto the ice, with Dave on the bottom and the other players on top. Dave sustained fractures of two thoracic vertebrae (T9 and T10) and a complete spinal cord injury, which resulted in paraplegia (muscle paralysis in both lower extremities). He has functioning lower abdominal and lower erector spinae muscles but no function in his hip or lower extremity muscles.

When we first meet Dave, his surgically repaired vertebral fractures have healed, and he is medically cleared to begin an aggressive rehabilitation program.

Dave's main goal at this point is to become independent as soon as possible, including being able to walk again. He admits that his legs probably will not regain their function, but he is determined to be able to get around with crutches.

His youth, his good physical condition, and the fact that he is used to the discipline required for participation in a varsity sport should be helpful in the rehabilitation process.

STATIC AND DYNAMIC POSTURES

Posture can be either static or dynamic. In *static posture*, the body and its segments are aligned and maintained in certain positions. Examples of static postures include standing, sitting, lying, and kneeling. *Dynamic posture* refers to postures in which the body or its segments are moving—walking, running, jumping, throwing, and lifting. An understanding of static posture forms the basis for understanding dynamic posture. Therefore, the static postures of standing and sitting are emphasized in this chapter. The dynamic postures of walking and running are discussed in Chapter 14.

The study of any particular posture includes kinetic and kinematic analyses of all body segments. Humans and other living creatures have the ability to arrange and rearrange body segments to form a large variety of postures, but the sustained maintenance of erect bipedal stance is unique to humans. The erect standing posture allows persons to use their upper extremities for the performance of large and small motor tasks. If the upper extremities need to be engaged by the use of crutches, canes, or other assistive devices to maintain the erect posture, an important human attribute is either severely compromised or lost.

CASE APPLICATION

Predicted Rehabilitation Progression case 13–1

Initially our patient, Dave, will be able to attain a standing posture on a tilt table, which will provide support for his entire body. He will progress to standing in the parallel bars, on which he can use his upper extremities to provide support. Standing will not be as much of a problem as walking, but he will have to learn how to transfer from wheelchair to standing and to maintain the standing posture before he can attempt walking. Later he will progress to walking in the parallel bars and finally will be able to use crutches. However, walking for any extended length of time or distance may not be a realistic goal for Dave because of the high energy cost involved when walking with crutches with the extent of his lower extremity paralysis.

Erect bipedal stance gives us freedom for the upper extremities, but in comparison with the quadrupedal posture, erect stance has certain disadvantages. Erect bipedal stance increases the work of the heart; places increased stress on the vertebral column, pelvis, and lower extremities; and reduces stability. In the quadrupedal posture, the body weight is distributed between the upper and lower extremities. In human stance, the body weight is borne exclusively by the two lower extremities. The human's base of support (BoS), defined by an area bounded posteriorly by the tips of the heels and anteriorly by a line joining the tips of the toes, is considerably smaller than the quadrupedal BoS (Fig. 13–1). The human's center of gravity (CoG) is the point where the mass of the body is centered and will be referred to in this chapter as the center of mass (CoM). The position of the CoM is not fixed and changes in different postures such as sitting and kneeling, with movements of the extremities or trunk, and when a person is carrying something.[1] When a person is wearing a leg cast on one leg, the CoM moves lower and toward the casted leg. In the sitting posture, the CoM of the body above the seat is located near the armpits.[2] In the young child in the standing posture, the CoM is located within the body about at the level of the 12th vertebra. As the child becomes less "top heavy," the CoM moves lower to a location in the standing adult at about the level of the second sacral segment in the midsagittal plane. The adult position of the CoM is relatively distant from the BoS but

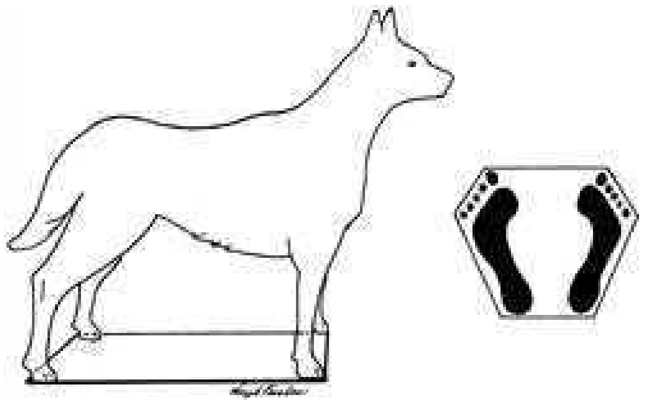


Figure 13–1 A comparison between the base of support (BoS) in quadripedal stance and bipedal stance. Note the small BoS and high center of mass (CoM) in the human figure, in comparison with the dog's relatively large BoS and low CoM.

despite the instability caused by a small BoS and a high CoM, maintaining stability in the static erect standing posture requires only low levels of muscle activity. Passive tension in the joint capsules, muscles, and ligaments is able to provide some of the forces needed to counteract gravity.

Postural Control

Although only a relatively small amount of muscular activity is required to maintain a stable erect standing posture, the control of posture is complex and is a part of the body's motor control system. The main focus of this text is not on the motor control aspects of human function; however, a discussion of some features of postural control is necessary for an understanding of postural stability in standing.

Postural control, which can be either static or dynamic, refers to a person's ability to maintain stability of the body and body segments in response to forces that threaten to disturb the body's equilibrium. According to Horak and associates,[3] the ability to maintain stability in the erect standing posture is a skill that the **central nervous system** learns, using information from passive biomechanical elements, sensory systems, and muscles. The central nervous system interprets and organizes inputs from the various structures and systems and selects responses on the basis of past experience and the goal of the response. **Reactive**[3] **(compensatory**[4]**)** responses occur as reactions to external forces that displace the body's CoM. **Proactive**[3] **(anticipatory**[4]**)** responses occur in anticipation of internally generated destabilizing forces such as raising arms to catch a ball or bending forward to tie shoes.

Major Goals and Basic Elements of Control

The major goals of postural control in the standing position are to control the body's orientation in space, maintain the body's CoM over the BoS, and to stabilize the head with regard to the vertical so that the eye gaze is appropriately oriented. According to DiFabio and Emasithi,[5] stabilizing the head with regard to the vertical is the primary goal of postural regulation. Maintenance and control of posture depend on the integrity of the central nervous system, visual system, vestibular system, and musculoskeletal system. In addition, postural control depends on information received from receptors located in and around the joints (in joint capsules, tendons, and ligaments), as well as on the soles of the feet. The central nervous system must be able to detect and predict instability and must be able to respond to all of this input with appropriate output to maintain the equilibrium of the body. Furthermore, the joints in the musculoskeletal system must have a range of motion that is adequate for responding to specific tasks, and the muscles must be able to respond with appropriate speeds and forces.

Absent or Altered Inputs and Outputs

When inputs are altered or absent, the control system must respond to incomplete or distorted data, and thus the person's posture may be altered and stability compromised. Alteration or absence of inputs may occur for a number of reasons, including, among others, the absence of the normal gravitational force in weightless conditions during space flight or decreased sensation in the lower extremities.

Example 13-1

Astronauts aboard the U.S. space shuttle *Discovery* in June 1985 assumed a position in space in which the neck, hip, and knee were flexed significantly more than they were in preflight.Initially when they returned to earth they maintained the same flexed posture.[6] Another group of astronauts exhibited changes in multijoint coordination that was attributed to a reweighting of inputs to the vestibular system in the gravity-eliminated condition.[7] The postural changes that have been observed in astronauts upon their return to earth are thought to be due to alterations in tactile, articular, vestibular, and proprioceptive inputs used in postural control.[6,7]

A more common example of altered inputs occurs when a person attempts to attain and maintain an erect standing posture when a foot has "fallen asleep." Attempts at standing may result in a fall because input regarding the position of the foot and ankle, as well as information from contact of the "asleep" foot with the supporting surface, is missing.

Another instance in which inputs may be disturbed is after injury. A disturbance in the kinesthetic sense about the ankle and foot after ankle sprains has been implicated as a cause of poor balance or loss of stability.[8] Forkin and colleagues,[9] in a study of gymnasts 1 to 12 months after an ankle sprain, found that these individuals were less able to detect passive range of motion in the previously injured ankle than they were in the uninjured ankle. The gymnasts in the study also reported that they believed that they were less stable in the standing posture than before their injury. Sometimes ankle sprains are followed by chronic functional instability.[10]

In addition to altered inputs, a person's ability to maintain the erect posture may be affected by altered outputs such as the inability of the muscles to respond appropriately to signals from the central nervous system. In sedentary elderly persons, muscles that have atrophied through disuse may not be able to respond with either the appropriate amount of force to counteract an opposing force or with the necessary speed to maintain stability.[11,12] In persons with some neuromuscular disorders, both agonists and antagonists may respond at the same time, thus reducing the effectiveness of the response.

CASE APPLICATION

Missing Inputs and Outputs

case 13–2

Dave is missing some of the inputs and outputs necessary for normal postural control. He is not able to receive input for standing postural control either from receptors located around his ankle, knee, and hip joints or from the soles of his feet. He is unable to provide output in response to signals from the central nervous system because his lower extremity muscles are paralyzed. Consequently, we must look at other aspects of postural control because Dave will have to rely on other mechanisms to monitor and maintain a standing posture. For example, his vestibular and visual systems are intact and able to provide input. Also proprioceptive input and output from his trunk and upper extremities will be of assistance in monitoring his standing posture.

Muscle Synergies

Although static posture is emphasized in this chapter, the term *static* can be misleading, especially with regard to standing posture, because the maintenance of standing posture is the result of dynamic control mechanisms. Postural control researchers have suggested that for any particular task such as standing on a moving bus, standing on a ladder, or standing on one leg, many different combinations of muscles may be activated to complete the task. A normally functioning central nervous system selects the appropriate combination of muscles to complete the task on the basis of an analysis of sensory inputs. Dietz[13] suggested that afferent input from Golgi tendon organs in the leg extensors signals changes in the projection of the body's CoM with regard to the feet. Variations in an individual's past experience and customary patterns of muscle activity will also affect the response. Allum and coworkers[14] suggested that proprioceptive input from the hip or trunk may be more important than input from the legs in signaling and initiating responses. According to these authors, muscle activation is based primarily on input from the hip and trunk proprioceptors. A second level of input includes cues from the vestibular system and proprioceptive input from all body segments.

Monitoring of muscle activity patterns through **electromyography (EMG)** and determinations of muscle peak torque and power outputs are some of the methods used to study postural responses during perturbations of upright postural stability. A **perturbation** is any sudden change in conditions that displaces the body posture away from equilibrium.[3] The perturbation can be sensory or mechanical. A **sensory perturbation** might be caused by altering visual input, such as might occur when a person's eyes are covered unexpectedly. **Mechanical perturbations** are displacements that involve direct changes in the relationship of the body's CoM to the BoS. These displacements may be caused by movements of either body segments or the entire body.[14] Even breathing can displace the CoM. Perturbations in standing that result from respiratory movements of the rib cage are counterbalanced by movements of the trunk and lower limbs. As detemined by electromyography, muscle activity in the trunk and hip muscles provides a counterbalance to motions of the rib cage.[15]

One method of studying how people respond to naturally occuring perturbations is to produce mechanical perturbations experimentally by placing subjects on a movable platform. The platform can be moved forward, backward, or from side to side. Some platforms can be tipped, and the velocity of platform motion can be varied. The postural responses to perturbations caused by either platform movement or by pushes and pulls are reactive or compensatory responses in that they are involuntary reactions. These postural responses are referred to in the literature as either *synergies*[3] or *strategies*.[4] Therefore, in this text, the terms will be used interchangeably. The synergies are task specific and appear to vary with a number of factors, including the amount and direction of motion of the supporting surface; width and compliance of the supporting surface and the location, magnitude, and velocity of the perturbing force; and initial posture of the individual at the time of the perturbation.

Fixed-Support Synergies

Horak and associates[3] described synergies as centrally organized patterns of muscle activity that occur in response to perturbations of standing postures. **Fixed-support synergies** are patterns of muscle activity in which the BoS remains fixed during the perturbation and recovery of equilibrium. Stability is regained through movements of parts of the body, but the feet remain fixed on the BoS. Two examples of fixed-support synergies are the ankle synergy and the hip synergy.

The **ankle synergy** consists of discrete bursts of muscle activity on either the anterior or posterior aspects of the body that occur in a distal-to-proximal pattern in response to forward and backward movements of the support platform, respectively. Forward motion of the platform results in a relative displacement of the line of gravity (LoG) posteriorly and would be similar to starting to fall backward in a free-standing posture (Fig. 13–2A). The group of muscles that responds to the perturbation is activated in an attempt to restore the LoG to a position within the BoS. Bursts of muscle activity occur in the ankle dorsiflexors, hip flexors, abdominal muscles, and possibly the neck flexors. The tibialis anterior muscle contributes to the restoration of stability by pulling the tibia anteriorly, and hence the body forward, so that the LoG remains or centers within the BoS (Fig. 13–2B). Backward motion of the platform results in a relative displacement of the LoG anteriorly and is similar to starting to fall

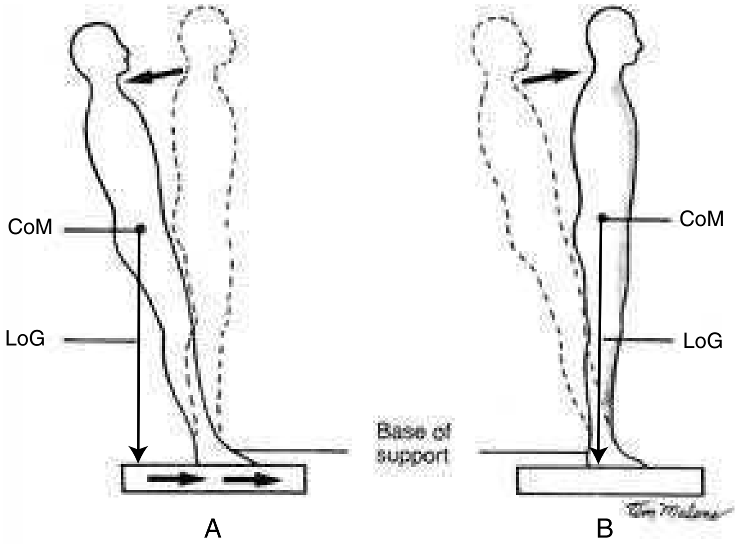


Figure 13–2 Perturbation of erect stance equilibrium caused by forward horizontal platform movement. **A.** Anterior (forward) movement of the platform causes posterior (backward) movement of the body and, as a consequence, displacement of the body's CoM posterior to the BoS. **B.** Use of the ankle strategy (activation of the flexors at the ankle, hip, trunk, and possibly neck) is necessary to bring the body's CoM back over the BoS and reestablish stability.

forward in a freestanding posture. The muscles respond in an attempt to restore the LoG to a position within the BoS (Fig. 13–3A). Bursts of activity in the plantarflexors, hip extensors, trunk extensors, and neck extensors are used to restore the LoG over the BoS (Fig. 13–3B).

The **hip synergy** consists of discrete bursts of muscle activity in a proximal-to-distal pattern of activation.[16] Maki and McIlroy[4] suggest that the fixed-support hip synergy may be used primarily in situations in which **change-in-support strategies** (**stepping** or **grasping synergies**) are not possible.

Change-in-Support Strategies

The change-in-support strategies include **stepping (forward, backward,** or **sidewise)** and **grasping** (using one's hands to grab a bar or other fixed support) in response to shifts in either the BoS or the entire body as in the **tether-release system.** The tether-release system simulates an unbalanced body at the beginning of a trip or slip by holding the person in a forward- or backward-inclined position by means of a horizontal tether attached either to the person's chest or waist. The tether is released suddenly to create the perturbation.[12]

Stepping and grasping differ from fixed-support synergies because stepping/grasping either moves or enlarges the body's BoS so that it remains under the body's CoM (Fig. 13–4).[17,18] Previously, it was thought that the stepping synergy was used only as a last resort, being initiated when ankle and hip strategies were insufficient to bring and maintain the CoM over the BoS.[15,19] However, Maki and McIlroy have suggested that change-in-support strategies are common responses to perturbations among both the young and the old.[4] Furthermore, these authors observed that change-in-support synergies are the only synergies that are successful in maintaining stability in the instance of a large perturbation.[4]

Comparisons of the stepping strategies used by the young and the old show that the younger subjects have a tendency to take only one step, whereas the elderly subjects have a tendency to take multiple steps that are shorter and of less height than those of their younger counterparts.[4,20] However, no differences are apparent in the speed at which

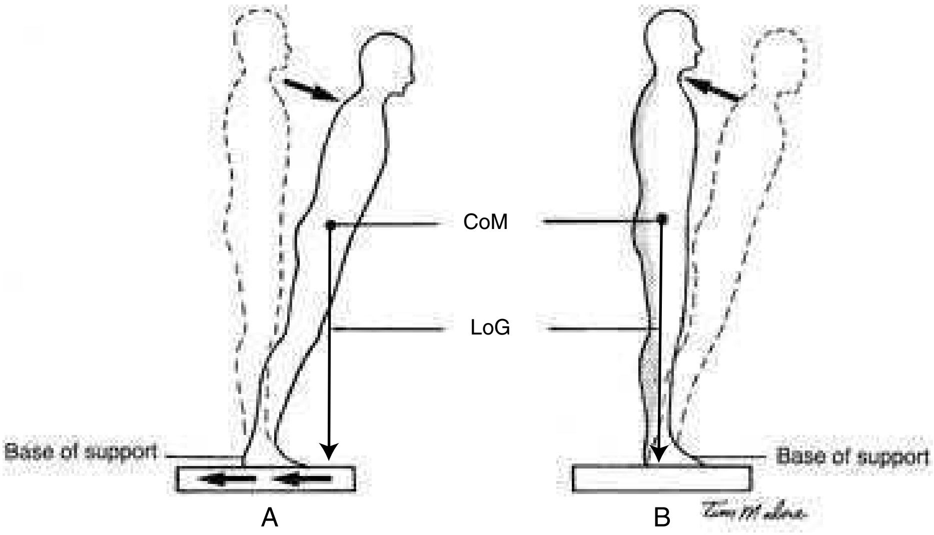


Figure 13–3 Perturbation of erect stance equilibrium caused by backward horizontal platform movement. **A.** Posterior movement of the platform causes anterior movement of the body and, as a consequence, displacement of the body's CoM anterior to the BoS. **B.** Use of the ankle strategy (activation of the extensors at the ankle, hip, back, and possibly neck) is necessary to bring the body's CoM over the BoS and reestablish stability.

Concept Cornerstone **13-1**

Summary of Fixed Support Strategies

Ankle Strategies

Perturbations

Forward translation of support surface (backward motion of the body)	Backward translation of support surface* (forward motion of the body)

Muscles Distal to Proximal Response

Tibialis anterior	Gastrocnemius
Quadriceps femoris	Hamstrings
Abdominals (neck flexors)[5]	Paraspinals (neck extensors)[5]

Hip Strategies

Perturbations

Forward translation of support surface (backward motion of the body)	Backward translation of support surface* (forward motion of the body)

Muscles Proximal to Distal Response

Abdominals	Paraspinals
Quadriceps femoris	Hamstrings
Tibialis anterior	Gastrocnemius

*Increasing the velocity of backward platform translations may lead to a mixed ankle-and-hip strategy.[16]

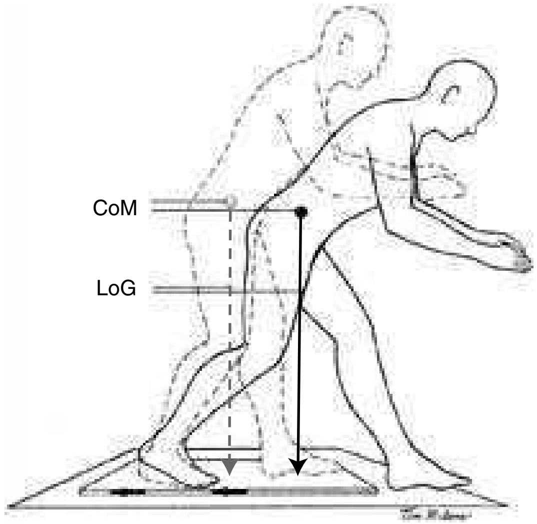


Figure 13–4 Perturbation of erect stance equilibrium caused by backward platform movement. The person in this illustration is using a stepping strategy to keep from falling forward in response to backward movement of the platform. Stepping forward brings the body's CoM over a new BoS.

the young and the elderly initiate the change-in-support stepping strategy. Luchies and associates[21] found that older subjects lifted their feet just as quickly as did the younger subjects. However, Wojcik and associates found distinct age and gender differences in the magnitudes of joint torques in the stepping extremities that were used to regain balance.[22] Maki and McIlroy[11] determined that the elderly had difficulty maintaining lateral stability when the stepping foot was off the ground and the CoM tended to fall toward the unsupported side. A decrease in muscle strength in the contralateral hip abductors may contribute to the lateral instability and the consequent use of multiple steps used by the elderly to recover balance. The elderly also tend to be more reliant on arm reactions than are young adults, but are less able to perform reach-to-grasp reactions rapidly. Van Wegen and colleagues determined that in quiet standing, the older person's CoP is located closer to the edge of the BoS than in younger subjects.[23] Therefore, older individuals may have less time to react to a perturbation before exceeding stability limits.

Head-Stabilizing Strategies

Two head-stabilizing strategies have been described by DiFabio and Emasithi.[5] These proactive strategies differ from the previously described reactive strategies because head-stabilizing strategies occur in anticipation of the initiation of internally generated forces caused by changes in position from sitting to standing. The head-stabilizing strategies are used to maintain the head during dynamic tasks such as walking, in contrast to ankle and hip strategies, which are used to maintain the body in a static situation. The authors described the following two strategies for maintaining the vertical stability of the head: head stabilization in space (HSS) and head stabilization on trunk (HST).[5] The HSS strategy is a modification of head position in anticipation of displacements of the body's CoG. The anticipatory adjustments to head position are independent of trunk motion. The HST strategy is one in which the head and trunk move as a single unit.

Continuing Exploration 13-1:

Evidence on the Nature of Postural Control

Although in the past muscle synergies have been considered fairly automatic responses to perturbations, some evidence suggests that postural control, instead of requiring only a minimal amount of attention, appears to require a significant amount of attention,[24–26] especially the sensory integration aspects.[27] The amount of attention required varies according to the complexity of the postural task (such as bending the trunk backward while standing with the feet close together), the age of the individual, balance abilities, and the type of secondary task (such as a math problem) being performed.[25,26,28] Postural challenges appear to influence reaction time for task performances in both young and old, but under some conditions, older individuals also demonstrate an increase in postural sway.[25,27]

In an attempt to determine if postural responses can be altered voluntarily, Weerdesteyn, Laing, and Rabinovitch[29] conducted a study in which standing subjects attempted either to recover their balance after a perturbation or to resist trying to recover their balance and instead to fall on a protected surface. Subjects were released either backward or sidewise from a tether that held them at 15° from the vertical. Electromyography showed similar reponses in both balance recovery and falling trials, indicating that a balance perturbation always elicits a postural response even when a specific recovery response is not desired. However, in the early stages of the falling trials the amplitude of muscle response was modulated, but did not replace the motor program. This suggests that triggering of postural responses is organized in a reflex-like manner with supraspinal control primarily contributing to adjust these responses in a functional goal-oriented manner.

KINETICS AND KINEMATICS OF POSTURE

The muscle strategies described in response to perturbations are examples of the active internal forces employed to counteract the external forces that affect the equilibrium and stability of the body in the erect standing posture. The following section examines the effects of both external and internal forces on the body in the standing posture. The external forces that will be considered are **inertia, gravity,** and **ground reaction forces.** The **internal forces** are produced by muscle activity and passive tension in ligaments, tendons, joint capsules, and other soft tissue structures. The sum of all of the external and internal forces and torques acting on the body and its segments must be equal to zero for the body to be in equilibrium. Stability is maintained by keeping the body's CoG over the BoS and the head in a position that permits gaze to be appropriately oriented.

Inertial and Gravitational Forces

In the optimal erect standing posture, little or no accleration of the body occurs, except that the body undergoes a constant swaying motion called **postural sway** or **sway envelope.**[18] The extent of the sway envelope for a normal individual standing with about 4 inches between the feet can be as large as 12° in the sagittal plane and 16° in the frontal plane.[18] Usually, the inertial forces that may result from this swaying motion are not considered in the analysis of forces for static postures.[30] However, in a postural study using laser technology, Aramaki and colleagues investigated angular displacements, including angular velocity and acceleration around the hip and ankle joints.[31] Naturally, inertial forces must be considered in postural analysis of all dynamic postures such as walking, running, and jogging in which the forces needed to produce acceleration or a change in the direction of motion are important for understanding the demands on the body.[32]

Ground Reaction Forces

Whenever the body contacts the ground, the ground pushes back on the body. This force is known as the **ground reaction force (GRF),** and the vector representing it is known as the **ground reaction force vector (GRFV).** The GRF is a composite (or resultant) force that represents the magnitude and direction of loading applied to one or both feet. The GRF is typically described as having three components: **a vertical component force** (along the y-axis), and **two force components directed horizontally.** One of the two horizontal forces is in a medial lateral direction (along the x-axis), whereas the other horizontal force is in an anterior posterior direction (along the z-axis) on the ground. The composite or resultant GRFV is equal in magnitude but opposite in direction to the gravitational force in the erect static standing posture. The GRFV indicates the magnitude and direction of loading applied to the foot.

The point of application of the GRFV is at the body's **center of pressure (CoP),** which is located in the foot in unilateral stance and between the feet in bilateral standing postures. If a person were doing a handstand, the CoP would be located between the hands. The CoP, like the CoG, is the theoretical point where the force is considered to act, although the body surface that is in contact with the ground may have forces acting over a large portion of its surface area. The path of the CoP that defines the extent of the sway envelope can be determined by plotting the CoP at regular intervals when a person is standing on a force plate system (Fig. 13–5). Much of the research on postural control uses the pattern of displacements of the CoP to evaluate the effects of attentional demands and perturbations on standing posture.

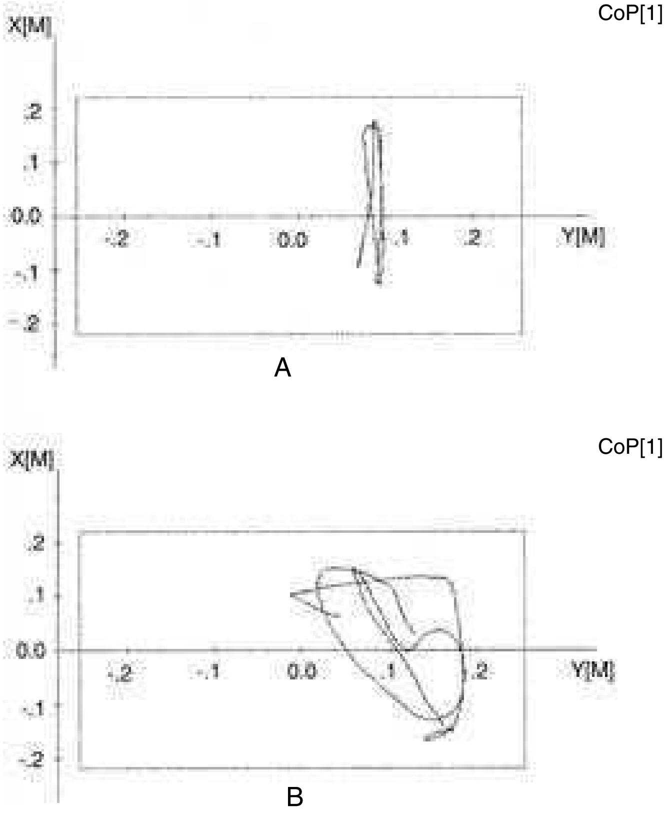


Figure 13–5 Path of the center of pressure (CoP) in erect stance. **A.** A CoP tracing plotted for a person standing on a force plate. The rectangle represents the outline of the force plate. The tracing shows a normal rhythmic anteroposterior "sway envelope" during approximately 30 seconds of stance. **B.** A CoP tracing showing relatively uncontrolled postural sway.

The GRFV and the LoG have coincident action lines in the static erect posture. The LoG represents the force of gravity-on-person and is generally equal in magnitude to and in the same direction as the force of person-on-ground. The ground reaction force is a more common name for the force of ground-on-person described in Chapter 1. In equilibrium during static stance, we would expect the force of gravity-on-person (represented by the LoG) to be equal in magnitude and opposite in direction to the GRF represented by the GRFV. In many dynamic postures, the intersection of the LoG with the supporting surface may not coincide with the point of application of the GRFV. The horizontal distance from the point on the supporting surface where the line of gravity intersects the ground and the CoP (where the GRFV acts) indicates the magnitude of the external moment that must be opposed to maintain a posture and keep the person from falling.

The technology required to obtain GRFs, the CoP, and muscle activity may not be available to the average evaluator of human function. Therefore, in the following sections, a simplified method of analyzing posture will be presented with the use of diagrams and with the combined action of the LoG and the GRFV as a reference.

Coincident Action Lines

In an ideal erect posture, body segments are aligned so that the torques and stresses on body segments are minimized and standing can be maintained with a minimal amount of energy expenditure. The coincident action lines formed by the GRFV and the LoG serve as a reference for the analysis of the effects of these forces on body segments (Fig. 13–6). When the LoG and the GRFV coincide, as they do in static posture, it is possible to assess the effects at each joint by using one or the other. However, the reader should be aware that the horizontal forces are not being considered separately. We will use the LoG in the remainder of this chapter. The location of the LoG shifts continually (as does the CoP) because of the postural sway. As a result of the continuous motion of the LoG, the moments acting around the joints are continually changing. Receptors in and around the joints of lower body segments and on the soles of the feet detect these changes and relay this information to the central nervous system.

CASE APPLICATION

Information From Knee and Ankle Receptors

case 13–3

Dave will not be able to get information either from the soles of his feet or from receptors in his knees and ankles, even though the receptors around these joints are able to transmit the information to the lower spinal cord below the injury. The information cannot go up the spinal cord beyond the level of the injury because of the disruption in the cord at the T10 vertebral level. Under normal conditions, the central nervous system would analyze the inputs and make an appropriate output response to maintain postural stability.

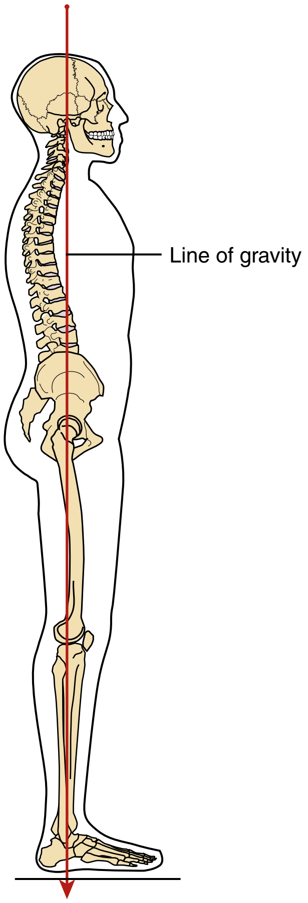


Figure 13–6 Location of the combined action line formed by the ground reaction force vector (GRFV) and the line of gravity (LoG) in the optimal standing posture.

External and Internal Moments

The effect of external forces on body segments during standing is determined by the location of the LoG in relation to the axis of motion of body segments. When the LoG passes directly through a joint axis, no **external** gravitational torque is created around that joint. However, if the LoG passes at a distance from the axis, an **external gravitational moment** is created. This moment will cause rotation of the superimposed body segments around that joint axis unless it is opposed by a **counterbalancing internal moment** (a muscle contraction). The **magnitude** of the gravitational moment of force increases as the distance between the LoG and the joint axis increases. The **direction** of the external gravitational moment of force depends on the location of the LoG in relation to a particular joint axis. If the LoG is located *anterior* to a **particular** joint axis, the gravitational moment will tend to cause anterior motion of the proximal segment of the body supported by that joint. If the LoG is *posterior* to the joint axis, the moment will tend to cause motion of the proximal segment in a posterior direction. In a postural analysis viewed from the side, external gravitational torques producing anterior and posterior motion of the proximal joint segment are referred to as either *flexion* or *extension moments.*

Example 13-2

If the LoG passes anterior to the ankle joint axis, the external gravitational moment will tend to rotate the tibia (proximal segment) in an anterior direction (Fig. 13–7). Anterior motion of the tibia on the fixed foot will result in dorsiflexion of the ankle. Therefore, the moment of force is called a **dorsiflexion moment.** An **internal plantarflexion moment** of equal magnitude will be necessary to oppose the external dorsiflexion moment and establish equilibrium.

Example 13-3

If the LoG passes anterior to the axis of rotation of the knee joint, the external gravitational moment will tend to rotate the femur (proximal segment) in an anterior direction (Fig. 13–8). An anterior movement of the femur will cause extension of the knee. Therefore, the moment of force is called an **extension moment**. An internal **flexion moment** of equal magnitude will be necessary to balance the external extension moment.

CASE APPLICATION

Tilt Table Standing

case 13–4

After his injury, Dave's first exposure to the standing posture will be on a tilt table. Standing on the tilt table will provide compression on Dave's bones and joints and will get him used to the upright position. Wide straps across his legs and trunk will hold Dave to the table and provide the necessary counterbalancing forces to oppose the external gravitational moments.

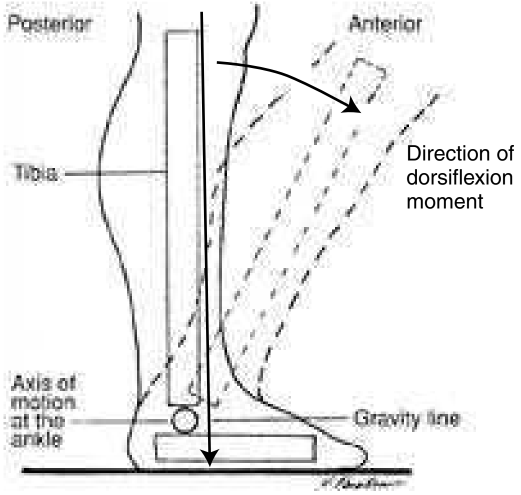


Figure 13–7 The anterior location of the LoG in relation to the ankle joint axis creates an external dorsiflexion moment. The arrow indicates the direction of the dorsiflexion moment. The dotted line indicates the direction in which the tibia would move if the dorsiflexion moment were unopposed.

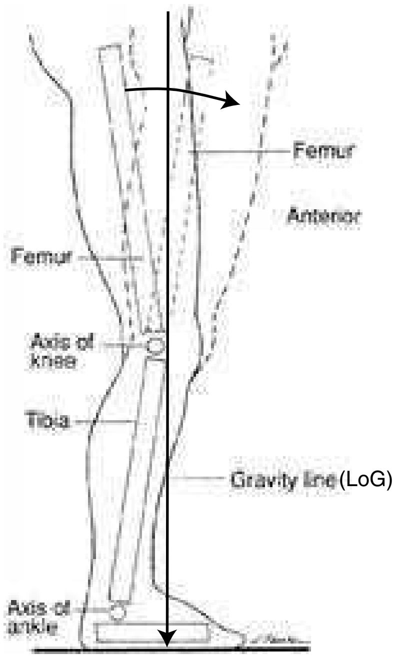


Figure 13–8 The anterior location of the LoG in relation to the knee joint axis creates an external extension moment. The arrow indicates the direction of the extension moment. The dotted line indicates the direction in which the femur would move if the extension moment were unopposed.

OPTIMAL POSTURE

Because the force of gravity is constantly acting on the body, an ideal standing posture is one in which the body segments are aligned vertically and the LoG passes through all joint axes. Normal body structure makes such an ideal posture impossible to achieve, but it is possible to attain a posture that is close to the ideal. In an optimal standing posture, the LoG is close to, but not through, most joint axes. Therefore, the external gravitational moments are relatively small and are balanced by internal moments generated by **passive capsular** and **ligamentous tension, passive muscle tension (stiffness),** and **a small but continuous amount of muscle activity.** The postural control system automatically activates the appropriate muscles to keep the body's CoG over the base of suppport.

Slight deviations from the optimal posture are to be expected in a normal population because of the many individual variations found in body structure. However, deviations from an optimal standing posture that are large enough to cause excessive strain in passive structures and to require high levels of muscle activity need to be identified, and remedial action taken. If faulty postures are habitual and assumed continually on a daily basis, the body will not recognize these faulty postures as abnormal, and over time, structural adaptations such as ligamentous and muscle shortening or lengthening will occur.

ANALYSIS OF STANDING POSTURE: VIEWED FROM THE SIDE

Observational analysis of posture involves locating body segments in relation to the LoG, which is represented by a **plumb line** (a line with a weight on one end). The line is dropped from the ceiling and can be used to assess a person's posture from either the lateral aspect or from the anterior or posterior aspect. In an anterior or posterior analysis, the LoG should bisect the body into two symmetrical halves. Evaluators of posture should be able to determine whether a body segment or joint deviates widely from the normal optimal postural alignment by using their observational skills. Simple photographs taken at the first evaluation may be used to review on-site analysis results and as a basis for comparison with subsequent photographs to determine either the effects of maturation or the results of treatment programs.

Concept Cornerstone **13-2**

Effects of Anterior and Posterior Gravitational Moments on Body Segments

In a lateral analysis, if the LoG passes anterior to the head, vertebral column, or joints of the lower extremities, the gravitational moment will tend to force the segment of the body superior to the joint in an anterior direction. Conversely, when the LoG passes posterior to the joints of the body, the gravitational moment will tend to force the body segment that is superior to the joint in a posterior direction. The rest of Chapter 13's Concept Cornerstone boxes will describe the optimal alignment of the following body structures (ankle, knee, hip, vertebral column, and head) and their relationships to the LoG, and the external and internal moments that are created will be identified.

Alignment and Analysis: Lateral View

Ankle

In the optimal erect posture, the ankle joint is in the neutral position, or midway between dorsiflexion and plantarflexion. The LoG passes slightly anterior to the lateral malleolus and, therefore, anterior to the ankle joint axis.[33,34] The anterior position of the LoG in relation to the ankle joint axis creates an external dorsiflexion moment that must be opposed by an internal plantarflexion moment to prevent forward motion of the tibia. In the neutral ankle position, there are no ligamentous checks capable of counterbalancing the external dorsiflexion moment; therefore, activation of the plantarflexors creates the internal plantarflexion moment that is necessary to prevent forward motion of the tibia. The soleus muscles contract and exert a posterior pull on the tibia and in this way is able to oppose the dorsiflexion moment (Fig. 13–9). Electromyographic studies have demonstrated that soleus[35,36] and gastrocnemius[36] activity is fairly continuous in normal subjects during erect standing. Mochizuki and colleagues[37] determined that the soleus muscles in each leg share a common function by acting to control anteroposterior sway between the legs.

Knee

In optimal posture, the knee joint is in full extension, and the LoG passes anterior to the midline of the knee and posterior to the patella. This places the LoG just anterior to the knee joint axis (see Figs. 13–8 and 13–9). The anterior location of the gravitational line in relation to the knee joint axis creates an **external extension moment** that tends to keep the knees extended.[38] The counterbalancing **internal flexion moment** created by passive tension in the posterior joint capsule and associated ligaments is usually sufficient to balance the gravitational moment and prevent knee hyperextension. However, a small amount of activity has been identified in the hamstrings. Activity of the soleus muscle may augment the gravitational extension moment at the knee through its posterior pull on the tibia as it acts at the ankle joint. In contrast, activity of the two-joint gastrocnemius muscle may tend to oppose the gravitational knee extension moment because the muscle crosses the knee posterior to the knee joint axis and will flex the knee as well as plantarflex the ankle.

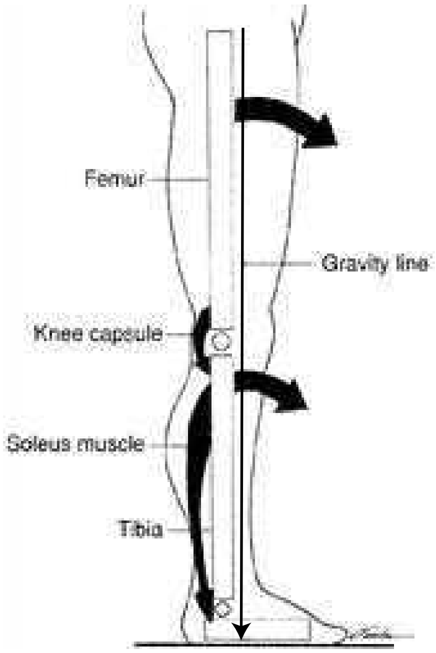


Figure 13–9 The external extension moment acting around the knee joint is balanced by an internal opposing moment created by passive tension in the posterior joint capsule. The external dorsiflexion moment at the ankle is counterbalanced by an internal moment created by activity of the soleus muscle.

CASE APPLICATION

How to Oppose External Gravitational Moments in Standing Posture Without Muscle Activity

case 13–5

When Dave progresses from standing on the tilt table to standing in the parallel bars, he will need some means of opposing the external gravitational moments affecting his lower extremities. He has no musculature capable of resisting the external dorsiflexion moment at the ankle and no tilt table straps to help him maintain the standing posture. Therefore, one or more forces will have to be found to substitute for the internal plantarflexor moment that would have been applied by activity in his plantarflexors. Orthoses (braces) at his ankles and the push of the parallel bars or crutches on his head and trunk will be able to provide the necessary opposing forces.

Concept Cornerstone **13-3**

Orthoses

All orthoses apply forces to the body that can be used either to resist motion or to protect a body part.[39] Orthoses are based on a three-point pressure system with one force acting in one direction and two forces directed in the opposite direction. The type of orthoses that Dave will use are knee-ankle-foot orthoses (KAFOs), which are deemed sufficient for low thoracic lesions (T9 to T12).[40] These orthoses have standard double metal uprights, posterior thigh bands, plates under the sole of the foot, and anterior knee flexion pads (Fig. 13–10). Locking joints are positioned at the knee and ankle; they are locked when the person is standing but can be unlocked to allow for sitting. There are locks at the ankle and knee to keep the knee and ankle stabilized during standing and to allow knee flexion when sitting.

CASE APPLICATION

Role of Knee-Ankle-Foot Orthoses

case 13–6

When Dave is standing, the knee pads on his knee-ankle-foot orthoses (KAFOs) will provide posteriorly directed external forces to oppose the gravitational dorsiflexion moments. The pads and locks at the knee will maintain the knees in extension in the event that the LoG passes behind the knee joint axis. The metal uprights will help support the weight of the body. Anteriorly directed external forces coming from the thigh bands and sole plates under the shoe will help ensure that his knees do not go into excessive hyperextension.

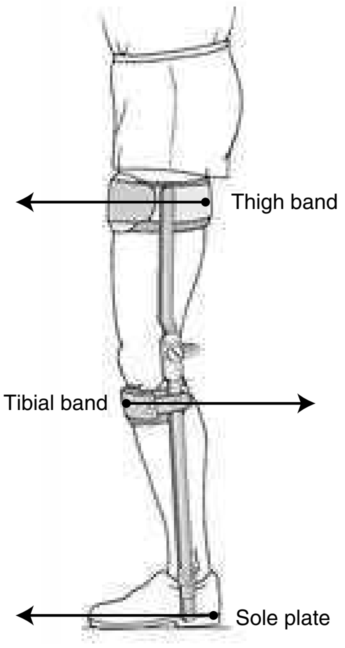


Figure 13–10 Features of the knee-ankle-foot orthosis (KAFO). The KAFO's sole plate attachment at the foot and posterior thigh band provide anteriorly directed forces, and the tibial pad below the knee provides a posteriorly directed force.

Hip and Pelvis

In optimal posture, according to Kendall and McCreary,[41] the hip is in a neutral position and the pelvis is level, with no anterior or posterior tilt (Fig. 13–11A). In a level pelvis position, lines connecting the symphysis pubis and the anterior superior iliac spines (ASISs) are vertical, and the lines connecting the anterior superior iliac spines and posterior superior iliac spines (PSISs) are horizontal.[41] In this optimal position, the line of gravity passes slightly posterior to the axis of the hip joint, through the greater trochanter.[33,34,38,42] However, during postural sway, the LoG may pass anterior to the hip joint axis, and contraction of the hip extensors may be required. The posterior location of the gravitational line in relation to the hip joint axis creates an external extension moment at the hip that tends to rotate the pelvis (proximal segment) posteriorly on the femoral heads[43] (Fig. 13–11B). Electromyographic studies have shown activity of the iliopsoas muscle during standing,[44] and it is possible that the iliopsoas is acting to create an internal flexion moment at the hip to prevent hip hyperextension. If the gravitational extension moment at the hip were allowed to act without muscular balance, as in a so-called relaxed or swayback posture,[41] hip hyperextension ultimately would be checked by passive tension in the iliofemoral, pubofemoral, and ischiofemoral ligaments.

In the swayback standing posture, the LoG drops farther behind the hip joint axes than in the optimal posture (Fig. 13–12). The pelvis is rotated posteriorly; the lumbar spine is flattened; the trunk is displaced posteriorly with an increased kyphosis; the head is forward; and the knees are hyperextended. The swayback posture does not require any muscle activity at the hip but causes an increase in the tension stresses on the anterior hip ligaments, which could

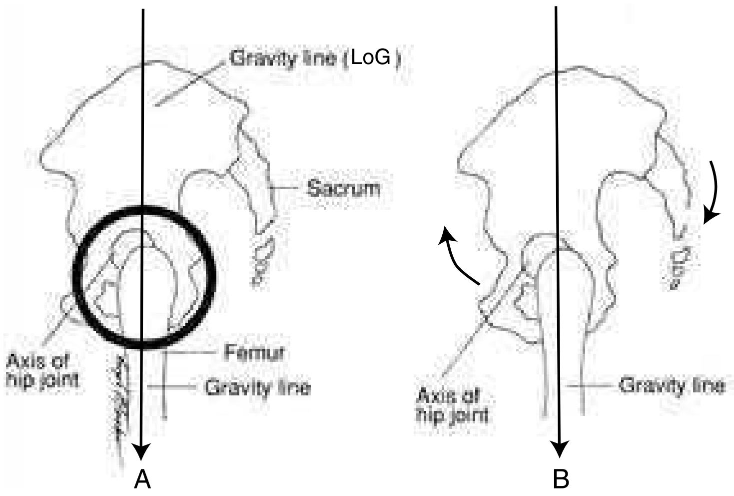


Figure 13–11 The location of the LoG in relation to the axis of the hip joint. **A.** The LoG passes through the greater trochanter and posterior to the axis of the hip joint. **B.** The posterior location of the LoG creates an external extension moment at the hip, which tends to rotate the pelvis posteriorly on the femoral heads. The arrows indicate the direction of the gravitational moment.

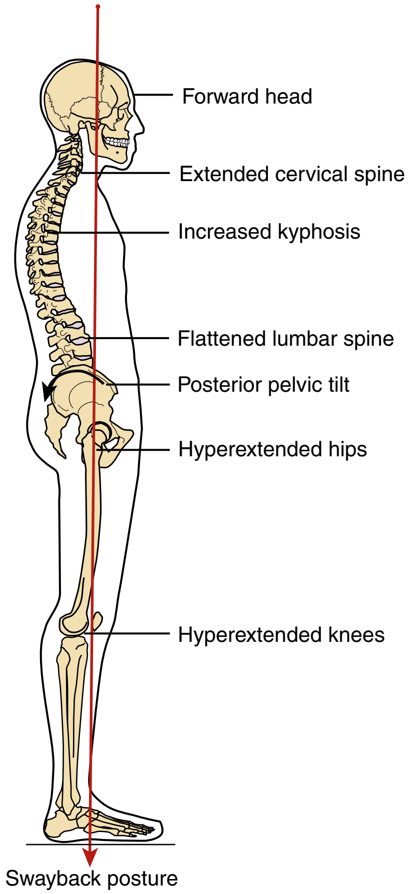


Figure 13–12 In the swayback posture the following deviations from optimal posture should be noted: a forward head with extension of the cervical spine, a backward displacement of the trunk with an increased kyphosis, flattening of the lumbar spine, posterior pelvic tilt, and hyperextended hips and knees.

lead to adaptive lengthening of these ligaments if the posture becomes habitual. Also, because of the diminished demand for hip extensor activity, the gluteal muscles may be weakened by disuse atrophy if the swayback posture is adopted habitually.[45] The relaxed standing or sway posture may also increase the magnitude of the gravitational torque at other joints in the body.

CASE APPLICATION

Swayback Posture[38,42]

case 13–7

Although a swayback posture is considered to be a poor posture partly because of its stress on anterior hip ligaments, Dave has to adopt a swayback standing posture in order to ensure that the LoG remains well behind his hip joints and in front of his knee and ankle joints In this posture, he does not need to have any hip extensors or any bracing at the hip to keep his hip in extension (Fig. 13–13). The KAFOs allow him to balance his weight over his feet with his hips hyperextended and help to prevent hyperextension at his knees.

Continuing Exploration 13-2:

Controversy: Relationship Between Sacral Inclination and Lumbar Lordosis

The nature of the relationship between sacral inclination and lumbar lordosis remains controversial. Youdas and associates,[48] in a study of 90 male and female subjects, found only a weak association between lumbar lordosis and sacral inclination. Conversely, Korovessis and coworkers,[49,50] using x-ray evaluations of erect posture, found that the sacral inclination correlated strongly with both thoracic kyphosis and lumbar lordosis.[49]

Lumbosacral and Sacroiliac Joints

The average lumbosacral angle measured between the bottom of the L5 vertebra and the top of the sacrum (S1) is about 30° but can vary between 6° and 30°.[46,47] Anterior tilting of the sacrum increases the lumbosacral angle and results in an increase in the shearing stress at the lumbosacral joint and may result in an increase in the anterior lumbar convexity in standing (Fig. 13–14A).

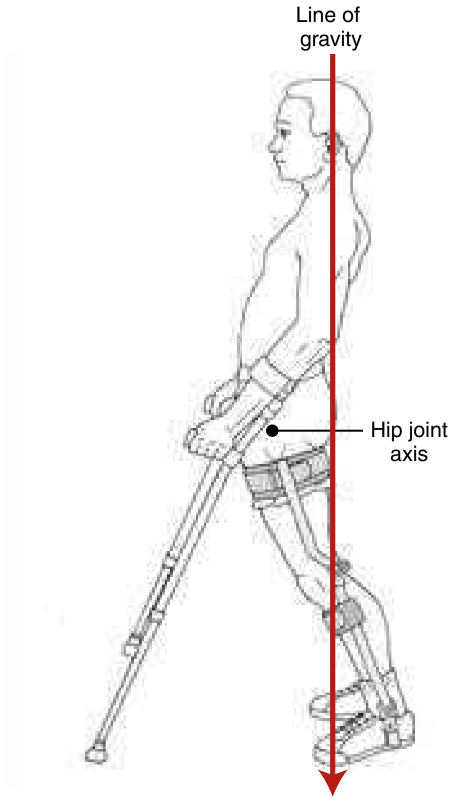


Figure 13–13 In the swayback posture, the LoG passes well posterior to the hip joint axis, which eliminates the need for activity of the hip extensor muscles.

In the optimal posture, the LoG passes through the body of the fifth lumbar vertebra and close to the axis of rotation of the lumbosacral joint. Gravity therefore creates a very slight extension moment at L5 to S1 that tends to slide L5 and the entire lumbar spine down and forward on S1. This motion is opposed primarily by the anterior longitudinal ligament and the iliolumbar ligaments. Bony resistance is provided by the locking of the lumbosacral zygapophyseal joints. When the sacrum is in the optimal position, the LoG passes slightly anterior to the sacroiliac joints. The external gravitational moment that is created at the sacroiliac joints tends to cause the anterior superior portion of the sacrum to rotate anteriorly and inferiorly, whereas the posterior inferior portion tends to move posteriorly and superiorly (Fig. 13–14B). Passive tension in the sacrospinous and sacrotuberous ligaments provides the internal moment that counterbalances the gravitational torque by preventing upward tilting of the lower end of the sacrum.[46]

The Vertebral Column

There is considerable variation among individuals, as can be seen in Table 13–1, but the average values are fairly close to one another in the studies presented. In the optimal configuration, the curves of the vertebral column should be fairly close to average or normal configuration described in Chapter 4. The optimal position of the plumb line is through the midline of the trunk (Fig. 13–15).

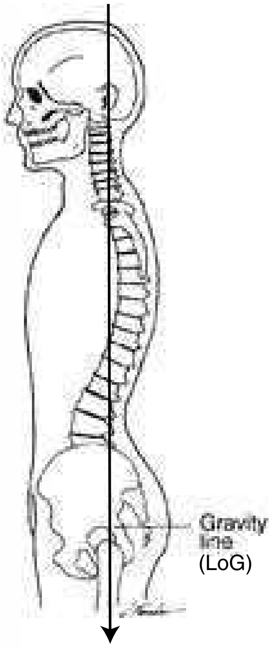


Figure 13–15 Location of the LoG in relation to the trunk.

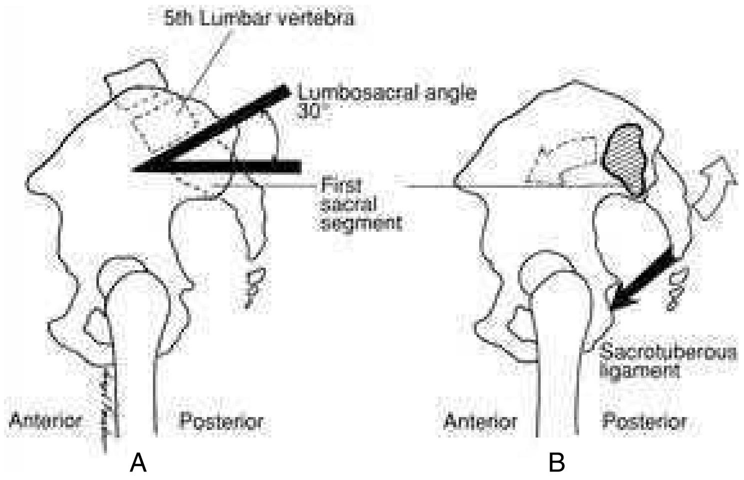


Figure 13–14 **A.** The average lumbosacral angle in optimal erect posture is about 30°. **B.** The gravitational moment tends to rotate the superior portion of the sacrum anteriorly and inferiorly. Consequently, the inferior portion tends to thrust posteriorly and superiorly. Passive tension in the sacrotuberous ligament prevents the upward motion of the inferior sacral segment.

Table 13–1 Variations in Spinal Curves in the Sagittal Plane in Standing Posture: Mean Values in Degrees

Authors	Hardacker et al[51]	Lord et al[52]	Jackson and McManus[53]*	Jackson et al[54]†	Jackson and Hales[55]‡	Gelb et al[56]
Ages	20–70 yr *n* = 100	21–83 yr *n* = 109	20–65 yr *n* = 100	26–75 yr *n* = 20	20–63 yr *n* = 75	40–70 yr *n* = 100
	VALUE					
	Mean (SD)	*Mean (SD)*	*Mean (SEM)*	*Mean (SD)*	*Mean (SD)*	*Mean (SD)*
Cervical	–40 (10)					
Thoracic	49 (11)		42 (9)	43 (9)	46 (11)	
Lumbar	–60 (12)	–49 (15)	–61 (12)	–62 (13)	–63 (12)	–64 (10)

*Ranges: Thoracic curve 22° to 68° and lumbar curve –88° to –31°.
†Ranges: Thoracic curve 23° to 61° and lumbar curve –81° to –38°.
‡Ranges: Thoracic curve 22° to 75° and lumbar curve was –90° to –35°.

Continuing Exploration 13-3:

Controversy: Location of the Line of Gravity Above the Fifth Lumbar Vertebra

The location of the LoG in relation to the vertebral column above the fifth lumbar level is controversial. Cailliet[57] reported that the LoG transects the vertebral bodies at the level of T1 and T12 vertebrae and at the odontoid process of the C2 vertebra. Duval-Beaupere and colleagues,[42] using x-ray examinations of the vertebral columns of 17 young adults, found that the LoG in these individuals was located anterior to the anterior aspect of the T8 to T10 vertebrae. According to Bogduk,[46] the LoG passes anterior to L4 and thus anterior to the lumbar spine in many individuals. In this instance, a flexion moment that would tend to pull the thorax and upper lumbar spine anteriorly. Activity of the erector spinae would be necessary to counteract the flexion moment and maintain the body in equilibrium.

According to Cailliet's frame of reference, the LoG will pass posterior to the axes of rotation of the cervical and lumbar vertebrae, anterior to the thoracic vertebrae, and through the body of the fifth lumbar vertebra. In this situation, the gravitational moments tend to increase the natural curves in the lumbar, thoracic, and cervical regions. Moreover, according to Cailliet,[57] the maximal gravitational torque occurs at the apex of each curve at C5, T8, and L3, because the apical vertebrae are farthest from the LoG and the moment arms are longest at these points. However, as Table 13–2 shows, there is considerable individual variation in the apices for both thoracic and lumbar curves, but the means are similar among investigators. Cailliet's apices are within the ranges shown in the table.

According to Kendall and McCreary,[41] the LoG passes through the bodies of the lumbar and cervical vertebrae and anterior to the thoracic vertebrae in the optimal posture. In this instance, the stress on the supporting structures would be greatest in the thoracic area, where the LoG would pass at greatest distance from the vertebrae. Stress in the lumbar and cervical regions would be comparatively less because the LoG passes close to or through the joint axes of these regions.

Although not confirming either Cailliet's or Kendall and McCreary's hypotheses, electromyographic studies have shown that the longissimus dorsi, rotators, and neck extensor muscles exhibit intermittent electrical activity during normal standing.[59] This evidence suggests that ligamentous structures and passive muscle tension are unable to provide enough force to oppose all external gravitational moments acting around the joint axes of the upper vertebral column. In the lumbar region, where minimal muscle activity appears to occur, passive tension in the anterior longitudinal ligament and passive tension in the trunk flexors apparently are sufficient to balance the external gravitational extension moment.

Head

The LoG in relation to the head passes slightly anterior to the transverse (frontal) axis of rotation for flexion and extension of the head and creates an external flexion moment (Fig. 13–16). This external flexion moment, which tends to tilt the head forward, may be counteracted by internal moments generated by tension in the ligamentum nuchae, tectorial membrane, and posterior aspect of the zygapophyseal joint capsules and by activity of the capital extensors.[57] Ideally, a plumb line extending from the ceiling should pass through the external auditory meatus of the ear, and the head should be directly over the body's CoM at S2.[57,60]

Continuing Exploration 13-4:

Configuration of the Cervical Spine in Standing

In a lateral radiographic study of the spines of 100 standing men and women between 20 and 70 years of age, plumb lines extending from the odontoid process to

Table 13–2 Variations in Apices of Thoracic and Lumbar Curves in the Sagittal Plane in Standing Posture

Authors	Vendantam et al[58]		Jackson and McManus[53]	Gelb et al[56]	
Ages	10–18 yr n = 88		20–65 yr n = 100	40–70+ yr n = 100	
	MEAN	RANGE	MEAN	MEAN	RANGE
Thoracic	T6	T3–T9	T7–78	T7	T5 disc–T10 apex disc
Lumbar apex	L4	L2–L5		L4	L2–L5

C7 fell within a relatively narrow range of 16.8 mm anterior to the center of C7. The greatest lordosis was at C1 to C2 (–31.9; standard deviation [SD] = 7.0), with little lordosis found in the rest of the cervical spine. On average, the occiput-C1 segment was kyphotic, and a segmental kyphosis of 5° or greater was present in 39% of the total group, although no total kyphosis (occiput to C7) was present. In this study, as cervical lordosis increased, thoracic kyphosis increased also.[51]

In a study in which they used slightly different reference points on radiographs, Visscher and colleagues identified the following two types of cervical spine configurations in 54 men and women students standing in a neutral position: a lordotic curvature and a predominately straight spine with an occasional high lordosis and low kyphosis.[61]

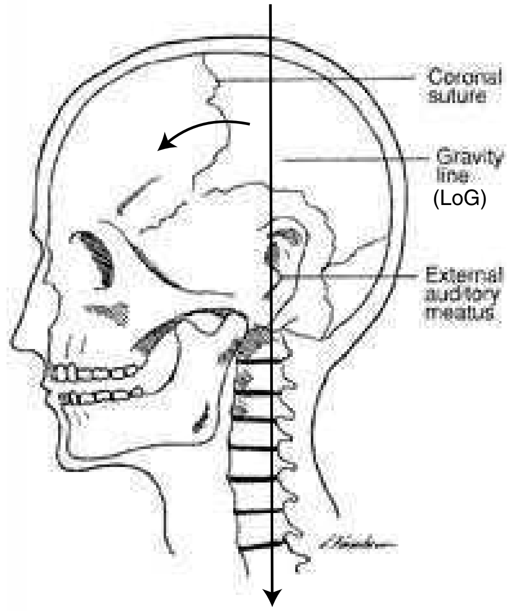


Figure 13–16 The anterior location of the LoG in relation to the transverse axis for flexion and extension of the head creates an external flexion moment.

Alignment of Body Segments Viewed From the Side

Table 13–3 shows the relationship of the LoG to various body segments in the lateral view. However, the reader must realize that the swaying motion that occurs in the normal erect posture will change the position of the LoG in relation to individual joint axes. The location of the center of pressure also will move during swaying. For example, if the amount of forward sway is large enough, the LoG may move from the optimal posterior location in relation to the hip joint axis to a position anterior to the hip joint axis. The CoP will move anteriorly toward the toes. The resulting external flexion moment at the hip created by the change in position of the LoG may be counteracted by activity of the hip extensors, which will move the LoG and CoP posteriorly. On the other hand, increased activity in the soleus muscles rather than in the hip extensors might be used to bring the entire body and thus the LoG back into a position posterior to the hip joint axis. Some independent motion may occur in each leg, and relative motion may occur between body segments in response to postural sway.[60]

If your body is suddenly thrust forward, either by someone bumping into you or by a sudden backward movement of the supporting surface, a large and forceful movement of the LoG will occur. Consequently, flexion moments will be created at the neck and head; cervical, thoracic, and lumbar spines; hip; and ankle. To counteract these moments, the neck, back, hip extensor, and ankle plantarflexor muscles may have to contract. The central nervous system responds with activation of a muscle or pattern of muscles that will counteract the inertial and flexion moments and reestablish static erect equilibrium and stability.

Deviations From Optimal Alignment Viewed From the Side

Minimizing energy expenditure and stress on supporting structures is one of the primary goals of any posture. Any change in position or malalignment of one body segment will cause changes to occur in adjacent segments, as well as changes in other segments, as the body seeks to adjust or compensate for the malalignment (closed-chain response to keep the head over the sacrum).[61] Large changes from optimal alignment increase stress or increase force per unit area

Table 13–3 Alignment in the Sagittal Plane in Standing Posture

JOINTS	LINE OF GRAVITY	EXTERNAL MOMENT	PASSIVE OPPOSING FORCES	ACTIVE OPPOSING FORCES
Atlanto-occipital	Anterior Anterior-to-transverse axis for flexion and extension	Flexion	Ligamentum nuchae and alar ligament; the tectorial, atlantoaxial, and posterior atlanto-occipital membranes	Rectus capitus posterior major and minor, semispinalis capitus and cervicis, splenius capitis and cervicis, and inferior and superior oblique muscles
Cervical	Posterior	Extension	Anterior longitudinal ligament, anterior anulus fibrosus fibers, and zygapophyseal joint capsules	Anterior scaleni, longus capitis and colli
Thoracic	Anterior	Flexion	Posterior longitudinal, supraspinous, and interspinous ligaments Zygapophyseal joint capsules and posterior anulus fibrosus fibers	Ligamentum flavum, longissimus thoracis, iliocostalis thoracis, spinalis thoracis, and semispinalis thoracis
Lumbar	Posterior	Extension	Anterior longitudinal and iliolumbar ligaments, anterior fibers of the anulus fibrosus, and zygapophyseal joint capsules	Rectus abdominis and external and internal oblique muscles
Sacroiliac joint	Anterior	Nutation	Sacrotuberous, sacrospinous, iliolumbar, and anterior sacroiliac ligaments	Transversus abdominis
Hip joint	Posterior	Extension	Iliofemoral ligament	Ilipsoas
Knee joint	Anterior	Extension	Posterior joint capsule	Hamstrings, gastrocnemius
Ankle joint	Anterior	Dorsiflexion		Soleus, gastrocnemius

on body structures. If stresses are maintained over long periods of time, body structures may be altered.

Muscles may lose sarcomeres if held in shortened positions for extended periods. Such adaptive shortening may accentuate and perpetuate the abnormal posture, as well as prevent full range of motion. Muscles may add sarcomeres if maintained in a lengthened position, and as a consequence, the muscle's length-tension relationship will be altered. Shortening of the ligaments will limit normal range of motion, whereas prolonged stretching of ligamentous structures will reduce the ligament's ability to provide sufficient tension to stabilize and protect the joints. Prolonged weight-bearing stresses on the joint surfaces increase cartilage deformation and may interfere with the nutrition of the cartilage. As a result, the joint surfaces may become susceptible to early degenerative changes.

The following sections illustrate how deviation from normal alignment of one or two body segments causes changes in other segments and increases the amount of energy required to maintain erect standing posture. Postural problems may originate in any part of the body and cause increased stresses and strains throughout the musculoskeletal system. Postures that represent an attempt to either improve function or normalize appearance are called **compensatory postures.**[62] Evaluators of posture need not only to identify the deviation but also to determine the cause of the deviation, compensatory postures, and possible effects of the deviation on bones, joints, ligaments, and muscles supporting the affected structures.

Foot and Toes

Claw Toes

Claw toes is a deformity of the toes characterized by hyperextension of the metatarsophalangeal (MTP) joint, combined with flexion of the proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints.[63–65] The abnormal distribution of weight may result in callus formation under the heads of the metatarsals or under the end of the distal phalanx. Sometimes the proximal phalanx may subluxate dorsally on the metatarsal head (Fig. 13–17A).[63] Calluses may develop on the dorsal aspects of the flexed phalanges from constant rubbing on the inside of shoes. In essence, this deformity reduces the area of the BoS and, as a result, may increase postural sway and decrease stability in the standing position.

A few of the many suggested etiologies for this condition are as follows: the restrictive effect of shoes, a cavus-type foot, muscular imbalance, ineffectiveness of intrinsic foot muscles, neuromuscular disorders, and age-related deficiencies in the plantar structures. Valmassy[64] has suggested that the claw toe deformity is actually the same condition as hammer toe because the only difference in the conditions is that a claw toe deformity affects all toes (second through fifth), whereas hammer toe usually affects only one or two toes.

Hammer Toes

In general, hammer toe is described as a deformity characterized by hyperextension of the metatarsophalangeal joint, flexion of the proximal interphalangeal joint,

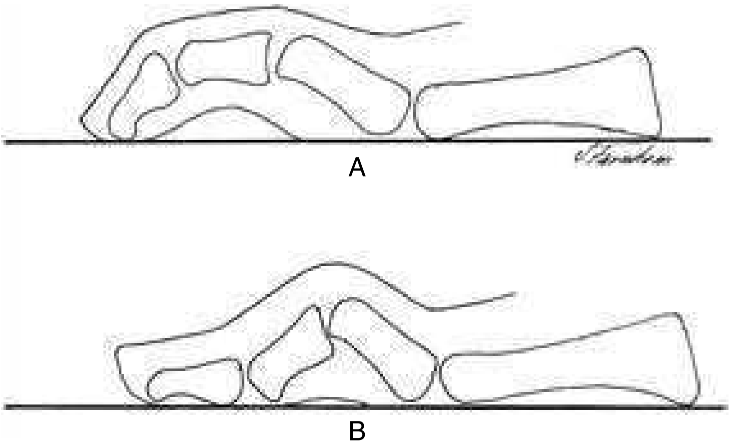


Figure 13–17 Claw toes and hammer toes. **A.** The drawing of claw toes shows hyperextension at the metatarsophalangeal joint and flexion of the interphalangeal joints. **B.** Hammer toes are characterized by hypertension of the metatarsophalangeal and distal interphalangeal joints and by flexion of the proximal interphalangeal joints.

and hyperextension of the distal interphalangeal joint (Fig. 13–17B). Callosities (painless thickenings of the epidermis) may be found on the superior surfaces of the proximal interphalangeal joints over the heads of the first phalanges as a result of pressure from the shoes. The tips of the distal phalanges also may show callosities as a result of abnormal weight-bearing.[64,65] The flexor muscles are stretched over the metatarsophalangeal joint and shortened over the proximal interphalangeal joint. The extensor muscles are shortened over the metatarsophalangeal joint and stretched over the proximal interphalangeal joint. If the long and short toe extensors and lumbrical muscles are selectively paralyzed, the instrinsic and extrinsic toe flexors acting unopposed will buckle the proximal interphalangeal and distal interphalangeal joints and cause a hammer toe.

Knee

Flexed Knee Posture

In the flexed-knee standing posture, which can result from knee flexion contractures, the LoG passes posterior to the knee joint axes. The posterior location of the LoG creates an external flexion moment at the knees that must be balanced by an internal extension moment created by activity of the quadriceps muscles in order to maintain the erect position. The quadriceps force required to maintain equilibrium at the knee in erect stance increases from zero with the knee extended to 22% of a maximum voluntary contraction (MVC) with the knee in 15° of flexion. A rapid rise in the amount of quadriceps force is required between 15° and 30° of knee flexion. When the knee reaches 30° of flexion, the necessary quadriceps force rises to 51% of an MVC.[66] The increase in muscle activity needed to maintain a flexed knee posture subjects the tibiofemoral and patellofemoral joints to greater-than-normal compressive stress and can lead to fatigue of the quadriceps femoris and other muscles if the posture is maintained for a prolonged period.

Other consequences of a flexed-knee erect standing posture are related to the ankle and hip. Because knee flexion in the upright stance is accompanied by hip flexion and ankle dorsiflexion, the location of the LoG also will be altered in relation to these joint axes. At the hip, the LoG may pass anterior to the hip joint axes, creating an external flexion moment. Activity of the hip extensors may be necessary to create an internal extensor moment to balance the external flexion moment acting around the hip. Increased soleus muscle activity may be required to create an internal plantarflexion moment to counteract the increased external dorsiflexion moment at the ankle (Fig. 13–18). The additional muscle activity subjects

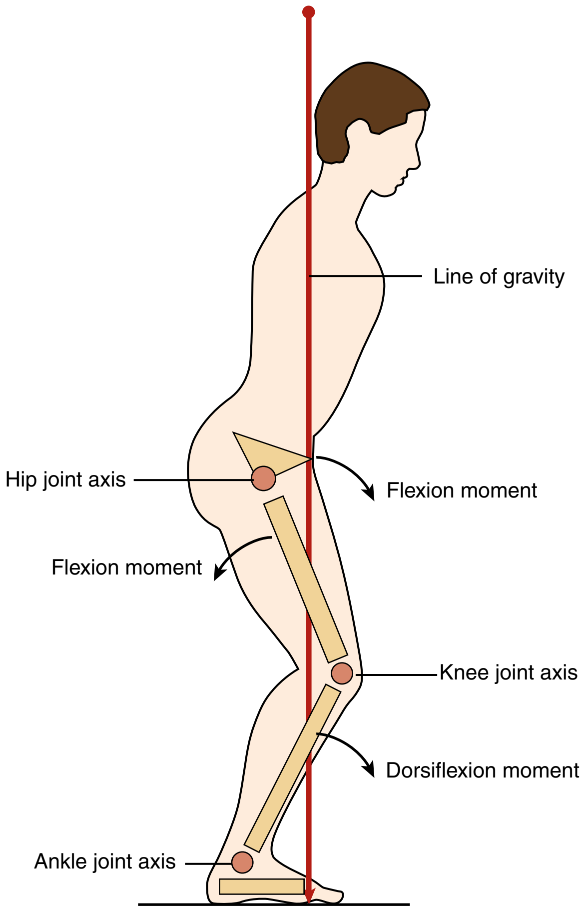


Figure 13–18 Gravitational moments in a flexed-knee posture. External flexion moments are present, acting around the hip, knee, and ankle joints. The external flexion moments are opposed by internal extension moments acting at the hip and knee and by a plantarflexion moment at the ankle.

the hip and ankle joints to greater-than-normal compression stress. Overall, the increased need for quadriceps, gastrocnemius, soleus, and, perhaps, hip extensor activity appears to substantially increase the energy requirements for stance.

CASE APPLICATION

Sitting From Standing

case 13–8

Dave needs to make sure that that the LoG does not pass anterior to the hip joint, because his hip extensors are paralyzed and unable to counteract a flexion moment at the hip. When he is going to sit down, he needs to make sure that he reaches back for the armrests on the wheelchair or other chair so that he can control the flexion moment at the hip when his trunk begins to flex in preparation for sitting. Therefore, development of his upper extremity strength is of utmost importance in his being able to control the lowering of his body into the chair.

Hyperextended Knee Posture (Genu Recurvatum)

The hyperextended knee posture (Fig. 13–19) is one in which the LoG is located considerably anterior to the knee joint axis. The anterior location of the LoG causes an increase in the external extensor moment acting at the knee, which tends to increase the extent of hyperextension and puts the posterior joint capsule under considerable tension stress. A continual adoption of the hyperextended knee posture is likely to result in adaptive lengthening of the posterior capsule and of the cruciate ligaments and, consequently, in a more unstable joint. The anterior portion of the knee joint surfaces on the femoral condyles and anterior portion of the tibial plateaus will be subject to abnormal compression and therefore are subject to degenerative changes of the cartilaginous joint surfaces. The length-tension relationship of the anterior and posterior muscles also may be altered, and the muscles may not be able to provide the force necessary to provide adequate joint stability and mobility.

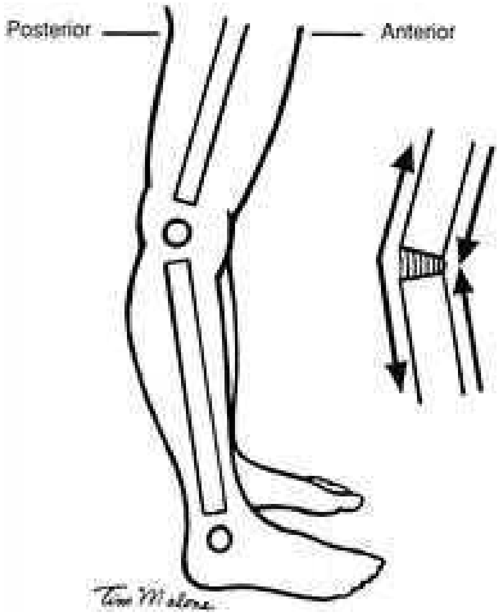


Figure 13–19 In a hyperextended knee posture, the anterior aspect of the knee is subjected to abnormal compressive forces, whereas the posterior aspect is subjected to abnormal tensile forces. Note the limitation of dorsiflexion at the ankle.

Hyperextension at the knee is usually caused either by limited dorsiflexion at the ankle or by a fixed plantarflexion position of the foot and ankle called **equinus.** It may also be the result of habits formed in childhood in which the child or adolescent always elects to stand with hips and knees hyperextended in the relaxed or swayback standing posture.

CASE APPLICATION

Knee Hypertension

case 13–9

Although our patient, Dave, needs to stand in a swayback posture he has some protection from knee hyperextension because of the anteriorly directed counterforce provided by the posteriorly placed thigh and calf pads on his KAFOs. However, we need to check for excessive knee hyperextension because Dave will not be aware of his knee joint position or be able to feel pain from his knee.

Pelvis

Excessive Anterior Pelvic Tilt

In a posture in which the pelvis is excessively tilted anteriorly, the lower lumbar vertebrae are forced anteriorly. The upper lumbar vertebrae move posteriorly to keep the head over the sacrum, thereby increasing the lumbar anterior convexity (lordotic curve). The LoG is therefore at a greater distance from the lumbar joint axes than is optimal and the extension moment in the lumbar spine is increased. The posterior convexity of the thoracic curve increases and becomes kyphotic to balance the lordotic lumbar curve and maintain the head over the sacrum. Similarly, the anterior convexity of the cervical curve increases to bring the head back over the sacrum (Fig. 13–20). Table 13–4 illustrates the changes that may result from an excessive anterior tilt.

In the optimal posture in erect standing, the lumbar discs are subject to tension anteriorly and compression posteriorly. A greater diffusion of nutrients into the anterior than into the posterior portion of the disc occurs in the optimal erect posture.[67] Increases in the anterior convexity of the lumbar curve during erect standing increases the compressive forces on the posterior annuli and may adversely affect the nutrition of the posterior portion of the intervertebral discs. Also, excessive compressive forces may be applied to the zygapophyseal joints.[68]

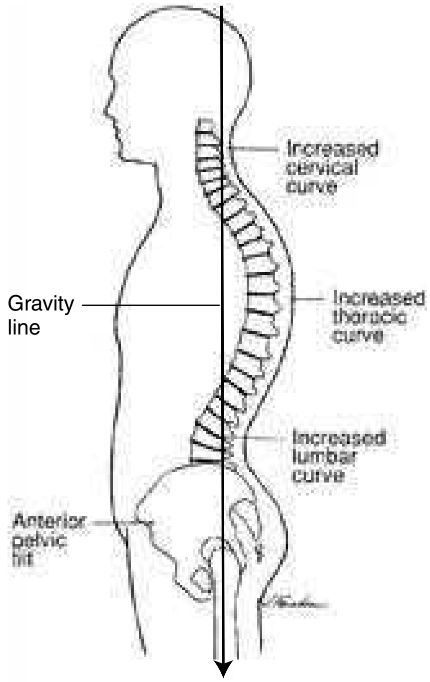
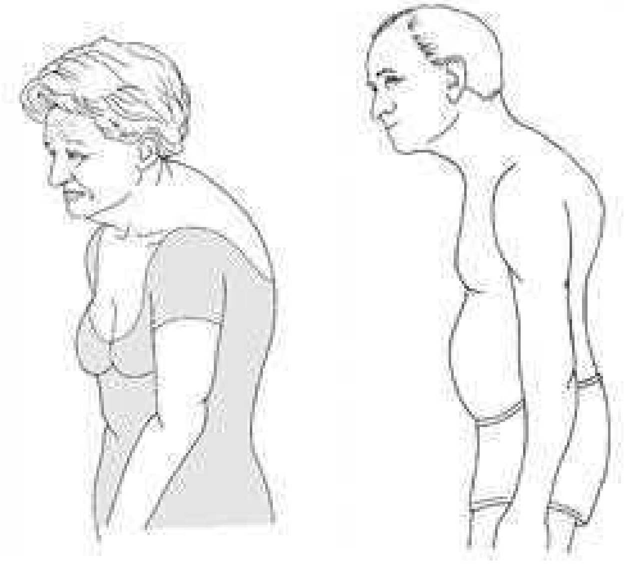


Figure 13–20 An excessive anterior pelvic tilt results in an increase in the lumbar anterior convexity. To compensate for the increased lumbar convexity, there is an increase in the posterior convexity of the thoracic region and an increase in the anterior convexity of the cervical curve.



A Dowager's hump B Gibbus deformity

Figure 13–21 **A.** Dowager's hump usually occurs as a result of compression fractures osteoporotic vetebrae. **B.** A Gibbus deformity may occur as a result of tuberculosis, which causes vertebral fractures.

Excessive Posterior Pelvic Tilt

A posterior pelvic tilt causes a straightening of the lumbar spine and the subsequent loss of flexibility. It also causes some loss of the ability of the vertebral column to withstand as high a load as it did when the lumbar spine had a normal lordosis.

Vertebral Column

Kyphosis and Lordosis

The term **kyphosis** refers to the normal posteriorly convex curves in the thoracic and sacral regions of the vertebral column.[69] Sometimes an abnormal increase in the normal thoracic posterior convexity may occur, and this abnormal condition also may be called a kyphosis.[70] This condition may develop as a compensation for an increase in the normal lumbar curve, as seen in Figure 13–20, or the kyphosis may develop as a result of poor postural habits or osteoporosis.

Two excessively kyphotic conditions, dowager's hump and gibbus deformity, both lead to vertebral fractures, althought they are due to different causes (Fig. 13–21A, B). Dowager's hump is found most often in postmenopausal women who have osteoporosis.[63] The anterior aspect of the bodies of a series of vertebrae may collapse as a result of osteoporotic weakening combined with increased compression stress on the anterior portions of the vertebral body (Fig. 13–22). The vertebral body collapse causes an

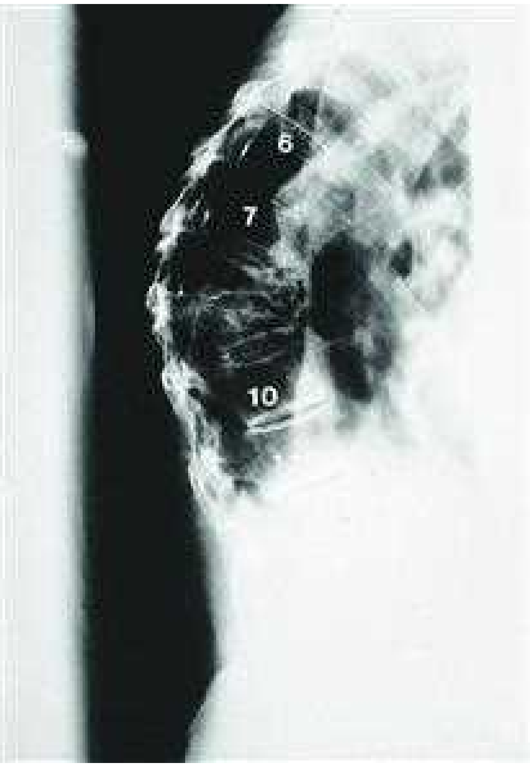


Figure 13–22 The x-ray shows an osteoporotic severely kyphotic thoracic spine in a 90-year-old woman. Compression fractures are present in the eighth and ninth thoracic vertebrae. *(From McKinnis, LN: Fundamentals of Orthopedic Radiology. Published with permission from FA Davis Company.)*

Table 13–4 Possible Effects of Malalignment on Body Structures

DEVIATION	COMPRESSION	DISTRACTION	STRETCHING	SHORTENING
Excessive anterior tilt of pelvis	Posterior aspect of vertebral bodies Interdiscal pressure at L5 to S1 increased	Lumbosacral angle increased Shearing forces at L5 to S1 Likelihood of forward slippage of L5 on S1 increased	Abdominal muscles	Iliopsoas, lumbar extensors
Excessive lumbar lordosis	Posterior vertebral bodies and facet joints Interdiscal pressures increased Intervertebral foramina narrowed	Anterior annulus fibers	Anterior longitudinal ligament	Posterior longitudinal ligament Interspinous ligaments Ligamentum flavum Lumbar extensors
Excessive dorsal kyphosis	Anterior vertebral bodies Intradiscal pressures increased	Facet joint capsules and posterior annulus fibers	Dorsal back extensors Posterior ligaments Scapular muscles	Anterior longitudinal ligament Upper abdominal muscles Anterior shoulder girdle musculature
Excessive cervical lordosis	Posterior vertebral bodies and facet joints Interdiscal pressure increased Intervertebral foramina narrowed	Anterior annulus fibers	Anterior longitudinal ligament	Posterior ligaments Neck extensors

immediate lack of anterior support for a segment of the thoracic vertebral column, which bends further forward, causing an increase in the posterior convexity and an increase in compression on the anterior aspect of the vertebral bodies and the anterior annulus. The LoG will pass at a greater distance from the thoracic spine, and create an increase in the gravitational moment arm. At the same time tensile stresses increase on the posterior aspect (convexity of the curve), which adversely affect the fibers of the posterior annulus and apophyseal joint capsules. Also, increased loading and changes in spinal posture may compromise the force-generating capacity of the back extensor muscles due to alterations in length-tension relationships, moment arm lengths, and force vector orientations.[71] A sustained increased curvature increases the likelihood of soft tissue creep and ossifcation of spinal ligaments as well as limitations in chest expansion.[71]

The term **lordosis** refers to the normal anterior convexity (posterior concavity) of the curves in the lumbar and cervical regions of the spine and also to abnormal increases in the curves usually in the lumbar region, as shown in Figures 13–18 and 13–20. Compression on the posterior aspects of the vertebrae and the posterior annulus are increased and tensile forces are increased on the anterior aspects of the vertebrae and the anterior longitudinal ligament.

Head

Forward Head Posture

A forward head posture is one in which the head is positioned anteriorly and the normal anterior cervical convexity is increased with the apex of the lordotic cervical curve at a considerable distance from the LoG in comparison with optimal posture. The constant assumption of a forward head posture causes abnormal compression on the posterior zygapophyseal joints and posterior portions of the intervertebral discs and narrowing of the intervertebral foramina in the lordotic areas of the cervical region. The cervical extensor muscles may become ischemic because of the constant isometric contraction required to counteract the larger than normal external flexion moment and maintain the head in its forward position. The posterior aspect of the zygapophyseal joint capsules may become adaptively shortened, and the narrowed intervertebral foramen may cause nerve root compression. In addition, the structure of the temporomandibular joint may become altered by the forward head posture, and as a result, the joint's function may be disturbed. In the forward head posture, the scapulae may rotate medially, a thoracic kyphosis may develop, the thoracic cavity may be diminished, vital capacity can be reduced, and overall body height may be shortened. Other possible effects of habitual forward head posture, including adverse effects on the temporomandibular joint, are presented in Table 13–5. Our patient Dave will need to be aware of his head and neck posture because most of the time he will be working and/or relaxing in a seated posture.

Optimal Alignment and Analysis: Anterior and Posterior Views

In both anterior and posterior views, the LoG bisects the body into symmetrical halves (Fig. 13–23). The head is straight, with no tilting or rotation evident, and the face is

Table 13–5 Forward Head Posture

DEVIATION	STRUCTURAL COMPONENTS	LONG-TERM EFFECTS ON STRUCTURAL FUNCTION
Forward Head	Anterior location of LoG causes an increase in the flexion moment, which requires constant isometric muscle tension to support head Stretch of suprahyoid muscles pulls mandible posteriorly into retrusion	Muscle ischemia, pain, and fatigue and possible protrusion of nucleus pulposus Retruded mandible position causes compression and irritation of retrodiscal pad and may result in inflammation and pain Reduction in range of motion
Increase in Cervical Lordosis	Narrowing of intervertebral foramen and compression of nerve roots Compression of zygapophyseal joint surfaces and increase in weight-bearing Compression of posterior annulus fibrosus Adaptive shortening of the posterior ligaments Adaptive lengthening of anterior ligaments Increase in compression on posterior vertebral bodies at apex of cervical curve	Damage to spinal cord and/or nerve roots leading to paralysis Damage to cartilage and increased possibility of arthritic changes; adaptive shortening and possible formation of adhesions of joint capsules with subsequent loss of ROM Changes in collagen and early disc degeneration; diminished ROM at the intervertebral joints Decrease in cervical flexion ROM Decrease in cervical extension ROM and decrease in anterior stability Osteophyte formation
Medial Rotation of the Scapula	Adaptive lengthening of upper posterior back muscles Adaptive shortening of anterior shoulder muscles	Increase in dorsal kyphosis and loss of height Decrease in vital capacity and ROM of shoulder and arm

LoG, line of gravity; ROM, range of motion.

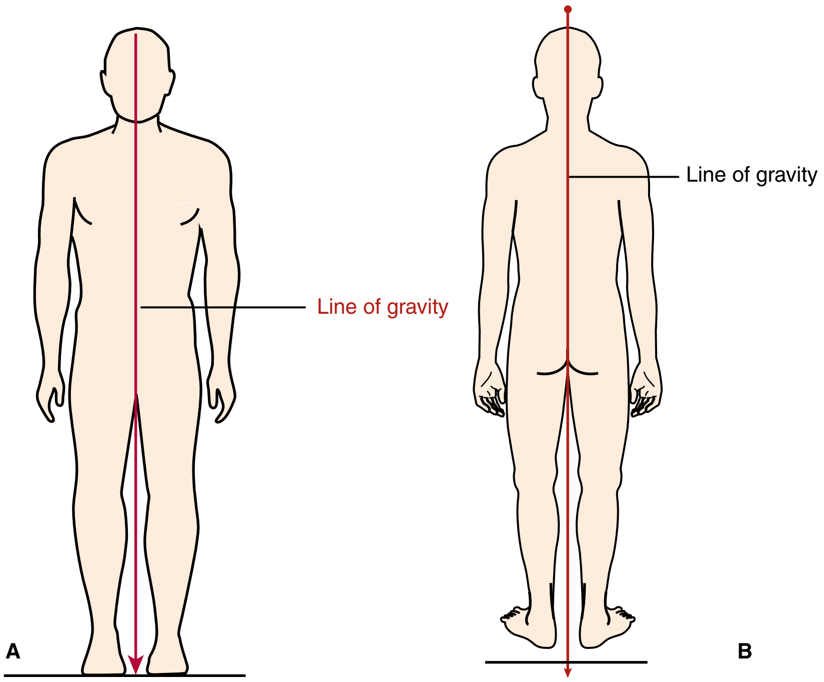


Fig. 13–23 In optimal posture, the LoG divides the body into two symmetrical parts. **A.** Front view. **B.** Back view.

bisected into equal halves. The eyes, clavicles, and shoulders should be level (parallel to the ground). In a posterior view, the inferior angles of the scapulae should be parallel and equidistant from the LoG. The waist angles and gluteal folds should be equal, and the anterior superior iliac spines and posterior superior iliac spines should lie on a line parallel to the ground, as well as being equidistant from the LoG. The joint axes of the hip, knee, and ankle are equidistant from the LoG, and the gravitational line transects the central portion of the vertebral bodies. When postural alignment is optimal, little or no muscle activity is required to maintain medial lateral stability. The gravitational torques acting on one side of the body are opposed by equal torques acting on the other side of the body (Tables 13–6 and 13–7).

Deviations From Optimal Alignment

Any asymmetry of body segments caused either by movement of a body segment or by a unilateral postural deviation will disturb optimal muscular and ligamentous balance. Symmetrical postural deviations, such as bilateral

Table 13–6 Alignment of Standing Posture: Anterior Aspect

BODY SEGMENT	LINE OF GRAVITY LOCATION	OBSERVATION
Head	Passes through middle of the forehead, nose and chin.	Eyes and ears should be level and symmetrical.
Neck/shoulders		Right and left angles between shoulders and neck should be symmetrical. Clavicles also should be symmetrical.
Chest	Passes through the middle of the xyphoid process.	Ribs on each side should be symmetrical.
Abdomen/hips	Passes through the umbilicus (navel).	Right and left waist angles should be symmetrical.
Hips/pelvis	Passes on a line equidistant from the right and left anterior superior iliac spines. Passes through the symphysis pubis.	Anterior superior iliac spines should be level.
Knees	Passes between knees equidistant from medial femoral condyles.	Patella should be symmetrical and facing straight ahead.
Ankles/feet	Passes between ankles equidistant from the medial maleoli.	Malleoli should be symmetrical, and feet should be parallel. Toes should not be curled, overlapping, or deviated to one side.

Table 13–7 Alignment of Standing Posture: Posterior Aspect

BODY SEGMENT	LINE OF GRAVITY LOCATION	OBSERVATION
Head	Passes through middle of head.	Head should be straight with no lateral tilting. Angles between shoulders and neck should be equal.
Arms		Arms should hang naturally so that the palms of the hands are facing the sides of the body.
Shoulders/spine	Passes along vertebral column in a straight line, which should bisect the back into two symmetrical halves.	Scapulae should lie flat against the rib cage, be equidistant from the line of gravity, and be separated by about 4 inches in the adult.
Hips/pelvis	Passes through gluteal cleft of buttocks and should be equidistant from posterior superior iliac spines.	The posterior superior iliac spines should be level. The gluteal folds should be level and symmetrical.
Knees	Passes between the knees equidistant from medial joint aspects.	Look to see that the knees are level.
Ankles/feet	Passes between ankles equidistant from the medial malleoli.	The heel cords should be vertical and the malleoli should be level and symmetrical.

genu valgum (knock knee), that disturb the optimal vertical alignment of body segments cause an abnormal distribution of weight-bearing or compressive forces on one side of a joint and increased tensile forces on the other side. The increased gravitational torques that may occur require increased muscular activity and cause ligamentous stress.

Foot and Toes

Pes Planus (Flat Foot)

An evaluation of standing posture from the anterior posterior aspect should include a careful evaluation of the feet. Normally the plumb line should lie equidistant from the malleoli, and the malleoli should appear to be of equal size and directly opposite from one another. When one malleolus appears more prominent or lower than the other and calcaneal eversion is present, it is possible that a common foot problem known as **pes planus,** or **flat foot,** may be present. Calcaneal eversion of 5° to 10° is normal in toddlers, but by 7 years of age, no calcaneal eversion should be present.[64]

Flat foot, which is characterized by a reduced or absent medial arch, may be either rigid or flexible. A rigid flat foot is a structural deformity that may be hereditary. In the rigid flat foot, the medial longitudinal arch is absent in nonweightbearing, toe-standing, and normal weight-bearing situations. In the flexible flat foot, the arch is reduced during normal weight-bearing situations but reappears during toe-standing or non-weightbearing situations.

In either the rigid or flexible type of pes planus, the talar head is displaced anteriorly, medially, and inferiorly. The displacement of the talus causes depression of the navicular bone, tension in the plantar calcaneonavicular (spring) ligament, and lengthening of the tibialis posterior muscle (Fig. 13–24). The extent of flat foot may be estimated by noting the location of the navicular bone in relation to the head of the first metatarsal. Normally, the navicular bone should be intersected by the Feiss line (Fig. 13–25). If the navicular bone is depressed, it will lie below the Feiss line and may

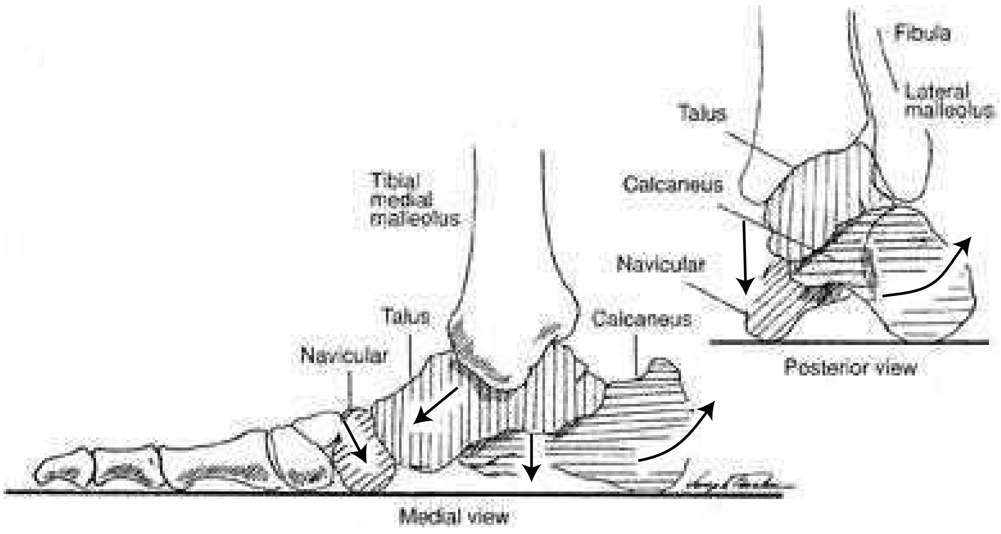


Figure 13–24 In pes planus ("flat foot"), there is displacement of the talus anteriorly, medially, and inferiorly; depression and pronation of the calcaneus; and depression of the navicular bone.

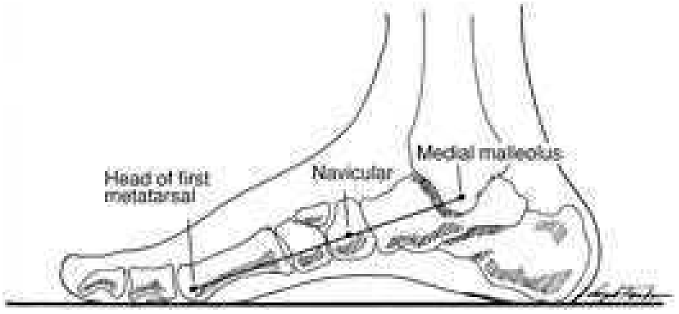


Figure 13–25 In the normal foot, the medial malleolus, the tuberosity of the navicular bone, and the head of the first metatarsal lie in a straight line called the Feiss line.

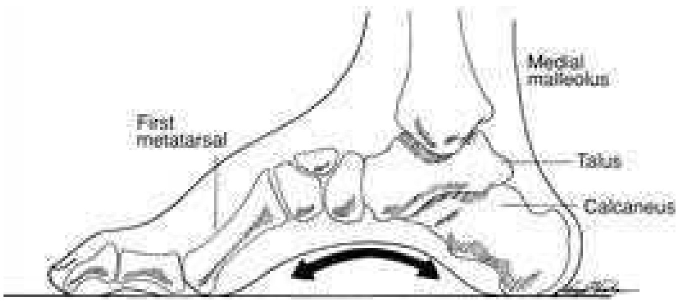


Figure 13–26 Pes cavus.

even rest on the floor in a severe case of flat foot. Flat foot results in a relatively overmobile foot that may require muscular contraction to support the osteoligamentous arches during standing. It also may result in increased weight-bearing on the second through fourth metatarsal heads with subsequent plantar callus formation, especially at the second metatarsal. Weight-bearing pronation in the erect standing posture causes medial rotation of the tibia and may affect knee joint function.

Pes Cavus

The medial longitudinal arch of the foot, instead of being low (as in flat foot), may be unusually high. A high arch is called **pes cavus** (Fig. 13–26). Pes cavus is a more stable position of the foot than is pes planus; however, the weight in pes cavus is borne on the lateral borders of the foot, and the lateral ligaments and the peroneus longus muscle may be stretched. In walking, the cavus foot is unable to adapt to the supporting surface because the subtalar and transverse tarsal joints tend to be near or at the locked supinated position. Therefore, the cavus foot is not a good shock absorber.

Knees

Genu valgum (knock knee) is considered to be a normal alignment of the lower extremity in children from 2 to 6 years of age.[72] However, by about 6 or 7 years of age, the physiological valgus should begin to decrease, and by young adulthood, the extent of valgus angulation at the knee should be only about 5° to 7°. In genu valgum, the mechanical axes of the lower extremities are displaced laterally. If the extent of genu valgum exceeds 30° and persists beyond 8 years of age, structural changes may occur. As a result of the increased external torque acting around the knee, the medial knee joint structures are subjected to abnormal tensile or distraction stress, and the lateral structures are subjected to abnormal compressive stress (Fig. 13–27A). The patella may be laterally displaced and therefore predisposed to subluxation.

The foot also is affected as the gravitational torque acting on the foot in genu valgum tends to produce pronation of the foot with an accompanying stress on the medial longitudinal arch and its supporting structures, as well as abnormal weight-bearing on the posterior medial aspect of the calcaneus (valgus torque). Additional related changes may include flat foot, lateral tibial torsion, lateral patellar subluxation, and lumbar spine contralateral rotation.[63]

Genu varum (bowleg) is a condition in which the knees are widely separated when the feet are together and the malleoli are touching (Fig. 13–27B). Some extent of genu varum is normal at birth and during infancy up to 3 or 4 years of age.[65,72] Physiological bowing is symmetrical and

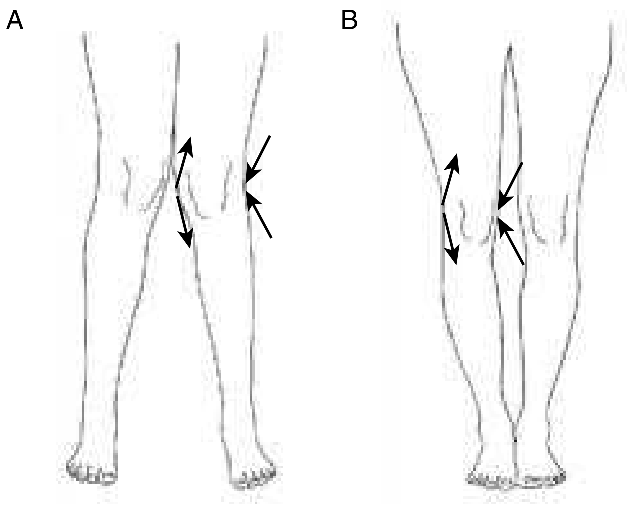


Figure 13–27 **A.** In genu valgum (“knock knee”), the medial aspect of the knee complex is subjected to tensile stress, and the lateral aspect is subjected to compressive stress. **B.** In genu varum (“bowleg”), the lateral aspect of the knee complex is subjected to tensile stress, and the medial aspect of the knee complex is subjected to compressive stress.

involves both the femur and the tibia. Cortical thickening on the medial concavity of both the femur and tibia may be present as a result of the increased compressive forces,[72] and the patellae may be displaced medially. Some of the more commonly suggested cause of genu varum are vitamin D deficiency, renal rickets, osteochondritis, or epiphyseal injury.

Vertebral Column

Scoliosis

Another segment of the body that requires special consideration when posture is evaluated from the front or back of the body is the vertebral column. Normally, when viewed from the posterior aspect, the vertebral column is vertically aligned and bisected by the LoG. The structures on either side of the column are symmetrical. The LoG passes through the midline of the occiput, through the spinous processes of all vertebrae, and directly through the gluteal cleft. In an optimal posture, the vertebral structures, ligaments, and muscles are able to maintain the column in vertical alignment with little stress or energy expenditure. If one or more of the medial lateral structures fails to provide adequate support, the column will bend to the side. The lateral bending will be accompanied by rotation of the vertebrae because lateral flexion and rotation are coupled motions below the level of the second cervical vertebra.

Consistent lateral deviations of a series of vertebrae from the LoG in one or more regions of the spine may indicate the presence of a lateral spinal curvature in the frontal plane called a **scoliosis** (Fig. 13–28). There are two classifications of curves: **functional curves** and **structural curves.** Functional curves are called nonstructural curves in that they can be reversed if the cause of the curve is corrected. These curves are the result of correctable imbalances such as a leg length discrepancy or a muscle spasm. Structural curves, as the name implies, involve changes in bone and soft tissue structures.

Although scoliosis is usually identified as a lateral curvature of the spine in the frontal plane, the deformity also occurs in the transverse (as vertebrae rotate) and sagittal planes (as the column buckles). Idiopathic (cause unknown) scolioses are categorized by age at onset: **infantile** (0 to 3 years), **juvenile** (4 to 10 years), and **adolescent** (older than 10 years).[73] The **adolescent idiopathic scoliosis (AIS)** type makes up the majority of all scolioses[74,75] and affects up to 4% of schoolchildren worldwide.[75] The curves in scoliosis are named according to the direction of the convexity and location of the curve. If the curve is convex to the left in the cervical area, the curve is designated as a left cervical scoliosis. If more than one region of the vertebral column is involved, the superior segment is named first (e.g., left cervical, right thoracic). A double major curve is present when there are two structural curves of the same size. A triple curve includes three regions of the vertebral column.The curve shown in Figure 13–28 is a structural curve called a right thoracic, left lumbar scoliosis. In a study of 606 AIS cases, the most prevalent (51%) type of curve was the main thoracic curve with its apex located between the body of T2 and the disc between T11 and T12.[76]

Investigators have postulated that AIS may result from a dysfunction in the vestibular system,[77] a disturbance in

Figure 13–28 A lateral curvature of the vertebral column that is convex to the right in the thoracic region and convex to the left in the lumbar region. Note the rotation of the vertebra and asymmetry of the rib cage.

control of the muscle spindle,[78] an inherited connective tissue disorder,[79] subcortical brain stem abnormalities,[80–83] developmental instability,[84,85] melatonin production abnormality,[86] growth hormone secretion, and platelet abnormalities.[87] Furthermore, Chan and associates found a genetic locus for AIS.[75]

Lidstrom and coworkers[88] found differences in postural sway in 100 children aged 10 to 14 years; 35 of the children were siblings of scoliotic patients, and 65 were control subjects. This is another finding that suggests the possibility of a genetic component as the causative agent. Nault and associates[83] found a decrease in standing stability among 43 girls diagnosed with scoliosis in comparison with 28 girls without scoliosis. Sway areas, as measured by variations in the CoP and CoM, were larger in the scoliotic group than in the nonscoliotic group. As of this writing, however, no evidence has been presented that unequivocally points to a single etiology for adolescent idiopathic scoliosis,[89] and Ahn and colleagues[87] suggest that the etiology is multifactorial. Despite the ambiguity surrounding the cause or causes of AIS, the effects of unequal torques on the structures of the body are dramatic and can be devastating to those affected. Adolescent idiopathic scoliosis involves changes in the structure of the intervertebral dics, vertebral bodies, transverse and spinous processes, ligaments, and muscles.[90] Growth on the compressed side (concavity) is inhibited or slower than on the side of the convexity of the curve.

Over a 1-year period, pressure profiles were measured in 25 scoliotic and 10 nonscoliotic discs from anesthetized recumbent patients with thoracolumbar curves. Interdiscal pressures and stresses in the scoliotic discs in patients were abnormal, asymmetrical, and high in magnitude, even in the absence of muscle loading. Stress profiles showed a peak in the vertical stress in the concave annulus with the peak being higher in 18 out of 25 discs on the concave side compared to the convex side. The abnormal stresses that were found in the absence of muscle loading was unexpected and unexplainable.[91] Abnormal scoliotic disc pressures provide direct evidence of the presence of assymetrical loading in the scoliotic spine. This assymmetry affects the vertebral end-plate which could calcify and adversely affect the nutrient supply to the disc. Also, abnormal loading can cause shape changes in the disc due to creep of the viscoelastic disc with fluid flow both within the disc and out of the disc to surrounding tissues. However the cause of the assymmetrical loading is still unknown.[91,92]

Example 13-4 depicts a hypothetical series of events for AIS. The first step in the process is unknown because researchers have been unable to identify the supporting structure or structures involved in the initial failure. However, Grivas[86] has postulated that the intervertebral disc, which is more easily deformed than the vertebral body, is the structure that is deformed first. This, in turn, leads to the assymmetrical growth of the vertebral body. Therefore, it is possible that the sequence of events begins with a developmental disturbance that results in asymmetry and failure of the intervertebral disc rather than a failure in the muscular or ligamentous support system, as suggested in the model.

Example 13-4

Hypothetical Series of Events in Adolescent Idiopathic Scoliosis (Fig. 13–29)

1. Possible failure of support as a result of a defect in muscular and/or ligamentous/fascial support systems during a period of rapid growth
2. Creation of an external lateral flexion moment
3. Asymmetrical loading of intervertebral discs
4. Deformation of the intervertebral disc and creation of abnormally high pressures within disc
5. Wedging of the disc
6. Deviation of the vertebrae with rotation
7. Compression of the vertebral body on the side of the concavity of the curve
8. Inhibition of growth of vertebral body on the side of the concavity of the curve in a still immature spine
9. Wedging of the vertebra in a still immature spine
10. Head out of line with sacrum
11. Compensatory curve
12. Adaptive shortening of trunk musculature on the concavity
13. Stretching of muscles, ligaments, and joint capsules on the convexity

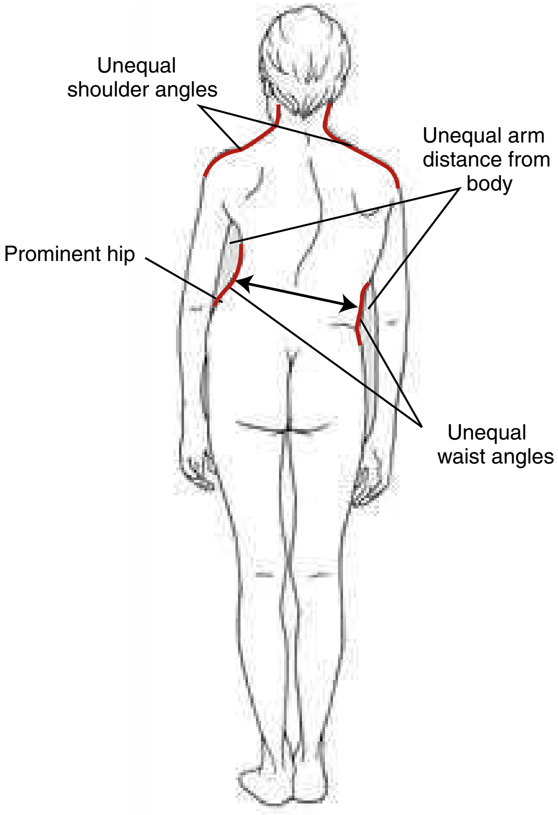


Figure 13–29 A lateral curvature of the vertebral column that is convex to the right in the thoracic region and convex to the left in the lumbar region.

The changes listed may progress to produce a severe deformity as growth proceeds unless intervention occurs at the appropriate time. Deformities can lead to pulmonary dysfunction, decreased vital capacity, and impaired exercise capacity and the function of other internal organs, as well as being cosmetically unacceptable. In the United States, observation combined with periodic follow-up to watch for curve progression is indicated for curves of less than 25°. Bracing may be used for flexible curves between 25° and 40°.[73] Adolescents whose vertebral columns are still immature and who have curves between 25° and 40° are considered to be at high risk because curves of this extent tend to progress.[90] Grivas[86] found girls with right thoracic curves before the age of menses were at high risk of curve progression. Boys were at high risk for curve progression if they had a right lumbar curve. According to Roach, bracing is successful in preventing additional progression of the curve in 70% to 80% of the cases in which it is used.[90] During periods of fast growth, braces worn for 23 hours per day are most effective in preventing curve progression.[93] Curves that have progressed beyond 40° may necessitate surgical intervention to prevent further progression. If a curvature is recognized early in its development, then measures may be instituted either to correct the curve or to prevent its increase.[94] However, some curves may progress even after surgery.[95]

According to the second phase of the 1988 Utah study in which a visual assessment (scoliosis screening) of 3,000 college-aged women (19 to 21 years of age) was performed in 34 states and 5 foreign countries, 12% of this population had a previously undetected lateral deviation of the spine.[96] In consideration of the fact that AIS may be progressive in some cases and lead to a considerable amount of deformity without treatment, early recognition is important. The vertebral deviations in scoliosis cause asymmetrical changes in body structures, and several of these changes may be detected through simple observation of body contours either at home or in the schools. Usually, home or school screening programs are designed for identification of the following: **unequal waist angles, unequal shoulder levels or unequal scapulae** (Fig. 13–30A), **rib hump, and obvious lateral spinal curvature** (Fig. 13–30 B).

The American Academy of Orthopedic Surgeon's Patient Service Brochure on scoliosis, which is available online, recommends that parents check their children's spines for any assymetry in body contours beginning at age 8 years. Parents are asked to inform the child's physician if any postural asymmetries are observed in their child.[97] The American Physical Therapy Association's (APTA's) pamphlet on scoliosis (also available online) suggests that home screening should take place every 6 months for both boys and girls starting at age 9 years and continue until the child is 14 years of age.[98] Grivas[86] advocates school screening not only to identify individuals with unrecognized AIS but also to identify those with a high risk of curve progression. Also, screening provides researchers with an important opportunity for expanding knowledge about the natural history of AIS.[86]

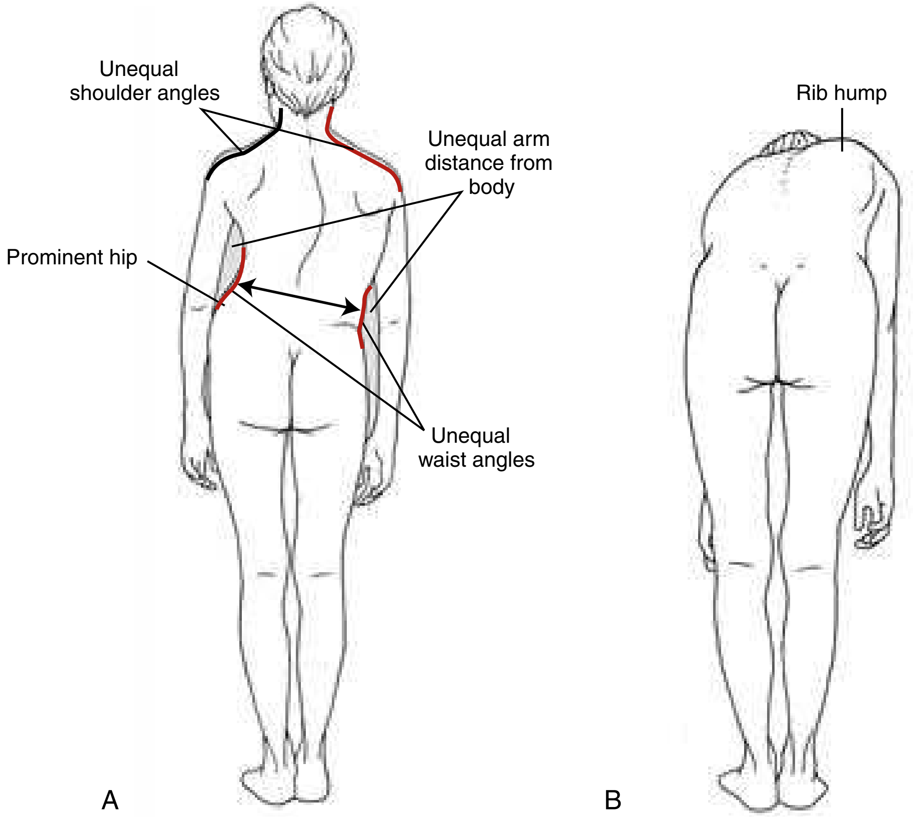


Figure 13–30 Typical changes in body contours used in scoliosis screening programs. **A.** Uneven waist angles or difference in arm to body space and unequal shoulder height or unequal scapula levels. **B.** A rib hump during forward trunk flexion.

Continuing Exploration 13-5:

Treatment

The International Society on Scoliosis, Orthopaedic and Rehabilitation Treatment (SOSORT) was established in Barcelona in 2004 to verify the scientific basis and efficacy of rehabilitation practices.[99] Its main purpose is to implement multidisciplinary reasearch to develop quantitative, objective data on the role of conservative treatment.[99] The new journal *Scoliosis* (the official journal of SOSORT) is the main forum for experts in conservative management of patients with spinal deformities.[100] In addition, the Scientific Exercises Approach to Scoliosis (SEAS) has been developed that is directed toward decreasing the rate of brace prescription and surgery.[101] SEAS is a personalized approach aimed at stabilizing the spine, strengthening the tone of antigravity muscles, improving balance and coordination, recovery, and maintaining physiological sagittal curves and full functional improvement. It is thought to be more effective than routine physical therapy as it involves self-correction actively performed by the patient without an external aid and can be taught by expert physical therapists.[101] A retrospective study over a 3-year period involving 112 AIS patients demonstrated that it was possible to reduce the rate of surgery considerably in AIS patients using SEAS plus orthoses.[102]

Some evidence exists in the form of prospective controlled studies that intensive rehabilitation and braces can alter the natural history of scoliosis, but no studies are available comparing the natural history with surgical treatment.[103,104] Proactive methods (physical therapy) used in European countries (France, Poland, Italy, Spain, Switzerland, Austria, and Germany) are in contrast to methods used in the United States, where physical therapy may be excluded in favor of surgery without scientific justification.

ANALYSIS OF SITTING POSTURES

The overall goal for sitting posture is the same as the goal for standing posture: to attain a stable alignment of the body that can be maintained with the least expenditure of energy and the least stress on body structures. In our analysis of standing posture, we saw that moments at the spine and extremity joints were created when the LoG was at a distance from either a portion of the vertebral column or the axes of the extremity joints. The greater the distance that the LoG was from the joint axes, the larger the moment that was created and, as a result, the more muscle activity and/or passive tension in ligaments and joint capsules that was required to maintain equilibrium and a stable posture. The necessary increase in muscle activity resulted in more energy expenditure and increased loads on body structures.

In a way, sitting postures are more complex than standing postures. The same gravitational moments as in standing posture must be considered, but, in addition, we must consider the contact forces that are created when various portions of the body interface with various parts of chairs, such as head, back, and foot rests, and seats. The location and amount of support provided to various portions of the body by the chair or stool may change the position of the body parts and thus the magnitude of the stresses on body structures.

Example 13-5

The use of a lumbar support can help to maintain the normal lumbar lordotic curve and reduce the compressive stress on the spine in comparison with sitting without a lumbar support.[105]

There are many different sitting postures, but we will direct our attention to the **active erect sitting posture,** which is defined in this chapter as an unsupported posture in which a person attempts to sit up as straight as possible. A consideration of **muscle activity, interdiscal pressures,** and **seat interface pressures** in the active erect sitting posture will be compared to forces in **relaxed erect, slumped,** and **slouched** sitting and to erect standing postures. In addition, we will discuss how these forces may affect Dave.

Muscle Activity

The LoG passes close to the joint axes of the head and spine in active erect sitting posture. (Fig. 13–31A, B). In the slumped posture, the LoG is more anterior to the joint axes of the cervical, thoracic, and lumbar spines than it is in either active or relaxed erect sitting (Fig. 13–31C). Therefore, we would expect that more muscle activity would be required in the slumped posture than in the other sitting postures. In contrast to these expectations, researchers have found that maintaining an active erect sitting posture requires not only a greater number of trunk muscles but also an increased level of activity in some of these muscles than in both relaxed erect and slumped postures. O'Sullivan and associates[106] used electromyography to monitor activity in the superficial lumbar multifidus, thoracic erector spinae, and internal oblique abdominal muscles in erect and slumped sitting postures. These authors found a significantly greater amount of activity in these muscles in erect sitting than in slumped sitting.

The **flexion relaxation (FR) phenomenon** may provide a possible reason why the slumped sitting posture requires less muscle activity than does the active erect sitting posture. Flexion relaxation is a sudden cessation of muscular activity, as manifested by electrical silence of the back extensors during trunk flexion in either sitting or standing postures. In a study by Callaghan and Dunk, flexion relaxation occurred in the thoracic erector spinae muscles (thoracic components of the longissimus thoracis and iliocostalis lumborum) in 21 of 22 subjects in slumped sitting and relaxed erect sitting but not in active erect sitting. Muscle activity in the lumbar erector spinae

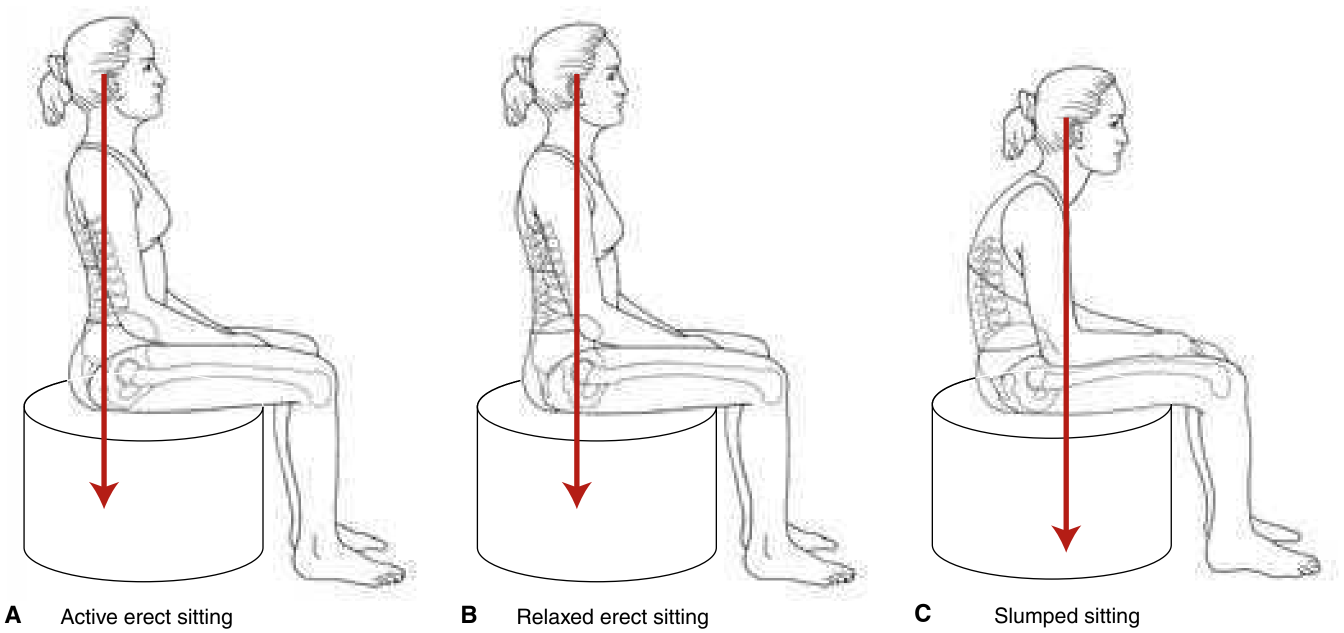


Figure 13–31 **A.** In the active erect sitting position, the LoG is close to the axes of rotation of the head, neck, and trunk. **B.** In the relaxed erect sitting posture, the LoG still is relatively close to those axes of rotation. **C.** In the slumped position, the LoG is relatively distant from the axes of rotation of the head, neck, and trunk.

remained the same in both postures. The authors postulated that the passive tissues were able to assume the load in the relaxed erect and slumped postures and that was why the thoracic erector spinae muscles ceased their activity.[107]

Muscle activity in the active erect sitting posture is greater than in both relaxed erect and slouched sitting. In relaxed erect sitting, the LoG is only slightly anterior to its position in active erect sitting. In the slouched posture, the LoG is posterior to the spine and hips, but body weight is being supported by the back of the chair, and so less muscle activity is required than in active erect posture (Fig. 13–32).[108]

Figure 13–32 In the slouched sitting posture, the LoG is at a distance from the axes of rotation at the head, neck, and trunk, but the back of the chair is providing support in lieu of muscle support.

Conversely, sustained slumped postures in which the lumbar spine is flattened or flexed leads to an increase in interdiscal pressure due to an increase in compressive stress and load on the anterior annulus and an increase in the tensile forces on the posterior annulus. If loading is sustained, creep may increase in the posterior annulus, which stretches and thins and finally may lead to disc degeneration.[109]

CASE APPLICATION

Strengthening of Trunk Muscles for Sitting and Transfers

case 13–10

Dave's trunk muscles are not paralyzed but are weakened from the surgery and subsequent bedrest. Therefore, one goal that we will have for Dave is to strengthen his trunk muscles so that he will be able to maintain his stability in sitting as well as in transfer and other daily living activities. (Individuals with a spinal cord injury below T3 may use the latissimus dorsi and lower trapezius muscles to help maintain stability in sitting, but it appears that Dave will not need these muscles to help in sitting.[110])

Muscle Activity in Sitting Versus Standing Postures

The amount of muscle activity employed to maintain a particular posture affects the amount of interdiscal pressure and energy expenditure. In general increases in muscle activity cause increases in interdiscal pressures and decreases in muscle activity are accompanied by decreases in interdiscal pressures. Callaghan and McGill[111] noted that the upper and lower erector spinae muscles shifted to higher levels of activity during active erect sitting than during standing. This increase in muscle activity has been attributed in part to the differences in the extent of lumbar lordosis observed between sitting and standing. Sitting forces the pelvis into a posterior tilt and, as a result, causes a reduction in the lumbar curve in comparison with that observed in standing.[105] In one radiographic study of 109 patients, the average lumbar curve (L1 to S1) was 15° less in active erect sitting than was an average lumbar curve of 49° in the same population in standing posture.[52] The LoG is more distant from the apex of the joint axes of the lumbar vertebrae in a flexed or more kyphotic lumbar spine than in a lordotic lumbar spine. Therefore, one would expect that more muscle activity would be required to maintain the active erect sitting posture than to maintain erect standing. However, the results of the following study raise questions about a more kyphotic lumbar spine being responsible for all of the increase in muscle activity in active erect sitting versus standing.

Continuing Exploration 13-6:

Muscle Activity and Lumbar Lordosis in Standing and Sitting Postures

When the lordotic lumbar curve in standing was replicated in unsupported active erect sitting in 30 subjects, the electromyographic activity level of the extensor muscles was significantly higher than in standing.[112] It thus appears from this study that the loss of lordosis in the sitting posture is not totally responsible for the greater amount of muscle activity observed in erect sitting than in standing.

Many variables need to be considered, and it is possible that investigators either did not employ identical conditions in their research or did not consider all of the variables that might affect muscle activity in erect sitting versus erect standing. For instance, something as simple as changing from a hard seat to a soft seat will decrease the amount of activity in the internal and external oblique muscles.[113] Supporting one's arms on a table when using both hands for data entry[114] can reduce the load on the left and right trapezius and erector spinae muscles in comparison with working with unsupported arms.

Interdiscal Pressures and Compressive Loads on the Spine

Direct determinations of interdiscal pressures have been made through the insertion of pressure-sensitive sensors or transducers[115–118] into one or more intervertebral discs. Indirect determinations of interdiscal pressures have used measurements of spinal shrinkage (creep)[119–122] and calculation of compressive forces based on information obtained from electromyography about muscle activity.[112,113] Pynt, Mackey, and Higgs reviewed the literature from 1985 to 2007 regarding the relationship between seated postures and the health of the lumbar spine. They concluded that sustained sitting postures in which the lumbar spine was kyphosed (flexed) were more harmful to the lumbar spine than lordosed (extended) sitting postures. According to these authors, kyphosed sitting postures increase the intervertebral disc shear force, posterior annulus tensile forces and anterior annulus load, hydrostatic pressure in the nucleus, and loading of the posterior ligamentous system and posterior fibers of the annulus.[109]

Active erect sitting requires co-contractions of trunk extensors (erector spinae muscles) and flexors (abdominal muscles), which cause higher pressures in the disc between L4 and L5 than does slumped sitting. One of the most well-known studies in which direct interdiscal pressure measurements in erect sitting were compared with pressures in erect standing and other postures was by Nachemson,[115] who reported that there was a 40% increase in pressures in the disc between L4 and L5 in erect sitting in comparison with erect standing. However, the results of some studies[116–118] in which advanced sensor technology was used suggest that interdiscal pressures/loads on the spine in active erect sitting are either only slightly higher than[116] or equal to[117] pressures in erect standing (Fig. 13–33).

Continuing Exploration 13-7:

Comparison of Interdiscal Pressures in Standing and Sitting

One of the problems in interpreting the results of investigations is that it is not always possible to determine whether the researchers are referring to active erect sitting or relaxed erect sitting. Undoubtedly, the controversy will continue until enough additional studies are performed to either confirm or deny Nachemson's findings. Some researchers agree that interdiscal pressures in relaxed or slouched sitting postures are lower than in erect standing.[116,117] Wilke and colleagues reported that the interdiscal pressure at the disc between L4 and L5 disc in relaxed erect sitting was slightly lower than in relaxed standing.[116] Straightening the back to attain an active erect sitting posture increased interdiscal pressures by approximately 10%.[116] Rohlmann and associates found that bending moments on an internal spinal fixation device were, on average, 13% lower for relaxed erect sitting than for relaxed standing[117] (Fig. 13–34).

The results of spinal shrinkage measurements comparing sitting and standing work lend some support to the findings that, in general, loads in sitting are less than in standing. Leivseth and Drerup[120] showed that shrinkage of the lumbar spine in sitting (1.73 mm) was much less than the shrinkage in standing (4.16 mm). In a comparison of spinal shrinkage between 2 hours of relaxed sitting versus 2 hours of work done in standing, there actually was a gain in stature in the lumbar spine in the relaxed sitting position. Working in a standing position caused a reduction in height of 0.8 mm per lumbar disc, in comparison with 0.3 mm for

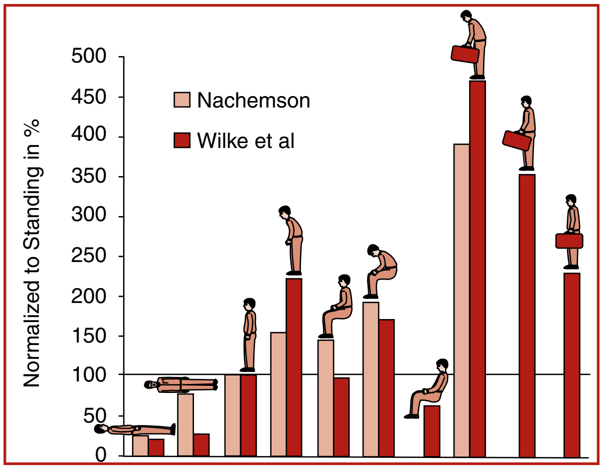


Figure 13–33 Interdiscal pressures in different sitting and standing positions. *(From Wilke H-J, Neef P, Caimi M, et al: New in vivo measurements of pressures in the intervertebral disc in daily life. Spine 24:755, 1999. Reprinted with permission from Lippincott Williams & Wilkins.)*

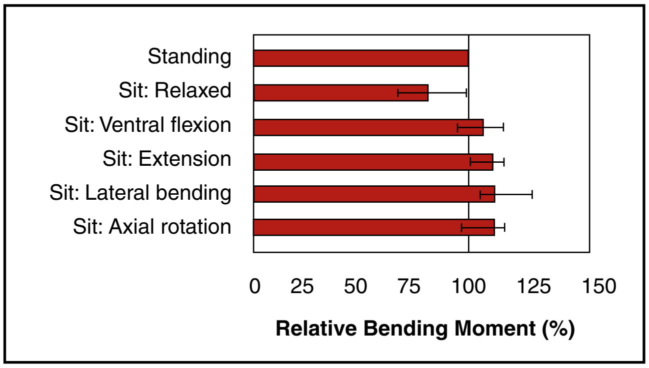


Figure 13–34 A comparison of average relative bending moments in internal fixation devices from 10 patients in different sitting positions. Values are related to the corresponding value for standing. Note that bending moment in relaxed sitting is less than the bending moment in standing, but the erect sitting (shown as extension) bending moment is greater than in standing. *(From Rohlmann A, Graichen F, Bergmann G: Loads on an internal spinal fixation device during physical therapy. Phys Ther 82:49, 2002. Reprinted with permission from the American Physical Therapy Association.)*

work in a sitting position.[120] In another study of spinal shrinkage, van Dieen and colleagues[119] found that a larger stature gain occurred when a person spent 2 hours working in dynamic chairs than in chairs with fixed seats and backs. Although dynamic chairs may be more desirable than fixed chairs, the task being performed had greater effects on spinal stature than did the type of chair.

Very few studies have investigated either the contribution of soft tissue deformation to measurements of total height loss or the shrinkage of the cervical spine. In an investigation of the soft tissue contribution to total seated height, one group of researchers found that deformation of muscle and fat below the sacrum contributed 28% to 30% at 5 and 10 minutes of loading, respectively. Soft tissue deformation always contributed a small amount to total height loss, but early in the loading cycle, height loss mostly occurred in the spine, whereas at the end of the loading cycle, the loss was a combination of spine and soft tissue.[121] Cervical spine shrinkage was investigated after 1 hour of television watching in a sitting position in the following three different head/neck positions: neutral, 20° of flexion, and 40° of flexion. The neutral head position had no effect on spinal shrinkage, in contrast to the head held at a 40° angle, when the most cervical shrinkage (approximately 1 mm) occurred in 1 hour.[122]

After a literature review, Harrison and colleagues[105] concluded that the lowest lumbar interdiscal pressures and lowest electromyographic readings are produced by seat back inclinations between 110° and 130°, combined with a lumbar support that protrudes 5 cm from the back of the seat back and a posterior seat inclination of 5°. The use of armrests produces further reductions in lumbar interdiscal pressures.[105]

Seat Interface Pressures

Pressure is force per unit area and is measured in pascals (Pa or N/m^2 [pounds per square inch]). The pressure caused by contact forces between the person's body and the seat is referred to as the **seat interface pressure.** Pressure mapping techniques using sensor-containing mats that can be placed on the seat of a chair are used to measure average and maximum seat interface pressures (Fig. 13–35A, B). **Average seat interface pressure** is the mean of pressure sensor values, and the **maximum seat interface pressure** is the highest individual sensor value.[123]

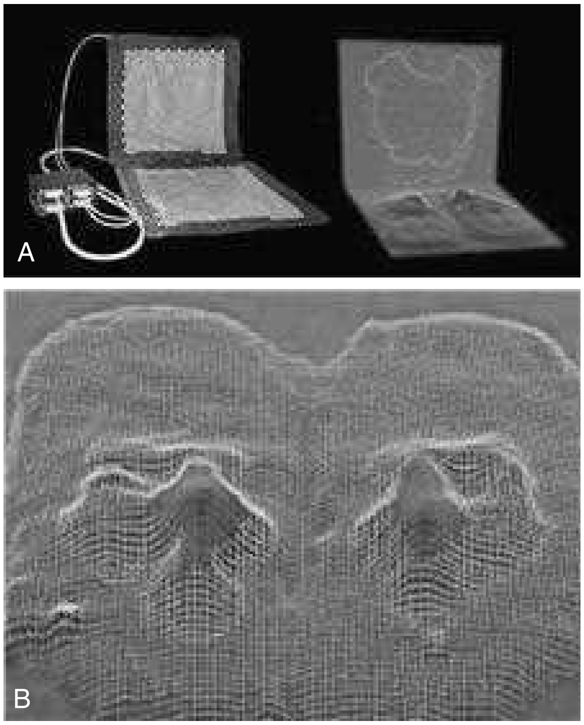


Figure 13–35 **A.** Tactilus seat pad sensor used for mapping seat pressures. **B.** Three-dimensional image of the pressure distribution, which shows where highest pressures are located. *(Courtesy of Sensor Products, Inc., 188 Rt. 10, Suite 307, East Hanover, NJ 07936.)*

Individuals with physical disabilities such as myelomeningocele and paraplegia have significantly higher seat interface pressures than do people without such disabilities.[124–126] Linder-Ganz[126] conducted a study using magnetic resonance imaging and finite element analysis in which six paraplegic wheelchair users were found to have significantly less muscle thickness under the ischial tuberosities compared to six healthy control subjects. The wheelchair users also had higher tension, compression, and shear strains in the gluteus muscles compared to the healthy subjects, quite possibly due in part to their reduced muscle thickness. Kernozek and colleagues studied peak interface pressures in a group of 75 elderly persons with different body mass indices (BMIs). Peak seat interface pressures were found to be highest in the thin elderly persons (those with the lowest BMI), who had the least amount of soft tissue over the ischiae.[127] These individuals probably had a smaller contact area with more concentration of pressure than did individuals with a greater body mass with increased surface contact area and better pressure distribution.

According to Regan, seat interface pressure has been found to be a good indicator of subcutaneous stress (with the latter being higher than the former).[128] However, it is unlikely that seat interface measurements alone can be used either to predict or prevent **pressure sores** (see Concept Cornerstone 13-4). Markshous recommends that interface pressure measurements should be used in conjunction with other measures such as transcutaneous partial pressures of oxygen and carbon dioxide measured from the buttocks overlying the ischial tuberosities. These measures provide evidence of the state of tissue perfusion deterioration and recovery.[132]

Concept Cornerstone **13-4**

Pressure Sores (Pressure Ulcers)

Superficial pressure sores initially affect the layers of skin near epidermal tissue and may progress downward into the subdermal layers. They are caused by frictional and shear forces, often in the presence of moisture and heat[132,133,126] Superficial pressure sores are usually considered reversible.

Deep pressure sores, termed “deep tissue injury” by the U.S. National Pressure Ulcer Advisory Council in 2005, initially affect subdermal tissue and originate in muscle underlying bony prominences and progress upward and through the epidermal tissue.[129,130] The deep tissue injuries are caused by compressive stress that interferes with blood flow, leading to local tissue ischemia. This ischemia is associated with failure of nutritive capillaries and leads to widespread tissue necrosis. Sustained elevated pressure leads to impaired lymphatic and venous circulation. The lack of a pain alarm system in spinal cord–injured patients prevents patients from recognizing when an area on the buttocks or heels becomes painful. Therefore, ischemia may be prolonged and eventually cause irreversible ischemic necrosis.[130] As the deep tissue injury progresses outward to the surface of the skin, fascia and subcutaneous tissues undergo necrosis as well.

CASE APPLICATION

Need to Minimize Seat Interface Pressure

case 13–11

Minimizing seat interface pressure is an extremely important consideration for our patient, Dave, who may have to spend a great deal of his time in a wheelchair. A decrease in seat interface pressures reduces the risk of developing **superficial pressure sores,** which are caused by unrelieved pressure, friction, and shearing forces. The greater danger, however, is the development of a deep tissue injury when soft tissue is compressed between the seat and bony prominences (e.g., the ischial spines). Dave is at high risk because he has lost muscle bulk in his buttocks as a result of the paralysis affecting his hip extensor muscles and the long periods of sitting in a wheelchair.

Effects of Changes in Body Posture

Changes in the posture of the body such as forward and lateral trunk flexion can be effective means of reducing seat interface pressures in individuals like our patient Dave who must spend long periods of time in a wheelchair. Hobson[125] compared seat interface pressure and shear in 10 healthy subjects and in 12 persons with spinal cord injuries. The individuals with spinal cord injuries had maximum seat interface pressures that, depending on which of the eight positions were assumed in the wheelchair, were 6% to 46% higher than the pressures found in the healthy group. Maximum seat interface pressures could be reduced from neutral position values by 9% when the trunk was flexed forward to 50° and reduced on the unweighted side by 30% to 40% when the trunk was laterally flexed to 15° (Fig. 13–36).

CASE APPLICATION

Pressure-Reducing Activities

case 13–12

The performance of seat interface pressure-reducing activities is an essential activity for our patient in order to relieve pressure and thus allow for tissue perfusion. Dave must be able to maintain stability while performing activities to alleviate pressure and be able to develop sufficient upper extremity strength to elevate his body vertically by performing **push-ups** on the armrests of his wheelchair. He needs to understand why these activities are important so that he will be motivated to perform them on a continual basis while he is in the wheelchair.

Effects of Alterations in the Position of the Chair

Alterations in the angulation of the chair’s back rest in combination with footrest and seat inclinations are other methods utilized to reduce seat interface pressure.

Figure 13–36 The patient is able to relieve interface pressure by leaning to the side. Leaning is recommended every few minutes. *(From O'Sullivan SB, Schmitz TJ: Physical Rehabilitation: Assessment and Treatment (ed.5). Philadelphia: FA Davis, 2007. With permission.)*

Continuing Exploration 13-8:

Comparison of Effectiveness of Wheelchair Push-Ups and the iPUP System

In a study involving paraplegic, tetraplegic, and normal individuals, Makhsous[132] compared the tissue relief and recovery effectiveness of wheelchair push-ups with an intelligent pressure ulcer prevention (iPUP) seating system developed at Northwestern University's Sitting Biomechanics Laboratory. The iPUP system is a pneumatic system consisting of a seat cushion, lumbar sacral support, pelvic stabilizer, and trunk support. The iPUP system's effects on tissue perfusion were compared with the effects using standard wheelchair push-ups. The iPUP system alternated every 10 minutes, whereas the wheelchair push-ups were performed every 20 minutes. Transcutaneous partial pressures of oxygen and carbon dioxide were measured over the ischial tuberosity, and interface pressures were measured at the seat back and buttocks. The researchers found that the wheelchair push-ups performed every 20 minutes provided pressure relief for about 49 seconds, but according to the results of the study, the amount of pressure relief time needed for full recovery of tissue perfusion is in the range of 200 to 300 seconds. The iPUP sytem, which drops the back of seat and transfers pressure to the thighs, was activated every 10 minutes and was successful in reducing pressure and for providing both tissue perfusion and recovery.[132] It would be interesting to see what the results would have been if the wheelchair push-ups were performed every 10 minutes.

Continuing Exploration 13-9:

Positioning to Reduce Seat Interface Pressure

Reclining the backrest posteriorly by 30° from 0° has been found to significantly reduce average seat interface pressure (but not maximum seat interface pressure). Supporting a person's feet on blocks of wood to produce 90° of flexion at the hips, knees, and ankles caused an increase in the average seat interface pressure.[123] In another study, the maximum seat interface pressure was observed to be significantly lower when sitting with a 0° backrest inclination when the feet were on the floor rather than when legs were supported on a rest.[134] Hobson[125] found that a recline of the back rest to 120° reduced the neutral backrest position values by 12%. Full body tilt to 20° reduced seat interface pressure values by 11%.

Also, cushions of various compositions and depths are used to reduce seat interface pressures. Materials used in the composition of cushions include synthetic materials, air, water, and gels of various kinds. Cushion thicknesses up to 8 cm have been found to be successful in reducing maximum subcutaneous stress inferior to the ischial tuberosity, but increasing the thickness beyond 8 cm failed to cause an additional decrease in seat interface pressure.[128] The use of a contoured foam cushion may be preferable to a flat foam cushion according to a Finite Element model that demonstrated that the former was more successful than the latter in reducing both internal and interface peak pressures.[133]

CASE APPLICATION

Cushion Selection

case 13–13

The selection of an appropriate cushion for Dave's wheelchair will be extremely important, and different materials will need to be tried. Ideally, the seat interface pressure should be mapped in order to customize his seating.

ANALYSIS OF LYING POSTURES

Interdiscal Pressures

In general, interdiscal pressures are less in lying postures than in standing and sitting postures. Wilke and colleagues[118] measured interdiscal pressures over a 24-hour period from a pressure transducer implanted in the nucleus pulposus of the nondegenerated disc between L4 and L5 of a 45-year-old healthy man. Interdiscal pressures in supine lying (0.10 MPa) were less than in either lying prone (0.11 MPa) or lying on the side (0.12 MPa), and in all of these postures the interdiscal pressure was less than in sitting and standing postures. Lying prone with the back extended and supported on one's elbows had the largest interdiscal pressure (0.25 MPa) among the lying postures tested and was only slightly less than in slouched sitting (0.27 MPa). Rohlmann and associates conducted a study of the bending moments on spinal fixation devices in 10 patients. Movements in the lying posture such as lifting an extended arm or leg in the supine and prone positions did not raise the bending moments above bending moments in standing (Fig. 13–37). However, when the patients raised both extended legs in the supine position, peak bending moments exceeded the moments in the standing posture.[117]

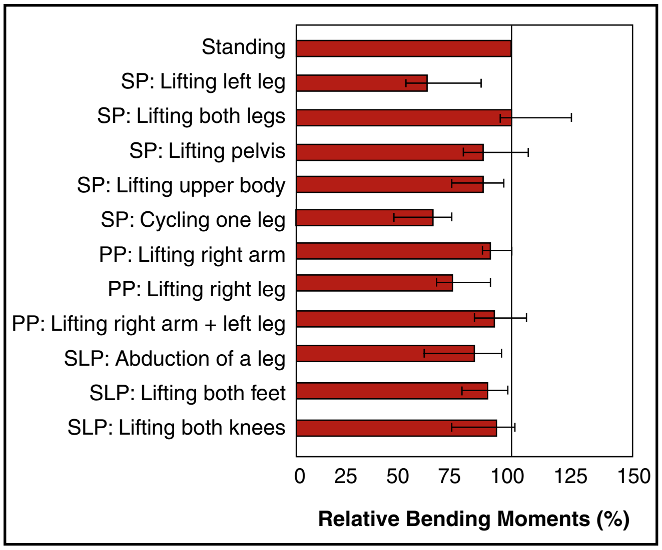


Figure 13–37 Bending moments that occurred when body parts were moved as they might be in an exercise program when the body is positioned in the supine lying position (SP), prone position (PP), and side-lying position (SLP).The bending moments are compared with the moments in standing. *(From Rohlman, A, Graichen, F, Bergmann, G: Loads on an internal spinal fixation device during physical therapy. Physical Therapy 82:48, 2002. Reprinted with permission from the American Physical Therapy Association.)*

Surface Interface Pressures

A uniform pressure distribution over the entire available surface is desirable to prevent sections of increased pressure over certain areas. Examples of some pressure-reducing mattress surfaces include foam, air, gas, water, and gel. Other pressure-relieving surfaces include movable surfaces, usually powered by a motor or pump, which can alternatively inflate and deflate.[135]

CASE APPLICATION

Risk of Pressure Sore Development

case 13–14

We need to be concerned about lying postures because of the danger of developing pressure sores. Dave is at risk for skin breakdown because his lower extremity muscles may have atrophied, with an accompanying loss of protective soft tissue over bony prominences. In the supine lying posture, the areas at risk are the backs of his heels and head, his scapulae, elbows, lower vertebral spine, and the sacrum. In the side-lying posture, the areas at risk are his ears, shoulders, medial aspects of the knees, lateral malleoli, and the greater tochanter of the femur and head of the fibula.

EFFECTS OF AGE, AGE AND GENDER, PREGNANCY, OCCUPATION, AND RECREATION ON POSTURE

Age

Infants and Children

Postural control in infants develops progressively during the first year of life, from control of the head to control of the body in a sitting posture and then to control of the body in a standing posture. Stability in a posture, or the ability to fix and hold a posture in relation to gravity, must be accomplished before the child is able to move within a posture. The child learns to maintain a certain posture, usually through co-contraction of antagonist and agonist muscles around a joint, and then is able to move in and out of the posture (sitting to standing and standing to sitting). Once stability is established, the child proceeds to controlled mobility and skill. Controlled mobility refers to the ability to move within the posture—for example, weight shifting in the standing posture. Skill refers to performance of activities such as walking, running, and hopping, which are dynamic postural activities.[136]

According to Woollacott,[137] by the time a child reaches 7 to 10 years of age, postural responses to platform perturbations are less variable and also comparable with those of adults in patterns of muscle activity and timing of responses. Responses of children younger than 7 years of age included greater coactivation of agonists and antagonists and slower response times for muscle activation than in either adults or older children.[137] Nolan and colleagues[138] investigated gender and age differences in CoP movement (postural sway) in 90 boys and 90 girls ages 9 to 16. In both the eyes open and eyes closed conditions, boys exhibited greater CoP movement (sway) than girls at 9 to 10 years old. Also antereroposterior sway velocity and greater sway path length were greater in 9- to 10-year-old boys than in the older groups of boys in the eyes closed condition. Very little age difference was noted in girls. The authors concluded that some aspects of postural control are still developing after age 9 or 10 in boys. Newell and colleagues[139] investigated CoP motion in different age groups from 3 years to 92 years of age. The young adult group of students in their 20s had the least amount of movement of the CoP; the individuals in the youngest and oldest groups had the greatest amount of CoP motion.

The erect standing posture in infancy and early childhood differs somewhat from postural alignment in adults, but by the time a child reaches the age of 10 or 11 years postural alignment in the erect standing position should be similar to adult alignment.[140] However, poor postural alignment in a 7- or 8-year-old child can be recognized because it is similar to poor postural alignment in adults. For example, the poor posture in these children may include forward head, kyphosis, lordosis, and hyperextended knees[41] (Fig. 13–38). La Fond, in a study of the standing posture of 1,084 children ages 4 to 12, found a significant linear trend between children's age and anterior translation of the head, shoulders, pelvis, and knees. A significant gender effect was also found for all variables except sagittal head translation. The authors concluded that the postural alignment of children relative to the vertical changes considerably between 4 and 12 years of age.[141]

Age and Gender

The following two studies investigated the effects of age and gender on the thoracic and lumbar curves. Widhe[142] monitored 90 Swedish boys and girls over a 10-year period, examining them first at 5 to 6 years of age and again at 15 to 16 years of age. Between the first and second measurements, both thoracic kyphosis and lumbar lordosis increased by 6° (Table 13–8). Sagittal mobility decreased in the thoracic region by 27° (9° in flexion and 18° in extension). Lumbar flexion decreased by 9° and extension by 5°. In the other study, 847 Finnish boys and girls were examined annually from ages 10.8 years to 13.8 years.The normal thoracic kyphosis was greater and the normal lumbar lordosis less in boys than in girls both at the beginning and at each annual examination.[143] Mean thoracic kyphosis increased and mean lumbar lordosis decreased with increasing age. Despite wide variations, thoracic kyphosis was between 20° and 40° and lordosis was between 20° and 50° in both genders.[143] The wide physiological variation in spinal posture already evident at puberty was also found in young Finnish adults at the age of 22 years; however, the trend of increasing mean male kyphosis continued throughout adolesence into young adulthood. In males, the degree of kyphosis at age 14 predicted the development of hyperkyphosis at age 22, which was found to be 10 times more prevalent in males than in females. In females at age 22, the mean lumbar lordosis was significantly more pronounced than in males.[145]

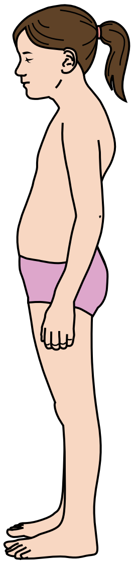


Figure 13–38 The forward head and kyphosis in a child are easily recognized as postural faults comparable to adult postural deviations in alignment.

Postural balance was investigated in a Finnish study involving a random sample of 2,726 males and 3,277 females between the ages of 30 and 80 plus years. The results showed that deterioration of postural control mechanisms began in the 40- to 49-year-old group who already performed more poorly than the 30- to 39-year-old group in four test conditions: standing with eyes open, eyes closed, one foot next to the arch of the other foot, and one foot in front of the other. Deterioration of postural control accelerated at about 60 years and the decline with age was more prominent in males. The authors recomended that due to gender differences separate gender normative values are needed.[146]

Elderly

Postural alignment in elderly people may show a more flexed posture than in the young adult; however, many elderly individuals in their 70s and 80s still demonstrate a close-to-optimal posture. Hardacker and colleagues found that cervical lordosis increased with increasing age[51] (Fig. 13–39). Hammerberg and Wood[144] in a study

Table 13–8 Age Variations in Spinal Curves in Standing Posture: Values in Degrees

Authors	Widhe[142]*		Vendantam et al[58]†	Gelb et al[56]	Hammerberg and Wood[144]
Ages	5–6 yr *n* = 90	15–16 yr *n* = 90	10–18 yr *n* = 88	40–49 yr *n* = 27	70–85 yr *n* = 50
			VALUE		
	Mean (SD)	*Mean (SD)*	*Mean (SD)*	*Mean (SD)*	*Mean (Range)*
Thoracic	29 (9)	35 (8)	38 (10)		52 (29–79)
Lumbar	−31 (8)	−38 (7)	−64 (10)	−68 (11)	−57 (−96 to −20)

*Widhe measured the thoracic curve from a point between the spinous processes of T2 and T3 to T12. The lumbar curve was measured from a point between T11 and T12 to a point between S1 and S2. The same 90 subjects were measured at different ages.

†Vendantam et al measured the thoracic curve from T3 to T12 and the lumbar curve from T12 to S1.

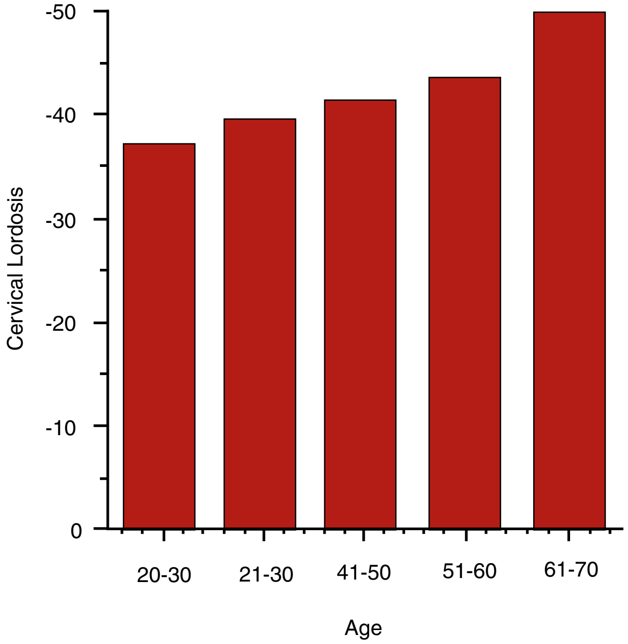


Figure 13–39 The graph indicates that for the 100 volunteers in the study, the mean cervical lordosis increased with increasing age. *(From Hardacker JW, Shuford RF, Capicotto PN, et al: Radiographic standing cervical segmental alignment in adult volunteers without neck symptoms. Spine 22:1477, 1997. Reprinted with permission from Lippincott Williams & Wilkins.)*

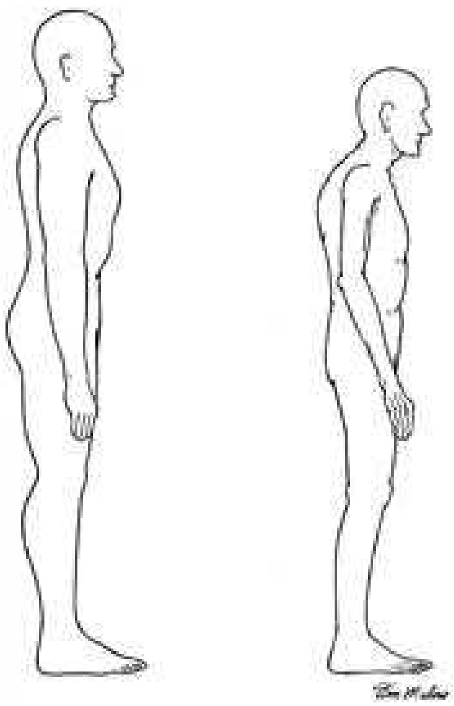


Figure 13–40 Changes in posture as a result of aging.

of the radiographic profiles of 50 elderly individuals 70 to 85 years of age showed an average kyphosis angle of 52°, with a range of 29° to 79°, and an anterior position of C7. The LoG passed, on average, 40 mm anterior to the posterosuperior corner of S1. Gelb and colleagues[56] in a study of 100 middle-aged and older volunteers (average age of 57 years) noted that as age increased, the LoG was located more anteriorly with a loss of lumbar lordosis and an increase in thoracic and thoracolumbar kyphosis. However, the mean values of 34° for thoracic kyphosis and 64° for lumbar lordosis values fell within normally accepted ranges for younger populations. No correlations were found between age and kyphosis either in the thoracic region or at the thoracolumbar junction. Only the loss of lumbar lordosis at the proximal levels showed the strongest correlation with age.[56] The flexed posture observed in some elderly persons is probably due to a number of factors, some of which may relate to aging processes (Fig. 13–40). Conditions such as osteoporosis may affect posture in elderly persons and lead to kyphosis. In kyphosis, the anterior trunk flexor muscles shorten as the posteriorly located trunk extensors lengthen. Teramoto and coworkers[147] evaluated the effects of kyphosis in subjects from 20 to 90 years of age. The authors found that the extent of kyphosis significantly decreased lung volume and maximal inspiratory pressure in the elderly subjects.

The range of motion at the knees, hips, ankles, and trunk may be restricted because of muscle shortening and disuse atrophy. Furthermore, as voluntary postural response times in elderly people appear to be longer than in young people, elderly persons may elect to stand with a wide BoS to have a margin of safety. Postural responses of older adults, aged 61 to 78 years, to platform perturbations show differences in timing and amplitude and include greater coactivation of antagonist and agonist muscles in comparison with younger subjects, aged 19 to 38 years. Iverson and associates,[148] who tested noninstitutionalized men 60 to 90 years of age on two types of balance tests that involved one-legged stance, found that balance time and torque production decreased significantly with age. In some of the tests, the authors found that torque production was a significant predictor of balance time; that is, the greater the torque production, the longer the balance time. These authors also found that men who exercised five to six times per week had greater torque production than did men who exercised less frequently. This finding suggests that high levels of fitness and activity may have beneficial effects on the aging person's ability to perform one-legged balancing activities that are needed for activities of daily living such as walking.[148]

CASE APPLICATION

Osteoporosis Risk

case 13–15

Osteoporosis may be a problem for Dave even before any significant aging because of the lack of weight-bearing on his lower extremities. However, if Dave happens to enter one of the newer rehabilitation programs that employ treadmill walking for spinal cord–injured patients, osteoporosis may

Continued

not be as much of a problem. It even may be possible that Dave might be able to gain back muscle strength in his legs and be able to walk with minimal assistance.[149,150]

Another aging consideration for Dave will be a decrease in the thickness of the skin that occurs with normal aging, including reductions in collagen, elastin, proteoglycan, and water content. Therefore, Dave will have to be extremely careful with his skin as he ages, because his skin is already at risk.

Pregnancy

Normal pregnancies are accompanied by weight gain, an increase in weight distribution in the breasts and abdomen, and softening of the ligamentous and connective tissue. The location of the woman's CoG changes because of the increase in weight and its distribution anteriorly. Consequently, postural changes in pregnancy include an increase in the lordotic curves in the cervical and lumbar areas of the vertebral column, protraction of the shoulder girdle, and hyperextension of the knees. Franklin and Conner-Kerr[151] compared postural evaluations of 12 pregnant volunteers in their first trimester with evaluations of the same women in their third trimester. These investigators found changes in lumbar angle, head position, and anterior pelvic tilt. The lumbar angle increased by an average of 5.9°, the anterior pelvic tilt increased by an average of 4°, and the head became more posterior as pregnancy progressed from the first through the third trimesters.[152] These changes in posture represent adaptations that help to maintain the CoM centered over the BoS. Softening of ligamentous and connective tissues, especially in the pelvis, sacroiliac joints, pubic symphyses, and abdomen, change the support and protection offered by these structures and predispose pregnant women to strains in supporting structures.[152] Many women experience backache during pregnancy, and all of the women in the study by Franklin and Conner-Kerr complained of backache.[151]

Continuing Exploration 13-10:

Effects of Pregnancy on Sitting and Standing Postures

Gilleard and coworkers,[153] however, did not find any significant effects of pregnancy on upper body posture of nine pregnant women during sitting and quiet standing in films taken at intervals during gestation and at 8 weeks postpartum. A flattening of the thoracolumbar curve was observed in some subjects in the sitting posture as pregnancy progressed. No significant differences were found between the postpartum group and a control group in the cervical or thoracic spines, but the postpartum group stood with more thoracolumbar flexion than did the control group.

Occupation and Recreation

Each particular occupational and recreational activity has unique postures and injuries associated with these postures. Bricklayers, surgeons, carpenters, and cashiers assume and perform tasks in standing postures for a majority of the working day. Others, such as secretaries, accountants, computer operators, and receptionists, assume sitting postures for a large proportion of the day. Performing artists often assume asymmetrical postures while playing a musical instrument, dancing, or acting. Running, jogging, and long-distance walking are dynamic postures with which very specific injuries are associated.

Different sitting postures and their effects on intradiscal pressures in the lumbar spine have been analyzed.[154] Wheelchair postures and the effects of different degrees of anterior posterior and lateral pelvic tilt on the vertebral column and trunk muscle activity in sitting postures in selected work activities also have been investigated.[155] A large portion of the research suggests that many back problems are preventable because they result from mechanical stresses produced by prolonged static postures in the forward stooping or sitting positions and the repeated lifting of heavy loads.

Many of the injuries sustained during both occupational and recreational activities belong to the category of "overuse injuries." This type of injury is caused by repetitive stress that exceeds the physiological limits of the tissues. Muscles, ligaments, and tendons are especially vulnerable to the effects of repetitive tensile forces, whereas bones and cartilage are susceptible to injury from the application of excessive compressive forces. A random sample of professional musicians in New York revealed that violin, piano, cello, and bass players were frequently affected by back and neck problems.[156] In a larger study involving 485 musicians, the authors found that 64% had painful overuse syndromes. The majority of problems were associated with the musculotendinous unit, and others involved bones, joints, bursae, and muscle. String players experienced shoulder and neck problems caused by the maintenance of abnormal head and neck positions, whereas flute players had shoulder problems associated with maintaining an externally rotated shoulder position that has to be assumed for prolonged periods during performances and practices.[157] Peripheral nerve disorders, including thoracic outlet syndrome, ulnar neuropathy at the elbow, and carpal tunnel syndrome, also appear to be common playing-related disorders.[158,159]

CASE APPLICATION

Succeptibility to Overuse Injuries

case 13–16

Dave will be susceptible to the same type of upper extremity overuse injuries that are incurred by any sitting worker performing a repetitive task such as data entry. If he chooses to use a hand-propelled wheelchair as his main method of transportation, he could be at risk for shoulder, elbow, and wrist overuse injuries. If he is able to use crutches, he may incur shoulder and wrist overuse injuries.

Each occupational and recreational activity requires a detailed biomechanical analysis of the specific postures involved to determine how abnormal and excessive stresses can be relieved. Sometimes the analysis involves not only a person's posture but also features of the worksite such as chair or table height, weight of objects to be lifted or carried, and weight and shape of a musical instrument or tool. Intervention may involve a combination of modifications of the environment, adaptations of the instrument or tools, and modifications of posture. For example, raising or lowereing the height of the computer monitor has been found to change the amount of muscle activity in the neck and back muscles in children aged 10 to 12[160,161] and in adults aged 20 to 40.[162]

SUMMARY

In this chapter, we introduced the basic aspects of postural control and analyzed normal postural alignment in the erect standing position. Also, we discussed some of the internal and external forces affecting sitting and lying postures primarily in relation to how they may affect our patient, Dave. The kinematic and kinetic information provided in this chapter and previous chapters forms the basis for the analysis of static posture as well as the dynamic posture of gait.

CASE APPLICATION

Future Progression From Static to Dynamic Postures

case 13–17

We have identified some of the problems that may face our patient, Dave, in static postures. He will now progress from static sitting and standing postures to walking. Because he is a former athlete, we hope that he may want to participate in activities such as wheelchair basketball or in wheelchair marathons. However, the treatment of spinal cord injuries is changing dramatically, and Dave may not be confined to a wheelchair. Advances in treatment are helping some individuals regain sufficient muscle strength to be able to walk without braces, and we hope that Dave will be able to benefit from these new treatment programs.[149,150]

STUDY QUESTIONS

1. What is a "sway envelope"?
2. Is quadriceps muscle activity necessary to maintain knee extension in static erect stance? Explain your answer.
3. Is activity of the abdominal muscles necessary to keep the pelvis level in static standing posture? Explain your answer.
4. How does the lumbar curve change from standing to sitting, and what effect does the change have on interdiscal pressures?
5. In which areas of the vertebral column would you expect to find the most stress in the erect standing posture? Why?
6. For the erect standing posture, identify the type of stresses that would be affecting the following structures: apophyseal joints in the lumbar region, apophyseal joint capsules in the thoracic region, anulus fibrosus in L5 to S1, anterior longitudinal ligament in the thoracic region, and the sacroiliac joints.
7. What effect might tight hamstrings have on the alignment of the following structures during erect stance: pelvis, lumbosacral angle, hip joint, knee joint, and the lumbar region of the vertebral column?
8. How would you describe a typical idiopathic lateral curvature of the vertebral column?
9. Describe the moments that would be acting at all body segments as a result of an unexpected forward movement of a supporting surface in a fixed support strategy. Describe the muscle activity that would be necessary to bring the body's LoG over the CoP.
10. Identify the changes in body segments that are commonly used in scoliosis screening programs.
11. Explain how our patient manages to stand when he lacks lower extremity musculature and elements of postural control.
12. In a change in support postural strategy, explain how postural responses to perturbations of the erect standing posture in elderly persons compare with responses of young adults who are in their 20s.
13. Compare a flexed lumbar spine posture with an extended posture in terms of the nutrition of the discs and stresses on ligaments and joint structures.
14. What is the relationship between the GRFV, LoG, and CoM in the erect static posture?
15. Explain how a hallux valgus deformity develops.

Continued

STUDY QUESTIONS—cont'd

16. Describe the effects of a forward head posture on the zygapophyseal joints and capsules, intervertebral discs, vertebral column ligaments, and muscles.
17. Explain the possible effects on body structures in a young person with a double major curve (right thoracic, left lumbar).
18. Explain how changes in body position affect seat interface pressures.
19. Compare interdiscal pressures in erect standing with erect, slumped, and relaxed sitting.
20. Explain the difference between the development of a superficial pressure sore and a deep tissue injury.

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Chapter 14

Gait

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INTRODUCTION

Gait Analysis

In human locomotion (ambulation, gait), the reader is given the opportunity to discover how individual joints and muscles function in an integrated manner both to maintain upright posture and to produce motion of the body as a whole. Knowledge of the kinematics and kinetics of normal ambulation provides the reader with a foundation for analyzing, identifying, and correcting abnormalities in gait.

Walking is probably the most comprehensively studied of all human movements, and the variety of technologies, coupled with the diversity of disciplinary perspectives, has produced a complex and sometimes daunting literature. The biomechanical requirements of the movements that explain gait are logical and easily understood if the detail is not permitted to cloud comprehension. The purpose of this chapter is to provide this comprehension of gait that will serve as the foundation for analysis of normal walking and of gait deviations.

In early gait analysis, investigators used cinematographic film and until about 20 years ago, sophisticated analysis required frame-by-frame hand-digitizing of markers that had been placed on body landmarks. These data were coupled with knowledge of the center of pressure (CoP) of the foot-floor forces derived from a force platform to give complete, if simplified, kinetic information. This is referred to as the **inverse dynamic approach** with link segment mechanics. Electrogoniometers fastened to joints were also commonly used to describe joint motion and still have applications.[1] Similarly, electromyography (EMG) has been used for many decades, although the expectation that it would be possible to convert those signals to force values in simple, useful ways has not been fulfilled. However, the past two decades have witnessed an explosion of technical advancements in motion analysis that offer the ability to collect and process large amounts of data. As with the development of any science, the knowledge available far exceeds its current applications.

A modern gait laboratory (Fig. 14–1) includes some kind of motion analysis system that employs precise marker locations that are subsequently used to model a several-segment body with joint centers and centers of mass. Also included are one or more force platforms that provide simultaneous foot-floor forces. EMG systems provide simultaneous information from surface electrodes, or, sometimes, indwelling electrodes. An excellent and engaging report of the evolution of clinical gait analysis, including motion analysis and EMG, can be found in Sutherland's articles.[2,3]

Human locomotion, or gait, may be described as a translatory progression of the body as a whole, produced by coordinated, rotatory movements of body segments.[1] The alternating movements of the lower extremities essentially support and carry along the head, arms, and trunk. The head, arms, and trunk constitute about 75% of total body weight, with the head and arms contributing about 25% of total body weight and the trunk contributing the remaining 50%.[4] To make full use of this chapter you should review the relevant biomechanics and anatomy. You must understand the basic biomechanical concepts presented in Chapter 1, the joint motion of major joints of the lower limbs, and have a thorough knowledge of the major muscle groups of the lower limbs and their actions.

Figure 14-1 A modern gait laboratory.

Major Tasks of Gait

To understand gait, let us first identify the fundamental purposes. Winter[5] proposed the following five main tasks for walking gait:

1. Maintenance of support of the head, arms, and trunk, that is, preventing collapse of the lower limb
2. Maintenance of upright posture and balance of the body
3. Control of the foot trajectory to achieve safe ground clearance and a gentle heel or toe landing
4. Generation of mechanical energy to maintain the present forward velocity or to increase the forward velocity
5. Absorption of mechanical energy for shock absorption and stability or to decrease the forward velocity of the body

Maintaining balance and stability is clearly important during ambulation, and there is an increasing body of literature on what might be called sub-tasks of gait that are potentially destabilizing. These include gait initiation,[6] and termination,[7] stair-climbing,[8] turning,[9] obstacle crossing,[9–11] and negotiating a raised surface.[12] Gait initiation and termination and stair-climbing will be introduced in this chapter.

Phases of the Gait Cycle

Gait has been divided into a number of segments that make it possible to describe, understand, and analyze the events that are occurring. A **gait cycle** spans two successive events of the same limb, usually initial contact of the lower extremity with the supporting surface. During one gait cycle, each extremity passes through two major phases: a **stance phase,** when some part of the foot is in contact with the floor, which makes up about 60% of the gait cycle,[13] and a **swing phase,** when the foot is not in contact with the floor, which makes up the remaining 40%[13,14] (Fig. 14–2). There are two periods of **double support** occurring between the time one limb makes initial contact and the other one leaves the floor at toe-off. At a normal walking speed, each period of double support occupies about 11% of the gait cycle, which makes a total of approximately 22% for a full cycle.[15] The body is thus supported by only one limb for nearly 80% of the cycle. The approximate value of 10% for each double-support phase is usually assigned to each of the two double-support periods.

Stance phase is divided into subphases by a number of events that mark the start and end of the subphases. Figure 14–3 identifies the events delimiting the stance phase as **initial contact,** sometimes referred to as **heel contact** or **heel strike** and **toe-off.** The gait cycle is divided into percentiles that will be used to clarify events and phases. Values for normal walking appear in the figures.

Events in Stance Phase

1. *Initial contact* refers to the instant the foot of the leading extremity strikes the ground.[16] In normal gait, the heel is the point of contact, and the event referred to as *heel contact* or *heel strike.* The word *strike* is actually a misnomer inasmuch as the horizontal velocity reduces to about 0.4 m/sec and only 0.05 m/sec vertically.[17] In abnormal gait, it is possible for the whole foot or the toes, rather than the heel, to make initial contact with the ground.
2. *Foot flat* in normal gait occurs after initial contact at approximately 7% of the gait cycle. It is the first instant during stance when the foot is flat on the ground.
3. *Midstance* is the point at which the body weight is directly over the supporting lower extremity, usually about 30% of the gait cycle.

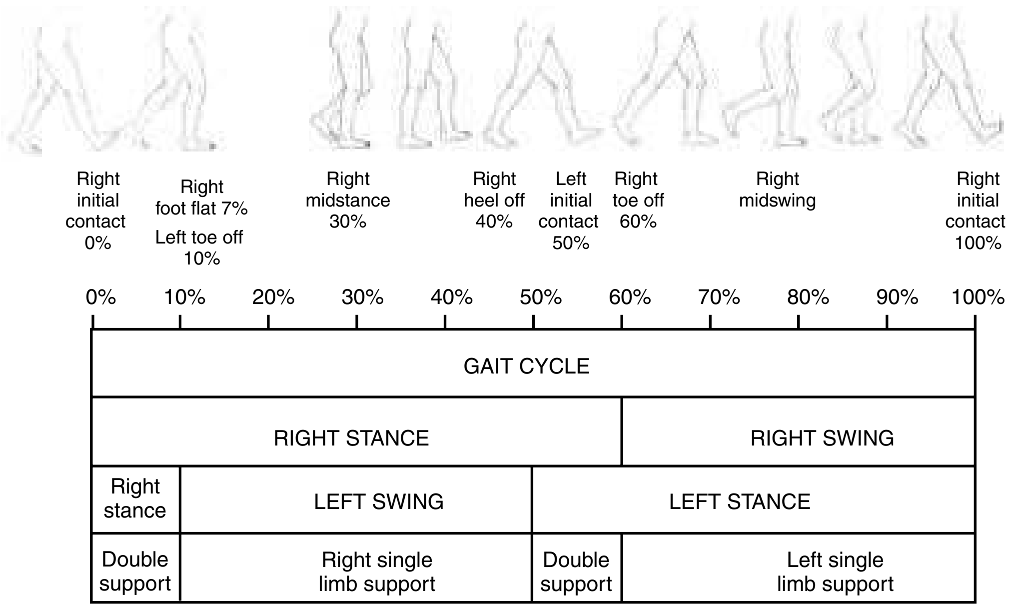

Figure 14-2 A gait cycle spans the period between initial contact of the reference extremity (right) and the successive contact of the same extremity. This figure shows the gait cycle with major events: stance and swing phases for each limb and periods of single and double support. The stance phase constitutes 60% of the gait cycle, and the swing phase constitutes 40% of the cycle at normal walking speeds. Increases or decreases in walking speeds alter the percentages of time spent in each phase.

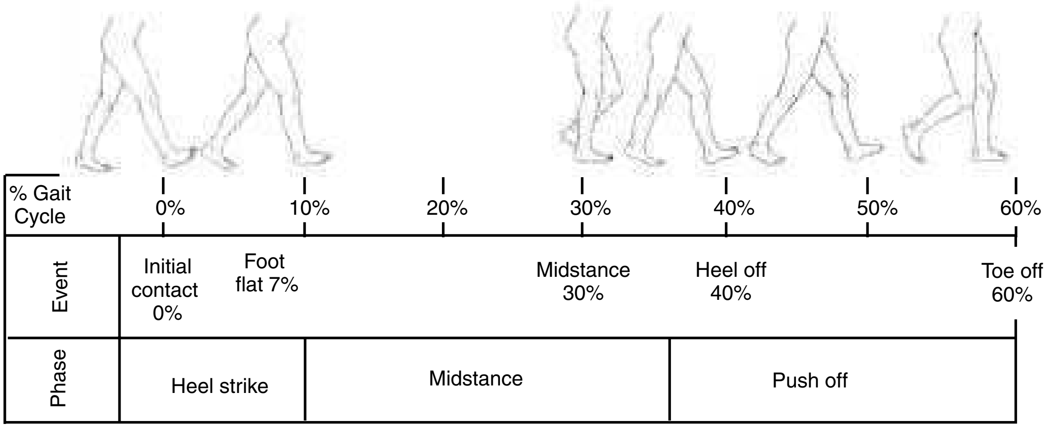

Figure 14-3 The stance phase of a gait cycle of the right lower limb. The events that delimit subphases are shown and expressed as percentages of full gait cycle: initial contact, foot flat, heel-off, midstance, and toe-off. Subphases are heel strike phase, midstance phase, and push-off phase.

4. *Heel-off* is the point at which the heel of the reference extremity leaves the ground, usually about 40% of the gait cycle.
5. *Toe-off* is the instant at which the toe of the foot leaves the ground, usually about 60% of the gait cycle.

Subphases of Stance Phase

1. *Heel strike phase* begins with initial contact and ends with foot flat and occupies only a small percentage of the gait cycle (see Fig. 14–3). Terminology originating in Rancho Los Amigos National Rehabilitation Center includes this phase in one called *weight acceptance*, or *loading response*, which begins at initial contact and ends when the contralateral extremity lifts off the ground at the end of the double-support phase[16] and occupies about 11% of the gait cycle.
2. *Midstance phase* begins with foot flat at 7% of the gait cycle and ends with heel-off at about 40% of the gait cycle.
3. *Push-off phase* begins with heel-off at about 40% of the gait cycle and ends with toe-off at about 60% of the gait cycle (see Fig. 14–3).

Sometimes authors refer to a *preswing phase*, which is terminology originating in Rancho Los Amigos National Rehabilitation Center.[16] Preswing refers to the last 10% of stance phase, beginning with initial contact of the contralateral foot (at 50% of the gait cycle) and ending with toe-off (at 60%).

Swing Phase

1. *Early swing phase* begins once the toe leaves the ground and continues until midswing, or the point at which the swinging extremity is directly under the body (Fig. 14–4). This phase is also referred to as *initial swing*, or the *acceleration phase*.
2. *Midswing* occurs approximately when the extremity passes directly beneath the body, or from the end of acceleration to the beginning of deceleration.
3. *Late swing* occurs after midswing when the limb is decelerating in preparation for heel strike. It is also known as *terminal swing*, or the *deceleration phase*.

14-1 Patient Case

Marlene Brown is a 63-year-old woman who sustained a stroke 15 days ago and shows weakness, or *hemiparesis*, of her right arm and leg. She has been in a rehabilitation unit for 10 days and is making good progress walking, although she is ambulating at only 0.20 m/sec. A value of about 1 m/sec would be typical for an able-bodied person of her age. She has weakness in several muscle groups of her lower limb, notably the ankle plantarflexors, ankle dorsiflexors, knee extensors, and hip flexors, with distal muscles more affected than proximal ones. Particularly troublesome is the inability to clear her foot during the swing phase of gait. She uses a cane with a large base (a four-point cane) in her unaffected hand for stability. As you read the Gait Terminology section, ask youself what differences from normal gait you might expect to see.

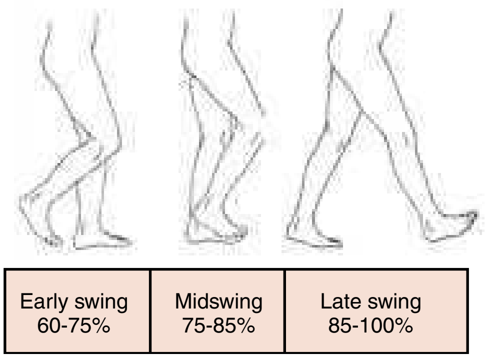

Figure 14-4 The swing phase of a gait cycle. Early swing phase is also known as initial swing or acceleration, and late swing is also known as terminal swing or deceleration.

GAIT TERMINOLOGY

Time and Distance Terms

Time and distance are two basic parameters of motion, and measurements of these variables provide a basic description of gait. **Temporal variables** include stance time, single-limb and double-support time, swing time, stride and step time, cadence, and speed. The **distance variables** include stride length, step length and width, and degree of toe-out. These variables, derived in classic research of over 30 years ago, provide essential quantitative information about a person's gait and should be included in any gait description.[16,18–21] Each variable may be affected by such factors as age, sex, height, size and shape of bony components, distribution of mass in body segments, joint mobility, muscle strength, type of clothing and footgear, habit, and psychological status. However, a discussion of all the factors affecting gait is beyond the scope of this text.

Stance time is the amount of time that elapses during the stance phase of one extremity in a gait cycle.

Single-support time is the amount of time that elapses during the period when only one extremity is on the supporting surface in a gait cycle.

Double-support time is the amount of time spent with both feet on the ground during one gait cycle. The percentage of time spent in double support may be increased in elderly persons and in those with balance disorders. The percentage of time spent in double support decreases as the speed of walking increases.

Stride length is the linear distance between two successive events that are accomplished by the *same* lower extremity during gait.[13] In general, stride length is determined by measuring the linear distance from the point of one heel strike of one lower extremity to the point of the next heel strike of the same extremity (Fig. 14–5). The length of one stride includes all of the events of one gait cycle. Stride length also may be measured by using other events of the same extremity, such as toe-off, but in normal gait, two successive heel strikes are usually used. A stride includes two steps, a right step and a left step. However, stride length is not always twice the length of a single step, because right and left steps may be unequal. Stride length varies greatly among

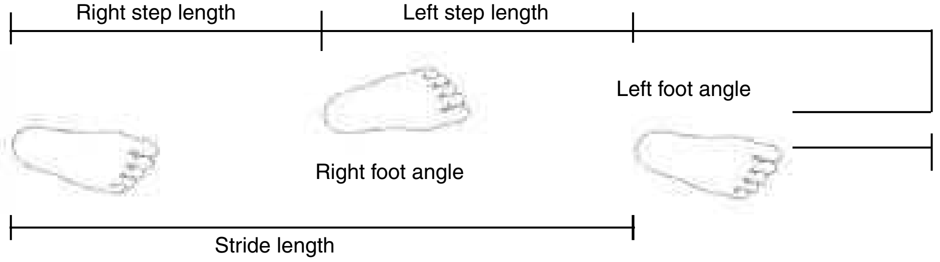

Figure 14-5 Stride length, step length, and width shown with foot angle placements. The midpoint of the heel is used as a point of reference for measuring step width.

individuals, because it is affected by leg length, height, age, sex, and other variables. Stride length can be normalized by dividing stride length by leg length or by total body height, so people of different sizes can be compared. Stride length usually decreases in elderly persons[14,15,22] and increases as the speed of gait increases.[23]

Stride duration refers to the amount of time it takes to accomplish one stride. Stride duration and gait cycle duration are synonymous. One stride, for a normal adult, lasts approximately 1 second.[24]

Step length is the linear distance between two successive points of contact of *opposite* extremities. It is usually measured from the heel strike of one extremity to the heel strike of the opposite extremity (see Fig. 14–5). A comparison of right and left step lengths will provide an indication of gait symmetry. The more equal the step lengths, the more symmetrical is the gait.

Step duration refers to the amount of time spent during a single step. Measurement usually is expressed as seconds per step. When there is weakness or pain in an extremity, step duration may be decreased on the affected side and increased on the unaffected (stronger) or less painful side.

Cadence is the *number* of steps taken by a person per unit of time. Cadence may be measured as the number of steps per second or per minute, but the latter is more common. A shorter step length will result in an increased cadence at any given velocity.[23] Lamoreaux found that when a person walks with a cadence between 80 and 120 steps per minute, cadence and stride length had a linear relationship.[13] As a person walks with increased cadence, the duration of the double-support period decreases. When the cadence of walking approaches 180 steps per minute, the period of double support disappears, and running commences. A step frequency or cadence of about 110 steps per minute can be considered as “typical” for adult men; a typical cadence for women is about 116 steps per minute.[4] Sometimes authors report values that refer to stride cadence, which is exactly half the step cadence.

Walking velocity is the rate of linear forward motion of the body, which can be measured in meters or centimeters per second, meters per minute, or miles per hour. Scientific literature favors meters per second. The term *velocity* implies that direction is specified, although this is frequently not included, and the more correct term **walking speed** should be used if direction is not reported. In instrumented gait analyses, walking velocity is used, inasmuch as the velocities of the segments involve specification of direction:

Walking velocity (meters/second) = distance walked (meters)/time (seconds)

Women tend to walk with shorter and faster steps than do men at the same velocity[23] due largely to height and leg length differences. Increases in velocity up to 120 steps per minute are brought about by increases in both cadence and stride length, but above 120 steps per minute, step length levels off, and speed increases are achieved with only cadence increases.

A person’s normal comfortable speed of gait may be referred to as **preferred, natural, self-selected,** or **free. Slow** and **fast** speeds of gait refer to speeds slower or faster than the person’s normal comfortable walking speed, designated in a variety of ways. There is a certain amount of variability in the way an individual elects to increase walking speed. Some individuals increase stride length to achieve a fast walking speed. Others increase cadence.

Step width, or width of the walking base, may be found by measuring the linear distance between the midpoint of the heel of one foot and the same point on the other foot (see Fig. 14–5). Step width has been found to increase when there is an increased demand for side-to-side stability, such as occurs in elderly persons and in small children. In toddlers and young children, the center of gravity is higher than in adults, and a wide base of support is necessary for stability. In the normal population, the mean width of the base of support is about 3.5 inches and varies within a range of 1 to 5 inches.

Degree of toe-out represents the angle of foot placement and may be found by measuring the angle formed by each foot’s line of progression and a line intersecting the center of the heel and the second toe. The angle for men normally is about 7° from the line of progression of each foot at free speed walking (see Fig. 14–5).[14] The degree of toe-out decreases as the speed of walking increases in normal men.[14]

Kinematic Terms

Kinematics is the term used to describe movements without considering the internal or external forces that caused the movements. These measures include positions, velocities, and accelerations of body markers or body segments. Sophisticated equipment—at first, stroboscopic photography, then cinematography and electrogoniometers, and, more recently, many types of computerized motion analysis systems—have provided comprehensive information about limb positions and their motions in normal and abnormal gait.[2,3,25–27]

Observational gait analysis employs a less sophisticated and less objective method in which an observer makes a judgment as to whether a particular joint angle or motion varies from a norm. Usually observational gait analysis is used to hypothesize causes of deviations and to direct treatment objectives. One disadvantage of the observational method of analysis is that it requires a great deal of training and practice to be able to identify the particular segment of gait in which a particular joint angle deviates from a norm while a person is walking. Videotaping the action and then viewing it using slow playback can improve this greatly. Another disadvantage of observational gait analysis methods is that they frequently have low reliability, although recent reports have identified some variables and conditions under which reliability is satisfactory.[28]

Trajectories, or the paths in space of particular parts, are of particular interest. For example, the path of the toe during gait is important in research into trips and falls.

Joint angles are of primary importance in recording gait. They may be expressed as absolute positions in space or, more commonly, as the relative angles of joints between adjacent segments, such as the hip or the knee. In most literature the anatomical position is called the "zero" position of the joint.

Kinetic Terms

Kinetics is concerned with the forces acting on the body that are the cause of the movement. A kinetic analysis is performed to understand the forces acting on the foot by the supporting surface, the forces acting on the joints, the forces produced by muscles, the moments produced by those muscles crossing the joints, the mechanical power generated or absorbed by those muscles, and energy patterns of the body during walking.

A **link-segment model** is most often used with an inverse dynamic approach. This means that we look at the body as a series of segments (e.g., foot, lower leg, thigh, etc.) with links at the joints. We begin the analysis starting with the segment touching the supporting surface (foot) and use Newtonian mechanics (Fig. 14–6).To do this we must know the positions of body markers through the gait cycle, usually from the motion analysis technology. We also need to know the forces acting on the body, and for the walking person, these are the ground reaction forces, derived from a force platform,[36] the weight of the body parts (gravity), and inertia of the body parts.[4,30] Inertia (units kg × m^2) is a measure of an object's resistance to changes in its rotation rate. It is the rotational analog of mass. It is particularly important in analyzing fast-moving parts of the body during the gait cycle, such as the swinging limb.

Using the **link segment model,** we can determine what forces and moments have been acting at each joint (e.g., the ankle) in order that the foot with that particular mass and moment of inertia moved with those particular linear and angular accelerations. Now we simply progress up the body segment by segment and solve for the more proximal joint. Larger numbers of segments and three-dimensional analyses are more complex than this, but the principles are the same.

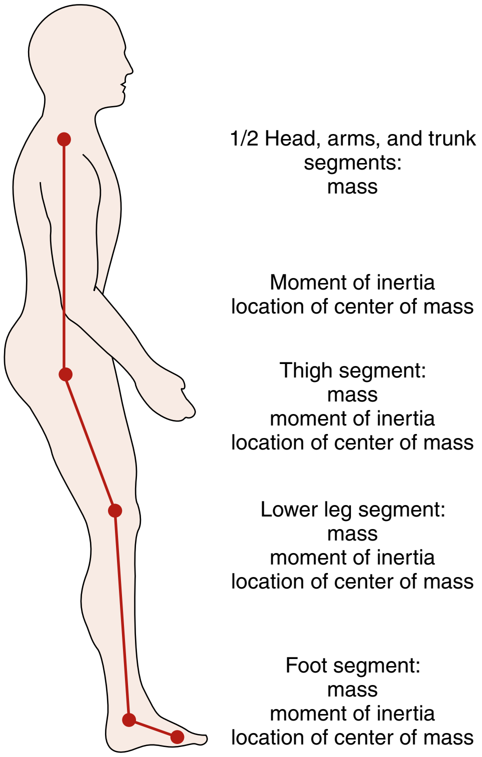

Figure 14-6 Simple four-segment link segment model of half a human body for use in gait studies. For each segment identified, the mass, moment of inertia, and location of the center of mass should be known for applications.

Ground reaction forces (GRFs) are the forces being applied to the foot by the ground when a person takes a step. These forces are equal in size (magnitude) but opposite in direction to the forces applied to the ground by the foot. GRFs are expressed using vertical, anteroposterior, and mediolateral axes.[4,31] If we combine the force components in two or thee planes, the vector sum is a single expression of the ground reaction force and is termed the ground reaction force vector (GRFV). When we refer to a single component, such as the vertical GRF, the abbreviation GRF will be used. If components are combined into a single vector (force[s] with a direction), GRFV will be used. In gait we frequently use the sagittal GRFV. The **center of pressure of the foot** on the supporting surface is the point where the resultant of all the floor-foot forces act. It moves along a path during gait and produces a characteristic pattern. GRFs, GRFVs, and CoPs that are characteristic of normal walking appear later in the chapter as Figures 14–10, 14–11, and 14–12.

A **moment of force,** usually referred to simply as **moment,** is the same as **torque,** which was introduced in Chapter 1. It is defined as the product of the force (usually muscle) and the perpendicular distance from its action line to the joint center. It can be thought of as the tendency to turn.

Continuing Exploration 14-1:

Understanding How the Kinetics of Gait Are Studied

The three equations on which the solutions are based for a two-dimensional analysis are simple: for each segment, the following three are applied:

$$\sum F_x = ma_x \ (1)$$
$$\sum F_y = ma_y \ (2)$$
$$\sum M_0 = I\alpha \ (3)$$

where

$\sum$ means "the sum of all of the"

F_x = forces in the designated x direction, in this case horizontal, in newtons (N)

m = mass of the segment, derived from anthropometric tables, in kilograms (kg)

a_x = acceleration of the center of mass in the x direction, derived from position and time data, in meters per second squared (m/sec^2)

F_y = forces in the designated y direction, in this case vertical, in N

a_y = acceleration of the center of mass derived from position and time data, in m/sec^2

M_0 = moment about selected point 0, the center of mass, in newton-meters (N·m), largely attributable to muscle activity, with ligaments, tendons, joint capsules, and bony components involved to a lesser extent

I = moment of inertia, a measure of an object's resistance to changes in its rotation rate. It is the rotational analog of mass. It is derived from anthropometric tables and expressed in kg·m^2

α = angular acceleration of segment derived from segment position and time data, expressed in radians per second squared (rad/sec^2). A radian is about 57°.

If we refer to Figure 14–7 with reference to the foot segment, equation (1) says that that the sum of all horizontal forces must equal the product of the mass of the foot and its acceleration in the horizontal direction. Equation (2) says that the sum of all vertical forces must equal the product of the mass of the foot and its acceleration in the vertical direction. Equation (3) says the sum of the moments about any designated center (we are choosing the center of mass) must equal the product of the moment of inertia (which can be visualized as the resistance to rotation) and the angular (rotatory) acceleration of the segment. Figure 14–7 shows that there are three things we do not know: the horizontal force at the ankle (E), the vertical force at the ankle (B), and the moment around the ankle (M_a). Foot-to-floor forces are shown as A and D, and are derived from a force plate. There is no moment about the free end of the segment.

Because we have three equations, we can solve for three unknowns but we cannot calculate the muscle moments on opposite sides of the joint if there is co-contraction. Applying these three equation results in numbers for the horizontal force of the tibia on the ankle, the vertical force of the tibia on the ankle, and the moment at the ankle. *In other words, we are finding out what forces and moments had to have been acting at the ankle in order that the foot with that particular mass and moment of inertia move with those particular linear and angular accelerations.* In this example[30] the downward force on the ankle (B in Fig. 14–7) was about 500 N, the horizontal force pushing backward on the ankle (E) was about 100 N, and the net moment being caused by the plantarflexors tending to plantarflex the foot (M_a) was about 80 N. Now if we were continuing the calculation we would simply progress up the body segment by segment and solve for the lower leg, then the thigh, then the trunk. Larger numbers of segments and three-dimensional analyses are more complex than this, but the principles are the same.

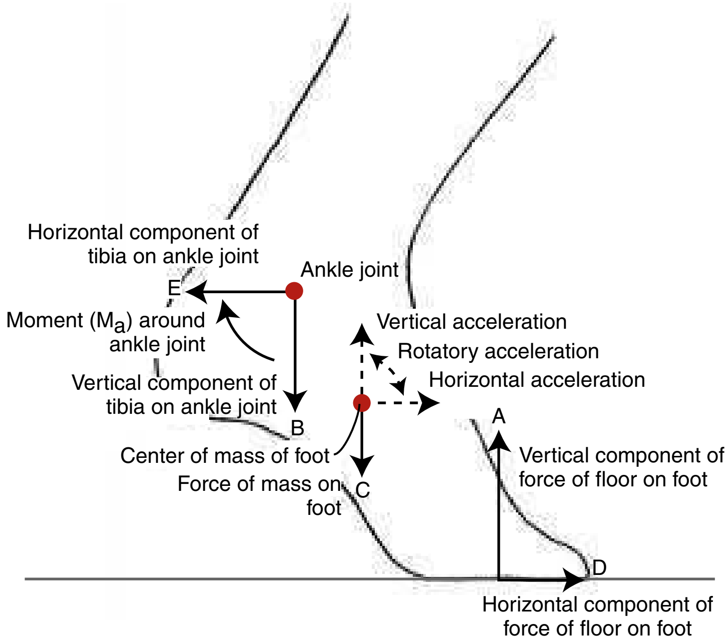

Figure 14-7 Diagram of the foot segment in stance shown with proximal joint (ankle) and all forces and moments acting on the segment. No moment is present at free end of the segment. Known forces and their location are the forces of the floor on the foot (A and D) and the force caused by the foot's mass (C); moments that can be calculated about the center of mass are moments caused by A and D, and the foot's moment of inertia. Unknown forces are joint reaction forces on the ankle (B and E), and the unknown moment is the net moment M_a at the ankle joint being caused by muscles, in this case, largely the ankle plantarflexors.

Internal moments are moments generated by the muscles, joint capsules, and ligaments to counteract the external forces acting on the body. External forces such as the ground reaction forces produce **external moments** about the joints. For example, when the weight is on the heel at initial contact (look at Fig. 14–11) an internal ankle dorsiflexor moment produced by anterior tibial muscles will oppose the external plantarflexion moment caused by the GRFV that is tending to plantarflex the foot. We will use the internal moment convention in this chapter. For ease of reading, the word *moment* will refer to an internal moment unless otherwise indicated.

Authors use different **moment conventions** to display internal moments in figures, but they indicate the direction of the moment by the words *flexor/extensor*; *abductor/adductor*; or *internal/external rotator*. The moment profiles presented in this chapter display the following internal moments as positive: ankle plantarflexor, knee extensor, and hip extensor.

Energy is the capacity to do **work,** and both work and energy are expressed in the same units, joules (J). Work is performed by the application of force, which produces accelerations and decelerations of the body and its segments. Muscles use metabolic energy to perform mechanical work by converting metabolic energy into mechanical energy. The main objective of locomotion is to move the body through space with the least expenditure of energy.

The overall metabolic cost incurred during locomotion may be measured by assessing the body's oxygen consumption (VO_2) per unit of distance traveled. If a long distance is traveled but only a small amount of oxygen is consumed, the metabolic cost of that particular gait is low. Approximately 32% of maximum oxygen consumption is needed by 20- to 30-year-olds to walk at a comfortable speed; this rises to 48% for 75-year-olds[32] and is even higher when a chronic condition such as stroke is present.[33] **Metabolic equivalents (METs)** are also used to express energy cost of the activity as multiples of the resting metabolic rate. Oxygen consumption for a person walking at 4 to 5 km/hour averages 100 mL/kg body weight per minute, typically 2.5 to 4 METS. The greatest efficiency is attained when the least amount of energy is necessary to travel a unit of distance. When asked to walk at a comfortable speed, people choose the speed at which they are most efficient, and if the speed of walking increases above this, the energy cost per unit of distance walked increases.[32] As the speed of walking decreases below self-selected walking speed, the energy cost increases. Both passive exchange of potential and kinetic energy and elastic energy utilization are responsible for the efficiencies,[34] and these are most effective near the self-selected speed.

Power generation is accomplished when muscles shorten (concentric contraction). They do positive work and add to the total energy of the body. Power, expressed in watts, is the work or energy value (in joules) divided by the time over which it is generated. The power of muscle groups performing gait is calculated through an inverse dynamic approach. The power generated or absorbed across a joint is the product of the net moment and the net angular velocity across the joint.[35] If both are in the same direction (flexors flexing, extensors extending, for example), positive work is being accomplished by energy generation. The most important phases of power generation and absorption during walking have been designated by joint (H = hip, K = knee, A = ankle) and plane (S = sagittal, F = frontal, T = transverse).[36]

Power absorption is accomplished when muscles perform a lengthening (eccentric) contraction. They do negative work and reduce the energy of the body. If joint motion and moment are in opposite directions, negative work is being performed through energy absorption.

Electromyography

Electromyography (EMG) is a technique for evaluating and recording the activation signal of muscles. EMG is performed using an instrument called an **electromyograph,** to produce a record called an **electromyogram,** in which the electrical activity generated by a muscle is recorded. Electrodes may be placed on the skin surface or inserted into the muscle. There is a great deal of information about EMG, the varieties of techniques that can be used, and the patterns obtained during gait.[35,37–41] EMG is often used in conjunction with force plates, goniometry, and/or motion analysis systems to link the muscle activity with other events during the gait cycle. The EMG record provides information about the time when particular muscles are acting and the relative level, or profile, of their activity. It does not tell why the muscles are acting or how much force the muscles are generating. The reader is encouraged to follow the developing literature on muscle function that is derived from elaborate mathematical simulations involving modeling precise muscle geometry and anthropometrics.[42–44] Although this work has just begun, it is already challenging conventional assumptions about the function of muscles.

CASE APPLICATION

Effects on Time and Distance Gait Variables

case 14–1

Ms. Brown walks with a speed of 0.20 m/sec and a cadence of 25 steps per minute. It is evident even on visual inspection that double-support time is considerably longer than normal on both steps. Stance phase is more than 60% of the gait cycle on her affected side, but she spends an even greater proportion of time in stance on the unaffected side. Her left step length (unaffected side) is shorter than her right (affected side), but her stride lengths are equal. Why? If you understand that a person walking in a straight line must have equal stride lengths but may have unequal step lengths, you understand the concepts of steps and strides.

CHARACTERISTICS OF NORMAL GAIT

Time and Distance Characteristics

Means and standard deviations of walking speed, stride length, and step cadence appear in Table 14–1. The values were derived from the classic work of Finley and Cody,[18] who surreptitiously measured the gait of 1,100 pedestrians, from Kadaba and colleagues,[19] Oberg and colleagues,[20] and colleagues of Ranchos Los Amigos National Rehabilitation Center[16] who obtained gait laboratory measurements.

Concept Cornerstone **14-1**

Normative Values for Time and Distance Gait Variables

It is helpful in clinical practice to keep approximate time and distance measures in mind. From Table 14–1 we can see that the length of a stride is usually between a meter and a quarter (1.25 m) and a meter and a half (1.50 m); the speed of walking is approximately this stride length (one stride) per second, and step cadence is approximately 120 per minute, or two steps per stride and one stride per second.

Sagittal Plane Joint Angles

Sagittal and frontal plane kinematics and kinetics have been reasonably well described, but transverse plane data are inconsistent and dependent on variations in joint positions and the specific methodologies used and will not be included here.

The approximate range of motion (ROM) needed in normal gait and the time of occurrence of the maximum flexion and extension positions for each major joint may be determined by examining the joint angle profiles in Figure 14–8. The standard deviation bars (dotted lines) around the mean profiles (solid lines) give an indication of how much person-to-person variation exists, demonstrating that 67% of subjects' values fell within the range shown. Results reported in gait studies vary with age, gender, and walking speed of subjects and with the method of analysis. Data presented here were derived from three-dimensional analyses.[35] For simplicity, the mean value shown in the figures will be referred to in the text, taken to the nearest 5°, and, to remind the reader that these are not fixed values, the "approximately" sign (~) will be used. In the anatomical position, the hip, knee, and ankle are at approximately 0°. Flexion for the hip and knee and dorsiflexion for the ankle are given positive values, and extension and plantarflexion are given negative values.

In Figure 14–8, it can be seen that the hip achieves maximum flexion (~+20°) around initial contact at 0% of the gait cycle and reaches its most extended position (~–20°) at about 50% of the gait cycle, between heel-off and toe-off. The knee is straight (0°) at initial contact and nearly straight again just before heel-off at 40% of the gait cycle. During the swing phase, the knee reaches its maximum flexion of ~+60° at ~70% of the gait cycle. Note also that a small knee flexion phase occurs at 10% of the gait cycle and peaks at ~+15°. The ankle reaches maximum dorsiflexion of ~+7° at approximately heel-off at about 40% of the gait cycle and reaches maximum plantarflexion (–25°) at toe-off (60%).

Table 14–1 **Normative Values for Time and Distance Variables**

CHARACTERISTIC	MALE: MEAN (SD)	FEMALE: MEAN (SD)	SOURCE
Speed of walking (m/sec)	1.37 (0.22)	1.23 (0.22)	Finley and Cody[15]
	1.37 (0.17)	1.32 (0.16)	RLA[8]
	1.22–1.32*	1.10–1.29*	Oberg et al[17]
	1.34 (0.22)	1.27 (0.16)	Kadaba et al[16]
Length of one stride (m)	1.48 (0.18)	1.27 (0.19)	Finley and Cody[15]
	1.48 (0.15)	1.32 (0.13)	RLA[8]
	1.23–130*	1.07–1.19*	Oberg et al[17]
	1.41 (0.14)	1.30 (0.10)	Kadaba et al[16]
Step cadence (steps/min)	110 (10)	116 (12)	Finley and Cody[15]
	111 (7.6)	121 (8.5)	RLA[8]
	117–121*	122–130*	Oberg et al[17]
	112 (9)	115 (9)	Kadaba et al[16]

*Range of means.
RLA, Ranchos Los Amigos.

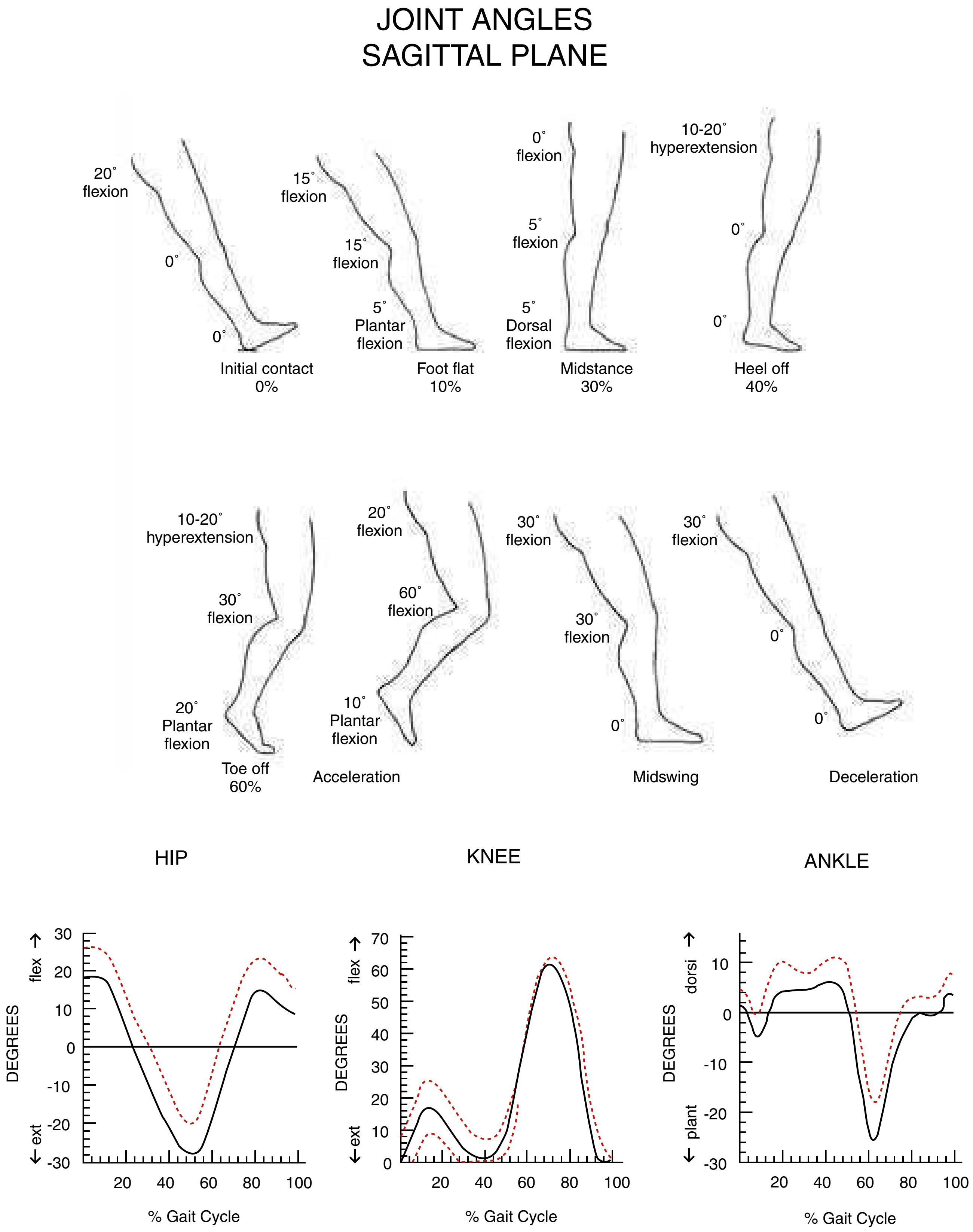

Figure 14-8 Joint angles in degrees at the hip, knee, and ankle in the sagittal plane. The dotted lines in the angle diagrams represent the standard deviation values, and the solid lines represent the mean values. *(Joint angle diagrams redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait Analysis: Theory and Application, pp 263-265. St. Louis, MO, Mosby-Year Book, 1994, with permission from Elsevier.)*

Concept Cornerstone **14-2**

Hip, Knee, and Ankle Range of Motion Needed for Normal Walking

For normal walking, we need a hip ROM from approximately 20° of extension to 20° of flexion, a knee range from straight (0°) to 60° flexion, and an ankle range from 25° of plantarflexion to 7° dorsiflexion. If these joint ranges are not available, a gait pattern would be expected to show considerable deviation from the norm.

Frontal Plane Joint Angles

During the first 20% of stance, the pelvis or the contralateral side drops about 5°, which results in adduction of the hip (Fig. 14–9). The hip abducts smoothly to about 5° of abduction, peaking about toe-off, then returns to neutral at initial contact. The knee remains more or less neutral, except for a brief abduction peaking at about 7° in midswing, and then returns to neutral.[27] From the figure, it can be seen that the ankle complex everts from about 5° of inversion to 5° of eversion in early stance and inverts about 15° during push-off.

CASE APPLICATION

Effects on Joint Angle Patterns

case 14–2

An examination of videotaped joint angles reveals that Ms. Brown has no knee flexion phase in early stance, and she tends to fully extend her knee in midstance. She has minimal ability to dorsiflex her ankle and has difficulty clearing the floor with her affected limb as a result of poor dorsiflexion and because she does not bend her knee more than a few degrees during swing phase. Instead, she tends to lift her pelvis ("hike") to clear her foot during swing on the affected side. Her affected hip does not extend beyond neutral. How do Ms. Brown's joint angle profiles vary from the normal in stance and swing phases?

Ground Reaction Force and Center of Pressure

The ground reaction forces have a typical pattern from initial contact to toe-off (Fig. 14–10).[31] In the vertical direction, the magnitudes are low at first but increase to values that are greater than body weight both in early stance and again in late stance, with lower values at midstance. The first hump is related to the weight acceptance when the body's downward velocity is being slowed (upward acceleration of the body's mass). The second hump is due to push-off and shows that the body's center of mass is being accelerated upward to increase its upward velocity. In the anteroposterior direction, the GRF is directed posteriorly against the foot that is making initial contact and prevents the foot from slipping forward. It reaches a maximum magnitude of about 20% of body weight. At midstance, the force becomes neutral, but as the body enters the second half of stance phase, the vector is directed anteriorly against the foot, enabling the person to push off. The mediolateral forces are small in magnitude and are variable across individuals. Figure 14–11 shows the GRFVs in the sagittal plane relative to the joint positions in early stance phase. You can see that the GRFV at initial contact passes behind the ankle (tending to plantarflex it) but in front of the knee and hip (tending to extend them). In early stance, the GRFV passes behind the knee (tending to flex it), then moves ahead of it until late stance. About midstance (Fig. 14–12), the GRFV passes behind the hip joint and remains there until swing phase begins.

The single point on the foot at which the resultant surface pressure may be considered to be acting is called the *center of pressure (CoP)*. This point is the starting point for the GRFV. The CoP of the foot on the supporting surface produces a characteristic pattern (Fig. 14–13). The pattern for normal individuals during barefoot walking differs from the patterns in which various types of footgear are used.[45] In barefoot walking, the CoP starts at the posterolateral edge of the heel at the beginning of the stance phase and moves in a nearly linear manner through the midfoot area, remaining lateral to the midline, and then moves medially across the ball of the foot with a large concentration along the metatarsal break. The CoP then moves to second and first toes during late stance.

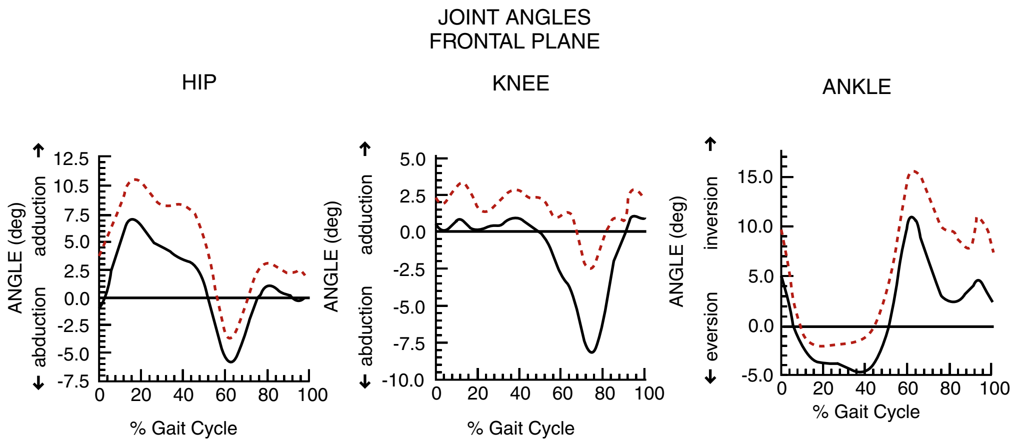

Figure 14-9 Joint angles in degrees at the hip, knee, and ankle in the frontal plane. *(Redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait Analysis: Theory and Application, pp 263-265. St. Louis, MO, Mosby-Year Book, 1994, with permission from Elsevier.)*

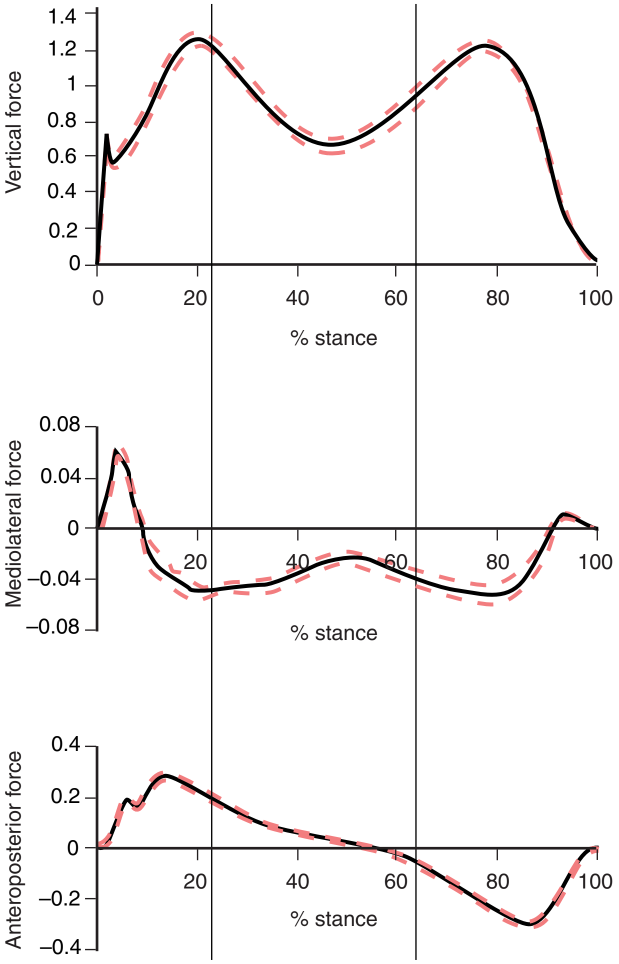

Figure 14-10 Ground reaction forces in newtons as a proportion of body weight over stance phase. Positive values represent upward, lateral, and posterior forces acting on the foot. *(Adapted and redrawn from Hunt AE, Smith RM, Torode M, et al: Intersegment foot motion and ground reaction forces over the stance phase of walking. Clin Biomech [Bristol, Avon] 16:592, 2001.)*

Continuing Exploration 14-2:

Why Don't Hip, Knee, and Ankle Moment Figures Look Familiar to Me?

Sometimes when we are reading journal articles that include figures and explanations of moments at the hip, knee, or ankle during walking, the shapes seem totally unfamiliar and the explanations do not make sense. In fact they often feel "reversed," which, in fact, is exactly what has happened. There are two ways of reporting moments. Here we have chosen to use "internal" moments, which refer to the *moments produced by the muscles and other structures around the joint* that had to have been acting to move body parts (with their known characteristics) in the directions and with the accelerations that we observe with motions capture systems. However, some authors use "external" moments, which refer to the moments produced by the external forces acting around the joint that must be acting in order that the body parts (with their known characteristics) were moving in the way they were. For example, if the internal moment of the knee in early stance was found to be an extensor moment, being caused by the quadriceps muscle, the external moment would be a flexor moment, caused largely by the GRFV passing behind the knee "trying to" flex it. Similarly, an internal abductor moment is an external adductor moment—but the magnitude is exactly the same. Therefore, internal moments must match the magnitude of the external moments, but in the opposite direction, to result in the positions and motions that we observe.

Sagittal Plane Moments

Before concerning ourselves with the individual sagittal plane moments about the hip, knee, and ankle, let us consider the concept of the support moment, which will help

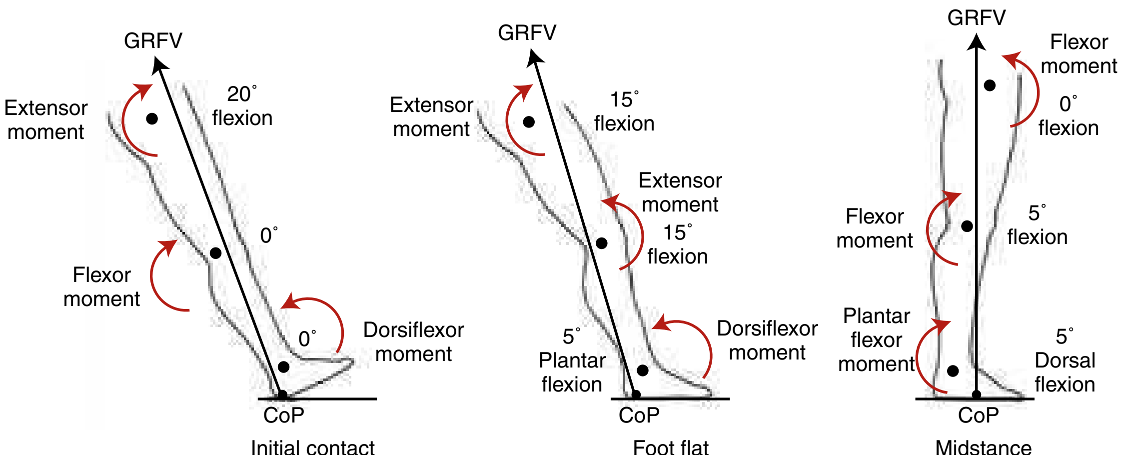

Figure 14-11 Diagram of joint positions with center of pressure (CoP), ground reaction force vector (GRFV), and **internal net moments** of force for gait cycle events of initial contact, foot flat, and midstance.

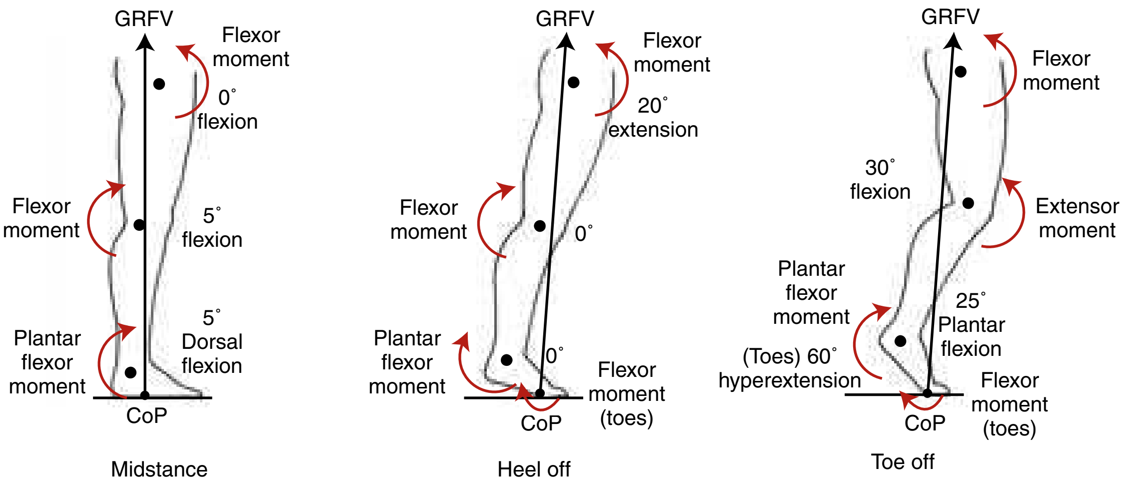

Figure 14-12 Diagram of joint positions with center of pressure (CoP), ground reaction force vector (GRFV), and **internal net moments** of force for gait cycle events of midstance, heel-off, and toe-off.

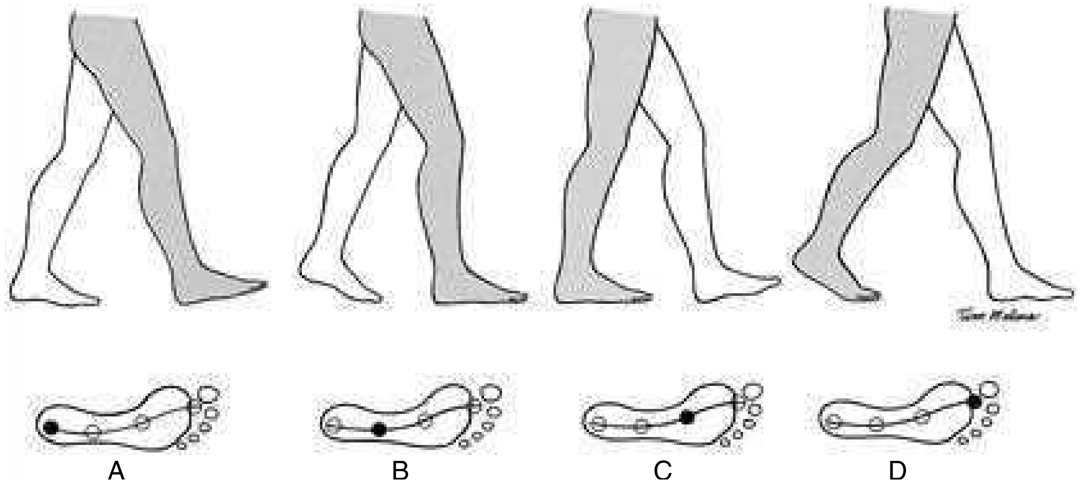

Figure 14-13 A center of pressure (CoP) pathway is shown by the position of the black dot at initial contact **(A),** at foot flat **(B),** just before heel-off **(C),** and just before toe-off **(D).**

us to understand the moment patterns through the gait cycle. The sum of the hip, knee, and ankle moments keeps the leg from collapsing during the stance phase. That is, for most of stance phase, the sum of all moments acting at the hip, knee, and ankle is a positive or extensor moment (Fig. 14–14). Winter[5] called this total limb synergy a **support moment.** He found the extensor support moment to be consistent for all walking speeds for both normal individuals and persons with disabilities. In Figure 14–14, notice that hip extension provides all of the positive moment in early stance, but it is soon joined by a knee extensor moment. (Remember that the moment identifies only the "supportingness" of the muscle group; it does not mean that muscle shortening or lengthening is occurring. To find out if the muscle is shortening or lengthening, one needs to consult the joint angle profiles: Even if there is no change in joint angle a moment may be present. In normal gait, the knee extensors are first lengthening and then shortening.) As stance phase proceeds, there is increasing support provided by the ankle plantarflexors until they become *the only support in most of late stance*. The support moment changes from a net extensor to a net flexor moment in late stance (55% to 60% of the gait cycle), which initiates swing. In late swing, a small net extensor moment appears again, to assist in the final positioning of the limb for initial contact.[5,45]

A clinically important feature of the support moment is that as long as the moments add up to be extensors, the body can vary how it accomplishes its support. For example, if the ankle plantarflexors are weak, the hip extensors and/or the knee extensors can compensate. If contraction of the knee extensors causes pain, the hip extensor and/or ankle plantarflexors can contract harder.

The support moment helps us to understand the joint moment profiles for each joint. The sagittal plane moment profiles for the hip, knee, and ankle are shown with GRFVs in Figure 14–15, and with joint angle profiles and muscle work in Figure14–16.

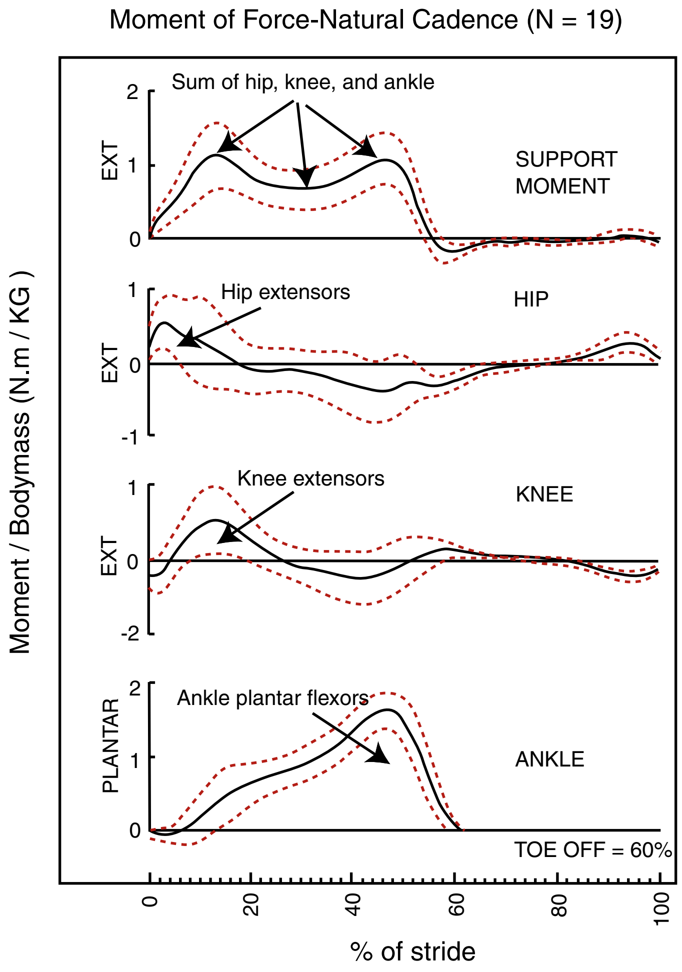

Figure 14-14 Typical pattern of sagittal plane moments at the hip, knee, and ankle, shown with their algebraic sum, the support moment. *(Redrawn from Winter DA: The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological [ed. 2]. Waterloo, Ontario, Waterloo Biomechanics, 1991, with permission from David A. Winter.)*

Frontal Plane Moments

Frontal plane moments are shown in Figure 14–17 and with joint angle profiles and muscle work in Figure 14–18. Large abduction moments of similar shapes occur at the hip and knee, and a smaller one occurs at the ankle. These are provided largely by ligament forces across the knee and ankle joints, and are necessary as the center of mass of the body is considerably medial to the point of support on the foot. There appears to be some active component at the knee, however (either muscular or passive spring related), because some power generation will be seen in the section on frontal plane powers.

CASE APPLICATION

Difficulty in Developing an Adequate Support Moment

case 14–3

Ms. Brown has low levels of activation of her ankle plantarflexors, knee extensors, and, to a lesser extent, hip extensors. This will make it difficult for her to develop an adequate support moment. To gain knee stability she tends to thrust her knee backward. We know that she has smaller deficits at the hip than at the ankle, and so we would encourage hip extension in early stance to assist with the support moment, as well as trying to stimulate the knee extensors. During this early poststroke period, we would expect some natural increase in force-generating capability of the muscles (strength) and we would be prepared to take advantage of it by strengthening the muscles during functional movements. If over time she is unable to provide enough support, an ankle orthosis would provide a passive extensor moment, but there may be energy costs to doing this.

Continuing Exploration 14-3:

Kinematics and Kinetics of the Foot and Ankle

Specific descriptions of the biomechanics of the foot during gait have been hindered by the complexity of the composite movements created by the ankle, subtalar, and transverse tarsal joints (and their noncardinal axes). These kinematics are referred to as *pronation* (a composite of dorsiflexion, eversion, and abduction) and *supination* (a composite of plantarflexion, inversion, and adduction). Usually, the foot movement is described as consisting of pronation early in stance, followed by progressive supination. However, three-dimensional analyses of the lower leg and foot, modeling the foot as a rearfoot segment and a forefoot segment, have provided more insight into its behavior during stance.[46,47] Although kinematics and kinetics are highly variable and depend on subject, foot position, terrain, and method of analysis, some general features are thought to occur. The rearfoot segment everts with respect to the lower leg early in stance and inverts during push-off. In the transverse plane, the rearfoot segment externally rotates, (abducts) in early stance, followed by internal rotation (adduction) through push-off. The forefoot segment adds considerably to dorsiflexion during early stance.

Text continued on page 540

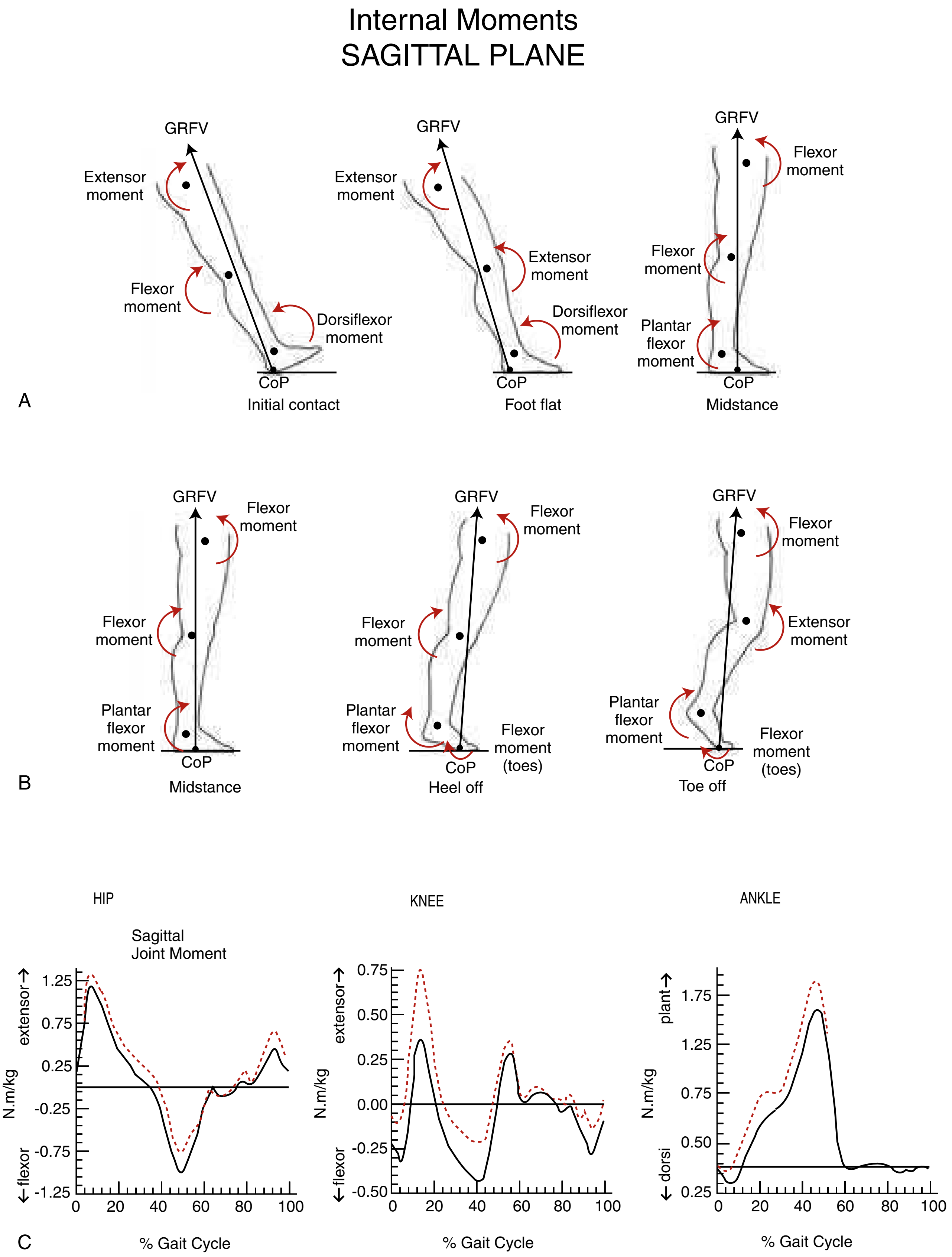

Figure 14-15 Patterns of internal moments in the sagittal plane at the hip, knee, and ankle with center of pressure (CoP) and ground reaction force vectors (GRFVs). The dotted lines represent the standard deviations, and the solid lines represent the mean values. *(Diagrams of internal moments redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait Analysis: Theory and Application, pp 263-265. St. Louis, MO, Mosby-Year Book, 1994, with permission from Elsevier.)*

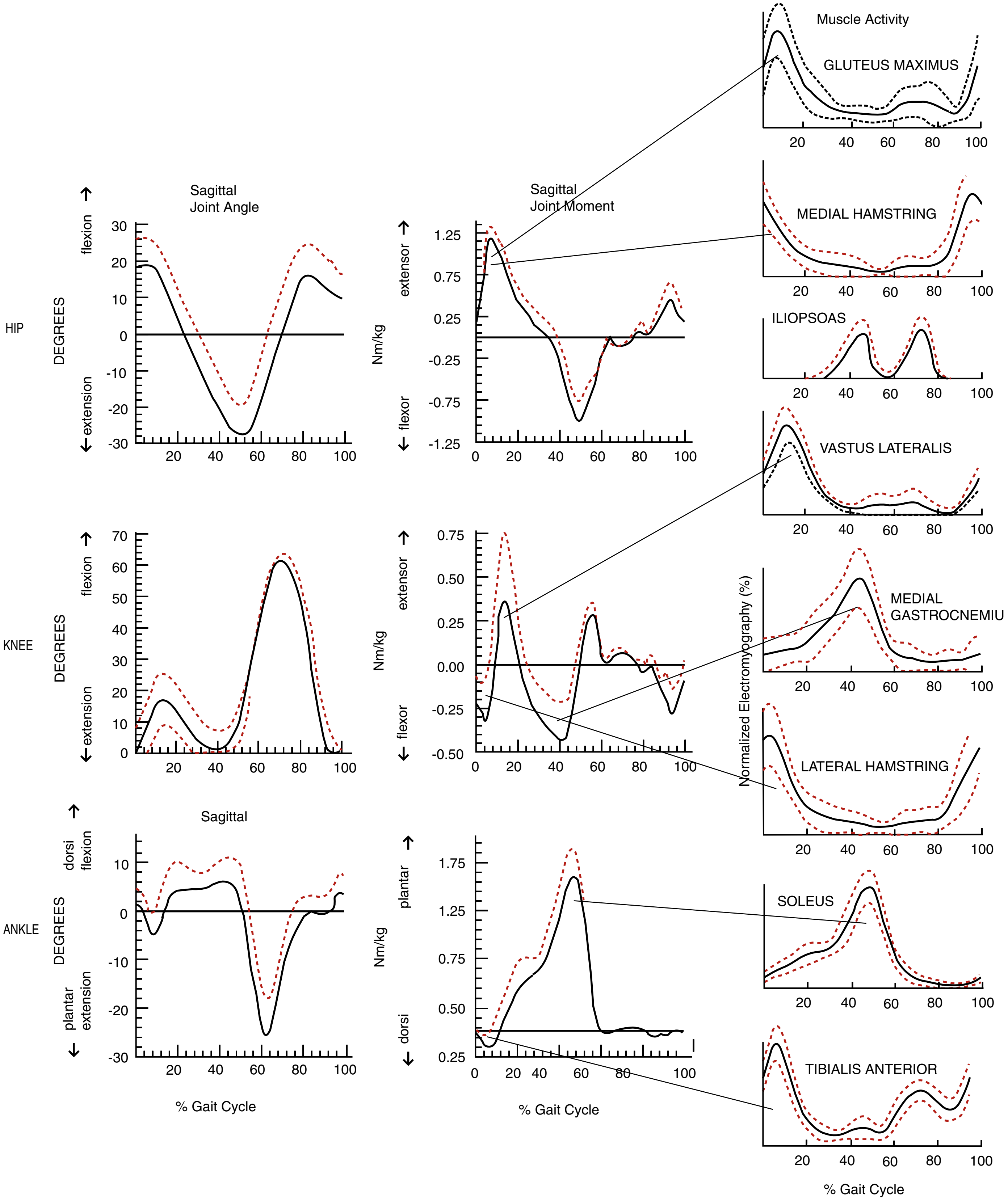

Figure 14-16 Joint angles and net joint moments in the sagittal plane, and EMG profiles of representatives of major contributors to joint moments of hip, knee, and ankle during adult gait. *(Angle and moment profiles redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait Analysis: Theory and Application, pp 263-265. St. Louis, MO, Mosby-Year Book, 1994, with permission from Elsevier. Muscle activity redrawn from Winter DA: The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological [ed. 2]. Waterloo, Ontario, Waterloo Biomechanics, 1991, with permission from David A. Winter. Iliopsoas muscle activity redrawn from Bechtol CO: Normal Human Gait. In American Academy of Orthopaedic Surgeons: Atlas of Orthotics, p 141. St Louis, MO, CV Mosby, 1974, with permission from Elsevier.)*

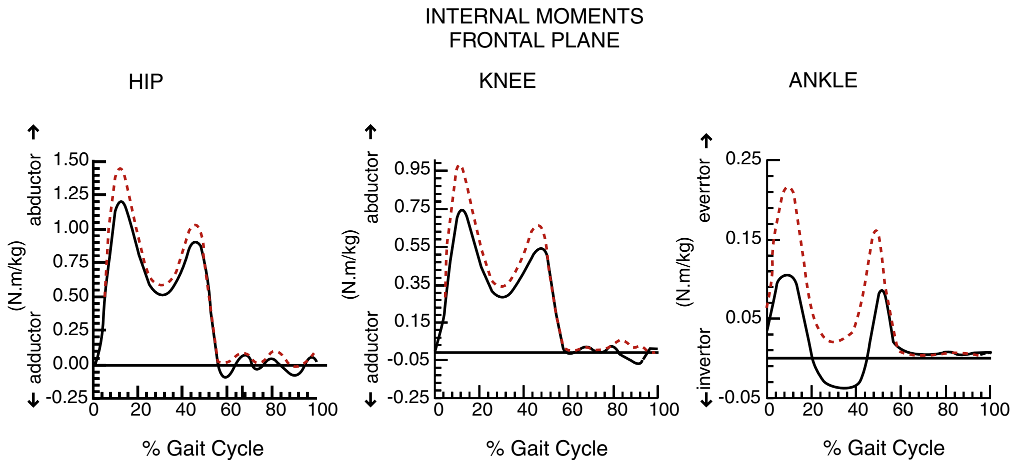

Figure 14-17 Patterns of internal moments in the frontal plane at the hip, knee, and ankle. The dotted lines represent the standard deviation, and the solid lines indicate the mean values. *(Redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait Analysis: Theory and Application, pp 263-265. St. Louis, MO, Mosby-Year Book, 1994, with permission from Elsevier.)*

Sagittal Plane Powers

Sagittal plane power profiles across the hip, knee, and ankle are compared with joint angle and major muscle activity profiles in Figure 14–19. A summary of the phases of power generation and absorption during the full gait cycle appear in Table 14–2. Table 14–3 on page 549 shows powers with joint motion, GRFs, moments, and muscle activity from initial contact to midstance, and Table 14–4 on page 550 for midstance to toe-off. Despite the complexity of the appearance of this information, the important facts are straightforward. Of greatest importance is the fact that most of the positive work of walking is provided by the ankle plantarflexors at push-off, the hip extensors in early stance, and the hip flexors in late stance and early swing ("pull-off"). Second, the knee extensors normally provide very little power, and act largely as absorbers. To make it possible to compare subjects, power values have been normalized by dividing the power in watts by the subjects' weight in kilograms.

In the sagittal plane (see Fig. 14–19), a burst of positive work (energy generation) occurs as the hip extensors contract concentrically during early stance (H1-S), while the knee extensors perform negative work (energy absorption) by acting eccentrically (K1-S) to control knee flexion during the same period.[48] Following a tiny period of unnamed negative work by the dorsiflexors at initial contact, negative work is performed by the plantarflexors (A1-S) as the leg rotates over the foot during the period of stance from foot flat to about 40% of the gait cycle. However, a small amount of positive work is done by the knee extensors at the beginning of this period (K2-S), extending the knee after foot flat.[48] Positive work of the plantarflexors at push-off (A2-S: late stance, ~40% to 60% of the gait cycle) and hip flexors at pull-off (H3-S: late stance and in early swing, ~50% to 75% of the gait cycle) increases the energy level of the body. During this 40% to 60% of the gait cycle when energy is being generated from A2-S and H3-S, simultaneous absorption is occurring by knee extensors (K3-S). In late swing, negative work is performed by the knee flexors (K4-S) as they work eccentrically to decelerate the leg in preparation for initial contact.

At slow and normal speeds of walking in healthy subjects, the hip flexors and extensors contribute about 25% of the total concentric work.[49] The ankle plantarflexors contribute about 66%, and the knee extensors about 8%.[49] Clearly, the ankle plantarflexors are of primary importance in walking (see "Continuing Exploration: On Rockers and Push-Off"). We can think of the ankle and hip muscles as primarily concerned with the function of generation of energy in walking and the knee muscles as absorbers of energy.[4,17]

Table 14–2 Summary of Major Phases of Power Generation and Absorption During Full Gait Cycle

GENERATION (CONCENTRIC CONTRACTION)			ABSORPTION (ECCENTRIC CONTRACTION)		
Name of Power Burst	*Major Muscle or Group*	*Occurrence in Gait Cycle*	*Name of Power Burst*	*Major Muscle or Group*	*Occurrence in Gait Cycle*
H1-S	Hamstrings, gluteals	Early stance	H2-S	Psoas, rectus	Midstance
K2-S (small)	Quadriceps	Early stance	K1-S	Hamstrings	Very early stance
H3-S	Psoas, rectus	Late stance, "pull-off"	K3-S	Rectus femoris	Late stance
A2-S	Plantarflexors	Late stance, "push-off"	K4-S	Hamstrings	Late swing
			A1-S	Plantarflexors	Early and midstance

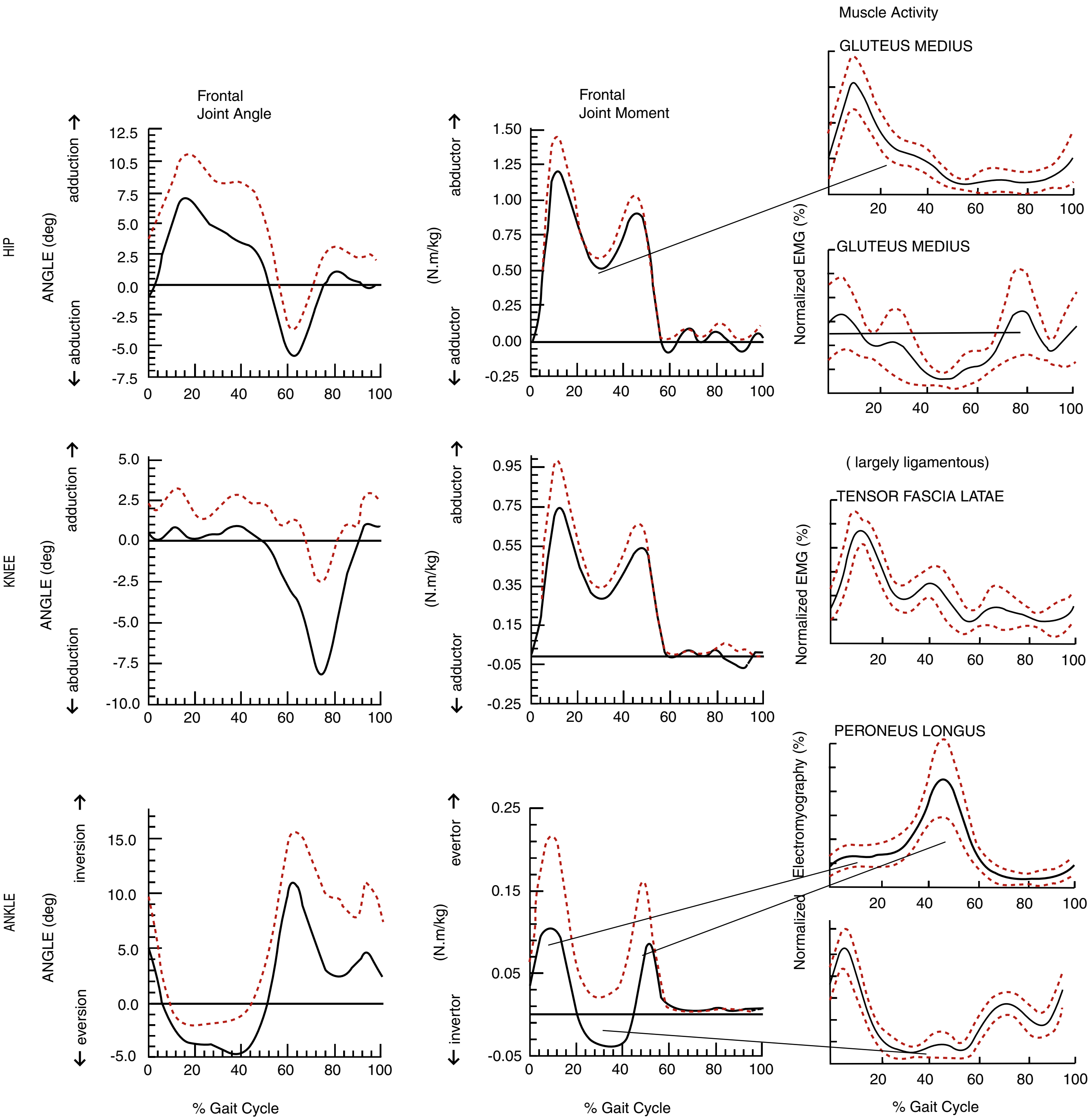

Figure 14-18 Joint angles and net joint moments in the frontal plane, and EMG profiles of representatives of major contributors to joint moments of hip, knee, and ankle during adult gait. *(Angle and moment profiles redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait Analysis: Theory and Application, pp 263–265. St. Louis, MO, Mosby-Year Book, 1994, with permission from Elsevier. Muscle activity redrawn from from Winter DA: The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological [ed. 2]. Waterloo, Ontario, Waterloo Biomechanics, 1991, with permission from David A. Winter.)*

CASE APPLICATION

Effects of an Ankle-Foot Orthosis

case 14–4

Recall that Ms. Brown hiked up her affected side (lifted hip and pelvis) in order to clear her foot during swing phase. This has been shown to have a serious energy cost[51] as the full weight of the upper body is raised and lowered, and there are no opportunities for savings by kinetic-potential exchange. Because the inability to adequately dorsiflex her foot appeared to be part of the problem, a light ankle-foot orthosis was prescribed, which held her ankle in a neutral position during swing phase. The orthosis also assisted with her inability to provide sufficient support during stance. The somewhat stiff orthosis material accomplished this by resisting dorsiflexion of her ankle during stance, as her body progressed forward over her foot. She gained the ability to bend her knee in later stance, thus avoiding excessive hip hiking. Watch for possible drawbacks while you read the section on power and work. If the orthosis does not permit any ankle plantarflexion during push-off, she will lose the energy that would otherwise be provided by plantarflexor generation.

Continuing Exploration 14-4:

Understanding Power and Work in Gait

Examination of power plots helps us identify and understand the contribution of lower extremity muscles to gait, and its phases. When slow or inefficient gait is a problem, knowledge of the sources of power enables a health practitioner to assist the client to compensate for deficiencies. Note that the scales used for power in the figures vary, and this must be taken into account when assessing the work (the area under the power curve). In Figure 14–19, identify the joint around which the largest amount of positive work (above zero line) is performed in the sagittal plane. It is apparent that the burst denoted A2-S (S denoting burst in the sagittal plane) is the largest, and this occurs at the ankle. We know that this positive work may be caused by either ankle plantarflexor activity with plantarflexion, or else active ankle dorsiflexors with dorsiflexion. We know this because positive work is occurring. A quick examination of the joint angle profile shows us that ankle plantarflexion is occurring at this time so we know the burst of positive work is caused by the plantarflexors. The second largest burst of positive power, denoted H1-S, is a hip extensor moment with hip extension occurring in early stance. The third burst, H3-S, occurs near A2-S. It is caused by a hip flexor moment and hip flexion that occur at the end of stance and the beginning of swing. It is frequently called *pull-off*.

The knee is not of major importance in energy generation. However, there is a small phase, denoted K2-S, in which the knee extensors extend the knee after the knee flexion phase of early stance. K2-S can be important in pathologies such as cerebral palsy when other sources of energy are not available.

Unless gait velocity is increasing, energy has to be systematically removed. The knee accomplishes this at K1-S (knee flexion with a knee extensor moment) and K3-S (small knee extensor moment in late stance occurring while the knee is flexing quite quickly). The latter may represent inefficiency in gait.[52] K4-S (knee flexor absorption with knee extension) occurs before initial contact. *Note that, as with all absorption phases, that is, when eccentric muscle activity is dominating, the dominating moment is opposite to the movement that is occurring.* In this case, a flexor moment is dominating (it is the net moment) while knee extension is occurring. The knee flexors are acting eccentrically, and the knee is being pulled into extension. A simpler example occurs when you squat. In this case, a knee extensor moment is dominating while knee flexion is occurring. Energy is being removed from the body, which is very evident as you end up in a lower position than you were; your potential energy is therefore lower.

Let us now examine the periods during which simultaneous positive and negative work normally occur, which, if excessive, represents inefficiency. The most important one occurs in late stance: Negative work done by the flexing knee (K3-S) occurs concurrently with both positive work by the hip flexors (H3-S—*pull-off*) and ankle plantarflexors (A2-S—*push-off*). Values of K3-S that are above normal represent excessive inefficiency and usually result in slower walking speeds.

Frontal Plane Powers

In the frontal plane (Fig. 14–20), an initial period of absorption by the hip abductors (H1-F) is followed by two small bursts of positive work in the remainder of stance (H2-F, H3-F). These bursts provide fine control of the mediolateral position of the center of mass of the body. At the knee, there is a very small generation pattern during the first half of stance (K1-F), followed by a small absorption (K2-F) both caused by abductor structures. Ankle power patterns are not shown as they are small and somewhat inconsistent.

Mechanical Energy of Walking

Walking at a constant speed is accomplished by the bursts of **positive (concentric)** and **negative (eccentric) muscle work** described above that result in small stride-to-stride fluctuations in body height and variations in the velocities of the body parts. One way the body saves energy is by making use of passive exchange of potential and kinetic energy through the stride, a process that is much like a ball rolling down one hill and up another.

Figure 14–21 is a diagram of kinetic energy of the head, arms, and trunk (bottom line), their potential energy (middle), and the two added together (top).[50] At initial contact of each foot, that is at 0% and 50% of the gait cycle, the body has the lowest potential energy but is moving the fastest, and as it moves into midstance, the potential energy rises and is exchanged for kinetic energy. Evidence of the energy exchange can be seen as the potential energy curve slopes downward following

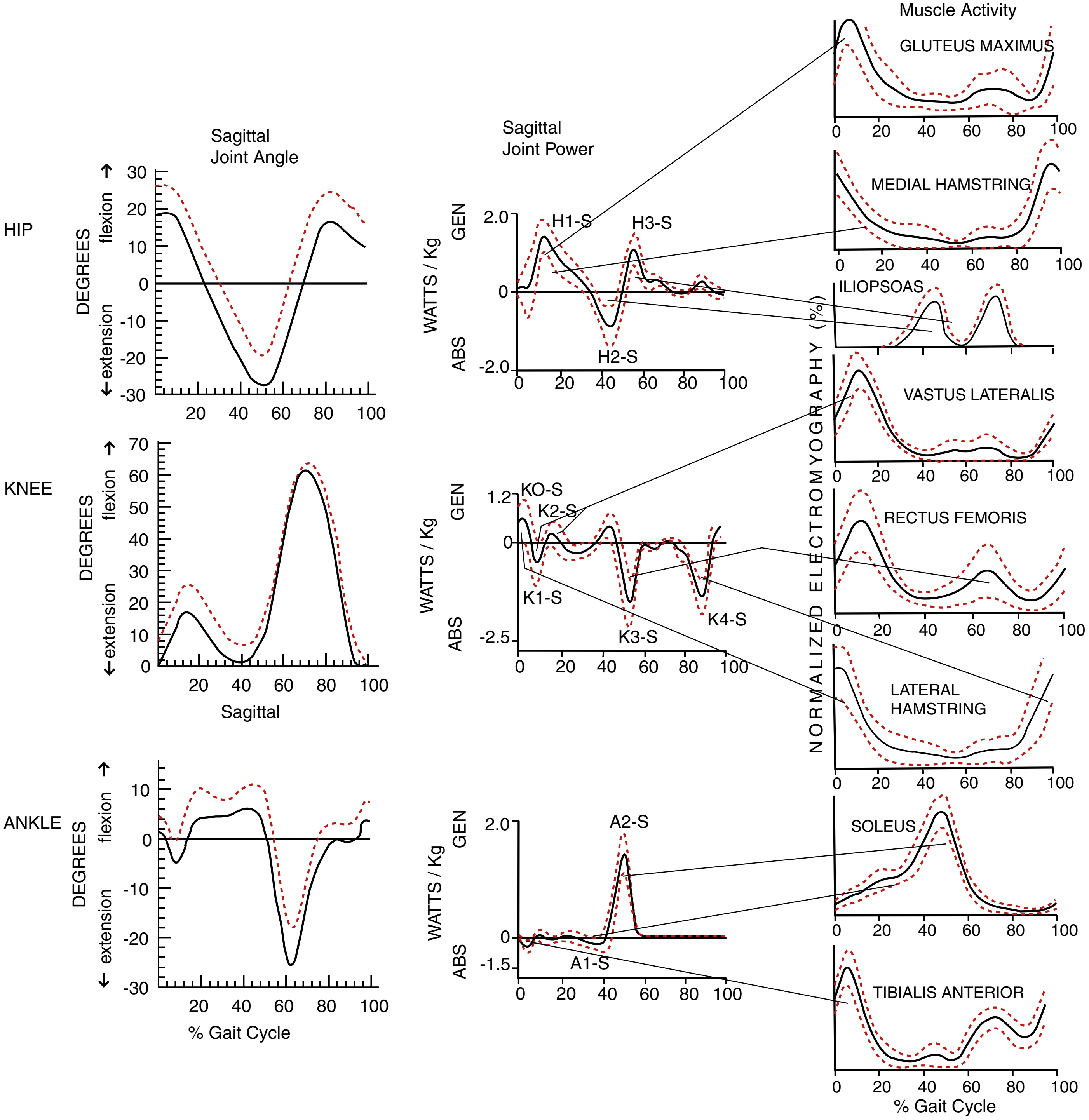

Figure 14-19 Joint angles and joint powers in the sagittal plane, and EMG profiles of representatives of major contributors to joint powers of hip, knee, and ankle during adult gait. *(Angle profiles redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait Analysis: Theory and Application, pp 263-265. St. Louis, MO, Mosby-Year Book, 1994, with permission from Elsevier. Power profiles redrawn from from Eng JJ, Winter DA: Kinetic analysis of the lower limbs during walking: What information can be gained from a three dimensional model? J Biomech 28:753, 1995, with permission from Elsevier. Muscle activity redrawn from Winter DA: The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological [ed. 2]. Waterloo, Ontario, Waterloo Biomechanics, 1991, with permission from David A. Winter. Iliopsoas muscle activity redrawn from Bechtol CO: Normal Human Gait. In American Academy of Orthopaedic Surgeons: Atlas of Orthotics, p 141. St Louis, MO, CV Mosby, 1974, with permission from Elsevier.)*

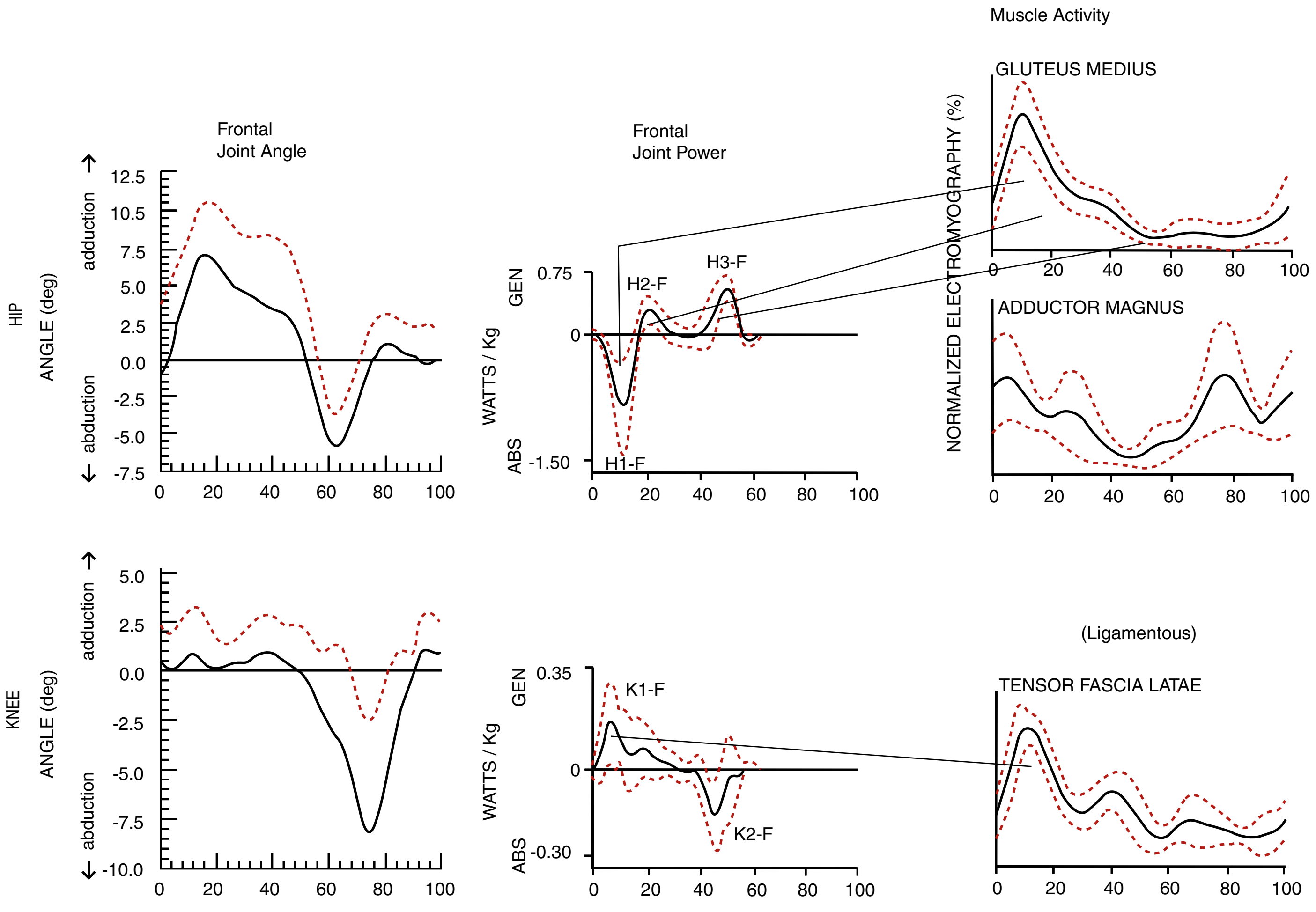

Figure 14-20 Joint angles and joint powers of hip and knee in the frontal plane, and EMG profiles of representatives of major contributors to joint powers of hip and knee during adult gait. *(Angle profiles redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait Analysis: Theory and Application, pp 263-265. St. Louis, MO, Mosby-Year Book, 1994, with permission from Elsevier. Power profiles redrawn from Eng JJ, Winter DA: Kinetic analysis of the lower limbs during walking: What information can be gained from a three dimensional model? J Biomech 28:753, 1995, with permission. Muscle activity redrawn from Winter DA: The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological [ed. 2]. Waterloo, Ontario, Waterloo Biomechanics, 1991, with permission from David A. Winter.)*

CASE APPLICATION

Continuing Gait Problems

case 14–5

It was noted that Ms. Brown tended to fully extend her knee in midstance and then flexed her knee only a few degrees during swing phase. A power analysis of Ms. Brown's affected limb, if available, would have shown a severely reduced A2-S and H3-S and virtual absence of an H1-S energy burst. The first two would be apparent to the therapist as no firm push-off[28] and no rapid hip flexion. This meant that Ms. Brown would be unable to push off strongly (A2-S) or pull off strongly (H3-S), because both actions require knee flexion. Every attempt was made during gait training to gain knee control in midstance, not permitting it to fully extend. Strong push-off and strong "pull-off" then could be encouraged. Because the hip extensors on the affected side were among the least affected muscles, Ms. Brown was encouraged to exploit H1-S, the "push from behind" in early stance. Stronger activity of A2-S, H1-S, and H3-S on the unaffected side would be encouraged, especially later in stages of rehabilitation, to provide interlimb compensation for the reduced activity on the affected side. Gait speed would increase to the degree that these efforts are effective.

Continuing Exploration 14-5:

On Rockers and Push-Off

There has been some controversy concerning the terms *rockers* and *push-off*. Orthopedic literature frequently refers to three rockers as characterizing normal kinematics of the foot and ankle during stance phase. The first rocker, or initial rocker, occurs from initial contact until foot flat and has a fulcrum about the heel (heel pivot). The second rocker, or midstance rocker, is described as occurring between foot flat and heel-off and has a fulcrum about the ankle joint. The third rocker, or terminal rocker, occurs between heel-off and toe-off (push-off), with the leg rotating about the forefoot. This terminology is frequently used to denote abnormalities occurring in particular phases, as, for example, "... walking with an ankle-foot orthosis impaired [the] third rocker"[53] and does not imply that there is no push-off. Although the terminology is not incorrect, it is preferable that standard terminology be used.

Before kinetic link segment analysis was common, there were objections to the term *push-off*. It was thought that the second peak in the vertical ground reaction force was passive and resulted from changes in body alignment, rather than an increasing force resulting from plantarflexor contraction. However, the work of Winter[54] and others[42,43,52] shows that push-off not only exists but is normally responsible for a major portion of the work of walking. It is clear that a person with no active ankle plantarflexion (hence no power generation at the ankle) can walk, however. Some compensation can be provided by the hip extensors in early stance (H1-S) and the hip flexors in late stance (H3-S) from both limbs. Zajac and colleagues[42,44] and other researchers have shown that the energy produced by the soleus muscle in late stance is delivered to the trunk to accelerate it forward, but the increase in trunk energy is more than that produced by the soleus muscle.[43,44] It appears that the soleus, rectus femoris, and gastrocnemius work synergistically to ensure forward acceleration of the trunk. These studies show that muscles not only generate and absorb energy but, in many cases, serve to redistribute energy.

midstance at the same times as the slope of kinetic energy rises. In this way there are great energy savings, as can be seen in the top curve, which is the sum of potential and kinetic components. It is also apparent that if kinetic energy does not match potential energy in magnitude—that is, if the person walks much more slowly or much more quickly—conservation is reduced. The lower limbs are larger contributors to the total energy costs than are the head, arms, and trunk, but the opportunities for modifications by training are limited.

Muscle Activity

EMG studies of gait are used to augment the understanding provided by a link segment analysis, to validate theoretical models that attempt to explain why muscles are needed for certain functions, and in theoretical models developed to explain the muscle activity found by EMG.[41] The EMG reported in the section that follows is derived from the work of Winter[17] and is shown in Figures 14–22, 14–23, and 14–24. EMG with respective joint angles and moments appears in Figures 14–16 and 14–18, and with repective joint angles and powers in Figures 14–19 and 14–20.

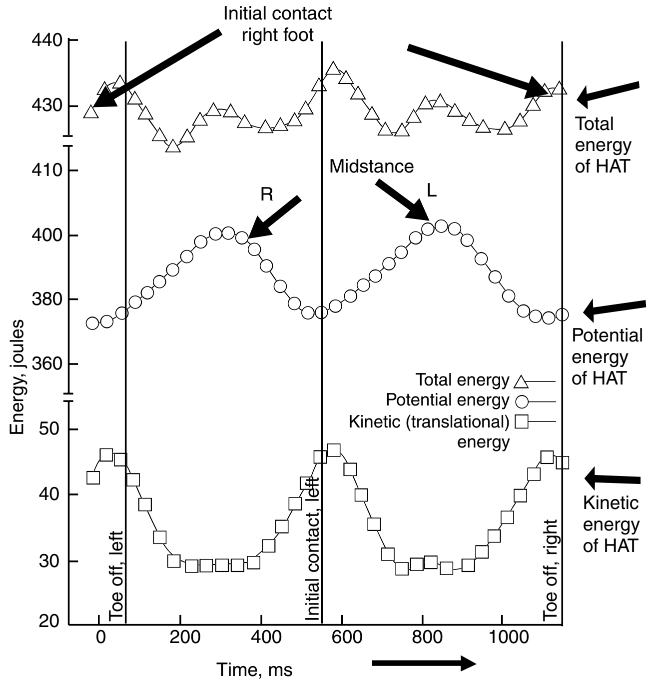

Figure 14-21 Potential and kinetic energy levels of the head, arms, and trunk (HAT) segment, and their sum for one stride in gait. *(Redrawn from Winter DA, Quanbury AO, Reimer GD: Analysis of instaneous energy of normal gait. J Biomech 9:253, 1976, with permission from Elsevier.)*

The muscle work of gait can be simplified by anticipating the muscle groups that must be functioning for specific purposes. Muscle work is logical, and the reader already knows that two major features are going to determine its patterns: **the need to provide a support moment through stance** and **the need to generate energy to move.**

Let us follow the logic of what muscle work around the hip, knee and ankle is expected if we consider what we know about the support moment (see Figs. 14–22 and 14–23). The support moment is made up of some combination of **hip extensors, knee extensors,** and **ankle plantarflexors.** We already have discussed the fact that the **hip extensors** produce an extensor moment early in stance. Next, **knee extensors** produce a knee extensor moment, and then **flexor moments** are produced at both the hip and knee before the hip and knee bend in late stance in preparation for swing phase. While these two muscle groups are flexing the segmented system, the **ankle plantarflexors** take the lead and provide support. We should not be surprised, then, to find activity in the hip extensors **(hamstrings)** early in stance and knee extensors **(quadriceps)** almost immediately after, followed by a smoothly increasing contraction of the **ankle plantarflexors** (**soleus, medial** and **lateral gastrocnemius muscles** and other minor contributors) that continues until mid-push-off and then declines and ceases at about 60% of the cycle.

Now let us look for the muscle work that is responsible for the main bursts of positive work, relating the muscle work shown in Figures 14–22 and 14–23 to power profiles

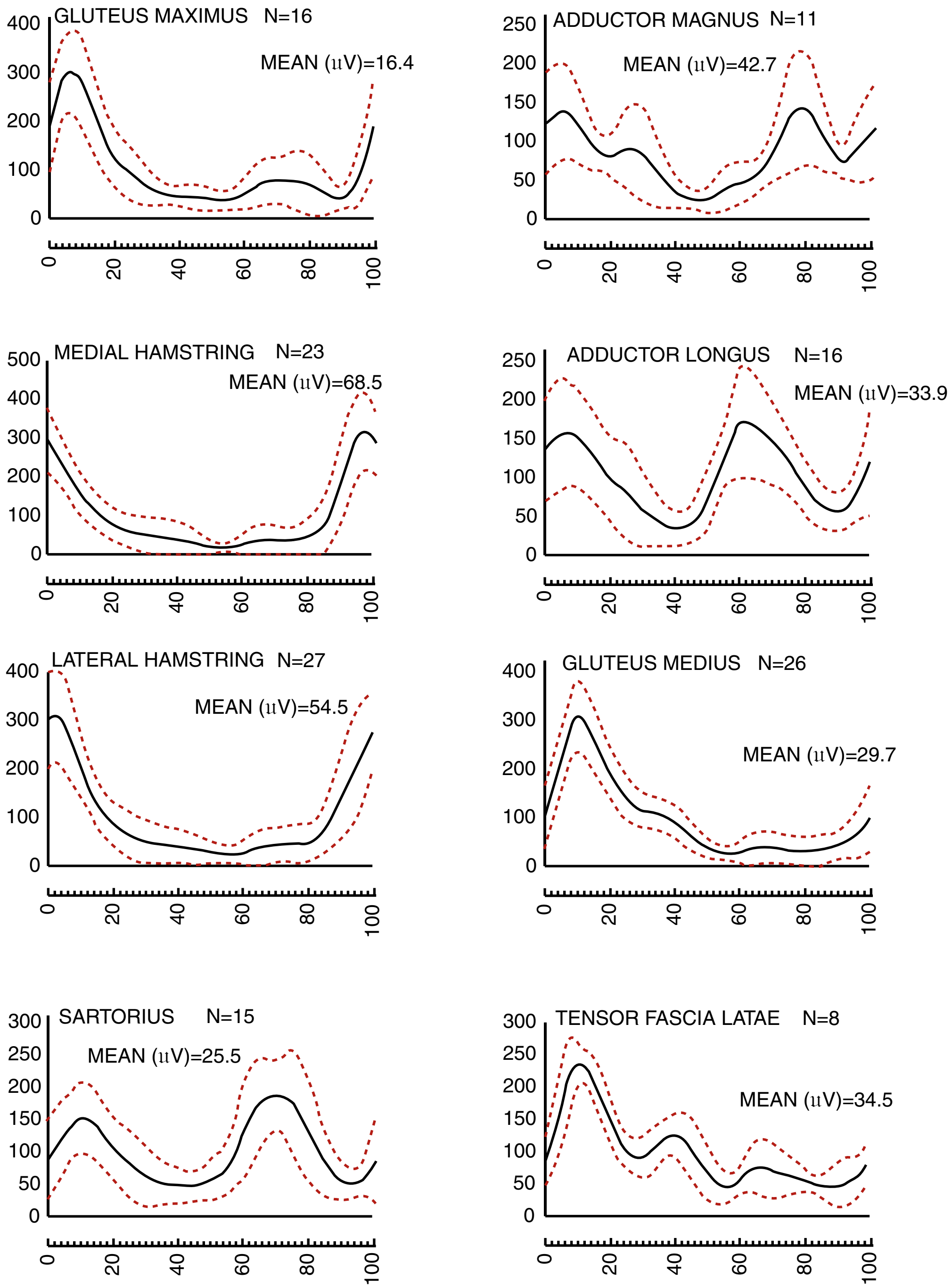

Figure 14-22 Electromyographic activity profiles of major muscle groups of the hip during one stride of gait with probable roles. Signals were derived from bipolar surface electrodes as data normalized to means for each subject. Note absence of the profile for the hip flexor, the iliopsoas muscle, which is inaccessible to surface electrodes. *(Redrawn from Winter DA: The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological [ed. 2]. Waterloo, Ontario, Waterloo Biomechanics, 1991, with permission from David A. Winter.)*

shown in Figures 14–19 and 14–20. In the sagittal plane, we note that the **hip extensors** (**gluteus maximus, medial hamstring,** and **lateral hamstring muscles**) are active in early stance (H1-S); indeed, they were serving the function of providing support during that period. The small energy-generating K2-S that peaks in early stance is reflected in activation of the **quadriceps** (**vastus medialis, vastus lateralis,** and **rectus femoris muscles**). Recall that the largest contribution to the work of walking comes from the **ankle plantarflexors** (largely **soleus, medial,** and **lateral gastrocnemius muscles**). These muscles work eccentrically (lengthening) from early stance until about 40% of the gait cycle, when they are controlling the forward movement of the tibia over the talus as the upper body passes over the foot. At about 40% of the gait cycle, they produce a burst of concentric activity (A2-S) ending at toe-off. A similar sequence of first eccentric and then concentric activity occurs in the **hip flexors: iliopsoas** and **rectus femoris muscle.** First, they lengthen, producing an energy-absorbing contraction as the hip extends (H2-S); then the muscle force overcomes the opposing ground reaction force and begins to act concentrically, causing an energy-generating "pull-off" phase (H3-S). The **iliopsoas** muscle is the major hip flexor, but it is inaccessible to surface electrodes. However, the **rectus femoris** muscle, being the only one of the quadriceps muscle that crosses the hip, also shows hip flexor function, and its activity in late stance correlates with that of iliopsoas in late stance. We can see **rectus femoris** activity

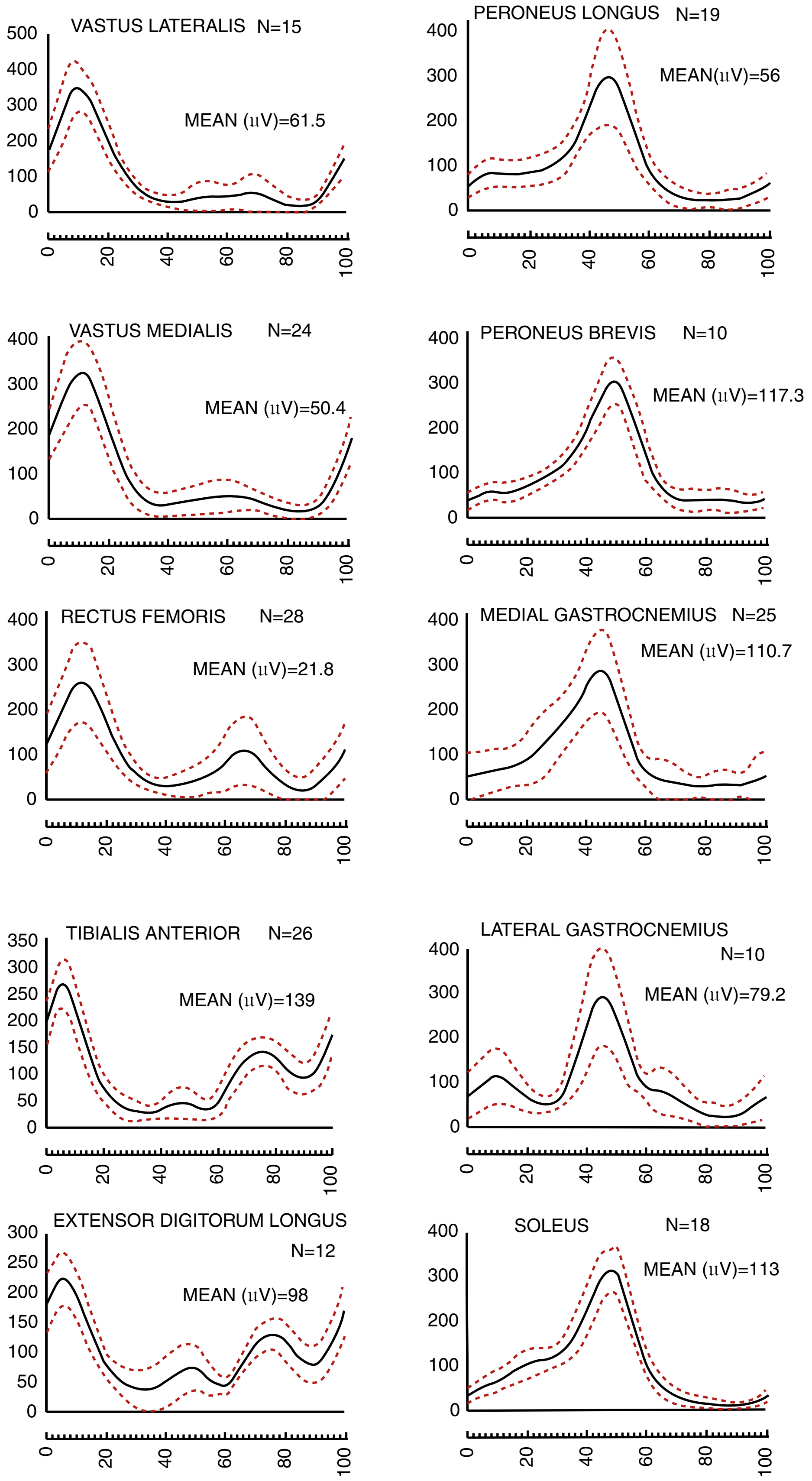

Figure 14-23 Electromyographic activity profiles of major muscle groups of the knee and ankle during one stride of gait with probable roles. Signals were derived from bipolar surface electrodes as data normalized to means for each subject. *(Redrawn from Winter DA: The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological [ed. 2]. Waterloo, Ontario, Waterloo Biomechanics, 1991, with permission from David A. Winter.)*

peaking around 70% of the cycle, reflecting this hip-flexor function.

Now let us look at the major energy-absorbing phases and the muscle groups that are responsible. K1-S, occurring before 20% of the cycle, is the eccentric phase of the **knee extensors** (**vastus lateralis, medialis, intermedius,** and **rectus femoris**), which precedes its concentric K2-S energy-generating phase. We have discussed H2-S, the eccentric action of the **hip flexors** (**iliopsoas** and **rectus femoris**), during midstance and late stance. Note that the **knee extensor** and the **hip extensor** muscles begin their contraction at the end of swing phase, although at this time the dominant moments are being provided by the knee flexors. K3-S is a small energy-absorbing internal knee extensor moment occurring while the knee is flexing rapidly (~50% to 70% of cycle), and this is reflected in low levels of contraction of the **vastus muscles,** particularly the **rectus femoris,** which, because it crosses both hip and knee, is active at that time as a **hip flexor** (H3-S). K4-S, energy absorption of the **knee flexors** at the end of swing, is reflected in EMG records as activation of the **medial** and **lateral hamstrings.** Note that the **gastrocnemius** muscles, which cross the knee as well as the ankle, also begin activity in late swing phase. At the ankle, A1-S absorption through much of early stance and midstance is attributable to **ankle plantarflexor** activity, which we have already discussed.

Now let us see whether we have missed any major EMG features shown in Figures 14–22 and 14–23 by deducing muscle activity from what we know about support moment and power profiles. First, in the sagittal plane, we have not considered the **ankle dorsiflexors** (**tibialis anterior, extensor digitorum longus,** and **extensor hallucis longus muscles**), which are eccentrically active in early stance before foot flat and act to lower the foot to the floor. They are

Continuing Exploration 14-6:

Ground Reaction Force and Muscle Activity

Before full dynamic biomechanical gait analyses were common, attempts were made to link the static information from the GRFV to the joint positions during gait in a visual way. There are errors in this kind of analysis because dynamic factors are not included, but during stance phase, these are minimal. Also, errors caused by using the GRFV are less at joints that are nearer the force platform. Errors are small at the ankle but become larger at the hip, especially at times of push-off and initial contact.[55] If they are used to attempt to reconcile with the internal moment profiles of Figures 14–11, 14–12, 14–16, and 14–18, however, they can add important understanding and helpful visualization of normal gait for stance phase of gait (of course, there is no GRF during swing phase). The general sequence of the most common pattern of GRFV in the sagittal plane for stance phase is shown in Figure 14–11 (initial contact to midstance) and Figure 14–12 (midstance to toe-off). These should be related to the moment profiles appearing in Figures 14–16 and 14–18. The GRFVs, joint positions, and muscle activity that were used to create the illustrations were derived from published studies on normal human walking.[4,16]

CASE APPLICATION

Hip and Knee Flexors

case 14–6

At heel-off (~40% of gait cycle), the GRFV tending to extend the hip and knee (see Fig. 14–12) is consistent with the opposition provided by the hip flexor moment (iliopsoas and rectus femoris muscles) and the knee flexor moment (gastrocnemius muscles) (see Fig. 14–16). Consider the effects of a larger than normal tendency to extend the knee, which occurred, for example, when Ms. Brown thrust her knee back into full extension in an effort to gain knee stability (Fig. 14–25). Because the knee flexors that are active at that time (gastrocnemius muscles) did not overcome this excessive moment, Ms. Brown had difficulty flexing the knee, which prevented flexion of both the hip and the ankle and reduced the opportunity to generate work from the ankle at A2-S and the hip at H3-S. Avoiding this "knee locking" in stance by rigorous encouragement of knee flexion at the end of stance and by temporary use of an ankle-foot orthosis was important in Ms. Brown's gait reeducation.

Concept Cornerstone **14-3**

What Gait Information Is Important?

Given the availability of information about walking, it is often not clear what information is helpful for any given situation. First, it is important to determine why you want the information. Usually the reasons include one or more of the following: (1) to gain an understanding of normal or pathological gait; (2) to assist movement diagnosis and identify specific causes of pathological gait; (3) to inform treatment selection; and (4) to evaluate the effectiveness of treatment. Second, you may ask what gait measures are important for the situation. For example, if you want to know whether energy costs are decreased with provision of an ankle orthosis, a measure of self-selected speed of walking may be sufficient. If you want to know which muscle groups could be exploited to gain increased walking speed in treating a person with a neurological condition, you would want to see a power analysis. If you wished to know whether a new ankle-foot orthosis really did return energy during push-off, you would also want a power analysis. If you wanted to determine whether surgery to realign the tibia and fibula was successful in decreasing a varus or valgus moment on the knee, you would want a moment analysis in the frontal plane. There is a tendency for power analyses to be more useful in neurological conditions and moment analyses in musculoskeletal conditions, particularly those involving pain.

active again during swing phase to hold the foot at a neutral angle, and show varying but small levels of activity at other times of the cycle, probably positioning the foot. We have not so far noted the activity in the frontal plane of the hip abductor **gluteus medius,** along with **gluteus minimus** and **tensor fasciae latae** muscles (not shown). These muscles control the lateral drop of the pelvis on the side of the swinging leg. Activity of these muscles diminishes during midstance and ceases when the opposite limb has contacted the ground.[14]

We have so far not noticed **adductor longus** and **brevis muscles,** which act in both the frontal and sagittal planes, and show two fairly equal peaks of activity at ~10% and 65% to 80% of the gait cycle. The first peak is concurrent with the **hip abductors** and may be providing stabilization of the hip joint; the second occurs early in swing, providing hip flexion to assist **iliopsoas** and **rectus femoris muscles.** Further information can be found in the literature.[37,39,40] Trunk muscle activity is discussed in a later section.

Table 14–3 **Summary of Gait Characteristics From Initial Contact to Midstance**

SAGITTAL PLANE ANALYSIS

Joint	*Motion*	*Ground Reaction Force*	*Internal Moment*	*Power*	*Major Muscle Activity*
Hip	Extends from +20°–+30° to 0°	Anterior to posterior	Extensor to neutral	Generation (hip extensors)	Gluteus maximus Hamstrings
Knee	Flexes from 0° to +15°, then extends +15° to +5°	Anterior to posterior to anterior	Flexor, then extensor then flexor	Generation (knee flexors) Absorption (knee extensors) Generation (knee extensors) Absorption (knee flexors)	Hamstrings Quadriceps Hamstrings Gastrocnemius
Ankle	Plantarflexes from 0° to −5°, then dorsiflexes to +5°	Posterior to anterior	Dorsiflexor, then plantarflexor	Absorption (dorsiflexors) Generation (plantarflexors)	Tibialis anterior Soleus Gastrocnemius

FRONTAL PLANE ANALYSIS

Joint	*Motion*	*Ground Reaction Force*	*Moment*	*Power*	*Major Muscle Activity*
Hip	Adduction from neutral to +5°, then abduction to 3°	Medial	Abductor	Absorption (hip abductors) Generation (hip abductors)	Gluteus medius
Knee	Small variation around neutral	Medial	Abductor	Small generation (knee abductors)	Tensor fasciae latae
Ankle	Inconsistent				

TRANSVERSE PLANE ANALYSIS

Joint	*Motion*	*Moment*	*Power*	*Major Muscle Activity*
Hip	Externally rotates a few degrees	External rotator	Absorption (internal rotators)	Tensor fasciae latae (Ligamentous) Gluteus medius Gluteus minimus
Knee	From several degrees of external rotation, rotates internally to neutral	Very small external rotator	Small absorption (external rotators)	
Ankle	Inconsistent			

Redrawn from Eng JJ, Winter DA: Kinetic analysis of the lower limbs during walking: What information can be gained from a three dimensional model? J Biomech 28:753, 1995; and from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA (eds): Gait Analysis: Theory and Application. St. Louis, MO, Mosby–Year Book, 1994.
Values are for young males.

Table 14–4 Summary of Gait Characteristics From Midstance to Toe-Off

SAGITTAL PLANE ANALYSIS

Joint	*Motion*	*Ground Reaction Force*	*Moment*	*Power*	*Major Muscle Activity*
Hip	Extends from 0° to −20° extension, then begins to flex	Posterior	Flexor	Absorption (flexors) Generation (flexors)	Iliopsoas Rectus femoris Iliopsoas Rectus femoris
Knee	From +5° extends a few degrees, then flexes to about +45°	Posterior	Flexor, then extensor	Absorption (extensors) Small generation (knee flexors) Large absorption (extensors)	Vastus Gastrocnemius Rectus femoris
Ankle	From +5° dorsiflexion, dorsiflexes a few more degrees, then rapidly plantarflexes to −25°	Anterior	Plantarflexor	Very small absorption (plantarflexors) Large generation (plantarflexors)	Soleus Gastrocnemius Soleus Gastrocnemius

FRONTAL PLANE ANALYSIS

Joint	*Motion*	*Ground Reaction Force*	*Moment*	*Power*	*Major Muscle Activity*
Hip	From +3° smoothly abducts to −5°	Medial	Abductor	Generation (abductors)	Gluteus medius
Knee	Neutral, then abducts to a few degrees −3°	Medial	Abductor	Absorption (abductors)	Tensor fasciae latae, ligaments
Ankle	Inconsistent				

TRANSVERSE PLANE ANALYSIS

Joint	*Motion*	*Moment*	*Power*
Hip	From −3°, external rotation a few more degrees	Internal rotator	Very small absorption (external rotators)
Knee	Remains near neutral	Internal rotator	Very small absorption (ligamentous)
Ankle	Inconsistent		

Redrawn from Eng JJ, Winter DA: Kinetic analysis of the lower limbs during walking: What information can be gained from a three dimensional model? J Biomech 28:753, 1995; and from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA (eds): Gait Analysis: Theory and Application. St. Louis, MO, Mosby–Year Book, 1994.
Values are for young males.

Gait Initiation and Termination

The previous sections have discussed gait as a continuing activity. However, both starting to walk and stopping walking require a certain sequence of motor events. These events are interesting because they are quite different from continuing gait, they are potentially destabilizing to a person's balance, and yet they are essential to the function of walking.

Gait initiation is defined as a stereotyped activity that includes the series or sequence of events that occurs from the initiation of movement to the beginning of the gait cycle. Gait initiation begins in the erect standing posture with an inhibition of the gastrocnemius and soleus muscles closely followed by activation of the tibialis anterior muscles.[56] Bilateral concentric contractions of the tibialis anterior muscles (pulling the tibias forward over stable feet) results in a sagittal moment that inclines the body anteriorly from the ankles. Initially, the CoP shifts posteriorly, and briefly, toward the first swing foot from its resting location between the feet and anterior to the ankle joints. As the heel of the

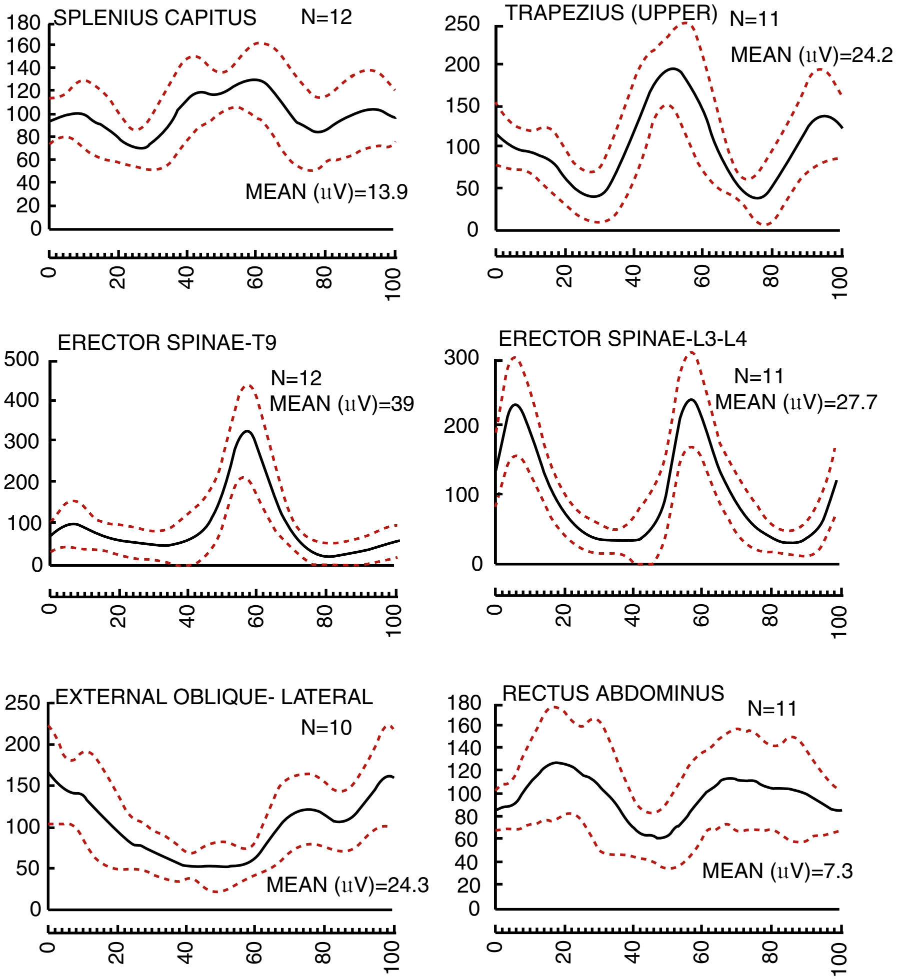

Figure 14-24 Electromyographic activity profiles of major muscle groups of the trunk during one stride of gait. Signals were derived from bipolar surface electrodes as data normalized to means for each subject. *(Redrawn from Winter DA: The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological [ed. 2]. Waterloo, Ontario, Waterloo Biomechanics, 1991, with permission from David A. Winter.)*

swing foot lifts off the floor the CoP moves rapidly to the stance foot heel; then, as the swing foot toe lifts off the floor, it progresses anteriorly to the forefoot.[6] Abduction of the swing hip, with activation of the gluteus medius, occurs almost simultaneously with contractions of the tibialis anterior. As weight is transferred to the stance limb, activiaton of gluteus medius on the stance side begins. According to Elble and colleagues,[57] the support limb hip and knee flex a few degrees (3° to 10°), and the CoP moves anteriorly and medially toward the support limb. A healthy individual may initiate gait with either the right or left lower extremity, and no changes will be seen in the pattern of events. The temporal and spatial patterns of gait initiation are preserved in the elderly, though there is a trend for displacements to be smaller and velocities lower.[6]

Termination of gait has received much less attention than has initiation (for a review, see Sparrow and Tirosh, 2004).[7] Anticipation of stopping appears to influence EMG activity even before initial contact.[58] The body attempts to maintain the stance limb anterior to the whole body center of mass, which is initiated first in the stance limb by reducing push-off and activating hip extensors and knee extensors as energy absorbers. The reduced push-off in the stance limb is accomplished by inhibition of soleus and strong activation of the tibialis anterior.[58] In the swing limb, the vastus lateralis is usually activated first.[7] Braking forces are then produced by the swing limb at initial contact with a large soleus muscle burst to control ankle plantarflexion, and reduced activity in the tibialis anterior to inhibit ankle dorsiflexion. To maintain knee extension and stabilize the trunk the vastus lateralis activity is continued and the erector spinae are activated.

CASE APPLICATION

Avoiding Instability in Initiation of Gait

case 14–7

Patients with hemiparesis, like Ms. Brown, demonstrate some differences in gait initiation depending on whether the step is started with the affected or unaffected limb. When the person stands on the unaffected limb and steps forward with the affected leg, the timing and pattern of events are practically the same as they would be in a nonaffected person, but when the person with paresis attempts gait initiation with the nonaffected leg, the weight must first be shifted onto the affected side. The result is an erratic pattern of events, and stability can be seriously threatened.[59]

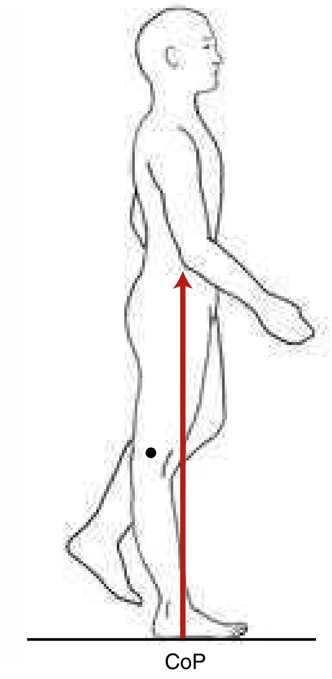

Figure 14-25 GRFV passing excessively anterior to knee joint center, making it difficult for the person to flex the knee.

TRUNK AND UPPER EXTREMITIES

Trunk

The trunk remains essentially in the erect position during normal free-speed walking on level ground, varying only 1.5°.[60] Krebs and coworkers[61] found that a flexion peak of low amplitude occurred near each heel strike and an extension peak of low amplitude occurred during single limb support. Winter explained[17] that at initial contact, the forward acceleration of the head, arms, and trunk is quite large and acts at a distance from the hip joint, thus producing an unbalancing moment that acts strongly to cause flexion of the trunk during weight acceptance. However, an almost equal and opposite moment is provided by the hip extensors (which we have seen before in their role in support and in energy generation at H1-S). In the frontal plane, a similar situation exists. The center of mass of the head, arms, and trunk, always being medial to the hip joint, exerts a considerable moment that is balanced primarily by an internal hip abductor moment from the supporting limb (see Fig. 14–18) and is assisted by the medial acceleration of the hip.

The biomechanical models used previously in the chapter do not distinguish between the pelvis and the trunk. In the sagittal plane, the pelvis moves in a sinusoidal curve up and down 4 or 5 cm with each step, the low point coinciding with initial contact of each foot and the high point coinciding with midstance. In the frontal plane, the pelvis translates from side to side about the same amount toward the standing limb and rotates downward about 5° toward the swinging limb on each side. In the transverse plane, looking at the pelvis from above, it rotates 4° to 8° counterclockwise during swing phase of the right limb, goes through neutral position about midstance, then continues rotating the same amount in preparation for left foot initial contact. When the pelvis rotates counterclockwise, preparing for initial contact of the right foot, the trunk rotates clockwise with regard to the pelvis to keep it directed forward. The amount of transverse rotation of the trunk during gait is slight and occurs primarily in a direction opposite to the direction of pelvic rotation (Fig. 14–26). As the pelvis rotates forward with the swinging lower extremity, the thorax on the opposite side rotates forward as well. Actually, the thorax undergoes a biphasic rotation pattern with a reversal directly after liftoff of the stance leg. The thorax is rotated backward during double support and then slowly rotates forward during single support. This trunk motion helps prevent excess body motion and helps counterbalance rotation of the pelvis. Krebs and coworkers found that at a free speed of gait, transverse rotation reached a maximum of 9° at 10% of the cycle after each heel strike.[61] In a study of treadmill walking, Stokes and associates[62] found that the movements and interactions of the trunk and pelvis were extremely complex when translatory and rotatory movements of the trunk were considered along with anterior and posterior pelvic tilting, lateral pelvic tilting, and rotation. Mediolateral translations of the trunk occur as side-to-side motions (leans) in relation to the pelvis. For example, the trunk is leaning or moving to

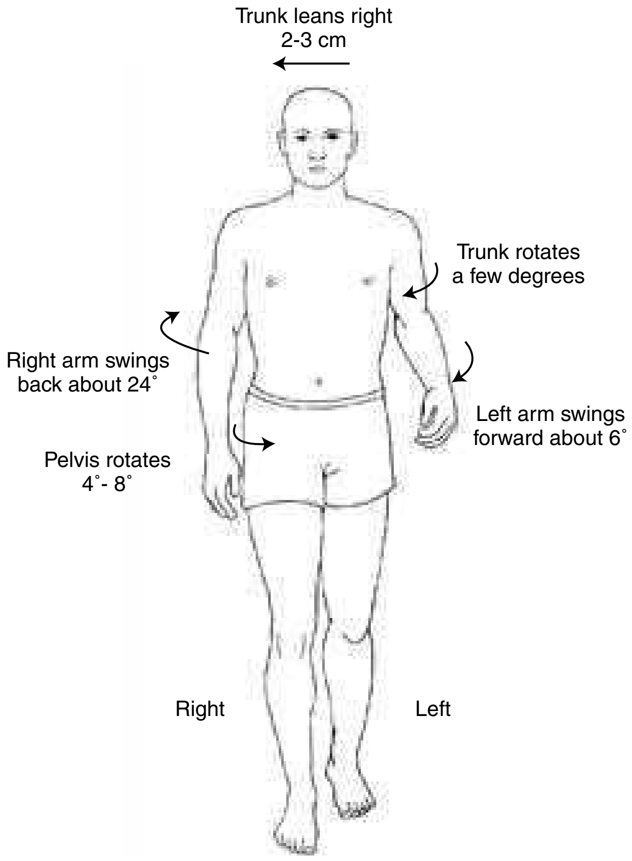

Figure 14-26 Pelvis, trunk, and arm motion. Note that the trunk and arms rotate in a direction opposite to that of the pelvis.

the right from right heel strike to left toe-off, at which point the trunk begins a lean to the left until right toe-off. The average total ROM that occurs during the mediolateral trunk leans is about 5.4 cm.[62] Hirasaki and coworkers[63] used a treadmill and a video-based motion analysis system to study trunk and head movements at different walking speeds. These authors found that the relationship between walking speed and head and trunk movements was the most linear in the range of walking speeds from 1.2 to 1.8 m/sec. At velocities above and below this range, head and trunk movements were less well coordinated.

EMG profiles and probable functional roles are shown for some trunk muscles in Figure 14–24. Recent EMG studies on the trunk muscles during gait have shown that there are subgroups of subjects who show similar patterns of muscle activity[64] when cluster analysis is used on the EMG data. This applies to the **internal oblique, external oblique, rectus abdominis,** and **lumbar erector spinae** muscles. Although most subjects showed low levels of activity throughout the gait cycle, the **internal oblique** and **erector spinae muscles** had more distinct bursts, usually occurring close to initial contact. Some researchers have shown two periods of activity[65] for the **erector spinae muscle,** one at initial contact and the second at toe-off. It is thought that its function is to oppose the unbalancing moment that acts strongly to cause flexion of the trunk during weight acceptance.

Upper Extremities

Detailed kinetics of the upper extremity during normal gait have not been reported, although extensive mapping of EMG was classically performed several decades ago.[65,66] Although the lower extremities are moving alternately forward and backward, the arms are swinging rhythmically. However, the arm swinging is opposite to that of the legs and pelvis but similar to that of the trunk (see Fig. 14–26). The right arm swings forward with the forward swing of the left lower extremity while the left arm swings backward. This swinging of the arms provides a counterbalancing action to the forward swinging of the leg and helps to decelerate rotation of the body, which is imparted to it by the rotating pelvis. The total ROM at the shoulder is not very large. At normal free velocities, the ROM is only approximately 30° (24° of extension and 6° of flexion). It is clear, however, that resticting arm motion increases energy costs of walking at prescribed speeds.[67]

The normal shoulder motion is the result of the combined effects of gravity and muscle activity. During the *forward* portion of arm swinging, the following medial rotators are active: **subscapularis, teres major,** and **latissimus dorsi muscles.** In *backward* swing, the **middle** and **posterior deltoid muscles** are active throughout, and the **latissimus dorsi** and **teres major muscles** are active only during the first portion of backward swing.[65] The **supraspinatus, trapezius,**[65] and **posterior** and **middle deltoid muscles**[65] are active in both backward and forward swing. It is interesting to note that little or no activity was reported in the **shoulder flexors** in these studies.[65,66] It appears that during forward swing, the **medial rotators** are acting eccentrically to control external rotation of the arm at the shoulder as the **posterior deltoid** acts eccentrically to restrain the forward swing. The **latissimus dorsi** and **teres major muscles,** as well as the **posterior deltoid,** may then act concentrically to produce the backward swing. The role of the **middle deltoid** is unclear, although it has been suggested that it functions to keep the arm abducted so that it may clear the side of the body.[45] Activity in all muscles increases as the speed of gait increases.[65]

Recent work supports neuronal coordination of arm and leg movement during locomotion. Deitz and colleagues[68] and Wannier and colleagues[69] reported the behaviors of the arm to leg corresponding to a system of two coupled oscillators. Results were compatible with the assumption that the proximal arm muscles are associated with the swinging of the arms during gait as a residual function of quadripedal locomotion.

TREADMILL, STAIR, AND RUNNING GAITS

Treadmill Gait

Treadmills used for gait measurement and training have a number of advantages including a small footprint, the availability of weight support for patient safety, and increasing sophistication of instrumentation that may include metabolic analysis as well as embedded force platforms. Comparisons of treadmill with overground walking have shown higher cadence and shorter stance times at comparable speeds,[70,71] although others do not report these differences.[72,73] Differences in joint ranges of motion have also been reported, but these have usually been less than 2.5° and not considered important.[70,74] Kinetic comparisons have revealed similar joint moments for treadmill and overground walking, although push-off forces and GRF maxima have generally been lower with treadmill walking.[73,74,75] It is still not certain whether metabolic costs are different. Whereas Pearce and colleagues[75] reported lower oxygen uptake in association with treadmill walking compared to overground walking at the same speed in young and older men, Parvataneni and colleagues[76] found significantly higher metabolic costs on the treadmill in healthy older adults when speeds on the treadmill were matched with overground speeds.[76] These findings are consistent with reports of higher heart rates by Grieg and colleagues.[72] More research is needed to assess energetic differences between treadmill and overground walking across age groups, and to determine their cause.

Stair Gait

Ascending and descending stairs is a basic body movement required for performing normal activities of daily living such as shopping, using public transportation, or simply getting around in a multistory home or building. Although many similarities exist between level-ground locomotion and stair locomotion, the difference between the two modes of locomotion may be significant for a patient population.

The fact that a patient has adequate muscle strength and joint ROM for level-ground walking does not ensure that the patient will be able to walk up and down stairs. Stair walking represents additional stress over level-ground walking and may reveal differences that are not apparent in level-ground walking.[77]

Krebs and coworkers[61] found that trunk ROM during level-ground gait was similar to trunk ROM during stair descent but differed from trunk ROM during stair ascent in all planes. The maximum ROM of trunk flexion in relation to the room during stair ascent was at least double the amount of trunk flexion found in either stair descent or in level-ground walking.[61]

Locomotion on stairs is similar to level-ground walking in that stair gait involves both swing and stance phases in which forward progression of the body is brought about by alternating movements of the lower extremities. Also, in both stair and level-ground gait, the lower extremities must balance and carry along the head, arms, and trunk. McFayden and Winter[8] (using step dimensions of 22 cm for the stair riser and 28 cm for the tread) performed a sagittal plane analysis of stair gait.[8] These investigators collected kinetic and kinematic data for one subject during eight trials. The stair gait cycle for stair ascent presented in Figure 14–27 is based on data from McFayden and Winter's study.[8] Although ankle moments were aproximately the same, the knee extensor moment in both stair ascent and descent was approximately three times larger than that of level-ground walking. This may cause particular mobility problems in patients with knee pain, which is common in such conditions as osteoarthritis and patello-femoral pain syndrome. Although the knee has primarily an absorptive function in level walking, it has a large generative role in stair ascent. Powers are largely generative in stair ascent and absorptive in descent for all joints.

The investigators divided the stance phase of the stair gait cycle into the three subphases and the swing phase into two subphases.[8] The subdivisions of the stance phase are weight acceptance, pull-up, and forward continuance. The subdivisions of the swing phase are foot clearance and foot placement. As can be seen in Figure 14–27, weight acceptance comprises approximately the first 14% of the gait cycle and is somewhat comparable to the heel strike throughout the loading phase of walking gait. However, in contrast to walking gait, the point of initial contact of the foot on the stairs is usually located on the anterior portion of the foot and travels posteriorly to the middle of the foot as the weight of the body is accepted. The pull-up portion, which extends from approximately 14% to 32% of the gait cycle, is a period of single-limb support. The initial portion of pull-up is a time of instability, inasmuch as all of the body weight is shifted onto the stance extremity when it is flexed at the hip, knee, and ankle. During this period, the task is to pull the weight of the body up to the next stair level. The knee extensors are responsible for most of the energy generation required to accomplish pull-up. The forward continuance period is from approximately 32% to 64% of the gait cycle and corresponds roughly to the midstance through toe-off subdivisions of walking gait. In the forward continuance period, the greatest amount of energy is generated by the ankle plantarflexors. Data regarding joint ROM and muscle activity for ascending stairs are presented in Tables 14–5, 14–6, and 14–7.[8] Similar findings have been reported by Protopapadaki,[78] Costigan,[79] Salsich,[80] and their colleagues. Variations between studies have been attributed to differences in stair dimensions, trunk inclination, foot contact positions, and differences in subject characteristics and methodologies.

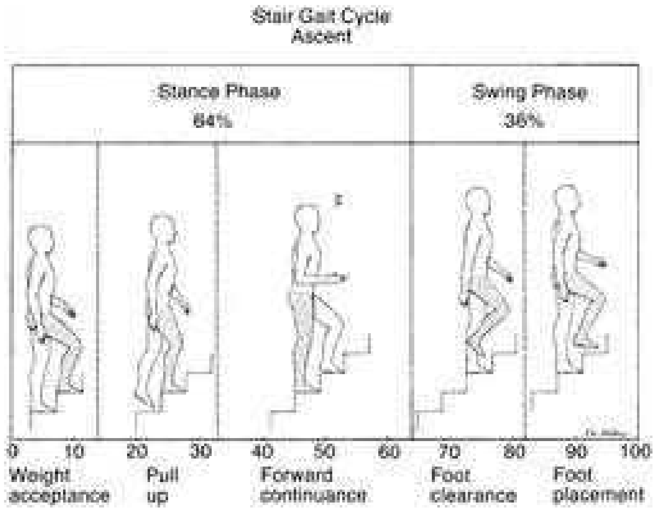

Figure 14-27 Stair gait cycle.

Concept Cornerstone **14-4**

Differences Between Level-Ground Gait and Stair Gait

Table 14–6 shows that considerably more hip and knee flexion are required in the initial portion of stair gait than are required in normal level-ground walking. Therefore, a patient would require a greater ROM for stair climbing (the same stair dimensions and slope) than for normal level-ground walking. Naturally muscle activity and joint ROMs will change if stairs of other dimensions are used.[8]

Ascending stairs involves a large amount of positive work that is accomplished mainly through concentric action of the rectus femoris, vastus lateralis, soleus, and medial gastrocnemius muscles. Descending stairs is achieved mostly through eccentric activity of the same muscles and involves energy absorption. The support moments during stair ascent, stair descent, and level-ground walking exhibit similar patterns; however, the magnitude of the moments is greater in stair gait, and, consequently, more muscle strength is required. Kirkwood and colleagues[81] found that the maximum

Table 14–5 Sagittal Plane Analysis of Stair Ascent (Fig. 14–29): Stance Phase–Weight Acceptance (0%–14% of Stance Phase) Through Pull-Up (14%–32% of Stance Phase)

JOINT	MOTION	MUSCLE	CONTRACTION
Hip	Extension: 60°–30° of flexion	Gluteus maximus Semitendinosus Gluteus medius	Concentric
Knee	Extension: 80°–35° of flexion	Vastus lateralis Rectus femoris	Concentric
Ankle	Dorsiflexion: 20°–25° of dorsiflexion Plantarflexion: 25°–15° of dorsiflexion	Tibialis anterior Soleus Gastrocnemius	Concentric Concentric

Table 14–6 Sagittal Plane Analysis of Stair Ascent (Fig. 14–29): Stance Phase–Pull-Up (End of Pull-Up) Through Forward Continuance (32%–64% of the Stance Phase of Gait Cycle)

JOINT	MOTION	MUSCLE	CONTRACTION
Hip	Extension: 30°–5° flexion Flexion: 5° to 10°–20° of flexion	Gluteus maximus Gluteus medius Semitendinosus Gluteus maximus Gluteus medius	Concentric and isometric Eccentric
Knee	Extension: 35°–10° of flexion Flexion: 5° to 10°–20° of flexion	Vastus lateralis Rectus femoris Rectus femoris Vastus lateralis	Concentric Eccentric
Ankle	Plantarflexion: 15° of dorsiflexion to 15°–10° of plantarflexion	Soleus Gastrocnemius Tibialis anterior	Concentric Eccentric

Table 14–7 Sagittal Plane Analysis of Stair Ascent (Fig. 14–29): Swing Phase (64%–100% of Gait Cycle)—Foot Clearance Through Foot Placement

JOINT	MOTION	MUSCLE	CONTRACTION
Hip	Flexion: 10°–20° to 40°–60° of flexion Extension: 40°–60° of flexion to 50° of flexion	Gluteus medius	Concentric
Knee	Flexion: 10° of flexion to 90°–100° of flexion Extension: 90°–100° of flexion to 85° of flexion	Semitendinosus Vastus lateralis Rectus femoris	Concentric Concentric
Ankle	Dorsiflexion: 10° of plantarflexion to 20° of dorsiflexion	Tibialis anterior	Concentric and isometric

peak internal abductor moment at the hip occurred during descending stairs.

Running Gait

Running is another locomotor activity that is similar to walking, but certain differences need to be examined. As in the case of stair gait, a patient who is able to walk on level ground may not have the ability to run. Running requires greater balance, muscle strength, and ROM than does normal walking. Greater balance is required because running is characterized not only by a considerably reduced base of support but also by an absence of the double-support periods observed in normal walking and the presence of float periods in which both feet are out of contact with the supporting surface (Fig. 14–28). The walking gait cycle in Figure 14–2 can be used to compare the gait cycle in walking with running gait. The percentage of the gait cycle spent in float periods will increase as the speed of running increases. Muscles must generate greater energy both to raise the head, arms, and trunk higher than in normal walking and to balance and support them during the gait cycle. Muscles and joint structures also must be able to absorb more energy to accept and control the weight of the head, arms, and trunk.

For example, in normal level-ground walking, the magnitudes of the vertical GRFs at the CoP at initial contact are approximately 70% to 80% of body weight and rarely exceed 120% of body weight during the gait cycle.[82,83] However, during running, the GRFs at the CoP have been shown to reach 200% of body weight and increase to 250% of body weight during the running cycle. Furthermore, the knee is flexed at about 20° when the foot strikes the ground. This degree of flexion helps to attenuate impact forces but also increases the forces acting at the patellofemoral joint. In addition, the base of support in running is considerably less than in walking. A typical base of support in walking is about 2 to 4 inches, whereas in running, both feet fall in the same line of progression, and so the entire center of mass of the body must be placed over a single support foot. To compensate for the reduced base of support, the functional limb varus angle increases. Functional limb varus angle is the angle between the bisection of the lower leg and the floor.[89] According to McPoil and Cornwall,[84] the functional limb varus angle increases about 5° during running in comparison with walking.

Joint Motion and Muscle Activity

Joint Motion

The ROM varies according to the speed of running and among different researchers. Comparisons in joint angles, moments, and powers between walking and running are shown in Figure 14–29.[85] At the beginning of the stance phase of running, the hip is in about 45° of flexion at heel strike and extends during the remainder of the stance phase until it reaches about 20° of hyperextension just after toe-off.[84] The hip then flexes to reach about 55° to 60° of flexion in late swing. Just before the end of the swing phase, the hip extends slightly to 45° to 50° in preparation for heel strike.[62] The knee is flexed to about 20° to 40° at heel strike and continues to flex to 60° during the loading response. Thereafter, the knee begins to extend, reaching 40° of flexion just before toe-off. During the swing phase and initial float period, the knee flexes to reach a maximum of approximately 125° to 130° in the middle of the swing phase. In late swing, the knee extends to 40° in preparation for heel strike.[83] In Figure 14–29, note these differences in joint angles from level-ground walking. At each joint, the maxima for running exceed those of walking. In the case of the hip, only flexion range is increased. The knee range in running is not very different from that in walking, but it takes place in approximately 25° more flexion. The ankle has greatly increased dorsiflexion and modestly increased plantarflexion.

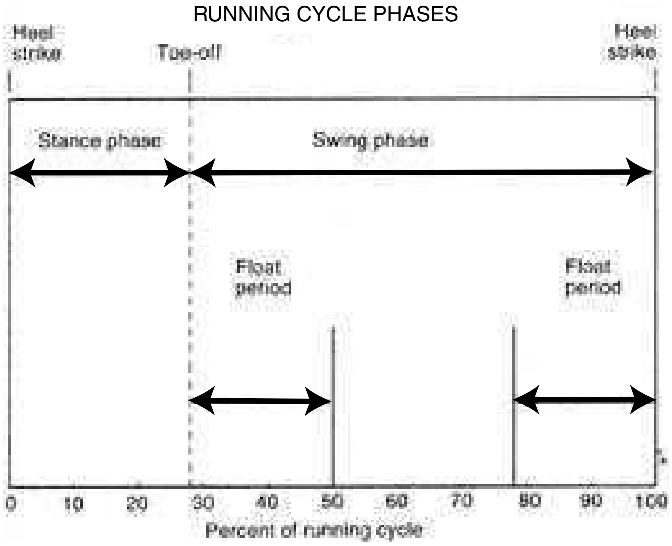

Figure 14-28 Running gait cycle.

The ankle is in about 10° of dorsiflexion at heel strike and rapidly dorsiflexes to reach about 25° to 30° dorsiflexion. The rapid dorsiflexion is followed immediately by plantarflexion, which continues throughout the remainder of the stance phase and into the initial part of the swing phase. Plantarflexion reaches a maximum of 25° in the first few seconds of the swing phase. Throughout the rest of the swing phase, the ankle dorsiflexes to reach about 10° in late swing in preparation for heel strike.[83]

The whole extremity begins to medially rotate during the swing phase. At heel strike, the extremity continues to medially rotate and the foot pronates. Lateral rotation of the stance extremity and supination of the foot begins as the swing leg passes the stance limb in midstance. The ROM in the lower extremities needed for running, in comparison with the ROM required for normal walking, is presented in Table 14–8. The largest differences in the total ROM requirements between the two activities appear to be at the knee and hip joints. At the knee joint, up to an additional 90° of flexion is required for running versus walking. At the hip joint, running requires about twice the amount of motion that was needed for normal walking.

Muscle Activity

The **gluteus maximus** and **gluteus medius** muscles are active both at the beginning of the stance phase and at the end of the swing phase. The **tensor fasciae latae** muscle is also active at the beginning of stance and at the end of swing but also is active between early and midswing. The **adductor magnus** muscle shows activity for about 25% of the gait cycle from late stance through the early part of the swing phase. Activity in the **iliopsoas** muscle occurs for about the same percentage of the gait cycle as the **adductor longus** muscle, but iliopsoas activity also occurs during the swing phase from about 35% to 60% of the gait cycle.

The **quadriceps** muscle acts eccentrically during the first 10% of the stance phase to control knee flexion when the knee is flexing rapidly. The quadriceps ceases activity after

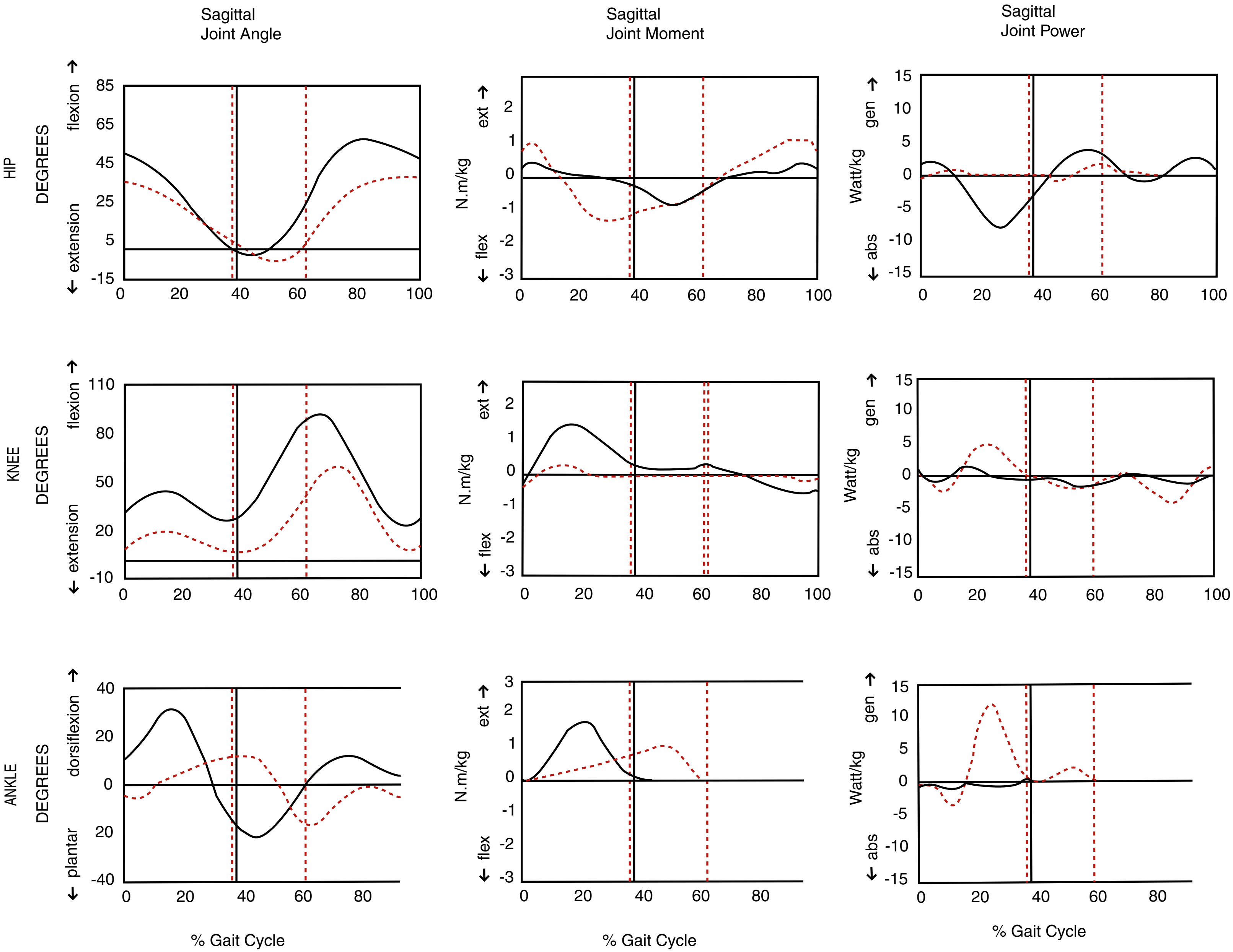

Figure 14-29 Sagittal plane joint angles, moments, and powers for running (solid line) and walking (dotted line). Joint angles are plotted with flexion and dorsiflexion positive. Moments are internal moments normalized to body mass, plotted with extensor and plantarflexor positive. Powers are normalized to body mass, plotted with generation positive and absorption negative. The vertical solid line near 40% of gait cycle represents toe-off for running; the vertical dotted line near 60% of gait cycle represents toe-off for walking. *(Joint angles, moments, and powers for running redrawn from Novacheck TF: The biomechanics of running. Gait Posture 7:77, 1998, with permission from Elsevier.)*

Table 14–8 Average Peak ROM at the Hip, Knee, and Ankle: Comparison Between Running[69–71] and Walking[8,71]

RUNNING		WALKING	
Hip joint		Hip joint	
Flexion	55°–65°	Flexion	30°
Extension	10°–20°	Extension	0°–20°
Knee joint		Knee joint	
Flexion	80°–130°	Flexion	40°–50°
Extension	0°–5°	Extension	0°
Ankle joint		Ankle joint	
Dorsiflexion	10°–30°	Dorsiflexion	10°
Plantarflexion	20°–30°	Plantarflexion	20°

the first part of stance, and no activity occurs until the last 20% of the swing phase, when concentric activity begins to extend the knee (to 40° of flexion) in preparation for heel strike. The **medial hamstrings** are active at the beginning of stance and through a large part of swing. For example, the medial hamstrings are active from 18% to 28% of the stance phase, from about 40% to 58% of initial swing, and for the last 20% of swing. During part of this time, the knee is flexing and the hip is extending, and the hamstrings may be acting to extend the hip and to control the knee. During initial swing, the hamstrings are probably acting concentrically at the knee to produce knee flexion, which reaches a maximum at midswing. In late swing, the hamstrings may be contracting eccentrically to control knee extension and to reextend the hip.

A comparison of walking and running muscle activity at the ankle shows that in walking, **gastrocnemius** muscle activity begins just after the loading response at about 15% of the gait cycle and is active to about 50% of the gait cycle (just before toe-off). In running, gastrocnemius muscle activity begins at heel strike and continues through the first 15% of the gait cycle, ending at the point at which activity begins in walking. The gastrocnemius muscle becomes active again during the last 15% of swing.

The **tibialis anterior** muscle activity occurs in both stance and swing phases in walking and running. However, the total period of activity of this muscle in walking (54% of the gait cycle) is less than it is in running, in which it shows activity for about 73% of the gait cycle. The difference in activity of the tibialis anterior muscle between walking and running is due partly to the differences in the length of the swing phases in the two types of gait. The swing period in walking gait is approximately 40% of the total gait cycle, whereas in running gait, the swing phase constitutes about 62% of the total gait cycle. Most of the activity in the tibialis anterior muscle during both walking and running gait is concentric or isometric action that is necessary to clear the foot in the swing phase of gait. The longer swing phase in running accounts for at least part of the difference in tibialis anterior activity between walking and running gaits. Tibialis anterior activity in the first half of the stance phase in running gait accounts for the remainder of the difference in activity in this muscle.

Moments, Powers, and Energies

In Figure 14–29, note the differences in internal moments and powers for running from those of walking (see Figs. 14–16 and 14–19). In all joints, the moments are greatly increased, particularly the knee extensor moment in early stance. Similarly, all the power generators are greatly increased in running. The chief generators are, again, A2, H3, and H1, but an additional source, K2, is now important. Note that the knee extensors in early stance (K2) are important in running. The absorption phases typical of walking are increased at all joints in running.

In walking, potential and kinetic energy of the head, arms, and trunk are out of phase, which results in considerable energy savings. In running, these savings cannot occur, but two other methods of gaining efficiency are present. First, there are storage and return of elastic potential energy by the stretch of elastic structures, particularly tendons. Second, there is transfer of energy from one body segment to another by two-joint muscles acting as "energy straps."[85]

Summary

As a result of the efforts of many investigators, our present body of knowledge regarding human locomotion is extensive. However, gait is a complex subject, and further research is necessary to standardize methods of measuring and defining kinematic and kinetic variables, to develop inexpensive and reliable methods of analyzing gait in the clinical setting, and to augment the limited amount of knowledge available regarding kinematic and kinetic variables in the gaits of children and elderly persons.

Standardization of equipment and methods used to quantify gait variables, as well as standardization of the terms used to describe these variables, would help eliminate some of the present confusion in the literature and make it possible to compare the findings of various researchers with some degree of accuracy. Some standardization has been provided by the manufacturers of motion analysis systems; as a result, investigators using similar systems tend to use the same conventions. At the present time, inexpensive, quantitative, and reliable clinical methods of evaluating gait are limited to time and distance variables of step length, step duration, stride length, cadence, and velocity.[86] These measures provide a simple means for objective assessment of a patient's status and for detecting overall change, but they provide virtually no help in determining treatment. Increases in step length and decreases in step duration may be used to document a patient's progression toward a more normal gait pattern; however, a normal gait pattern may not be appropriate for many patients. Although many movement practitioners feel that it is best to aim for normal gait during early stages of rehabilitation, it is also important to identify the means by which a person can use the flexibility of the human body to compensate for deficiencies.[5,52] Automated gait analysis computer programs can provide the clinician with information about all of the kinematic and kinetic gait parameters related to a particular subject or patient. However, the researcher or clinician must have sufficient knowledge of the kinematics and kinetics of normal gait to interpret and use the information for the benefit of the patient.

EFFECTS OF AGE, GENDER, ASSISTIVE DEVICES, AND ORTHOSES

Age

The gait of young children has received less attention than that of adults. The relatively few studies of children's gait that have been conducted have shown that the age at which independent ambulation begins is extremely variable among individuals and that this variability continues throughout the developmental stages of walking. Cioni and coworkers[87] found that for 25 full-term infants, the age at which independent walking was attained (ability to move 10 successive steps without support) varied between 12.6 and 16.6 months. In the first stage of independent walking, none of the toddlers had heel strike, reciprocal arm swinging, or trunk rotation. However, 4 months after attainment of independent walking, nealy half of the children had heel strike, and nearly two thirds had reciprocal arm swinging and trunk rotation.

The toddler has a higher relative center of mass than does the adult and walks with a wider base of support, a decreased single-leg support time, a shorter step length, a slower velocity, and a higher cadence in comparison with normal adult gait. A study of 3- and 5-year-old children showed that some relationships between these variables were similar to the relationships found in adult gaits.[88] For example, as a group, the 3- and 5-year-old children showed significant increases in stride length adjusted for leg length, step length, and cadence from a slow to a free speed and from a free to a fast speed of gait. In a study that included children from 6 to 13 years of age, Foley and associates[89] reported that the ROMs for flexion and extension of the joints of the lower extremities were almost identical to the values obtained for adults. However, linear displacements, velocities, and accelerations were found to be consistently larger for these children than they were for adults.[89]

Sutherland and colleagues,[90] who studied 186 children from 1 to 7 years of age, suggested that the following five gait parameters could be used as indicators of gait maturity: duration of single-limb support, walking velocity, cadence, step length, and the ratio of pelvic span to ankle spread (inter-ankle distance measured during periods of double support, and indicative of base of support). Increases in all of these parameters except for cadence are indicative of increasing gait maturity. In Sutherland and colleagues' study, the duration of single-limb stance increased from about 32% of the gait cycle in 1-year-olds to 38% in 7-year-olds (normal mean adult value is 40%). Walking velocity also increased steadily, whereas cadence decreased with age.[90] Beck and colleagues[91] found that time and distance measures and GRF measurements depended on speed of gait and age of the child. Increases in height and age were the major factors in determining changes in time and distance measures with age. Average stride length was 76% of the child's height at a walking speed of 104 m/sec, regardless of a child's age. It is generally agreed that children's gait patterns have matured at least by the age of 7 years, with certain variables considerably earlier.[91,92] Studies involving young children are difficult to perform and often complicated by the fact that the child's musculoskeletal and nervous systems are in various stages of development. However, Sutherland and colleagues attempted to provide evaluators with guidelines for assessing children's gait by developing a group of prediction regions for the kinematic motion curves in normal gait. A test of the prediction regions indicated that they were capable of detecting a high percentage of abnormal motion and therefore could be used as an initial screen to identify deficits in lower extremity function in children.[93]

In contrast to the dearth of gait studies of young children, the effects of aging on gait continue to be the object of many studies.[20,45,91,95] Some of this interest in elderly gait has been prompted by the large number of hip fractures and falls experienced by elderly persons. Fifty percent of elderly people who were able to walk before a hip fracture are not able to either walk or live independently after the fracture.[96] Furthermore, it is estimated that an elderly person experiences at least two falls per year,[96] with the incidence higher in women and the rate increasing with age.[97] Increased sway and physiological decline are thought to be implicated and many falls occur during walking and while turning the head.[98] Therefore, many studies are directed toward determining what constitutes normal elderly gait and whether falls are caused by deficits in motor functioning or control, by other deficits that may accompany normal aging, or by adaptations employed that are thought to make their gait safer.[99]

The use of different age groups and levels of activity (sedentary versus active groups) among investigators has made it difficult to draw definitive conclusions about the effects of normal aging. With respect to temporal and kinematic variables, elderly persons, in comparison with younger groups, usually demonstrate a decrease in natural walking speed, shorter stride and step lengths, longer duration of double-support periods, and smaller ratios of swing to support phases,[20,94,100] all of which are thought to increase stability. Himann and associates[94] found that between 19 and 62 years of age, there was a 2.5% to 4.5% decline in the normal speed of walking per decade for men and women, respectively. After age 62, there was an accelerated decline in normal walking speed, that is, a 16% and 12% decline in walking speed for men and women, respectively.[94] Winter and associates,[17,45] comparing fit and healthy elderly subjects with young adults, found that the natural cadence in the elderly subjects was no different from that in young adults but that the stride length of the elderly subjects was significantly shorter and both stance time and the double support times were longer in the elderly subjects than in young adults. A significantly higher horizontal heel velocity at initial contact was also reported, which would increase the potential for slip-induced falls, and which occurred even though gait speed in the elderly was lower than that of the young subjects. Kerrigan and associates[100] found that older persons, in comparison with younger persons, had reduced plantarflexion ROM, peak hip extension ROM, and increased anterior pelvic tilt, and suggested that subtle hip flexion contractures and plantarflexor weakness might be causes of the joint changes in elderly people.

Differences in kinetic measures of gait have also been reported. Winter and colleagues reported less vigorous push-off by ankle plantarflexors in power analyses, as did Kerrigan and associates.[100] Mueller and coworkers found that plantarflexor peak torque and ankle dorsiflexion were interrelated.[101] These authors suggested that walking speed and step length might be improved by increasing ankle plantarflexor peak torque and dorsiflexion ROM.[101] Bohannon and colleagues[102] found that hip flexor strength was one of the variables that predicted gait speed, although these authors did not test plantarflexor strength. Strengthening and conditioning programs aimed at changing kinetics typically show similar results to those of Lord and associates,[103] who conducted an exercise program for women 60 years of age and older. After the program, the authors found significant increases in cadence and stride length, as well as reductions in stance time, swing time, and stance duration. However, detraining may be substantial. Connelly and Vandervoort[104] showed a strength decline of 68.3% one year after a quadriceps training program ended for a group of elderly persons having a mean age of 82.8 years. The speed of self-selected gait in this group also declined by 19.5%.

That a less efficient gait is typical of aging is certain, although the importance of possible causes awaits further research. Winter[17] reported both lower push-off work by the ankle plantarflexors and higher levels of absorption by the knee extensors (K3) at the same time in the gait cycle. Although several causes for the lower push-off are possible, the pistonlike action of a strong push-off may be perceived as more destabilizing, and avoided by older subjects. The reduced amount of work done by the plantarflexors to generate the work of walking may itself explain much of the inefficiency of the gait of older people, as shown by Kuo.[105] Using a simple theoretical model, Kuo persuasively showed that using larger amounts of ankle plantarflexor work (A2) is much more efficient than using the other two muscle groups, the hip extensors (H1) and hip flexors (H3), to generate the positive work of walking.[105] Less plantarflexor work and more hip work is typical of the older walker, and hence would be expected to be much less efficient. Note, however, that the increased absorption by the knee extensors also reported by Winter[17] would also produce a less efficient gait. Another possible cause of inefficiency could be reduction in the kinetic-potential exchange, described earlier in the Mechanical Energy of Walking section in this chapter, but there is no evidence for this. However, there is mounting evidence for increased co-contraction of antagonist muscles in the gait of older subjects,[106] which would reduce efficiency. In addition, age-related changes appear to affect lateral balance, and the resulting compensations may also explain some of the increased energetic cost of walking in older adults.[107]

Walking ability has important implications for health in the older adult population. Measures of walking are not only indicators of independence and general health,[108] but also have predictive value for future health. Self-paced gait speed is the most common outcome measure for gait and reflects the ability to transport the body from one place to another in a timely manner. Perry and colleagues[109] have suggested that an average self-paced walking speed of 0.4 m/sec is the minimum criterion for limited community ambulation, and 0.8 m/sec for unlimited community ambulation. Daily step counts are also a new and popular method of assessing walking function. It has been suggested that less than 5,000 steps a day reflect a sedentary lifestyle and more than 12,000 a highly active lifestyle.[110] Mean values for healthy older adults are 5,000 to 6,000 per day.[111,112] Walking function has predictive value. The ability to walk 400 meters and the time taken to do so have been found to be important predictors for mortality, cardiovascular disease, and mobility disability in a large sample of community-dwelling older adults.[113]

Walking is known to be important for bone health. For example, poorer walking endurance, as measured by a 6-minute walk test,[108] or lower ground reaction forces during walking in people with stroke, has been shown to correlate with lower paretic hip bone density, a condition that contributes to hip fracture risk. Fast walking also appears to have positive benefits: The osteogenic index,[114] a reflection of peak vertical GRF (or body weight), the number of loading cycles, and number of walking sessions, has been shown to be higher in fast walking than in walking at self-selected speeds,[115] and may promote greater bone health.

Although the particulars vary, it is clear that there are both kinematic and kinetic differences in the gait of elderly people. These can stem from two sources: degeneration of strength and balance control system or adaptation to make gait safer. It is likely that both are responsible.

Gender

The research regarding gender differences in gait is fraught with the same difficulties as found in gait research with regard to age. Variations among methods, technologies, and subjects used in various studies make it difficult to come to many conclusions regarding the effects of gender. When differences in height, weight, and leg length between the genders are considered, gender differences are not very great. Oberg and associates[116] found significant differences between men and women for knee flexion/extension at slow, normal, and fast speeds at midstance and swing. They found a significant increase in joint angles as gait speed increased. For example, the knee angle at midstance increased from 15° to 24° in men and from 12° to 20° in women. However, Oberg and coworkers looked at only the knee and hip. In another study, the authors looked at velocity, step length, and step frequency.[20] Gait speed was found to be slower in women than in men (118 to 134 cm/sec for men and 110 to 129 cm/sec for women), and step length was shorter in women than in men. Kerrigan and associates[117] found that women had significantly greater hip flexion and less knee extension during gait initiation, a greater internal knee extension moment in late stance, and greater peak mechanical joint power absorption at the knee at that time (K3). Kinetic data were normalized for both height and weight. These authors also found that women had a longer stride length in proportion to their height and that they walked with a greater cadence than did their male counterparts.[117]

Assistive Devices

Walking without the use of assistive devices (crutches, canes, and walkers) is preferred by most people. However, such devices often are necessary either after a lower-extremity fracture, when the healing bone is unable to bear full body weight, or as a more permanent adjunct for a balance, to compensate for muscle deficiencies or for joint pain. Recall from Chapter 10 that a very small force at the hand is needed to produce a large balancing moment about the standing leg because of the long perpendicular distance from the point of application of the force on the hand to the hip joint center. You may wish to review the sections in Chapter 10 on the use of a cane and the accompanying figures at this time. Canes have typically been used on the side contralateral to an affected lower extremity to reduce forces acting at the affected hip, the reason being that a lower abductor muscle force on the affected side would be necessary to balance the weight of the upper body during single-limb stance if an upwardly directed force was provided by the hand on a cane at some distance from the hip joint center. Krebs and coworkers[118] tested the effect of cane use on reduction of pressure through the use of an instrumented femoral head prosthesis that quantified contact pressures at the acetabular cartilage. The prosthetic head contained 13 very sensitive pressure-sensing transducers. The magnitude of acetabular contact pressure was reduced by 28% on one transducer and by 40% on another transducer in cane-assisted gait in comparison with unaided gait. The reduction in pressure at the hip coincided with reductions in EMG amplitude in comparison with the same pace in unaided gait trials. The authors concluded that the use of a cane on the contralateral side apparently allows the person to increase the base of support and to decrease muscle and ground reaction forces acting at the affected hip. The hip muscle abductor force was reduced, and gluteus maximus activity was reduced approximately 45%. Similar conclusions were reported by Neumann in an article that includes clear biomechanical explanations and figures.[119]

Recall from our Patient Case that Ms. Brown used a four-point cane on her unaffected side, because her arm was also affected. For her, the most important objective was to gain balance and stability, not to reduce joint forces.

Orthoses

The function of orthoses in gait is to alter the mechanics of walking. They are used to support normal alignment, to prevent unwanted motion, to help prevent deformity, to reduce unwanted forces or moments,[120–122] and, more recently, to augment joint power.[123] Although a full discussion is beyond the scope of this book, the student can deduce the effects of various types of devices with knowledge of the mechanics of gait. In all cases, the wish is to reduce or prevent unwanted movement or undesirable forces while permitting as normal mechanics as possible. For example, one may wish to limit ankle plantarflexion in a child with cerebral palsy. However, in so doing, one would prefer to encourage the active energy-generating activity of the ankle plantarflexors (A2) during push-off. Formerly orthoses did not permit any ankle motion, but more recent designs include hinged orthoses that prevent excessive plantarflexion by use of a posterior stop but permit the ankle to move into dorsiflexion in late stance. Therefore, a child with cerebral palsy is able to generate some power through the plantarflexion that follows. There is some evidence of decreases in energy costs with use of an orthosis composed of elastic straps that assist the hip, knee, and ankle movements in persons with stroke.[124] Attempts have also been made to make use of mechanical characteristics of a leaf-spring design to return energy to the foot during push-off. Although good in concept, designs to date have failed to show return of energy.[94] Recently a carbon-fiber-spring ankle-foot orthosis and a carbon-fiber knee-ankle-foot orthosis showed promising results when tested on children with ankle plantarflexion weakness caused by myelomeningocele or arthrogryposis. The orthosis supplemented ankle power and work and enabled longer strides in almost all participants.[125]

CASE APPLICATION

Gait Status at Discharge

case 14–8

Recall that Ms. Brown was prescribed an ankle-foot orthosis to assist with providing an adequate support moment during stance phase and to prevent foot drop and the resulting energy-costly hip hiking. However, if the orthosis was rigid, it would make it impossible for her to achieve any A2-S push-off during late stance. Two options were considered: (1) a hinged orthosis that permitted unlimited dorsiflexion but had a stop beyond 10° of plantarflexion, thus allowing limited push-off; and (2) a flexible ankle-foot orthosis that narrowed posterior to the ankle, thus permitting a limited range of both dorsiflexion and plantarflexion. Because it was hoped that use of the device would be temporary, the latter was chosen. Gait reeducation included progressive training without the orthosis as strength was gained, and at discharge, Ms. Brown used her orthosis only for outdoor use. As her stability improved, she was able to progress from a four-point cane to a straight cane. When she was discharged from outpatient treatment, she was walking at 0.55 m/sec.

ABNORMAL GAIT

Both quantitative and qualitative evaluations of gait are useful for assessors of human function. The most important quantitative variable is gait speed, which has been shown to be related to all levels of disablement.[126] An individual's gait pattern may reflect not only physical or psychological status but also any defects or injuries in the joints or muscles of the lower extremities. In assessing an abnormal gait, it is helpful to separate the cause or causes of deviations into structural impairment(s) and/or functional impairment(s). Functional impairments can be further categorized by geneal cause: whether directly due to abnormalities of the muscular or nervous system or their control, by pain, or as a result of compensations/adaptations to the abnormalities or pain. Although an extensive review is beyond the scope of this book, commonly encountered examples will provide a framework for examining abnormal gait.

Structural Impairment

These are structural malformations that are congenital, caused by injury or by structural changes occurring secondary to injury.

A common structural abnormality is leg length discrepancy. Kaufman and coworkers[127] undertook a study to determine the magnitude of limb length inequality that would result in gait abnormalities. Many minor limb inequalities are found in the general population but many of these do not necessitate any particular treatment or intervention because they do not have any significant effects on normal gait. The authors concluded that a limb length discrepancy of 2.0 cm resulted in an asymmetrical gait and had the potential for causing changes in articular cartilage. Song and colleagues[128] evaluated neurologically normal children who had limb discrepancies of 0.8% to 15.8% of the length of the long extremity (0.6 to 11.1 cm). The compensatory strategies observed were equinus position of the ankle and foot of the short limb (toe walking), vaulting over the long limb, increased flexion of the long limb, and circumduction of the long limb. Children who used toe walking had a greater vertical translation of the body's center of mass during gait than did normal controls.

Structural problems may be implicated in running injuries. Increases in the Q-angle, tibial torsion, and pronation of the foot may contribute causes to patellofemoral syndromes. In running, stresses are greater than in walking, and so there is an accompanying increase in the likelihood of injury. In a survey of the records of 1,650 running patients between the years 1978 and 1980, 1,819 injuries were identified.[129] The knee was the site most commonly injured in running, and patellofemoral pain was the most common complaint.

At the foot, pes cavus and pes planus cause alterations in weight and may cause abnormal stresses at the hip or knee. In pes cavus, the weight is borne primarily on the hindfoot and metatarsal regions, and the midfoot provides only minimal support.[130] In running, the metatarsals bear a disproportionate share of the weight. In pes planus, the weight is borne primarily by the midfoot rather than being distributed among the hindfoot, lateral midfoot, metatarsals, and toes, as it is in the normal walking foot. The propulsive phase of gait is severely compromised.

Functional Impairment

This group includes all causes in which the timing and/or amplitude of muscle activity is abnormal, whether due to abnormalities of the muscular or nervous system or their control, pain, or compensations/adaptations to the abnormalities or pain.

Abnormalities in the central nervous system are responsible for conditions such as Parkinson's disease, stroke, and cerebral palsy. These all produce characteristic gaits that are easily recognized by a trained observer. The parkinsonian gait is characterized by an increased cadence, shortened stride, lack of heel strike and toe-off, and diminished arm swinging. The muscle rigidity that characterizes this disease interferes with normal reciprocal patterns of movement.[131] Cerebrovascular incidents, commonly known as strokes, produce gait abnormalities that usually include reduction of strength and power of muscles of one lower limb that are usually worse distally. These deficits are frequently compounded by some degree of spasticity and deficits in motor control. Children with cerebral palsy sustain brain lesions that are similar to lesions in adults who have had strokes; however, the children sometimes have both limbs affected, and the immature system produces shortened muscles that do not keep up with bone growth. Conversely, children with cerebral palsy often show compensations that people with stroke do not use.

When the plantarflexors, which are the major source of mechanical energy generation in gait, are unable to generate sufficient power, muscles at other joints may compensate and provide even more energy than is typical of normal gait.[4] For example, Winter[132] found that individuals with below-the-knee amputations and below-the-knee prostheses used the gluteus maximus, semitendinosus, and knee extensor muscles as energy generators to compensate for loss of the plantarflexors. Olney and associates[49] found that in children with cerebral palsy who had unilateral plantarflexor weakness, the involved plantarflexors produced only 33% of the energy generation, in comparison with the 66% produced in normal gait.

The quadriceps is normally active at initial contact throughout early stance when the GRFV is tending to extend the knee. It is common for people with patellar pain to inhibit quadriceps contraction and produce a gait that appears kinematically normal. The quadriceps function is easily compensated for if a person has normal hip extensors and plantarflexors. The gluteus maximus and soleus muscles pull the femur and tibia, respectively, posteriorly, which results in knee extension. Additional compensation, if the hip extensor and ankle plantarflexor activity is inadequate, may be accomplished by forward trunk bending and a rapid plantarflexion after initial contact. The forward shifting of the weight moves the GRFV anterior to the knee (at initial contact and during the loading response period). It also may force the knee into hyperextension and eliminate the need for any quadriceps activity. This tendency is often seen in the early weeks after stroke, if not corrected with gait training.

Concept Cornerstone **14-5**

Paresis of Dorsiflexors

The normal functions of the dorsiflexors in gait are (1) to maintain the ankle in neutral so that the heel strikes the floor at initial contact; (2) to control the external plantarflexion moment at heel strike; (3) to dorsiflex the foot in initial swing; and (4) to maintain the ankle in dorsiflexion during midswing and late swing. If these functions are absent, the following would be expected to occur: (1) The entire foot or the toes would strike the floor at initial contact; and (2) the amount of flexion at the hip and knee would have to increase to clear the foot in swing phase. An orthosis that supports the foot, called an *ankle-foot orthosis (AFO)*, *is frequently used to avoid these problems.* An orthosis that incorporates an electical stimulation at the appropriate time, called *functional electrical stimulation (FES)*, or *functional neuromuscular stimulation (FNS)*, has also been shown to be effective.[133]

In patients with bilateral lower extremity paresis or paralysis, such as that caused by a spinal cord injury, walking usually involves the use of long leg braces and crutches. In this form of gait, the trunk and upper extremity muscles must perform all of the work of walking, and the energy cost of walking is much greater than normal. Functional electrical stimulation is currently being used to activate the paralyzed lower extremity muscles so that these muscles can generate energy for walking. However, the energy cost of functional electrical stimulation–induced walking is still well above that of normal gait.[134]

Pain

This group includes all causes of variations that are attributable primarily to pain. All overuse injuries and most joint pathologies fall into this category. It is common for people with patellar pain to inhibit quadriceps contraction and produce a gait that appears kinematically normal. The quadriceps function is easily compensated for if a person has normal hip extensors and plantarflexors, a situation that is described above with reference to stroke.

Many gait variations can be seen in osteoarthritis.[135] For example, hip joint pain causes an individual to reduce the level of contraction of the hip abductor, the gluteus medius muscle, during single limb stance. This results in a typical pattern called Trendelenburg gait. Normally, the gluteus medius stabilizes the hip and pelvis by controlling the drop of the pelvis during single-limb support. If gluteus medius activity on the side of the stance leg is reduced, the pelvis, accompanied by the trunk, will tend to fall excessively toward the swing side, which would result in a loss of balance. To prevent the trunk and pelvis from falling toward the unsupported side, the individual may laterally bend the trunk over the stance leg at initial contact, during weight acceptance, and through single stance. This trunk motion enables the person to bring the center of mass closer to the hip joint, thus reducing the need for such a large hip abductor contraction and the pain it would have caused (see Chapter 10).

Knee joint pain is the principal clinical problem in 10% of people over 55 years of age with osteoarthritis of the knee, and one quarter of these are severely disabled, with extensive gait limitations.[136] As knee varus is commonly associated with osteoarthritis, the internal knee abductor moment (often reported in the literature as the external adductor moment, or simply “adductor moment”) is also high, and is associated with abnormally large forces across the knee. Efforts to limit knee forces in the frontal plane include trunk lean to the affected side, similar to the compensation for hip joint pain. Conservative means of reducing forces include weight loss, use of a cane, external rotation of the foot while walking, and use of inner soles that change the GRFV.

Pain also appears to be a factor leading to an increase in oxygen consumption. As pain increases, oxygen consumption has been found to increase.[137]

Adaptation/Compensation

This group includes all causes of variations that occur when a structural or functional impairment is present and other structures alter their pattern to adapt to the abnormal conditions, or attempt to maintain gait function by compensating for the reduced function. Sometimes both are present. The human body is remarkable in its ability to compensate for losses or disturbances in function. Most of the compensations that are made are performed unconsciously, and if the disturbance is slight, such as occurs in excessive pronation of the foot, the individual may not be aware that the gait pattern is in any way unusual. However, most compensations will result in an increase in energy expenditure over the optimal amount and may result in excessive stress on other structures of the body.

Asymmetries of the lower extremities that result in gait adaptations may have structural or functional primary causes, such as contractures of soft tissues around the joints, bony ankylosis, and muscle weakness or spasticity.

Example 14-1

One might see excessive plantarflexion at the ankle during stance phase in a limb that is normal. The primary cause could be in the other limb: for example, an inability to clear the toes in swing phase as a result of inadequate knee flexion. The excessive plantarflexion by the stance limb would be an adaptation.

Example 14-2

A somewhat different example of adaptation could result from a knee flexion contracture. When the affected extremity is weight-bearing, the normal extremity will have difficulty swinging through in a normal manner, as it appears to be too long. A method of “equalizing” leg lengths is necessary for the swing leg to swing through without hitting the floor. Extra plantarflexion during push-off of the affected side, described previously, would be one means. In this case, there is no structural or functional abnormality in the adaptive movement. Alternatively, the person could increase the amount of flexion at the hip, knee, and ankle of the unaffected side. Again, the limb showing the adaptation has no structural or functional impairment. Other methods that produce relative shortening of the swinging leg are hip hiking, or circumduction of the leg. Each of these compensations makes it possible to walk, although they increase the energy requirements above normal levels.

Many compensations for inadequate power generation have been identified.[51] For example, persons with hemiparesis resulting from stroke frequently show greater than normal power generation from ankle plantarflexors at push-off (A2-S) of the unaffected limb (interlimb compensation) or in hip extensors in early stance (H1-S) on the affected side (intralimb compensation).

SUMMARY

The objectives of gait analysis are to identify deviations from the norm and their causes. Once the cause has been determined, it is possible to take corrective action aimed at improving performance, eliminating or diminishing abnormal stresses, and decreasing energy expenditure. Sometimes the corrective action may be as simple as using a lift in the shoe to equalize leg lengths or developing an exercise program to increase strength or flexibility at the hip, knee, or ankle. In other instances, corrective action may require the use of assistive devices such as braces, canes, or crutches. However, an understanding of the complexities of abnormal gait and the ability to detect abnormal gait patterns and to determine the causes of these deviations must be based on an understanding of normal structure and function. The study of human gait, like the study of human posture, illustrates the interdependence of structure and function and the large variety of postures and gaits available to the human species.

STUDY QUESTIONS

1. What percentage of the gait cycle is occupied by the stance phase in normal walking? How does an increase in walking speed affect the percentage of time spent in stance?
2. What percentage of the gait cycle is spent in double support? How is double support affected by increases and decreases in the walking speed?
3. Describe the subdivisions of the stance and swing phases of the walking gait cycle.
4. During which period of the gait cycle does maximum knee flexion occur?
5. What are the approximate values of maximum flexion and extension required for normal gait at the knee, hip, and ankle?
6. How does the total range of motion required for normal gait at the knee, hip, and ankle compare with the range of motion required for running and stair gaits?
7. What is the difference between an internal moment and an external moment?
8. What is the concept of the support moment, and what major muscle groups are responsible?
9. What moments are acting at the ankle, knee, and hip at initial contact? Answer the same question with regard to different gait events: foot flat, midstance, heel-off, and toe-off.
10. What is largely responsible for the abductor moment at the knee in stance phase?
11. What are the roles of the hamstrings in normal gait? Do they contribute to support and/or to power?
12. What are the major muscle groups that contribute to the positive work of walking, and when in the gait cycle do their contributions occur?
13. Why does walking faster than normal and walking slower than normal usually result in increased energy costs?
14. Why do long double-support times usually result in increased energy costs?
15. What is the role of the quadriceps muscle during walking gait?
16. What is the function of the plantarflexors during walking gait?
17. What are the functions of the dorsiflexors in normal walking gait?
18. How is the swinging motion of the upper extremities related to movements of the trunk, pelvis, and lower extremities during walking gait?
19. Compare muscle action in walking gait with muscle action in running gait.
20. Explain what would happen in walking and running if a person's plantarflexors were weak. What compensations might you expect?

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