RAPID INTERPRETATION OF EKG's



Dr. Dubin's classic, simplified methodology for understanding EKG's



Dale Dubin, MD

RAPID INTERPRETATION

 $\overset{ ext{of}}{ ext{EKG's}}$

... an interactive course

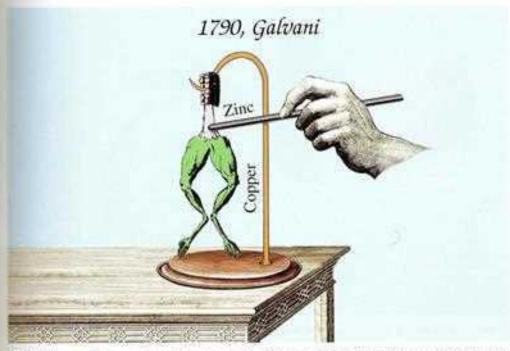


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Chapter 1: Basic Principles



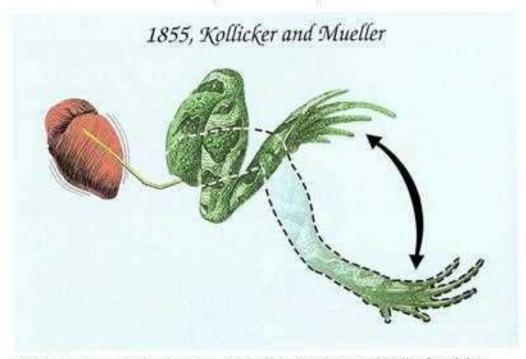
In 1790, an audience of usually sedate scientists gasped in disbelief as Luigi Galvani, with a flare of showmanship, made a dead frog's legs dance by electrical stimulation.

Galvani knew that complete metals to the legs of a recer stimulating	ng a circuit connecting dissimilar itly deceased frog would create a current.	electrical
	rent would stimulate the frog's legs stimuli he could make them	dance

Note: But in those times, bringing a dead frog "back to life" was a shocking and ghastly "supernatural" feat.

(And Galvani loved it!)*

^{*} Get yourself a warm cup of coffee, relax and enjoy... the rest is just as easy and entertaining.



While conducting basic research around 1855, Kollicker and Mueller found that when a motor nerve to a frog's leg was laid over its isolated beating heart, the leg kicked with each heartbeat.

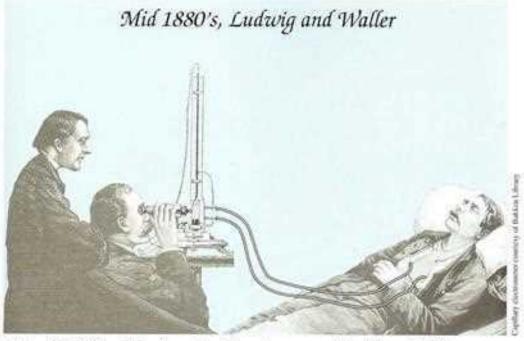
"Eureka!" they thought, "the same electrical stimulus that	
causes a frog's leg to kick must cause the heart to	_"

beat

So it was logical for them to assume that the beating of the heart must be due to a rhythmic discharge of stimuli.

electrical

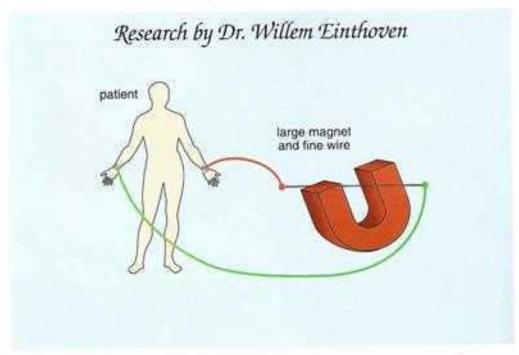
Note: And thus an association between the rhythmic pumping of the heart and electrical phenomena was scientifically established. Very basic and very important.



In the mid 1880's, while using a "capillary electrometer," Ludwig and Waller discovered that the heart's rhythmic electrical stimuli could be monitored from a person's skin.

This apparatus consisted of sensor electrodes that were placed on a man's and connected to a Lippman capillary electrometer, which used a capillary tube in an electric field to detect faint electrical activity.	skin
The fluid level in the capillary tube moved with the rhythm of the subject'sbeat very interesting.	heart
This apparatus was a little too unsophisticated for clinical application, or even for economic exploitation, but it was interesting.	very

Note: This momentous achievement opened the door for recording the heart's electrical activity from skin surfaces.

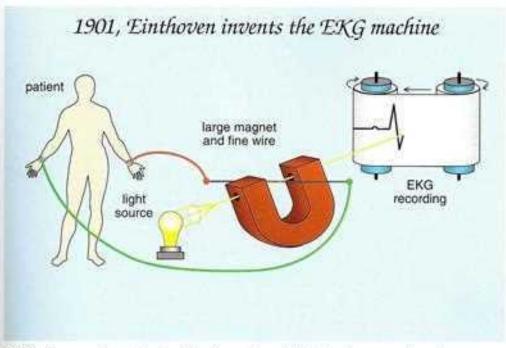


Enter Dr. Willem Einthoven, a brilliant scientist who suspended a silvered wire between the poles of a magnet.

Two skin sensors (electrodes) placed on a man were then connected to the ends of the silvered wire, which ran between the two poles of the	magnet
The silvered (in the magnetic field) twitched to the rhythm of the subject's heartbeat.	wire
This was also were interesting but	Einthoven

This was also very interesting, but

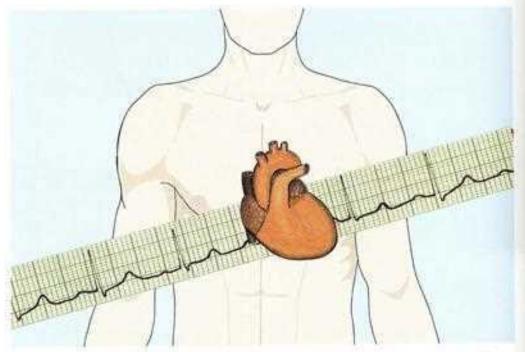
wanted a timed record.



So Einthoven projected a tiny light beam through holes in the magnet's poles, across the twitching silvered wire. The wire's rhythmic movements were recorded as waves (that he named P, QRS, and T) on a moving scroll of photographic paper.

Very clever, that Einthoven! The movements of the wire (representing the heartbeat) created a bouncing shadow		rhythmic
that was recorded as a of distinct waves in repeating cycles.	series	rhythmic
He named the waves of each cycle (alphab P. QRS, and	etically)	7

Note: "Now," thought the clever Einthoven, "we can record a heart's abnormal electrical activity... and compare it to the normal." And thus a great diagnostic tool, his "electrokardiogram" (ElectroKardioGram), evolved around 1901. Let's see how it works...



The electrocardiogram (EKG) records the electrical activity of the heart, providing a record of cardiac electrical activity, as well as valuable information about the heart's function and structure.

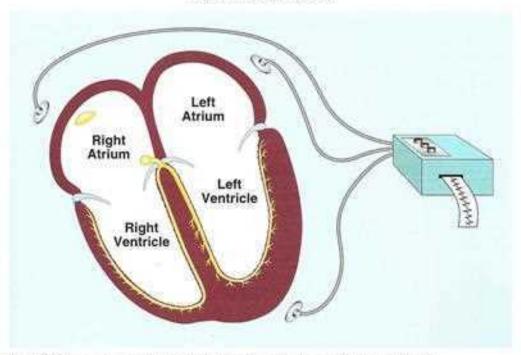
The electrocardiogram is known by the three letters ____; it provides us with a record of cardiac electrical activity and valuable information about the heart's function and structure.

EKG

Note: Since the time of Einthoven's "electrokardiogram," the medical profession has used the letters EKG to represent the electrocardiogram. Some say that "ECG" is more correct, and you may see it used in some texts. However, Medicine honors tradition, and EKG has been used for years. Also, ECG sounds like EEG (the brain wave recording), and this can cause misunderstanding and confusion.

The EKG is inscribed on a ruled paper strip that gives us a permanent ______ of cardiac activity and the health status of the heart. Cardiac monitors and cardiac telemetry provide the same information in real time.

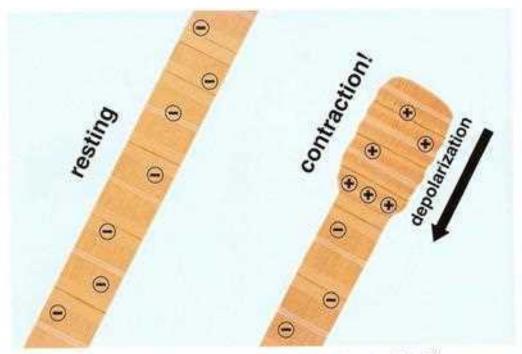
record



The EKG records the electrical activity of contraction of the heart muscle ("myocardium").

The information recorded on the EKG represents the heart'activity.	s electrical
Most of the information on the EKG represents electrical activity of of the myocardium.	contraction
Note: The EKG also yields valuable information about the heart's rate and rhythm.	e
When the myocardium (cardium = heart, myo = muscle) is electrically stimulated, it	contracts

Note: This illustration is intended to familiarize you with the simplified cross-section of the heart. The chambers are identified, and you should know them, for this diagram will be used often.



The interiors of heart muscle cells (myocytes*) are negative ("polarized") at rest, but when "depolarized" their interiors become positive and the myocytes contract.

	cell being	-ly charged.	negative
negatively cha	arged interior and	ting polarized cell has a a positively charged outside der only the negative interior	
	resting myocytes s are depolarized,	[1] [4] [4] [4] [5] [5] [5] [5] [6] [6] [6] [6] [6] [6] [6] [6] [6] [6	
	and the ce		positive
		e through the myocardium, imulates the heart's myocyt	es.
they become po			contract

* Just as the heart muscle is called the myocardium, its cells are called "myocytes".



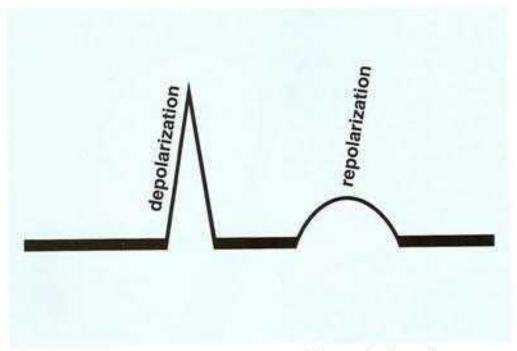
As a wave of depolarization progresses through the heart, it causes contraction of the myocardium.

Depolariza	tion may be considered an advancing wave	
of	charges within the heart's myocytes.	positive

Note: The depolarization wave initiates contraction of the resting myocytes as the charge within each cell changes to positive.

The advancing wave of depolarization causes progressive contraction of the myocardium as this wave of ______ charges positive passes through the interiors of the myocytes.

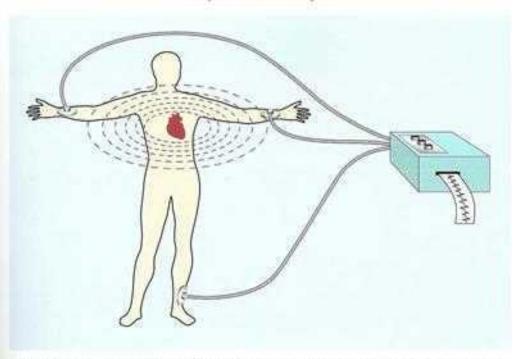
Note: The cell-to-cell conduction of depolarization through the myocardium is carried by fast-moving sodium (Na⁺) ions, the +'s in the illustration above.



The depolarization wave (cell interiors become positive), and a phase of repolarization (cell interiors return to negative) that follows, are recorded on the EKG as shown.

of the myocytes to contract.			positive
Then the myocyte inter- charge during the	iors regain their res	ting negative ase that follows.	repolarization
Note: Repolarization begins immediately a we see on EKG is the	fter depolarization.	nomenon that, in realist The broad hump that of repolarization.	ıy.
Myocardial contraction the myocytes, which re	is caused by	of	depolarization
The recovery phase tha	t follows denolariz	ation is known	

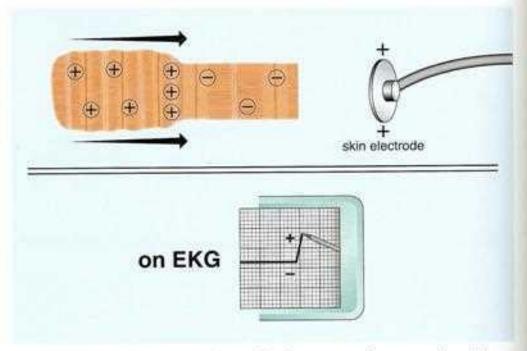
electrodes



Sensors called "electrodes" are put on the skin to detect the heart's electrical activity. The EKG machine records this activity on moving paper as an electrocardiogram.

Both depolarization and repolarization of the myocardium are phenomena caused by the movement of ions.	electrical
The heart's electrical activity may be detected and recorded from the surface by sensitive monitoring equipment, including EKG machines, cardiac monitors, and telemetry devices.	skin
The EKG records the electrical activity of the heart	

using skin sensors called _



As the positive wave of depolarization within the myocytes flows toward a positive electrode, there is a positive (upward) deflection recorded on EKG.

Note: "Positive electrode," of course, refers to a positive electrode actively recording a patient's EKG.

A wave of depolarization advar is a moving wave of	cing through the myocardium charges.	positive
(Here come the Na* ions!)	5-04-007918010	
When this wave of positive cha toward a positive electrode, the	20 - 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1	

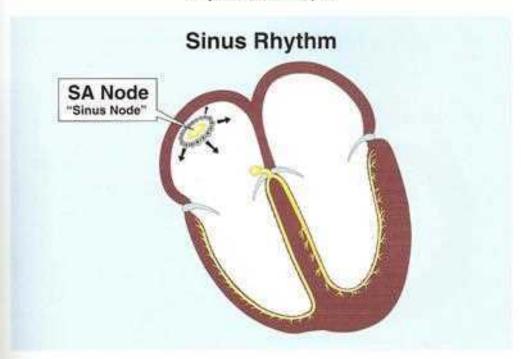
In general, when you see an upward wave on EKG, you know that it represents a depolarization wave

moving toward a ______electrode.

upward deflection recorded on the

positive

EKG

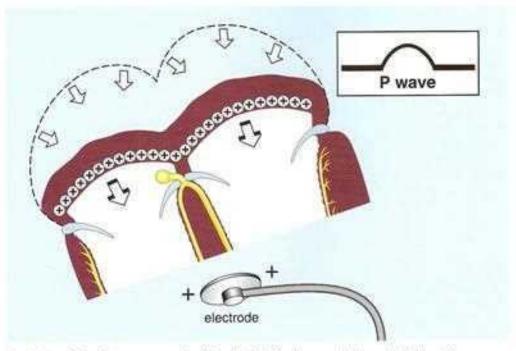


The heart's dominant pacemaker, the SA Node, initiates a wave of depolarization that spreads outward, stimulating the atria to contract as the circular wave advances.

Note: The SA Node ("Sinus Node") is the heart's dominant pacemaker, and its pacing activity is known as a "Sinus Rhythm." The generation of pacemaking stimuli is **automaticity**. Other focal areas of the heart that have automaticity are called "automaticity foci."

The SA Node, located in the upper-posterior wall of the right, initiates a depolarization wave at regular intervals to accomplish its pacemaking responsibility.	atrium
Each depolarization wave of + charges (Na* ions) proceeds outward from the SA Node and stimulates both atria to	contract
The ability of the SA Node to generate pacemaking stimuli	automaticity

Note: The depolarization wave flows away from the SA Node in all directions. Imagine the atria as a pool of water. A pebble dropped in at the SA Node produces an enlarging, circular wave (depolarization) that spreads outward. Atrial depolarization (and contraction) is a spreading wave of positive charges within the atrial myocardial cells. Let's read this page again.

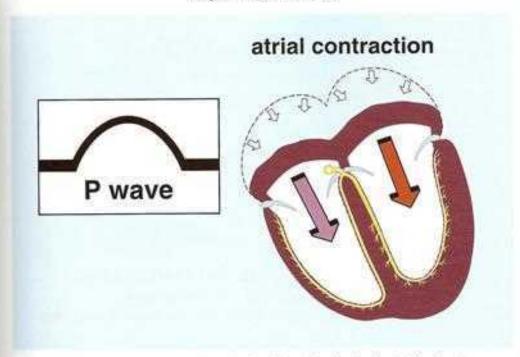


Each depolarization wave emitted by the SA Node spreads through both atria, producing a P wave on the EKG.

Note: The illustration depicts the positive wave of atrial depolarization advancing toward a positive skin electrode, producing an upward (positive) P wave on EKG.

The wave of depolarization sweeping through the atria can be detected by sensitive electrodes.	skin
Atrial depolarization is recorded as a wave on EKG.	P
So when we see a P wave on an electrocardiogram, we know that, electrically speaking, it represents atrial	depolarization

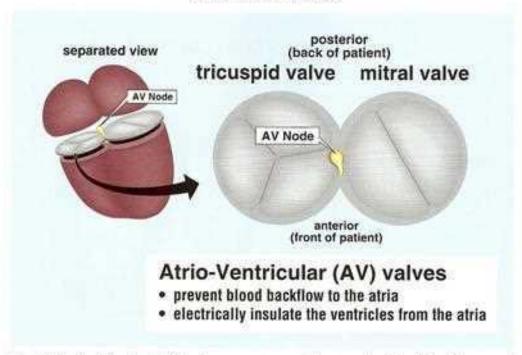
Note: The atria have a specialized conduction system, which we will examine later (page 101, if you're curious).



Thus the P wave represents the electrical activity (depolarization) of both atria, and it also represents the simultaneous contraction of the atria.

As the wave of depolarization moves through both atria, there is a simultaneous wave of atrial	contraction
So the P wave represents the depolarization and	atria

Note: In reality, contraction of the atria lasts longer than the duration of the P wave. However, we'll still consider that a P wave = atrial contraction. This simultaneous contraction of the atria forces the blood they contain to pass through the Atrio-Ventricular (AV) valves between the atria and the ventricles.



The Atrio-Ventricular (AV) valves prevent ventricle-to-atrium blood backflow, and they electrically insulate the ventricles from the atria... except for the AV Node, the only conducting path between the atria and the ventricles.

When the ventricles contract, the blood they contain cannot flow back into the atria due to the very efficient ____ valves.

AV

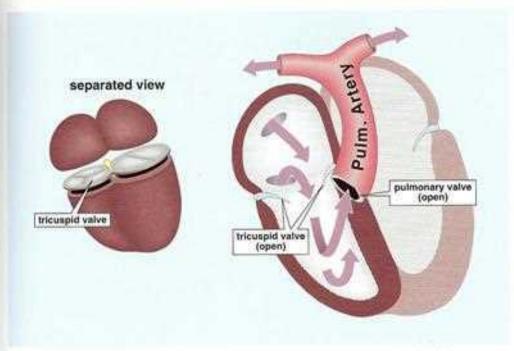
The mitral and tricuspid (AV) valves lie between the atria and the ventricles, thereby acting to electrically _____ the ventricles from the atria...

insulate

... leaving only the ___ Node as the sole pathway to conduct the depolarization stimulus through the fibrous AV valves to the ventricles.

AV

Note: The AV Node is just above, but continuous with, a specialized conduction system that distributes depolarization to the ventricles very efficiently. Next we will review the movement of blood through the heart's chambers.



Oxygen-depleted venous blood enters the right atrium. Atrial contraction forces blood through the *tricuspid valve* into the right ventricle, which pumps it into the lungs.

Note: Tricuspid is right side.

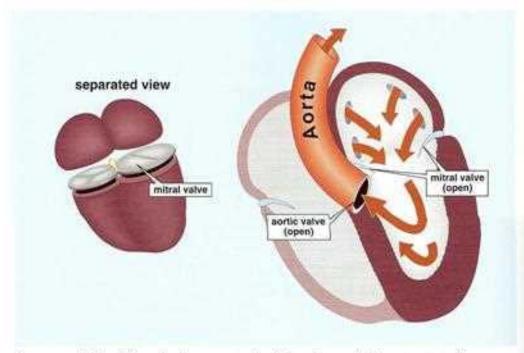
The right side of the heart (right atrium and right ventricle)	
receives under-oxygenated venous blood from all over the	
body, and pumps it into the	

lungs

The right ventricle contracts, forcing the under-oxygenated venous blood through the **pulmonary valve** into the pulmonary _____, and thence to the lungs.

artery

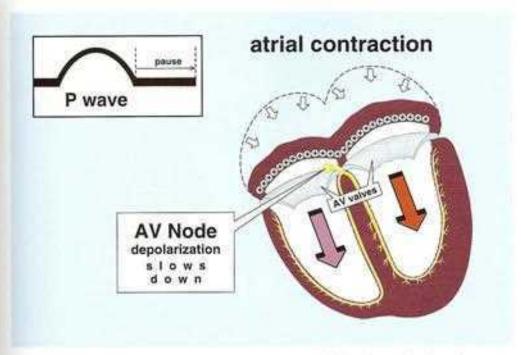
Note: Remember, both atria contract simultaneously, and also both ventricles contract together. However, the right and left sides of the heart have different responsibilities.



Oxygenated blood from the lungs enters the left atrium, which contracts to force blood through the *mitral valve* into the left ventricle. The powerful left ventricle, in turn, pumps blood through the **aorta** to all areas of the body.

Note: Mitral is left side.

The left atrium contracts, forcing oxygenated blood through the valve into the left ventricle.	mitral
Then the muscular left ventricle contracts, forcing oxygenated blood through the aortic valve into the (That's too easy!)	aorta
Both atria contract simultaneously, then both	ventricles

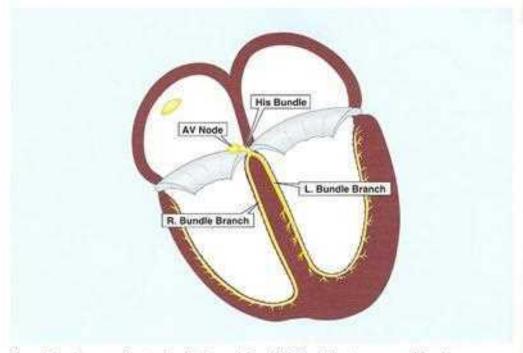


When the wave of atrial depolarization enters the AV Node, depolarization slows, producing a brief pause, thus allowing time for the blood in the atria to enter the ventricles. Slow conduction through the AV Node is carried by calcium (Ca⁺⁺) ions.

Note: Of course you remember that the AV Node is the only electrical conduction pathway between the atria and the ventricles.

Because depolarization slows there is a brief delay or		pause
depolarization is conducted to		15
This brief pause allows the bl	2 - 3 - 3 - 3 - 3 - 3 - 3 - 3 - 3 - 3 -	973545725400 (#F606
through the AV valves and in	to the	ventricles

Note: At this point, we are correlating electrical activity with mechanical physiology. The atria contract, forcing blood through the AV valves, but it takes a little time for the blood to flow through the valves into the ventricles (hence the necessary pause that produces a short piece of flat baseline after each P wave on the EKG). Please review the illustration again.

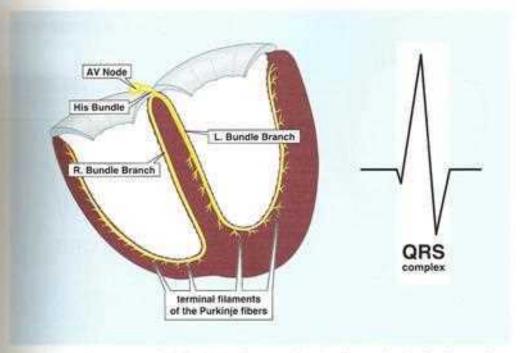


Depolarization conducts slowly through the AV Node, but upon reaching the ventricular conduction system, depolarization rapidly shoots through the His Bundle and the Left and Right Bundle Branches and their subdivisions.

Depolarization conducts slowly through the AV Node, since it is carried by slow-moving Ca ⁺⁺ ions, but depolarization shoots rap through the ventricular conduction system beginning in the		His
Depolarization conducts slowly through the AV Node, then rapidly through the His Bundle to the Right and		
Left Branches.	I	Bundle
Depolarization shoots rapidly through the His Bundle and the Bundle Branches and their subdivisions, so depolarization is quickly distributed to the myocytes of the	ver	ntricles

Note: The ventricular conduction system originates at the His Bundle, which penetrates the AV valves, then immediately bifurcates (in the interventricular septum) into the Right and Left Bundle Branches. The His "Bundle" and both "Bundle" Branches are "bundles" of rapidly conducting Purkinje fibers*. Like the myocardium, Purkinje fibers use fast-moving Na* ions for the conduction of depolarization.

Texts in the past (including this one) have incorrectly implied that only the terminal filaments were Purkinje fibers. Not so! Study "Note" and learn it correctly.

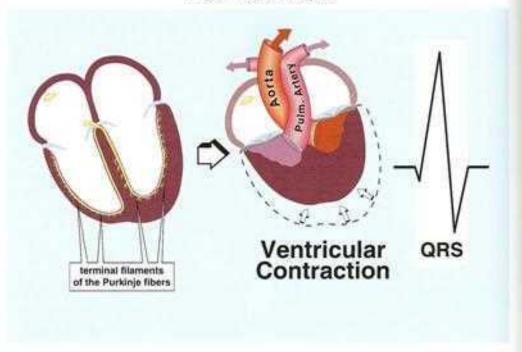


The terminal filaments of the Purkinje fibers rapidly distribute depolarization to the sentricular myocytes. Depolarization of the entire ventricular myocardium produces a QRS complex on EKG.

Note: The ventricular conduction system is composed of bundles of rapidly-conducting Purkinje fibers that carry depolarization away from the AV Node at high speed. The Purkinje fibers terminate in tiny filaments that directly depolarize the ventricular myocytes. The (rapid) passage of depolarization down the ventricular conducting system is too weak to record on EKG; however, depolarization of the ventricular myocardium records as a QRS complex.

Depolarization conducts slowly through the AV Node (using Ca⁺⁺ ions), and then conducts rapidly (using Na⁺ ions) through the His Bundle to the Right and Left Bundle Branches into the terminal filaments of the Purkinje fibers, which depolarize the _____ myocytes. ventricular

Note: The terminal filaments of the Purkinje fibers spread out just beneath the endocardium that lines both ventricular cavities, therefore ventricular depolarization begins at the lining and proceeds toward the outside surface (epicardium) of the ventricles. The Purkinje fibers branch and subdivide just beneath the endocardial lining, but they really do not penetrate into the myocardium. Since that's almost impossible to depict in a two-dimensional drawing, please recognize the limits of the illustration and remember it correctly.



The entire ventricular conduction system consists of rapidly conducting Purkinje fibers. The terminal filaments of the Purkinje fibers depolarize the ventricular myocardium, initiating ventricular contraction while inscribing a QRS complex on EKG.

The terminal filaments of the Purkinje fibers rapidly conduct
to the myocytes that lie just beneath the the endocardial lining of both ventricles.

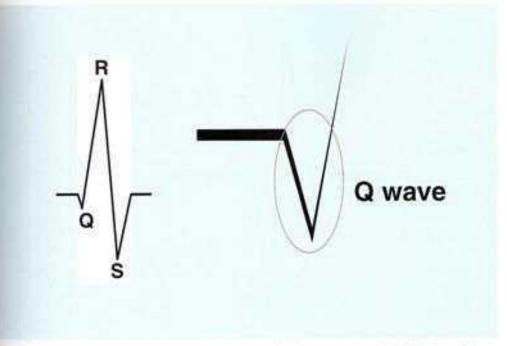
depolarization

Note: Remember, the entire ventricular conduction system, i.e., the His Bundle through the terminal filaments, is composed of Purkinje fibers that use fast-moving Na⁺ ions for conduction.

Depolarization of the ventricular myocytes produces a ____ complex on the electrocardiogram and initiates contraction of the ventricles.

QRS

Note: The QRS complex actually represents the beginning of ventricular contraction. The physical event of ventricular contraction actually lasts longer than the QRS complex, but we will still consider the QRS complex as generally representing the occurrence of ventricular contraction. So the QRS complex is an electrocardiographic recording of ventricular depolarization, which causes ventricular contraction. Still with me?



The Q wave is the first downward wave of the QRS complex, and it is followed by an upward R wave, however the Q wave is often absent on EKG.

The Q* wave, when present, always occurs at the of the QRS complex and is the first downward deflection of the complex.

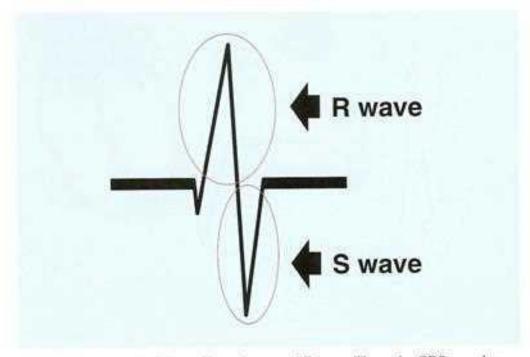
beginning

The downward Q wave is followed by an upward ____ wave.

R

Note: If there is any upward deflection in a QRS complex that appears before a "Q" wave, it is not a Q wave, for by convention, when present, the Q wave is always the first wave in the complex.

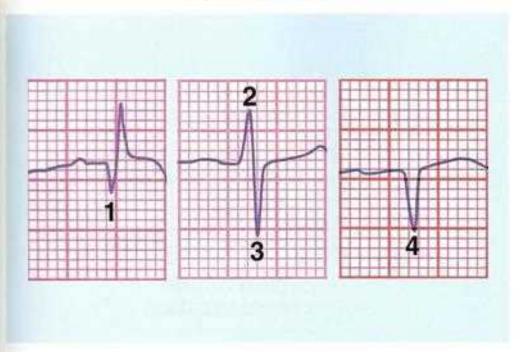
⁹ It is now popular to use small (non-capital) letters to designate small waves in the QRS complex, for instance a "q" (small, lower case q) wave is a small wave.



The upward R wave is followed by a downward S wave. The entire QRS complex represents ventricular depolarization.

The first upward wave of the QRS complex is the	R wave	
Any downward wave PRECEDED by an upward wave is an	S wave	
The complete QRS complex can be said to representdepolarization (and the initiation of ventricular contraction).	ventricular	

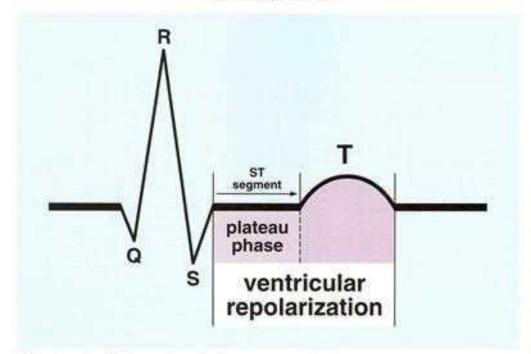
Note: An upward wave is always called an R wave. Distinguishing between the downward Q and downward S waves really depends on whether the downward wave occurs before or after the R wave. The Q occurs before the R wave, and the S wave follows the R. Just remember your alphabet.



Name the numbered waves in each QRS complex.

1	Q wave
2.	R wave
3	S wave
4	QS wave

Note: Number 4 is a little unfair. Because there is no upward wave, we cannot determine whether number 4 is a Q wave or an S wave. Therefore it is called a **QS wave**, and it is considered to be a Q wave when we look for Q's.



Following the QRS complex, there is a segment of horizontal baseline known as the ST segment, and then a broad T wave appears.

The horizontal segment of baseline that follows the QRS complex is known as the segment.

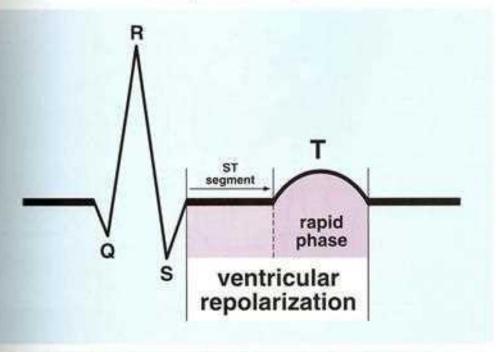
ST

After the QRS there is a segment of horizontal baseline, followed by a broad hump called the _____ wave.

77

Note: The ST segment is horizontal, flat, and most importantly, it is normally level with other areas of the baseline. If the ST segment is elevated or depressed beyond the normal baseline level, this is usually an sign of serious pathology that may indicate imminent problems.

Note: The ST segment represents the "plateau" (initial) phase of ventricular repolarization. Ventricular repolarization is rather minimal during the ST segment.



The T wave represents the final, "rapid" phase of ventricular repolarization, during which ventricular repolarization occurs quickly and effectively.

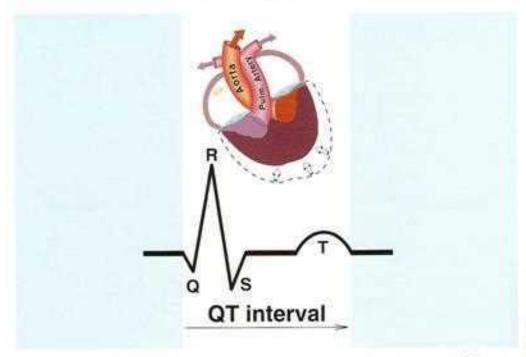
so they can be depolarized again.	negative
Even though the T wave is usually a low, broad hump, represents the phase of ventricular repolarization.	rapid
Repolarization of the ventricular myocytes begins immediately after the QRS and persists until the end of the wave.	т

Note: Repolarization (both phases) is accomplished by potassium (K+) ions leaving the myocytes.

Repolarization occurs so that the ventricular myocytes

Note: Ventricular systole* (contraction) begins with the QRS and persists until the end of the T wave. So ventricular contraction (systole) spans depolarization and repolarization of the ventricles. This is a convenient physiological marker.

^{*} Pronounced "SISS-toe-lee"



Since ventricular systole lasts from the beginning of the QRS until the end of the T wave, the QT interval has clinical significance.

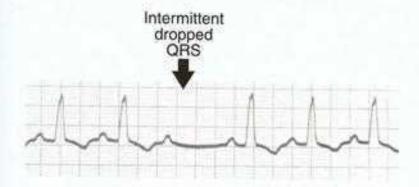
The QT interval represents the duration of ventricular and is measured from the beginning of the ORS until the end of the T wave.

systole

Note: The QT interval is a good indicator of repolarization, since repolarization comprises most of the QT interval. Patients with hereditary Long QT interval ("LQT") syndromes are vulnerable to dangerous (or even deadly) rapid ventricular rhythms. If you routinely examine the QT interval in all EKG's, eventually you will detect this anomaly, and probably save a patient's life during your career.

Note: With rapid heart rates both depolarization and repolarization occur faster for greater efficiency, so the QT interval varies with heart rate. Precise QT interval measurements are corrected for rate, so they are called QTc values. As a simple rule of thumb, the QT interval is considered normal when it is less than half of the R-to-R interval at normal rates.

Intermittent Mobitz (2° AV Block)



Occasional dropped QRS due to permanent BBB (one side) with intermittent BBB of the other side.

Simultaneous RBBB and LBBB prevents depolarization from reaching the ventricles; this is a complete (3°) AV block. So, block of one Bundle Branch with intermittent block of the other produces intermittent complete AV block, intermittent Mobitz.

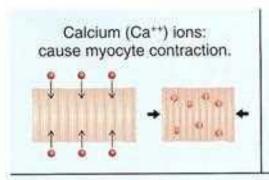
Right BBB plus intermittent Left BBB will record on EKG
scontinuous Right BBB pattern QRS's with intermittent
spisodes of complete AV block (P waves without _____ response).

QRS.

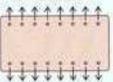
Left BBB plus intermittent Right BBB will record on EKG as continuous Left BBB pattern QRS's with intermittent episodes of complete AV block (P waves without _____ response).

ORS

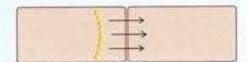
Note: An EKG tracing or cardiac monitor display with a continuous BBB pattern QRS with an occasional missing QRS indicates intermittent complete AV block. The intermittent block may worsen, eventuating in a constant complete AV block. This intermittent Mobitz texactly what it is) flashes an important warning sign. Intermittent Mobitz is the heart's warning that eventually it will need an artificial pacemaker to drive the ventricles at a normal rate. Don't let it slip by you unnoticed... for the patient's sake!



Potassium (K*) ions: outflow causes repolarization of myocytes.



Sodium (Na+) ion movement produces cell-to-cell conduction (of depolarization) in the heart...



° → ° → ° → ° →

except the AV Node, which depends on the (slow) movement of Ca** ions.

The movement of three types of ions determines all aspects of cardiac conduction, contraction, and repolarization.

The release of free Ca** ions into the interiors of the myocytes produces myocardial _____.

contraction

Following depolarization, repolarization is due to the controlled outflow of ____ ions from the myocytes.

K-

Cell-to-cell conduction (of depolarization) through the myocardium is carried by Na* ions, however, AV Node conduction is due to the slow movement of ____ ions.

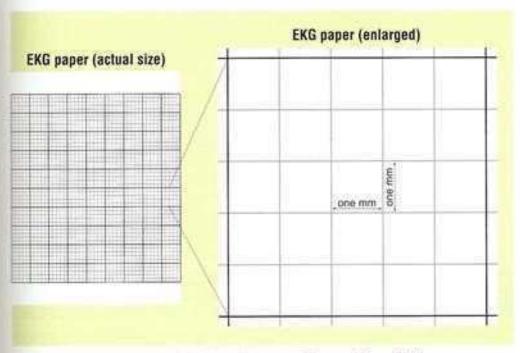
 C_3

Note: This information may seem incongruous in an EKG text, however, during this millennium this page will prove to be the most important of all. Movement of these three ions is the very basis of cardiac physiology; this knowledge will serve you well in the future.

Note: Very soon, all health care professionals will understand (so easy!) cardiac function on the ionic-molecular level by reading *Ion Adventure* in the Heartland, which demonstrates how and why the electrical messages of the heart are displayed on EKG. See pages 331 and 332.

5

Chapter 2: Recording the EKG



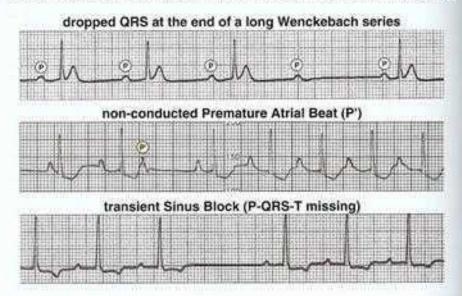
The EKG is recorded on ruled (graph) paper. The smallest divisions are one millimeter (mm) squares.

The EKG is recorded on a long strip of paper, although some EKG machines record many different leads simultaneously on a large sheet.	ruled (graph)
The smallest divisions are onehigh.	millimeter millimeter

Between the **heavy** black lines there are ___ small squares. Each large square is formed by **heavy** black lines on each side, and each side is five mm long.

Note: As with all graphs, the time axis is horizontal and moves left to right, like we read. So timed events on EKG are measured left to right and similarly, cardiac monitors display a time axis that reads from left to right.

Innocuous Imitators of Intermittent Mobitz



Since intermittent Mobitz may herald a complete AV block requiring a pacemaker, it is important that we recognize its characteristic span of clear baseline after a normal P wave. But, innocuous conditions can also produce a span of empty baseline.

A Wenckebach series (innocuous) produces a barren span of baseline after the terminal, punctual P _____, which is not conducted (review page 180).

WZ

A non-conducted Premature Atrial Beat (innocuous) strikes the AV Node while it is still refractory, so no stimulus is conducted to the ______ (page 128); notice the peculiar, premature P before the barren baseline.

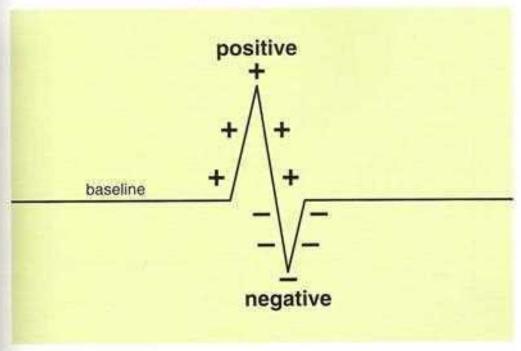
ventricis

A transient Sinus Block (usually innocuous, but the patient should be followed) can produce a pause before pacing resumes, or an automaticity focus may respond to the pause with an escape _____; in either case there is never an isolated P wave preceding the pause (review page 174).

bea

Note: Simply stated:

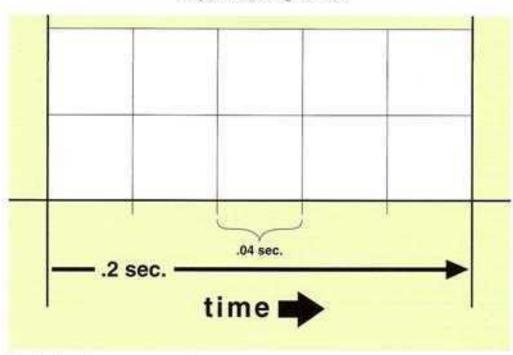
- punctual P wave (no QRS response)... 2º AV block; Mobitz vs. Wenckebach
- premature P' wave (no QRS response)... non-conducted PAB
- missed P-QRS-T cycle... SA Node transiently blocked (Sinus Block)



Upward deflections are called "positive" deflections, Downward deflections are called "negative" deflections.

Positive deflections are	on the EKG.	upward	
NA. ATT. OF PROJECT CO.	and FVC	donoused	
Negative deflections are	on the EKG.	downwa	

Note: When a wave of stimulation (depolarization) advances toward a positive skin electrode, this produces a positive (upward) deflection on EKG. You will recall that depolarization is an advancing wave of positive charges within the cardiac myocytes. So with depolarization, the advancing wave of positive intracellular charges produces a positive deflection on EKG as this wave moves toward a positive electrode. Be positive!

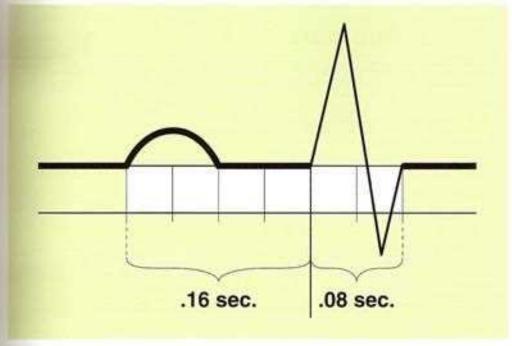


The horizontal axis represents time.

perween the r	ieavy black	lines there at	re smaii squ	ares.

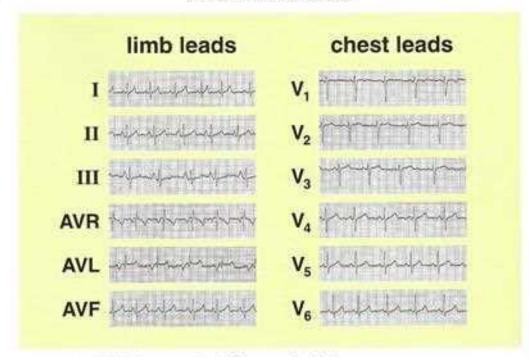
5

Each small division (measured horizontally between two fine lines) represents ______. .04 of a second (that's four hundredths!)



By measuring along the horizontal axis, we can determine the duration of any part of a cardiac cycle.

The duration of any wave may be determined by measuring along the horizontal	axis
Four of the small squares represents of a second.	.16 (sixteen hundredths)
The amount of EKG graph paper that passes out of the EKG machine in .12 second is small squares. You don't have to be a mathematician to read EKG's.)	three (3)

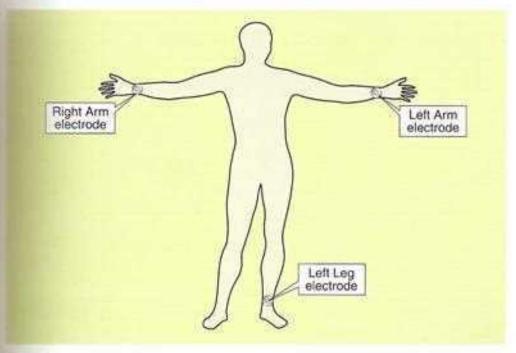


The standard EKG is composed of 12 separate leads.*

A standard EKG is composed of six limb recorded by using arm and leg electrodes and		leads
there are also six a suction cup electrode at	leads obtained by placing six different positions on the chest.	chest

Note: Leads not considered "standard" may be monitored from various locations on the body as required for special diagnostic purposes.

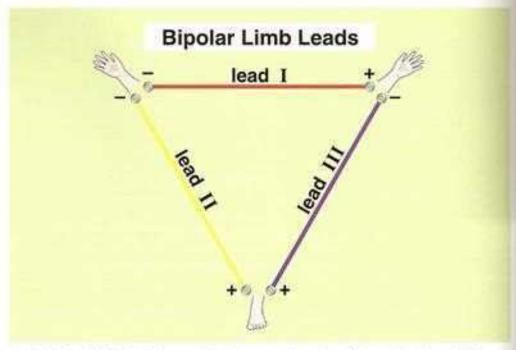
Rhymes with seeds.



To obtain the limb leads, electrodes are placed on the right arm, the left arm, and the left leg. A pair of electrodes is used to record a lead.

e can obtain and record the _	[20] [11 - [12] [12] [13] [13] [14] [14] [15] [15] [15] [15] [15] [15] [15] [15	eg, limb
	hree locations for limb electro d standard for recording the E	
The placement of these	is the same	electrodes

Note: Two electrodes are used to record a lead. A different pair is used for each lead.

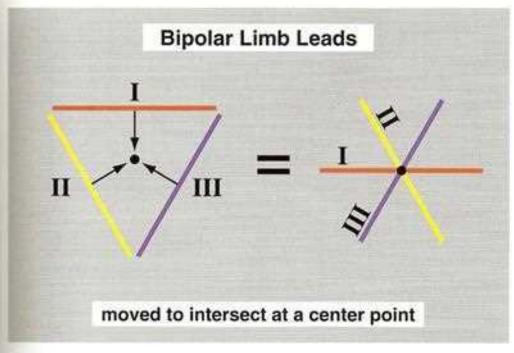


Each bipolar limb lead is recorded using two electrodes. So by selecting a different pair of electrodes for each lead, we create three separate bipolar limb leads (lead I, lead II, and lead III) for recording.

Each limb lead consists of a pair of electrodes, one is positive and one is, so these leads are called "bipolar" limb leads.	negative
Lead I is horizontal, and its left arm electrode is, while its right arm electrode is negative.	positive
When we consider lead III, the left arm electrode is now, and the left leg electrode is positive.	negative

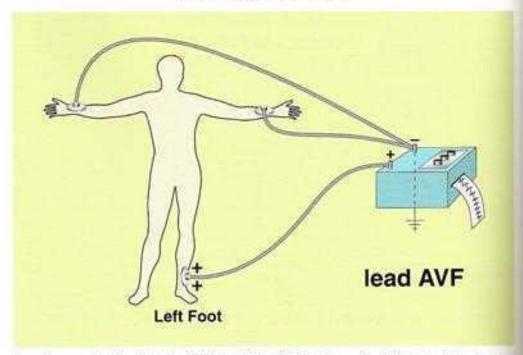
Note: The engineering wonders of the EKG machine permit us to make any skin electrode positive or negative depending on which pair of electrodes (that is, which lead) the machine is recording.

Note: The bipolar limb lead configuration is sometimes called "Einthoven's triangle."



By pushing the three (bipolar) limb leads to the center of the triangle, we produce three intersecting lines of reference.

The triangle has a center, and each may be moved to that center point.	lead
By pushing leads I, II, and III to the center of the triangle, three intersecting lines of are formed.	reference
Although the three bipolar limb leads may be moved to the of the triangle, they remain at the same angles relative to each other. (They're still the same leads, yielding the same information.)	center

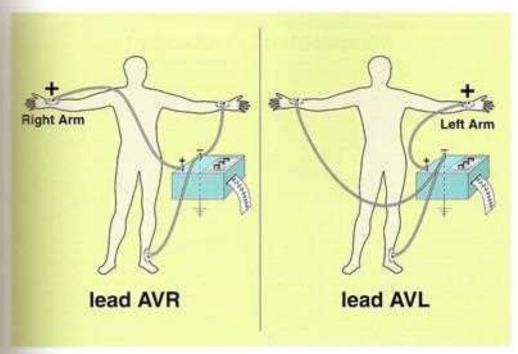


Another standard lead is the AVF lead. The AVF lead uses the left foot electrode as positive and both arm electrodes as a common ground (negative).

The AVF lead uses the left foot electrode as	positis
In AVF both the right and left arm electrodes are channeled into a common ground that has a	
charge.	negativ

Note: Dr. Emanuel Goldberger, who designed and introduced the "Augmented" limb leads, discovered that in order to record a lead in this manner, he had to amplify (Augment) the Voltage in the EKG machine to match the wave magnitude of leads I, II, and III. He named this lead: A (Augmented), V (Voltage), F (left Foot), and he went on to produce two more leads using this same technique.

Aside: Your deductive mind tells you that lead AVF is a combination of leads II and III... just what Dr. Goldberger was trying to accomplish! Therefore lead AVF is a cross between (and oriented between) those two bipolar limb leads. Now, let's create two more augmented leads.



The remaining two augmented limb leads, AVR and AVL, are obtained in a similar manner.

For the AVR lead the Right arm electrode is positive, and the remaining two electrodes are _______ negative

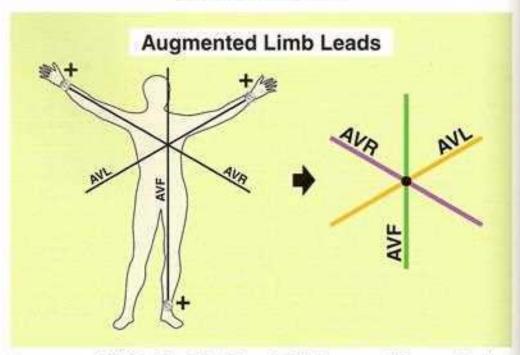
To obtain the AVL lead, the Left arm electrode is made
_____; the other two electrodes are negative. positive

Note: AVR — Right arm positive

AVL — Left arm positive

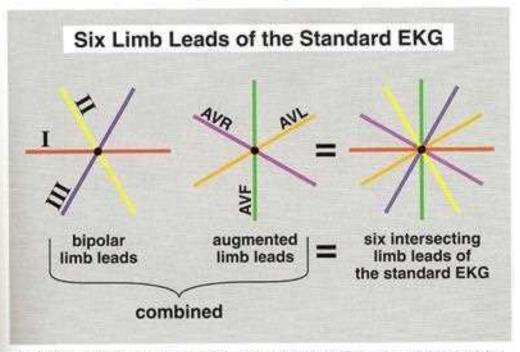
AVF — Foot (left foot) positive

These augmented limb leads are sometimes called the "unipolar" limb leads, stressing the importance of the positive electrode.)



The augmented limb leads, AVR, AVL, and AVF, intersect at different angles than those produced by the bipolar limb leads, and they produce three other intersecting lines of reference.

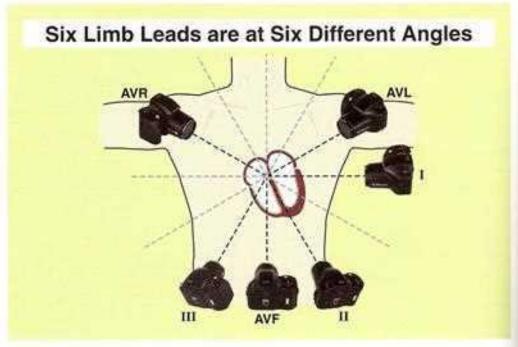
AVR, AVL, and AVF are the augmented (or "unipolar")leads.	limb
These augmented limb leads at 60 degree angles, but the angles differ from those formed by bipolar limb leads, I, II, and III.	intersect
Leads AVR, AVL, and AVF intersect at angles from leads I, II, and III. In fact, leads AVR, AVL, and AVF split the angles formed by leads I. II. and III.	different



All six limb leads (I, II, III, and AVR, AVL, and AVF) meet to form six intersecting leads that lie in a flat "frontal" plane on the patient's chest.

The six limb leads consist of the three bipolar leads, I, II, III, and three augmented leads,, and	AVR, A	VL, and AVF
If the bipolar limb leads I, II, and III are superimpos on augmented limb leads AVR, AVL, and AVF, we intersecting leads in a flat plane on the patient's		chest
The flat plane of the limb leads is called the	plane.	frontal

Note: Don't get bedazzled by the kaleidoscope of limb leads. Bear with me for a few pages, and soon you will understand their utility, and a simplified way to visualize this concept.



Each camera* position represents the positive electrode of a standard limb lead. Each limb lead (I, II, III, AVR, AVL, and AVF) records from a different angle (viewpoint), to provide a different view of the same cardiac activity,

Note: The heart's electrical activity remains constant, but the positive electrode position changes from lead to lead. Therefore the tracing looks slightly different in each lead, as the angle from which we record the electrical activity changes with each lead. Remember, a wave of depolarization is a progressive wave of POSITIVE charges passing through the myocardial cells. So, when a depolarization wave moves toward a POSITIVE electrode, a POSITIVE (upward) deflection is produced on the EKG (or monitor) for that particular lead. (A little repetitious, but it is so important!)

The EKG records the same cardiac in each lead.	activity
The waves look different in various leads because the heart's electrical activity is recorded from a	
different for each lead.	ang

ang= (viewpoin

^{*} If this were a video camera, it could record the information for a cardiac monitor.



It is conceptually necessary for you to visualize the six intersecting limb leads. Why? Can you identify this car?

Note: This page sure seems empty, doesn't it?

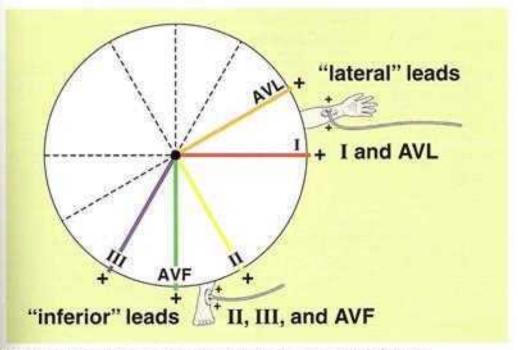
Note: Automobile experts are encouraged not to recognize the car for the sake of understanding the concept.



By observing the same object from six different angles, you will obtain a great deal of information, and in this case, perhaps even identify the car.

Note: You can't see the car's rear bumper in the photo at top left. But with progressively different views, you can determine more about the bumper (or even the driver). Similarly, it may be difficult to see a specific wave in a given lead, but with six different lead positions, it is certain to show up better in other leads.

Note: Observation from six different angles is better than one. Thus recording cardiac electrical activity from six different angles gives us a much greater and more accurate perspective. At this point you can take a sip of coffee and relax. By the way, the car is a 1965 Ford Thunderbird, but it is far more important that the concept (not the car) always remain in your mind.

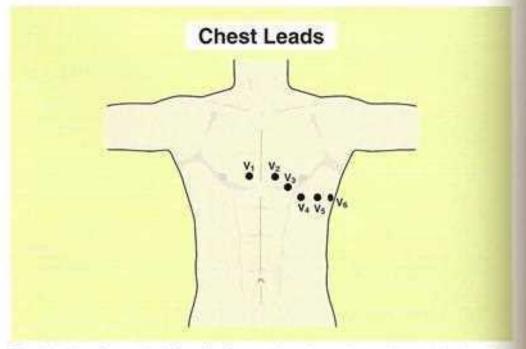


The importance of the positive electrode's position is emphasized by the conventional grouping of limb leads. A positive left arm electrode is used to record "lateral leads" I and AVL, and a positive left foot electrode is used to record "inferior leads" II, III and AVF. The location of the positive electrode is the key.

Leads I and AVL are called the lateral	leads (left lateral	
understood) because each has a	electrode	positive
positioned laterally on the left arm.		

Leads II, III, and AVF are called the *inferior leads* because each of these leads has a positive electrode positioned foot

Note: So now you can determine if depolarization is moving toward for even away from) the patient's left side, and the same for depolarization directed inferiorly toward (or even away from) the left foot. The "inferior leads" and the "lateral leads" include 5 of the 6 limb leads. These are not arbitrary designations. These terms are common cardiology parlance and have important clinical/diagnostic agnificance. Know and understand them.



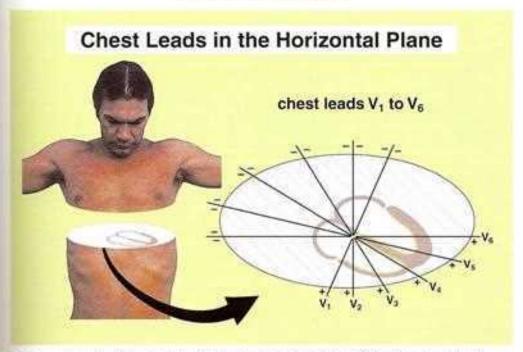
To obtain the six standard **chest leads**, a positive electrode (suction cup) is placed at six different positions (one for each lead) on the chest.

The six chest leads are recorded from six progressively different positions around the, (See illustration.)	ches
For each of the chest leads, the suction cup electrode that is placed on the chest is considered	positive
The chest leads are numbered from V ₁ to V ₆ and are positioned in successive steps from the patient's right to the left side of the chest. Notice how the chest leads	

Note: Traditionally a suction cup electrode records the chest leads, however adhesive electrodes are now commonly used. Because the electrode for the chest leads is always positive, a depolarization wave moving toward a given chest electrode produces a positive (upward) deflection in that chest lead of the EKG tracing.

within the chest.

negative



In general, each of the chest leads* is oriented through the AV node and projects through the patient's back, which is negative.

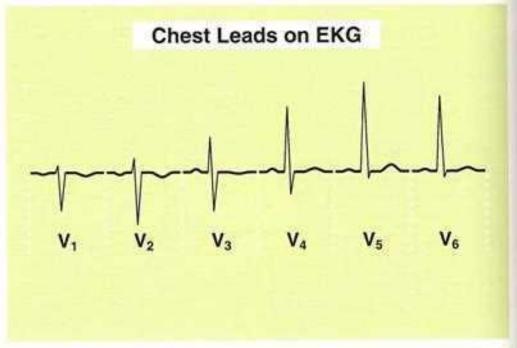
Note: The plane of the chest leads (called the "horizontal" plane) cuts the body into top and bottom halves.

	ach of the chest leads is always (positive or negative).	positive
	V ₆ are imagined to be the spokes ter of the wheel is the	AV Node
\$2000 miles 1000 0000 000 0000 0000 0000 0000 000	a straight line directly from the front patient. In lead V ₂ the patient's back	

__ (positive or negative).

a considered

^{*} The chest leads, also called the "precordial" (in front of the heart) leads, were introduced by Dr. Frank Wilson.

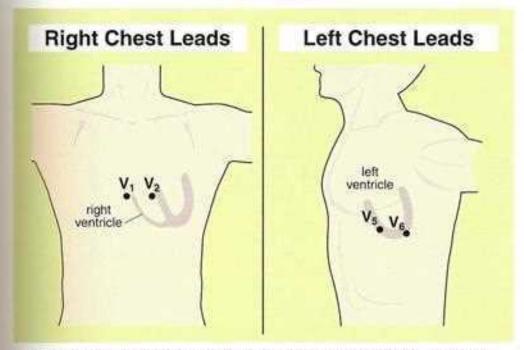


By examining an EKG, you will notice that the waves in the six chest leads show progressive changes from V_1 to V_6 .

Note: When observing the chest leads from V₁ to V₆ you will see gradual changes in all the waves (as the position of the positive electrode changes for each successive lead).

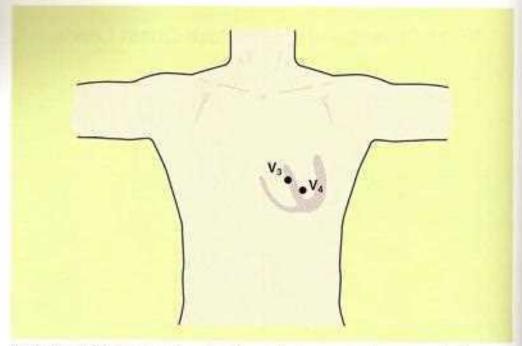
look at page 12).

mainly (positive or negative) normally.	negative
In chest lead V ₆ the QRS complex is usually mainly (positive or negative), Understand why,	positive
Observing the V ₆ chest lead, we know that the mainly positive QRS complex is produced by ventricular depolarization moving the POSITIVE chest electrode of V ₆	toward



Leads V_1 and V_2 are oriented over the right side of the heart, while V_5 and V_6 are oriented over the left side of the heart.

Leads V_1 and V_2 are called the "" chest leads,	right
two chest leads oriented over the left side of the heart and, (and are called the "left" chest leads).	V_5 and V_6
A depolarization wave moving toward the (positive) chest electrode in lead V ₆ causes an deflection on the EKG tracing of this lead. (Now you understand!)	upward (positive)

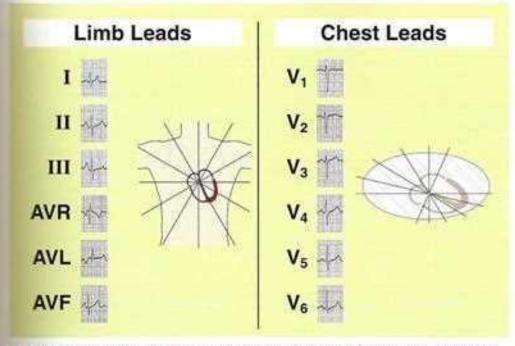


Leads V_3 and V_4 are generally oriented over the interventricular septum. V_3 is nearer the right ventricle, and V_4 is nearer the left ventricle.

Leads V ₃ and V ₄ are oriented over the area of	
the interventricular	

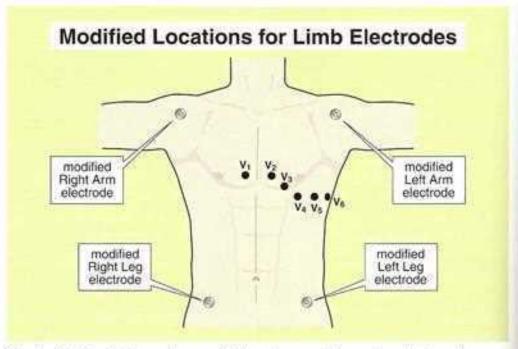
septum

Note: The interventricular septum is a common wall shared by the right and left ventricles, so this septum separates the cavity of the right ventricle from the cavity of the left ventricle. The Right and the Left Bundle Branches course through the interventricular septum.



On the standard EKG tracing there are six chest leads and six limb leads. This is the 12 lead electrocardiogram.

be visualized on the patient's chest	THE STANFACTOR OF THE STANFACT	frontal
The six chest leads lie in the horizontal amanged in progressive order from V ₁		V ₆
The six chest leads are recorded using a such is placed at six specific anatomic maching the heart in the	cal positions on the chest,	horizontal



The six limb leads also can be recorded by using carefully positioned electrodes on the trunk of the patient. The special electrode placement (above) used for exercise ("stress") testing, can be used to record each of the twelve EKG leads.

Note: An EKG recorded from a carefully positioned trunk* electrode can record the same information (same accuracy and same amplitude) as an ankle or wrist electrode for a given limb lead. In this way, a standard twelve lead EKG can be recorded using trunk electrodes.

Cardiac monitoring in hospital rooms, as well as in the emergency department, surgery, recovery room, coronary care, and intensive care, is carried out using modified electrode positions on the patient's ______ to monitor classical limb (and other) leads.

trunk

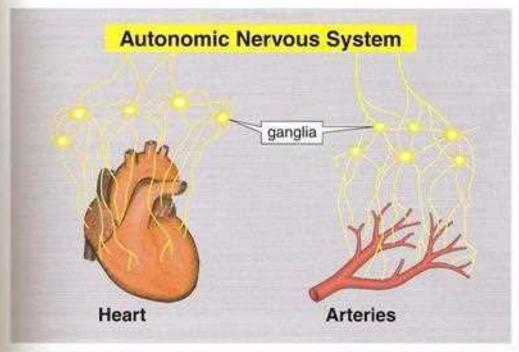
Paramedics and many Emergency Medical Technicians (EMT's) use trunk* ______ for diagnostic purposes and also for telemetry transmission.

electrodes

Now we're ready to tackle the autonomic nervous system... O.K.?

[&]quot;These are "trunk" but not truly "chest" electrodes, for they often use the shoulders and abdomen as electrode locations. A variety of modifications are commonly used to monitor patients in various settings and circumstances (see page 346).

Chapter 3: Autonomic Nervous System



The Autonomic Nervous System (ANS) regulates vital functions of all organs by both reflex and central nervous system control, but not by conscious control.

Although the ANS controls all organs and organ systems,	
main concern is autonomic control of the,	3
and also of the systemic arteries as they relate to blood pressure.	

heart

Note: The ANS has two divisions that sometimes seem difficult to comprehend, because one division may stimulate an organ, yet inhibit another organ. However, these two divisions have well defined soles in the heart and the systemic arteries; one division stimulates, and one division inhibits. It's that simple! One division stimulates cell function, while the other division opposes that stimulation. Each division operates like an electrical system that controls its own terminal switches called "receptors" that modulate the function of cells.

Sote: A stimulus originating in the ANS is transmitted to a ganglion*

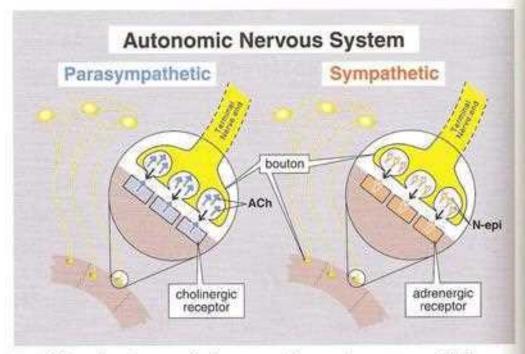
of secondary nerve cells for processing. The nerves of the ganglion

elay the stimulus to their nerve ends, each of which terminates as a

osc called a bouton (bouton is French for button) that covers the

ecceptors of a cardiac cell (or an arterial muscle cell). See next page.

[&]quot;Ganglion" is singular, "ganglia" is plural.



The ANS consists of a sympathetic system and an opposing parasympathetic system. Each of these two systems secretes its own neurotransmitter from its terminal boutons in order to activate specific cell receptors in the cell membrane.

The terminal ends (bouto	ns) of sympathetic nerves secrete
Nor-epinephrine* (N-ep	i), an adrenaline-like neurotransmitter
that activates specific	receptors called adrenergic receptors.

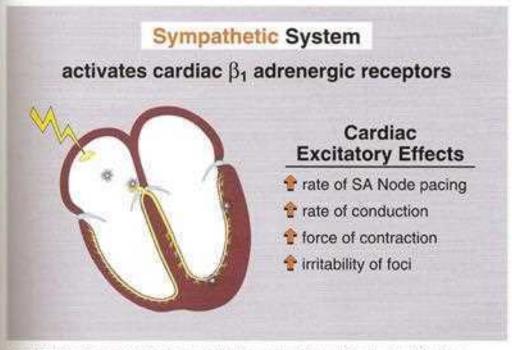
cell

Note: In the heart, the sympathetic and parasympathetic nervous systems have opposite functions. Interestingly, the parasympathetic exercises some direct control of the sympathetic.

The terminal parasy	mpathetic nerve ends (boutons) secrete
the neurotransmitter	Acetylcholine (ACh), which exclusively
activates cell	called <i>cholinergic</i> receptors.

receptors

Nor-epinephrine, hyphenated for recognition purposes, will be "Norepinephrine" from now on. Although the abbreviation "N-epi" is used here, some texts use "NE."

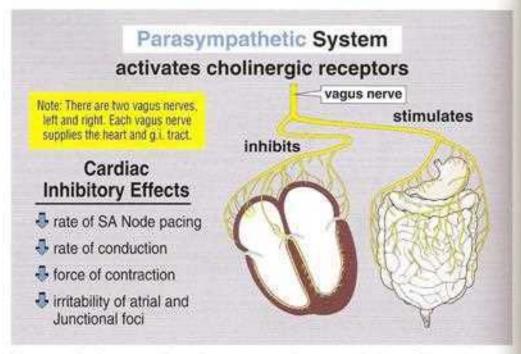


The heart is stimulated by the sympathetic system through its terminal boutons. The boutons deliver N-epi to the β, (adrenergic) receptors; this activates the 5. receptors,* producing an excitatory response at the cellular level.

Screpinephrine (N-epi), the neurotransmitter of the supathetic system, activates the heart's β ₁ (adrenergic) sceptors, stimulating the SA Node to faster. Sepi also:	pace
improves AV Node conduction and accelerates conduction mough the atrial and ventricular,	myocardium
increases the force of myocardial,	contraction
and increases the irritability of atrial and Junctional (page 123) and minimally affects ventricular foci.	foci
N-epi's brother, epinephrine ("adrenaline") is secreted integrated by the adrenal glands. Epinephrine is an even <i>more potent</i> .	o the

amulator of the heart's β, receptors.

^{3.} adrenergic receptors" is often shortened to "β₁ receptors," but adrenergic is understood.



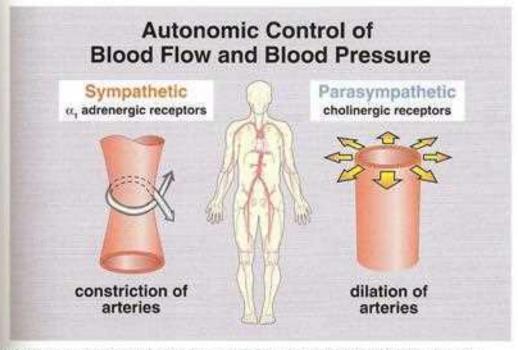
Parasympathetic nerves release the neurotransmitter acetylcholine (ACh), which activates cardiac cholinergic receptors (most are within the atria) to produce a cardiac inhibitory effect. Conversely, the gastrointestinal tract is stimulated by its parasympathetic innervation.

Parasympathetic activation of cholinergic receptors by ACh:

inhibits the SA Node, decreasing the heart,	rate
decreases the speed of myocardial conduction, and depresses the AV	Node
diminishes the force of myocardial,	contraction
and depresses irritability of automaticity,	foci

Note: The vagus nerves are the body's main parasympathetic pathway, so "vagal" stimulation means parasympathetic stimulation, with the understanding that vagal "stimulation" of the heart is inhibitory.

Note: Despite the parasympathetic system's inhibiting effect on the heart, parasympathetic activation of cholinergic receptors stimulates the gastro-intestinal tract. Recalling the agony of severe vomiting or an episode of painful, crampy diarrhea will help you remember the effect of excessive parasympathetic stimulation of the stomach and the bowel.



Besides controlling the SA Node's pacing rate, the Autonomic Nervous System controls blood flow and blood pressure by regulating constriction and dilation of arteries throughout the body.

Sympathetic stimulation of arterial α_1 (adrenergic) receptors constricts arteries throughout the body, increasing blood pressure and blood ______, The α_1 receptors are more responsive the neurotransmitter N-epi than to circulating epinephrine.

flow

Note: By pulling both ends of the Greek alpha, the center loop of the "α" constricts the artery (see arrows on the α in the illustration). Now you will always remember alpha adrenergic sympathetic effects on systemic arteries.

Parasympathetic activation of arterial (cholinergic) receptors

filates the same arteries as above, reducing blood

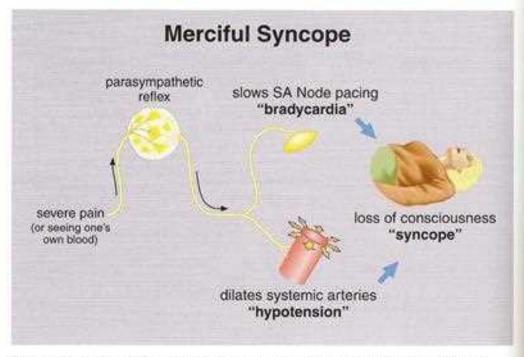
and blood flow. Besides the direct cholinergic inhibition of the

arteries, there is also an indirect inhibitory parasympathetic effect

the sympathetic ganglia that send nerve fibers to the vessels.

pressure

Note: Blood flow is also very dependent on the heart rate: sympathetic stimulation increases the SA Node pacing rate, while the parasympathetic decreases it. Autonomic control of the heart rate and systemic blood pressure involves delicate regulation of the parasympathetic—sympathetic balance to maintain circulatory homeostasis (the ideal status quo).



Severe pain and/or seeing one's own blood may induce a reflex parasympathetic response that causes syncope* (loss of consciousness).

Severe pain and/or seeing one's own blood often initiates

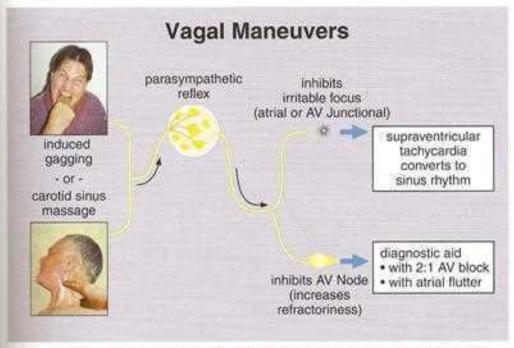
reflex parasympathetic activity that slows SA No	ode	W 184
pacing, known ascardia.		brady
The same reflex parasympathetic response dilate	es systemic	
arteries causing hypotension, as the blood	falls	pressure

Note: A devastating injury, which causes excruciating pain/awareness of bleeding, can induce a parasympathetic response that dramatically lowers the blood pressure and slows the heart. This merciful reflex effectively reduces the brain's blood supply to the point of syncope.

Aside: Perhaps you have encountered (oversensitive) patients who lose consciousness upon seeing their own blood drawn for lab tests, or if they experience minimal pain. Be compassionate; their body is only responding to a normal parasympathetic reflex.

^{*} This and other types of vagally mediated syncope are sometimes called "vaso-vagal syncope." Syncope is pronounced "SINK-oh-pee."

parasympathetic



Cardiovascular sensors provide ("afferent") input for parasympathetic reflexes that counterbalance sympathetic effects. Vagal maneuvers may be employed to produce a reflex parasympathetic response for both diagnostic and therapeutic purposes.

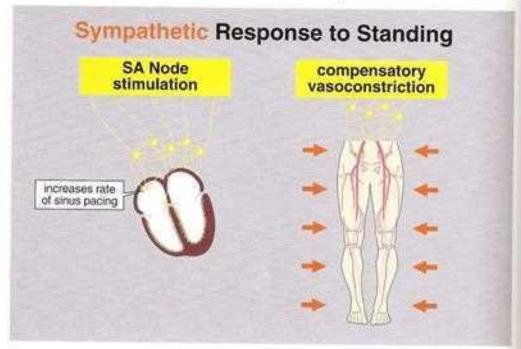
constrointestinal stimulation (e.g., gag reflex) may be comployed to produce a parasympathetic	response
Carotid sinus massage may be used in carefully selected	
aroug sinus massage may be used in carefully selected	

response.

Note: An induced parasympathetic response may be used therapeutically to depress an irritable focus in the atria or AV Junction. An induced parasympathetic response may be diagnostically employed to transiently slow AV Node conduction, or to make the AV Node more refractory to depolarization usee pages 160 and 183).

nutients" to produce a

^{*}Injudicious use of carotid sinus massage in some patients can dislodge a piece of atheromatous plaque, sending plaque emboli to the brain (careful!). Use discretion when employing diagnostic procedures that can cause introgenic stroke, which can disable a patient and incite a feeding frenzy of lawyers.



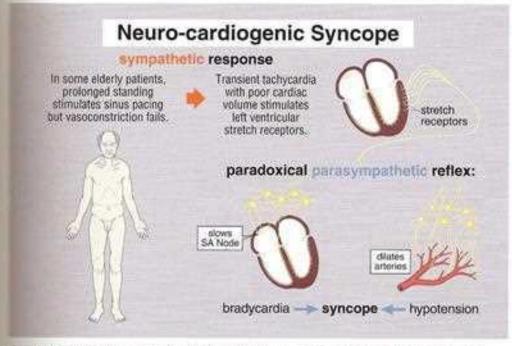
It seems as though standing would allow blood to gravitate into the lower extremities. However, standing produces a compensatory sympathetic response that constricts peripheral arteries to prevent distal blood pooling, and stimulates sinus pacing.

Note: The body has "pressure" receptors* that detect low blood pressure, particularly with standing. These pressure receptors initiate a sympathetic reflex that constricts peripheral arteries and increases the heart rate slightly, thereby conserving blood flow to the brain.

Impaired function of this normal sympathetic response to standing can diminish blood flow to the brain, causing	120	syncope
Orthostatic hypotension is an abrupt fall in blood pressure caused by failure of these compensatory		
sympathetic mechanisms upon		standing

^{*} Careful! These "receptors" are cardiovascular sensors, called baroreceptors, that the body uses to detect changes in blood pressure. Please don't confuse them with the cell membrane receptors that are activated by N-epi or ACh.

syncope



Standing produces a sympathetic vasoconstriction response to maintain adequate circulation. This compensatory mechanism may fail with prolonged standing in circular elderly patients, triggering a paradoxical parasympathetic response causing scope.

Pooling of blood in the lower extremities from prolonged anding is normally compensated by a reflex sympathetic increase in blood pressure and heart rate. However, in some elderly patients, sus pacing accelerates, but peripheral vasoconstriction is inadequate. So, the partially filled ventricles contract rigorously, stimulating masympathetic mechanoreceptors in the left ventricle. This initiates undesirable parasympathetic reflex that slows SA Node pacing and aduces blood pressure; so blood flow to the brain is reduced, causing sencope. This is neuro-cardiogenic syncope.

cardiogenic syncope, a (paradoxical) parasympathetic conse to prolonged standing, causes vasodilation and ing of the pulse, resulting in a loss of	. consciousness
ander controlled circumstances, a Head Up Tilt ("HUT") test	

ms the diagnosis of neuro-cardiogenic

- 1. Rate
- 2. Rhythm
- 3. Axis
- 4. Hypertrophy
- 5. Infarction

Knowing basic cardiac principles and understanding the autonomic nervous system ensures mastery these five general areas of routine EKG interpretation.

Proper interpretation of an _____ requires consideration of Rate, Rhythm, Axis, Hypertrophy, and Infarction. They are all equally important.

EKG

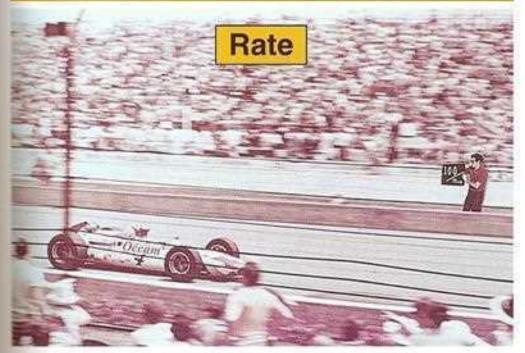
Note: Take a moment and examine page 334 to observe the simple methodology that will become your routine.

Before you begin each chapter, preview its summary (pages 335 to 346). Then, as you progress through the chapter, little "aha's" of recognition will flash in your brain, and you will appreciate how each concept is carefully woven into this simplified methodology. Your understanding evolves rapidly; this is the foundation of your permanent knowledge.

Ready? Let's go!

Chapter 4: Rate

Before you begin, look at this chapter's summary on pages 334 and 335.



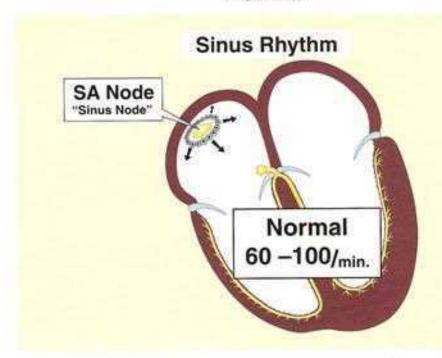
When reading an EKG, you should first consider the rate.

Note: The sign in this picture is not informing the driver* about the rate of his race car. The man holding the sign is a physician who has been monitoring the driver's transmitted EKG. The sign is telling the driver about his current heart rate (he's a little excited).

Shen examining an EKG, you should determine first.	rate
The rate is read as cycles per,	minute

let's examine where and how the normal heart rate originates...

With a sincere dedication to Billy Occam, deceased long ago, who made simplicity a virtue of science.



The SA Node (Sinus Node), the heart's pacemaker and the dominant center of automaticity, generates a Sinus Rhythm. The SA Node paces the heart in the normal rate range of 60 to 100 per minute.

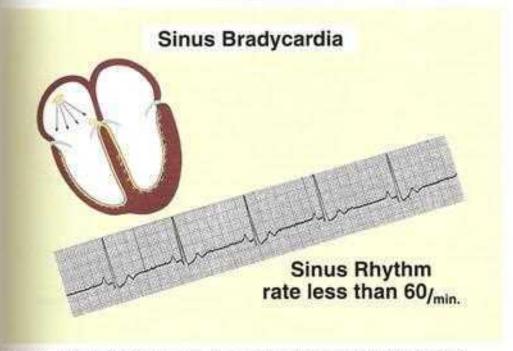
The heart's normal pacemaker, the, generates a continuous series of regular, pacemaking stimuli (this is its "automaticity").	
The SA Node is located within the upper-posterior wall of the right The SA Node emits a regular series of pacemaking (depolarization) stimuli.	atrium

Note: The Sinus Node (SA Node) is the heart's dominant center of automaticity, and the normal, regular rhythm that it generates is called the Sinus Rhythm.

At rest, the Sinus Rhythm maintains a rate of 60 to _____ beats per minute, which is the normal range of the pacing rate.

TERRORS OF NO DE DE

100



the Sinus Node (SA Node) paces the heart at a rate slower than 60 per minute, is Sinus Bradycardia.

Note: "Brady" = slow; "cardia" = heart.

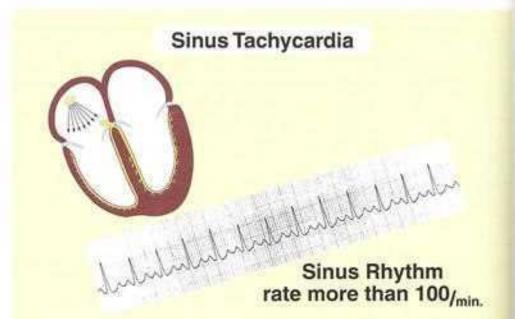
* bythm originating in the heart's normal pacemaker, the Node, with a rate slower than 60 per minute is called

Bradycardia

Sinus Bradycardia most often results from parasympathetic excess, as we see in conditioned athletes at rest. Sometimes an extremely slow heart rate may reduce blood flow to the brain causing loss of consciousness (syncope). See pages 60 and 63.

Bradycardia is present if the SA Node produces

second (careful!)



If the Sinus Node (SA Node) paces the heart at a rate greater than 100 per minute, this is Sinus Tachycardia.

Note: "Tachy" = fast; "cardia" = heart.

A rhythm originating in the SA Node (Sinus Node) is called Sinus Tachycardia if the rate is greater than per minute.

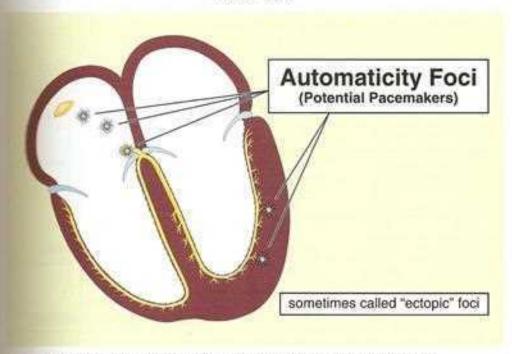
100

Exercise produces sympathetic stimulation of the SA Node; this is the most common cause of Sinus ______

Tachycard

Note: There are focal areas of automaticity in the heart known as automaticity foci. * They are potential pacemakers that are capable of pacing in emergency situations. Under normal circumstances, these foci are electrically silent (that's why they are referred to as "potential" pacemakers).

[&]quot;Automaticity foci" refers to more than one "automaticity focus"; in fact, when the word "foci" is used alone, "automaticity foci" is understood. Foci is pronounced "FOE-sigh."



sormal SA Node pacemaking fails, other potential pacemakers known as maticity foci (also called "ectopic" foci) have the ability to pace (at their interent rate). They are in the atria, the ventricles, and the AV Junction.

SA Node ceases to function, one of the potential pacemakers, as an automaticity focus, will assume pacemaking activity assumerent (only one focus assumes pacing responsibility).	rate
The atria have automaticity of potential pacemakers are within the atrial conduction system (see page 101), they are called atrial automaticity foci.	foci
The proximal end of the AV Node has no automaticity foci, sever the middle and distal regions of the AV Node, an area as the AV Junction does have automaticity foci that are alled Junctional automaticity foci.	
Terainje fibers have automaticity foci, so there are foci of	

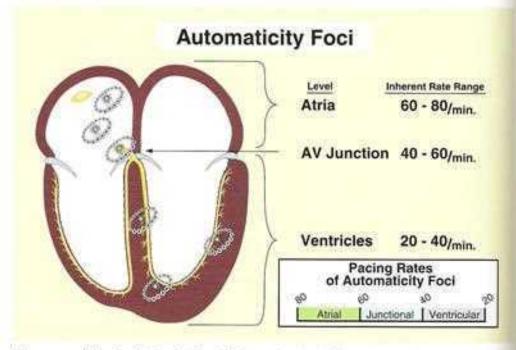
in the His Bundle and in

Bundle Branches and their subdivisions; these foci

ed ventricular automaticity foci.

pacemakers

and potential

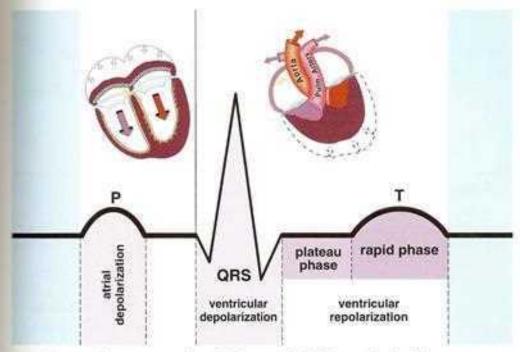


The automaticity foci of each "level" (the atria, the AV Junction, and the ventricles are each a "level") have a general range of pacemaking rate. Although all foci of a given level pace within a general rate range, each individual automaticity focus has its own precise inherent rate at which it paces.

Each automaticity focus of the atria has a specific inherent rate at which it paces, but its inherent rate falls within the general range of ____ to 80 per minute.

The automaticity foci of the AV Junction all pace in the range of _____ to 60 per minute, but any single Junctional focus paces at its individual inherent rate.

Ventricular automaticity foci all pace in the ____ to 40 per minute range, but any specific ventricular focus has a distinct inherent rate of pacing.



A cardiac cycle is represented by the P wave, the QRS complex, the T wave, and the baseline that follows until another P wave appears. This cycle is repeated continuously. Please study the illustration to make certain that you understand every event in sequence,

Note: Physiologically, a cardiac cycle represents atrial systole (atrial contraction), followed by ventricular systole (ventricular contraction), and the resting stage that follows until another cycle begins.

Atrial depolarization (and contraction) is represented by the wave.

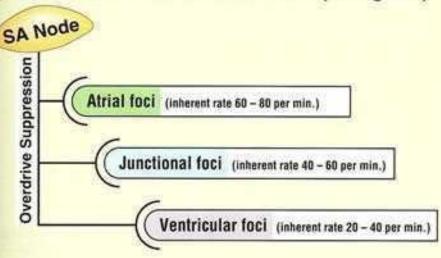
P.

Ventricular depolarization (and contraction) is represented by the _____ complex.

ORS

Note: In reality, atrial contraction lasts longer than the P wave, and ventricular contraction lasts longer than the QRS complex, but you already knew that.

SA Node overdrive-suppresses all foci (since all foci have a slower inherent pacing rate)



— pid automaticity (pacemaking activity) suppresses slower automaticity pacemaking activity) – this is overdrive suppression, a very important fundamental characteristic of all automaticity centers.

Note: Overdrive suppression is characteristic of all centers of automaticity (including the SA Node and all automaticity foci).

Simply stated: any automaticity center will overdrive-suppress*

others that have a slower inherent pacemaking rate.

SA Node overdrive-suppresses the (slower)

between pacemaking activity of all the automaticity

below it; this provides the SA Node with the luxury

thaving to compete with slower pacemaking

below it is the second in the seco

foci

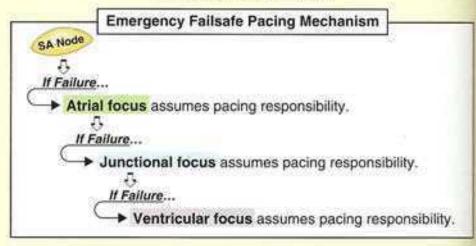
a fact, once an automaticity focus actively begins pacing, all overdrive-____ all lower (slower) foci,

suppress

eliminating any competition, Well Designed!

"Then used as a verb, "overdrive-suppress" is hyphenated... so says the publisher.

Overdrive Suppression provides emergency backup pacing at 3 separate levels



Overdrive suppression is the heart's failsafe pacing mechanism, providing three separate levels of backup pacing, by utilizing automaticity foci in the atria, the ventricles, and the AV Junction.

Note: An automaticity focus actively pacing at its inherent rate, overdrive-suppresses all slower foci including slower foci at its own level.

activity at all levels that are	it.

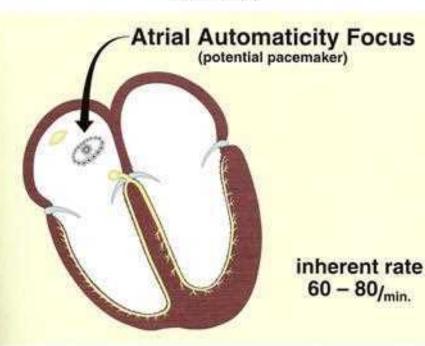
Therefore, an automaticity focus only emerges to function as a pacemaker when it is no longer _ -suppressed. For instance, in SA Node failure...

Del III

overg

...a focus from a lower level - no longer overdrive-suppressed by regular pacing stimuli from above – can emerge to pace. Very well Designed!

Let's do that once again, slowly.



The atria have automaticity foci of potential pacemakers, any one of which can assume active pacemaking responsibility in its inherent rate range of about 50 to 80 per minute, if normal pacemaking fails.

SA Node pacing fails, an atrial automaticity focus can
sume the active pacemaking responsibility in its inherent
range of about 60 to ____ per minute (close to the
Node's normal rate).

80

SA Node fails, an atrial automaticity

whin the atrial conduction system) may then

me active pacing responsibility to become the

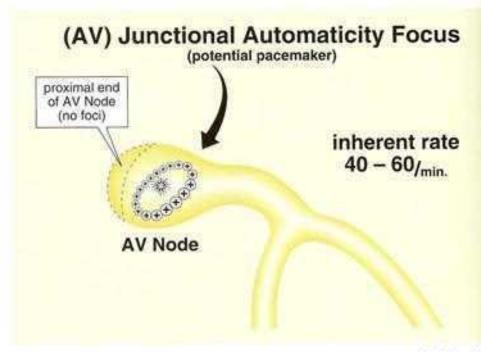
meant pacemaker. (The last sentence on this page

sentence on this page

focus

Second SA Node pacing, an atrial automaticity focus
 Second Samuel Samu

inherent



The AV Junction has automaticity foci (potential pacemakers), one of which will emerge to actively pace in its inherent rate range of 40 to 60 per minute if there is an <u>absence</u> of regular pacing stimuli progressing down from the atria.

Note: The AV Junction is that portion of the AV Node that has foci of automaticity. The proximal end of the AV node has no foci. The AV Junction has foci of automaticity called "Junctional foci."

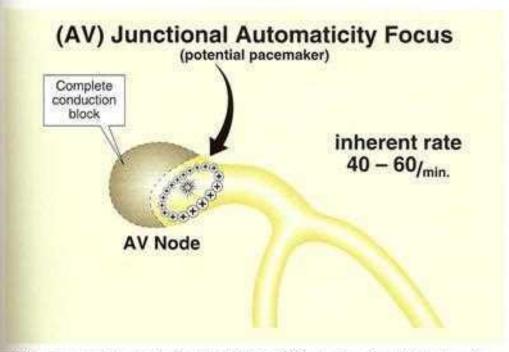
An automaticity focus in the AV Junction begins active backup pacing only in the absence of pacing stimuli coming down from the atria. Then, no longer overdrive-suppressed, it emerges to actively pace in its inherent _____range of 40 to 60 per minute, and it overdrive-suppresses all lower (slower) automaticity foci, becoming the dominant pacemaker.

A Junctional focus actively pacing at its inherent rate

(___ to 60 per minute range), produces an idio-junctional* rhythm.

Note: A Junctional focus (that is, an automaticity focus in the AV Junction) emerges as the active pacemaker if it is no longer overdrive-suppressed by regular pacing stimuli from above. This can occur if the SA Node and all atrial foci fail. But wait — something else can prevent a Junctional focus from being depolarized by regular pacing stimuli from above. Next page!

^{*} The prefix "idio" is of Greek origin, and it means "one's own." Idiojunctional is usually not hyphenated.

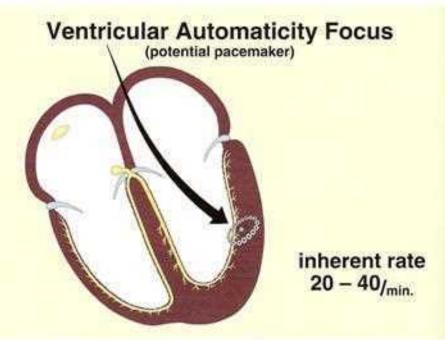


If there is a complete conduction block in the AV Node above the AV Junction, then no regular paced depolarization stimuli from above reach the automaticity foci in the AV Junction.

Note: You will recall that the AV Node is the only conduction link between the atria and the ventricular conduction system below.

BE AV Junction, an automaticity in the AV Junction Below, receives no pacing stimuli from above	focus
so, no longer overdrive-suppressed, the Junctional focus scapes to become the active pacemaker for the ventricles. and that Junctional focus paces the ventricles at its inherent rate	40 - 60
per minute while overdrive-suppressing lower, wer) ventricular foci.	40 - 60

It is possible for the AV Junction (together with all its maticity foci) to suffer a complete block. In that instance, only an maticity focus in the Purkinje fibers of the ventricles can come to the rescue to pace the ventricles. Let's see how...



The ventricles have automaticity foci (potential pacemakers), any one of which will assume pacing in its inherent rate range of 20 to 40 per minute, if the usual overdrive suppression (due to regular pacing stimuli from above) is absent.

Note: Ventricular automaticity foci are composed of specialized Purkinje fibers. These pacemaking foci are in the His Bundle, the Bundle Branches, and all their subdivisions, since they are all composed of Purkinje fibers.

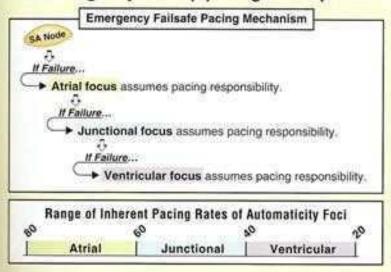
Without overdrive suppression from above, a ventricular automaticity focus emerges to actively pace in its inherent rate range of ____ to 40 per minute; this is an *idio-ventricular** rhythm.

Note: A ventricular focus emerges as the active ventricular pacemaker only if it is no longer overdrive-suppressed by regular, paced stimuli from above. This occurs:

- if all pacemaking centers above it have failed.
- if there is a complete block of conduction below the AV Node (including the AV Junction) that prevents any pacing stimulus above it (i.e., from the SA Node, an atrial focus, or a Junctional focus) from reaching the ventricles

^{*}Hyphenated here for ease of recognition, idioventricular should not be hyphenated.

OVERDRIVE SUPPRESSION provides emergency backup pacing at 3 separate levels



SA Node pacing fails, an automaticity focus in the atria, or the AV

tion, or even the ventricles (in that order) is available to assume the pacemaking

bility at its own inherent rate. This provides three levels of backup pacing.

SA Node should cease pacing, an atrial automaticity focus

acce in its inherent rate range of 60 to 80 per minute; failing

bushup pacing by a Junctional focus will assume the active

responsibility in its (slightly slower) inherent rate range

for per minute.

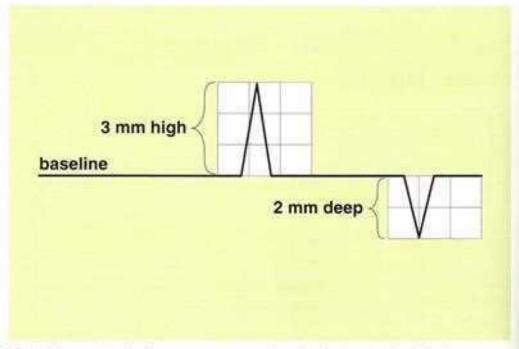
pacing

rate range of 20 to 40 per minute, if the focus
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rate range of 20 per minute, if the

inherent

a physiological or pathological emergency, an irritable subcity focus may suddenly discharge at a rapid rate. This rate (150 to 250 per minute) is approximately the faci of all levels.

something real easy...



The height and depth of a wave are measured vertically from the baseline in millimeters, and this vertical amplitude represents a measure of voltage.

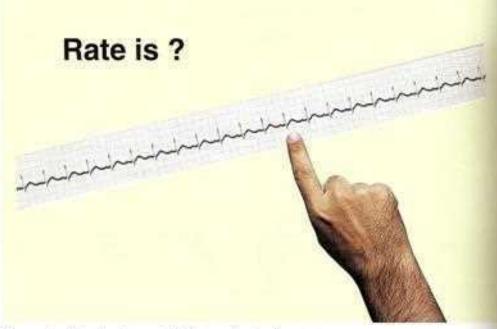
The height or depth of waves is measured from the	
baseline in millimeters and is a measure of	voltage

Note: The deflection of a wave is the direction in which it records on EKG; for instance, the "upward deflection" or "downward deflection" of a wave. However, the amplitude of a wave is the magnitude (in millimeters) of upward deflection or downward deflection. The height or depth of a wave (i.e., its amplitude) is a measure of voltage.

The first wave in the illustration has an upward	
deflection of 3 mm in	amplitude

Note: The elevation or depression of segments of baseline is also measured vertically in millimeters, just as we measure waves.

^{*}Ten millimeters vertically represents one millivolt (mV), however, in practice, one usually speaks of "millimeters" of height or depth (waves) and the same for elevation or depression of baseline segments.



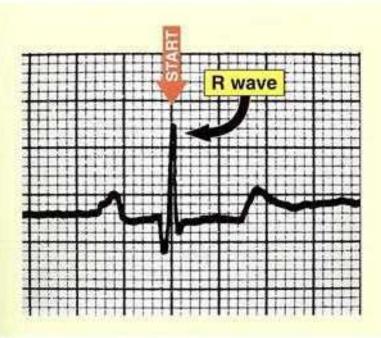
Our main objective is to rapidly determine the heart rate.

After finishing this chapter you will be able to determine the rapidly.	T.
No special devices, calculators, rulers, or awkward mathematical computations are needed in order to the rate.	determine

Note: In emergency situations, you probably will not be able to find, much less use a calculator; and you may not have the presence of mind (or the time) to do mathematical calculations.

Observation alone can give us the ______

Tale



First: Find an R wave that peaks on a heavy black line (our "start" line).

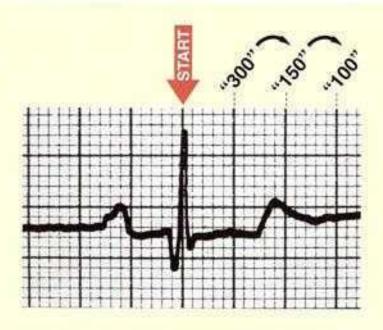
alculate rate, you should first look at the ___ waves.

R

find one that peaks on a heavy black line,

we will call it the "line."

start



Next: Count off "300, 150, 100" for the three thick lines that follows the start line, naming each line as shown. Memorize these numbers.

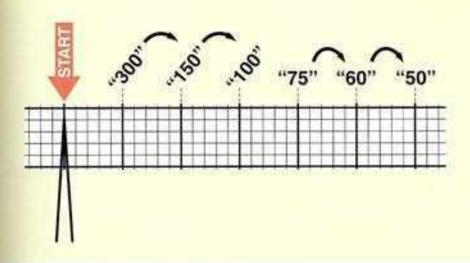
An R wave peaks on a heavy black start line...

th	e <u>next</u> heavy b	lack line is named "" followed by	300
	" and "	" for the next two heavy black lines.	150, 10

Note: The line that the R wave peaks upon is the start line; we only name the heavy lines that follow the start line.

The three lines following the start line (where the R wave falls) are named "_____, ____, "in succession. 300, 150, IIII (Say them out loud!)

Again!



Then: Count off the next three lines after "300, 150, 100" as "75, 60, 50,"

The next three	lines after	"300.	150,	100"	are
med "	. 60, 50,"				

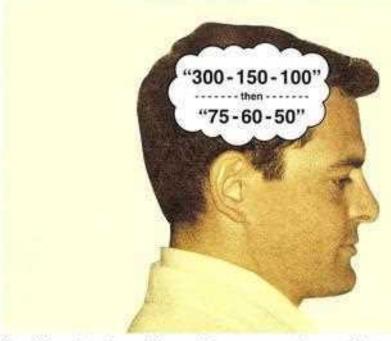
75

Remember the next three lines together as:

75, 60, 50

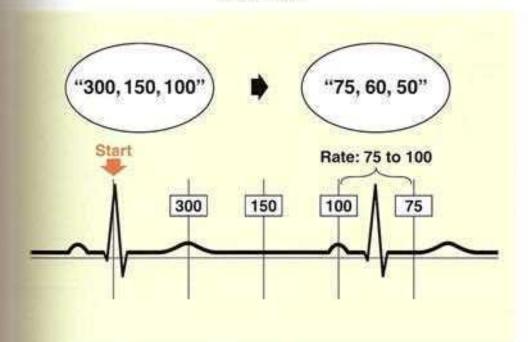
Once more out loud, please.

good!



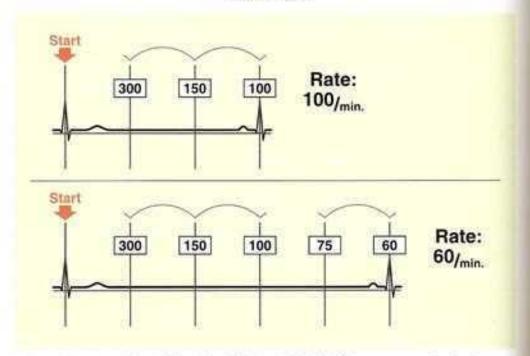
Now: Memorize these triplets until they are second nature. Make certain that you can say the triplets without using the picture.

These triplets, "300, 150, 100" and "75, 60, 50" must be	memoriza
Be able to name the lines that follow the start line on which an R wave; it is easy to remember them as triplets, and so easy to use immediately. (Can hardly wait!)	peal (fall
Do not count those lines that follow the start line - name them with the as you go.	triple



Where the next R wave falls, determines the rate. It's that simple.

an R wave peaked upon a heavy black (start) line, took for the R wave.	next
There is no need for mathematical computations.	rate
the next R wave falls on "75"	minute
Note: You may have noticed that the illustration shows the sormal rate range of 75 to 100.	



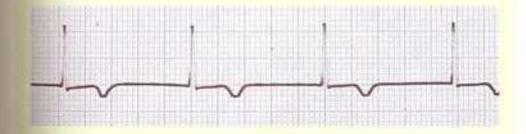
By knowing the triplets "300, 150, 100" then "75, 60, 50" you can merely look at an EKG and tell the approximate rate immediately.

The triplets are: first "_____, ____." 300, 150, 100 then "_____, ___." 75, 60, 50

By simply naming the lines following the start line, you can identify the rate immediately using the ______.

triplets

Practice Tracing



is an EKG tracing from a resting patient, whose heart rate is slower than the last rate one would see with a Sinus Rhythm, Let's examine the rate.

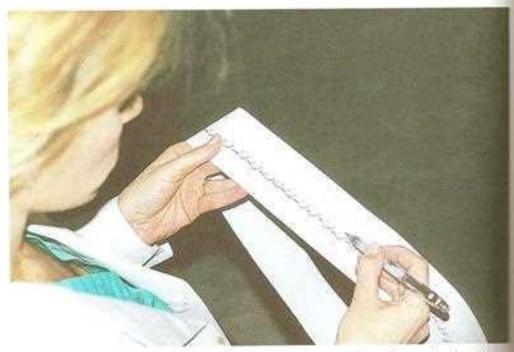
the rate in the above tracing is about ____ per minute.

60

were told that this rhythm probably originated in an maticity focus, by the rate alone, you would suspect the pacemaker) to be in the

AV Junction

This is indeed a rhythm originating in the AV Junction, and why you don't see P waves. This elderly woman has a very leased heart. Her SA Node failed, then all the atrial automaticity foci led. Fortunately, a Junctional focus came to the rescue. This natural backup system is wonderfully effective.

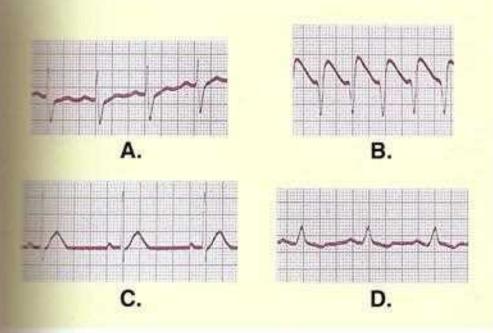


You do not need to depend on mathematical computations in order to calculate the rate. Observation alone will do it!

You can rapidly	determine the rate on an EKG tracing	0.00000
by	alone.	observa

There is no need to depend on annoying math or calculators (where did I put that darn thing?) in order to determine the

Note: You will always have your brain with you (at least until that time when brain transplants would provide you with someone else's brain). Just remember to name the lines that follow the *start* line using the triplets, and say: "300, 150, 100" then "75, 60, 50." Enough, enough... let's try it!



let's determine the approximate rates of these EKG tracings.



As you may have discovered for yourself, any prominent wave the S wave in example B.) can be used to determine the rate.

The distance between the heavy black lines represents 1/300 min.

So two 1/300 min. units = 2/300 min. = 1/150 min. (or 150/min. rate)

and three 1/300 units = 3/300 = 1/100 min. (or 100/min. rate)

There is a logical explanation for the seemingly unusual rate determinations using the triplets.

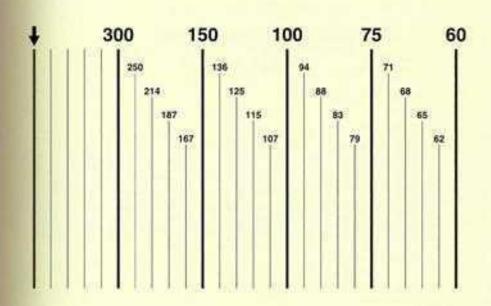
Note: The unit of time (duration) between two heavy black lines is .2 sec., which is 1/300th of a minute.

The number of time units between five consecutive heavy black lines is ____.

So this represents 4/300 minute or a rate of _____ per minute.

Therefore if a heart contracts 75 times per minute, there will be a span equal to the distance between five heavy black lines between the ______complexes.

Note: Reasonable instructors should not require students to master this page. As author, I have not personally memorized the text material on this boring page. Let's keep it simple and practical.

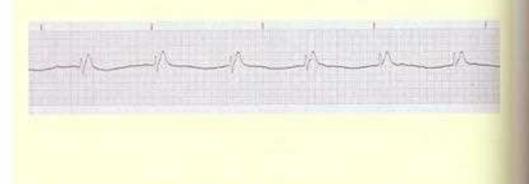


Due fine line divisions can provide more precise rate determination. Memorizing them is impractical, so when determining fast rates, most of us use a reference like the one provided in the Personal Quick Reference Sheets (page 335).

Sore: It is admittedly a great task to memorize the fine line
subdivisions, so you can use page 335 as a personal reference
when you need it. Determining the rate range using the triplets
smore than adequate in most cases.

Sote: For rates less than sixty per minute, see the next few pages a simple way to determine rate when you see a bradycardia.

Bradycardia (slow rates)



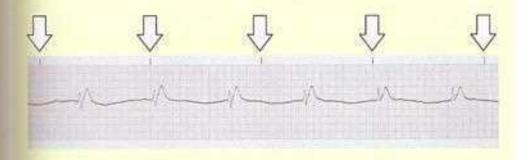
For very slow rhythms, there is an easy method for quickly determining the rate.

The proper term for slow heart rate is _______. bradycards

Note: The triplets give us a very large range of rates. By using the triplets "300, 150, 100" then "75, 60, 50" you can determine rates ranging from 300 to 50. Bradycardia means a rate slower than 60 per minute.

For bradycardia you can use another easy method to determine the _____. I'll show you on the next page...

"3 second" marks



On the top margin of every EKG strip, there are small marks that identify the "three second" intervals.

There	are small marks above the graph portion o
the_	tracing. Find a strip of EKG tracing
and ex	amine it.

EKG

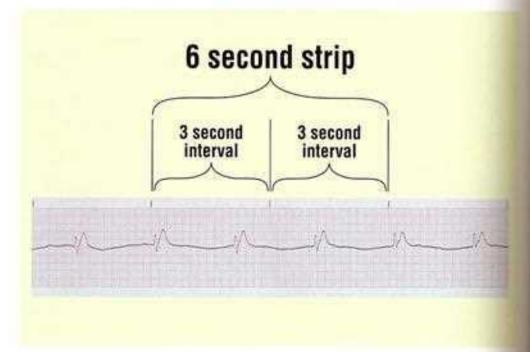
Two of these marks enclose a three second ______.

interval

Note: Some EKG paper has "3 second intervals" that are marked with a dot, circle, triangle, or a vertical line.

when an EKG machine is running, the span of paper between two of these "3 second interval" marks basses under the stylus needle in ______.

3 seconds

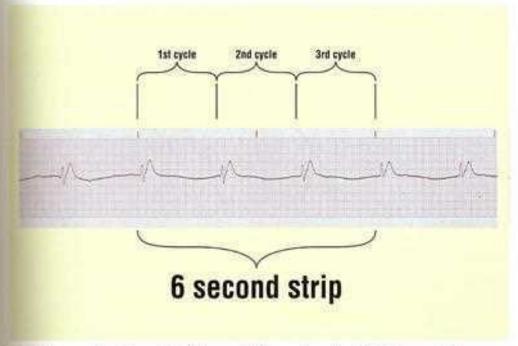


Taking two of the three second intervals, we have a 6 second strip.

Note: A three second interval is obviously the distance between two consecutive 3 second interval marks.

Taking two of the three second intervals gives us a 6 second ______.

This 6 second strip represents the amount of paper used by the machine in six seconds (one-_____ of a minute).



Count the number of complete (R wave to R wave) cycles in this 6 second strip.

In the marked bradycardia, there will be few cycles per 6 second strip.

length of a cardiac can be measured as a specific wave until the wave is repeated again.	cycle
So R wave to wave gives us the duration (length) Come cardiac cycle.	R
Count the number of cycles in the 6 second	strip

Find the rate by multiplying the number of cycles in the six second strip by ten (10)

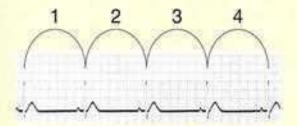
Time to

Ten of the 6 second strips equals one _____ (time) of EKG tracing.

The number of cycles per minute is the _____.

So cycles per 6 second strip multiplied by ____ equals the rate. Simple!

So, if there are 4 cycles per six sec. strip...

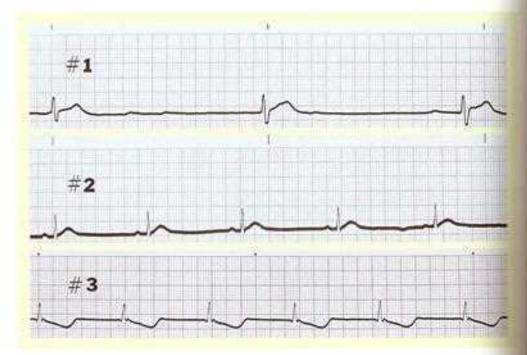


the rate is 40

The can just place a zero on the right of the number of cycles per six second strip,

slow heart rates (bradycardia), you should	
m find a six second,	strip
the number of in this strip,	cycles
and multiply by to get the rate.	10

Multiplying by ten may be done by placing a zero on the sade of the number of cycles per six second strip. For instance, together the second strip gives a rate of 50.



Let's determine the approximate rates of these EKG's.

Rates: No. 1. ____ per minute

No. 2. ____ per minute

No. 3. ____ per minute

usually determined using this method.

about -

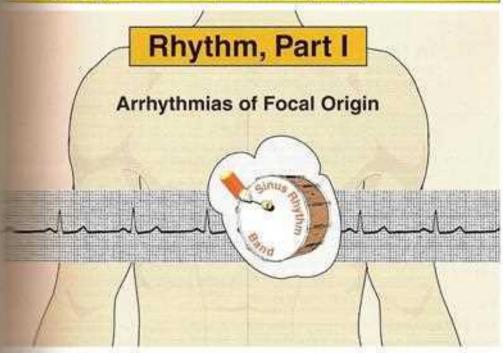
Note: The general, average rates of irregular rhythms are

Why don't you obtain some EKG tracings and amaze yourself (and your friends) at how easily you can determine the rate.

Note: Take a minute to review the illustrations in this chapter, then turn to the Personal Quick Reference Sheets at the end of this book for a simplified summary of determining rate (page 335).

Chapter 5: Rhythm, Part I

size you begin, look at this chapter's summary on pages 334 and 336-338.



EKG provides the most accurate means of identifying a cardiac arrhythmia mormal rhythm), which can be diagnosed easily, once we understand the arrophysiology of the heart.

rhythm
ased to denote any abnormal rhythm. The term
bythmia" (bad rhythm) has the same meaning,
also is commonly used in medical literature.

EKG records the heart's electrical phenomena that

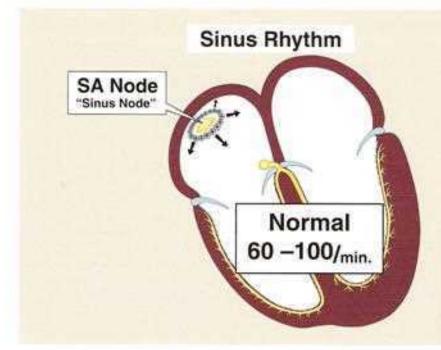
not be seen, felt, or heard on physical examination,

EKG is a very accurate means of recording

disturbances.

rhythm

To understand the arrhythmias, you must first become familiar the normal electrophysiology of the heart, including the normal addition pathways.



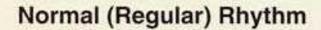
The SA Node generates a regular* Sinus Rhythm that paces the heart. Each pacemaker impulse from the SA Node (Sinus Node) spreads through both atria as an advancing wave of depolarization.

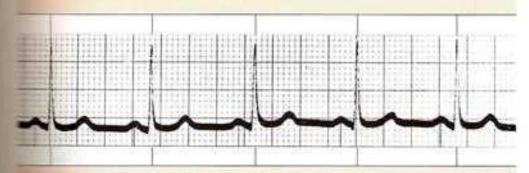
It is the <u>automaticity</u> of the Sinus Node (SA Node) that generates the regular* cadence of depolarization stimuli for pace—______activity.

Normally, the SA Node discharges regular pacing impulses (60 to 100 per minute) that depolarize the _____.

Note: We know that the SA ("Sino-Atrial") Node is the same as the Sinus Node, so we understand that the terms "Sinus" and "Sino" imply SA Node origin.

^{*} The term "regular" indicates a rhythm of constant rate. See next page...





equal distances between identical waves

- EKG there is a consistent distance (duration) between similar waves during a likely regular cardiac rhythm, because the SA Node's automaticity precisely mains a constant cycle duration between the pacing impulses that it generates.
- All automaticity foci pace with a regular rhythm,
- SA Node generates pacing impulses at a constant,
 ung rate, producing cycles of equal length, so the rhythm
 teart is said to be ______. This characteristic
 of regularity is typical of SA Node pacing.*

regular

repeating cycle, there is a predictable regularity

smilar (named) waves. Therefore, irregularities
are easy for you to spot on EKG.

rhythm

- We can visually scan an EKG and appreciate the repetitive unity of a regular rhythm. But breaks in that continuity, such as a the presence of too-early (premature) beats, or sudden, dramatic ange, immediately catch our attention, warning us of a rhythm persence.
- Normal Sinus Rhythm varies imperceptibly with respiration.

Sinus "Arrhythmia"

SA Node's pacing rate normally varies with respiration



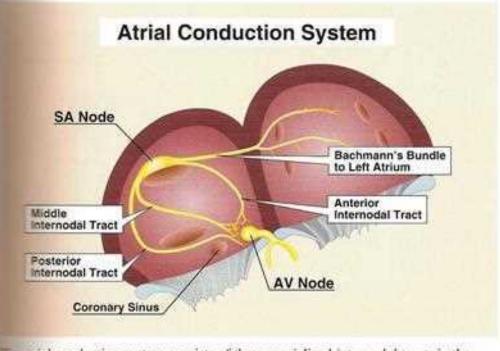
A normal physiological mechanism, Sinus Arrhythmia, sounds pathological ("arrhythmia" = abnormal rhythm), but it functions in all humans at all times. The autonomic nervous system causes barely detectable rate changes in Sinus pacing that relate to the phases of respiration. This is not a true arrhythmia.

Note: Sinus Arrhythmia is a normal, but extremely minimal, increase in heart rate during inspiration, and an extremely minimal decrease in heart rate during expiration.

Sinus Arrhythmia represents normal, minimal variation	ons
in the SA Node's pacing rate in association with the	maran.
phases of	respira

Note: The slight increase in the heart rate is due to inspiration-activated sympathetic stimulation of the SA Node. The slight decrease in pacing rate is due to expiration-activated parasympathetic inhibition of the SA Node. Perhaps you knew that already, since Sinus pacing is regulated both divisions of the Autonomic Nervous System.

Note: This variability of Sinus Rhythm is normal. In fact, if the heart rate variability is reduced, this is pathological and is a valuable indicator of increased mortality, particularly after infarction. Parameters of "Heart Rate Variability" are being established for determining patient prognosis in many types of heart disease.



atrial conduction system consists of three specialized internodal tracts in the atrium (the Anterior, the Middle, and the Posterior), and one conduction tract as Bachmann's Bundle that innervates the left atrium.

conduction pathways in the right atrium cour	se from
SA Node to the AV Node (thus the term "Intern	odal").
are the Anterior, the Middle, and the	
modal Tracts.	

Posterior

mann's Bundle originates	in the S	A Node and	distributes
rization to the left			

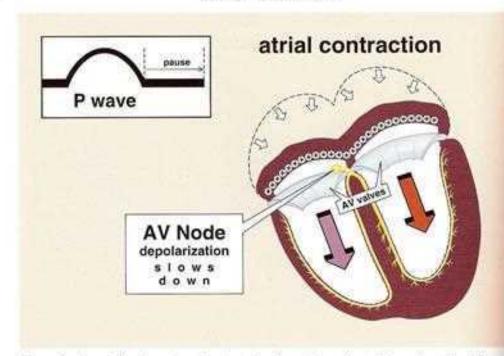
atrium

does not record on EKG; however, depolarization of the myocardium produces a ____ wave on EKG.

P

Just as ventricular automaticity foci are within the ventricular markinje fibers, similarly, atrial automaticity foci are within the mecialized atrial conduction system. Because there is a concentration merging atrial conduction tracts in the immediate region of the AV Node near the coronary sinus, * considerable automaticity activity meginates in that area.

The heart's own venous drainage (i.e., from the myocardium) empties into the right atrium the coronary sinus.



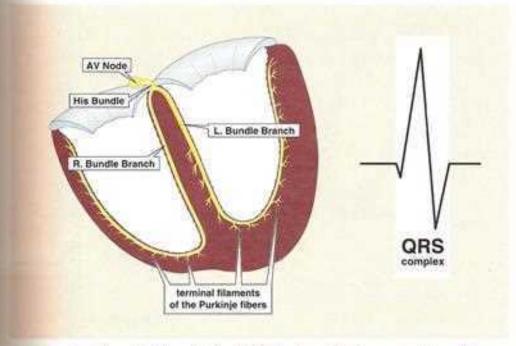
When the depolarization stimulus (passing down from the atria) reaches the AV Node, the stimulus s I o w s in the AV Node, producing a pause on EKG.

District

Atrial depolarization eventually reaches the AV Node, but conduction of depolarization slows within the AV Node, recording a on EKG.

This pause (during which blood from the atria passes into the ventricles) is represented by the horizontal piece of baseline between the P wave and the _____ complex.

Note: The AV Node is named for its position between the Atria and the Ventricles (thus "AV"). The proximal end of the AV Node has no automaticity foci. However, the remainder of the AV Node, an area known as the AV Junction, does have automaticity foci. These foci are essential for backup pacing should there be a total failure of all pacemaking activity from above (SA Node as well as atrial foci), of (this is important) if a complete conduction block of the proximal end of the AV Node occurs, preventing all (SA Node or atrial foci) pacing stimuli from being conducted to the ventricles.



After passing (s I o w I y) through the AV Node, depolarization proceeds rapidly mough the His Bundle, Bundle Branches and their subdivisions, and through the minal Purkinje filaments to distribute depolarization to the ventricles. Ventricular epolarization produces a QRS complex on EKG.

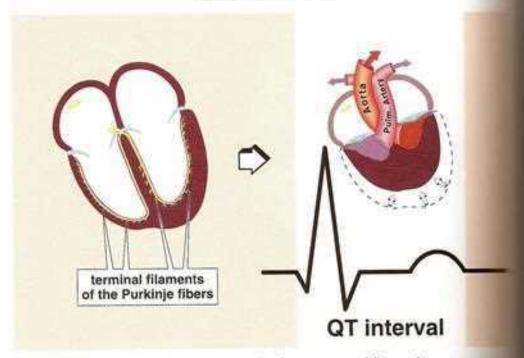
Note: The His Bundle and the Bundle Branches are "bundles" of apidly conducting Purkinje fibers. Depolarization passing through the Purkinje fibers of the ventricular conduction system is too weak precord on EKG; this is a form of "concealed" conduction.

The second secon	ing through the AV Node, depolarizated races through the His, and	N771 1 100 1
andivisions to	Right and Left Bundle Branches and the rapidly transmit depolarization via the nje filaments to the <i>endocardial</i> surface	ir
of the	myocardium.	ventricular

Sen the ventricular myocardium depolarizes, produces a _____ complex on EKG.

QRS

Note: The Purkinje fibers of the ventricular conduction system contain automaticity foci (you knew that already).



The Purkinje fibers of the ventricular conduction system rapidly conduct depolarization away from the AV Node to the endocardial surface of the ventricles when the ventricles depolarize, it produces a QRS complex on EKG.

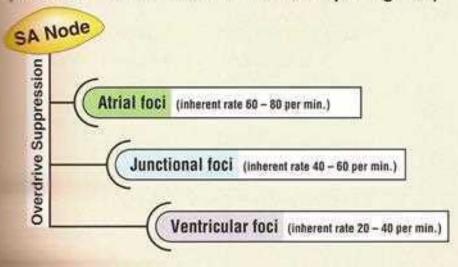
Note: Ventricular depolarization begins midway down the interventricular septum, where the Left Bundle Branch produces fine terminal filaments. The Right Bundle Branch does not produce terminal filaments in the septum. So left-to-right depolarization of the septum occurs immediately before the rest of the ventricular myocardium depolarizes. (Examine the illustration.)

Ventricular depolarization initiates ventricular contraction, which persists (through both phases of repolarization) to the end of the __ wave.

Ventricular contraction begins and ends during the ____ interval.

Note: Repolarization of the Purkinje fibers takes longer than ventricular repolarization. That is, the end of the T wave marks the end of ventricular repolarization; however, repolarization of the Purkinje fibers terminates a little later — beyond the end of the T wave. The final phase of Purkinje repolarization may record a small hump, the U wave (following the T wave), on EKG.

SA Node overdrive-suppresses all foci (since all foci have a slower inherent pacing rate)



three levels of automaticity foci (atrial, Junctional, and ventricular) that can backup pacemaker responsibility if pacing activity fails. The foci of each characteristic inherent rate range, giving the SA Node a failsafe three levels of backup pacing.

ed of automatic	city foci	has a consi	stent range
rate.			

inherent

The SA Node and all automaticity foci are centers of maticity ("automaticity centers"), which means that they generate regular pacing stimuli.

mendrive suppression allows the automaticity center with the pacemaker (no competition).

dominant

the highest pacemaking center fail, an automaticity focus
the next highest level (no longer overdrive-suppressed)
the next highest pacemaking overdrive-suppressing
the next highest level (no longer overdrive-suppressing)
the next highest level (no longer overdrive-suppressing)
the next highest level (no longer overdrive-suppressing)
the next highest level (no longer overdrive-suppressed)

foci

A very "irritable" automaticity focus may suddenly pace rapidly.

Arrhythmias

• Ir	regular	Rhythms	(page 107)
------	---------	---------	------------

• Esca	pe	(page 112)
	Po	(proget itself

The arrhythmias can be divided into a few general categories, according to the arrhythmia's mechanism of origin. The best students, I've noticed, apply index tabs to the pages that begin each arrhythmia category (see above); try it – you will find it very helpful!

Note: Although arrhythmia literally means "without rhythm," generally it describes any rhythm disturbance, that is, any variance from a Normal Sinus Rhythm. Some authors prefer the term "dysrhythmia" rather than arrhythmia.

Note: The illustration is a simplified arrhythmia classification that is categorized according to the mechanism of origin, so the arrhythmias will be easy for you to understand.

Note: The underlying mechanisms that are basic to the heart's function are very satisfying to learn. But more importantly, conceptual understanding of the basic mechanisms facilitates and perpetuates your knowledge. Don't memorize patterns; your knowledge will be vital to others! <u>Lasting knowledge results from understanding</u>.

Irregular Rhythms

- Wandering Pacemaker
- Multifocal Atrial Tachycardia
- Atrial Fibrillation

The irregular rhythms presented in this section are usually caused by multiple, active automaticity sites.

that lack a constant duration between cycles are said to be _____.

irregular

The term "irregularly irregular" is an old designation that a cribes an irregular and chaotic rhythm that has no predictable arring pattern.

In some hearts with structural pathology or hypoxia,

functioning automaticity foci may suffer from entrance block,

ereby any incoming depolarization is blocked, "protecting" them

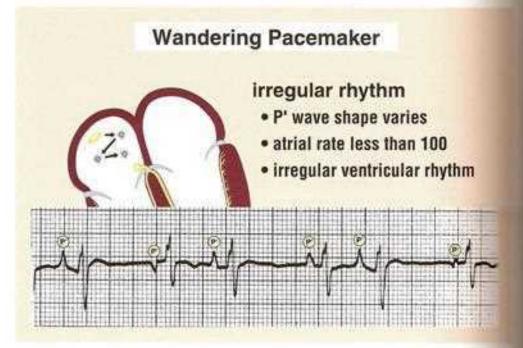
passive depolarization by any other source. Such "protection" is

healthy. By being insensitive to passive depolarization, they cannot

werdrive-suppressed, while their own automaticity is still conducted

strounding tissue. When an automaticity focus has entrance block,

said to be parasystolic (the focus paces, but can't be overdrive
messed).



Wandering Pacemaker is an irregular rhythm produced by the pacemaker activity wandering from the SA Node to nearby atrial automaticity foci. This produces cycle length variation as well as variation in the shape of the P' waves. The overall rate, however, is within the normal range.

Note: The P' (pronounced "P prime") wave represents atrial depolarization by an automaticity focus, as opposed to normal Sinus-paced P waves.

Note: Each automaticity focus has a specific inherent rate at which it paces. In a given lead, each atrial automaticity focus produces its own morphological signature, that is, it produces a P' wave of a distinctive shape related to the anatomical location of that focus within the atria.

Wandering Pacemaker is an irregular rhythm (normal rate range); the pacemaking activity wanders from the SA Node to ______ foci...

... so the cycle lengths vary, and __ wave morphology (shape) varies as the pacemaking center moves.

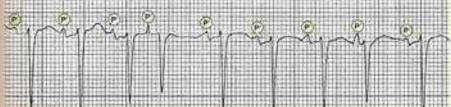
Note: Should the rate accelerate into a tachycardia (greater than 100 per minute), it becomes Multifocal Atrial Tachycardia. Next page...

Multifocal Atrial Tachycardia



irregular rhythm

- · P' wave shape varies
- atrial rate exceeds 100
- irregular ventricular rhythm



Multifocal Atrial Tachycardia (MAT) is a rhythm of patients with Chronic Estructive Pulmonary Disease (COPD). The heart rate is over 100 per minute with P' waves of various shapes, since three or more atrial foci are involved.

MAT, we can recognize each P' wave from a particular focus by its morphological signature, i.e., P' waves the same focus look the same in a given lead.

atrial

MAT is an arrhythmia of patients who are very ill* with COPD.

The atrial automaticity foci are also ill, showing early signs of

arrasystole (entrance block) by developing a resistance to overdrive

appression. That is why no single focus achieves pacemaking

minance, so they all pace together.

Because of the multifocal origin of MAT, each individual atrial
locus paces at its own inherent _____, but the total, combined
locus paces at its own inherent _____, but the total, combined
locus of multiple unsuppressed foci produces a rapid,
locus are represented by the combined of t

rate

and in a given lead, each focus produces P' waves with a specific perphological signature, i.e., __ waves of a distinct shape (note that some P' waves are identical, since they're from the same focus).

In the product of the same focus of the sam

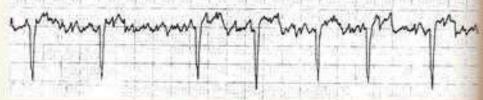
P

^{*}MAT is sometimes associated with digitalis toxicity in patients with heart disease.

Atrial Fibrillation

irregular rhythm

- continuous chaotic atrial spikes
- · irregular ventricular rhythm



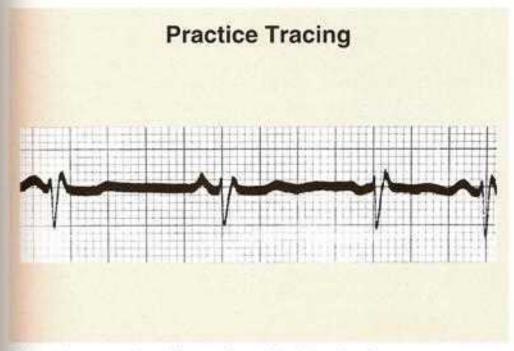
Atrial Fibrillation is caused by the continuous rapid-firing of multiple atrial automaticity foci. No single impulse depolarizes the atria completely, and only an occasional, random atrial depolarization reaches the AV Node to be conducted to the ventricles; this produces an irregular ventricular (QRS) rhythm.

Note: Atrial Fibrillation is NOT an arrhythmia of healthy, young individuals. It is the result of multiple "irritable" atrial foci, suffering from entrance block, pacing rapidly. These multiple atrial foci are parasystolic, so they're all insensitive to overdrive suppression; therefore, they all pace at once. What chaos!

During Atrial Fibrillation, no single impulse completely depolarizes both ______, so there are no P waves, just a rapid series of tiny, erratic spikes on EKG.

Only the occasional atrial impulse gets through the AV Node to initiate a ____ complex. The irregular ventricular response may result in either a slow or rapid ventricular rate, but it is always irregular.

Note: You must determine and document the general ventricular rate in Atrial Fibrillation (QRS's per six second strip times 10).



This tracing was monitored from a patient with an irregular pulse.

This practice tracing	has an irregular	rhythm in	which
we see discernible _	waves, so we	know that	it is not
Atrial Fibrillation.			

P

The "P" waves are not identical, and the rate does not gradually increase and gradually decrease, so we immediately know that this is not ______ Arrhythmia.

Sinus

The rate is less than 100 (which rules out MAT), the thythm is irregular, and the P' waves are of different shapes. This is most likely Pacemaker.

Wandering

Easy, isn't it!

Note: Just to solidify your knowledge of these irregular rhythms, study the simplified review and tracings of *Irregular Rhythms* on page 336.

Escape

Escape Rhythm - an automaticity focus escapes overdrive suppression to pace at its inherent rate:

- Atrial Escape Rhythm
- · Junctional Escape Rhythm
- · Ventricular Escape Rhythm

Escape Beat - an automaticity focus transiently escapes overdrive suppression to emit one beat:

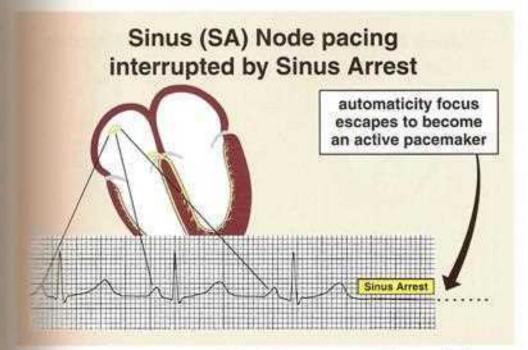
- Atrial Escape Beat
- Junctional Escape Beat
- Ventricular Escape Beat

"Escape" describes the response of an automaticity focus to a pause in the pacemaking activity.

The SA Node's regular	pacing overdrive-suppresses all
automaticity foci, but a	brief pause in SA Node pacing permits
an automaticity	to escape overdrive suppression.
The state of the s	

If SA Node pacing ceases entirely, an automaticity focus will escape to pace at its inherent _____, thereby producing an *Escape Rhythm*. We will, however, need to identify the focus (atrial, Junctional, or ventricular) that escapes to actively pace.

If the pause in pacing is brief (only one cycle missed), an automaticity focus may ______ to emit a single Escape Beat before the normal Sinus rhythm returns. So, we will need to identify that focus (atrial, Junctional, or ventricular).



Saus Arrest occurs when a very sick SA Node ceases pacemaking completely.

The heart's efficient, failsafe mechanism provides three separate levels of maticity foci for backup pacemaking. Divine Design.

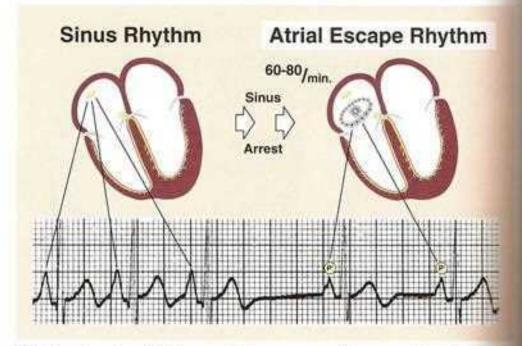
With Sinus Arrest, the SA Node ceases pacing; then, absent endrive suppression by the SA Node, an automaticity focus (with the stest inherent pacing rate) escapes to become an active pacemaker.

In since it has the fastest inherent rate, it overdrive-suppresses all below, to become the dominant pacemaker.

An automaticity focus is overdrive-suppressed if it is regularly colorized by a pacing rate faster than its own inherent pacing rate. If an automaticity focus is not overdrive-suppressed – regardless the cause – it escapes to initiate its own pacemaking activity.

Each specific focus has its own individual, inherent rate of being. However, the inherent pacing rates of all foci of a given level for example, the inherent rates of all Junctional foci) are within a rate

With a Sinus Arrest, the SA Node ceases pacing, so absent serdrive suppression from above, an automaticity focus escapes to moduce an Escape Rhythm. However with Sinus Block, the SA Node assess one pacing cycle, producing only a transient pause. So an automaticity focus escapes to emit an Escape Beat, which actually presents the first beat of the attempt by the focus to pace, but the sum of SA Node pacing overdrive-suppresses it again.



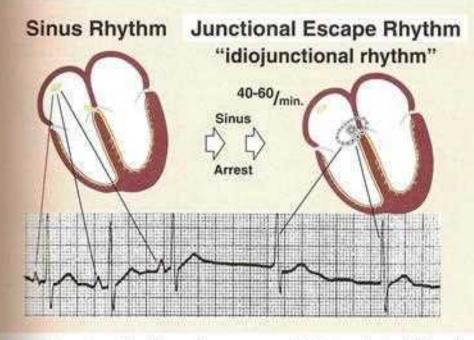
With Sinus Arrest an atrial focus quickly escapes overdrive suppression to become the dominant pacemaker at its inherent rate. This is an Atrial Escape Rhythm.

With a Sinus Arrest, an automaticity focus in the highest level of foci, the _____, escapes overdrive suppression to become an active pacemaker in its inherent rate range of 60 to 80 per minute.

Note: The active atrial automaticity focus overdrive-suppresses all lower, slower foci to become the dominant pacemaker. It also paces at its inherent rate, which differs from (i.e., is slower than) the previous Sinus rate. (See illustration.)

When an atrial focus assumes pacing responsibility in the absence of a Sinus Rhythm, this is an Atrial ______ Rhythm.

SAN



escape overdrive suppression to become an active pacemaker producing a secape Rhythm in its inherent rate range: 40 to 60 per minute.

A Junctional focus escapes the influence of overdrive pression if there is a Sinus Arrest, and the atrial foci also function properly...

w if there is a complete conduction block in the proximal end the AV Node. In either case, the Junctional focus is not regularly lated by pacing depolarizations from above.

producing a Junctional Escape Rhythm, and it becomes
minant pacemaker of the ventricles at a rate ranging from
per minute (it's also called an "idiojunctional rhythm").*

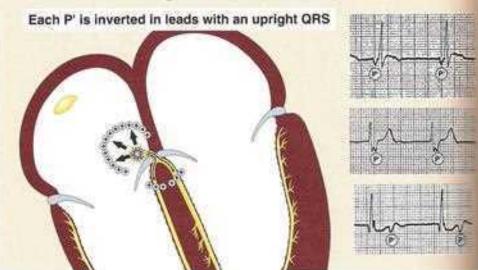
60

memorial Escape Rhythm usually conducts mainly to memorial escape a series of lone ____ complexes. the next page for an interesting exception.

ORS

Section of the inherent Junctional pacing rate may accelerate beyond its usual range to the same an Accelerated Idiojunctional Rhythm.

A Junctional Automaticity Focus May Cause Retrograde Atrial Depolarization



Because each Junctional automaticity focus is located within the AV Node, each pacing stimulus originating there will conduct to the ventricles as expected, but the paced stimuli may also (unexpectedly) depolarize the atria from below ("retrograde"), producing inverted P' waves in EKG leads with an upright QRS.

Note: The illustration shows that atrial depolarization and ventricular depolarization proceed in opposite directions from a pacing Junctional focus. Also, most EKG leads produce an upright QRS.

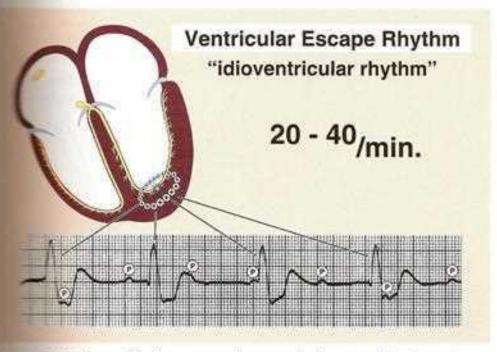
With a Junctional Escape Rhythm, every paced stimulus will depolarize the ventricles, but the pacing may also depolarize the atria from below in a retrograde fashion, producing P' waves in EKG leads with an upright QRS.

Note: The AV Node conducts very slowly, so depolarization from a Junctional focus may delay <u>either</u> ventricular depolarization or retrograde atrial depolarization (if present)...

... as a result, if there is retrograde atrial depolarization from a Junctional focus, it may record on EKG with one of these three patterns:

- · retrograde (inverted) P wave immediately before each QRS
- · retrograde (inverted) P' wave after each QRS
- retrograde (inverted) P' wave buried within each QRS (not shown)

1058



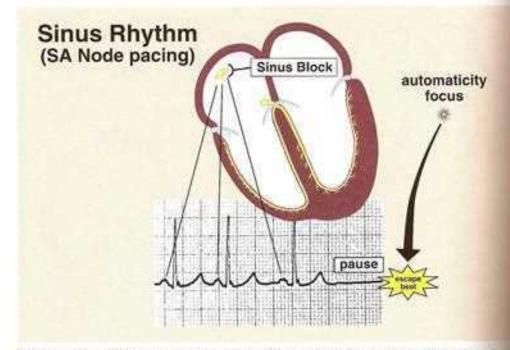
stimulated by paced depolarizations from above, so it escapes overdrive to emerge as the ventricular pacemaker with an inherent rate in the range 40 per minute* (so it is also called an "idioventricular rhythm"). Notice the ventricular complexes.

Ventricular Escape Rhythm usually results from one of two

complete conduction block high in the ventricular conduction system
below the AV Node), the ventricular foci are not stimulated by atrial
conductions from above (see P waves in illustration), so a ventricular
escapes to pace the ventricles at its inherent rate.

failure of the SA Node and all automaticity foci above the ventricles a rare and grave condition called "downward displacement of the secondary". In extremis, a ventricular focus escapes to become the ventricular pacemaker in a final, futile attempt to sustain life.

Pacing from a ventricular focus is often so slow that blood flow brain is significantly reduced to the point of unconsciousness pe). This is Stokes-Adams Syndrome. This unconscious airway must be monitored and maintained... constantly.



During a Sinus Rhythm, a transient Sinus Block makes the SA Node miss a pacing stimulus (one missed cycle), producing a pause in pacing. So an atrial automaticing focus escapes overdrive suppression to emit an Escape Beat.

OVE

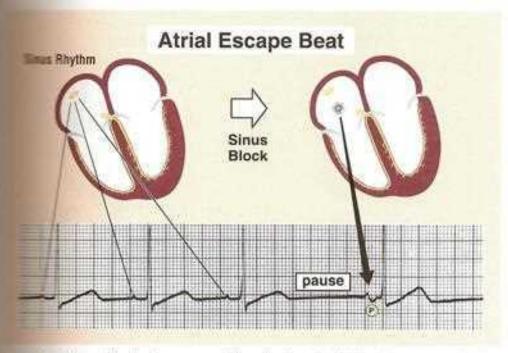
With a transient Sinus Block, an unhealthy SA Node misses one pacing stimulus. This missed cycle produces a ______ during which the heart is electrically silent.

If this pause is long enough (see NOTE below), then an automaticity focus will "escape" ______ suppression.

Note: If there is a "sufficient" pause — longer than the inherent (pacing) cycle length of an automaticity focus — that focus will "escape" the SA Node's overdrive suppression to emit a stimulus.*

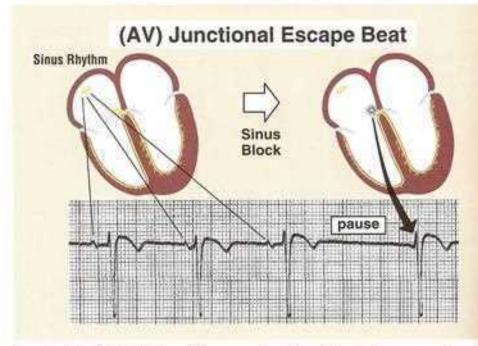
If the SA Node misses only one cycle, it will then resume pacing, and the SA Node's overdrive suppression of all automaticity ______ resumes as well.

^{*} If you don't understand the Note, don't worry. Just be aware of the escape mechanism



Sent Sinus Block of one pacemaking stimulus (SA Node misses one cycle) is a sent pause for an atrial automaticity focus to escape overdrive suppression and Atrial Escape Beat. Notice that the P wave differs from the Sinus-generated

ring one pacemaking stimulus, thus producing a electrical silence for one pacing cycle.	SA Node
cont enough to remove the overdrive suppression arial automaticity and	focus
Escape Beat (on EKG, a pause followed by a P' that from the P waves). Then the SA Node quickly resumes so the atrial focus issuppressed again.	overdrive



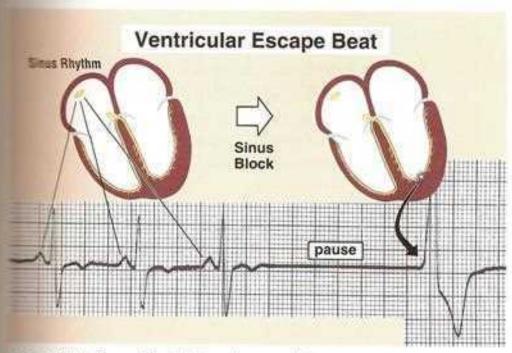
An unhealthy SA Node that suffers a transient Sinus Block misses one pacing cycle, This pause can induce a Junctional automaticity focus to escape overdrive suppression and emit a Junctional Escape Beat.

ults and	pa
	ippressin
14	nal automaticity

88

The depolarization stimulus emitted by the Junctional focus passes down the ventricular conduction _______ to depolarize the ventricles in a normal fashion, so a normal QRS complex results. Then the SA Node resumes pacemaking, overdrive-suppressing the Junctional focus,

Note: A single Junctional Escape Beat may produce retrograde atrial depolarization that records an inverted P' immediately before the QRS or an inverted P' after the QRS.



Ventricular Escape Beat originates in a ventricular automaticity focus that is no per overdrive-suppressed by regular pacing stimuli from above. A ventricular typically produces this enormous ventricular (QRS) complex.

focus

be atrial foci and all the Junctional ____ would fail foci

and so rare? Here's how...

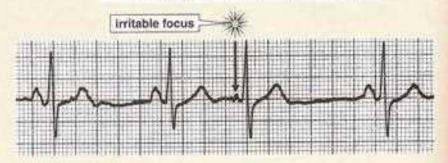
Cardiac parasympathetic innervation inhibits the SA Node and also bibits the atrial and Junctional foci (see illustration, page 58), but not the extracular foci. Therefore, a burst of excessive parasympathetic activity expresses the SA Node (producing a pause) and also depresses the atrial and Junctional foci, which leaves only the ventricular foci to respond to the see. So a ventricular automaticity focus escapes overdrive suppression and discharges, depolarizing the ventricles, producing an enormous entricular complex. Such a burst of excessive parasympathetic activity susually transient, so the SA Node resumes its pacemaking activity.

Sole: Please study the organized review of Escape on page 337, with a focus on understanding.

Premature Beats

Premature Beat - an irritable focus spontaneously fires a single stimulus:

- Premature Atrial Beat
- Premature Junctional Beat
- Premature Ventricular Beat



A premature beat (premature stimulus) originates in an <u>irritable</u> automaticity focus that fires spontaneously, producing a beat (on EKG we see evidence of a depolarization) earlier than expected in the rhythm.

Note: Those things that make you irritable can do the same to an atrial or Junctional automaticity focus. Quickly peek at the next page and you'll see.

A premature	beat,	like a	premature	baby, appears	earlier
than					

expect

When we see a premature beat, we recognize that it was fired by an irritable automaticity _____, so we need to identify the focus (atrial, Junctional, or ventricular).

18000

Note: Ventricular automaticity foci are the world's most sensitive O₃ sensors. When they sense low O₃, they become irritable... and they react!

Note: Premature beats can cause peculiarities in the rhythm that may mimic more serious problems such as pathological conduction blocks. While some premature beats are not serious, others are a dire warning – we'll explore them all. You should be cautious and know the difference – lives will depend on it! *Understanding* the basics provides answers, and understanding facilitates rapid judgement.

Atrial and Junctional foci become irritable because of:

- adrenaline (epinephrine) released by adrenal glands
- increased sympathetic stimulation*
- presence of caffeine, amphetamines, cocaine, or other β₁ receptor stimulants
- excess digitalis, some toxins, occasionally ethanol
- hyperthyroidism
 (direct stimulation plus heart oversensitive to adrenergic stimulants)
 atrial focus
- stretch
- ... and to some extent, low O2

decreasing or blocking parasympathetic effects may accomplish this.

Junctional focus

accompanient focus in the atria or in the AV Junction may become irritable contaneously fire an impulse or even suddenly pace very fast. The cause of a strial and Junctional foci is usually adrenergic substances (page 57).

an atrial or Junctional automaticity focus become irritable,*

fire a spontaneous impulse that depolarizes the surrounding

so we can recognize it on _____ as a premature beat.

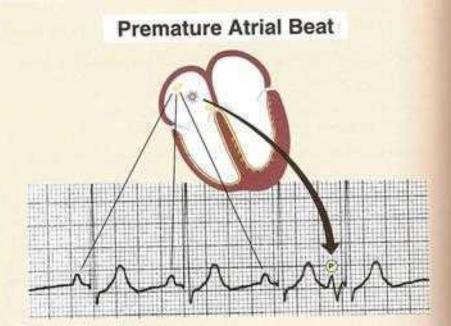
EKG

atrial or Junctional focus may fire
of rapid pacing impulses to become the dominant
ker, overdrive-suppressing all automaticity centers.

irritable

- Conditions/substances that can make an atrial focus (usually) or lanctional focus (occasionally) irritable:
- excess of epinephrine or norepinephrine, the natural substances that activate the adrenergic receptors (of foci).
- adrenergic chemicals that mimic this effect.
- substances or conditions that increase the release of epinephrine
 norepinephrine.
- much coffee), you will remember that upper level foci can also become "irritable"

 and causes) and spontaneously fire a stimulus.



A Premature Atrial Beat (PAB) originates suddenly in an *irritable* (see previous page) atrial automaticity focus, and it produces a P wave earlier than expected. On EKG, P is atrial depolarization by an automaticity focus.

A Premature Atrial Beat (PAB) originates in an irritable atrial automaticity focus that spontaneously fires a depolarization stimulus earlier than the normal ____ wave on EKG.

But because an atrial focus is the origin of this premature atrial depolarization (not the SA Node), the stimulus produces a premature and unusually shaped P' wave* that does not look like a normal (Sinus-generated) P______.

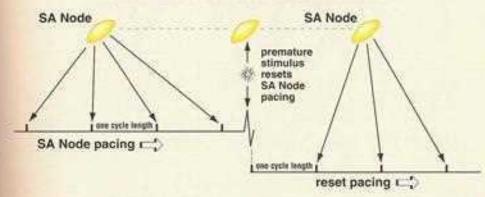
Note: On EKG, a PAB records as a P'. The P' may be difficult to detect when it's hiding on the peak of a T wave; the giveaway is a too-tall T... taller than the other T waves in the same lead.

Note: Each PAB depolarizes the SA Node; the effect of this ... (next page)

^{*} Atrial depolarization from a focus near the SA Node produces a generally upright P was whereas a focus in the lower atrium depolarizes the atria in a "bottom-upwards" (refregation to record an inverted P wave in most leads.

premature

An active automaticity center "resets" to continue pacing one cycle length from a Premature Stimulus



Pacing resets to begin in step with* the premature stimulus

*one cycle length after

All centers of automaticity reset, a characteristic of automaticity. A center of automaticity resets its rhythm when it is depolarized by a premature stimulus, so its pacemaking activity resets in step with the premature beat. Observing the illustration from left to right, helps clarify the concept.

SA Node) is depolarized by a	premature
pacemaking activity resets in step lus, so that the next pacing stimulus cycle length from the premature	bear (stimulus
SA Node is depolarized by a prema	

In order to reset, the dominant (active) center of automaticity

be depolarized by the premature beat. When there is a premature

bulus that does not reach the dominant pacing center, its pacing is

reset.

stimulus.

from the

Sinus Rhythm resets Sinus Rhythm resets Sinus Rhythm (riuset) Sa Node resets in step with Premature Atrial Beat

A Premature Atrial Beat from an irritable atrial automaticity focus produces a too-early depolarization of the atria that depolarizes the SA Node as well. So the SA Node resets its rhythm in step with the Premature Atrial Beat (P').

Note: The P' on EKG is the funny-looking atrial depolarization wave produced by an automaticity focus. It appears different from all SA Node-generated P waves in the same EKG lead, but a normal QRS follows.

If a Regular Sinus Rhythm produced by the SA _____ is interrupted by a spontaneous Premature Atrial Beat (from an atrial automaticity focus), the SA Node, which lies within the atria, is depolarized as well so...

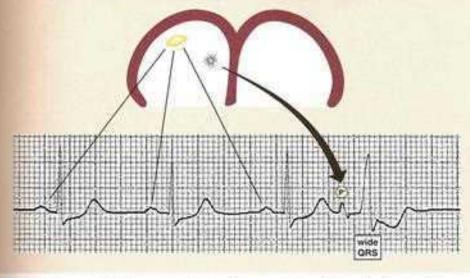
rh

... the SA Node resets, making the P' the first beat of its (reset)
_____. The "?" in the illustration marks where the P wave
would have occurred, if the SA Node weren't reset.

Note: The reset thythm of the SA Node resumes the same rate (same cycle length) as before the premature stimulus, but it continues one cycle length from (i.e., in step with) the P. The pacing rate of the SA Node before and after the PAB remains the same.

Note: In reality, the first cycle after a PAB is a little lengthened due to a transient (baroreceptor) parasympathetic effect on the SA Node, which resumes pacing during systole. (Understanding the mechanism is not important.)

Premature Atrial Beat with aberrant ventricular conduction

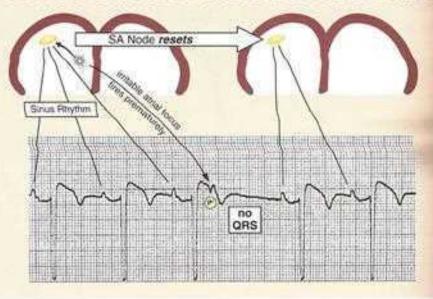


The ventricular conduction system is usually receptive to being depolarized by a permature Atrial Beat, but one Bundle Branch may not have completely repolarized that is, it's still a little refractory) when the other is receptive. This "aberrant sentricular conduction" produces a slightly widened QRS for that premature cycle may.

When a Premature Atrial Beat (P') is conducted to the sentricles, the ventricles are also depolarized earlier than usual.

metimes a Premature Atrial Beat can produce aberrant entricular conduction, because one of the Bundle Branches completely, and therefore is temporarily effectory to depolarization.	repolarized
depolarization of one ventricle is immediate, while polarization of the other ventricle is slightly	delayed
he non-simultaneous depolarization of the ventricles records a slightlyQRS complex after the P on the EKG.	widened

Non-conducted Premature Atrial Beat



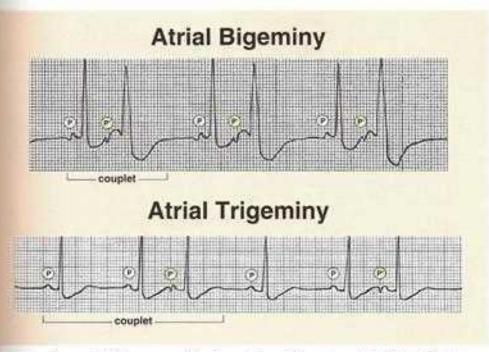
At times, the AV Node is completely unreceptive to a premature atrial depolarization stimulus because it reaches the AV Node prematurely, that is, while the AV Node is still in the refractory period of its repolarization. This results in a "non-conducted" (to the ventricles) Premature Atrial Beat.

A Premature Atrial Beat may be unable to depolarize the AV Node if it (the AV Node) is not fully repolarized and still to an extra-stimulus.

refrac

On EKG, this records as a too-early, unusual ____ wave that has no ventricular (QRS-T) response.

Note: Warning! Although a "non-conducted" PAB (on EKG, a premature P' without a QRS response) does not depolarize the ventricles, it does depolarize the SA Node, which resets its pacemaking one cycle length after the premature stimulus. The combination of reset pacing plus the missing QRS-T creates a harmless, but dangerous-looking, span of empty baseline... which has the sinister appearance of a "some-kind-of-block." And one day you will have the satisfaction of correcting someone who guessed the wrong diagnosis.



Occasionally, an irritable automaticity focus fires a Premature Atrial Beat (P') that couples to the end of a normal cycle, and repeats this process by coupling a PAB to be end of each successive normal cycle. This is **Atrial Bigeminy**.

Note: The cycle containing the premature beat together with the cycle ecycles to which it couples, is called a "couplet."

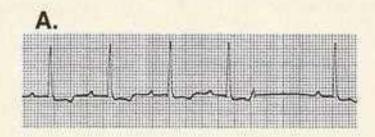
Semetimes, an irritable atrial focus may prematurely fire after
normal cycles; when this couplet _____ continuously.

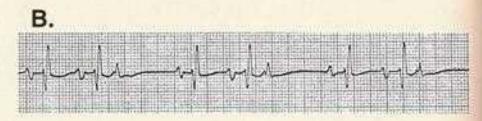
The art of Atrial Trigeminy.

With both Atrial Bigeminy and Atrial Trigeminy, each premature simulus (from the irritable atrial focus) depolarizes the SA Node and resets producing a span of clear baseline between the couplets. So a series run") of couplet groups called "group beating" is often seen with Atrial Bigeminy, Atrial Trigeminy, etc. Just look for the premature (P') beat in each couplet. It's that simple! This is mentioned because group beating may secur with a type of AV conduction block to be discussed later (page 180).

^{*}As you may have noticed, there is a slightly widened (aberrant) QRS after each P' in the apper tracing. Aberrant ventricular conduction can occur after any premature atrial are Junctional) beat.

Practice Tracings





Can you determine what is occurring on each of these practice EKG tracings?

Tracing A:

This tracing is from a medical student who had a few cups of coffee in order to study late. She went to the Emergency Room because her pulse seemed irregular

The intern on duty thought that the tracing showed "intermittent complete AN Block" and was about to call the attending physician (at 4:00 am) to schedule an emergency artificial pacemaker implantation. Explain the EKG strip to the intermusing only what you have learned so far (before he wakes the attending physician and discovers the real meaning of "irritable").

Tracing B:

This transmitted telemetry tracing is from a known drug abuser who took a large quantity of amphetamines before his emergency ride to the hospital.

Someone in the ambulance suggested what sounded like "Winky bok block," when the telemetry was transmitted. Utilizing only what you have read and understand so far in this book, you will recognize things that you have just learned. Notice that in each grouping only two of the P waves are identical. Carefully analyze what you see, so you can explain it to others,

Note: Carefully examine each tracing and contemplate its subtle information. The answers will appear as you continue reading... somewhere.

Premature Junctional Beat

AV Junctional Beat (PJB) occurs when an *irritable* automaticity focus

AV Junction suddenly fires a premature stimulus that is conducted to, and

policies, the ventricles (and sometimes the atria, in retrograde fashion).

an irritable focus (see page 123) in the AV Junction

semaneously fires a stimulus, this produces a Premature

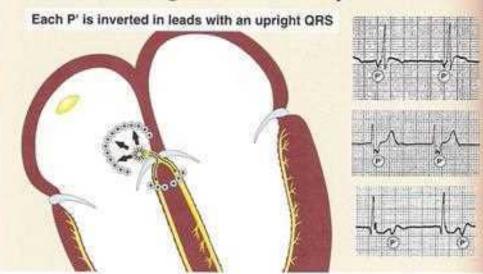
Beat on EKG.

Junctional

After heart tissue depolarizes, it immediately repolarizes, and mag repolarization that tissue is refractory to another stimulus remature stimulus). As the ventricles repolarize, one Bundle Branch repolarize slower than the other. So the too-early depolarization a PJB may conduct through one Bundle Branch, but the impulse temporarily delayed in the other, still refractory, Bundle Branch smally the Right). So, instead of depolarizing simultaneously, one entricle depolarizes punctually and the other is delayed, producing a phtly widened QRS complex typical of a Premature Junctional Beat the aberrant ventricular conduction.

you see a premature QRS complex th	nat is slightly widened,	
should consider that it may be due	to a Premature Junctional	
Premature Atrial) Beat with	ventricular conduction.	aberran

A Junctional Automaticity Focus May Cause Retrograde Atrial Depolarization



A Premature Junctional Beat originates in an irritable Junctional focus within the AV Node. We expect such a premature stimulus to conduct to the ventricles, but it may also depolarize the atria in a bottom-up "retrograde" fashion that records as an inverted P wave in EKG leads with an upright QRS.

Since atrial and ventricular depolarization move in opposite directions from the Junctional focus, the premature P' wave is ________i.e., opposite the upright ORS.

inverted

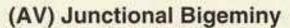
If a PJB produces retrograde atrial depolarization, it may record an inverted P' wave immediately before the premature _____ complex.

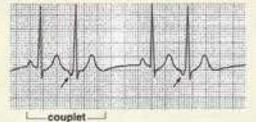
ORS

Sometimes an inverted ___ wave associated with a PJB follows the QRS. Occasionally the P' disappears within the QRS when atrial and ventricular depolarization occur simultaneously (not shown in illustration).

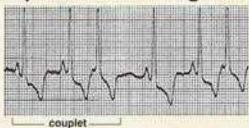
P

Note: Retrograde atrial depolarization from a PJB usually depolarizes the SA Node as well. So the SA Node pacing is *reset* in step with the retrograde *atrial* depolarization.





(AV) Junctional Trigeminy



An irritable focus in the AV Junction may initiate a Premature Junctional Beat after each normal (SA Node-generated) cycle. This is **Junctional Bigeminy**. When a PJB is coupled with two consecutive, normal cycles in a repeating series of these couplets, this is **Junctional Trigeminy**.

maintable Junctional automaticity focus may fire a premature semalus coupled to the end of each normal (SA Node paced)

Bigeminy

irritable Junctional focus may fire a stimulus after two
secutive, normal Sinus-generated cycles. A repeating
eries of these couplets is Trigeminy. Junctional

Note: Don't forget that on EKG you may see an inverted (retrograde)

P wave (arrows in upper tracing) with every PJB in either Junctional

Bigeminy or Junctional Trigeminy. Also, the SA Node will reset its

sacing with each retrograde atrial depolarization; this can produce

alarming (but innocent) gaps of empty baseline between couplets.

A ventricular focus can be made irritable by:

Low O₂

Low K+

Reduced serum potassium ("hypokalemia")

Pathology Mitral Valve Prolapse, stretch, myocarditis, etc.

... and to a lesser degree, epinephrine-like substances (β, adrenergic stimulants).

A ventricular focus may become irritable from under-oxygenation ("hypoxia") due to various circumstances and conditions. Hypokalemia, QT-prolonging medications, mitral valve prolapse, cardiac pathology, and stretch, can do the same.

Poor oxygenation (hypoxia) can make a ventricular automaticity

become irritable and fire a spontaneous impulse,
producing a premature ventricular beat on EKG.

foca

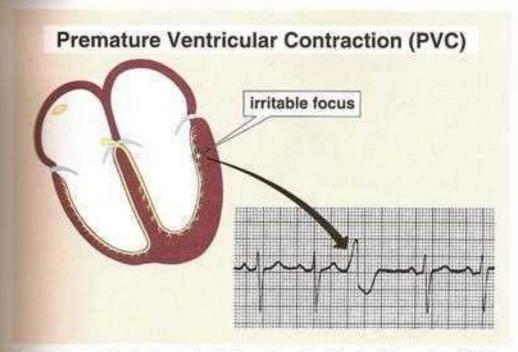
A very irritable ventricular focus may be so excessively provoked by hypoxia or "ischemia" (diminished blood supply) that it suddenly fires a series of rapid impulses, overdrive-suppressing the ______ Sinus rhythm...

normal

... so it becomes the heart's dominant pacemaker.

Note: If you study the illustration for a moment, you will quickly realize that there are numorous mechanisms that can reduce the oxygen supply to these sensitive ventricular automaticity foci. In a clinical setting, most (but not all) "deadly" ventricular tachycardias are due to coronary insufficiency or infarction. Know the other causes of ventricular focal irritability (see illustration).

Note: Cocaine is known to make atrial and Junctional foci irritable, but it has more dangerous effects. Cocaine causes coronary spasm, making ventricular foci hypoxic and very irritable; dangerous ventricular arrhythmias may ensue.



A premature ventricular beat called a **Premature Ventricular Contraction** (PVC) sognates suddenly in an *irritable* ventricular automaticity focus and produces a pant ventricular complex on EKG.

mtable (qui	ckly review the previous page, please)
ericular focus	may suddenly fire a stimulus and produce
200 FBC 01 TO 200 F 10 G	Ventricular Complex* (PVC) on EKG.

Premature

Note: PVC's occur early in the cycle. Easily recognized by their meat width and enormous amplitude (height and depth), they are upward, PVC's are mainly downward).

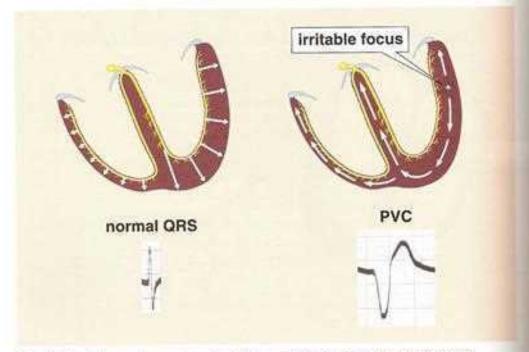
The most likely reason for a ventricula	r automaticity focus
become irritable is under-	(hypoxia).

oxygenation

Note: PVC denotes a ventricular "contraction." When you see a PVC, emember that this represents a (premature) ventricular contraction, and an associated premature pulse beat, albeit weaker than normal the prematurely stimulated ventricles are not completely filled).

^{*}PVC may stand for Premature Ventricular "Contraction" or "Complex."

This issue remains unresolved.

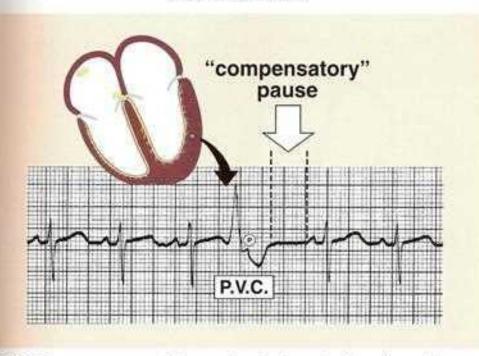


The PVC originates in an automaticity focus within the ventricular conduction system, usually in a ventricular wall. Thus, one area of the ventricular wall begins to depolarize before the rest of the ventricle, long before the other ventricle depolarizes.

Note: After a normal, Sinus-generated depolarization stimulus has passed through the AV Node, the stimulus is quickly transmitted to the entire endocardial lining of both ventricles at once. This simultaneous depolarization of both ventricles produces a slender QRS complex.

Note: When an irritable ventricular automaticity focus suddenly fires an impulse, that ventricular region depolarizes before the rest of the ventricle, and then the depolarization wave creeps to the other ventricle, which then depolarizes... producing an enormously wide ventricular complex.

Note: Normally, ventricular depolarization passes through the entire thickness of both ventricles at once. Left ventricular depolarization in the leftward direction tends to be counterbalanced by the simultaneous right ventricular depolarization in the opposite direction. This minimizes the QRS amplitude. But depolarization originating in a remote ventricular focus (as with a PVC), gradually spreads without simultaneous opposition from the other side, and in its slow course, produces (unopposed) deflections of immense amplitude.



The PVC is an enormous ventricular complex that is much wider, taller, and deeper than a normal QRS. There is a pause after the PVC, but it is not caused by resetting of the SA Node; in fact, sometimes you can see the punctual, but ineffective, P wave within the PVC (see P in the illustration).

Be PVC is a gigantic ventricular complex that jumps out a you from the EKG, warning you that there is a ventricular scus that is irritable, usually because of	hypoxia
PVC's depolarize only the, not the SA Node, the SA Node discharges on schedule. In fact, by measuring P eycles, you can often locate the punctual P wave within a PVC.	ventricles
But that timely P wave occurs while the ventricles are still refractory (from the PVC) and not fully Then this normal stimulus arrives, they can't depolarize	repolarized
so there is a as the ventricles finish repolarizing,	pause*

Note: Interpolated PVC's are rare, but are somehow sandwiched between the beats of a normal rhythm, producing no pause and no shythm disturbance.

^{*}The pause, sometimes called a "compensatory" pause, doesn't "compensate" for anything.

Multiple PVC's from an irritable focus

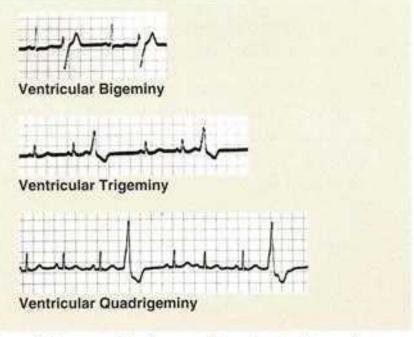
Numerous PVC's may emanate from the same ventricular focus, warning that the focus is very irritable, usually because of its poor state of oxygenation. Six or more PVC's per minute is considered pathological.

While monitoring a given lead, you notice identical PVC's appearing quite often; since each PVC is identical, we know that they all originated in the same irritable focus. (These are "unifocal" PVC's.)

ventric

Frequent unifocal PVC's usually indicate poor oxygenation of a single ventricular focus – often because the blood supply to that focus is diminished. Remember, ______ PVC's per minute is pathological. Don't ignore this patient!

Note: There are situations when the coronary blood flow is adequate, but the blood is poorly oxygenated (e.g., drowning, pneumothorax, pulmonary embolus, tracheal obstruction, etc.). When a highly irritable ventricular focus is warning you with multiple PVC's... you must respond! Low serum potassium, as well as certain medications, can also irritate a ventricular focus. In addition, adrenergic stimulants like epinephrine can aggravate the situation.



A very irritable ventricular automaticity focus may fire a stimulus that couples to one more normal cycles to produce Ventricular Bigeminy, or Ventricular Trigeminy, etc.

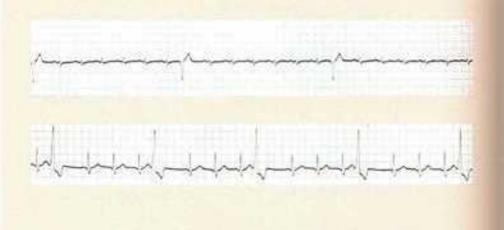
Note: By convention, 6 PVC's per minute is considered pathological.

A continuous run of any of these couplet patterns quickly exceeds that criterion and usually indicates that a very irritable focus is hypoxic and calling for help.

the pattern continues with every cycle in succession, adentify this as Ventricular	Bigeminy
See a repetitive pattern of a PVC coupling with every sormal cycles, this is a run of Ventricular	Trigeminy

Ventricular automaticity foci are the heart's hypoxia early saming system. Respond!

Ventricular Parasystole



Ventricular Parasystole is produced by a ventricular automaticity focus that suffers from entrance block (but is not irritable). The parasystolic focus is not vulnerable to overdrive suppression, so it paces at its inherent rate, and the ventricular complexes that it generates poke through the dominant Sinus Rhythm.

Note: A solitary ventricular focus suffering from entrance block is "parasystolic", that is, it can't be overdrive-suppressed yet it can deliver pacing stimuli at its inherent rate.

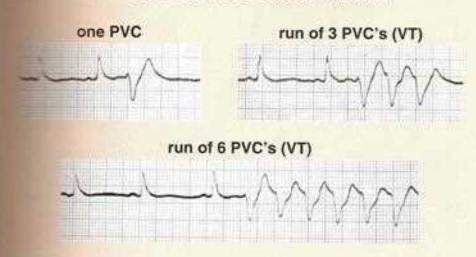
Note: A parasystolic ventricular focus suffers from entrance block, insulating it from depolarization by outside sources. Absent overdrive suppression, it paces at its inherent rate. The result is a dual rhythm with pacing from two sources, the SA Node and the ventricular focus.

When you see PVC's that appear to be coupled to a long series of normal cycles, you should suspect Ventricular ______,

Parasysto ...

Note: Because this represents two unrelated, independent rhythms (from two different pacing locations), the interval between the normal cycle and the large ventricular complex is not always consistent. Occasionally a large ventricular complex may fail to appear because the ventricular focus happens to discharge during the refractory period of the (Sinus-paced) ventricles.

A very irritable ventricular focus can emit consecutive stimuli



eventricular automaticity focus may fire once or, if extremely irritable extremely, it may fire a rapid series of impulses to produce a run of PVC's.

oxygen supply decreases further, the focus may be provoked series of discharges in ______ succession.

rapid

are more serious than occasional PVC's same focus, particularly in patients suffering an myocardial infarction.

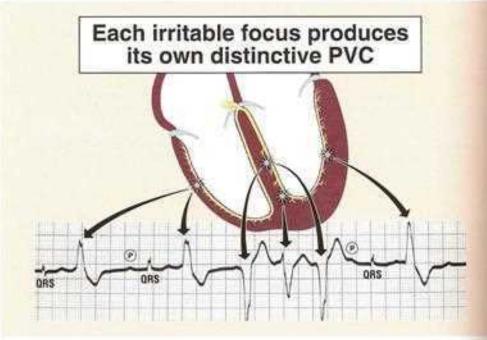
focus

A run of three or more PVC's in rapid succession is really a run

Ventricular Tachycardia (VT), Two of the examples in the above

Bustration are VT. If a run of VT lasts longer than 30 seconds, it is

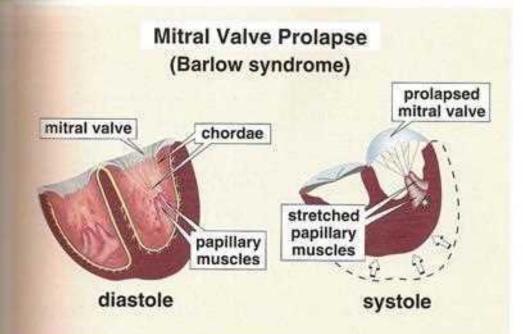
Elled "sustained" VT.



Severe cardiac hypoxia can cause Multifocal PVC's – a desperation measure produced by multiple, exceptionally irritable (hypoxic), ventricular foci. Each focus produces its own unique, identifiable PVC every time it fires.

In a given lead, PVC's originating in a specific ventricular focus all appear the ______.

Note: Severe cardiac hypoxia can cause numerous multifocal PVC's to apear. This is indeed dangerous and requires immediate intervention. Because a single irritable ventricular focus can suddenly fire a series of rapid discharges to produce a dangerous tachy-arrhythmia (e.g., Ventricular Tachycardia), the presence of numerous multifocal PVC's means that a number of extremely irritable foci are discharging, and trouble is imminent. The chance of developing a dangerous or even deadly arrhythmia (e.g., Ventricular Fibrillation) under these dire circumstances is obviously enhanced. With infarction patients, this is an alarm of crisis proportions!



Witral Valve Prolapse (MVP) causes PVC's, including runs of VT and multifocal PVC's, yet it is considered a benign condition. With MVP the mitral valve is "toppy" and billows into the left atrium during ventricular systole.

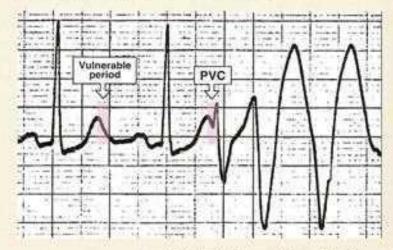
Sole: First described by Dr. J. B. Barlow in 1968 (MVP is also alled "Barlow Syndrome"), this is quite common; 6% to 17% of emales, and about 1.5% of males. Females with MVP typically have beliender body with a slight chest deformity, experience "dizzy" spells, and are anxiety prone. They first experience symptoms after age 20.

wing ventricular systole, the billowing valves pull on the
induce that tether them to the papillary muscles in the left
incle. In the author's opinion, this traction on the papillary
incles causes localized stretch and ischemia, irritating adjacent
includer automaticity _______.

foci

Note: MVP patients usually have a mid-systolic click with a secrescendo murmur on auscultation.

If a PVC falls on a T wave...



watch this patient closely.

If a PVC falls on a T wave ("R on T phenomenon"), particularly in situations of hypoxia or low serum potassium, it occurs during a "vulnerable period" and dangerous arrhythmias may result. Notice how a PVC hits the second T wave directly in its vulnerable period (ouch!)... and see what happens!

PVC's are, of course, premature and usually occur just after the ___ wave of a normal cycle.

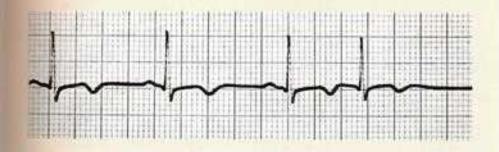
When a PVC falls on the peak of a T wave or on the initial part of its downslope, it catches the ventricles during a vulnerable period, particularly in the presence of _______ (often caused by cardiac ischemia from a narrowed coronary artery) or in the presence of low potassium.

hypo

Note: Repolarization of Purkinje fibers (along with their vulnerable period) extends beyond the T wave, so a PVC falling just after the T wave may, in fact, occur during the vulnerable period of ventricular foci. Ischemia can extend Purkinje repolarization even further.

Note: This well known warning sign, "R on T" is often noted after the fact, during the review of an EKG strip from a patient who suffered a dangerous or deadly arrhythmia. By being cautious and vigilant, you can respond quickly.

Practice Tracing



The discerning eye of a coronary care nurse detected a beat that appeared a little too arriv on the EKG strip taken from a patient's monitor. Let's determine the location of the irritable focus that produced the premature beat.

last QRS complex in the strip catches your eye because securs prematurely, and it is not preceded by a ____ wave,

P

last QRS complex looks the same as the other QRS's, so we know although premature, the last QRS resulted from depolarization passed (in a normal fashion) down the ventricular conduction therefore, it is not from a _______ focus. ventricular

Note: Sure you understand this, but it probably would be a good idea to take a minute to review this section, and study the simplified wisew of *Premature Beats* on page 337. Then, let's take a break, and right here when you return.

Tachy-arrhythmias

rapid rhythms originating in very irritable automaticity foci

s 1	50	350
Paroxysmal Tachycardia	Flutter	Fibrillation
		multiple foci discharging

A "tachy-arrhythmia" originates in a very irritable focus that paces rapidly, Sometimes more than one active focus is generating pacing stimuli at once.

Note: Tachy-arrhythmia ("rapid arrhythmia"), hyphenated for recognition purposes here, is not usually hyphenated, so henceforth the hyphen will be omitted.

The rate ranges of the tachyarrhythmias are:

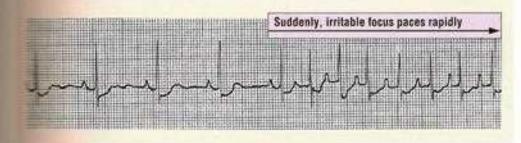
Paroxysmal Tachycardia	to	/minute.	150 to 25
Flutter	to	/minute.	250 to 35
Fibrillation	to	/minute	350 to 45

Note: A tachyarrhythmia is easily recognized by rate alone, but the specific diagnosis requires that we identify the origin, that is, we must determine the location of the irritable automaticity focus (atrial, Junctional, or ventricular). You already have a solid understanding* of normal conduction in the heart, so we merely need to get up to speed (pun intended) in learning the behavior of very irritable automaticity foci, and how they record on EKG.

[&]quot;"Understanding is a kind of ecstacy." Carl Sagan (from Broca's Brain.)

Paroxysmal (sudden) Tachycardia a very irritable automaticity focus suddenly paces rapidly:

- Paroxysmal Atrial Tachycardia
- Paroxysmal Junctional Tachycardia
- Paroxysmal Ventricular Tachycardia



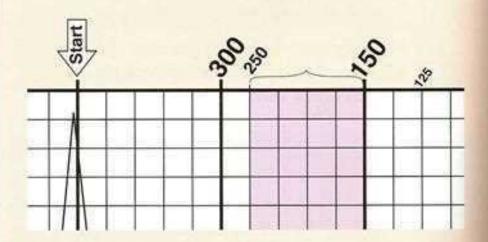
**Torysmal ("sudden") tachycardia ("rapid heart rate") indicates rapid pacing to 250 per minute) by a very irritable automaticity focus. Once we recognize **Torysmal tachycardia, we need only identify the focus (atrial, Junctional, or **Toricular*) of its origin.

medical term for rapid heart rate is	 tachycardia
moxysmal means	sudden

Paroxysmal tachycardia arises <u>suddenly</u> from a very irritable <u>su</u>

contrast, Sinus tachycardia is th	e SA Node's gradual response
exercise, excitement, etc. Altho-	ugh the SA Node's rate of
may eventually become qu	ite rapid, Sinus tachycardia is
mer sudden nor does it originat	e in an automaticity focus,
by definition, it is not a	tachycardia.

Paroxysmal Tachycardia



The rate range of the paroxysmal tachycardias is 150° to 250 per minute, so they are easy to recognize. Locating the causative irritable focus (atrial, Junctional, or ventricular) gives us the diagnosis.

When calculating rate, we find an R wave that peaks on a heavy black "start" line. The next three heavy black lines are called "300, 150, ____."

The fine line immediately to the right	of the heavy black "300" line
is the thin "250" line. Therefore, if an	R wave falls on the "start" line
(see illustration) the next R wave will	fall within the shaded area
during paroxysmal	

tachye

You can instantly recognize a paroxysmal tachycardia by noting the rate range of ______ to 250 per minute. Now you have to determine at which of three levels there is a very irritable automaticity focus causing the tachycardia. Easy enough!

Some authors now set the lower rate limit of paroxysmal tachycardia at 125 per minuse

Paroxysmal Atrial Tachycardia 150 - 250/min. Suddenly, irritable focus paces rapidly

irritable* atrial automaticity focus. You may see the beginning of this amia only occasionally, so become familiar with its general appearance.

win a very irritable focus in one of the _____. The rate usually 150 to 250 per minute, so it overdrive-suppresses

atria

the origin of this tachyarrhythmia is a very irritable locus, the atrial depolarizations of PAT are ____ waves not look like the Sinus-generated P waves.

P

focus depolarizes the atria and then is conducted
the ventricular conduction system to the ventricles,
normal-appearing P'-QRS-T cycles.

atrial

A premature stimulus from another focus may set off PAT.

PAT with (AV) Block



- rapid rate, spiked P' waves
- · 2:1 ratio of P':QRS

Suspect digitalis excess or toxicity.

Paroxysmal Atrial Tachycardia with AV block has more than one P' wave spike for every QRS response. Suspect digitalis excess or toxicity; atrial foci are very sento the irritating effects of digitalis preparations.

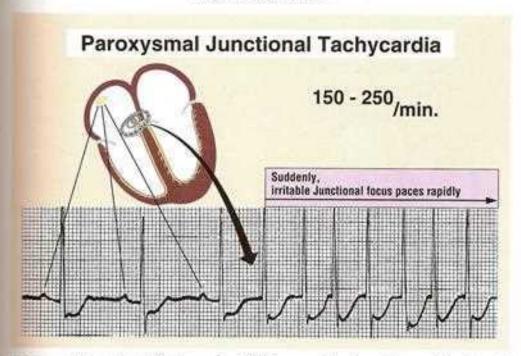
Note: Excess digitalis can provoke an atrial focus into such an irritable state that it suddenly paces rapidly. At the same time, digitalis markedly inhibits the AV Node, so that only every second stimulus conducts to the ventricles (every-other atrial stimulus is blocked in the digitalis-inhibited AV Node).

PAT with block* is a tachyarrhythmia that has two P' waves for each QRS response on EKG, because the ______blocks the conduction of every-other atrial stimulus.

AVN

PAT with block is usually a sign of _____ excess or toxicity, particularly if the patient has a low serum potassium, so careful administration of intravenous potassium can help. Also, digitalis antibodies can be employed to reduce toxicity.

^{*} The "AV" is sometimes omitted, but is always understood.



Paroxysmal Junctional Tachycardia (PJT) is caused by the sudden rapid pacing of a very irritable automaticity focus in the AV Junction. The Junctional focus may suddenly initiate tachycardia pacing, because of marked irritability induced by stimulants and/or by a well-timed premature beat from another focus.

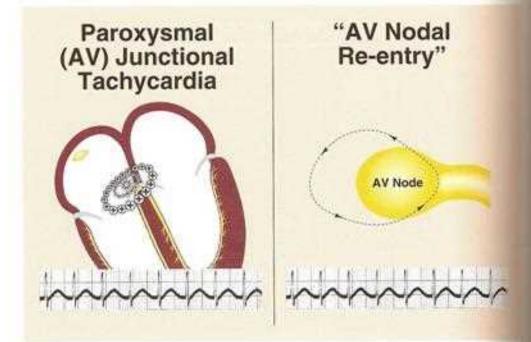
material Tachycardia is due to a very irritable* focus
the AV Junction that paces at the rate of to 250 per minute. 150

Note: A rapidly pacing (irritable) Junctional focus may also sepolarize the atria from below in retrograde fashion to record:

- an inverted P' immediately before each upright QRS, or (see illustration
- an inverted P' after each upright QRS, or page 132)
- · an inverted P' buried within each QRS (difficult to detect).

Note: Each stimulus from a rapidly pacing (irritable) Junctional focus may occur at a time in the cycle when the Left Bundle Branch has fully repolarized (i.e., recovered from its refractory period), but the Right Bundle Branch is still refractory (in some patients, the reverse occurs). As a result, this aberrant ventricular conduction depolarizes the left ventricle before the right, to produce somewhat widened QRS's during the tachycardia.

^{*}One more look at page 123, and I'll never bother you again.



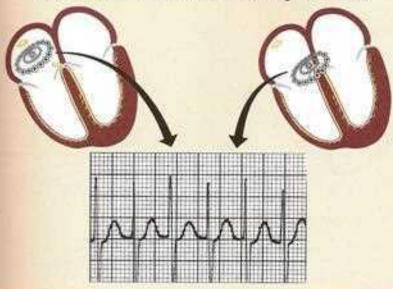
Another type of Junctional Tachycardia is AV Nodal Reentry* Tachycardia (AVNRT). In theory, a continuous reentry circuit develops (which includes the AV Node and the lower atria) and rapidly paces the atria and ventricles.

Note: A theoretical "reentry circuit" may continuously circle (like perpetual motion) through the AV Junctional region, giving off a depolarization stimulus to the atria and to the ventricles with each pass in the circuit. This is "circus reentry," an aptly named tachycardia that looks suspiciously like PJT.

Note: In AVNRT, each pacing stimulus first records from an origin near the coronary sinus — an area loaded with automaticity foci. Although the putative reentry circuit includes a broad area around the AV Node, only catheter ablation of the focus-laden region can successfully eliminate this tachycardia (very suggestive of focal automaticity origin). Dogmatic loyalty to this theoretical reentry model persists. The jury is still out.

Pronounced "ree-EN-tree".

Supraventricular Tachycardia



Sery irritable* automaticity foci that produce both Paroxysmal Atrial
Serial and Paroxysmal Junctional Tachycardia originate above the ventricles,
Serial Serial

entricular tachycardia (the word "paroxysmal" is often

PJT

"supraventricular" imparts the understanding that all atrial

ventricles

Paroxysmal Atrial Tachycardia can be so rapid that the

sees run into the preceding T waves to become indistinguishable.

can make differentiation between PAT and PJT very difficult.

see treatment for both is so similar, the umbrella term

sepentricular Tachycardia (SVT) suffices, and further distinction

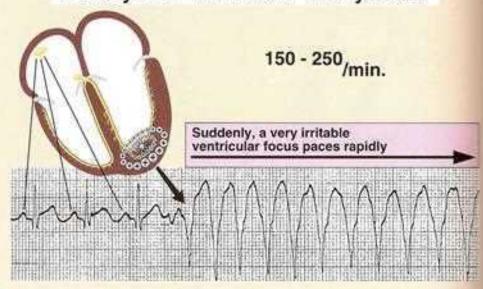
ean the two is unnecessary. Certain conditions may widen the

sin SVT, so it may then resemble Ventricular Tachycardia

page).

an atrial or Junctional focus is made irritable by adrenergic stimulants, but a focus
be further provoked into tachycardia pacing by a premature stimulus from another
be focus.

Paroxysmal Ventricular Tachycardia



Paroxysmal Ventricular Tachycardia (PVT or VT)* is produced by a very irritable ventricular automaticity focus that suddenly paces in the 150 to 250 per minute range. It has a characteristic pattern of enormous, consecutive PVC-like complexes. Please conscientiously review page 134 now.

Paroxysmal Ventricular Tachycardia originates suddenly in a very ______ ventricular automaticity focus, producing a (ventricular) rate of 150 - 250.

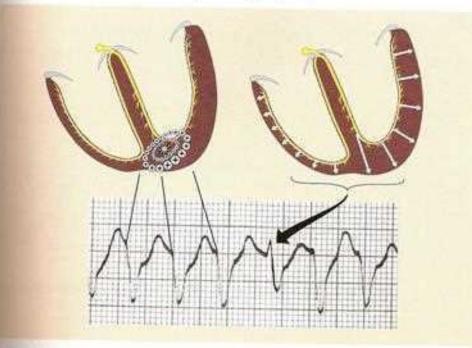
irrit

Sudden runs of Ventricular Tachycardia* resemble a rapid series of ______'s (which in reality, they are).

pus

Note: During Ventricular Tachycardia, the SA Node still paces the atria, but the large, dramatic ventricular complexes hide the individual P waves that can be seen only occasionally. So, there is independent pacing of the atria and the ventricles... a type of AV dissociation.

^{*} The "Paroxysmal" is often left off, so "Ventricular Tachycardia" or "VT" are used commonly.



Ventricular Tachycardia, the SA Node continues to pace the atria (AV sociation), but an occasional atrial depolarization catches the AV Node in a sprive state, and then this depolarization stimulus conducts to the ventricles.

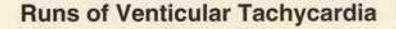
sepolarizations finds the AV _____ receptive to

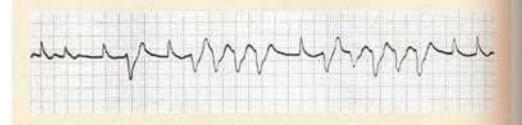
Node

stimulus passes through the AV Node to depolarize

conduction

On occasion during VT, a (Sinus-paced) depolarization stimulus
the atria finds the entire ventricular conduction system receptive
charization and produces a normal-appearing QRS (capture beat)
andst of the ventricular tachycardia. More commonly during VT,
depolarization finds a receptive AV Node, but ventricular
relation only proceeds so far before it meets ventricular
relation progressing from the ventricular focus. This produces a
but, which is a blending on EKG of a normal QRS with a PVCpolex (see illustration). The presence of "captures" or "fusions"
the diagnosis of Ventricular Tachycardia, because they could





Runs of (Paroxysmal) Ventricular Tachycardia may signify coronary insufficiency (ischemia) or other causes of cardiac hypoxia that make a ventricular automaticity focus very irritable.

Ventricular Tachycardia appears like a run of _____'s.

PVC

Paroxysmal Ventricular Tachycardia often indicates coronary _______, causing poor oxygenation of the heart (and ventricular foci). For other causes of cardiac hypoxia see page 134.

insufficience

Note: This rapid ventricular rate suddenly erupts from an irritable (hypoxic) ventricular focus. The rapid rate is too fast for the heart to function effectively, particularly in the elderly with compromised coronaries. It should be treated quickly (but cautiously) in patients with a myocardial infarction.

Caution: A rapid (Junctional or atrial) Supraventricular Tachycardia with aberrant conduction can produce a tachycardia with widened QRS's that mimics VT. Also, pre-existing Bundle Branch Block with SVT will widen the QRS complexes to give the same impression. NEVER give medications for SVT to a patient with VT.

Distinguishing Wide QRS complex SVT from Ventricular Tachycardia

Helpful Clues	Wide QRS Complex SVT	Ventricular Tachycardia
Patient with coronary disease or infarction	uncommon	very common
QRS width (duration)	less than .14 sec.	greater than .14 sec.
AV dissociation showing captures or fusions	rare	yes
Axis: Extreme Right Axis Deviation (R.A.D., see page 231)	rare	yes

A few clues and good judgement can help you distinguish between VT and wide QRS complex SVT (with aberrant ventricular conduction). Begin with the history, and get a 12 lead EKG.

nations with VT is most likely elderly and suffering

mygen supply to the ventricular foci.	flow
Signs of AV dissociation (e.g., presence of fusions or captures, Extreme R.A.D. (-90° to -180°) are characteristic of	VT

Sote: If the QRS complex can be measured with accuracy, the QRS

SVT, even if widened by aberrant ventricular conduction, is usually

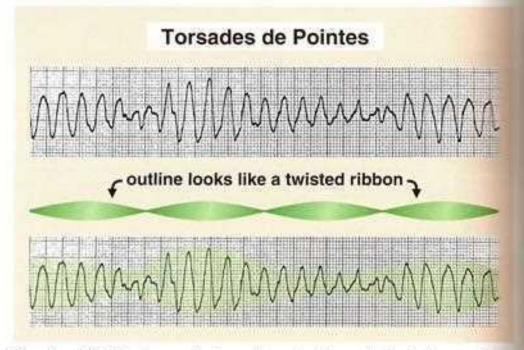
14 sec. or less in duration. However the ventricular complexes in

T are very wide, .14 sec. or greater. There are many criteria for

Estinguishing VT from SVT with aberrancy, but probably the most

estable to date are in:

Bragada et al: The differential diagnosis of a regular tachycardia with a wade QRS complex on the 12 lead ECG. PACE 1994; Vol.17: 1515-1524.



Torsades de Pointes is a peculiar form of (very) rapid ventricular rhythm caused by low potassium, medications that block potassium channels, or congenital abnormalities (e.g., Long QT syndrome), all of which lengthen the QT segment. The rate is a variable 250 to 350 per minute, usually in brief episodes.

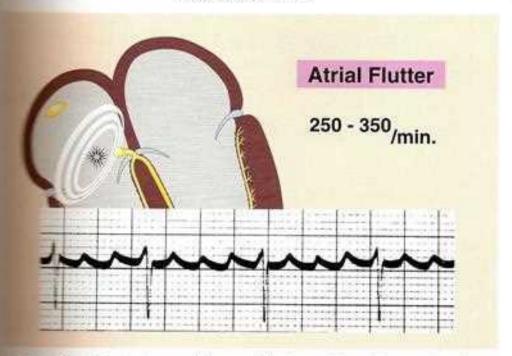
Note: Torsades* de Pointes means "twisting of points," which refers to the series of ventricular complexes that are upward-pointing then downward-pointing in a repeating continuum. In 1966, Dr. F. Dessertenne presented the first scientific description of this arrhythmia. He theorized that it was caused by two competitive, irritable foci in different ventricular areas – an explanation that seems quite plausible.

The rate of this arrhythmia is 250 to ____ per minute, but fortunately it usually occurs only in brief self-terminating bursts, for at that rate there is no effective ventricular pumping.

Note: On EKG, the amplitude of each successive complex gradually increases and then gradually decreases, so when viewed as a whole, the general outline or silhouette of the tracing looks like a series of end-to-end spindle shapes. Some say the tracing outline resembles a twisted ribbon. If unresolved, it can lead to a deadly arrhythmia.

35

[&]quot;This is the correct spelling; don't forget the "s" at the end of "Torsades", even though it is not pronounced.



Flutter originates in an atrial automaticity focus. The rapid succession of back-to-back atrial depolarization waves, "flutter" waves, suggest a reentry some experts (see the last paragraph on the next page).

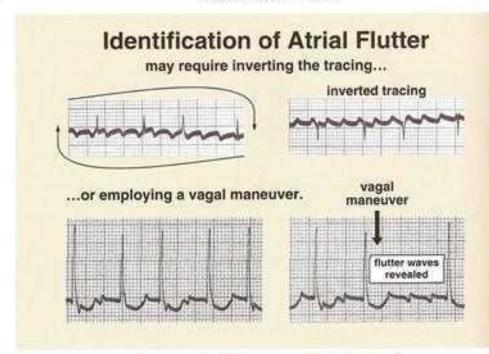
flutter an extremely irritable atrial automaticity focus
at a rate of 250 to 350 per minute, producing a rapid
depolarizations.

atrial

On EKG, atrial flutter is characterized by consecutive, second "flutter" waves in rapid back-to-back succession.

baseline appears to vanish between the back-to-back flutter and because the waves are identical, they are described baving the appearance of the teeth of a saw or a "saw tooth" seine. Turn back to PAT with (AV) block to make sure that secognize the difference.

The AV Node has a long refractory period, so only one series of flutter waves conducts to the ventricles. Therefore, very rapid series of atrial depolarizations cannot drive the scies at the same excessive rate; perhaps only one of two series are the same excessive rate; perhaps only one of two series the ventricles in atrial flutter.



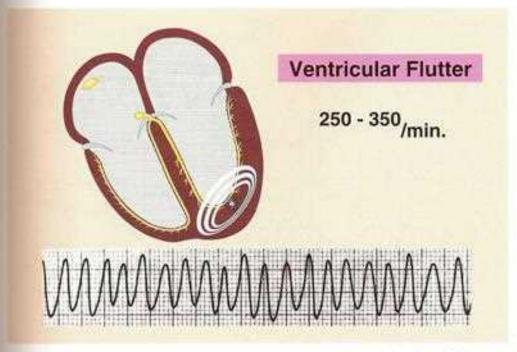
Inverting a tracing of suspected atrial flutter can help in its identification. Also, vagal maneuvers can be an effective diagnostic aid (see page 61.)

When in doubt about atrial flutter, inverting the _____ tracing may be helpful.

Note: With atrial flutter there may be a rapid QRS response rate, particularly in 2:1 ratios (flutter waves:QRS response), masking the flutter waves. Vagal maneuvers increase AV Node refractoriness, allowing fewer flutter waves to be conducted to the ventricles. This produces a longer series of flutter waves that are easier to identify.

Note: The "Maze" surgical procedure cuts (and resutures) the atria into a maze of channels that provides a continuous pathway from the SA Node to the AV Node. This procedure eliminates any possibility of reentry circuits. Yet a study of patients recovering from the maze procedure, revealed that 47% developed atrial flutter (or atrial fibrillation) postoperatively. This raises considerable doubt that the origin of atrial flutter could be reentry.

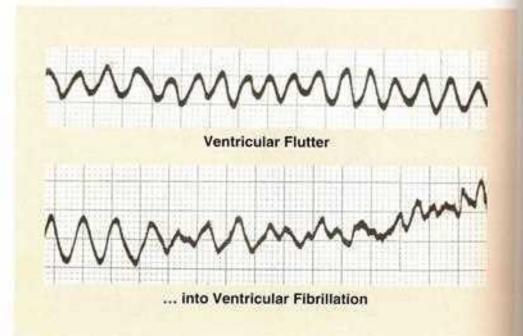
EKG



Ventricular Flutter is produced by a single ventricular automaticity focus firing at an exceptionally rapid rate of 250 to 350 per minute. It produces a rapid series of smooth sine-waves of similar amplitude,

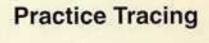
beas that is desperately discharging at a rate of to per minute.	250 350
The ventricular rate in ventricular flutter is so rapid that the hardly have enough time to fill – even partially, this arrhythmia rapidly deteriorates into a deadly arrhythmia.	ventricles
The smoothwave pattern of ventricular flutter is its stinguishing characteristic.	sine

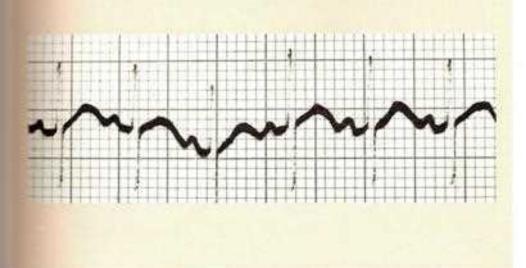
Note: Ventricular flutter produces a rapid series of smooth sine-waves of similar amplitude, whereas the waves of Torsades de Pointes get progressively larger, then smaller, producing a general outline of connected spindle shapes (page 158). Ventricular Flutter rarely self-resolves and is nearly always a prelude to a deadly arrhythmia... see next page.



True ventricular flutter almost invariably deteriorates into ventricular fibrillation, which requires immediate Cardio-Pulmonary Resuscitation and defibrillation.

Note: During ventricular flutter, the ventricles are contracting at an alarming rate. The above (separated but continuous) tracing shows ventricular flutter at a rate of about 300 per minute, which is five contractions per second. Blood is a viscous fluid, and the ventricles cannot properly fill (and empty) at a rate of 5 times per second; in fact, they hardly fill at all. For this reason, there is no effective cardiac output. Therefore, the coronary arteries are not receiving blood, and the heart itself has no blood supply. Ventricular Fibrillation ensues, as many profoundly hypoxic ventricular automaticity foci desperately try to compensate... in vain.





a monitored patient became very concerned about a sudden pounding in his chest.

By the history and the rate (which you determined by observation),	
identify the rhythm as a paroxysmal	tachycardia
www. we'll determine the causative irritable automaticity focus.	

Because this paroxysmal tachycardia has narrow, normal backing QRS's, it could <u>not</u> have originated in an irritable focus; therefore it must be some type of sprayentricular tachycardia.

ventricular

There appear to be P' waves present, so we are probably dealing
with an automaticity focus in the ______. You have already ruled
at a Junctional focus, because any retrograde depolarizations
that it might have produced, would record as inverted P' waves
which are usually adjacent to the QRS when they precede it).

atria

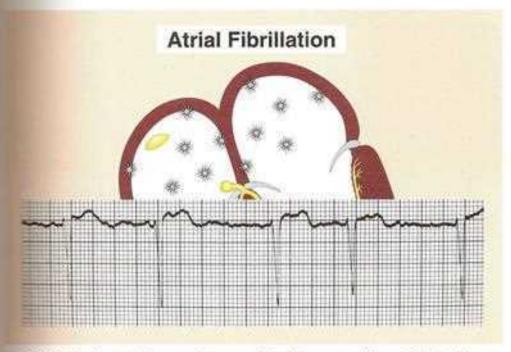
Note: This is Paroxysmal Atrial Tachycardia (PAT), and because each P' wave produces a QRS response, it could not be PAT with block. Quickly review the illustrations for the paroxysmal tachycardias and flutter before we go on. Take your time.



"Fibrillation" is a totally erratic rhythm caused by continuous, rapid rate discharges from numerous automaticity foci in either the atria or in the ventricles.

Note: Fibrillation is caused by rapid discharges from numerous profoundly irritable automaticity foci in the atria (Atrial Fibrillation), or due to numerous profoundly irritable foci in the ventricles rapidly discharging (Ventricular Fibrillation). Both types represent a pathological condition: these irritable foci all suffer from entrance block, so they are parasystolic. Since they cannot be overdrive-suppressed, they all pace rapidly at once. The resulting rhythm is so erratic and uncoordinated that distinct, complete waves are not distinguishable, thus rates are impossible to determine. The involved chambers merely twitch rapidly.

Note: The "rate" 350 to 450 per minute is not a true rate, since many of the foci discharge simultaneously. The number and the tachy-rate of individual foci is conjectural. The range of "rate" is more relative and hypothetical than real, because fibrillating chambers do not effectively pump at all.



Fibrillation (AF) is caused by many irritable <u>parasystolic</u> atrial foci (with mane block) firing at rapid rates, producing an exceedingly rapid, erratic atrial mater. The atrial "rate" is 350 to 450 per minute. Notice the irregular ventricular posses.

fibrillation occurs when many irritable atrial foci fire

addy, but since they are parasystolic, none of them can be

addrive-_____; they all rapidly pace at once to produce suppressed

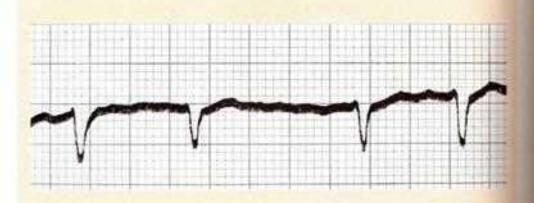
accessively rapid series of tiny, erratic spikes on EKG.

Because so many atrial foci are rapidly firing, no single appolarization spreads very far. Only a small portion of the atria depolarized by any one discharge from an atrial focus. Depolarizations from foci near the AV Node conduct to the entricles, producing a very irregular ventricular rhythm. see rhythm strip, page 351.

Sote: With a Normal Sinus Rhythm, each pacing impulse that the SA Node generates spreads through the atria like an enlarging, circular wave; much like a pebble dropped into a pool of water. However, the multiple erratic depolarizations of atrial fibrillation are analogous to a rain shower striking the same pool.

^{*} Atrial fibrillation is usually initiated by parasystolic foci in the pulmonary vein ostia of the left atrium.

Atrial Fibrillation



Atrial Fibrillation often appears as a wavy baseline without identifiable P or P' waves. The QRS response is not regular and may be fast or slow.

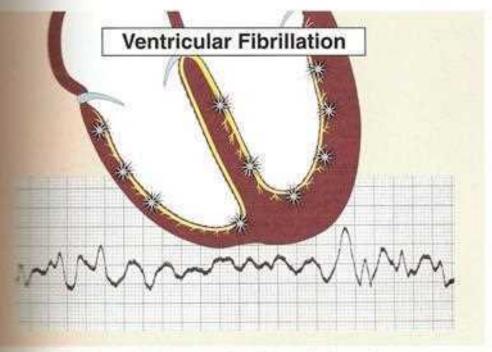
Atrial Fibrillation may cause such small, erratic spikes that it appears like a wavy baseline without visible __ waves (and without distinguishable P' waves either).

Note: Only discharging foci near the AV Node can (occasionally) stimulate it, but the AV Node sorts out a normal ventricular rate.

The AV Node is irregularly stimulated during atrial fibrillation, so the ______(QRS) response is irregular. On EKG, you may see only random QRS's (see illustration), so the pulse is irregular also.

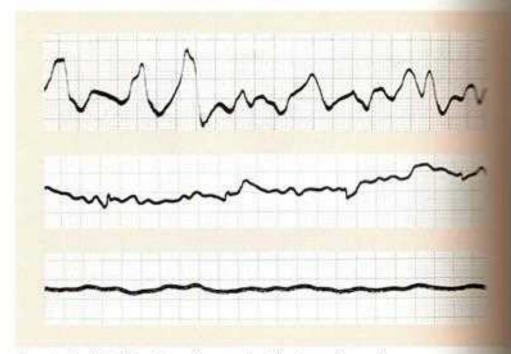
ventra

Note: With Atrial Fibrillation, the ventricular rate depends on the AV Node's duration of refractoriness after it is stimulated. During AF, the AV Node usually allows a relatively normal range of ventricular rate, albeit always irregular. Sometimes the AV Node permits an increased number of depolarization stimuli to pass through, producing a rapid ventricular rate that may require pharmacological control. Always determine the ventricular (pulse) rate (QRS's per 6-second strip times 10) and document it. If the ventricular rate is out of a safe range for the patient, it should be treated appropriately.



ricular Fibrillation (VF) is caused by rapid-rate discharges from many
less, parasystolic ventricular automaticity foci, producing an erratic, rapid
log of the ventricles (ventricular "rate" is 350 to 450 per minute).

cular Fibrillation is due to numerous parasystolic ventricular seeing rapidly (each of them suffering from entrance block, can not besuppressed); this produces an twitching of the ventricles.	overdrive
cause there so many ventricular firing rapidly, one repeatedly depolarizes only a small area of ventricle, results in a rapid, ineffective twitching of the ventricles.	foci
erratic twitching of VF has been called a "bag of worms," this is the way the ventricles actually appear. On EKG the is totally erratic, without identifiable, and the erricles do not provide mechanical pumping. Emergency!	waves



Ventricular Fibrillation is easily recognized by its totally erratic appearance and of any identifiable waves on the electrocardiogram.

Note: These three strips are from a continuous tracing of the same patient's dying heart. Notice how the amplitude of the deflections diminishes as the heart dies.

We recognize Ventricular Fibrillation by its completely erratic appearance on the EKG tracing. Even with large deflections, there are no identifiable ______.

There is no predictable pattern with ______ Fibrillation. As you can see, it looks different at every moment, but it is so erratic that it is difficult to miss, thank Goodness!

If you do recognize any repetition of pattern or regularity of deflections, you probably are not dealing with _____.

Venu



Emericular Fibrillation is a type of cardiac arrest, for there is no pumping action
 the heart; this is a dire emergency! VF requires immediate CPR and defibrillation,
 some type of electrical defibrillator.

micular Fibrillation is a type of cardiac _____. There is no metric cardiac output, because the ventricles are only twitching _____.

There is no ventricular pumping, so there is no circulation.

arrest

WF requires immediate defibrillation. Cardiac Arrest is an mergency that demands immediate intervention. Cardio-Pulmonary tesuscitation (cardiac massage and assisted respiration) is carried out marder to circulate oxygenated blood by external mechanical means. The technique of CPR was originally taught only to hospital and abulance personnel, but it is imperative that every person master technique. Only when CPR skills are universally known, can all actims of cardiac arrest get immediate lifesaving care at any location.

There are two other types of cardiac arrest. Cardiac Standstill

Ssystole") occurs when there is no detectable cardiac activity on

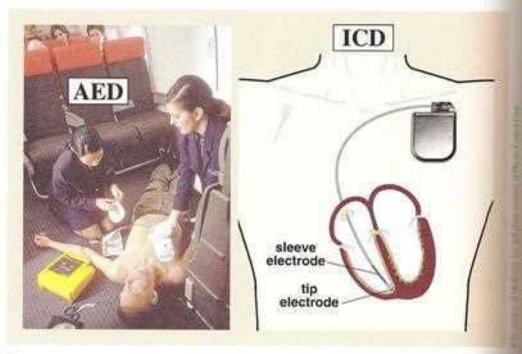
E.G. This is a rare circumstance when the SA Node and the escape

mechanisms of all the foci at all levels are unable to assume pacing

sponsibility. Pulseless Electrical Activity (PEA) is present when a

sing heart produces weak signs of electrical activity on EKG, but

me moribund heart cannot respond mechanically (no detectable pulse).

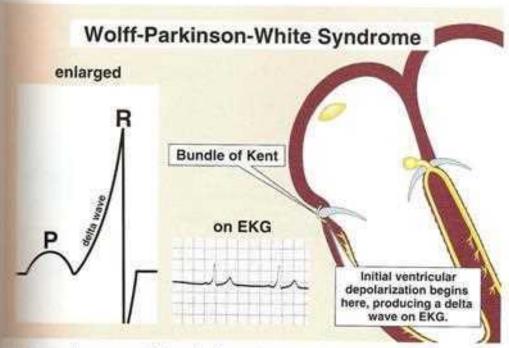


There are now computerized defibrillators that can detect Ventricular Fibrillation and immediately deliver a defibrillating shock. One type, the AED, is a portable unit that can be operated by the public. Another type, the ICD, is a small unit that is implanted under the chest skin to automatically defibrillate appropriate patients as needed.

Note: The Automated External Defibrillator (AED) is a small portable unit. When its electrodes are placed on the chest of an unconscious person, it is programmed to identify VF and deliver a defibrillating shock.

Note: The Implantable Cardioverter Defibrillator (ICD) is implanted under the chest skin of patients likely to develop Ventricular Fibrillation. Wire leads from the ICD are attached to the heart to detect VF and deliver a defibrillating shock. This mini-computer can also identify other arrhythmias and treat them with timed electrical stimuli, and it can pace if a bradycardia ensues. A technological wonder!

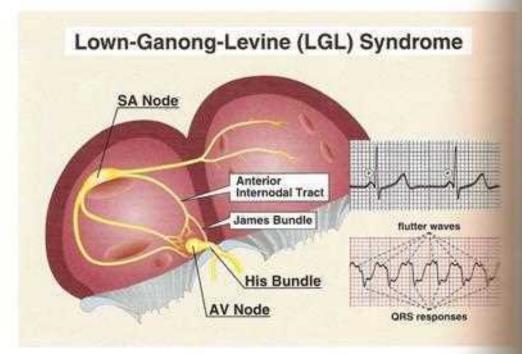
Please review all "fibrillation" illustrations.



abnormal, accessory AV conduction pathway, the bundle of Kent, can "short "short the (usual) delay of ventricular conduction in the AV Node. This prematurely polarizes ("pre-excites") a portion of the ventricles (producing a delta wave on usual statement of the polarization begins.

causes ventricular causes ventricular causes ventricular (WPW) syndrome.	Kent
delta wave creates the illusion of a "shortened" PR interval lengthened" QRS. The delta wave actually records the	
pre-excitation.	ventricular

- WPW syndrome is very important because persons with such an accessory pathway can have paroxysmal tachycardia by three possible accessory.
- apid conduction supraventricular tachycardia (including atrial flutter or atrial fibrillation) may be rapidly conducted 1:1 through this accessory pathway producing dangerously high ventricular rates.
- some Kent Bundles have been found to contain automaticity foci
 foci
- re-entry ventricular depolarization may immediately restimulate
 atria in a retrograde fashion via the accessory pathway causing
 beoretical circus re-entry loop.



In patients with LGL syndrome, the AV Node is bypassed by an extension of the Anterior Internodal Tract. Absent the conduction delay in the AV Node, this "James bundle" conducts atrial depolarizations directly to the His Bundle without delay. The can pose a serious problem with rapid atrial arrhythmias like atrial flutter.

VCT

Ordinarily the AV Node filters rapid supraventricular rates, in order to transmit depolarization to the _____ at a physiologically reasonable rate.

Absent the filtering effect of the AV Node, patients with LGL syndrome can transmit rapid atrial rates directly (1:1) to the His Bundle, driving the ventricles at very _____ rates.

With this syndrome, the AV Node is bypassed by the James tract, so there is no significant PR interval delay; the P ______ are adjacent to their QRS's on the EKG.

Note: You may now review Rhythm, Part I by turning to the Personal Quick Reference Sheets on page 336-338, and relate this to the simplified methodology that is summarized on page 334.

Chapter 6: Rhythm, Part II

Before you begin, look at this chapter's summary on pages 334 and 339.

Rhythm, Part II

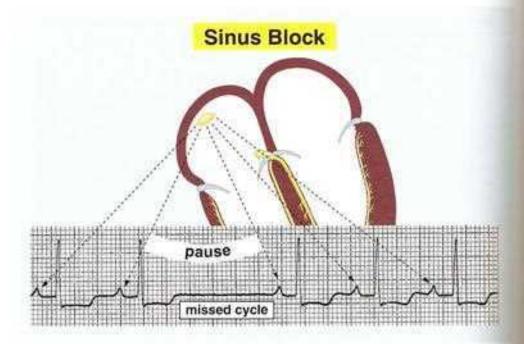
Blocks

- Sinus Block
- AV Block
- Bundle Branch Block
- Hemiblock (begins on page 295, Chapter 9)

Blocks retard or prevent the conduction of depolarization; they can occur in the SA Node, the AV Node, or in the larger divisions of the ventricular conduction system. The general public and the media often refer to them as "heart blocks".

Blocks may develop in any of these areas: the AV Node, the His Bundle, the Bundle Broom the two subdivisions of the Left Bundle Broom the Left Bundle Broom the Left Bundle Broom the Broo	anches, or in either	
These are blocks of electrical conduction that our retard) the passage of	t prevent _ stimuli.	depolarization

Note: When examining the rhythm on a tracing, always check for all the varieties of block, because the same patient can have more than one type of block.



An unhealthy SA Node (Sinus Node) may temporarily fail to pace for at least one cycle, this is **Sinus Block**, then the SA Node resumes pacing. Notice that the missed cycle has no P wave; a very important feature.

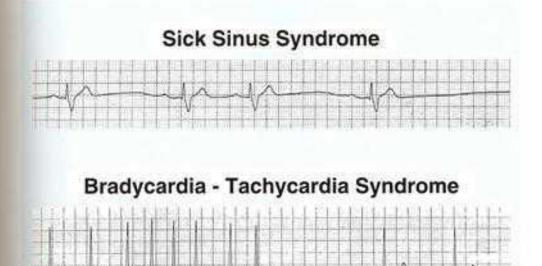
CNC

TOCH

With Sinus Block (also called "SA Node Block" or simply
"SA Block"), an unhealthy SA Node* stops its pacing activity for
at least one complete , so the block is usually transient.

Note: The P waves before and after the pause are identical, since they originate in the SA Node. The SA Node continues to generate atrial depolarizations with the same timing as before the block. However a long pause may elicit an escape beat from an automaticity focus before the SA Node can resume pacing (see pages 119-121).

^{*} Some experts claim that the SA Node does generate a stimulus, but that it is blocked from leaving the SA Node. This is referred to as Sinus "Exit" Block.



Sick Sinus Syndrome (SSS) is a wastebasket of arrhythmias caused by SA Node dysfunction associated with unresponsive supraventricular (atrial and Junctional) automaticity foci, which are also dysfunctional and can't employ their normal escape mechanism to assume pacing responsibility.

Note: Sick Sinus Syndrome most often occurs in elderly individuals who have heart disease. It is usually characterized by marked Sinus Bradycardia, but without the normal escape mechanisms of atrial and Junctional foci. SSS may also present as recurrent episodes of Sinus Block or Sinus Arrest associated with faulty (or absent) escape mechanisms of all supraventricular foci.

Note: Because of the exclusive parasympathetic innervation to the SA Node and all supraventricular (atrial and Junctional) foci, excessive parasympathetic activity depresses the pacing rate of the SA Node, and depresses the atrial and Junctional foci as well. Therefore, young, bealthy individuals (e.g., conditioned athletes like marathon runners) who often have parasympathetic hyperactivity at rest, appear to exhibit convincing signs of SSS ("pseudo" Sick Sinus Syndrome).

Sometimes even Atrial Flutter or Atrial Fibrillation) mingled with the Smus Bradycardia. This is **Bradycardia-Tachycardia Syndrome**.

AV Block

1° (first degree) AV Block

2° (second degree) AV Block

3° (third degree) AV Block

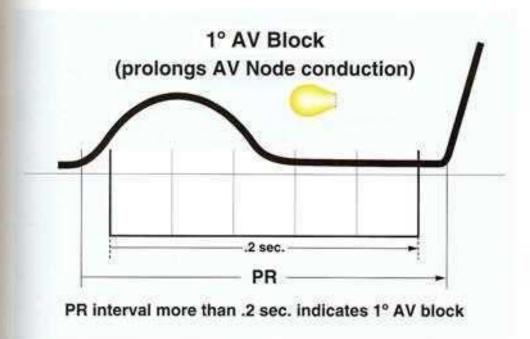
AV (Atrio-Ventricular) blocks either retard or eliminate (or both!) conduction from the atria to the ventricles.

Minor AV blocks lengthen the brief pause between atrial depolarization and ventricular	depolarization
Most AV blocks completely block some (or all)	ventricles

Note: The AV blocks are:

- first degree (1°) AV block (lengthens the delay between atrial and ventricular depolarization)
- second degree (2°) AV block (Wenckebach and Mobitz types)
- third degree (3") AV block (completely blocks the conduction of atrial stimuli to the ventricles)

Note: Whether "first degree" is written out or the shorthand notation, 1°, is used, both have the same meaning. This book will alternately use both methods for all degrees of AV block, since you will see both in the current literature. Although it is currently popular to omit the "AV" it is always understood.



First degree (1°) AV block retards AV Node conduction, prolonging the PR interval more than one large square (.2 sec.) on EKG.

Note: Technically, a "segment" is a portion of baseline, while an "interval" contains at least one wave. So the PR interval includes the P wave and the baseline that follows, up to the point where the QRS complex begins. Therefore, the PR interval is measured from the beginning of the P wave to the beginning of the QRS complex.

The delay caused by 1° AV block prolongs the ___ interval. PR

The PR interval normally should be less than one large square, which is less than ___ second. .2 (2/10)

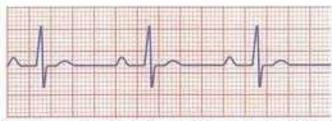
Note: You must observe (and record) the PR interval for every EKG. Some kind of AV block is present, if the PR interval is longer than one large square anywhere on an EKG.

1° AV Block





"Measure" PR by observation (one large square).



PR remains consistently lengthened cycle-to-cycle.

A first degree (1") AV block is characterized by a PR interval greater than .2 second (one large square). The PR prolongation is consistent in every cycle.

Once you recognize a prolonged PR ______, you should determine the type of AV block that is present.

interv

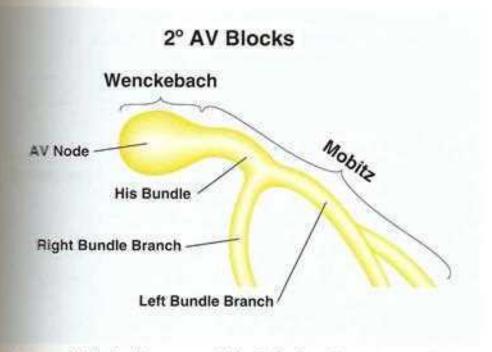
Some type of AV block is present if any PR interval is longer then ____ second, anywhere in the tracing.

(two-tenth

A 1º block® is present when the PR interval is consistently prolonged the same amount in every ______, and the P-QRS-T sequence is normal in every cycle also.

C3/C=

Whenever you hear "I" block," understand that it means "I" AV block."



degree AV blocks allow some atrial depolarizations (P waves) to conduct sentricles (producing a QRS response), while some atrial depolarizations are leaving lone P waves without an associated QRS. There are two general 2° AV block; those that occur in the AV Node, and those that occur below Node.

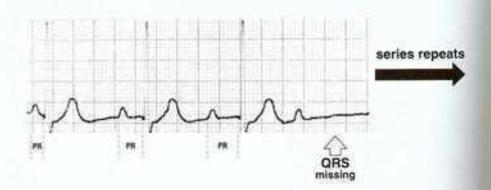
There are two types of 2" AV blocks:

- They produce a series of cycles with progressive blocking of AV Node conduction until the final P wave is totally blocked in the AV Node, eliminating the QRS response. Each repeating Wenckebach series has a consistent P:QRS ratio like 3:2, 4:3, 5:4, etc. (one less QRS than P waves in the series).
- * 2" blocks of Purkinje fiber bundles (His Bundle or Bundle Branches) are "Mobitz" (formerly called "Type II"). They usually produce a series of cycles consisting of one normal P-QRS-T cycle preceded by a series of paced P waves that fail to conduct through the AV Node (no QRS response). Each repeating Mobitz series has a consistent P:QRS ratio, like 3:1, 4:1, 5:1, etc.

Don't be intimidated by these descriptions, once you see the mings on the next few pages, you will understand immediately.

Pronounced "WINKY-bok"

"Wenckebach" 2° AV Block



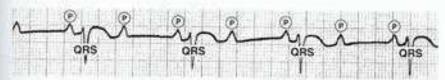
Wenckebach 2° AV block occurs in the AV Node. On EKG, the PR interval gradually lengthens in successive cycles, but the last P wave of the series fails to conduct to the ventricles (the final P lacks a QRS response). This series repeats.

On EKG, Wenckebach (2° AV block) gradually prolongs the PR interval in each successive cycle, until the final P wave of the series fails to produce a response ("dropped QRS").

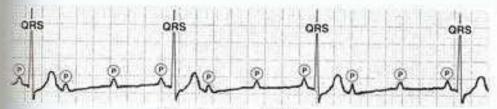
Each P wave and its associated QRS get progressively farther apart in successive cycles; the last P wave stimulus (totally blocked in the AV Node) stands alone at the end of the series. This typical Wenckebach pattern ("footprint") consists of anywhere from two to eight or more ______.

Note: Wenckebach is usually located in the AV Node. Wenckebach is sometimes caused by parasympathetic excess (inhibits the AV Node) or drugs that mimic or induce parasympathetic effects. Carefully examine EKG's for this characteristic, progressive lengthening of the PR in consecutive cycles, ending in a final lone P wave (see page 329). Repeating short series of Wenckebach footprints can produce "group beating" that looks somewhat like couplets of premature beats. Don't be fooled.

"Mobitz" 2° AV Blocks



2:1 Mobitz AV block



3:1 Mobitz AV block

Mobitz* 2° (AV) Block totally blocks a number of paced atrial depolarizations. P waves) before conduction to the ventricles is successful. This produces 2:1 two P waves to one QRS) or 3:1 (three P waves to one QRS) or even higher AV ratios. The series repeats. Mobitz is a serious problem; notice the extremely slow ventricular rates, which may produce loss of consciousness (syncope).

**stwo P waves to one ____ response, often referred to as **21 AV block" (or simply "2:1 block").

ORS

Note: Mobitz sometimes blocks three atrial depolarizations

(P waves) producing a single ventricular response (QRS); this is
written "3:1 AV block," or just "3:1 block", which describes the
mechanism of conduction. Poorer conduction ratios (e.g., 4:1, 5:1, etc.)
relate to increased severity of the block and are sometimes called
"advanced" Mobitz block.

warning! With Mobitz, every cycle that is missing its QRS has a regular, punctual P wave — but never a premature P wave see Note, page 128). This distinction is critical!

Previously called "Type II" or "Mobitz II."

2:1 AV Block Wenckebach vs. Mobitz

Most likely Wenckebach...

if the PR interval is lengthened, but the QRS is normal.

Most likely Mobitz...

if the PR interval is normal, but the QRS is widened.

Both Wenckebach and Mobitz have missing ("dropped") QRS's, so how can we differentiate between 2:1 Wenckebach and 2:1 Mobitz? Wenckebach is considered innocuous and Mobitz is considered pathological, so we should differentiate.

Note: On EKG, a 2:1 AV block could be a short, two-cycle Wenckebach. For example, if the first cycle is fairly normal but in the second cycle the PR lengthens just enough to prevent conduction through the AV Node, this is 2:1 Wenckebach. But by its appearance alone, most of us would probably interpret (correctly?) a 2:1 block as Mobitz. Perhaps the following will help...

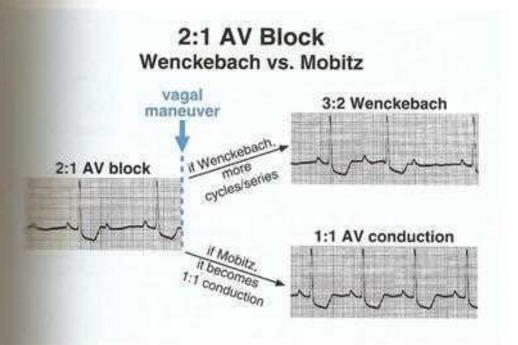
Because Wenckebach commonly originates in the AV _____.

a 2:1 AV block of this origin often has an initial lengthened PR with no wide QRS pattern* (typical of Bundle Branch Block).

But since Mobitz originates below the AV Node, in the His Bundle or the Bundle Branches, we recognize that it often has a normal PR with a widened _____ (Bundle Branch Block) pattern.9

Note: Since differentiating between these two types of 2:1 AV Block is clinically very important, we may need to employ bedside diagnostic techniques to make the distinction (next page).

^{*} The wide QRS pattern, typical of Bundle Branch Block, is soon explained (pages 191-201)



block) is important clinically. In order to determine which type of 2:1 (2° AV)

a patient has, we can carefully employ vagal maneuvers (see page 61).

Occasionally an EKG of 2:1 block (like the one on the cover of book) has criteria (i.e., for PR length and QRS width) that fit both be bach and Mobitz. This may require the judicious use of a vagal beaver to differentiate between the two.

tion, so vagal	inhibit the AV Node,	maneuvers
----------------	----------------------	-----------

increase parasympathetic parasympathetic increasing the number of series to produce 3:2 or 4:3 Wenckebach.

Node

the 2:1 block is Mobitz (i.e., the block is in the ventricular), vagal maneuvers either eliminate the block, soring 1:1 AV conduction, or they have no effect.

system

On every EKG check:

1. PR Interval

- increased consistently in 1° AV block
- progressively increases in each series of cycles with Wenckebach
- totally variable in 3° AV block
- decreased in WPW and LGL syndromes

2. P without QRS response

- Wenckebach and Mobitz 2° AV blocks
- 3° AV block independent atrial and ventricular rates

Let's take a moment to see why routine EKG examination requires both checking the PR interval and looking for P waves missing their QRS response. Routinely checking these two parameters can reveal the entire spectrum AV conduction problems.

A prolonged PR	interval can alert	you to the existence
of 1° AV block,	2° AV block, and	3° AV

An EKG with P waves lacking a QRS response can expose 2° AV blocks and 3° ____ block.

Note: Let's pause to contemplate how these two diagnostic parameters relate to each type of AV block. Really, take a moment and try this. It's not just a meaningless exercise. You have consumed a great deal of practical knowledge about AV blocks. Now you can easily detect these blocks by checking both parameters on every EKG you see. In each instance, you should consider not only the anatomical origin of the problem, but its prognostic significance to the patient as a person. Congratulations on your progress; you should take pride in your knowledge.

Practice Tracing



examining physician noticed that a patient had an irregular pulse. The doctor was seed to feel a group of three pulse beats followed by a pause, and this group seed to repeat over and over. Let's share the patient's EKG.

see scan the PR intervals and discover that the cycle has a PR interval that exceeds .2 sec..

Block.

 $\Delta \lambda$

booking for P waves that lack a QRS response,

lone P wave with no _____ response following

complete cycle.

ORS

at first, but becomes progressively longer
successive cycle. We now recognize
block.

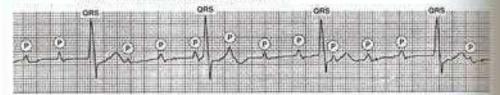
Wenckebach

Complete (3°) AV Block



When the conduction of supraventricular depolarizations to the ventricles is totally blocked...

focus escapes to pace the ventricles



an automaticity focus escapes to pace the ventricles at its inherent rate.

Complete (3°) AV block is a total block of conduction to the ventricles, so atrial depolarizations are not conducted to the ventricles. Therefore, an automaticity focus below the complete block escapes to pace the ventricles at its inherent rate.

3° block is a complete block that prevents sinus-paced	
atrial depolarizations from reaching the	
THE COLOR WINE TO ALL THE WAY A COLOR DESIGNATION OF THE COLOR DESIGNAT	

ventro

A single block of the AV Node or the His Bundle can be "complete," but more distally in the ventricular conduction system, there must be complete blocks of all subdivisions (branches) to eliminate ________ to the ventricles.

conduc

Absent paced depolarizations from above, an automaticity focus below the complete block escapes to pace the ventricles at its inherent.

Note: The location of the escaping focus depends on the location of the complete (3°) block. Next, let's look at the possibilities.

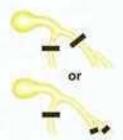
Forms of Complete (3°) AV Block



Complete block in the upper AV Node leaves Junctional foci to escape and pace the ventricles.



Complete block of the entire AV Node or in the His Bundle leaves only a ventricular focus to pace.



Below the His Bundle, all paths are completely blocked, so a ventricular focus escapes.

below the block in the AV Node, leaving only a ventricular focus to below the ventricles, or... the block may be below the AV Node, leaving only a ventricular focus to be and pace the ventricles. To be a "complete block," all avenues of AV blocked.

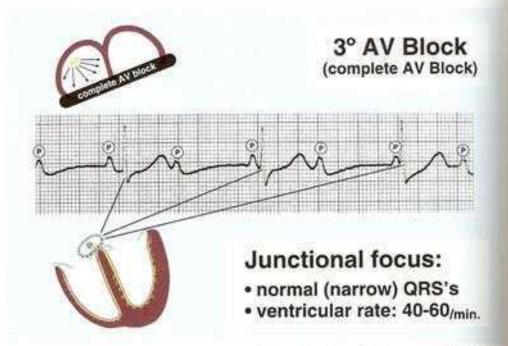
manufacture block is high in the AV Node, a Junctional focus
lastest-pacing focus below the block), escapes to
at its inherent rate.

ventricles

Node (for instance in the His Bundle), that leaves only

sentricular focus escapes to pace at its (slow) ______ rate. inherent

Regardless of the location of the focus that escapes to pace
entricles, the atria remain independently paced by the SA Node,
EKG, we see a Sinus-paced atrial (P wave) rate and a totally
wordent, focus-paced, slow ventricular (QRS) rate. Complete
lock produces this "AV dissociation" that records on EKG as
ally normal) P wave rate superimposed over an independent,
QRS rate. The AV dissociation (on EKG or cardiac monitor)
aff that there is probably a complete AV block.



If a complete AV block occurs above the AV Junction (i.e., in the upper AV Node), then a Junctional focus, no longer overdrive-suppressed, escapes to pace the ventricles. On EKG, we see Sinus-paced P waves and a slower, independent QRS rate, usually with normal QRS complexes.

Note: If the complete AV block is in the AV Node, above the AV Junction, then a Junctional focus, no longer overdrive-suppressed, escapes to pace the ventricles. This is an "idiojunctional rhythm."

With a complete AV block, if the QRS's appear normal
(because each pacing stimulus passes down the ventricular
conduction system), we know that a Junctional focus must
be pacing the

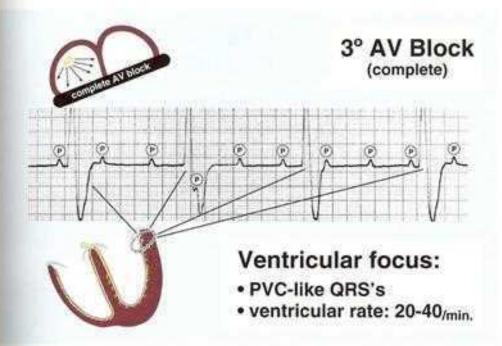
ventracas

Note: Sometimes paced depolarizations from a Junctional focus may have to pass through diseased regions in the ventricular conduction system, delaying depolarization in some areas of the ventricles, producing wide QRS complexes.

If the ventricular rate ranges between 40 and 60, then a focus in the ______ is probably pacing the ventricles.

AV Jun

^{*} Pacing by a Junctional focus may accelerate to become an accelerated idiojunctional rise



When a complete AV block occurs below the AV Junction, a ventricular focus escapes overdrive suppression to pace the ventricles at its slow inherent rate of only 20 to 40 per min.; so slow, in fact, that cerebral blood flow is compromised and sencope may ensue.

RS) rates, you should check the morphology of the QRS's. Sen we see large, wide, PVC-like complexes, we know that	
sentricles are probably being paced by a focus.	ventricular
The also see that the ventricular rate is within the inherent rate	
tage (20 to 40 / min.) of a ventricular	focus.

We understand that a ventricular focus could only escape to pace

there were no Junctional focus available above it. So the complete

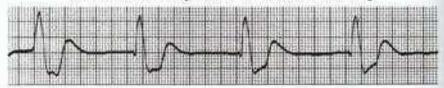
AV block either obliterated the entire AV Node or it occurred below

AV Junction (i.e., below the AV Node).

In 3° (complete) AV block the ventricular rate may be so slow that blood flow to the brain is inadequate, and the patient may lose asciousness (syncope). This is Stokes-Adams Syndrome. Patients the complete AV block need continuous surveillance and maintenance airway... many die needlessly without. Respond! Patients with AV block eventually need an artificial pacemaker.

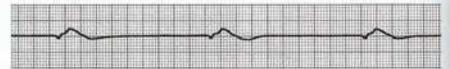
Downward displacement of the pacemaker

No visible supraventricular activity



prognosis worse for:

· wider complexes · diminished amplitude · slower ventricular rate



The above tracing is not caused by a 3° AV block. Don't be trapped by assuming that wide complex bradycardia is always due to a 3° block. Can you see signs of independent atrial activity? In practice, you should check all leads.

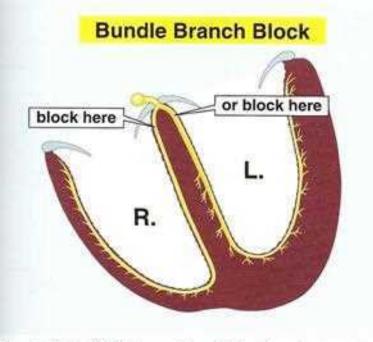
Bradycardia with wide ventricular complexes is not always pathognomonic for complete AV block, so identify AV dissociation (independent atrial and ventricular activity) before calling any wide complex bradycardia a 3° AV ______.

Note: The absence of atrial activity with wide complex bradycardia indicates that neither the SA Node nor supraventricular foci are viable enough to pace the atria. This failure of all automaticity centers above the ventricles, called "downward displacement of the pacemaker" usually carries an unfavorable prognosis. Before pronouncing this "downward displacement," make certain that the flat baseline is not due to atrial fibrillation.

Note: Extremely high serum K⁺ concentrations "hyperkalemia" can severely depress the SA Node and supraventricular foci, producing the same EKG findings. Hyperkalemia can cause cardiac asystole, a form of cardiac arrest.

Why don't we all take a break. Then, a little refreshed, we can look at Bundle Branch Block... next page.

bl-s



Bundle Branch Block (BBB) is caused by a block (of conduction) in the Right or in the Left Bundle Branch. The blocked Bundle Branch delays depolarization to the maricle that it supplies.

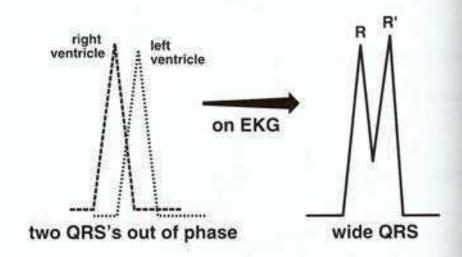
somally, the Right Bundle Branch quickly conducts the	
Bundle Branch does the same to the ventricle. See depolarization stimulus is conducted to both ventricles same time (i.e., simultaneously).	left

delay depolarization of the ventricle that it supplies.

Ordinarily both ventricles are depolarized simultaneously.

But with Bundle Branch Block, the unblocked Bundle Branch conducts smally, while depolarization in the blocked Bundle Branch has to seep slowly through the surrounding muscle (which conducts more why than the specialized Bundle Branch) to stimulate the Bundle Branch below the block. After the delay, depolarization proceeds apidly again below the block. However, the delay in the blocked Branch allows the unblocked ventricle to begin depolarizing before the blocked ventricle (see next page).

Bundle Branch Block



Therefore in Bundle Branch Block, one ventricle depolarizes slightly later than the other, causing two "joined QRS's" to appear on EKG.

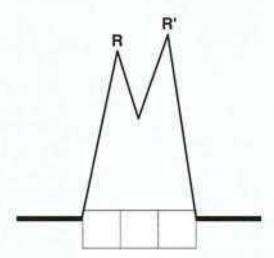
When a Bundle Branch Block is present, either the left or the right ventricle may depolarize late, depending on which Bundle is blocked.

Brans

Note: Individual depolarization of the right ventricle and depolarization of the left ventricle are still of normal duration. Because the ventricles do not depolarize simultaneously, this produces the "widened QRS" appearance that we see on the EKG. The two "out-of-sync" QRS's are superimposed on one-another, and the machine records this combined electrical activity as a widened QRS with two peaks.

Note: Because the "widened QRS" represents the non-simultaneous depolarization of both ventricles (one punctually depolarized, the other slightly delayed), we usually see two R waves named in sequential order: R and R'. The R' (pronounced "R-prime") represents delayed depolarization of the blocked ventricle.

Bundle Branch Block



Bandle Branch Block the "widened QRS" increases in duration to three small ares (.12 sec.) or greater, and two R waves (R and R') appear. The R' designates te delayed depolarization of the blocked ventricle.

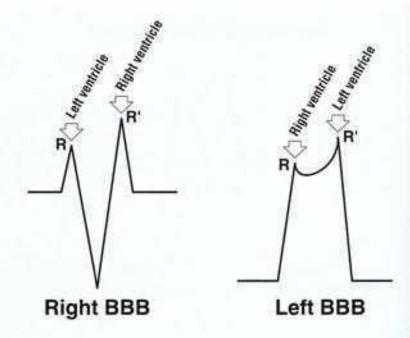
Simultaneous depolarization of the ventricles normally occurs in than twelve hundredths second, producing a QRS that is less than mee small squares in duration.

a diagnosis	of Bundle Branch Block is mainly based on the	
milesed	(.12 sec. or more duration).	QRS

morder to make the diagnosis of Bundle Branch Block, the complex should be at least small squares wide three (3) second). Check the QRS width of every EKG that you read!

The needle that records the EKG tracing moves rapidly enough record most of the heart's electrical activity accurately. However, great deflections the needle lags a bit mechanically, sometimes going us an exaggerated duration on the tracing. Therefore, it is best wheck the limb leads for QRS duration (where QRS amplitude is main, rather than the chest leads where the QRS deflections are great.

If a patient with BBB develops a supraventricular tachycardia, rapid succession of widened QRS's may imitate Ventricular Tachycardia, Careful!



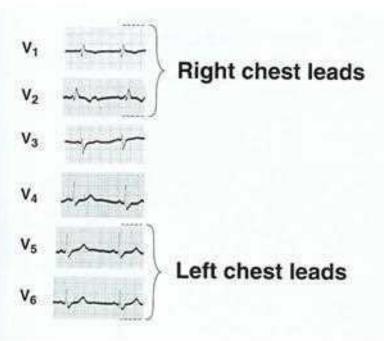
In Left Bundle Branch Block (LBBB), left ventricular depolarization is delayed. In Right Bundle Branch Block (RBBB), right ventricular depolarization is delayed.

In Bundle Branch Block, you first notice the widened _____
complex. Then you should be able to find the R.R' configuration in the chest leads.

In Right Bundle Branch Block, the left ventricle depolarizes punctually, so the R represents left ventricular depolarization, and the R' represents delayed ______ ventricular depolarization.

In Left Bundle Branch Block, left ventricular depolarization is delayed, so the right ventricle depolarizes punctually (R), and the R' represents delayed ventricular depolarization.

Kind of easy to understand, isn't it?



where is a Bundle Branch Block, look at leads V_1 and V_2 (right chest leads) and leads V_5 and V_6 (left chest leads) for the R,R'.

the QRS complex is wide enough (.12 sec. or more)

make the diagnosis of BBB, we immediately look at the
and left chest leads for the

R.R

During ventricular depolarization and just afterward (up to the wave), any additional stimulus cannot depolarize the caricles, that is, they are refractory to a premature stimulus. The bandle Branches have a refractory period, but the Left and Right Branch refractory periods are not identical, so with a survaventricular tachycardia one Bundle Branch is receptive to mulation before the other. At a certain critical rapid rate, one added Branch conducts before the other, producing non-simultaneous polarization of the ventricles, So this rate-dependent Bundle Branch broduces a tachycardia with wide QRS's that imitates incular Tachycardia.

be night chest leads are V₁ and ____.

V,

Right Bundle Branch Block





023

Right Bundle Branch Block produces an R.R' in the right chest leads, V1 or V2

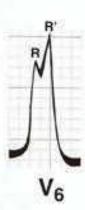
With a wide ____ (and a diagnosis of BBB), check the right and left chest leads for R,R'.

Then, if there is an R,R' in the right chest leads V₁ or V₂ there is probably a ______ Bundle Branch Block.

In Right Bundle Branch Block, the right ventricle is depolarizing slightly later than the left ventricle, so the R' in the above illustration represents the delayed depolarization to the (blocked) ______ ventricle.

Left Bundle Branch Block





BBB, an R,R' in the left chest leads V₃ or V₆ means that Left Bundle Branch is present. The R' represents delayed depolarization of the *left* ventricle.

chest electrode is located over the left ventricle in enest leads ___ and V₆.

V5

messionally, the R,R' in V₅ or V₆ will appear only as a messed peak with two tiny points in _____ Bundle Block. (Examine the QRS in V₅ in the illustration).

Left

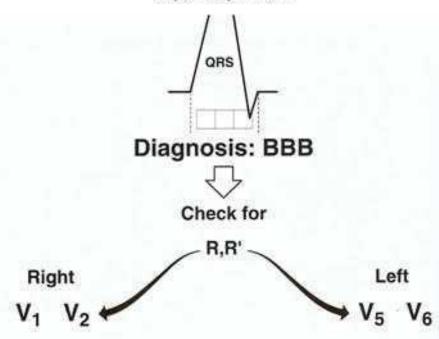
BBB the right ventricle depolarizes before the left ventricle,
the first portion of the wide QRS represents _____

right

Make a mental note of the typical QRS pattern (i.e., shape) of the pattern and Left BBB. A diagnosis is often made by appearance alone. The patterns are important because sometimes a PVC or the ventricular applexes in VT are said to have a "RBBB" or "LBBB" pattern; you would understand what that means. The same is true of the ventricular applexes (on EKG) produced by artificial pacemaker electrodes.

The Left Bundle Branch has two subdivisions ("fascicles");

sicks of these fascicles are called *Hemiblocks* (pages 295 - 305).



Remember, a wide QRS (three small squares) indicates BBB, and you should identify which Bundle Branch is blocked by checking the left and right chest leads.

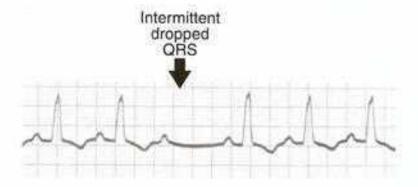
To diagnose BBB, the QRS complex must be at least ____ of a second in duration. Now, just for smiles, let's identify the type of BBB in the illustration on page 193.

Note: In some individuals recovery from refractoriness (during the last stage of repolarization) differs slightly in duration between bundle branches. So only at a particular critical rate of tachycardia, one ventricle depolarizes after the other to produce a rate-dependent Bundle Branch Block (see Note, page 195).

The R.R' pattern may occur in only one chest _____. It is often difficult to see the R', but usually it can be found in the right chest leads V_1 or V_2 or in the left chest leads V_5 or V_6 .

Note: Occasionally you will see an R,R' in a QRS of normal duration. This is called "incomplete" BBB.

Intermittent Mobitz (2° AV Block)



Occasional dropped QRS due to permanent BBB (one side) with intermittent BBB of the other side.

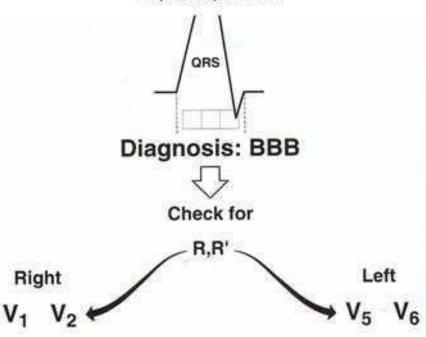
Simultaneous RBBB and LBBB prevents depolarization from reaching the ventricles; this is a complete (3°) AV block. So, block of one Bundle Branch with intermittent block of the other produces intermittent complete AV block, intermittent Mobitz.

Right BBB plus intermittent Left BBB will record on EKG as continuous Right BBB pattern QRS's with intermittent episodes of complete AV block (P waves without _____ response). ORS

Left BBB plus intermittent Right BBB will record on EKG as continuous Left BBB pattern QRS's with intermittent episodes of complete AV block (P waves without response).

ORS

Note: An EKG tracing or cardiac monitor display with a continuous BBB pattern QRS with an occasional missing QRS indicates intermittent complete AV block. The intermittent block may worsen, eventuating in a constant complete AV block. This intermittent Mobitz (exactly what it is) flashes an important warning sign, Intermittent Mobitz is the heart's warning that eventually it will need an artificial pacemaker to drive the ventricles at a normal rate. Don't let it slip by you unnoticed... for the patient's sake!



Remember, a wide QRS (three small squares) indicates BBB, and you should identify which Bundle Branch is blocked by checking the left and right chest leads.

12

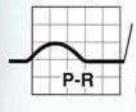
To diagnose BBB, the QRS complex must be at least ____ of a second in duration. Now, just for smiles, let's identify the type of BBB in the illustration on page 193.

Note: In some individuals recovery from refractoriness (during the last stage of repolarization) differs slightly in duration between bundle branches. So only at a particular critical rate of tachycardia, one ventricle depolarizes after the other to produce a rate-dependent Bundle Branch Block (see Note, page 195).

The R.R' pattern may occur in only one chest _____. It is often difficult to see the R', but usually it can be found in the right chest leads V_1 or V_2 or in the left chest leads V_5 or V_6 .

Note: Occasionally you will see an R,R' in a QRS of normal duration. This is called "incomplete" BBB.

Rhythm: always observe



... for AV Block (also P's missing a QRS response)



QRS... for Bundle Branch Block

that you must always visually measure* the duration of the PR intervals duration of the QRS complex when examining the rhythm on an EKG.

EKG's, you must always measure* the PR intervals because prolonged more than one large square, then there is some Block present (and, of course, look for missing QRS's, indicate that a 2° or 3° AV Block is present).

AV

EKG's, the QRS duration must be measured* for if it is seged to .12 second or _____ there is a Bundle Branch Block.

more

Always check the PR intervals and the QRS duration when minizing the rhythm on any EKG. This must be part of any EKG appreciation. The spontaneous appearance of Mobitz AV block or made Branch Block may be an early warning of impending section.

Hemiblocks commonly occur with infarction, so they are scribed in the Infarction chapter. A hemiblock is a block of one two subdivisions ("fascicles") of the Left Bundle Branch.

Cace you visually check these criteria on EKG, you should record the accise PR and QRS duration.

Bundle Branch Block

Vector = ?

Ventricular Hypertrophy?

The Mean ONS Vector. AXIS (NV) Set to this in the next chapter) and ventered hypertrophy cannot be determined accurately in the presence of Bundle Branch Block.

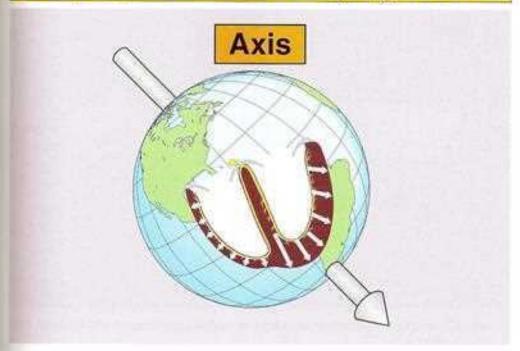
Note: Because the Mean QRS Vector represents the general direction of the simultaneous depolarization of the ventricles, with BBB it is very difficult to represent such a vector. This is because with BBB the ventricles do not depolarize simultaneously, so there are really two separate (right and left) ventricular vectors.

Note: The criteria for ventricular hypertrophy (enlargement) are based on a normal QRS. Bundle Branch Block produces large QRS deflections because each ventricle lacks the (usual) simultaneous electrical opposition from depolarization of the other ventricle. Therefore the EKG diagnosis of ventricular hypertrophy should be very guarded with BBB. However, atrial hypertrophy can be diagnosed in the presence of BBB.

Note: Let's review all the illustrations in this chapter. Then see "Blocks" in the Personal Quick Reference Sheets on page 339, and relate this to the simplified methodology that is summarized on page 334.

Chapter 7: Axis

Before you begin, look at this chapter's summary on pages 334 and 340.



Axis" refers to the direction of the movement of depolarization, which spreads throughout the heart to stimulate the myocardium to contract.

Note: The axis around which the earth rotates has nothing to do with electrocardiography, but we can borrow the large arrow ("Axis") in the illustration.

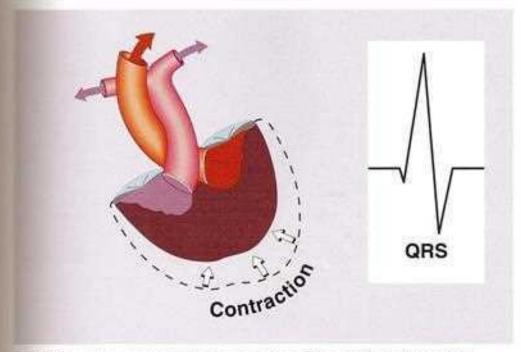
The progressive depolarization of thenoves in a certain direction.	myocardium
exis refers to the of	direction

^{*} Sometimes called "electrical axis."

Direction Of Vector Electrical Stimulus

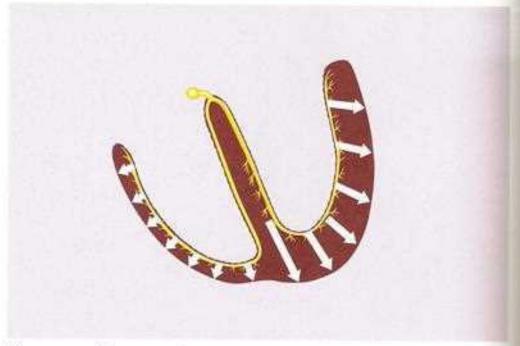
To demonstrate the direction in which depolarization is moving, we use an arrow that is called a "vector."

	nstrate the general direction of the depolarization by using a	vector
The vector sho	ows the direction in which is moving.	depolarization
When interpre	ting EKG's, a vector shows the of depolarization in the heart.	direction



The QRS complex represents the depolarization of the ventricular myocardium.

QRS complex represents the simultaneous	ventricles
Ventricular depolarization and contraction can be said occur at the same time, (but we know that	contraction
Depolarization of the ventricles and their contraction sepresented by the complex.	QRS



We can use small vectors to demonstrate ventricular depolarization, which begins at the endocardium that lines both ventricles and proceeds toward the outside surface (epicardium) in all areas at once.

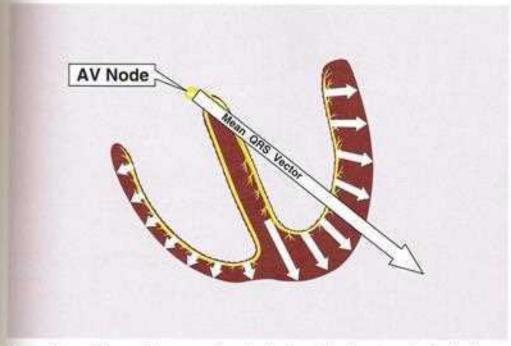
Note: Once depolarization is beyond the AV Node, the ventricular conduction system conducts this stimulus to the ventricles with great speed. In this way, ventricular depolarization begins at the endocardial lining of the ventricles and proceeds through the thickness of the ventricular wall in all areas at about the same time. (We will not yet address depolarization of the ventricular septum).

	ibers transmit depolarization to the myocardial cell e endocardium that lines both ventricles; this	s
	that depolarization begins at the general level	
of the	in all areas at about the same time.	endocardium

Depolarization of the ventricles generally proceeds from the endocardial lining to the outside (epicardial) surface through the full thickness of the _____ wall in all areas at once. (See small vectors in the illustration).

ventricul

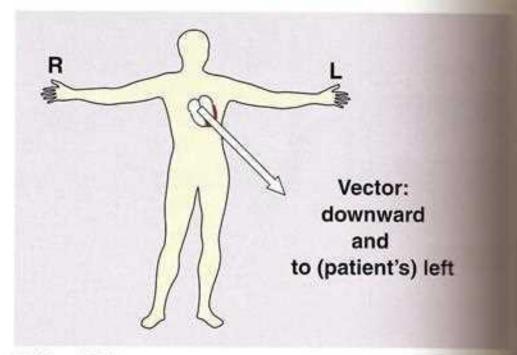
Note: Notice that the thicker left ventricular wall has larger vectors.



If we add up all the small vectors of ventricular depolarization (considering both direction and magnitude), we have one large "Mean QRS Vector" that represents the general direction of ventricular depolarization.

The Mean QRS Vector is the sum of all the smaller vectors of depolarization.	ventricular
By convention we consider the origin of the Mean QRS Vector to be the AV Node, so the "tail" of the Vector is always the,	AV Node
Because the small depolarization vectors of the thicker left ventricle are larger (previous page), the Mean QRS Vector points more toward the	left

Note: Remember that a vector represents both direction and magnitude of depolarization... bigger vectors represent greater magnitude.



The Mean QRS Vector normally points downward and to the patient's left, be this is the general direction of ventricular depolarization.

The ventricles are in	the left s	ide of the	chest and
angle downward and	to the	-	

The ______ Vector points downward and toward the patient's left side.

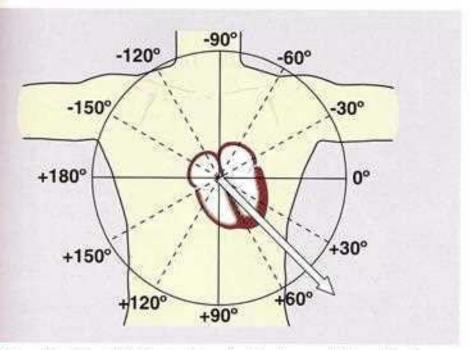
3.Faire

Note: From now on, we occasionally will use the word "Vector"

(with a capital "V") to represent the Mean QRS Vector, which depicts
the general direction and magnitude of ventricular depolarization.

Visualize the Vector over the patient's chest, and remember that it
begins in the AV Node.

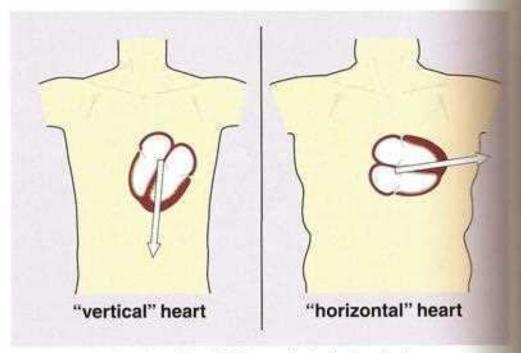
Note: Depolarization is an advancing wave of Na* ions.



The position of the Mean QRS Vector is described in degrees within a circle drawn wer the patient's chest. This circle is in the *frontal* plane. The limb leads are used to determine the position ("Axis") of the Mean QRS Vector in the frontal plane.

e can locate the position of the Mean QRS Vector thin a large around the heart.	circle
The center of the circle is the	AV Node
The Vector normally points downward and to the patient's left, that is, between 0 and degrees.	+90 (don't forget the +)

Note: The "axis" of the heart is simply the Mean QRS Vector when located by degrees in the frontal plane. For example, the axis of the heart in the above illustration is about +40 degrees. Review the illustration and note that 0° is on the patient's left, and that the lower half of the circle is "positive" degrees. The top half of the circle is "negative" degrees. Axis is often denoted in medical literature by an "A" as in "A $+30^{\circ}$ " or "A $= +30^{\circ}$ ", and it may be called "electrical axis."



If the heart is displaced, the Mean QRS Vector is also displaced in the same direction. The AV Node is always the tail of the Vector.

If the heart is rotated toward the patient's right side	¥
then the Mean QRS Vector moves toward the	as well
This is common in tall, slender individuals (see illu	istration).

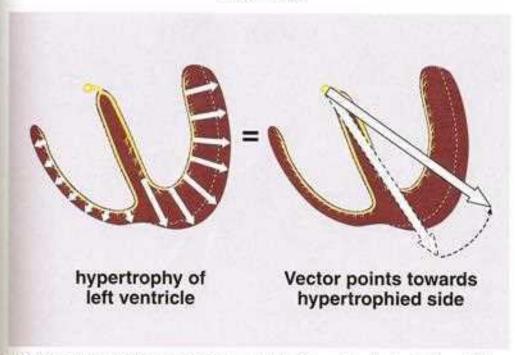
In very obese people the diaphragm is pushed up (and also the heart), so the Mean QRS Vector may point directly to the patient's _____. (See illustration).

The tail of the Vector is the

AV Note

Note: In obese individuals the increased abdominal pressure often pushes the diaphragm upward so the position of the displaced heart may be called a "horizontal heart". By the same token, a tall, slender individual may have a so called "vertical heart".

hypertrophied

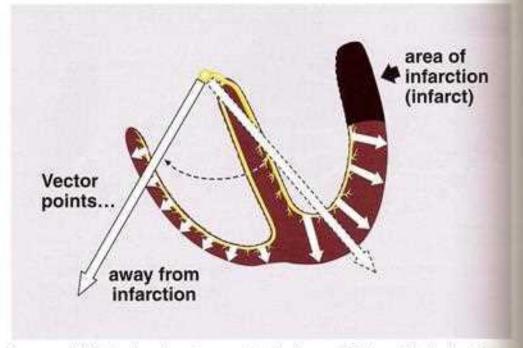


With hypertrophy (enlargement) of one ventricle, the greater depolarization activity of the hypertrophied side displaces the Mean QRS Vector toward the hypertrophied side.

ventricle.	500 BSC 5000 BC FF 4.75
50, the Mean QRS Vector deviates toward the	ventricle

Note: A hypertrophied ventricle has more (and larger) vectors, which draw the Mean QRS Vector in that direction.

There is increased depolarization in a



In myocardial infarction there is a necrotic (dead) area of the heart that has lost its blood supply and does not depolarize. The unopposed vectors from the other side draw the Mean QRS Vector away from the infarct.

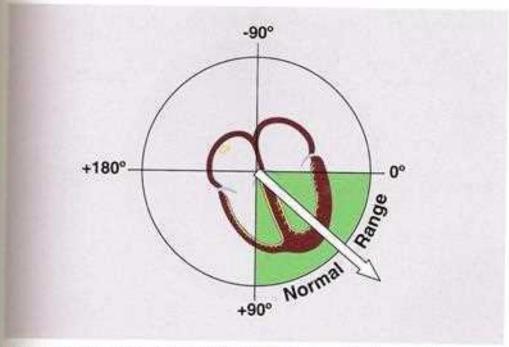
Note: Myocardial infarction occurs when a branch of one of the coronary arteries (the heart's own source of blood supply) becomes occluded. The area of myocardium supplied by this blocked coronary artery has no blood supply and becomes electrically dead (can't depolarize).

In myocardial infarction (coronary occlusion) there is an area in the ventricles that has no _____ supply. This infarcted area cannot depolarize, and therefore it has no vectors.

Since there is no depolarization (and no vectors) in the infarcted area, the vectors from the opposite side are unopposed, so the Mean QRS Vector tends to point away from the

blood

infar



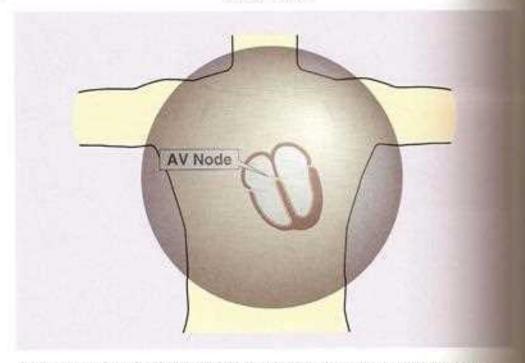
Now you understand why the Mean QRS Vector is diagnostically so valuable.

Axis" is the Mean QRS Vector when given in degrees, and the normal axis range to +90° in the frontal plane.

me patient's, that is, in the 0° to +90° range.	left
The Mean QRS Vector gives us valuable information about the position of the, and	heart
insight into ventricular, and	hypertrophy
apocardial	infarction

Note: The Mean QRS Vector tends to point toward ventricular typertrophy, and away from myocardial infarction. These basic principles of axis are so logical and easy to understand that you should employ this diagnostic* tool whenever a twelve lead EKG is available.

The diagnosis of Hemiblocks (pages 295-305) is based on changes in QRS Axis.

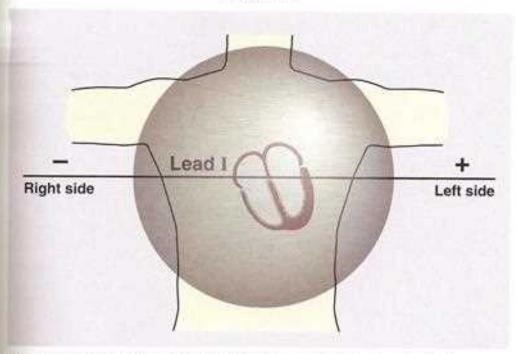


To determine the direction of the Vector, visualize a sphere surrounding the heart, with the AV Node at the center of the sphere.

Visualize a large _____ surrounding the heart.

The AV Node is the _____ of the sphere.

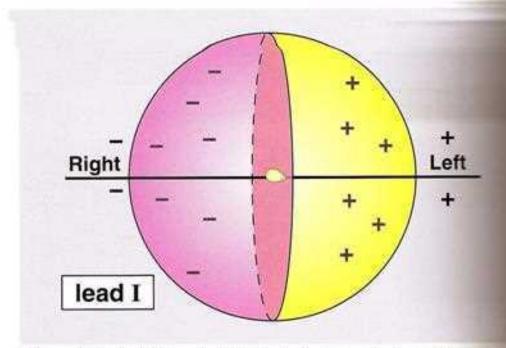
Note: The Mean QRS Vector has the AV Node as its tail, and the tip of the arrow touches somewhere on the surface of this hypothetical sphere.



With the sphere in mind, consider lead I (left arm with the positive electrode, right arm with the negative electrode).

Lead I uses the right and left for recording.	arms
If lead I is introduced into the sphere, the patient's left side left arm) is	positive
lm lead I the right arm is,	negative

Note: Lead I passes through the center of the sphere, which is the AV Node.

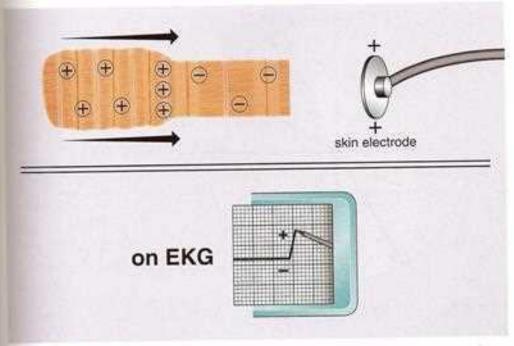


Still considering lead I, the patient's left-hand side of the sphere is positive, and the right half is negative. The center of the sphere is the AV Node.

We are considering only lead ___ at this time.

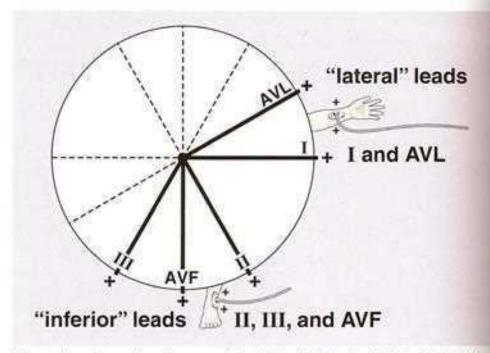
We will now consider the lead I sphere in two ______.

The patient's right half of the sphere is _____.



As the positive wave of depolarization within the myocardial cells moves toward a positive (skin) electrode, there is a simultaneous upward (positive) deflection recorded on EKG.

An advancing wave of	f depolarization may be considered charges.	positive
skin ele	esitive charges is moving toward a ectrode, there is a simultaneous upward ecorded on the EKG.	positive
that instant a depol	(upward) wave on EKG, it means arization stimulus is moving toward ode that is being used to record the EKG.	positive

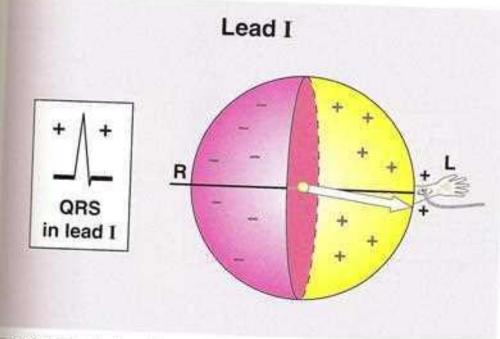


The positive electrode used to record the *inferior* limb leads, II, III, and AVF, is on the left "foot." The positive electrode that is used to record the *lateral* limb leads. I and AVL, is on the left arm.

Let's focus our attention on the only horizontal lead, that is, lead I, which uses a positive electrode on the arm.

Next, we will look at the only vertical lead, AVF, which uses a ______ electrode on the left leg ("Foot").

That was fast... let's move on.



the QRS complex is positive (mainly upright) in lead I, the Mean QRS Vector is pointing somewhere into the patient's left half (i.e., the positive half) of the sphere.

and I. complex in

QRS

We check the QRS complex because it represents

QRS in lead I is mainly upright,

(positive or negative)...

positive

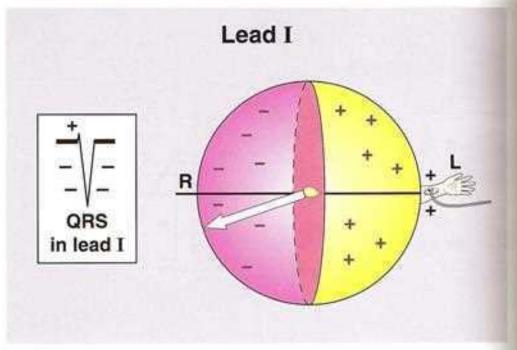
and if the QRS is positive in lead I, then the Mean QRS

ector points positively, that is, into the _____ half of the sphere

ward the positive skin electrode on the patient's left arm).

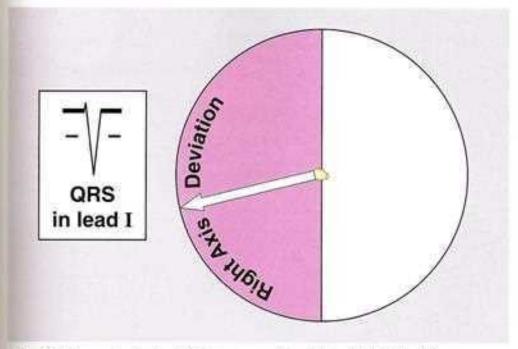
left

This point becomes clearer if you go back and reread the previous page and continue directly with this page. It comes into better on the second go 'round.



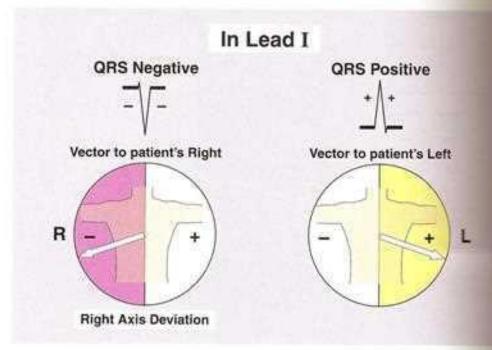
Still considering lead I on the tracing, if the QRS complex is mainly negative (downward), the Vector points to the patient's right side.

In lead I, if the QRS complex is mainly below the baseline, it is (positive or negative).	negative
Now checking the lead I sphere surrounding the patient, a Vector pointing into the negative half of the sphere points to the patient's side.	right
So if the QRS in lead I is mainly negative, then the Mean Vector points to the patient's right side (away from the positive electrode on the patient's left arm).	QRS



If the QRS is negative in lead I (Vector toward the right), this is Right Axis Deviation.

the Mean QRS Vector points toward the right, we expect the QRS complex in lead I to be	negative
the Mean QRS Vector points to the patient's right side the right of a vertical line drawn through the AV Node), his indicates Right Deviation.	Axis
Bis finiteates Right Deviation.	AXIS
so if the QRS complex is negative in lead, this indicates	Ī



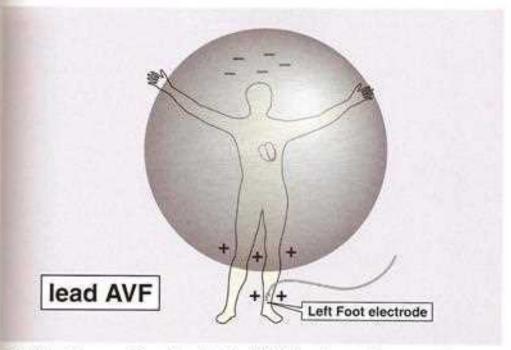
By simple observation, we can tell whether the Mean QRS Vector points to the patient's left or right side.

Lead I is the best lead for detecting Right ______

Deviation.

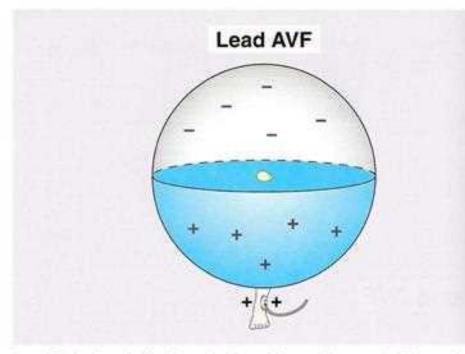
If the QRS complex is positive in lead I (which it usually is), this indicates that there is no R.A.D., because the Vector is pointing to the _____ side of the patient.

When we record lead I on an EKG, the patient's left arm has the ______ electrode.



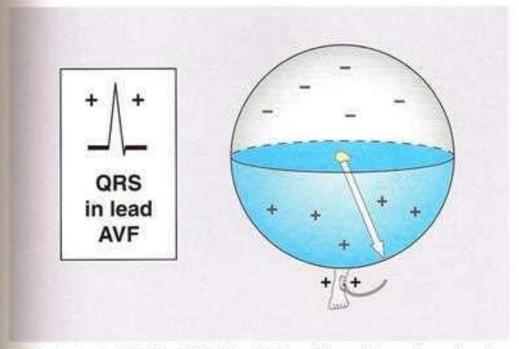
The left foot has a positive electrode in lead AVF. Imagine a sphere around the patient for lead AVF.

lgnore the lead discussed on the previous page. We are considering only lead at this time.	AVF
Note: We are now considering a completely different sphere – the of that surrounds the body when we record lead AVF on the EKG machine. We need to re-orient ourselves as to the positive and nega- halves of the sphere in AVF.	
When we switch the EKG machine to monitor lead AVF, the machine makes the electrode on the left positive.	foot
The lower half of this sphere is	positive
The center of this sphere is the	AV Node



For AVF the lower half of the sphere is positive, and the upper half is negative.

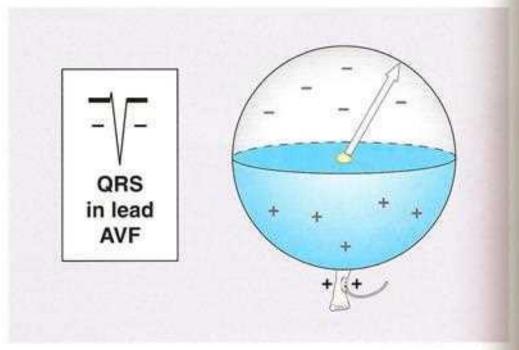
The lower half of the AVF sphere is the location of the positive left foot electrode, so we know that the lower half of this sphere is	position
The upper portion of the sphere (above the AV Node) is (positive or negative).	negative
The sphere in AVF has two halves, the upper half is	negative
and the lower half of the AVF sphere is	positive



Considering lead AVF of the EKG, if the QRS is mainly positive on the tracing, then the Mean QRS Vector points downward into the positive half of the sphere, toward the positive (lead AVF) electrode.

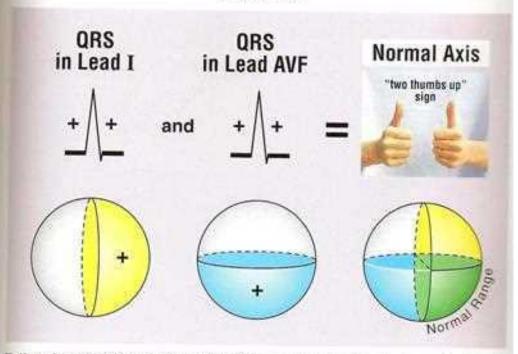
the Mean QRS Vector points downward, then
QRS complex in lead AVF is ______ upright (or positive)

Note: Don't get confused just because the positive QRS is upright, yet the Vector points downward. You must remember that the Vector points into the positive half of the sphere (toward the positive left foot electrode) when the QRS is positive. The lower half of the sphere just happens to be the positive half in lead AVF.



In AVF, if the QRS is negative, the Vector points into the negative half of the sphere.

The center of the sphere is the _		AV Node
The upper half of the (lead AVI	sphere is	negative
A negative QRS complex in lea Mean QRS Vector points	d AVF tells us that the into the negative	upwara
half of the sphere (i.e., it is point electrode on the left foot),	ting away from the positive	-



Follow the illustration closely. If the QRS is positive in lead I and also positive in AVF, the Vector points downward and to the patient's left. This is the normal axis range; the area that is both yellow and blue (yellow plus blue equals green).

A mainly positive QRS in lead I indicates that the Mean

QRS Vector points to the _____ side of the patient, and... lef

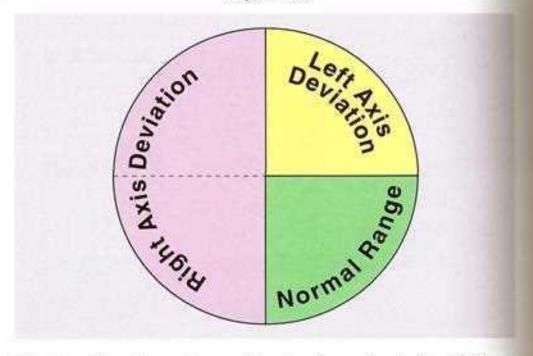
... a mainly positive QRS complex in lead AVF means
that the Vector points _____ downward

In the same patient, if the Vector points leftward and also points downward, the Vector must be in the only quadrant of the _____ that satisfies both criteria (and it happens to be the normal range).

πď

sphere

Note: Since the ventricles angle downward to the left, and ventricular depolarization moves downward and leftward, it should not surprise you that this is the normal range of the Vector. Remember, Vector position is stated in terms of the patient's left or right. If the QRS is upright in both lead I and AVF (the "two thumbs up" sign), the Vector ("Axis") is within the normal range.



In the frontal plane, there are four possible axis quadrants where the Mean QRS Vector may point. Visualize this large circle on the patient's chest in the frontal plane.

Note: In the frontal plane, we determine if there is any Deviation of Axis out of the normal range.

If the Vector points upward (from the AV Node) and to the patient's left, this is Left ______ Deviation (L.A.D.).

Axis

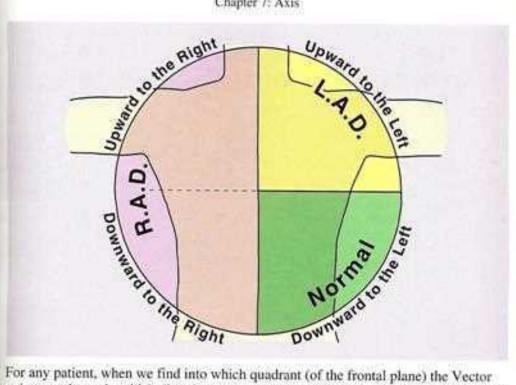
If the Vector points to the patient's right side, this is _____ Axis Deviation (R.A.D.).

Right

If the Vector points downward to the patient's left, it is in the _____ range (i.e., Normal Axis).

norm

Note: Remember, Axis is merely the position (that is, the direction) of the Mean QRS Vector, which indicates the general direction of ventricular depolarization.

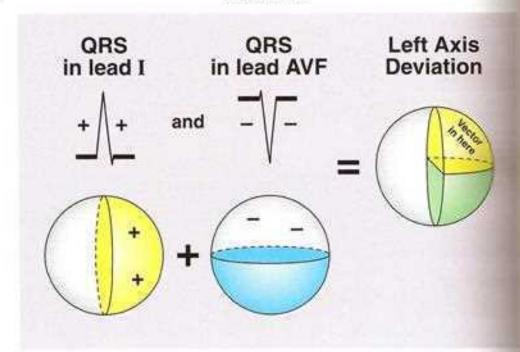


For any patient, when we find into which quadrant (of the frontal plane) the Vector points, we know in which direction the ventricular depolarization is going. The small type in the illustration relates to the patient's right or left.

Note: This is how you should visualize the four axis quadrants in a large circle (AV Node is the center) drawn on the patient's chest in the frontal plane. On some EKG charts the Mean QRS Vector is depicted in a similar circle (which represents the frontal plane).

The upper left quadrant represents Axis Deviation L.A.D.).

Left



If the QRS is positive in lead I, and negative in AVF, that places the Vector in the upper left quadrant. This is Left Axis Deviation.

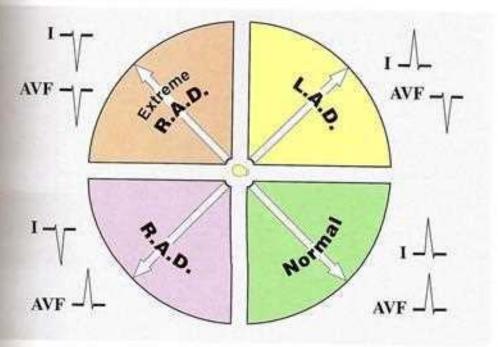
belie

27.0

If the QRS in lead I is upright, the Vector points to the patient's _____.

If the Vector is pointing upward, then the QRS in lead AVF is mainly ______ the baseline.

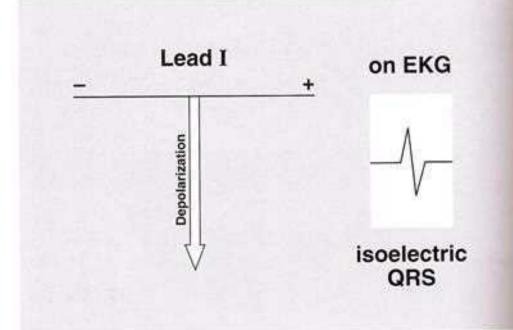
And when the Vector points upward and to the patient's left, this is Left _____ Deviation (L.A.D.).



Now, by looking at the QRS complex in I and AVF, you can locate the Mean QRS Wector in an Axis quadrant (in the frontal plane as it relates to the patient).

Axis Deviation (R.A.D.); and when the Vector points upward (and to the patient's right), this is commonly "Extreme" R.A.D.	Right
But if the QRS is positive in lead I and negative in lead AVF,	Deviation
So if the Mean QRS Vector points downward and to the patient's left, expect the QRS complexes in leads I and AVF to be usually (upright). And of course, they usually are, sace this is normal.	positive

Note: You also can calculate the vector for a portion of a QRS complex for instance, the initial or terminal .04 sec.) in exactly the same manner as for the Mean QRS Vector.



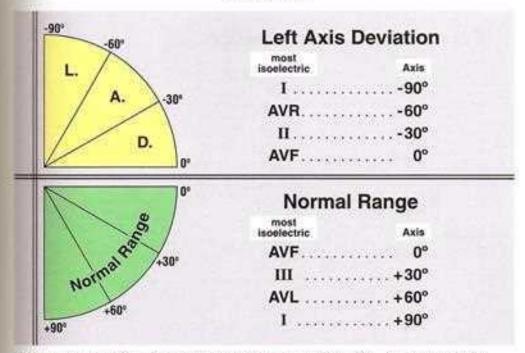
When depolarization moves in a direction perpendicular to the orientation of a lead, the deflection is minimal and/or "isoelectric." An isoelectric QRS records equal magnitudes of upward (positive) and negative (downward) deflection.

isoelec

a lead, is directed negligibly toward either electrode, so the recorded deflection is as much negative as positive and is called	
The word "isoelectric" literally means "same voltage," so it is used when the positive and the negative portions of the QRS complex are about	
Although the positive and negative deflections of an isoelectric QRS are equal in amplitude, they are generally small in the limb	

Depolarization that moves perpendicular to the orientation of

Note: First, locate the Mean QRS Vector in an axis quadrant (i.e., Normal, L.A.D., R.A.D., or Extreme R.A.D.). Then, find the limb lead in which the QRS is the most isoelectric, so you can more precisely locate the Vector in degrees (Axis). The Axis is about 90° from the orientation of the most isoelectric lead. It is really very easy... next page.



To locate the position of the Vector (Axis) more precisely (i.e., in degrees) in the frontal plane: <u>first</u> locate the axis quadrant, and <u>then</u> note the <u>limb lead</u> in which the ORS is most isoelectric.

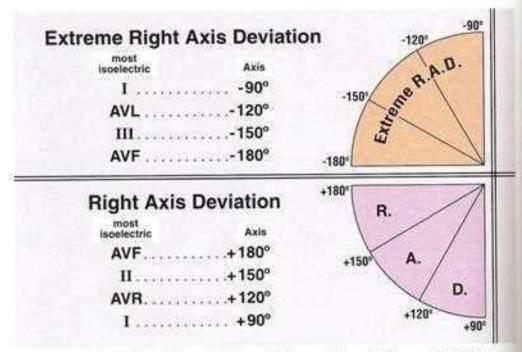
Note: Please refer to the illustration on this page (and the page that follows) to determine the exact position of the Mean QRS Vector (Axis) in degrees. For exams and in "real life" situations you need a reference. Accuracy is far more important than memory. You may copy page 340; it's yours for real life use.

Note: Let's review. First, locate the appropriate axis quadrant. Then, to determine the exact position of the Vector (Axis), find the lead where the QRS is most isoelectric. Refer to the illustration as you contemplate the hypothetical examples below.

A patient with Left Axis Deviation has a Mean QRS Vector
of between 0 and _____ degrees (QRS positive in 1 and _____ -90
segative in AVF). Check the illustration. (don't forget the negative)

A young lady has a Mean QRS Vector in the normal range. If the QRS in lead III is isoelectric, then she has an electrical axis of _____. Please don't proceed to the next page until you feel comfortable with this exercise.

+30°



The exact position of the Vector (Axis) can be located in a similar way for Right Axis Deviation and Extreme Right Axis Deviation. Refer back to the illustration for each sentence below.

Note: After the axis quadrant is determined, the limb lead with the most isoelectric QRS is noted.*

Consider a patient with R.A.D. You find that the QRS is isoelectric in lead II, so the Axis is _____.

+150

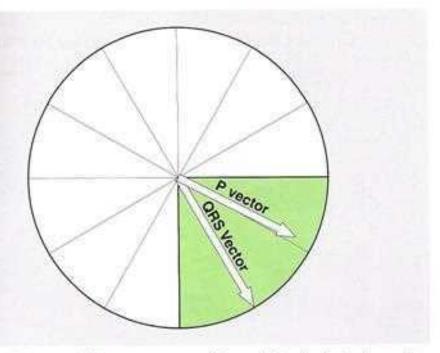
You have a patient with numerous widened, premature QRS's, and you need to know whether it is a PVC or an aberrant Junctional beat. The wide QRS is negative in I and AVF, which places its Vector in the Extreme ____ quadrant (how could that be?)...

RAD

... the wide QRS is also isoelectric in AVL, so its Axis is ____. For ventricular depolarization to progress in that direction, it must have originated in a focus (or pacemaker electrode) at the apex of the left ventricle, rather than from a Junctional focus. Let's think about that.

Note: An Axis of 180° is either + or - depending on whether the Vector is in the R.A.D. or Extreme R.A.D. quadrant respectively.

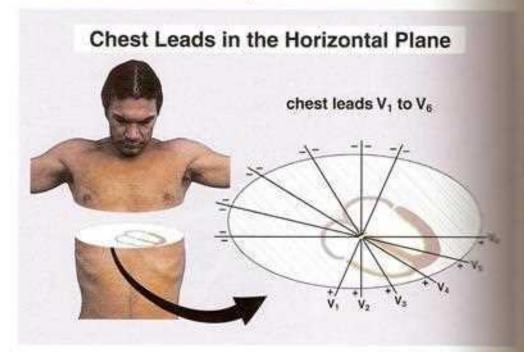
^{*} This is summarized for you (page 340) of your Personal Quick Reference Sheet.



The Mean QRS Vector, which represents normal biventricular depolarization, points downward and to the patient's left, The P wave vector, which represents normal matrial depolarization, points downward to the left side of the patient.

Note: The P wave vector points generally downward, toward the positive electrode on the patient's left foot (for *inferior* leads II, III, and AVF), so the P waves are usually upright in those leads. The P wave vector also points leftward, toward the positive electrode on the patient's left arm (for leads I, and AVL), producing generally upright P waves in those leads. So, if we see an inverted "P wave" in any of those leads, it is probably a P' depolarizing upwards from a low atrial focus, or retrograde atrial depolarization moving upward from the AV Node.

Note: Most PVC's emanate from a peripheral focus in a ventricular wall, depolarizing the ventricles in a general bottom upward direction, so they are usually mostly negative in the *inferior* and *lateral* limb leads where the QRS is usually upward. Exception: PVC's that are mainly upward like the QRS's in those leads, probably originate in a septal ventricular focus and follow a near-normal path.



The sphere has three dimensions, so it is important to note the general position of the Mean QRS Vector in the horizontal plane as well.

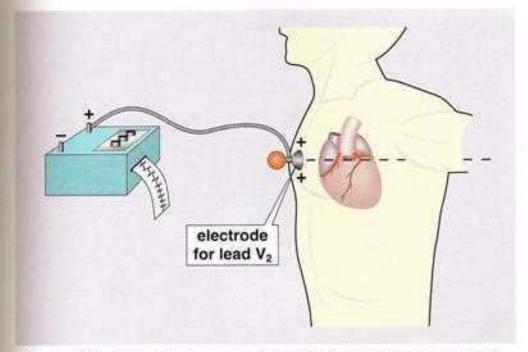
The horizontal _____ divides the body into top and bottom halves.

The chest leads form the ______plane.

hon

Note: To determine changes ("rotation") of the Mean QRS Vector in the horizontal plane, we examine the chest leads.

Note: Although the Axis may "deviate" in the frontal plane, the Vector is said to "rotate" in the horizontal plane. This is conventional (universally accepted) terminology used in communication and in medical literature.



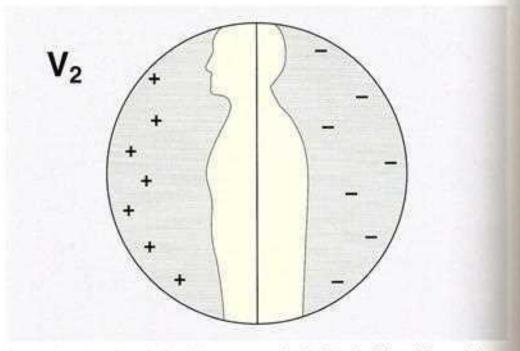
Chest lead V₂ is obtained by placing a positive electrode on the chest along the left side of the sternum (at the fourth interspace).

The chest elec-	trode used for recording lead V2 is	
always	(positive or negative).	positive

Note: The electrode for the chest leads is a suction cup that is moved to a different position on the chest for each of the six chest leads (which form the horizontal plane). In each case, the suction cup electrode is positive.

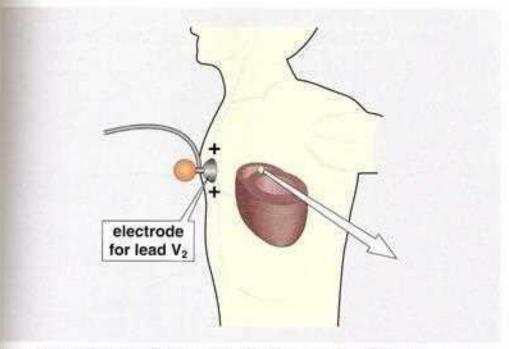
The position of the (suction cu	 p) electrode for recording lead V₂ 	
places it in front of the heart, a	t the fourth interspace to the left of	
the sternum, so it is just	to the AV Node.	anterior

Note: We already know that metal electrodes affixed with conductive gel are often used for recording the chest leads, so let's refocus on the conceptual material.



Considering the sphere for lead V_2 we can see that the front half is positive and the back half is negative.

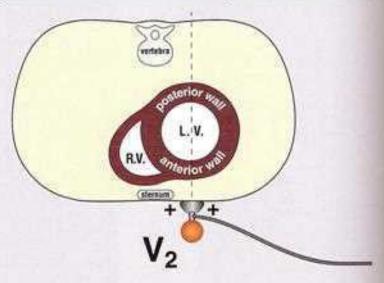
Considering a lead V ₂ sphere, we view the patient from the side. The center of the is still the AV Node.		sphere
The patient's back is when considering lead V ₂ .	(negative or positive),	negative
The front half of the sphere is _	in lead V ₂ .	positiva



Normally the QRS in lead V_2 is negative. Therefore, the Mean QRS Vector points backward, because of the (generally) posterior position of the thick left ventricle.

complex is usually (below the baseline).	negative
Therefore, the Mean QRS Vector usually points into the negative half of the sphere.	backward (posteriorly)
Normally, most of the ventricular depolarization moves away from the positive V ₂ electrode, toward the thicker and more posteriorly positioned ventricle.	left

Body Cross Section Through Ventricles



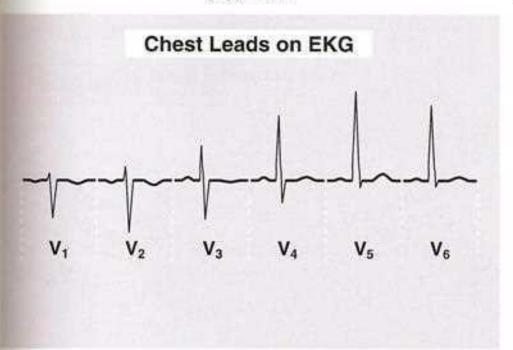
The orientation of chest lead V₂ makes it the most informative lead for the determination of both Anterior and Posterior Infarction.

The orientation of lead V₂ projects through the anterior wall and the posterior wall of the ______ventricle.

So V₂ reflects the most reliable information concerning Anterior Infarction and ______ Infarction of the left ventricle.

Poster

Note: As you will soon see, both ventricular depolarization and repolarization should be scrutinized in the right chest leads, because they reveal subtle vector changes caused by both anterior and posterior infarctions (of the left ventricle).



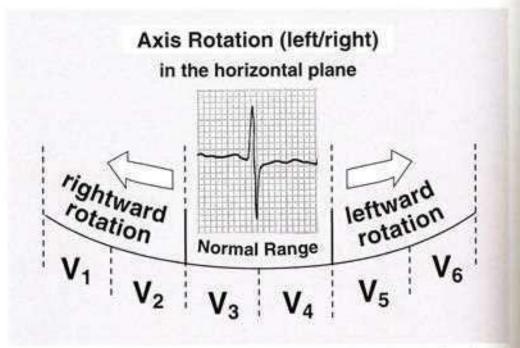
in the chest leads, there is a gradual transition from the generally negative QRS in V_1 to the generally positive (upright) QRS in V_6 .

in lead V ₆ .	positive
Examining chest leads V ₁ through V ₆ to observe the gradual	
becomes as much positive as negative or ""	isoelectric
lead V ₃ or V ₄ . This is the transitional zone.	

Note: You will recall that an isoelectric QRS is 90° away from the Mean QRS Vector. So a shift ("rotation") of the Vector in the horizontal plane is reflected as a similar change in position of the transitional" (isoelectric) QRS in the chest leads. You will better understand and appreciate this, when you see the next page.

RS is mainly negative in lead V and

the Vector changes its position (rotates) in the horizontal plane,	
See Vector's tail remains anchored to the	



Rotation of the Vector in the horizontal plane is described from the patient's point of view as "rightward" or "leftward." Check the chest leads for the isoelectric QRS.

Note: The Vector can rotate in the horizontal plane with its tail anchored to the AV Node. When the isoelectric ("transitional") QRS has rotated to the patient's right (into leads V₁ or V₂) this is rightward rotation. But if the transitional QRS is found in the patient's left chest leads, V₅ or V₆ this is leftward rotation. Anatomically speaking the heart is not capable of much rotation in the horizontal plane. But, we do know that the Vector shifts toward Ventricular Hypertrophy and away from Infarction.

Note: In older literature you may still see the terms "clockwise" (meaning leftward) rotation or "counterclockwise" (meaning rightward) rotation of the Vector in the horizontal plane. These terms have become obsolete since they do not relate well to clocks, and much confusion resulted.

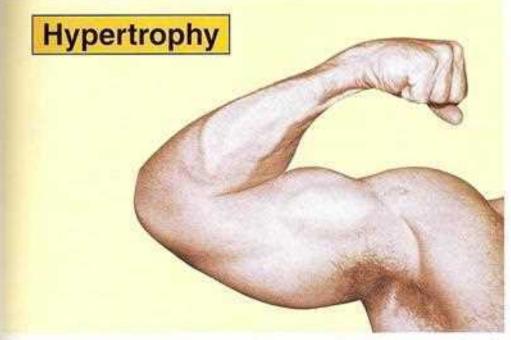
Reminder: Axis deviation is in the frontal plane.

Axis rotation is in the horizontal plane.

Note: Please observe the simplified technique for determining Axis by turning to page 340. A quick review of the methodology may be found in the Personal Quick Reference Sheets on page 334.

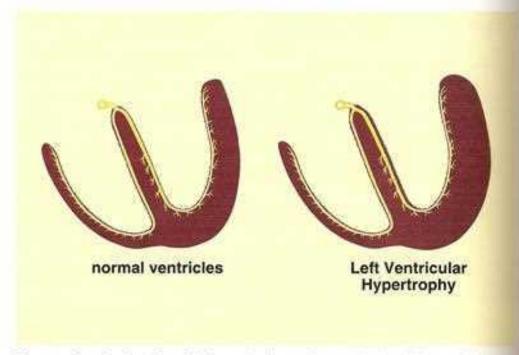
Chapter 8: Hypertrophy

Before you begin, look at this chapter's summary on pages 334 and 341.



Hypertrophy usually pertains to an increase in size, but when relating to muscle as in myocardium, this term refers to increase in muscle mass.

Note: The photo above is the arm of a weight-lifter. I had contemplated using a photo of my own arm, but I soon abandoned the idea because then I would have to title this section "hypotrophy" (if there is such a word).



Hypertrophy of a chamber of the heart implies an increase in the thickness of the chamber wall, but some dilation is always present also.

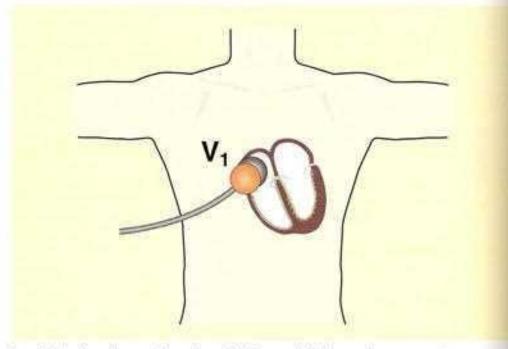
Hypertrophy of a chamber of the heart means that the muscular wall of that chamber has dilated and thickened	
beyond thickness.	nom
Hypertrophy may increase the volume that the contains, and the wall of that chamber is thicker than normal.	chami
The increase in the muscular thickness of the wall of a hypertrophic chamber, as well as dilation of a chamber of the heart may be diagnosed on	Đ

P wave

Since the P wave represents the depolarization and contraction of both atria, we examine the P wave for evidence of atrial enlargement. (See Note.)

The depolarization of both atria causes their simultaneous	contraction
The depolarization of both atria is recorded on EKG as a wave.	P
Signs of atrial enlargement can be detected by examining P wave on the twelve lead	EKG

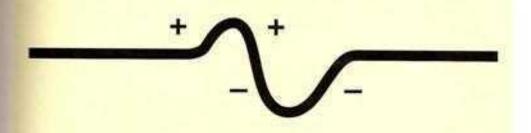
Note: Although the designation "atrial hypertrophy" is commonly used, enlargement of an atrium is usually due to dilation of the atrium. Therefore, the general term atrial enlargement is preferred, since it includes both dilation and hypertrophy. Whereas when referring to the ventricles, "ventricular hypertrophy" predominates.



Lead V_1 is directly over the atria, so the P wave in V_1 is our best source of information about atrial enlargement.

The chest electrode that records lead V ₁ is considered (positive or negative).	positive
When lead V ₁ is recording, the electrode is positioned just to the right of the sternum in the 4th interspace; this places the electrode directly over the	atria
Because the V_1 electrode is close to the atria, the P wave in lead V_1 gives us the most accurate information about atrial	enlargemen

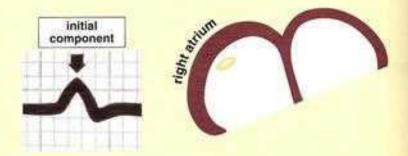
Diphasic P Wave



With atrial enlargement, the P wave is usually diphasic (both positive and negative).

called a	wave (two phased w	- CONTROL OF THE PROPERTY OF T	diphasic
A diphasic P was	ve has deflections above a	nd	baseline
	vave is characteristic of atr	ial enlargement,	atria

Right Atrial Hypertrophy



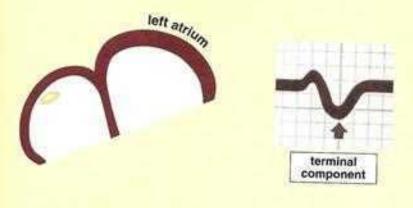
If the initial component of a diphasic P wave (in lead V₁) is the larger, then this is Right Atrial Enlargement.

If the P w	ave in lead V ₁ is, then we know	diposissi
that one o	of the atria is enlarged.	
If the initi	ial portion of the diphasic P wave is	
the	of the two phases, then there	large
is Right A	Atrial Enlargement.	

A diphasic P wave in V₁ with a large, often peaked, initial component tells us that this patient's ______ atrium is probably thicker and more dilated than the left.

Note: If the height of the P wave in any of the limb leads exceeds 2.5 mm (even if it's not diphasic), suspect Right Atrial Enlargement.

Left Atrial Hypertrophy



If the terminal portion of a diphasic P wave in V₁ is large and wide, there is Left Atrial Enlargement.

A patient who has enlargement of the left atrium, because the mitral valve is stenosed*, will have a diphasic P wave in lead	
	V
This patient's diphasic P wave in lead V ₁ has a small initial component and a larger component.	terminal
The terminal component of a diphasic P wave in lead V ₁ is usually (positive or negative).	negative

Mitral stenosis (narrowed mitral valve opening) can cause left atrial enlargement, but systemic hypertension is the most common cause.

Normal QRS in Lead V1



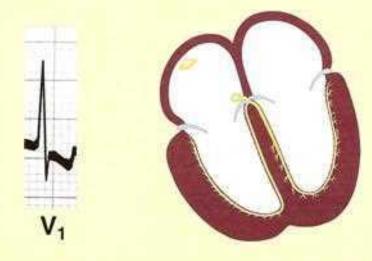
Now let's consider the QRS complex in V₁. Normally the S wave is much larger than the R wave in this lead.

so we would expect the QRS to reflect the presence of ventricular	hypertropie
venureurai	112 1200

In lead V₁ the QRS complex is mainly _____, and therefore the R wave is usually very short. negative

Note: The V₁ electrode is positive. Ventricular depolarization moves downward to the patient's left side and also posteriorly (the thicker left ventricle is more posteriorly located). Because ventricular depolarization is moving <u>away</u> from the (positive) V₁ electrode, the QRS in V₁ is usually mainly negative. Remember that the positive wave of depolarization moving toward a positive electrode records a positive deflection on EKG. By the same token, depolarization moving away from a positive electrode records negatively.

Right Ventricular Hypertrophy



However, with Right Ventricular Hypertrophy (RVH) there is a large R wave in V1.

Right Ventricular Hypertrophy there is a large wave in lead V1.

R

Note: With Right Ventricular Hypertrophy, the wall of the right ventricle is very thick, so there is much more (positive) depolarization (and more vectors) toward the positive V1 electrode. We would therefore expect the QRS in lead V1 to be more positive (taller) than usual.

The S wave in lead V₁ is smaller than the ___ wave

R

Right Ventricular Hypertrophy. (See illustration).

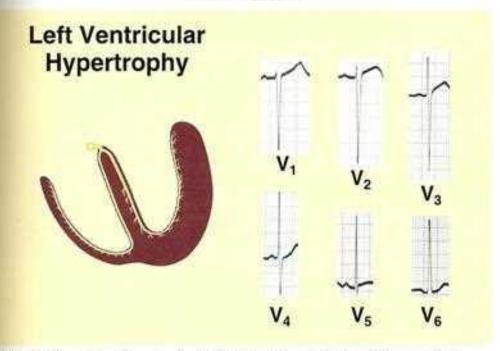
Right Ventricular Hypertrophy V1 V2 V3 V4

With Right Ventricular Hypertrophy, the large R wave of V_1 gets progressively smaller from V_2 to V_3 to V_4 etc.

When Right Ventricular Hypertrophy is present, there is a large R wave in lead ____ that becomes progressively smaller in chest leads V₂, V₃, and V₄.

The progressive decrease in the height of the ____ wave is gradual, proceeding from the right chest leads to the left chest leads.

Note: The enlarged right ventricle adds more vectors toward the right side, so there is Right Axis Deviation (in the frontal plane), and in the horizontal plane there is rightward rotation of the (Mean QRS) Vector. Visualize the reasons for these (Mean QRS) Vector shifts and the criteria will become very logical.



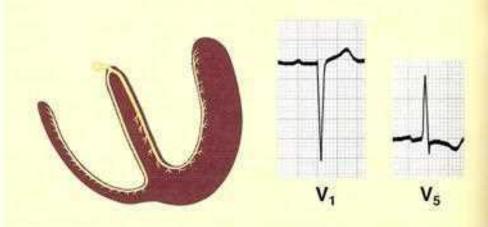
With Left Ventricular Hypertrophy (LVH), the left ventricular wall is very thick, causing great QRS deflections in the chest leads.

The heart chamber with the thickest muscular walls

s the ventricle.	left
Hypertrophy of the left ventricle produces QRS co	
especially in the leads.	chest

Note: Normally the S wave in V₁ is deep. But with Left Ventricular Hypertrophy, even more depolarization is going downward to the patient's left – away from the positive V₁ electrode. Therefore, the S wave is even deeper in V₁. There is Left Axis Deviation, and often the Vector is displaced in a leftward direction in the horizontal plane. Visualize and understand the reason for these shifts of the Vector. Lasting knowledge results from understanding.

Left Ventricular Hypertrophy



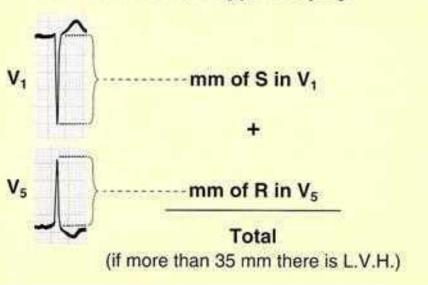
With Left Ventricular Hypertrophy there is a large S in V1 and a large R in V5.

With Left Ventricular Hypertrophy there is a very tall ____ wave in lead V₄

Note: Lead V_5 is over the left ventricle, so the increased depolarization is going toward the electrode of V_5 when there is L.V.H. This results in more (positive) depolarization going toward the (positive) electrode of V_5 which produces a very tall R wave in that lead.

In Left Ventricular Hypertrophy, there is a very tall R wave in lead ____, and this excessive depolarization moving away from the V₁ electrode produces a deep S wave in lead V₁.

Left Ventricular Hypertrophy



Depth (in mm) of S in V₁ plus the height of R in V₅... if greater than 35 mm, there is Left Ventricular Hypertrophy.

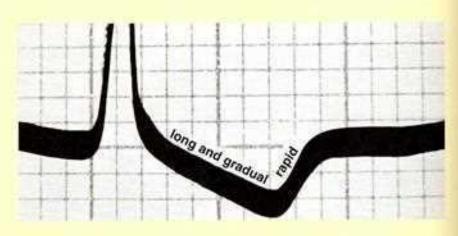
To check an EKG for Left Ventricular Hypertrophy, just add the depth of the S wave in V1 to the height of the wave in V5. R

If the depth (in mm) of the S wave in V, added to the reight (in mm) of the R wave in V₅ is greater than 35 (mm). ben _____ Ventricular Hypertrophy is present.

Left

Note: The sum of the S in V, plus the R in V, should be routinely checked (mere observation will usually suffice) with every twelve lead EKG. When providing a written EKG interpretation, however, one should measure and document the amplitude of these waves in millimeters.

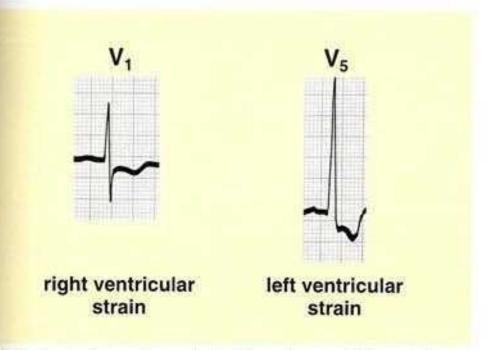
Left Chest Leads in LVH



inverted T wave

The T wave may show "Left Ventricular Hypertrophy" characteristics. Often there is T wave inversion with T wave asymmetry.

associated with Ventricular Hypertrophy.	Let
Since the left chest leads (V ₅ or V ₆) are over the left, these are ideal leads to check for this characteristic T wave that we find with LVH.	ventricle
With LVH, the inverted T wave has a gradual downslope and a very steep return to the, making it asymmetrical.	baseline

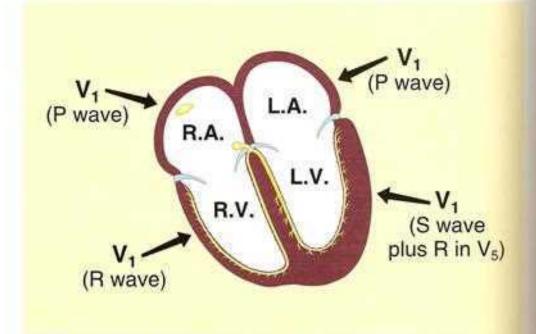


Wentricular hypertrophy may be associated with a strain pattern. With ventricular strain, the ST segment becomes depressed and humped.

Sentricular strain is characterized by depression	
of the ST	segment

Note: Strain is usually associated with ventricular hypertrophy, which is logical, since a ventricle that is straining against some kind of resistance (e.g., increased resistance from a narrowed valve or from hypertension) will become hypertrophied in its attempt to compensate.

entricular strain depresses the ST segment, which generally	
tumps upward in the middle of the	segmen



Note that lead V₁ provides most of the information concerning hypertrophy of the heart's chambers.

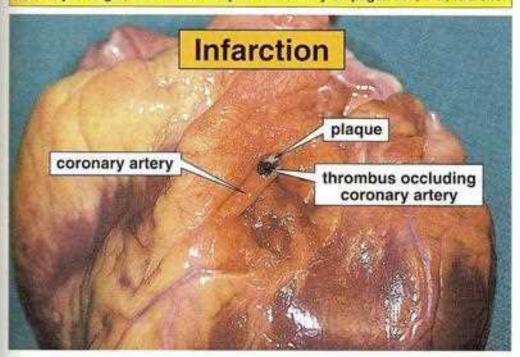
When routinely reading a 12 to see if there is	ead EKG, you should check of any of the chambers.	hypertrophy
First, check lead V ₁ to see if t	he P waves are	diphasia

Second, check the R wave in V_1 ... and then check the S wave in V_1 and the ___ wave in V_5 .

Note: You may now review *Hypertrophy* by turning to the Personal Quick Reference Sheets on page 341 and relate this to the simplified methodology that is summarized on page 334.

Chapter 9: Infarction (includes Hemiblock)

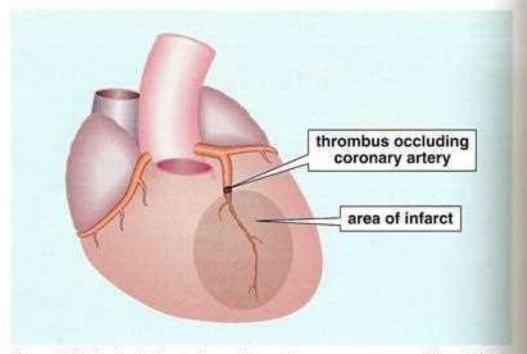
Before you begin, look at this chapter's summary on pages 334, 342, and 343.



Myocardial Infarction (M.I.) results from the complete occlusion of a coronary artery. The infarcted area of myocardium becomes necrotic (dead), so it can't depolarize or contract.

Note: Although the heart's chambers are filled with blood, the myocardium's own blood supply is provided exclusively by the coronary arteries. A coronary artery can be gradually narrowed by lipid deposits that become atheromatous plaque beneath the intima lining of the vessel. The intima may eventually rupture, exposing the plaque to the blood within the artery. This initiates the immediate formation of a clot (thrombus). The vessel, already narrowed by the plaque, becomes totally occluded by the thrombus. Instantly the infarcted area of the ventricle (without a blood supply) becomes necrotic. Ventricular foci in the hypoxic area around the infarct become very irritable; this can produce deadly ventricular arrhythmias.

Note: Myocardial Infarction implies the complete occlusion of a coronary artery, which we can diagnose with the EKG. The electrocardiogram will also tell us which coronary artery (or coronary branch) is occluded, and it can even reveal any blocks in the ventricular conduction caused by the infarction. By careful interpretation of the EKG, we can also determine if a coronary vessel is narrowed, rendering a decreased blood supply to the heart. Practical lifesaving knowledge. Let me show you...



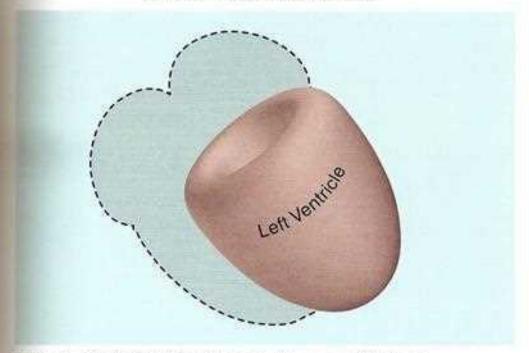
Myocardial infarction is due to the occlusion of a coronary artery supplying the leftventricle, so an area of the heart* is without a blood supply and suffers necrosis.

The terms "myocardial and "heart attack" refer to the	," "coronary occlusion," same serious problem.	infarction
경기 없으면서, 제 20~시간 [26] 이 경기 입에 있다면 하는데 (1.15) 모이 없었다며 다고!	od supply from the rtery or one of its major branches ocardium is without blood supply.	coronary

The infarcted (necrotic) area is primarily in the _____ ventricle; deadly arrhythmias may result.

Note: We understand that the coronary arteries also supply the right ventricle, so there is often some involvement of the right ventricle. But since most of the critical problems originate in left ventricular infarcts, myocardial infarction is usually conceptualized in terms of the left ventricle.

In this illustration, the pulmonary artery has been "surgically" removed to show the origin of the coronary arteries at the base of the aorta.



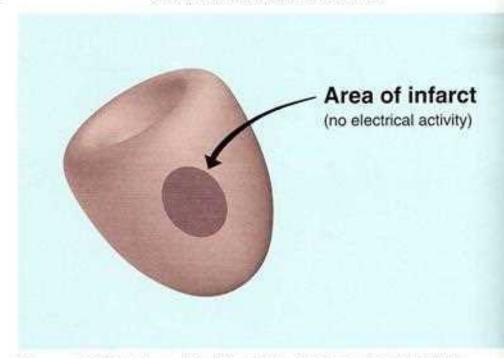
Commonly, it is the thick left ventricle that suffers myocardial infarction.

artery

Blood is pumped to all parts of the body by the powerful, bick, _____ ventricle.

left

Note: When we describe infarcts by location, we are speaking of an area within the left ventricle. Coronary arteries to the left ventricle usually send smaller branches to other regions of the heart, so an infarction of the left ventricle can include a small portion of another chamber.



The necrotic infarcted area of the left ventricle (that has no blood supply) is electrically dead and cannot depolarize.

left	of the wall of the	ventricle
An area of infarction cannot be de the cells there are without a are necrotic (functionally dead).	polarized because supply, so they	blood

Note: This necrotic infarcted area produces an electrical void, while the rest of the heart (with an adequate blood supply) functions as usual. The infarcted region does not depolarize, so it does not contract, thereby impairing the muscular function of the left ventricle. Also, hypoxic ventricular foci nearby are often the source of serious ventricular arrhythmias.

	Infarction
	• Ischemia
	• Injury
	• Necrosis
The myocardial infare he three may occur al	tion triad is "ischemia," "injury," and "necrosis," but any of one.
	th) of a ventricular region produces

dead myocardial cells that cannot depolarize.

The myocardial infarction triad is the basis for recognizing and diagnosing a _ infarction.

myocardial

The word hypoxia means decreased oxygen; in the heart it susually caused by ischemia, which literally means reduced supply (diminished blood flow).

blood

Note: Ischemia, injury, and necrosis need not all be present at once in order to establish the diagnosis of myocardial infarction. Routine EKG interpretation requires checking these infarction criteria.

Ischemia: reduced blood supply



investor

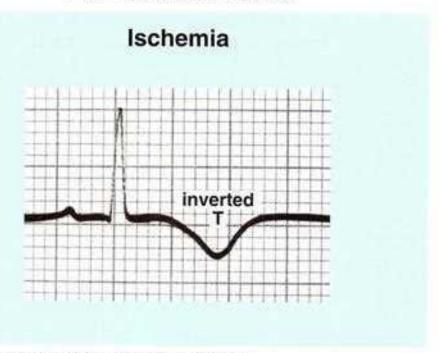
Ischemia (decreased blood supply) is characterized by inverted T waves,

Ischemia means reduced _____ supply (from the coronary arteries); the ischemic area is at the periphery of the infarct.

The characteristic sign of ischemia is the ______
T wave. It may vary from a slightly inverted to a deeply inverted T wave.

Inverted ___ waves may indicate ischemia in the absence of myocardial infarction. Coronary blood flow can decrease without producing an infarction.

Note: Cardiac ischemia alone can cause chest pain known as angina, which is usually associated with transient T wave inversion.



The typical ischemia T wave is symmetrically inverted.

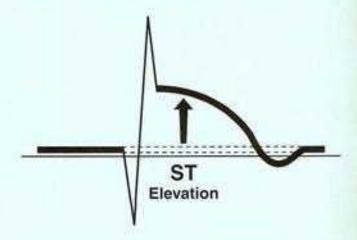
Note: You should check every EKG that you read for T wave inversion. Since the chest leads are nearest the ventricles, T wave changes are most pronounced in these leads. Always run down V₁ to V₆ (as well as the limb leads) and check for T wave inversion to see if there is diminished coronary flow.

The T wave of ischemia is both inverted and
; that is, the right and left sides
the inverted T wave are mirror images.

symmetrical

Note: In adults flat (nonexistent) T waves or minimal T wave inversion may be a normal variant in any of the limb leads (frontal plane). However, any T wave inversion in leads V₂ through V₆ is considered pathological. Marked T wave inversion in leads V₂ and V₃ the hallmark Wellens syndrome, alerts us to stenosis of the anterior descending acronary.

Injury: means acute or recent



Injury indicates the acuteness of an infarct. Elevation of the ST segment denotes "injury" sometimes called the "current of injury."

Note: "Acute" means recent or new.

The ST segment is that section of baseline between the QRS complex and the ____ wave. The ST segment contains no waves.

Elevation of the ST segment signifies "injury," The ST segment may be elevated only slightly, or as much as ten or more millimeters above the

MWS AU

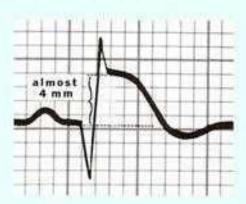
ST segment elevation tells us that a myocardial infarction is ______. It is the earliest consistent sign of infarction to record on EKG.

acu

haseSime

Note: Angina without exertion, "Prinzmetal's" angina, can cause transient ST elevation in the absence of an infarction.

ST Elevation



If there is ST elevation, this indicates that the infarction is acute. ST elevation, alone, can indicate an infarction.

Note: Once you have made a diagnosis of infarction, it is important to know whether the infarction just occurred and needs immediate treatment, or if the infarction is old — maybe years old.

rises above the baseline with an acute starction, in fact it is usually the earliest EKG sign of an afarction. With time, the ST segment returns to the baseline.

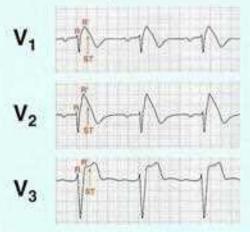
segment

Note: If the ST segment is elevated without associated Q waves, this may represent non-Q wave infarction, which is usually a small infarction that may herald an impending larger infarct. Significant ST changes require enzyme studies and close scrutiny.

Note: A ventricular aneurysm (the outward ballooning of the wall of a ventricle) can cause persistent ST elevation in most of the chest leads; but in this case, the ST segment does not return to the baseline with time. Pericarditis (next page) produces a unique type of ST segment elevation that may also elevate the T wave off the baseline.

Brugada Syndrome

- RBBB pattern QRS with ST elevation in V₁ V₃
- sudden cardiac arrest (in absence of coronary obstruction)



Brugada syndrome is a hereditary condition that can cause sudden death in individuals without heart disease. It is characterized by Right Bundle Branch Block with ST elevation in leads V₁ to V₃. Look for it; this malady is not rare.

Sudden cardiac death (cardiac arrest) can occur spontaneously in patients with Brugada

SYD

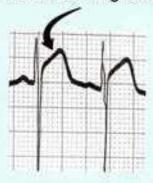
In Brugada syndrome there is RBBB and ST elevation in leads V₁ to V₃. The elevated ____ segments have a peculiar, peaked downsloping shape, particularly in V₁ and V₂.

Note: Brugada syndrome is a familial condition caused by dysfunctional cardiac Na⁺ (sodium) channels. Prophylaxis against the deadly arrhythmias requires ICD implantation in order to immediately treat cardiac arrest (usually ventricular fibrillation.)

Note: This syndrome is responsible for nearly one-half of the sudden deaths in healthy young individuals without structural heart disease.

Pericarditis

flat or concave elevated ST segment



ST segment and T wave elevated off baseline (dashed line)



With *pericarditis*, the ST segment is elevated and usually flat or concave. The entire T wave may be elevated off the baseline.

Note: Pericarditis is inflammation of the membrane (pericardium) that surrounds the heart. Pericarditis may be caused by a virus, bacteria, cancer, or other sources of inflammation, including myocardial infarction.

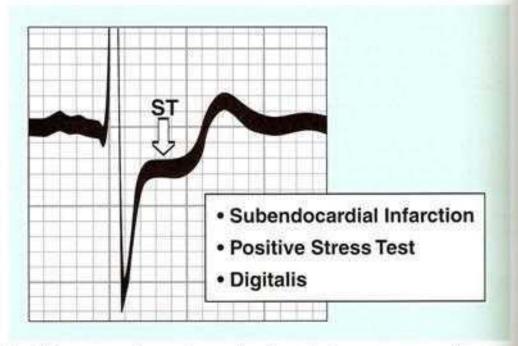
Pericarditis can elevate the ____ segment. It usually produces = elevated ST segment that is flat or slightly concave = addle sags downward). This resolves with time.

ST

Pericarditis seems to elevate the entire __ wave off the baseline; that is, the baseline gradually angles back down (often including the P wave) all the way to the next QRS (illustration on right).

T

Note: The characteristics shown in the left illustration are found in leads in which the QRS is usually mainly negative (like the right chest leads). The pattern shown in the right illustration is seen in leads where the QRS is mainly positive (such as the lateral and inferior limb leads). Sometimes PVC's are produced.



The ST segment may become depressed under certain circumstances or conditions.

Note: During an angina* attack, the ST segment may be temporarily depressed.

A subendocardial infarction, an infarct that does not extend through the full thickness of the ______ ventricular wall, will depress the ST segment.

left

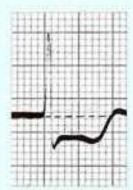
When a patient with narrowed coronaries exercises, the myocardium demands more blood flow than its arteries can deliver. A stress (or "exercise") test will record depression of the ____ segment on EKG when such a patient is exercised.

SI

Digitalis can cause ______ of the ST segment, however it has a unique, unforgettable appearance (see page 317). depression

Chest pain caused by diminished coronary blood flow (without infarction).

Subendocardial Infarction



(flat) ST depression

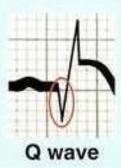
Subendocardial infarction causes flat depression of the ST segment; however, any significant ST depression (in leads where the QRS is upright) indicates compromised coronary blood flow until proven otherwise.

Subendocardial infarction (often referred to as subendocardial injury) is identified by flat ST ______ depression, which may be either horizontal or down-sloping.

Note: Subendocardial infarction, a type of "non-Q wave infarction" involves only a small area of myocardium just beneath the endocardial lining. Classical myocardial infarction is said to be transmural; that is, the full thickness of the left ventricular wall is damaged in the infarcted area. Even though subendocardial infarction involves only a small area of the myocardium, it must be respected as a true M.I. that requires appropriate care. A subendocardial M.I. may enlarge or extend and become more life-threatening.

Note: Any patient with acute ST depression (or elevation), particularly if it persists, should have an immediate, complete workup including cardiac enzymes.

Necrosis: dead tissue



(diagnostic for infarction)

The Q wave indicates necrosis (dead tissue) and makes the diagnosis of infarction.

The diagnosis of myocardial infarction is usually based on the presence of significant ____ waves produced by an area of necrosis in the wall of the left ventricle.

Note: The Q wave is the first downward stroke of the QRS complex, and it is never preceded by anything in the complex. In the QRS complex, if there is any positive wave — even a tiny spike — before the downward wave, the downward wave is an S wave (and the upward wave preceding it is an R wave).

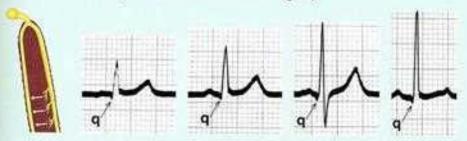
Significant Q _____ are absent in normal tracings.

We use a capital "Q" to designate a significant Q wave, however "q" (small, lower case q) waves are not significant (see next page).

Water

insignificant q waves

The earliest ventricular depolarization is initiated by Left Bundle Branch fibers at mid-septum (and moves left-to-right)...



... producing tiny q's in some leads.

Normally, ventricular depolarization begins midway down the interventricular septum. Septal depolarization (initiated at mid-septum by the Left Bundle Branch) is left-to-right, and this <u>initial</u> rightward ventricular activation may produce tiny, insignificant q (small q) waves in leads where the QRS is usually upright.

The Right Bundle Branch traverses the septum vertically without branching, however the _____ Bundle Branch gives off terminal Purkinje filaments at mid-septum.

Left

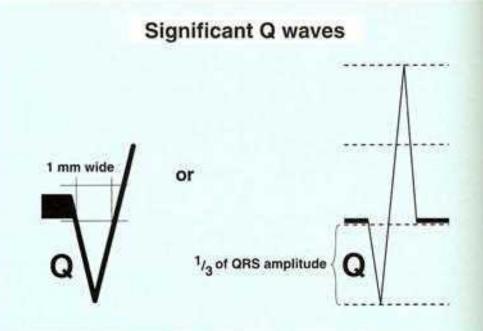
So this initial mid-septal depolarization moves left-to-right, away from:

- · the positive left arm electrode of lateral leads I and AVL, and...
- the positive left foot electrode of inferior leads II, III, and AVF, and...
- the positive chest electrode of left chest leads V₅ and V₆...

... to occasionally record tiny, insignificant __ waves in those leads.

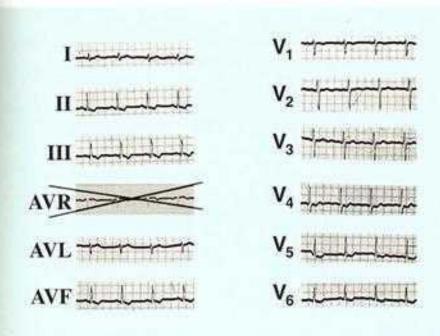
Note: This mid-septal depolarization is brief since the efficient ventricular conduction system quickly transmits depolarization to the endocardial surface of both ventricles. So brief is this mid-septal depolarization that only a tiny q wave of less than .04 second is recorded. Insignificant q waves are, by definition, less than one millimeter (.04 sec.) in duration.

q



A significant Q wave is at least one small square wide (.04 sec.) or one-third of the entire QRS amplitude. Significant Q waves indicate the necrosis of a myocardial infarction.

Significant Q waves are indicative of the necrosis of a myocardial	infarction
A significant Q wave is one small square (one millimeter) or more wide, and therefore is at least second or more in duration.	.04
An old, but persistent, criterion of the significant Q wave is when the Q wave is the amplitude (height and depth) of the entire QRS complex.	one-third



When looking at an EKG tracing, note which leads have significant Q waves. Omit lead AVR. Keep in mind the leads that make up the *lateral*, *inferior*, and *chest* lead designations.

To check for an infarction,	we scan all leads (except AVR)	
for the presence of	Q waves:	significant

Note: Forget about lead AVR, since this lead is positioned in such a way that data regarding Q waves are unreliable. Lead AVR is like an upside-down lead II, so the large Q waves that are commonly seen in lead AVR are really the upside-down R waves from lead II. Even if you don't understand the logic behind AVR's phony Q's, don't bother to check it for signs of infarction.

When examining a tracing, either a long strip or mounted, write down those _____ in which you find significant Q* waves, ST segment elevation (or depression), and inverted T waves.

leads

^{*} For proper documentation, insignificant q waves should be recorded as well.

Left ventricular depolarization moves in opposite directions (simultaneously) in opposing walls

Left Ventricle sagital section



Left Ventricle top view



Purkinje fibers conduct so rapidly that depolarization is initiated in all endocardial surfaces lining the left ventricle nearly simultaneously. So, depolarization passes from endocardium to epicardium in all left ventricular areas at once.

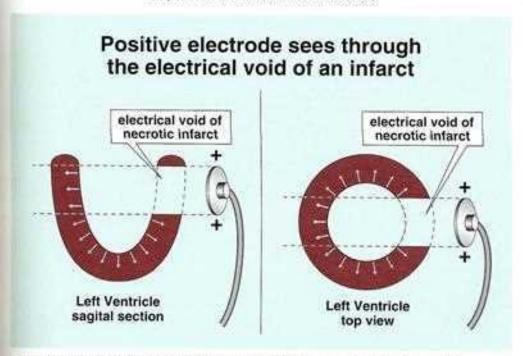
Note: Vectors describe the path of myocardial conduction (endocardium to epicardium), so left ventricular depolarization moves in opposite directions in opposing walls simultaneously.

In the left ventricle, depolarization of the lateral wall moves toward the patient's left, while depolarization of the medial (septal) wall moves toward the

right

Depolarization of the anterior left ventricular wall moves anteriorly, while simultaneously, depolarization of the posterior left ventricular wall moves in a ______ direction.

posterior



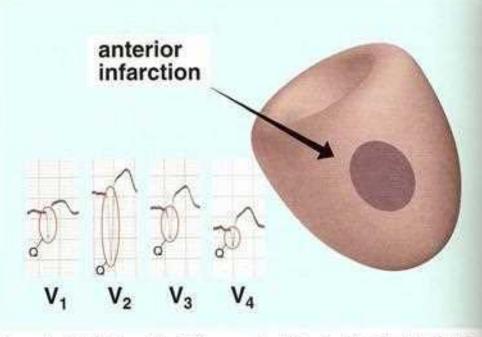
An infarct is necrotic; it cannot depolarize and has no vectors. So, the **positive** electrode nearest the infarct detects no "toward" vectors, it sees only the "away" vectors from the opposite wall (through the necrotic void). Therefore, a Q wave is inscribed on EKG in the leads which use that positive electrode for recording.

Note: Depolarization moving <u>away from</u> a positive electrode records a <u>negative</u> wave (in this case a Q wave) on EKG.

Note: Take your time and visualize each sentence as you read it.

In recording the initial left ventricular depolarization:

- with an anterior infarct, the positive (chest) electrode detects only the initial "away" vectors from the opposite side, so a Q is inscribed on EKG in leads V₁ - V₄ which use that positive electrode for recording.
- with a lateral infarct, the positive left arm electrode detects only the initial "away" vectors from the opposite side, so a Q is inscribed on EKG in leads I and AVL, which use that positive electrode for recording.
- with an inferior infarct, the positive left foot electrode detects only the initial "away" vectors from the opposite side, so a Q is inscribed on EKG in leads II, III, and AVF, which use that positive electrode for recording.



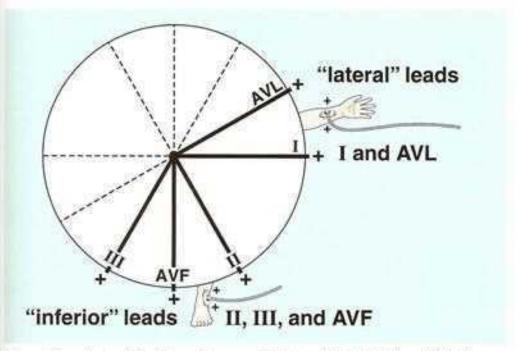
Q waves in V₁, V₂, V₃, or V₄ signify an anterior infarction. The infarction in the illustration is definitely acute, because of the ST elevation in all four leads.

Note: The chest leads are mainly placed anteriorly, so this is a good way to remember the leads for anterior infarction.

The presence of Q waves in V₁, V₂, V₃, or V₄ indicates an infarction in the anterior wall of the ______ ventricle.

Note: The anterior portion of the left ventricle includes part of the interventricular septum. Some cardiologists say that when isolated Q waves appear in V₁ and V₂, the infarction includes the septum, so it is called an antero-septal infarction. Similarly, isolated Q waves in V₃ and V₄ (more laterally located chest leads) are said to represent an antero-lateral infarction. Remember that (insignificant) q waves are seen normally in V₅ and V₆.

Note: Statistically, anterior infarctions are very deadly, but fortunately, immediate treatment with intravenous thrombolytic medications or angioplasty with stenting has improved the survival rate substantially.



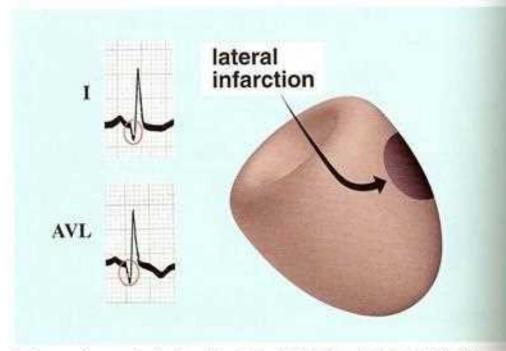
The positive electrode that is used to record the *lateral* limb leads, I and AVL, is on the left arm. The positive electrode used to record the *inferior* limb leads, II, III, and AVF, is on the left "foot."

The lateral limb leads are I and AVL; they are recorded	
by a positive left arm	electrode

Yawn... excuse me.

The inferior leads are II, III, and AVF; they are recorded by a positive electrode on the left ______. foot

Note: Yes, it is necessary to have this page here. You'll see why in just a few seconds.



If there are Q waves in the lateral leads, I and AVL, there is a lateral infarction.

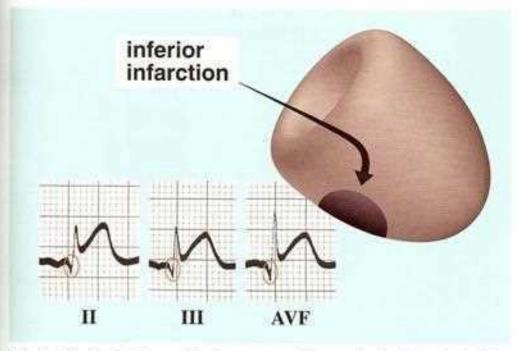
Note: Depolarization moving <u>away from</u> a positive electrode records a <u>negative</u> wave (in this case a Q wave) on EKG.

A lateral infarction involves the lateral portion of the _____ ventricle.

In lateral infarction, the positive left arm electrode senses only the initial "away" vectors from the opposite (septal) wall, so it records a Q wave in the lateral ______, I and AVL.

When a lateral infarction occurs, Q waves appear in the lateral leads, which are leads I and ____; Q waves are produced by the initial "away" vectors recorded by the positive left arm electrode through the void of the necrotic lateral infarct.

Note: One might abbreviate Lateral Infarction as L.I., which is diagnosed using leads AVL and I.



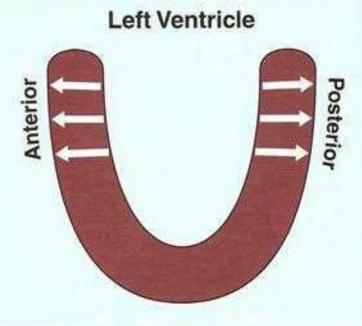
Inferior infarction is diagnosed by the presence of Q waves in the inferior leads, II, III, and AVF. Check the ST segments to see if this infarction is acute.

Note: Depolarization moving <u>away from</u> a positive electrode records a <u>negative</u> wave (in this case a Q wave) on EKG.

The inferior wall of the left ventricle rests upon the diaphragm, so the alternate term "diaphragmatic" infarction is occasionally	No. Contractor Contrac
used instead of "inferior"	infarction
In inferior infarction, absent the initial "toward" vectors, the positive left foot electrode senses only the initial "away"	
vectors from the opposite wall, so it records a Q wave in the	
inferior, II, III, and AVF.	leads
An inferior infarction is identified by significant Q waves	
in inferior leads II III and O waves are produced by	A 3712

Note: Autopsy data show that about one-third of inferior infarctions also include portions of the right ventricle.

the initial "away" vectors recorded by the positive left foot electrode through the void of the necrotic inferior infarct.



Depolarization of the anterior wall and depolarization of the posterior wall of the leftventricle are in opposite directions.

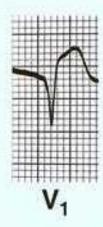
Note: Left ventricular depolarization may be said to proceed from the endocardium (inner lining) to the epicardium (outer surface).

Depolarization of the anterior wall of the left ventricle

proceeds from the inner endocardium, which lines the ventricle, through the full thickness of the ventricular wall to the outer ventricular surface ().	epicardium
Similarly, depolarization of the posterior wall of the ventricle proceeds from the endocardium to the epicardium.	left
So, vectors representing depolarization of the anterior and the posterior portions of the left ventricle point in directions.	opposite

Acute Anterior Infarction

(note ST elevation)





If an acute anterior infarction produces Q waves and ST elevation in V₁ and V₂ then a posterior infarction would appear the opposite.

An acute anterior infarction produces significant Q waves with ST in the first few chest leads.

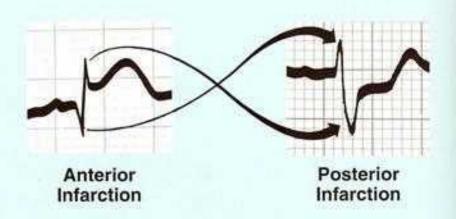
elevation

Considering only V₁ and V₂ the appearance of significant Q waves and ST elevation indicates an acute ______ infarction.

anterior (antero-septal)

Note: Acute posterior infarction of the left ventricle would produce the exact opposite to the pattern of acute anterior infarction, because the anterior and posterior walls of the left ventricle depolarize in opposite directions. This will be clarified on the next page.

Lead V₁ or Lead V₂



In acute Posterior Infarction there is a large R wave (the opposite of a Q wave) in V_1 and V_2 .

Note: In lead V₁ a Q wave turned upside-down would look like an R wave (and as you recall, R waves in lead V₁ are normally very tiny).

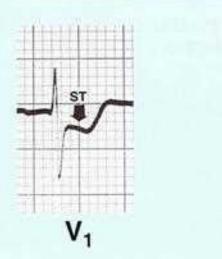
A significant "Q wave" from an infarction in the posterior portion of the _____ ventricle will cause a large R (positive deflection) wave to appear in lead V₁.

Suspect a true posterior infarction when you see a large $_$ wave in V_1 or V_2 — even though Right Ventricular Hypertrophy can also produce a large R in V_1 .

lett.

R

Acute Posterior Infarction



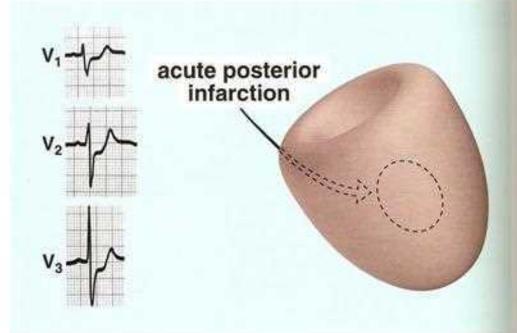


In acute posterior infarction, there is ST depression (the opposite of the usual ST elevation of Injury) in V_1 or V_2 .

Acute anterior infarction produces Q waves in the chest leads and the ST segments are ______.

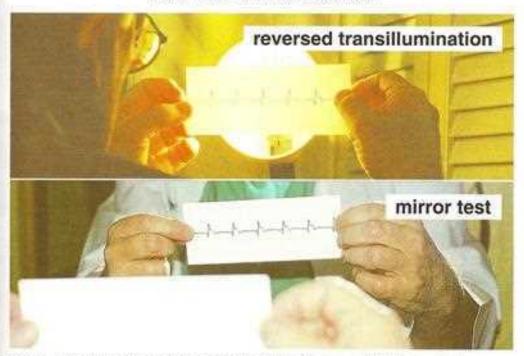
elevated

Note: Since the posterior wall of the left ventricle depolarizes in a direction opposite to that of the anterior wall, an acute infarction of the posterior wall causes ST depression in V₁ or V₂.



In summary, acute posterior infarction is characterized by a large R wave and ST depression in V_1 or V_2 (sometimes even in V_3).

Note: Always be suspicious of ST segment depression in the right chest leads, for it could indicate an acute posterior infarction. If you do not remember those things that can cause ST depression, look back at page 270. For instance, the diagnosis of an "anterior subendocardial infarction" (because of depressed ST segments in chest leads, see page 271) should be made only with extreme caution, because this ST depression may actually represent an acute true posterior infarct.



If you suspect an acute posterior infarction (large R wave and ST depression in V_1 or V_2), then try "reversed trans-illumination" or the "mirror test." You must follow the instructions for each test precisely.

Note: If acute posterior infarction is suspected because of tall R waves and ST depression in V₁ or V₂ — try reversed trans-illumination or the mirror test. Both of these tests require that you invert the tracing first, then hold the blank (unprinted) back side towards your face.

- Reversed trans-illumination: First, invert the EKG tracing, then hold the inverted tracing so that it faces a strong light. Observe the back side of the tracing to check for "Q waves and ST elevation" in the inverted V₁ and V₂ leads.
- Mirror Test: First, invert the EKG tracing, then observe it in a mirror. If there is an acute posterior infarction, you will see the classic signs of "Q waves and ST elevation" in the reflection of the inverted V₁ and V₂ leads.

Note: With either test, remember to first *invert the tracing*. Then face the tracing toward a mirror for the mirror test; or for reversed transillumination, place the tracing in front of a strong light, viewing the EKG through its back side.

Always Check V₁ and V₂ for:

- 1. ST elevation and Q waves (Anterior Infarct)
- 2. ST depression and large R waves (Posterior Infarct)

Although posterior infarctions are severe, they are easy to overlook.

When making your routine reading of an EKG, pay special attention to leads V₁ and ___ while looking for signs of infarction.

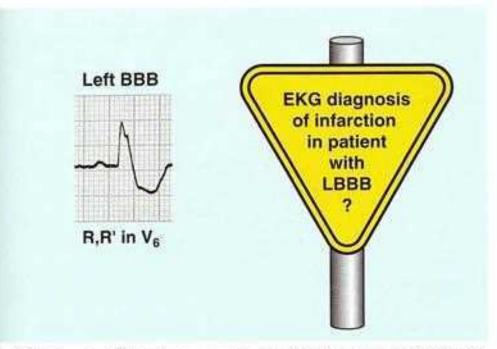
٧.

Note: ST changes in V₁ and V₂ are always significant and important... both depression and elevation.

Check for Q waves in V₁ and V₂ and be sure to observe the height of the __ waves.

R

Note: And remember how important T wave inversion can be in all leads.



The EKG diagnosis of infarction is generally not valid in the presence of Left Bundle Branch Block.

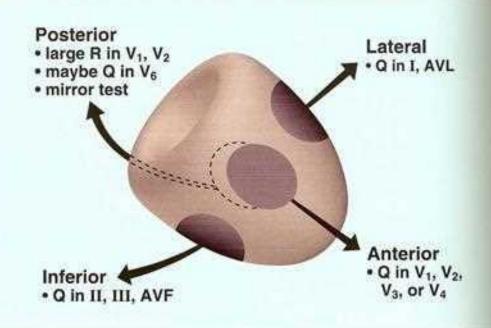
In Left Bundle Branch Block, the left ventricle (generally, the main chamber to suffer infarction) depolarizes after the ventricle depolarizes.

right

So any Q wave originating in the left ventricle could not appear at the beginning of the QRS ______ (with Left BBB); rather, it would fall somewhere in the middle of the QRS complex. In this instance it would be difficult to detect significant Q waves.

complex

Note: One special exception is possible. The right and left ventricles share the interventricular septum in common. So an infarct in the septal area would be shared by the right ventricle, which depolarizes first in Left BBB. This would produce Q waves at the beginning of the wide QRS. Therefore, even in the presence of Left BBB, Q waves in the chest leads might suggest (but not confirm) septal (anterior) infarction.



Locating an infarct is important because treatment modalities and prognosis depend on the location of the infarction.

There are four general locations within the _____ ventricle where infarctions commonly occur.

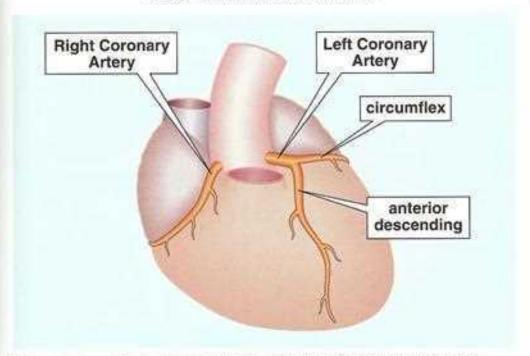
left

Note: More than one area of the left ventricle may infarct. One infarction may be very old, while another is very recent (acute). So correlate the ST elevation with the appropriate leads both to locate, and to determine the acuteness of each infarct. If ST elevation is present in leads without Q waves, "non-Q wave infarction" must be ruled out.

Be careful about diagnosing an infarction in the presence of ______ Bundle Branch Block.

Lett

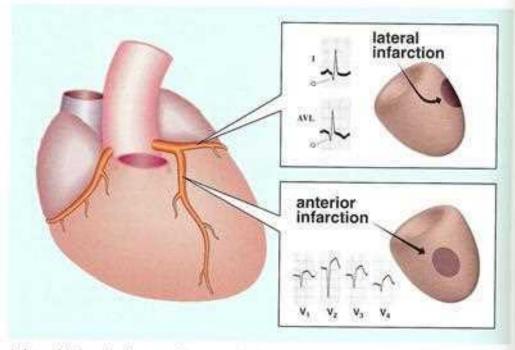
Note: Isolated areas of Ischemia (T wave inversion) or ST elevation without Q's (for non-Q wave infarction) can also be "located" by using the same location criteria.



It is common practice to determine the location of an infarction, but with a little anatomical knowledge of the heart's coronary blood supply*, we can make a far more sophisticated diagnosis.

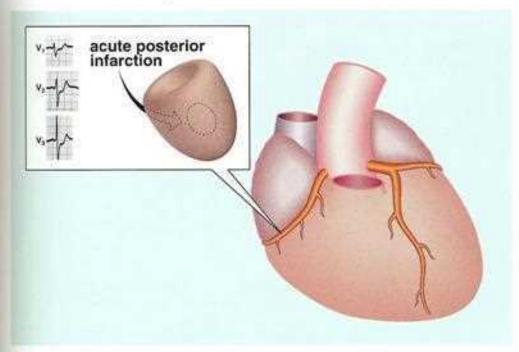
There are two coronary arteries th continuous supply of oxygenated	at provide the heart with a	blood
Quickly review the illustration. The Left Coronary Artery has two Circumflex branch and the	major branches; they are the Descending branch.	Anterior
The Right Coronary Artery curve	s around the right	ventricle

^{*} The pulmonary artery has been "surgically" removed in this illustration to show the origin of the coronary arteries at the base of the aorta.



A lateral infarction is caused by an occlusion of the Circumflex branch of the Left Coronary Artery. An anterior infarction is due to an occlusion of the Anterior Descending branch of the Left Coronary Artery.

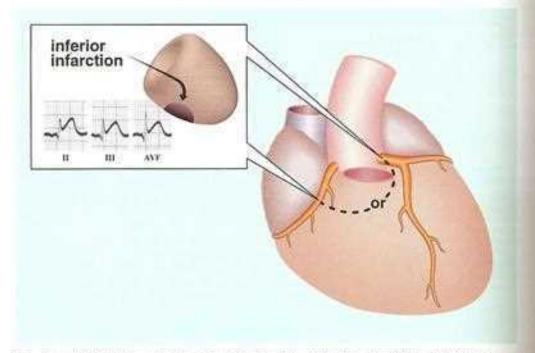
The Circumflex branch of the Left Coronary Artery distributes blood to the portion of the left ventricle.	lateral
The Anterior Descending branch of the Left Coronary Artery supplies blood to the anterior portion of the ventricle.	left
The Circumflex and the Anterior Descending are the two main branches of the Coronary Artery.	Left



True posterior infarctions are generally caused by an occlusion of the Right Coronary Artery or one of its branches.

The Right Corona	ry Artery wrap	is around the right ventricle	
posteriorly to supp	oly the	portion of the left ventricle.	posterior
So, a posterior infa branch of the	arction usually Coronary	is caused by an occlusion of a Artery.	Right

Note: For a long time the Right Coronary Artery was thought to play only a minor role in supplying blood to the heart. Sophisticated techniques of cardiac catheterization and coronary angiography have shown that the Right Coronary Artery usually provides the blood supply to the SA Node, the AV Node, and the His Bundle. It is no wonder that acute posterior infarction is often associated with serious arrhythmias. Wise health care providers treat posterior infarction with concern and respect.



The base of the left ventricle receives its blood supply from branches of either the Right or the Left Coronary Artery, depending on which artery is "dominant."

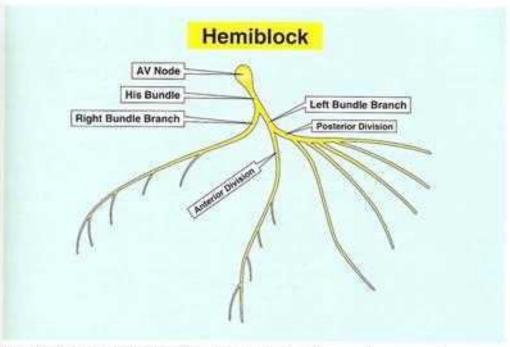
Inferior ("diaphragmatic") infarctions are caused by an occluded terminal branch of either the Right or the _____ Coronary Artery.

Lett

So the diagnosis of inferior infarction does not necessarily identify the artery branch that is occluded, unless you have a previous coronary arteriogram (an x-ray highlighting the coronary arteries) to identify which ______ artery supplies the inferior portion of that patient's left ventricle.

coronan

Note: Left or Right Coronary "dominance" denotes which coronary artery is the major source of blood supply to the base of the left ventricle, Right Coronary dominance is by far most common in humans.

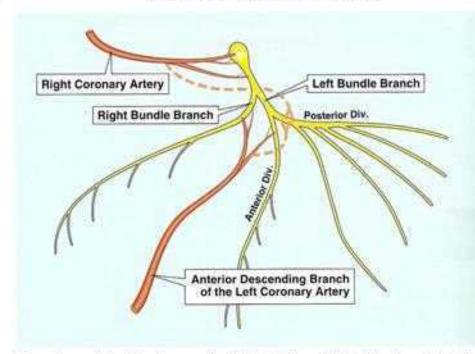


Hemiblocks are presented in this section (Infarction) because they commonly occur with infarction and an associated diminished blood supply to one of the two divisions of the Left Bundle Branch.

Note: The Left Bundle Branch subdivides into two divisions.

Posterior Division of the Bundle	T-0-1-200 T-0-200 T-0-10-10-10-10-10-10-10-10-10-10-10-10-1	Left
Hemiblocks are commonly due to loss of either the Anterior or the Posterior		Division

Note: The Right Bundle Branch does not have consistent, named subdivisions of either clinical or electrocardiographic importance (yet).



To understand hemiblocks, you should be familiar with the blood supply to the AV Node and the ventricular conduction system. Follow text and illustration closely.

to the AV Node, Bundle of His and	1. (C. 1912) 1. (C. 1913) 1. (C. 1914) 1. (
Posterior Division of the Left	Branch.	Bundle
The Left Coronary Artery also sen the Posterior Division of the Left I	20.00 C C C C C C C C C C C C C C C C C C	Branch

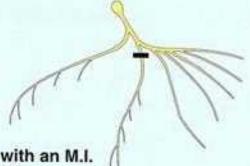
A total occlusion of the Anterior Descending branch of the
Left Coronary Artery may cause a subsequent Right Bundle Branch
with an Anterior Hemiblock (a block of the Anterior
Division of the Left Bundle Branch). Study the illustration carefully.

Block

Note: The key to knowing hemiblocks is understanding that an infarction may be due to an occlusion of a vessel at any of numerous locations, and therefore may cause a variety of blocks of the Bundle Branch system. There can be single blocks of a bundle or division, or combinations of these blocks, that spare one or more branches. A coronary obstruction that is not quite complete may cause an *intermittent* block.

^{*} Let's not forget that the SA Node is usually dependent on the right coronary artery.

Anterior Hemiblock



- LAD usually assoc. with an M.I. (or other heart disease)
- Normal or slightly widened QRS
- Q₁S₃

Anterior Hemiblock refers to a block of the Anterior Division of the Left Bundle Branch, and the above criteria are used in the diagnosis.

The slight delay of conduction to the antero-lateral and superior area of the left ventricle causes (late) unopposed depolarization upward and leftward, recognized on EKG as Left _____ Deviation. Acute LAD is usually what makes you suspect Anterior Hemiblock.

Axis

With pure Anterior Hemiblock, the QRS is widened only 10 to .12 sec., but association with other blocks of the Bundle ______ system will widen the QRS more.

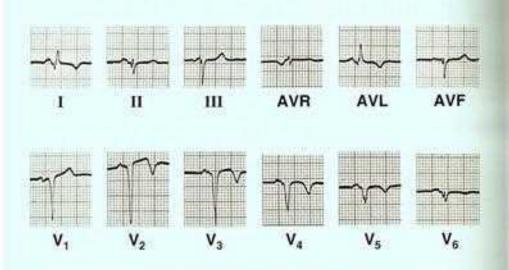
Branch

Anterior Hemiblock is a block of the Anterior Division of the Left Bundle Branch. Finding a Q in I and a wide and/or deep in III ("Q₁S₃") helps to confirm the diagnosis of Anterior Hemiblock.

S

Note: The patient's previous EKG's are essential in making a diagnosis of Anterior (or any) Hemiblock. You must always rule out pre-existing sources of Left Axis Deviation, e.g., Left Ventricular Hypertrophy, "horizontal heart," or Inferior Infarction.

Anterior Hemiblock



An occlusion of the Anterior Descending coronary artery produces an Anterior Infarction, and about one-half of these patients develop Anterior Hemiblock. Study the illustration on page 296.

Anterior Hemiblock is a block of the Anterior Division of the Left Bundle Branch, which produces unopposed, late superior-leftward depolarization in the left ventricle,	
resulting in Left Axis	Deviation
An occlusion of the Anterior Descending coronary artery will produce an Anterior Infarction, which often causes	
Anterior (That's easy to remember.)	Hemiblock
If a patient with an acute Anterior Infarction has an associated	
Axis change from normal to -60°, you should suspect Anterior (and look for Q ₁ S ₃).	Hemiblock
But if a patient with an Inferior Infarction develops Left Axis	

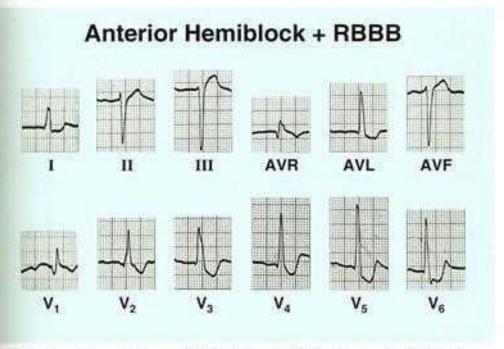
Hemiblock may not be the

Anterior

Deviation, don't jump to hasty conclusions! Inferior Infarction

can cause LAD, so __

culprit.

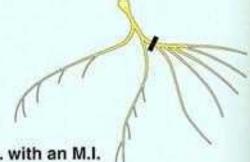


An infarction of the anterior wall of the left ventricle (due to an occluded Anterior Descending branch of the Left Coronary Artery) may cause Anterior Hemiblock and Right Bundle Branch Block. Review the illustration on page 296.

Note: Don't forget that the Anterior Descending also renders blood supply to the Right Bundle Branch, so Anterior Infarction may have an associated Right Bundle Branch Block, depending on the location of occlusion.

With Right Bundle Branch Block, the Mean QRS Vector s within the normal range or shows minimal Right Axis	Deviation
However, when a patient develops a Right Bundle Branch Block with Left Axis Deviation as well, this is probably caused by Anterior Hemiblock, particularly if there is an	
scute Anterior	Infarction

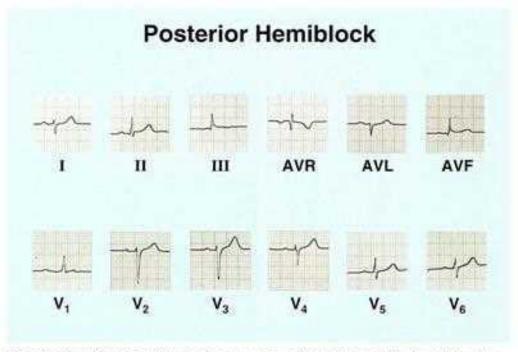
Posterior Hemiblock



- RAD usually assoc. with an M.I. (or other heart disease)
- · Normal or slightly widened QRS
- . S1Q3

Pure, isolated *Posterior Hemiblock* is rare because the posterior division is short, thick, and commonly has a dual blood supply. See the illustration on page 296.

An inferior infarction may impair the blood su Posterior division of the Left Bundle	pply to the	Branch
Posterior Hemiblocks cause Right Axis to the late, unopposed depolarization forces to right.	due ward the	Deviation
When Posterior Hemiblock is suspected, look unusually wide S in I and Q in III (known as S confirm the of Posterior Hemiblock)	Q3) to help	diagnosis



Posterior Hemiblock is always to be respected, and all Inferior Infarctions should be scrutinized to rule it out.

A lateral infarction, either recent or old, can cause

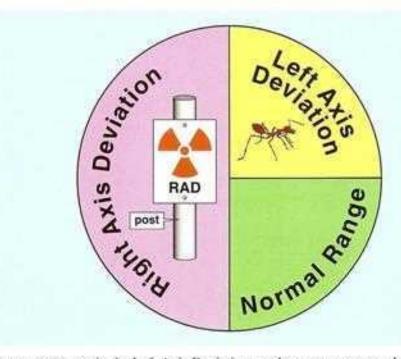
Right Axis Deviation, which can be confused with

Posterior Hemiblock. In the presence of a ______ M.I., lateral
the EKG diagnosis of Posterior Hemiblock is equivocal.

Make certain that by history and previous EKG's,
chronic Right Axis Deviation due to slender body build
("vertical heart"), ______ Ventricular Hypertrophy,
and pulmonary disease, etc. are ruled out.

Note: Posterior Hemiblock is serious, and when associated with Right Bundle Branch Block, this combination is considered very dangerous because of the tendency to progress into AV Blocks.

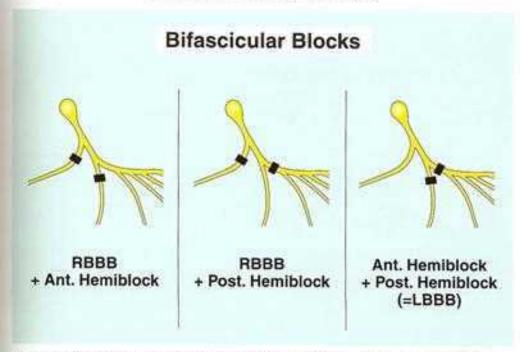
Important!: AV Block refers to "atrio-ventricular block", that is, a block between atrial depolarization and ventricular depolarization, so we commonly think of a block in the AV Node or in the His Bundle. However, simultaneous blocks of both Bundle Branches can block AV conduction. Also, RBBB in association with the simultaneous blocks of both divisions of the Left Bundle Branch can produce a block of AV conduction. Please contemplate that for a while.



Yes, you see an ant in the Left Axis Deviation quadrant, a memory tool for Left axis shift suggests Anterior Hemiblock. And, that's a "rad" (short for "radiation") sign on a post that represents a shift to Right Axis Deviation suggesting Posterior Hemiblock. For some reason this silly illustration will stick in your mind.

When a patient with a normal axis shifts into an abnormal axis, particularly when associated with a serious cardiac event,	
we suspect	hemibled
A shift from normal axis to Right Axis Deviation (remember the "rad post") is characteristic	
of Posterior	Hemiblio
A shift from normal axis to Left Axis Deviation (get that @#&%!* insect off my illustration!)	
is characteristic of Anterior	Hemibled

^{*} I wonder how the Japanese translators will deal with that?



The word "fascicle" means bundle (bundle of Purkinje fibers), so any main division of the ventricular conduction system is a fascicle. Both Bundle Branches as well as both divisions of the Left Bundle Branch are fascicles.

Note: Previously, "Bundle" implied only the Right or the Left Bundle Branch. But now, to avoid confusion for combinations of blocks (e.g., Hemiblock + Bundle Branch Block) we use a more inclusive term, "fascicular" block, to denote a Bundle Branch Block with a Hemiblock.

Note: "Bifascicular" block means that two fascicles are blocked.

Anterior Hemiblock plus Posterior Hemiblock is clinically the same as
Left Bundle Branch Block. So Bifascicular Block generally refers to
Right Bundle Branch Block together with a block of either the Anterior
Division or the Posterior Division (of the Left Bundle Branch).

Note: A block of both the Right and the Left Bundle Branch is a Complete AV Block. Right BBB plus a block of both the Anterior and Posterior Divisions (of the Left Bundle Branch) is also a Complete AV Block. Complete AV Block is very serious since only a ventricular focus remains to slowly pace the ventricles... so slowly that syncope often occurs (airway!), and a patient's life is at stake.

Note: When Bundle Branch Blocks or fascicular blocks are intermittent, we don't see them continuously on monitor or EKG tracing; just occasionally.

Intermittent Block

Intermittent block of one fascicle:

continuous normal EKG pattern -

- with intermittent wide QRS pattern characteristic of the type of intermittent Bundle Branch Block present.
- or with intermittent change of QRS Axis (i.e., QRS orientation changes intermittently) typical of the type of intermittent hemiblock present.

Permanent block + Intermittent block:

 continuous EKG signs of one permanent block with intermittent EKG signs of another block, as long as a third fascicle conducts normally.

Fortunately, combinations of (fascicular) blocks are often intermittent, making them quite obvious. Intermittent change in QRS axis (e.g., upright QRS's that transiently change to downward QRS's) usually indicates intermittent hemiblock, and a steady rhythm with transiently widened QRS's suggests intermittent BBB.

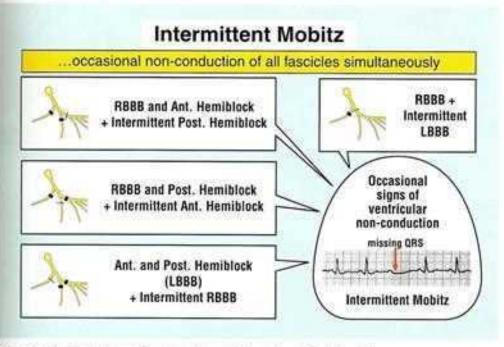
Intermittent block may exist in more than one fascicle in t	he same
patient, producing a variety of transient changes of/ (intermittent [anterior or posterior] hemiblock) or	Axis

ORS

...transiently widened QRS's typical of intermittent (left or right) _____ on EKG or cardiac monitor*. Don't ignore these intermittent changes; document them and give proper notification. BBB

Note: Like a failing light bulb that occasionally flickers, sick fascicles may suffer intermittent block. As a failing, flickering light bulb eventually burns out, similarly, intermittent fascicular blocks often warn of impending permanent block of the fascicle. With a pre-existing permanent block of another fascicle, intermittent fascicular block can be a timely warning (the only warning!) of an imminent complete block (that's why the first word on this page is "fortunately"). In most cases, permanent block plus intermittent block is an indication for an artificial pacemaker.

It is important and challenging to differentiate between intermittent anterior and posterior hemiblock, as well as intermittent right and left Bundle Branch Block. You know how already, but a little review wouldn't hurt.



Considering the three pathways of ventricular depolarization, it becomes apparent that one fascicle must remain functional at least intermittently to provide AV conduction. Early detection allows for early intervention (see page 199).

more of the fascicular blocks is intermittent otherwise there would be no AV conduction. Also, the diagnosis of "bilateral" (Left and Right) Bundle Branch

is made only if one of the Bundles has an intermittent block (or there would be no AV conduction).

Block

Note: If all fascicles are permanently blocked except one that has an intermittent block, then an intermittent Mobitz pattern (occasional nonconduction to the ventricles) will emerge. If that Mobitz pattern becomes more frequent in the tracing, or if a continuous Mobitz 2:1 pattern begins, or worse yet, if there is a continuous high ratio Mobitz block, there is a strong likelihood that complete AV block is imminent and an implantable pacemaker is needed. Knowledge plus vigilance saves lives.

Warning! With Mobitz, every cycle missing its QRS has a regular, punctual P wave - but never a premature P wave (see Note, page 128). This distinction is critical!

Note: Quickly review from page 295 to this page.

"Trifascicular" blocks are diagnosed only when one or



Patients with acute myocardial infarctions are placed in coronary care units and monitored continuously. In most hospitals patients with symptoms (only) of myocardial infarction receive the same cautious care. Patients with no physical symptoms of infarction but with definite EKG criteria of acute infarction ("silent infarction") require admission and monitoring also.

Note: Just as medical treatment of arrhythmias changes with the times, so do the attitudes toward indications for artificial pacemakers, angioplasty with stenting, coronary bypass procedures, and thrombolytic treatment, Keep up with the changing standards in your local medical community, read the current literature, and always know the basics.

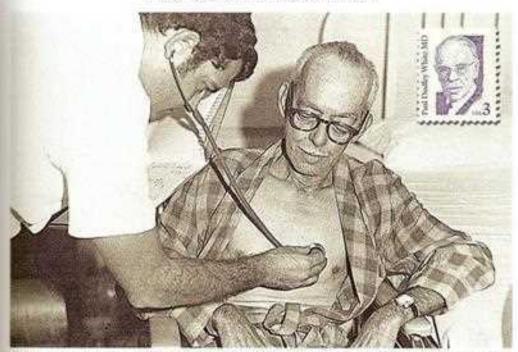
You should always know how to determine the loca	tion of
an infarction and the vessel(s) involved, as well as the	heir
association with	Hemiblock
POWER ENGINEERING FOR THE SECOND SECO	

In patients with myocardial infarction, be alert for subtle changes of Axis (change of QRS orientation in the same lead), and also rhythm changes that may indicate impending

______AV block. Vigilance is critical.

226 728 2200

complete



Remember that the patient's history and clinical diagnosis are still the most valuable tools you have (using your knowledge and judgment) in determining infarction and infarction-related problems.

The EKG has never become obsolete because it provides more information than any other diagnostic modality.	cardiac
There is no substitute for obtaining an accurate, even if it is volunteered by witnesses to an event.	history
Although the laboratory provides much useful information, the is an immediate diagnostic gift for those skilled in its interpretation.	EKG

Note: The value of an EKG increases multifold when it is compared to a patient's previous tracings — get them as soon as possible! Incidentally, is this a photo of Dr. Paul Dudley White, and who is his examining physician with the Elvis sideburns?

Note: Review Infarction by turning to the Personal Quick Reference Sheets on pages 342 and 343, and again, look at your simplified methodology (page 334).

- 1. Rate
- 2. Rhythm
- 3. Axis
- 4. Hypertrophy
- 5. Infarction

You now have the knowledge and certainly the interest and enthusiasm to interpret EKG's, but always do it methodically. Begin with Rate, then Rhythm, Axis, Hypertrophy, and Infarction. Get accustomed to this routine.

Note: In the excitement of an emergency you may be tempted to hunt for Q waves. By breaking the routine you will inevitable miss important diagnostic information - valuable information necessary for the proper treatment of the patient. Keep a cool head and read every EKG properly. Your patients will benefit from your thoroughness.

Note: Take one final look at page 334 and review each step of the entire methodology. Then (please) slowly review all of the PQRS pages from 334 to 343. But before you close this book there is some very helpful information in the Miscellaneous chapter. It is next. No, you aren't done yet.

Chapter 10: Miscellaneous

Before you begin, look at this chapter's summary on pages 344 to 346.

Miscellaneous Effects

Pulmonary

Electrolytes

Medications

Artificial Pacemakers

Heart Transplants

The above effects are common to, but not necessarily diagnostic of, certain conditions or situations that can produce recognizable changes on the EKG.

Note: Certain effects may be recognized by their characteristic appearance on the electrocardiogram or on cardiac monitor. For most of the conditions to be discussed in this section, the electrocardiographic signs merely alert us to be aware of existing conditions, certain pathology, or drug or electrolyte effects. But to confirm your suspicion, you should review the medical history, carry out a detailed physical exam, and obtain proper diagnostic tests. Rarely is a diagnosis based entirely on any of the following EKG findings, however they are exceptionally helpful.

Three Important Syndromes detection can save a life

Brugada Syndrome

- RBBB with ST elevation in V₁, V₂, and V₃ (see page 268)
- susceptible to deadly arrhythmias



Wellens Syndrome

- · marked T wave inversion in V2 and V3
- Ant. Descending Coronary stenosis



Long QT Syndrome

- QT interval longer than ½ of the cardiac cycle
- · predisposed to ventricular arrhythmias



Each of these perilous syndromes are easily detected in relatively asymptomatic patients. Routine examination of all EKG's for these innocuous-looking hallmarks can avoid an inevitable demise. Conventional treatment offers the patient reasonable longevity. The satisfaction of saving a human life is your reward for your vigilance.

Patients with Brugada Syndrome, a familial condition, may succumb to deadly arrhythmias; implantation with an ICD can prevent sudden

death

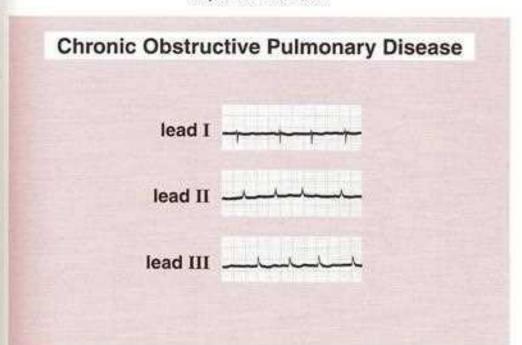
Wellens Syndrome, caused by a stenosed anterior descending coronary artery, is easily recognized. Angioplasty with stenting or a coronary bypass graft can remove the imminent peril of impending myocardial _______.

infarction

There are six known forms of (hereditary) Long QT Syndrome; these patients are predisposed to dangerous ventricular arrhythmias. A long QT interval exceeds one-half of the ______ cycle.

cardiac

Note: If everyone who reads this book becomes familiar with, and routinely looks for, these important diagnostic signs, it will serve humanity immensely. A glance at the right chest leads and observing the QT interval is sufficient.



Chronic Obstructive Pulmonary Disease (COPD) often produces low voltage amplitude in all leads, and there is usually Right Axis Deviation.

Chronic Obstructive Pulmonary Disease (COPD) commonly produces QRS complexes of small amplitude* in all leads. In fact, all waves in the EKG are minimized in _____.

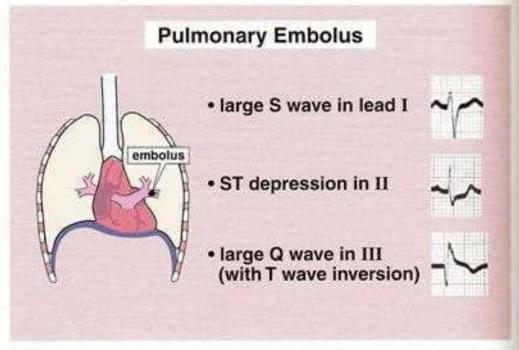
COPD

With COPD, the right ventricle works against considerable resistance, so there is usually some degree of Right Ventricular Hypertrophy and therefore associated ______ Axis Deviation notice negative QRS's in lead I).

Right

Note: Multifocal Atrial Tachycardia (MAT) is also seen with COPD.

Low voltage in all leads also appears with hypothyroidism and chronic constrictive pericarditis.



With Pulmonary Embolus we usually see a large S wave in lead I, and a Q wave and an inverted T wave in lead III $(S_1Q_3 L_3)^*$.

S₁Q₃ L₃ syndrome characterizes acute *cor pulmonale* resulting from pulmonary embolus. It is called S₁Q₃ L₃ because of the large S wave in lead L and there is a Q wave and an inverted T wave in lead ____.

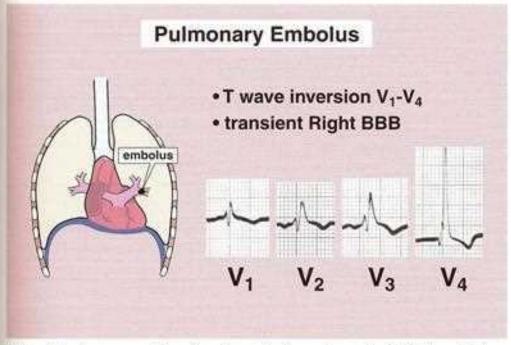
III

Note: Notice the typical tendency toward Right Axis Deviation (lead I).

There is usually ST segment ______ in lead II.

depression

^{*} Don't be confused by the inverted T in the printed text. It's a great memory tool, even if the publisher dislikes it.

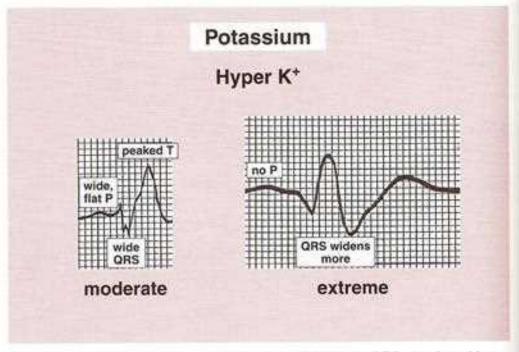


Also with pulmonary embolus, there is usually T wave inversion in V₁ through V₄.

Often there is Right Bundle Branch Block.

wave inversion in the chest leads (particularly in eads V ₁ through V ₄) is a very important diagnostic ign of pulmonary	embolus
Pulmonary embolus may cause Bundle Branch Block. This block often subsides after the patient improves.	Right
We can recognize the presence of Right Bundle Branch Block by the R,R' in the right leads.	chest

Note: Occasionally the Right Bundle Branch Block may be "incomplete" (QRS of normal width, but R,R' is present).

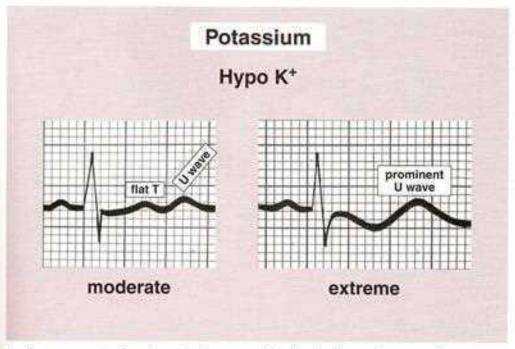


With elevated serum potassium the P wave flattens down, the QRS complex widens, and the T wave becomes peaked.

Note: The potassium ion (K*) plays an extremely important role in cardiac electrophysiology. The range of normal serum K* concentration is very narrow. In medical parlance we add the suffix "-emia" to the end of the ion name to denote its presence in the blood... but it sounds funny with "potassium." So its chemical symbol, K, is pronounced verbally, and the prefix "hyper" for increased, or the prefix "hypo" for decreased, is added to communicate deviations from normal. Now you'll understand both hyper- and hypo-kalemia* (pronounced "kay-LEE-mia"). And that's right, it's written "kalemia." That should help you and also your friends who might be perplexed...

serum potassium is the T wave.	peaked
The P wave widens and flattens with increased scrum sotassium, and with extreme hyperkalemia the wave nearly disappears.	P
When a patient has hyperkalemia, ventricular depolarization takes longer, so the QRS complex	widens

The "I" is added to enhance the phonics, so you don't have to get the "I" out of there (chuckle!).



As the scrum potassium drops below normal levels, the T wave becomes flat (or inverted) and a U wave appears.

With hypokalemia, as the serum potassium concentration drops, the __ wave flattens out, and if the K* concentration drops lower, the T wave inverts.

T

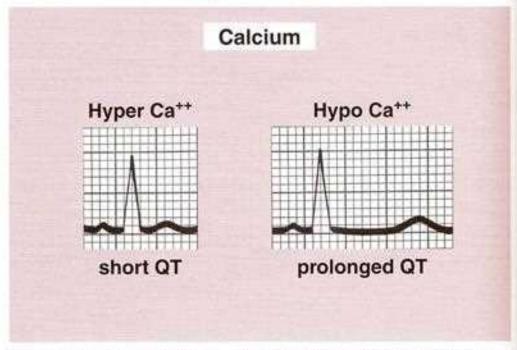
Note: I always think of the T wave as a tent housing potassium ions. When there is an increase in potassium ions, the tent peaks up, but lowering of potassium ions lowers the height of the tent.

With hypokalemia a __ wave appears. This wave becomes more pronounced as the loss of potassium becomes more severe.

U

Note: Potassium is not just "one of those serum electrolytes."

Potassium plays a critical role in repolarization and also in maintaining a precise resting potential. A decrease in potassium makes ventricular automaticity foci extremely irritable. In fact, low potassium can initiate Torsades de Pointes, and it can also evoke dangerous ventricular tachyarrhythmias. Hypokalemia also enhances the toxic effects of digitalis excess.



With hypercalcemia, the QT interval shortens; however hypoculcemia prolongs the QT interval.

Note: Since you already understand "hyper-" and "hypo-", I only need mention that "-calcemia" is pronounced "cal-SEE-mia".

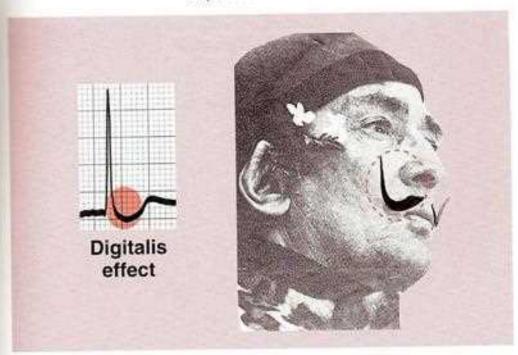
Hypocalcemia will prolong the ____ interval.

QT

Note: The QT interval is measured from the beginning of the QRS complex to the end of the T wave. Normally, the QT interval should be less than half of the cycle length.

An increase in calcium (Ca*+) ions accelerates both ventricular depolarization and ventricular repolarization. This is manifested as a short QT_____.

interval



Digitalis causes a gradual downward curve of the ST segment, to give it the appearance of Salvador Dali's mustache. Notice that the lowest portion of the ST segment is depressed below the baseline.

Digitalis produces a unique, gradual downward curve of the ____ segment; this is the classical "digitalis effect."

ST

Note: To identify the classical pattern of digitalis effect, you should observe a lead with no demonstrable S wave. The downward portion of the R wave gradually thickens as it curves down into the ST segment, which is usually depressed. The downward limb of the R wave has a gentle, curving slope that gradually blends into the depressed ST segment. Look for it the next time you have a patient on a digitalis preparation.

Note: Digitalis in therapeutic doses has a parasympathetic effect. With a Sinus Rhythm, digitalis slows the SA Node pacing rate. Conduction through the AV Node is slowed, and digitalis also inhibits the AV Node's receptiveness to multiple stimuli, allowing fewer stimuli to reach the ventricles (necessary with Atrial Flutter and Atrial Fibrillation) to permit a more physiological and more efficient ventricular response rate. Digitalis has a very narrow range of therapeutic effectiveness, and should this therapeutic range be exceeded, a multitude of undesirable effects can result. See the next two pages...

Excess Digitalis

- atrial & Junctional premature beats
- · PAT with block
- · Sinus block
- AV blocks

Excess digitalis tends to cause AV Blocks of many varieties, and may even induce Sinus (SA) Block.

Note: Supraventricular (particularly atrial) foci are exceptionally sensitive to digitalis, so premature atrial beats (PAB's) are often the earliest warning sign that your patient has elevated levels of digitalis. Atrial automaticity foci are very effective digitalis sensors.

Digitalis in excess may cause transient Sinus	Block
Digitalis retards conduction of depolarization through the AV Node; and in excess, it can cause various types	
of Block, particularly rate-dependent AV Block.	AV
Automaticity foci of the atria and the AV Junction can	
become irritable when preparations are present in excessive concentrations in your patient.	digitalis

Note: Low serum potassium can enhance the toxicity of digitalis, so that digitalis, even in therapeutic concentrations, can produce undesirable signs of toxicity if the serum potassium is low.

Digitalis Toxicity

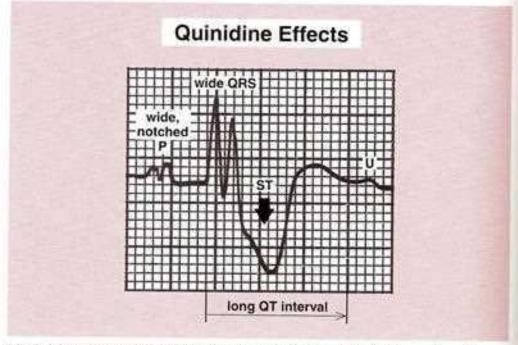
- atrial & Junctional tachy-arrhythmias
- PVC's
- Ventricular Bigeminy, Trigeminy
- Ventricular Tachycardia
- Ventricular Fibrillation

Atrial and Junctional automaticity foci are very likely to become irritable in the presence of excessive digitalis. In fact, marked digitalis toxicity can even provoke ventricular foci into rapid and dangerous rhythms.

The foct of the atria and AV Junction are most sensitive to	
excessive digitalis, but with marked digitalis,	toxicity
even ventricular foci may become so irritable that they	110000
spontaneously emit PVC's.	

Marked digitalis toxicity can make ventricular foci so irritable that they may suddenly fire multiple discharges that initiate dangerous ______tachy-arrhythmias. ventricular

Note: Digitalis preparations have been used medicinally by civilized people since the thirteenth century. But like most other cardiac medications, in certain circumstances or in high concentrations, digitalis can induce deadly arrhythmias.



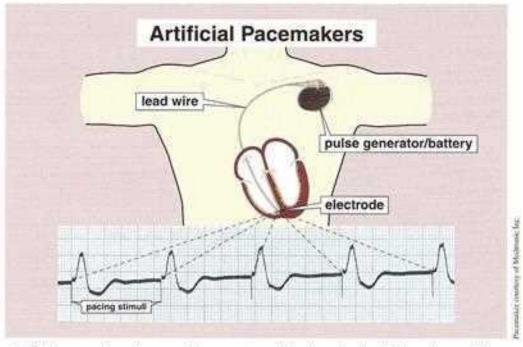
Quinidine causes widening of the P wave and widening of the QRS complex. There is often ST depression with a prolonged QT. The presence of U waves is typical as well.

Note: Quinidine retards depolarization and repolarization through both the atrial and the ventricular myocardium. Most of the effects of quinidine that we see on EKG relate to its pharmacological effects on sodium and potassium ion channels.

Quinidine causes a wide, notched ____ wave on EKG, and the QRS complex is also widened.

Quinidine prolongs the ____ interval, and depresses the ST segment. Look for U waves (which represent delayed repolarization of the ventricular conduction system).

Note: Episodes of Torsades de Pointes – a rapid and dangerous ventricular rhythm can result from quinidine toxicity (see page 158). QT



Artificial pacemakers have a pulse generator with a long-lasting lithium battery. The pacemaking stimuli are designed for ventricular or atrial (or both) pacing modalities, and a wide variety of sensing features are available.

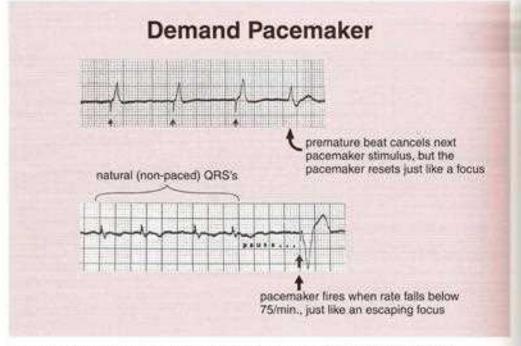
Note: Artificial pacemakers are surgically implanted as a permanent pacemaking source. Originally, they were designed to counter the bradycardia that attends Complete AV Block and Sick Sinus Syndrome. Now, the uses and variety of pacemaker types is well beyond the scope of this book, so we will review only basic principles of artificial cardiac pacing. In most cases the electrode lead wire is passed transvenously into the right side of the heart; however, sometimes the stimulating electrode is surgically attached to the epicardial surface of the heart.

The pacemaker	generator emits	regular	pacing	stimuli,	which
record on the _	as a narrow '	vertical	spike.		

EKG

The pacemaker emits regular, paced electrical _____,
and each stimulus should "capture" (i.e., depolarize) the
myocardial tissue in contact with the electrode. The depolarization
stimulus then conducts through the myocardium.

stimuli



The demand feature of many artificial pacemakers is designed to imitate the physiological mechanisms of an automaticity focus (great idea!). The demand pacemaker is programmed with an "inherent rate" that is overdrive-suppressed by normal Sinus pacing.

Note: The illustration depicts the EKG of a demand pacemaker with a ventricular sensing electrode and a ventricular pacing electrode.

A demand pacemaker is overdrive-suppressed by normal Sinus pacing, but should the Sinus rate drop below the pacemaker's programmed inherent rate, the pacemaker, no longer overdrive-suppressed, escapes to assume pacemaking responsibility at its inherent _____.

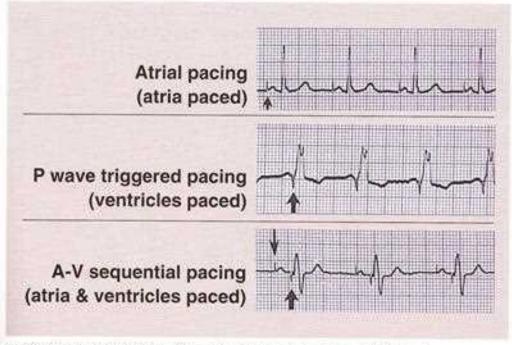
rate

But if the SA Node resumes pacing at a normal rate (which is faster than the _____ rate of the demand pacemaker) the demand pacemaker is overdrive-suppressed and stops pacing.

inherent

The demand pacemaker is designed to reset just like an automaticity focus. When the demand pacemaker senses a PVC, it resets its pacing (at the cycle length of its inherent rate) in step with the ____. This provides for uninterrupted cardiac function (clever engineers design to imitate Nature).

PVC



Contemporary pacemakers offer many features that can be used to treat many types of cardiac dysfunction and pathology.

With failure of the SA Node, Atrial Pacing can be used when the AV Node and ventricular conduction system function normally, so the artificially paced atrial stimuli are properly conducted from the atria to the _______ ventricles

A complete AV block prevents normal Sinus pacing from conducting to the ventricles and may require P wave triggered pacing", which senses the patient's ____ wave, then after a brief pause (imitating normal AV conduction) it generates a stimulus for ventricular depolarization.

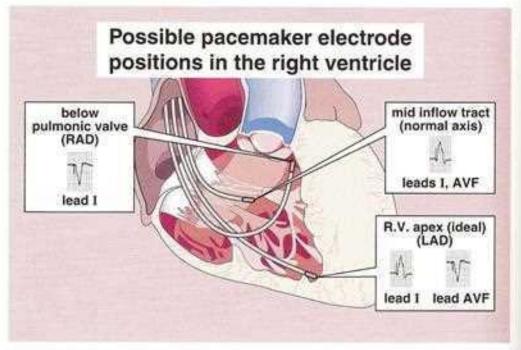
P

SA Node malfunction combined with complete AV block sometimes necessitates A-V sequential pacing, which provides a stimulus for atrial depolarization followed by a brief pause, then the ventricles are

depolarized

Note: Modern pacemakers are computerized wonders that can detect and respond to physiological needs such as decreased rate during sleep and increased rate during exercise.

^{*} Also called "atrial synchronous" or "atrial tracking" pacing.

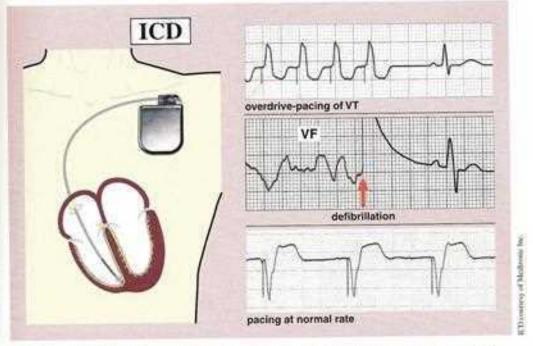


Usually a right ventricular electrode is used for cardiac pacemaking; the electrode tip of the lead is positioned within the cavity of the right ventricle. Three possible catheter lead positions are shown with the way they record on EKG.

Note: The ideal location of the tip electrode of a right ventricular pacemaker, is in the apex of the right ventricle. The resultant QRS complex has a Left Bundle Branch Block pattern with Left Axis Deviation.

When a paced QRS shows a LBBB patter normal axis, the electrode tip is in the mid		
of the right		ventricle
But if you notice a paced QRS with a LBI	3B pattern and	
Right Axis Deviation, the tip of the below the pulmonic valves	is just	electrode

Note: Certain cardiac patients may have a surgically implanted "pacemaker", called an *Implantable Cardioverter Defibrillator* (ICD, see next page) that can pace, detect and interpret rhythm disturbances, and treat tachyarrhythmias by overdrive pacing or cardioversion, even defibrillate in the event of ventricular fibrillation. Oh, Brave New World!



The Implantable Cardioverter Defibrillator (ICD) is a self-contained, computerized device that can instantly analyze and treat most dangerous cardiac arrhythmias. It can simulate normal sinus pacing, institute overdrive (suppression) pacing to treat ventricular tachycardia, provide cardioversion, and even defibrillate VF.

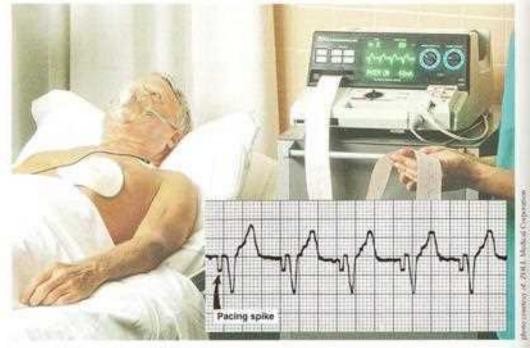
The ICD can detect and treat certain arrhythmias with cardioversion (a precisely timed electrical shock), and...

it can diagnose ventricular tachycardia and respond with overdrive pacing to suppress the causative ventricular _____. focus

The ICD can detect ventricular fibrillation and instantly defibrillate the heart, and...

should the SA Node perform sluggishly after defibrillation, the ICD will provide pacemaking stimuli at a physiological ______

Note: The ICD is a technological masterpiece!



There is an external non-invasive pacemaking device that effectively delivers pacing stimuli to the heart through intact skin in emergency situations.

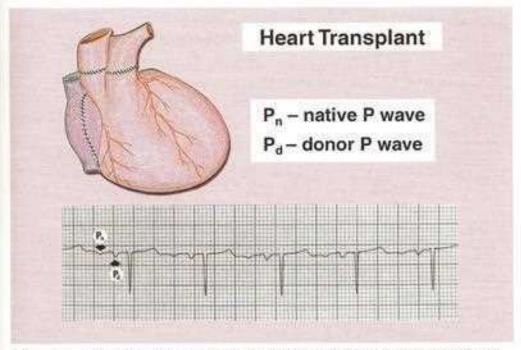
Sophisticated pacemakers are available that can painlessly pace the heart through the intact _____. These external, non-invasive pacemakers are ideal for temporary pacing.

skin

Pacing the heart through the body surface requires an impulse of longer duration than that of intracardiac pacemakers so each pacing spike is wide with a _____ end.

flat

Note: Another externally applied emergency device, the Automated External Defibrillator (AED), records and analyses the patient's EKG, and then automatically defibrillates the patient if a deadly arrhythmia is detected. The AED is very accurate in its computerized recognition of Ventricular Fibrillation and high rate Ventricular Tachycardia; it is easily operated by moderately trained personnel. Numerous trials and studies have proven the AED to be a very effective method of defibrillation in a non-hospital setting. See page 170.



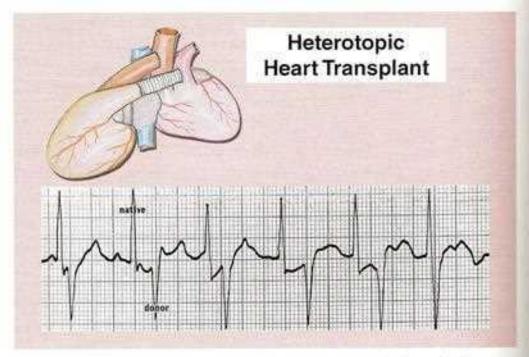
A heart transplant procedure leaves portions of the recipient patient's "native" atria in place. These portions of atria contain the patient's own SA Node, so the transplant patient has his native SA Node, plus the SA Node of the donor heart.

Note: To expedite these procedures, the portions of the native atria that contain the large vessel orifices are left behind to be sutured to the atria of the transplanted heart. So the recipient patient retains the native SA Node, and the donor heart that the patient receives also has a functioning SA Node.

each producing waves.	P
The native SA Node produces depolarizations (P _n) that do not pass beyond the suture line, so they do	
not depolarize the donor	atria

The transplanted "donor" heart has its own functional SA Node that remains the dominant pacemaker, so all of its P waves (P_d) are followed by _____ complexes.

QRS



A heterotopic heart transplant is a procedure that leaves the native heart in place, while a temporary, donor heart is surgically attached to assist the pumping effort.

In order to assist	in pumping, a heterotopic heart transplant
gives the patient	(temporarily) two

hearts

So the EKG in this temporary, emergency situation displays the simultaneous recording of the electrical activity of two separate ______.

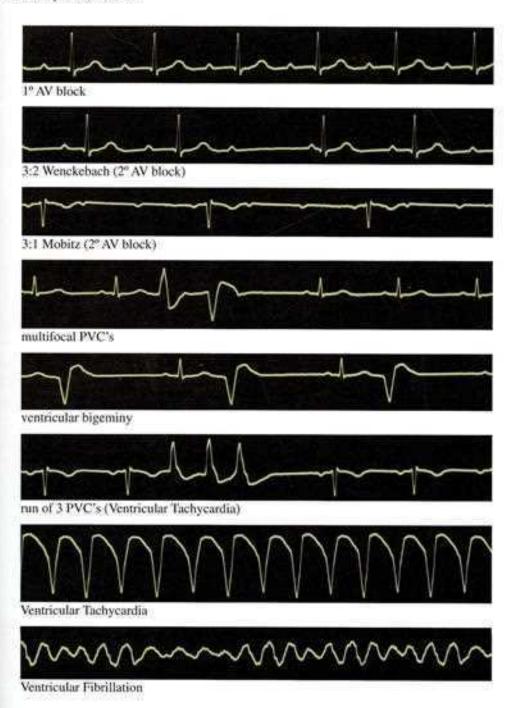
hearts

Note: With great advances in medical technology and increasing sophistication of biomechanical engineering, attempts are constantly being made to devise an efficient artificial heart. It is unlikely that a totally artificial heart will ever approach the efficacy and safety of that Designed by Nature.

Let me know if your understanding was a kind of ecstacy.
(It has been for me.) -DD

Cardiac Monitor Displays

Cardiac monitors display the same information as recorded on a standard 12 lead EKG. Some initial apprehension may arise because of lack of familiarity with the display. The EKG tracing is in bright green on a black background, and the amplitude of waves (height and depth) is increased. Because the "leads" of a cardiac monitor are modifications of standard leads with exaggerated amplitudes to aid in visualization at a distance, voltage (height and depth) criteria can not be utilized. But don't despair, this is just another method of displaying the heart's electrical activity... and familiarity eventually breeds content.



Electrocardiography was your challenge; knowledge, your achievement.

Now that you are certainly pleased with your understanding of basic electrocardiography, and proud of your ability to interpret the information on EKG's and cardiac monitors, you realize how logical and marvelously Designed is the heart.

You're probably ready for *Ion Adventure in the Heartland*, Dr. Dubin's highlyacclaimed, entertaining text. For your giant leap into the 21st century, your knowledge needs to be on a molecular level, so let Dr. Dubin be your guide and simplify your understanding.

Ion Adventure in the Heartland is an exciting full-color expedition deep into the secret molecular wonderland of cardiac physiology with a splash of biochemistry. We will explore the vivid inner world of the magnificent "ion movers," a dynamic microcosm of exotic ion channels, ion pumps, ion exchangers, the mysterious connexons, and the fast moving ions that they control. You will be immersed in this never-before-seen, living wonderland that generates the heart's electrical energy and power in responce to physiological demands. What a performance to behold, as we expose the private activities of these ion movers, exquisitely orchestrated by the autonomic nervous system. The book is narrated in Dr. Dubin's entertaining, easy to understand style. You will discover what really makes the healthy heart tick, yet falter with stress and disease. Though this adventure is not likely to become a great movie, it is an easy to understand, illustrated story of the intimate lifestyles of the ions and their movers as recorded by the surface EKG.

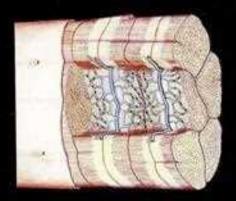
To learn more:

www.lonAdventure.com



P.O. Box 1092, Tampa, FL 33601 U. S. A. Ion Adventure in the Heartland, is an exciting adventure in living color, providing vital knowledge for the medical profession in millennium 2000.

Scientists and researchers in the twentieth century found the microstructure of the cells of the heart to be an engineering wonder. Research continues to reveal intriguing information, while raising many new questions. Current concepts may seem complex—even intimidating—to medical professionals, although, in reality, they are easy to understand.

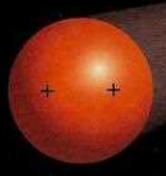


The key to cardiac function is at the ionic-molecular level, where autonomic control occurs, and where medications work. All the electrical and mechanical properties of the heart are due to the movement of only three types of ions...yes, three little ions!

Let me have them come forward to introduce them by name:



Sodium ion (Na*)

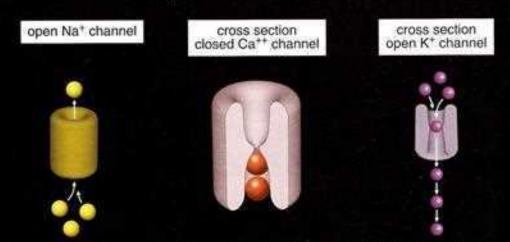


Calcium ion (Ca**)

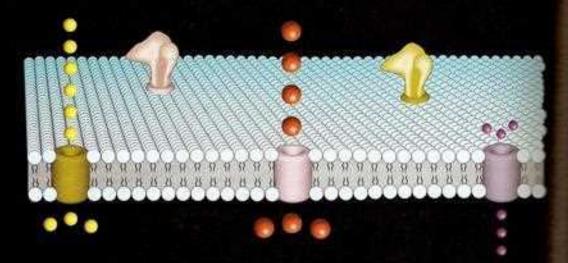


Potassium ion (K*)

Ion-moving ("ion-kinetic") structures of the cell membrane (and cell interior) produce ion movement. Most of these structures are sophisticated molecular portals that employ precision mechanisms to control and regulate the movement of Na⁺, Ca⁺⁺, and K⁺ ions. Each variety of ion-kinetic structure has its own unique behavior.



We are launching an expedition to explore this incredible ionic-molecular microcosm to learn just how these mechanisms move Na⁺, Ca⁺⁺, and K⁺ ions to govern the heart's function. We would love to have you join us on this fascinating adventure.



Our fantastic journey is narrated by a five year old boy named Dale, so certainly anyone who has read Rapid Interpretation of EKG's can easily master this vital medical knowledge, which is so useful and necessary in millennium 2000.

Hurry... your knowledge is needed!

www.lonAdventure.com

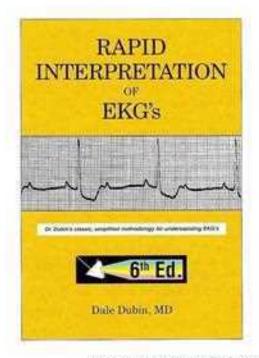
(pages 333 to 346)

from: Rapid Interpretation of EKG's

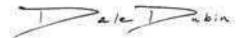
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May humanity benefit from your knowledge.



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Physicians and medical students: www.theMDsite.com

Nurses and nurses in training: www.CardiacMonitors.com

Emergency medical personnel: www.EmergencyEKG.com

Dubin's Method for Reading EKG's

from: Rapid Interpretation of EKG's

by Dale Dubin, MD
COVER Publishing Co., P.O. Box 1092, Tampa, FL 33601, USA

1. RATE (pages 65-96)

Say "300, 150, 100" "75, 60, 50"

 but for bradycardia: rate = cycles/6 sec. strip X 10

2. RHYTHM (pages 97-202)

Identify the basic rhythm, then scan tracing for prematurity, pauses, irregularity, and abnormal waves.

- Check for: P before each QRS. QRS after each P.
- Check: PR intervals (for AV Blocks).
 QRS interval (for BBB).
- . If Axis Deviation, rule out Hemiblock.

3. AXIS (pages 203-242)

- QRS above or below baseline for Axis Quadrant (for Normal vs. R. or L. Axis Deviation),
 For Axis in degrees, find isoelectric QRS in a limb lead of Axis Quadrant using the "Axis in Degrees" chart.
- Axis rotation in the horizontal plane: (chest leads) find "transitional" (isoelectric) QRS.

4. HYPERTROPHY (pages 243-258)

Check V, P wave for atrial hypertrophy.

R wave for Right Ventricular Hypertrophy. S wave depth in V,...

+ R wave height in V, for Left Ventricular Hypertrophy.

5. INFARCTION (pages 259-308)

Scan all leads for:

- Q waves
- Inverted T waves
- · ST segment elevation or depression

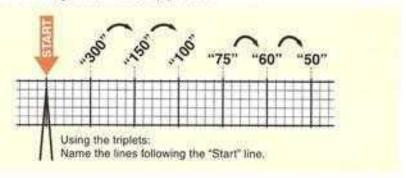
Find the location of the pathology (in the Left ventricle), and then identify the occluded coronary artery.

Rate (pages 65 to 96)

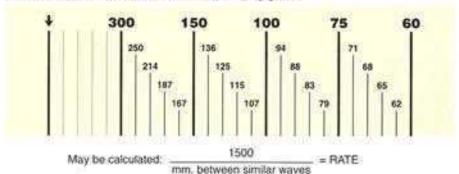
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Determine Rate by Observation (pages 78-88)



Fine division/rate association: reference (page 89)



Bradycardia (slow rates) (pages 90-96)

- Cycles/6 second strip × 10 = Rate
- When there are 10 large squares between similar waves, the rate is 30/minute.

Sinus Rhythm: origin is the SA Node ("Sinus Node"), normal sinus rate is 60 to 100/minute.

- Rate more than 100/min. = Sinus Tachycardia (page 68).
- Rate less than 60/min. = Sinus Bradycardia (puge 67).

Determine any co-existing, independent (atrial/ventricular) rates:

Dissociated Rhythms: (pages 155, 157, 186-189)
 A Sinus Rhythm (or atrial rhythms) may co-exist with an independent rhythm from an automaticity focus of a lower level. Determine rate of each.

Irregular Rhythms: (pages 107-111)

 With Irregular Rhythms (such as Atrial Fibrillation) always note the general (average) ventricular rate (QRS's per 6-sec. strip × 10) or take the patient's pulse.

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Personal Quick Reference Sheets

Rhythm (pages 97 to 111)

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* Identify basic rhythm...

...then scan entire tracing for pauses, premature beats, irregularity, and abnormal waves.

* Always:

- Check for: P before each QRS. QRS after each P.
- Check: PR intervals (for AV Blocks). QRS interval (for BBB).
- Has QRS vector shifted outside normal range? (to rule out Hemiblock).

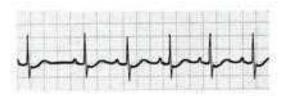
Irregular Rhythms (pages 107-111)

Sinus Arrhythmia (page 100)

Irregular rhythm that varies with respiration.

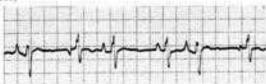
All P waves are identical.

Considered normal.



Wandering Pacemaker (page 108)

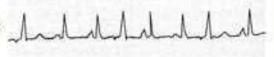
Irregular rhythm. P waves change shape as pacemaker location varies. Rate under 100/minute...



...but if the rate exceeds 100/minute, then it is called

Multifocal Atrial Tachycardia

(page 109)



Atrial Fibrillation (pages 110, 164-166)

Irregular ventricular rhythm. Erratic atrial spikes (no P waves) from multiple atrial automaticity foci. Atrial discharges may be difficult to see.



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Personal Quick Reference Sheets

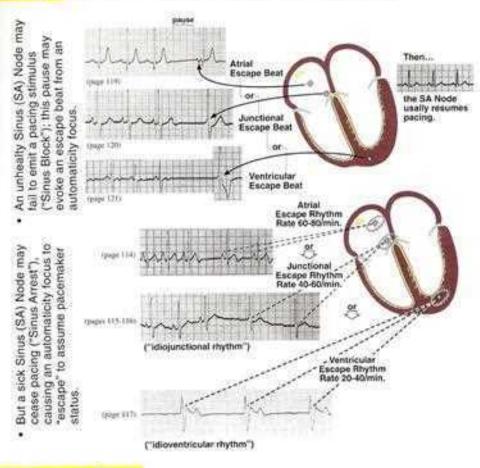
Rhythm continued (pages 112 to 145)

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Escape

(pages 112-121) - the heart's response to a pause in pacing



Premature Beats (pages 122-145) - from an irritable automaticity focus

An irritable automaticity Premature Atrial Beat focus may suddenly discharge, producing (pages 124-130) Premature Junctional Beat (pages 131-133) Premature Ventricular Contraction (pages 135-141) PVC's may be multiple, multifocal, in runs, or coupled with normal cycles.

"Supraventricular Tachycardia" (page 153)

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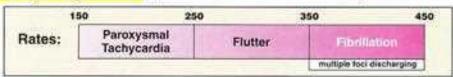
Personal Quick Reference Sheets

Rhythm continued (pages 146 to 172)

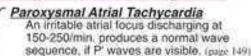
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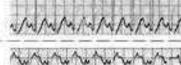
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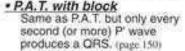
Tachyarrhythmias (pages 146-172), "focus" = automaticity focus

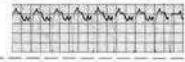


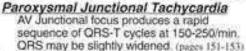
Paroxysmal (sudden) Tachycardia ...rate: 150-250/min. (pages 146-163)



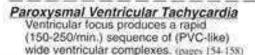










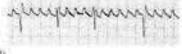




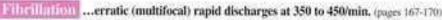
Flutter ...rate: 250-350/min.

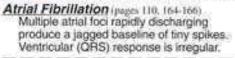
Atrial Flutter

A continuous ("saw tooth") rapid sequence of atrial complexes from a single rapid-firing atrial focus. Many flutter waves needed to produce a ventricular response. (pages 159, 160)

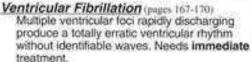


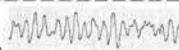
Ventricular Flutter (pages 161, 162) also see "Torsades de Pointes" (pages 158, 345) A rapid series of smooth sine waves from a single rapid-firing ventricular focus; usually in a short burst leading to Ventricular Fibriliation.







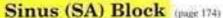




Rhythm: ("heart") blocks (pages 173 to 202)

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An unhealthy Sinus (SA) Node misses one or more cycles (sinus pause)...

the Sinus Node usually resumes pacing, but the pause may evoke an "escape" response from an automaticity focus. (pages 119-121)

AV Block (pages 176-189)

Blocks that delay or prevent atrial impulses from reaching the ventricles.

1º AV Block ... prolonged PR interval (pages £76-178). PR interval is prolonged to greater than .2 sec (one large square).



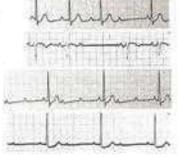
2° AV Block ... some P waves without QRS response (page 179-185)

Wenckebach ... PR gradually lengthens with each cycle until the last P wave in the (pages 180-182, series does not produce a QRS. 183)

Mobitz ...some P waves don't produce a QRS response. If "intermittent," an (pages 181-183) occasional QRS is droped.

> More advanced Mobitz block may produce a 3:1 (AV) pattern or even higher AV ratio (page 181).

2:1 AV Block ... may be Mobitz or Wenckebach. (pages 182, 183) PR length and QRS width or vagal maneuvers help differentiate.

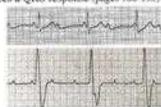


3° ("complete") AV Block ... no P wave produces a QRS response (pages 186-190)

3" Block: (page 188)

P waves-SA Node origin. QRS's-if narrow, and if the ventricular rate is 40 to 60 per min., then origin is a Junctional focus:

P waves-SA Node origin. 3° Block: QRS's-if PVC-like, and if the (page 189) ventricular rate is 20 to 40 per min., then origin is a Ventricular focus.



Bundle Branch Blockfind R,R' in right or left chest leads (pages 191-202)

Right BBB (pages 194-196)

Left BBB (pages 194-197)

* Always Check:

· is QRS within 3 tiny squares?



With Bundle Branch Block the criteria for ventricular hypertrophy are unreliable.



Caution With Left BBB infarction is difficult to determine on EKG:

QRS in V₁ or V₂

QRS in Vs or Va

Hemiblock ... block of Anterior or Posterior fascicle of the Left Bundle Branch. (pages 295-305)

* Always Check:

· has Axis shifted outside Normal range?

Anterior Hemiblock

Axis shifts Leftward → L.A.D: look for Q,S, (pages 297-299)

Posterior Hemiblock

Axis shifts Rightward -+ R.A.D. look for S.Q. (pages 300-302)

 PR intervals less than one large square?
 Is overy P wire followed by a ORS! . Always Check:

Axis (pages 203 to 242)

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General Determination of Electrical Axis (pages 203-242)

Is QRS positive (--) or negative (--) in leads I and AVF?

Is Axis Normal? (page 227)

QRS in lead I (pages 315-222)
...If the QRS is Positive (mainly above baseline), then the Vector points to positive (patient's left) side.

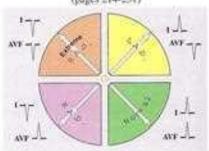
Normal:

QRS upright in I and AVF "two thumbs-up" sign



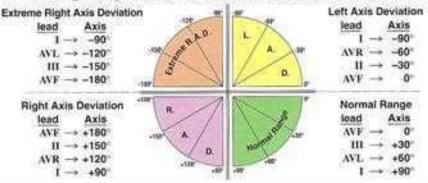
QRS in lead AVF (pages 223-226)
...if the QRS is mainly Positive, then
the Vector must point downward to
positive half of the sphere.

First Determine Axis Quadrant

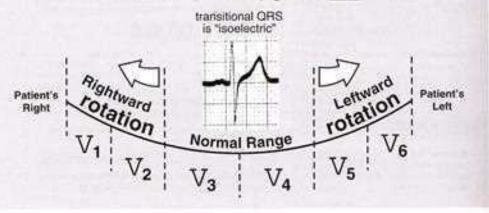


Axis in Degrees (pages 233, 234) (Frontal Plane)

After locating Axis Quadrant, find limb lead where QRS is most isoelectric:



Axis Rotation (left/right) in the Horizontal Plane (pages 236-242) Find transitional (isoelectric) QRS in a chest lead.



Hypertrophy (pages 243 to 258)

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Atrial Hypertrophy (pages 245-249)

Right Atrial Hypertrophy (page 248).

· large, diphasic P wave with tall initial component



Left Atrial Hypertrophy (page 249).

· large, diphasic P wave with wide terminal component



Ventricular Hypertrophy (psgus 250-258)

Right Ventricular Hypertrophy (pages 250-252)

- . R wave greater than S in V, but R wave gets progressively smaller from V, - Ve-
- . S wave persists in V, and V,
- · R.A.D. with slightly widened QRS.
- Rightward rotation in the horizontal plane.

Left Ventricular Hypertrophy (pages 253-257)

S wave in V, (in mm.)

+ R wave in V, (in mm.)

Sum in mm, is more than 35 mm, with L.V.H.

- · L.A.D. with slightly widened QRS.
- · Leftward rotation in the horizontal plane.

Inverted T wave: slants downward gradually,



but up rapidly.

Infarction (pages 259 to 308)

from: Rapid Interpretation of EKG's

by Dale Dubin, MD COVER Publishing Co., PO. Box 1092, Tampa, FL 33601, USA

Q wave = Necrosis (significant Q's only) (pages 272-284)

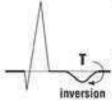


- Significant Q wave is one millimeter (one small square) wide, which is .04 sec. in duration...
 ... or is a Q wave 1/3 the amplitude (or more) of the QRS complex.
- Note those leads (omit AVR) where significant Q's are present
 ... see next page to determine infarct location, and to identify
 the coronary vessel involved.
- Old infarcts: significant Q waves (like infarct damage) remain for a lifetime. To determine if an infarct is acute, see below.

ST (segment) elevation = (acute) Injury (pages 266-271) (also Depression)

- Signifies an acute process, ST segment returns to buseline with time.
- elevation
- ST elevation associated with significant Q waves indicates an acute (or recent) infarct.
 - A tiny "non-Q wave infarction" appears as significant ST segment elevation without associated Q's. Locate by identifying leads in which ST elevation occurs (next page).
 - ST depression (persistent) may represent "subendocardial infarction," which involves a small, shallow area just beneath the endocardium lining the left ventricle. This is also a variety of "non-Q wave infarction." Locate in the same manner as for infarction location (next page).

T wave inversion = Ischemia (pages 264, 265)



- Inverted T wave (of ischemia) is symmetrical (left half and right half are mirror images). Normally T wave is upright when QRS is upright, and vice versa.
- Usually in the same leads that demonstrate signs of acute infarction (Q waves and ST elevation).
- Isolated (non-infarction) ischemia may also be located; note those leads where T wave inversion occurs, then identify which coronary vessel is narrowed (next page).

NOTE: Always obtain patient's previous EKG's for comparison!

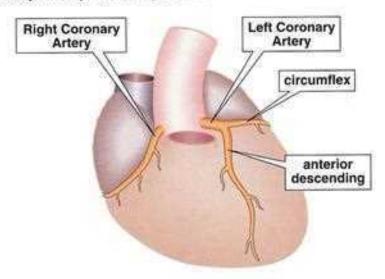
Infarction Location — and — Coronary Vessel Involvement

(pages 259 to 308)

from: Rapid Interpretation of EKG's

by Dale Dubin, MD COVER Publishing Co., P.O. Box 1092, Tampa, FL 33601, USA

Coronary Artery Anatomy (page 291)

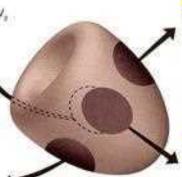


Infarction Location/Coronary Vessel Involvement (pages 278-294)

Posterior

 large R with ST depression in V, & V₂
 mirror test or reversed transitumination test (Right Coronary Artery)

(pages 282-286)



Inferior

(diaphragmatic) O's in inferior leads II. III, and AVF (R. or L. Coronary Artery) (pages 281, 294)

Lateral

Q's in lateral leads I and AVL (Circumflex Coronary Artery) (pages 280, 292)

Anterior

O's in V., V., V., and V. (Anterior Descending Coronary Artery) (pages 278, 292)

Miscellaneous (pages 309 to 328)

from: Rapid Interpretation of EKG's

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Pulmonary Embolism (pages 312, 313)

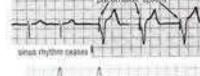
- · S,Q,L, wide S in I, large Q and inverted T in III
- · acute Right BBB (transient, often incomplete)
- · R.A.D. and clockwise rotation
- inverted T waves V_i → V_a and ST depression in II

Artificial Pacemakers (pages 324-326)

Modern artificial pacemakers have sensing capabilities and also provide a regular pacing stimulus. This electrical stimulus records on EKG as a tiny vertical spike that appears just before the "captured" cardiac response.

Demand Pacemakers; (page 322)

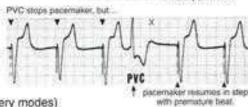
 are "triggered" (activated) when the patient's own rhythm ceases or slows markedly.



are "inhibited" (cease pacing) if the patient's own rhythm resumes at a reasonable rate.



 will "reset" pacing (at same rate) to synchronize with a premature beat.



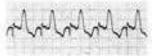
Pacemaker Impulse (delivery modes)



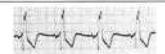
Ventricular Pacemaker (page 323) (electrode in Right Ventricle)



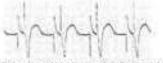
(Asynchronous) Epicardial Pacemaker Ventricular impulse not linked to atrial activity.



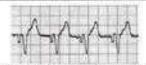
Atrial pacemaker (page 323)



Atrial Synchronous Pacemaker (page 323)
P wave sensed, then after a brief delay,
ventricular impulse is delivered.



Dual Chamber (AV sequential) Pacemaker (page 323)



External Non-invasive Pacemaker (page 326)

Miscellaneous continued

from: Rapid Interpretation of EKG's

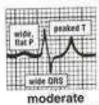
by Dale Dubin, MD COVER Publishing Co., P.O. Box 1092, Tampa, FL 33601, USA

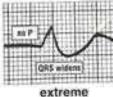
Electrolytes

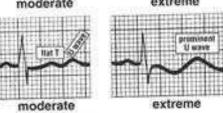
Potassium (pages 314, 315)

Increased K* (page 314) (hyperkalemia)

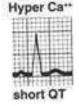
Decreased K* (pages 315) (hypokalemia)







Calcium (page 316)



Hypo Ca**

prolonged QT

Digitalis (pages 317-319)

EKG appearance with digitalis ("digitalis effect")

- remember Salvador Dali.
- · T waves depressed or inverted.
- · OT interval shortened,

Digitalis Excess ----> Digitalis Toxicity

- SA Block
- · P.A.T. with Block
- + AV Blocks

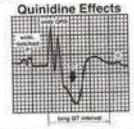
(blocks)

AV Dissociation

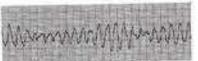
- (irritable foci firing rapidly)
- Atrial Fibrillation
- Junctional or Ventricular Tachycardia
- · multiple P.V.C.'s
- Ventricular Fibrillation

Quinidine (page 320)

EKG appearance with quinidine (page 320)



 Excess quinidine or other medications that block potassium channels (or even low serum potassium) may initiate Torsades de Pointes (page 158)



Torsades de Pointes

Practical Tips

from: Rapid Interpretation of EKG's

by Dale Dubin, MD COVER Publishing Co., P.O. Box 1092, Tampa, FL 33601, USA

Dubin's Quickie Conversion —for— Patient's Weight from Pounds to Kilograms

Patient wt. in kg. = Half of patient's wt. (in lb.) minus 1/10 of that value.

Examples:

180 lb. patient (becomes 90 minus 9) is 81 kg 160 lb, patient (becomes 80 minus 8) is 72 kg

Q052-031021Q2503-0345030

140 lb. patient (becomes 70 <u>minus</u> 7) is 63 kg.

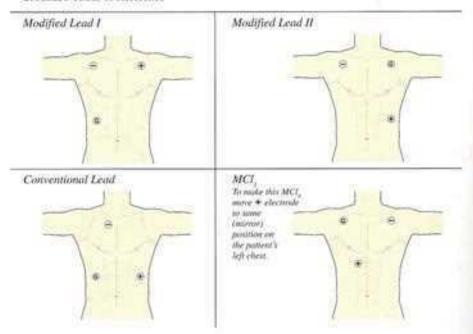
Modified Leads

—for— Cardiac Monitoring

Locations are approximate. Some minor adjustment of electrode positions may be necessary to obtain the best tracing. Identify the specific lead on each strip placed in the patient's record.

	Identification		
Sensor Electrode	Letter	er Color (inconsistent)	
+	R (or RA)	red	
<u> </u>	L (or LA)	white	
G°	G (or RL)	variable	

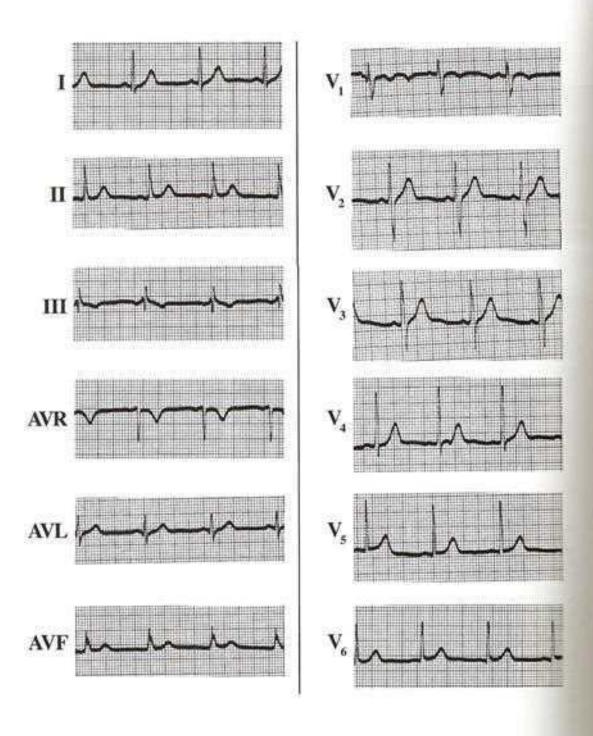
* Ground, Neutral or Reference



Cupyright & 2000 COVUR Inc.

EKG Tracings

This section contains EKG tracings (and their interpretation) from various patients. The tracings and interpretations are provided so that you can see how this method of reading EKG's actually works. Try these few examples so that you grow accustomed to this systematic approach. Once you learn how to read an EKG systematically, you will soon become very skilled at routine EKG interpretations. Patient D.D. is a 29 year old white male known to be a hypochondriac with numerous complaints.



EKG Tracings

EKG Interpretation

Patient:

D.D.

Rate:

about 70/minute

Rhythm:

Regular Sinus Rhythm

PR less than .2 sec. (No AV Block). ORS less than .12 sec. (No BBB).

...but note the R,R' in III suggesting incomplete

Bundle Branch Block.

Axis:

Normal Range (about +30°).

Rightward rotation in the horizontal plane.

Hypertrophy: No atrial hypertrophy.

No ventricular hypertrophy.

Infarction:

No significant Q waves.

Coconary vascular status)

ST segments: not elevated, except for V, and V, where ST is elevated 1/2 mm. due to "early repolarization."*

Twaves: generally upright.

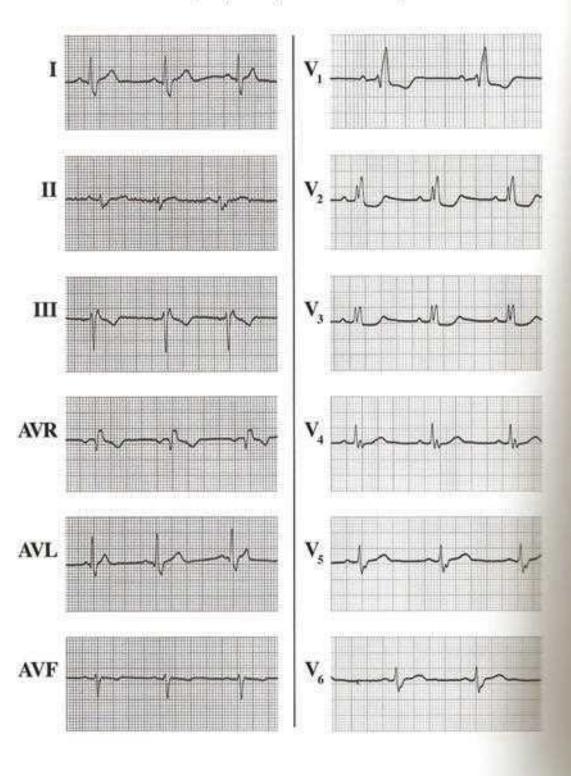
Comment:

This is an essentially normal tracing. This is the author's own EKG,

however he is no longer 29 years old.

^{*} Early repolarization is characterized by (minimal) ST elevation in the left chest leads, often with rightward rotation (horizontal plane). It is a normal finding in young athletic males.

This is the followup EKG on patient D.D., 30 years after his last EKG (see previous page.); he has had labile hypertension for the last 25 years. This EKG clearly demonstrates the value of obtaining the patient's prior EKG's for comparison.



EKG Interpretation

Patient:

D.D.

Rater

about 58/minute

Rhythm:

Sinus Bradycardia

PR less than .2 sec. (No AV Block)

ORS greater than .12 sec. reveals Bundle Branch Block.

Leads V, and V, show R,R' complexes typical of Right Bundle

Branch Block.

Axis:

Left Axis Deviation of about -25° (in lead I, R wave greater than S wave) and Q,S, indicate probable Anterior Hemiblock. Axis rotation in the horizontal plane difficult to assess due to RBBB.

Hypertrophy: Left atrial enlargement.

Left ventricular hypertrophy verified by other tests (difficult to assess on EKG with RBBB present).

Infarction:

No significant Q waves.

ST segments: depressed in V, and V, as related to RBBB

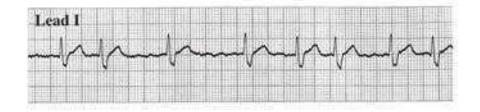
Ecoronary vascular status)

Twaves: generally upright; some T inversion in inferior leads.

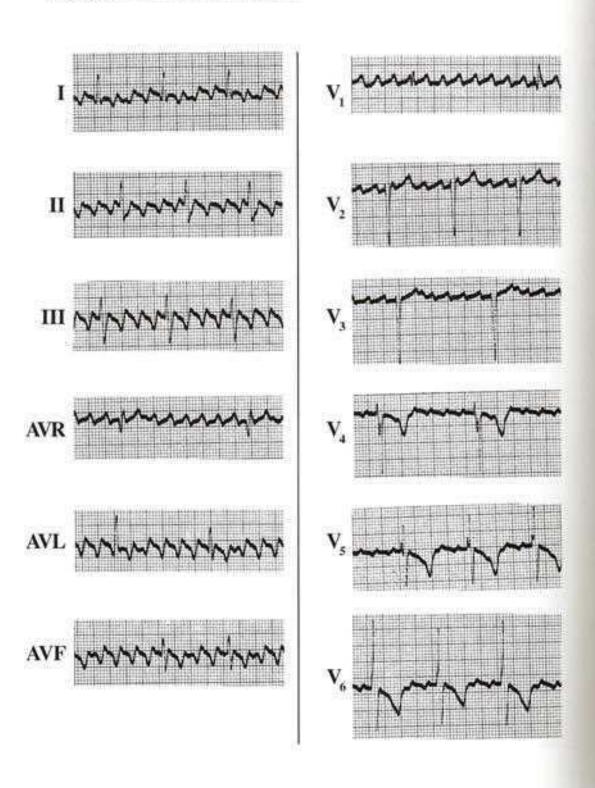
possibly distorted by RBBB.

Comment:

Compared to his previous, normal EKG (see page 348), this patient has developed significant changes. There is a sinus bradycardia. The new Right Bundle Branch Block (previously incomplete) and Anterior Hemiblock have occurred in the absense of infarction. After 25 years of poorly compensated hypertension, the patient has developed left ventricular hypertrophy. The associated left atrial enlargement may stretch irritable atrial foci in the ostia of the pulmonary veins, initiating atrial fibrillation. A few days later, the patient's rhythm strip of lead I (below), shows just that. All EKG's are, unfortunately, authentic.



Patient R.C. is a 45 year old black male with a history of coronary vascular disease. Blood pressure was 210/100 on admission.



EKG Interpretation

Patient: R.C.

Rate: Atrial rate of 300/minute

Ventricular rate generally 60/min. but occasionally slower.

Rhythm: Atrial Flutter (with inconsistent ventricular response, i.e.,

no fixed AV ratio).

PR is variable.

QRS is less than .12 sec. (No BBB).

Axis: Left Axis Deviation (-30°).

Leftward rotation in the horizontal plane.

Hypertrophy: Atrial hypertrophy difficult to determine.

No ventricular hypertrophy.

Infarction: Q waves: Q in lead I (also note large S in Lead III).

(coronary ST segments are generally isoelectric.

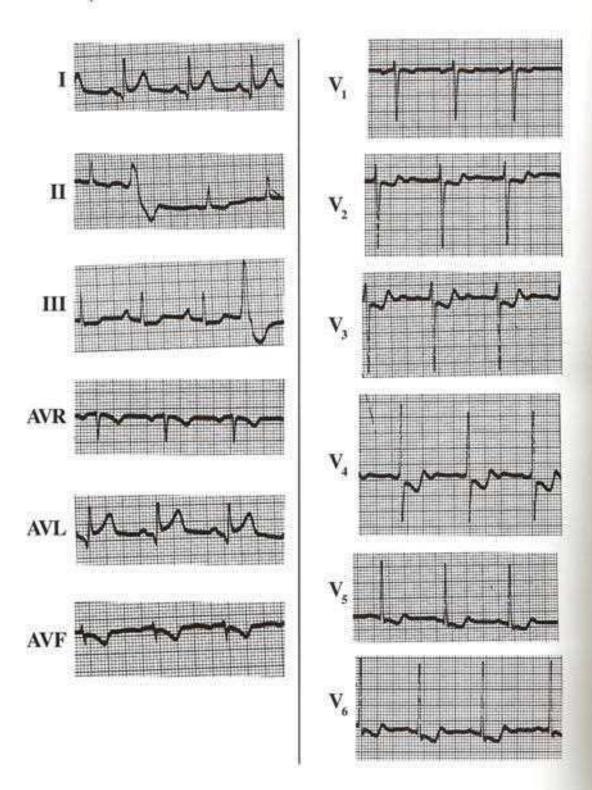
vascular status) T waves are inverted in I and AVL (look closely)

and the mid-to-left chest leads.

Comment:

The most obvious problem is Atrial Flutter with an atrial rate of 300/min, and a variable irregular ventricular rate (average 60/min.) caused by the variable AV conduction ratio between 3:1 and 7:1. An old occlusion of the Left Circumflex Coronary artery is evidenced by the old lateral infarction. New involvement of the Anterior Descending Coronary artery is suggested by anterior ischemia (T wave inversion in V_a, V_b, as well as by the probable Anterior Hemiblock (shift to Left Axis Deviation with QS, configuration; previously R.A.D. with his old lateral M.I.). Note that if one scrutinizes the T wave regions (somewhat obscured by flutter waves) in the limb leads, the flutter waves dip lower (suggestive of negative T waves) rather than higher (if superimposed on upright T waves) in all but AVR, indicating a generalized cardiac ischemia, as well as the obvious compromise of both branches of the Left coronary Artery.

Patient K.T. is a 61 year old obese, black male who was brought into the emergency department by his family. This patient had a sudden episode of severe left chest pain. Blood pressure was 95/65.



EKG Interpretation

Patient: K.T.

Rate: about 75/minute

Rhythm: Generally regular Sinus Rhythm with occasional PVC's.

PR is exactly .2 sec. so we will have to say there is a

borderline first degree AV Block, QRS is less than .12 sec. (No BBB).

Axis: Left Axis Deviation (nearly -90°).

No rotation in the horizontal plane.

Hypertrophy: Probable left atrial hypertrophy.

Left ventricular hypertrophy.

Infarction: Significant Q waves in I and AVL.

(commany ST segments are elevated in I and AVL, ST segments vascular

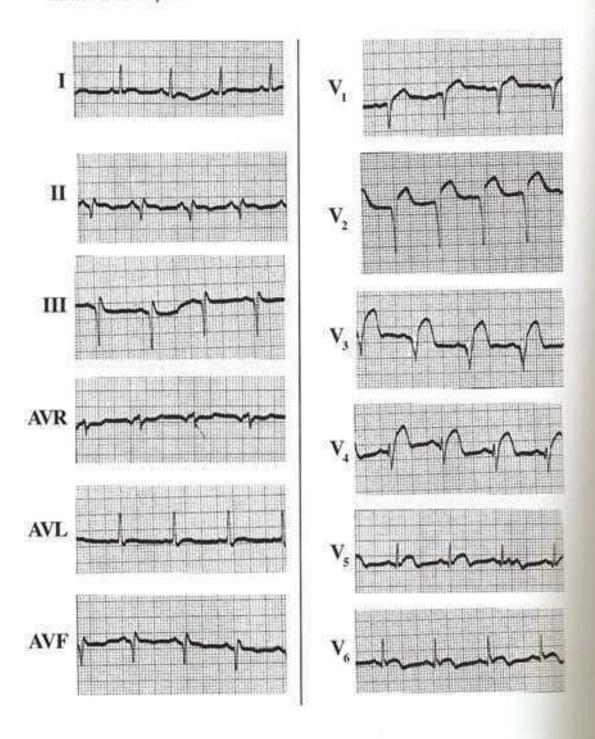
are depressed in V_1 , V_2 , V_3 , and V_4 .

Twaves are flat or inverted in II, III, and AVF and all chest leads.

Comment:

This patient has a classical acute lateral infarction caused by an occlusion of the Left Circumflex Coronary Artery. Coincident with this is a probable occlusion of the Right Coronary Artery characterized by prominent R waves with ST depression in the (V₁ to V₂) chest leads. Also, T wave inversion in II, III, and AVF suggests Right Coronary compromise. T wave inversion in all chest leads is indicative of ischemia of the Anterior Descending Coronary Artery. Note also the tall, peaked T waves in I and AVL known as "hyperacute T waves," which, although uncommon, characterize a very acute M.I. The Left Axis Deviation appeared in this patient's previous EKG's and is most likely related to his left ventricular hypertrophy rather than implicating Anterior Hemiblock (also, the Bundle Branch System appears to conduct normally). Occasional PVC's caused by the ischemia, depending on frequency and multiplicity of origin, may forebode more serious arrhythmias.

Patient G.G. is a 45 year old Asian male who was doing heavy work when he was overcome by severe, crushing, anterior chest pain. Blood pressure was 110/40 on admission to the hospital.



Patient: G.G.

Rate: about 100/minute but variable.

Rhythm: Sinus Rhythm, somewhat irregular due to Sinus Arrhythmia.

PR less than .2 sec. (No AV Block). QRS less than .12 sec. (No BBB).

Axis: Left Axis Deviation (-30° to -60°).

Leftward rotation in the horizontal plane.

Hypertrophy: No atrial hypertrophy.

No ventricular hypertrophy.

Infarction: Significant Q waves in II, III, and AVF.

(coronary vascular status)

There are also very large Q waves in V₁, V₂, V₃, and V₄,
ST segments are elevated in V₁, V₂, V₃, and V₄.

Twaves are difficult to distinguish, but inverted T waves

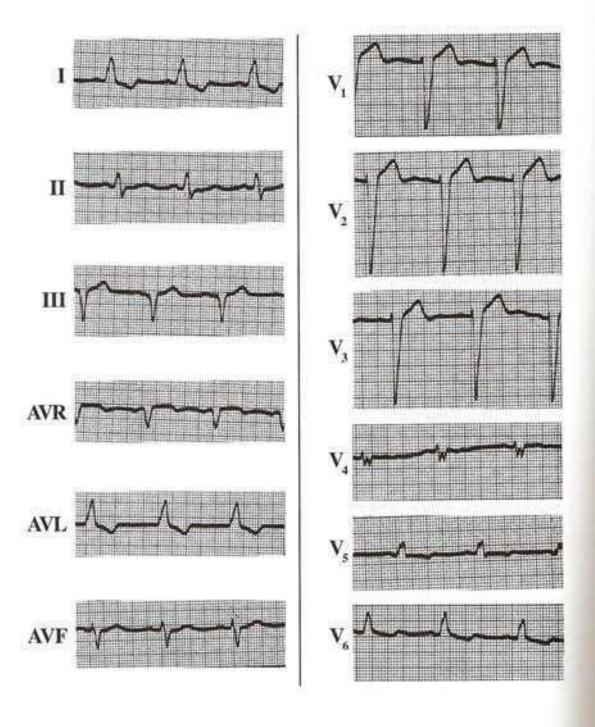
are noted in V, V, and V.

Comment: This patient has an acute antero-septal infarction, probably representing

an occlusion of the Anterior Descending branch of the Left Coronary. Generalized ischemia of the myocardium is evident by the flat-to-inverted T waves in nearly every lead. The old inferior infarction demonstrated on this EKG was noted on the patient's previous hospital record and is the documented etiology of his Left Axis Deviation (no Hemiblock). Note that the QRS becomes isoelectric between V₄ and V₅ but this is not within the normal (V₃, V₄) range; this represents minimal leftward rotation away from the septal infarction. Old EKG's

showed no anterior involvement on his previous admission.

Patient E.M. is a 65 year old Hispanic female. She was admitted to the hospital because of constant left chest pain for twelve hours. Blood pressure on admission was 110/75.



Patient:

E.M.

Rate:

60/minute

Rhythm:

Sinus Bradycardia

PR is about .2 sec. so there is probably a

first degree AV Block.

QRS is more than .12 sec. (it is .16 sec. wide). R,R' is present in

V₄ and V₆ so there is a Left Bundle Branch Block.

Axis:

Suggestive of Left Axis Deviation, but not reliable because of the

presence of Bundle Branch Block.

Hypertrophy: No atrial hypertrophy.

Ventricular hypertrophy is difficult to determine

because of Bundle Branch Block.

Infarction: Looronary

Q Waves: not a reliable criterion of infarction in the presence of

Left Bundle Branch Block.

vascular status)

ST segments: not reliable in the presence of Left Bundle

Branch Block:

T Waves are flat in V, V, and V, but not reliable with

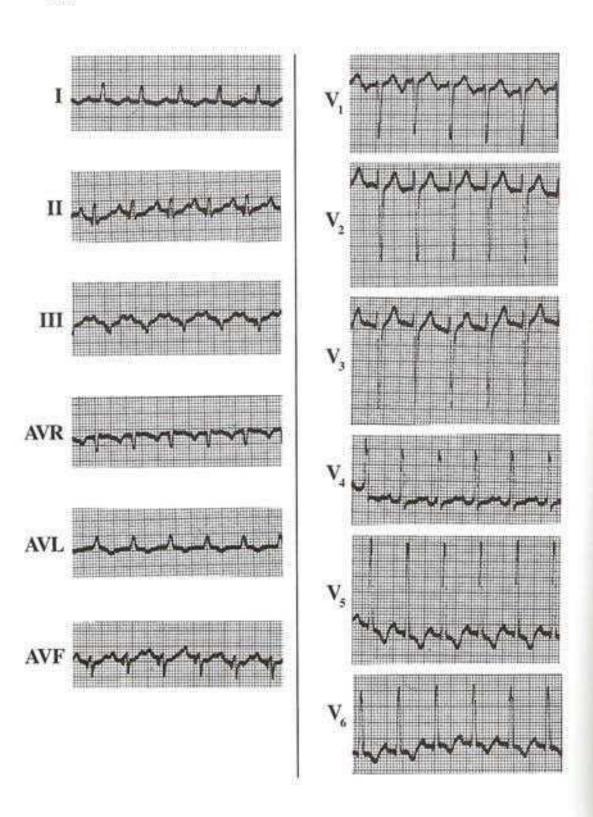
Left Bundle Branch Block.

Comment:

Enzyme studies confirmed a presumptive diagnosis of myocardial

infarction. The patient's chest pain made us suspicious.

Patient M.A. is a 75 year old black female with a long history of marked hypertension.



Patient:

M.A.

Rate:

about 125/minute

Rhythm:

Sinus Tachycardia

PR is less than .2 sec. (No AV Block). QRS is less than .12 sec. (No BBB).

Axis:

Left Axis Deviation (minimal amplitude of QRS in limb leads.)

make exact axis determination difficult).

No rotation in the horizontal plane.

Hypertrophy: Left atrial hypertrophy.

Left ventricular hypertrophy with strain.

Infarction:

Q waves are present in II, III, and AVF.

(coronary vascular status's ST segments: generally isoelectric (on baseline),

but V, and V, show strain pattern.

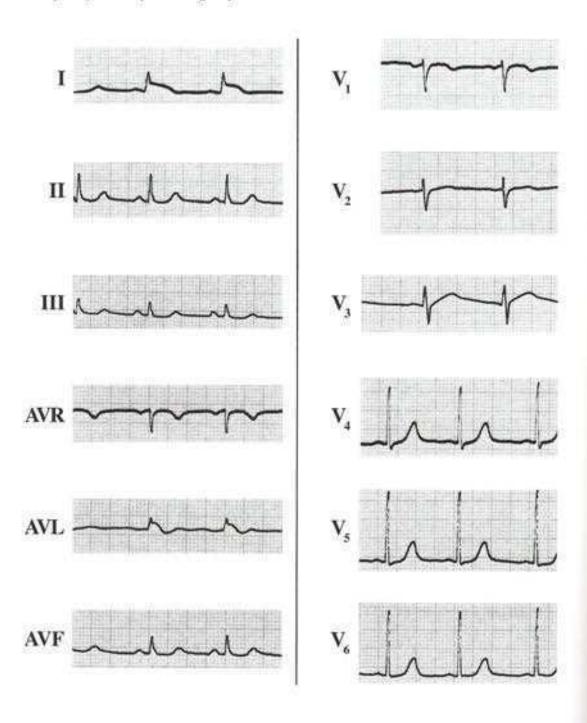
Twaves are inverted in I and AVL, and also in V, V,

Comment:

This patient has hypertrophy of both the left atrium and left ventricle with a left ventricular strain pattern. The patient also had an old inferior infarction. The Left Axis Deviation is caused by the Mean QRS Vector pointing away from the (old) inferior M.I. and toward the thickened left ventricle. It does not represent Hemiblock. There is currently (lateral) ischemia in the distribution of the Left Circumflex Coronary

Artery.

R.M., an anxious, obese, 57 year old white male whose law practice was failing, complained of "tight, squeezing" pain in his anterior chest, An electrocardiogram was quickly taken by an Emergency Medical Technician.



Patient: R.M.

Rate: 75/minute

Rhythm: Sinus Rhythm

PR.16 sec. (No AV Block). QRS.08 sec. (No BBB).

Axis: about +45° (Normal).

No rotation in the horizontal plane.

Hypertrophy: Possible minimal left atrial hypertrophy.

No ventricular hypertrophy.

Infarction: Q waves: no significant Q waves.

(commary ST segments: elevated 2+ mm. in I and AVL.

status) T waves: inverted in I and AVL.

Comment: It is interesting that in this innocuous appearing EKG there is a subtle-

non-Q wave infarction in the lateral left ventricle, which very soon developed into a serious lateral infarction, Symptomatology suggestive

of M.I. always must be investigated and scrutinized.

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