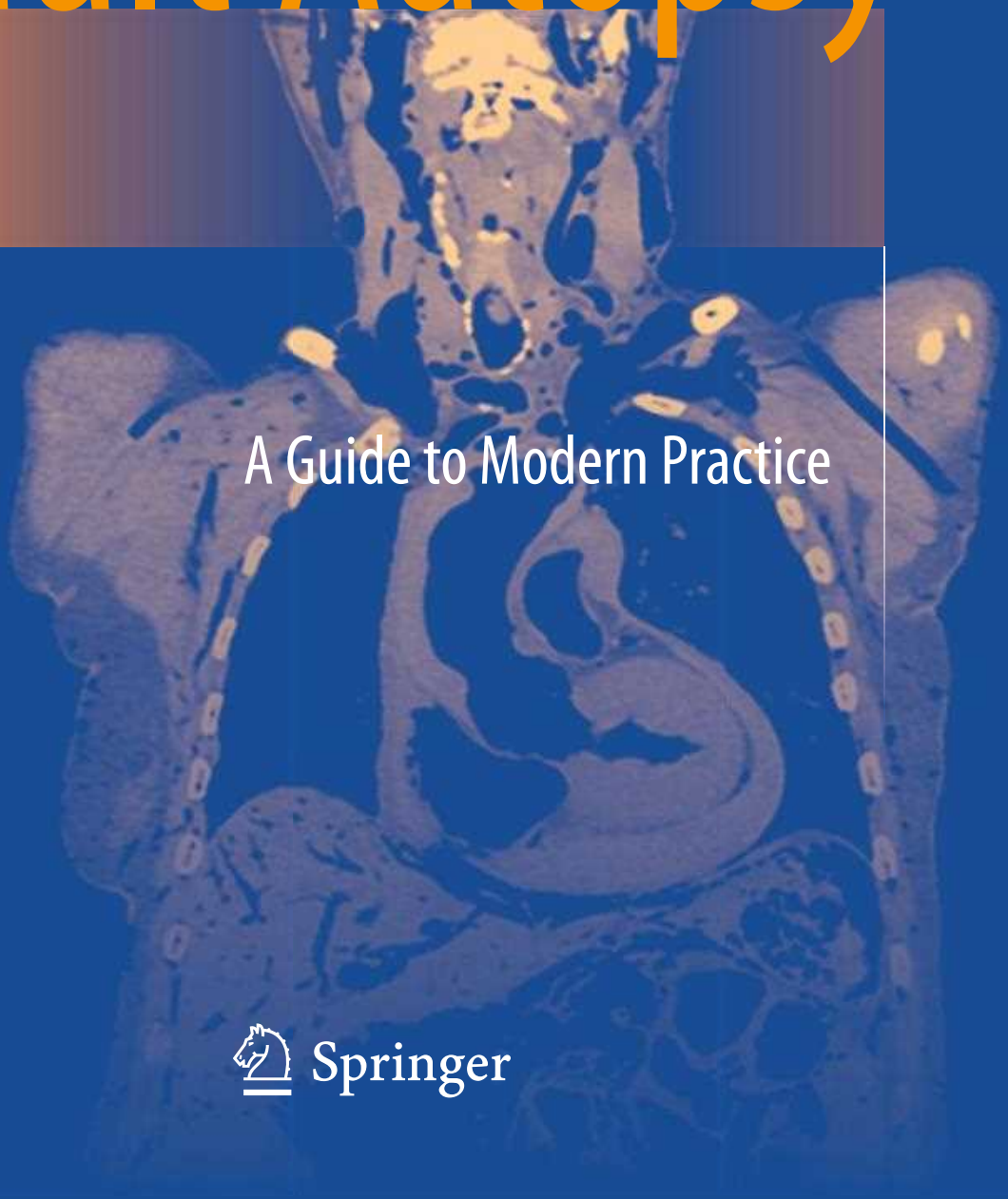
S. Kim Suvarna

# Atlas of Adult Autopsy

## A Guide to Modern Practice



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# Preface

The autopsy has been declining, not only in the UK but also across the rest of the developed world. This decline reflects advances in ante-mortem special tests (blood tests, surgical biopsies, various ante-mortem radiological techniques, etc.) that provide a much better insight into internal pathology than previously existed. Another factor has been the objections from some religious groups and the public disquiet about the practices of a few pathologists who have created a scandal at some centres, with the result that a number of 'consented' autopsies, relying on permission from relatives, have fallen. Contrastingly, and certainly in the UK, the medicolegal autopsy has continued to be used widely.

Unlike virtually every other aspect of medical practice, the invasive autopsy is still similar to the autopsies performed historically (certainly over the last 100 years or more). It is still considered to have a vital place in terms of the evaluation of ante-mortem disease and can highlight undeclared pathologies. The autopsy also has a role in the teaching of pathologists and students of medicine, in providing access to tissues for research, in helping with medicolegal queries, and in validating ante-mortem tests.

What has changed in the past century is that the macroscopic autopsy is now assisted by a variety of special tests. Furthermore, the autopsy can be augmented not only by the standard of microscopy but also by immunohistochemistry, microbiology (for bacteria, fungi, viruses, and other organisms), serological studies, photography, genetic studies, and mass spectroscopy, to name but a few of the available tests—and all can be recorded by photography.

It can be argued, however, that the autopsy currently is at a crossroad. With the better understanding of disease driven by molecular ante-mortem tests and modern imaging, there is a view that the 'gold standard' invasive autopsy may be less relevant. Certainly, digital autopsy techniques, especially CT radiology, are increasingly coming into play and diminishing the need for invasive studies.

However, if the autopsy is in decline, then the opportunity to learn by seeing and doing will diminish. In an arena where competence is recognized to be experience driven, this could be a problem. The aim of this book is therefore to provide a training standard of the protocol for an autopsy and to provide insight into the pathological lesions that may be found at autopsy. It attempts to deal with the various aspects of the autopsy in terms of grouped areas for dissection and special investigations. It also endeavours to cover the basics of forensic pathology, the radiological autopsy, and toxicology. It is hoped that it will both support students and provide trained practitioners with a ready reference.

The images and data in a book of this size cannot be all encompassing, but the majority of common lesions are displayed, along with some rarities. Most of the images are derived from autopsy cases, with a few surgical macroscopy images and some histology. Most images are largely left to speak for themselves but, where provided, the red reference bar is 10 mm.

Suggestions for further reading and references also are provided. One is mindful that autopsy pathology is often the same pathology as seen in the living, but simply demonstrated in the deceased. It follows that a good grounding in general surgical pathology is required to be a competent autopsy practitioner. There are so many books one could recommend in this arena, but only a few are cited, and it is left to readers to select their favourites to add to the understanding of autopsy cases.

It is also important, given occasional scandals (Bristol, Alder-Hey) and conflicts with social and religious custom, that each autopsy is appropriate and respectful. It is vital to remember that the deceased was someone's mother, father, daughter, or son. I therefore believe that the deceased should be afforded the same status as any other patient. Due respect must always be given, and one must have an open attitude about discussing cases with the bereaved and with medicolegal agencies, other relevant medical authorities, family practitioners, and society in general. That does not mean open access, and it is important that the autopsy is not abused for inappropriate commentary or gratuitous display.

Knowledge of the method by which one undertakes a high-quality autopsy will undoubtedly remain part of twenty-first-century medical practice, but this expensive and time-consuming technique needs to find its place in the ever-increasing range of medical investigations that can be performed. Ultimately, it is hoped that this atlas will be of use as a reference for the next generation of pathologists and anatomical pathology technicians, as well as for medicolegal practitioners and others.

Sheffield, UK S. Kim Suvarna

# Acknowledgements

The autopsy, and indeed the training in this area of medical practice, for me has always been a particular area of interest. This aspect of pathology straddles consent and medicolegal autopsy practice and has been a bedrock of understanding disease for the past few centuries. Despite this, it is clear that the classic dissection-style autopsy is in decline and that pathologists are coming to active practice with far less experience than previous trainees enjoyed.

This book is therefore aimed at providing a manual of current practice and knowledge resource for case examination in the form of an atlas. It is primarily aimed at practising pathologists, particularly in training grades. It is also hoped that this book will be of interest to those working as anatomical technicians in autopsy suites, as well as parties with a legal interest in autopsy practice. I would hope this atlas will provide sufficient resource for those approaching the practical examination of autopsy practice assessment in the UK and the equivalent farther afield.

It goes without saying that any publication is not the work of solely one individual, and grateful thanks are expressed to all who have assisted this project. As always there are so many to mention, and I can only use so much space. So, to start, I am strongly mindful of the enthusiasm and collaboration from co-authors, the support from Springer staff, and the vital contribution made by other autopsy practitioner colleagues and the trainees within Sheffield, UK. Specific mention must go to the mortuary staff in Sheffield: Maxine Coe, Joanne Dawn, Nigel Prestidge, Sally Smith, Tim Wild, and Paul Wood, as well as Astrid Rowlands, Neville Udall, and Neil White. I am greatly indebted to Seonaid Ashby for the secretarial support; without whose help the book preparation would have been considerably slower and more painful! Additional thanks are expressed to the various coroners across the UK, but in particular Mr. Dorries (HM Coroner for South Yorkshire, West). The coroners have provided me with the opportunity to work and train with very interesting cases.

One must not lose sight of the fact that the images from this book are almost all derived from the deceased—with some surgical pathology cases. Immense gratitude is expressed to the relatives who have given consent for images to be retained and used to teach the pathologists of the future. This generosity cannot be understated.

Finally, my thanks also go to Grace, Miranda, and Elara. Without their understanding and generous support, all this would not have been possible.

Sheffield, UK
March 2016

S. Kim Suvarna

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# General Considerations and Safety

1

Maxine S. Coe and S. Kim Suvarna

Preparation for the autopsy and consideration of the case, before entering the autopsy room or putting knife to skin, are key in getting the best results from the examination. This chapter commences with the components of an instruction to perform an autopsy. The relevant paperwork (usually legal and/or consent permissions) is discussed.

The practical realities of completing an autopsy require a modern, well-lit, and dedicated room or facility for this examination; views of some facilities are shown. Other requirements are appropriate clothing, protective face masks, gloves, and other body-wear. The standard tools and the workstation are presented. This process permits a safe working environment for the autopsy practitioner and will enable the pathologist or technician to efficiently examine the case.

All these efforts will maximize macroscopic information, but additional tests are often required to realize the full potential of the autopsy. The role of specialist investigations (histology, toxicology, microbiology) is described, and macroscopic photography is also considered. Finally, the methods for recording the data derived are described.

## Paperwork: Permissions and Instructions

Before starting the autopsy, it is vital to confirm that there is a clear instruction to perform the examination from the relevant authorities. This data may come from legal sources (*e.g.*, for the United Kingdom, HM Coroner, Procurator Fiscal), the police, or another state body. Alternatively, it may be driven by a request from the medical staff or relatives, in the form of a "consent" autopsy.

## Identification

As with all medical procedures, identification of the body in question is required, using a variety of different case identifier data. The name, date of birth, and relevant hospital number may be important, but other identifying information can come from third parties (confirming the identity of the deceased). Specific identification marks, including piercings, tattoos, scars, and other body features, may be relevant in allowing certification of identity. Correlation with a previous photographic record (*e.g.*, passport, identity cards, or other resources) is sometimes of use.

Ideally, bodies are retrieved soon after death, and little decomposition will have occurred, but some bodies will have been significantly degraded, with autolysis and maggot infestation. In such circumstances, other data such as dental record comparison, registry numbers of implants, or evidence of prior disease events or correlates may be needed.

Once confirmation of identity has been established, the body is 'booked-in'. This necessitates the assignment of the body to a unique case identification number (*e.g.*, 679H/16, reflecting the case number given, with a check letter and the year).

The body, usually identified by a name tag or bracelet, is then stored to await examination. Bodies are normally stored in refrigerators, generally at 4° Celsius (40° Fahrenheit). Alternatively, if storage is needed for a prolonged period, then freezing at −20° Celsius (−4° Fahrenheit) is appropriate.

## The Background Story

The examination generally follows review of information from those requesting the examination, and/or consideration of the clinical record and medical history data. This review may yield not only information to guide the examination in

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terms of likely pathology but also information that may be pertinent to infection risks for those working in the autopsy suite. Potential risks in the form of various viral infections (*e.g.*, drug users, those brought back from abroad, etc.) may be important. Nevertheless, *in all cases* the various risks of infection must be assessed before examining a body.

Cases with Category 1 and 2 infections can proceed without special measures. Category 3 risk cases need additional clothing and protection. Ideally, such cases should be performed by those with appropriate experience. Category 4 risk cases should be examined only by experienced practitioners in specialist facilities with the appropriate techniques and safety measures. It cannot be over-emphasized, however, that caution with all cases must be the standard for working in the mortuary, and relevant immunizations are mandatory for all personnel.

## Getting Ready

One cannot perform autopsies wearing one's own clothing. The mortuary must provide clothing designed to be comfortable and yet protective for the staff. At this point, it is worth stressing that there should always be at least two people in the autopsy room when it is active, for safety reasons.

Fresh clothing (usually theatre scrubs, stored in dedicated changing rooms with wash and shower facilities) should be worn, with an outer gown. Layers of flexible waterproof and stab-resistant (Kevlar/chainmail) gloves must be worn. Additional waterproof lower arm covers are recommended. Face covers (generally masks and visors) are required, and one may also select hair nets or caps. Specialist masks may be required for various infection risks. A waterproof apron must be worn, covering the torso and legs, down to below the top of waterproof wellington boots (with toe protection).

## The Autopsy Room, Workbench, and Autopsy Table

The autopsy room, along with adjoining dedicated rooms, must be available. The layout of an autopsy room needs good lighting, down-draft ventilation, running water, work surfaces capable of being chemically cleaned and sterilised, nonslip flooring, and space to work.

The workstation should be equipped with a range of scalpel blades and other long knives. There should be scissors (various sizes) and 'toothed' forceps. A ruler, measuring jug, and accurate electronic scales are vital. A range of bowls, ladles, string, wire-cutters, saws, shears, and other tools must be available.

A digital camera is particularly valuable in the autopsy room to record the stages of dissection as well as specific lesions or gross morphology. Imaging is of vital importance in some cases that have a legal bearing, such as surgical procedures with complications or retained foreign bodies.

## Additional Testing Materials

Within easy reach should be containers for swabs, culture bottles, toxicology kits, formalin pots, and tissue cassettes.

Histological examination is a common adjunct to the autopsy. This test requires the availability of formalin and appropriately numbered cassettes for securing the samples taken. The cassettes should be identified in terms of the case number and the cassette number (*e.g.*, case 679H/16, a1, where the designator a1 is the cassette number that can be specified with regard to the contents). The tissue type and number of tissue fragments should be recorded for the content of each cassette. Standard paraffin-based tissue processing is commonplace, but glutaraldehyde for electron microscopy may also be relevant. Handling tissues for frozen section must also be possible.

Microbiology samples may be taken in possible cases of bacterial, fungal, or viral disease. Those with a significant infection risk (*e.g.*, Category 3 risk) must be labeled accordingly.

Toxicology tests may also be needed, with samples of various body fluids (vitreous, blood, CSF, urine) and tissues being reserved for analysis.

Rarely, other specialist techniques find their way into the autopsy room. For example, samples may be taken for mass spectroscopy (*e.g.*, metal analysis) or serology (*e.g.*, hormone assay, serum tryptase).

## The Results

Examining a body will generate a lot of information, such as organ weights, pathology descriptions, and measurements. These must be annotated by tape or digital recording or by writing onto a standard proforma (see Appendix).

The paperwork or recording must itemize whether samples have been taken. If so, the type and number of samples should be stated. At this point, one may be able to provide a cause of death, which should be in the standard format (*i.e.*, 1a, 1b, 1c, and 2).

Figures 1.1, 1.2, 1.3, 1.4, 1.5, 1.6, 1.7, 1.8, 1.9, 1.10, 1.11, 1.12, 1.13, 1.14, 1.15, 1.16, 1.17, 1.18, 1.19, 1.20, 1.21, 1.22, 1.23, 1.24, 1.25, 1.26, 1.27, 1.28, 1.29, 1.30, 1.31, and 1.32 illustrate the points made in this chapter.

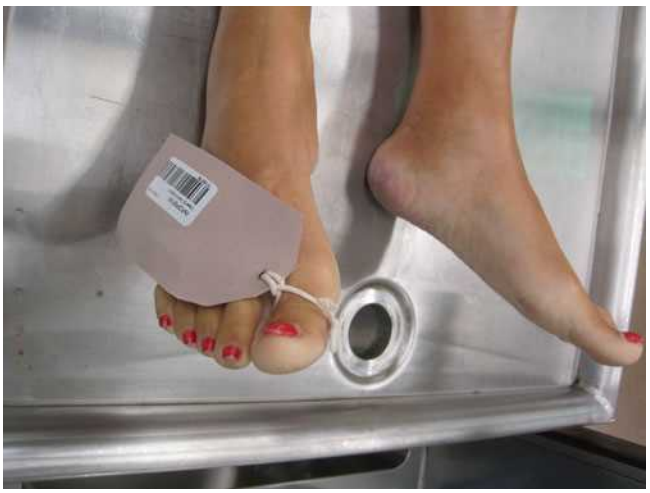

**Fig. 1.1** A toe tag attached to the body is seen with some of the numerical patient data of the deceased. The patient's name is on the reverse of this form

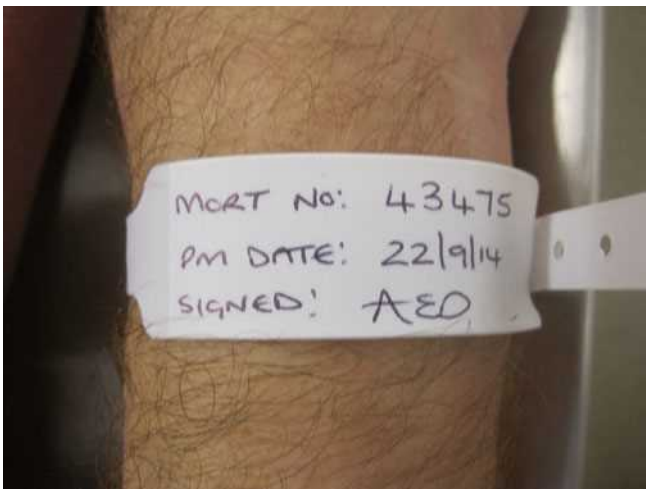


**Fig. 1.3** Additional data may be added to the body, indicating when the autopsy is due and the case number. The wrist or ankle band contains relevant details of the deceased

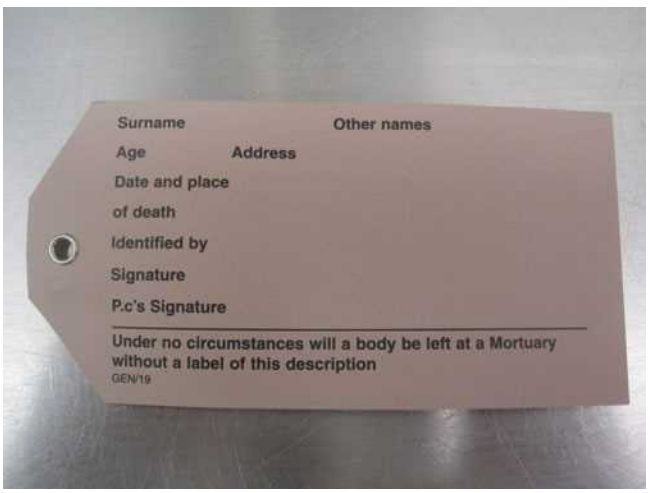


**Fig. 1.2** The tag should show relevant identifier data: full name, place and date of death, address, and confirmation as to how the identification was verified

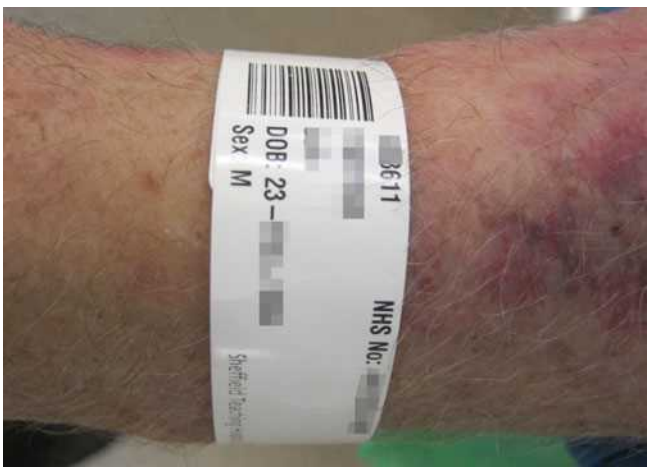


**Fig. 1.4** The arrival of a body from a medical unit often comes with bar-coded identifier wrist bands and other information. The name in this case has been obscured intentionally

**Fig. 1.5** Additional information confirming the person's identity may be seen in the form of external jewellery (*e.g.*, metal ear rings or piercings)

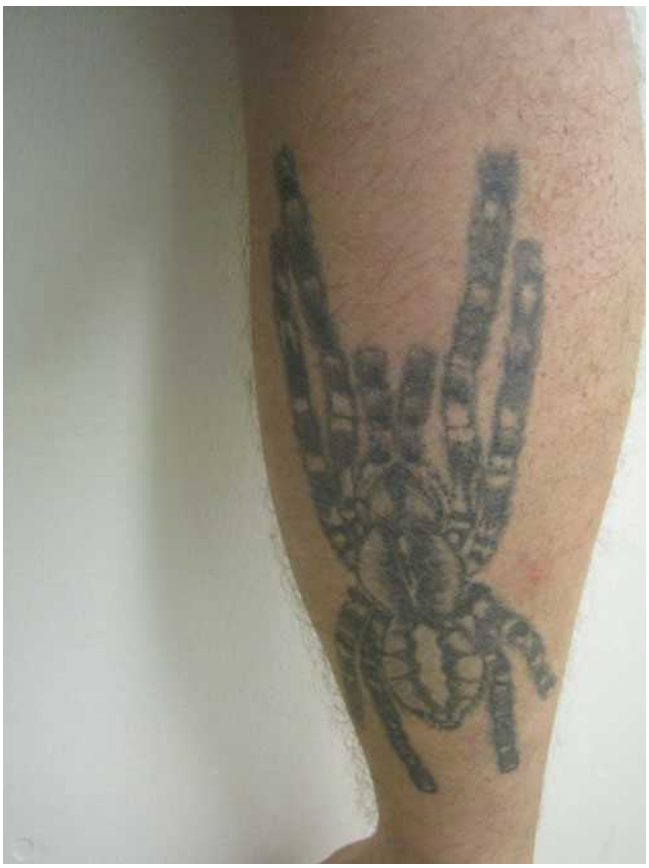

**Fig. 1.6** Tattoos are also a good identifier, particularly if unique or unusual. This leg tattoo of a spider is not a common design and may be helpful when correlating to other information about the deceased or photographs taken in life

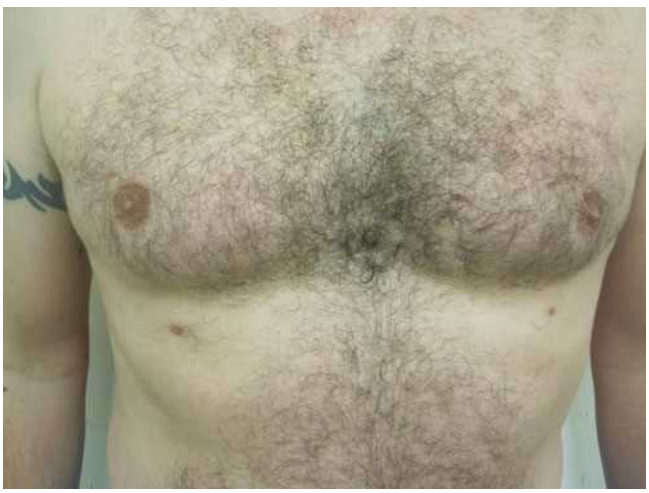

**Fig. 1.7** The body may have a variety of distinguishing marks that aid identification. This image shows the front of the chest with multiple nipples

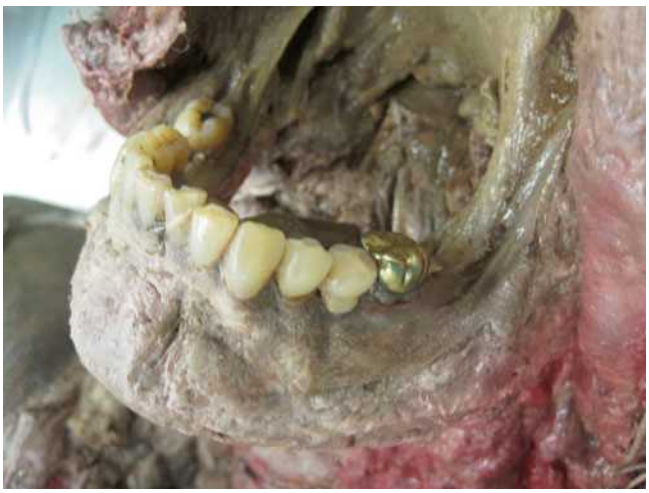

**Fig. 1.8** Even badly decomposed bodies may contain useful clues for identification, including dental work. Here the mouth is seen to have dental amalgam filling and a gold tooth

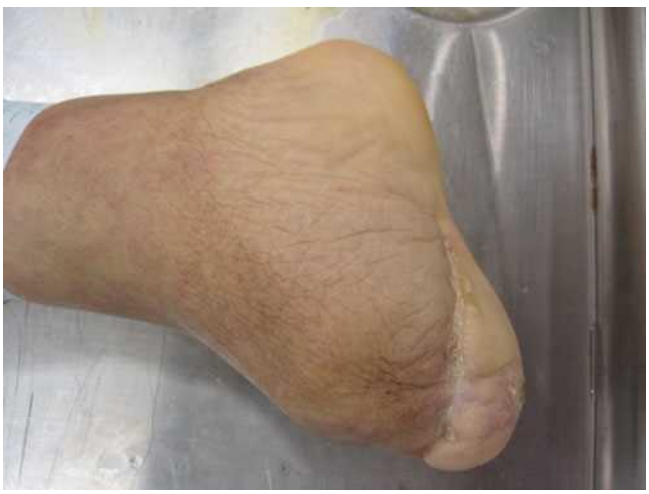

**Fig. 1.9** Amputations and other scars may serve to confirm identity, particularly when matched to hospital records

**Fig. 1.10** Often the body may be fully or partly dressed. The clothing can be mapped against witness information as to the clothes the deceased was last seen wearing. Precautions must be taken when removing clothing from a body. The pockets may contain sharp objects or may be covering jagged, protruding bone. One should use forceps to expose pocket contents

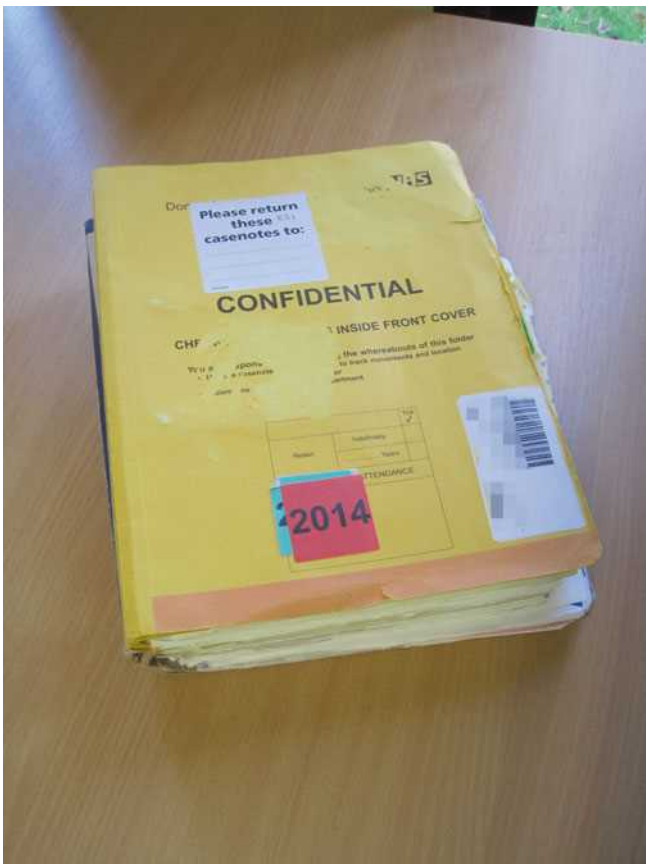


**Fig. 1.11** The notes of the deceased often contain abundant medical, nursing, and other data that help to identify the deceased. The clinical record may also point to hazards to be considered before the autopsy (*e.g.*, infection risks)


**Fig. 1.12** Body storage fridge containing horizontal tiers of roll in/roll out trays are seen within a refrigerated compartment. The trays travel with the body during the stay in the mortuary. These trays and bodies are usually arranged in tiers of three to five. The units have doors at the head and the feet ends and are run at a temperature of 4 °C (40 °F) or freezer temperature of −20 °C (−4 °F)

**Fig. 1.13** The acceptable minimum protective clothing to perform a post mortem: scrub (tunic and trousers); trousers tucked inside reinforced (toe-cap) wellingtons and plastic apron close to the floor; gloves and oversleeves. There should be a mask with an attached or integral clear plastic visor to cover the face

**Fig. 1.14** This photograph of a mask and inbuilt visor shows the importance of protecting the eyes and the mouth. The visor has prevented small blood splashes from striking the face

**Fig. 1.15** This image shows a specialised mask being used in Category 3 risk cases, with separate clear goggles and a hair net. Covering the hair provides additional protection

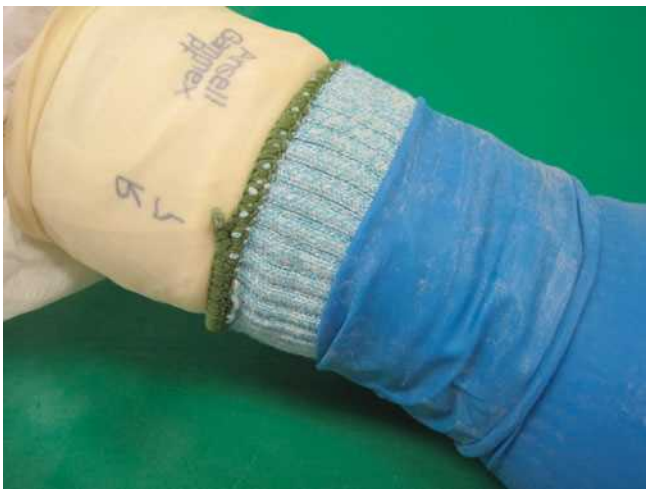

**Fig. 1.16** Layering of the gloves limits the risk of cutting and of tissue fluid penetration to the operator's skin. Here the inner beige surgical glove sits next to the skin. Then there is a Kevlar (or chain-mail as an alternative) glove. Finally, there is a heavy-duty waterproof blue glove. This ensemble will interlock with the oversleeves and afford good protection for those working with body dissection

**Fig. 1.17** Dealing with high-risk cases may require additional measures for protection. Here, a disposable suit is worn over the standard mortuary wear. In cases with high risk of infection, if one wishes to use a battery-operated full facial unit, it should be put in place before entering the autopsy room

**Fig. 1.18** Moving bodies around in the mortuary requires safe working practices to avoid mortuary staff injury. Here a hydraulic unit is employed to allow the fridge tray and body to be moved safely to the operating station

**Fig. 1.19** The general layout of a post-mortem facility, with a central dissecting station. The tray on which the deceased rests is not yet in place; it is still in the fridge. Indeed, the tray stays with the individual to avoid risks associated with lifting heavy objects. There is good lighting, drainage, and access to fridges and other equipment. For illustration purposes in this view, there is a ladder for an assistant to take photographs during dissection. Dissection tools and other equipment are on a mobile trolley adjacent

**Fig. 1.20** The dissection trolley layout shows the instruments that can be used during the autopsy

**Fig. 1.21** After evisceration, the organs are often carried to a separate work area, allowing the autopsy assistant to start reconstructing the body. The work area should be well lit, with down-draft ventilation and good access to running water and drainage. All surfaces (as with other areas in the mortuary) are capable of chemical cleaning and sterilisation later

**Fig. 1.22** The pathologist dissection bench is shown with the essential instruments for examinations. These include a large or long knife, scissors, small and large scalpels, forceps, sponge, and ruler. Also needed are local access to running water, a measuring jug, and histology cassettes


**Fig. 1.24** This two-tiered sample trolley contains materials for additional tests. The upper shelf has syringes, needles, blood culture bottles, and swabs for bacteriology and virology. The lower tier holds specimen pots for histology samples, together with containers or tubes for blood, urine, tissue, and faeces

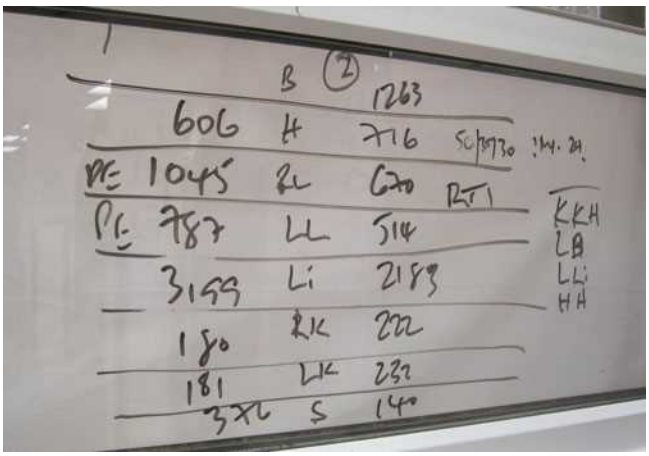

**Fig. 1.23** Recording of weights and other notes during the examination can be made on wipe-clean surfaces using nonpermanent markers

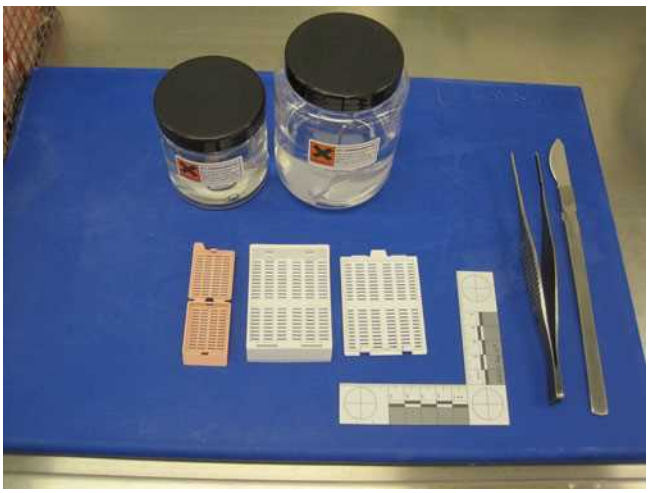

**Fig. 1.25** Histology cassettes of different sizes are used, to ensure that the amount of tissue taken can be processed appropriately. The smaller (standard) pink cassettes are generally used for thumbnail or smaller tissue fragments. The larger white cassettes are useful for larger samples that merit large area architecture analysis (*eg*, samples of brain, lung, or heart). Sharp scalpel blades and forceps should be used when samples are taken for histology

**Fig. 1.26** The samples of tissue for histology may be placed in cassettes in single pieces, or, as in this case, as multiple fragments. The tissue samples should be recorded ideally. This multiple sample technique means that special stains may be used on multiple bits of tissue with only one additional slide per stain being needed

**Fig. 1.28** A messy work station (here mocked-up for illustration purposes) poses a risk to others. Aside from the widespread contamination of the work area, the discarded and contaminated blades pose a significant hazard. This is not good practice

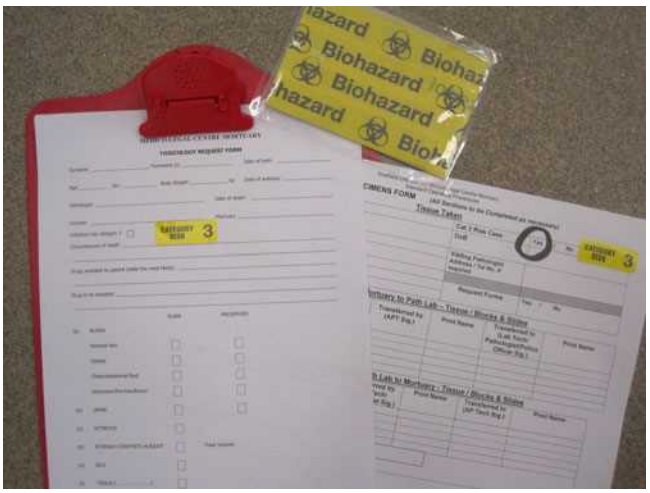


**Fig. 1.27** Any samples that are taken must have the correct corresponding paperwork to accompany the specimen to the laboratory. High-risk cases must be labelled with high-risk (yellow) stickers to alert laboratory staff to risks and dangers


**Fig. 1.29** Different types of cameras can be used in the autopsy room. In the recent years, even compact digital cameras have good resolution and autofocus, comparing favourably with single lens reflex type cameras. For health and safety reasons, cameras ideally should not be removed from the autopsy room. Data should be remotely downloaded and stored onto a secure (often encrypted) computer

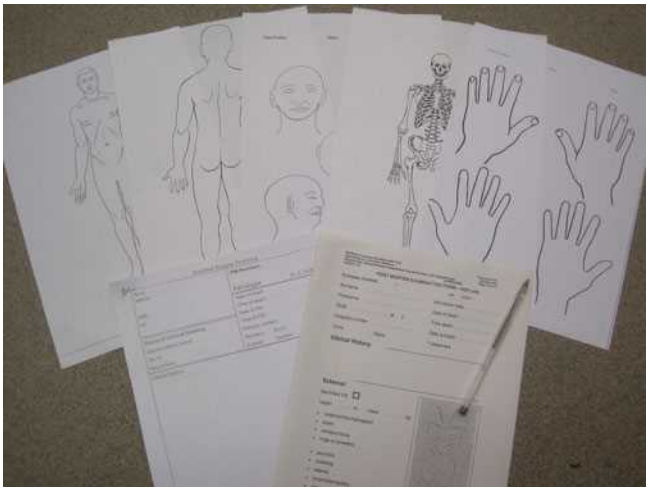

**Fig. 1.30** Paperwork at the completion of the examination will vary depending on the standard operating procedures of the department. The basic paperwork includes sheets for data and results, and anatomical diagram proformas

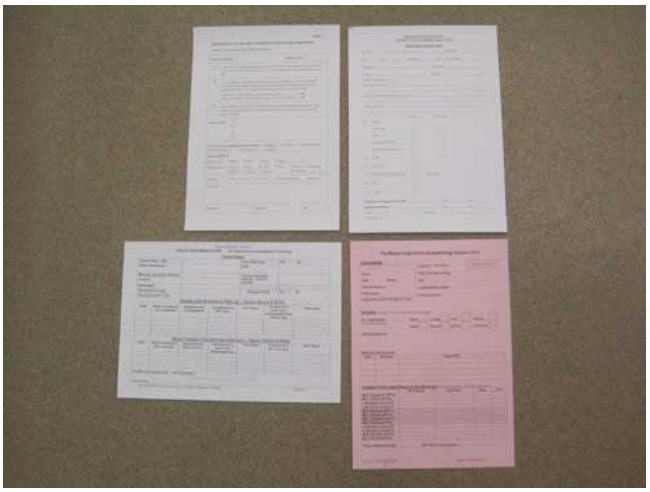

**Fig. 1.31** Other paperwork includes 'cause of death' documents, histology tissue sheets (*i.e.*, the record of tissues sampled), microbiology specimen forms, toxicology sample documents, and other items

**Fig. 1.32** After the examination, bodies are reconstituted and returned to the shelving in the fridges. Those with significant tissue degradation and decomposition may require heavy bagging ('cracker-wrapping') to avoid tissue leakage. Contaminated waste materials should be placed into clearly marked (in this case, orange) bags for incineration

## Suggested Reading

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# External Examination

# 2

Kirsty L. Lloyd and S. Kim Suvarna

## Introduction

The aim of the external examination is to identify the deceased and to formally document relevant positive and negative findings related to natural disease or injury. The examination of the outer surfaces of a body provides supporting information for later clinicopathological correlation, and it may (in its own right) help to determine the cause of death.

The 2006 report from the National Confidential Enquiry into Patient Outcome and Death (a national review of the UK autopsy system) [1] states 'Before evisceration of a body, the pathologist must inspect the body first. This is to confirm identity, to observe any external features that might modify the process of examination and to consider the possible need for a forensic examination.' Guidance from The Royal College of Pathologists [2] also emphasises that this is a vital part of the autopsy.

The images in this chapter are organised in the manner of one going around the body at the start of an autopsy examination:

- Head (Figs. 2.1, 2.2, 2.3, 2.4, 2.5, 2.6, and 2.7)
- Face (Figs. 2.8, 2.9, 2.10, 2.11, 2.12, and 2.13)
- Eyes (Figs. 2.14, 2.15, 2.16, 2.17, and 2.18)
- Neck (Figs. 2.19, 2.20, 2.21, 2.22, 2.23, 2.24, 2.25, 2.26, 2.27, and 2.28)
- Upper limbs (Figs. 2.29, 2.30, 2.31, 2.32, 2.33, 2.34, 2.35, and 2.36)
- Hands (Figs. 2.37, 2.38, 2.39, 2.40, 2.41, 2.42, 2.43, and 2.44)
- Chest (Figs. 2.45, 2.46, 2.47, 2.48, 2.49, and 2.50)
- Abdomen (Figs. 2.51. 2.52, 2.53, 2.54, 2.55, 2.56, 2.57, and 2.58)
- Groin and external genitalia (Figs. 2.59, 2.60, 2.61, 2.62, 2.63, 2.64, and 2.65)
- Lower limbs (Figs. 2.66, 2.67, 2.68, 2.69, 2.70, 2.71, 2.72, 2.73, 2.74, 2.75, 2.76, 2.77, 2.78, 2.79, 2.80, 2.81, 2.82, 2.83, and 2.84)
- Feet and toes (Figs. 2.85, 2.86, 2.87, 2.88, 2.89, and 2.90)
- Lateral aspect (Figs. 2.91 and 2.92)
- Back (Figs. 2.93, 2.94, 2.95, and 2.96)

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S.K. Suvarna (ed.), *Atlas of Adult Autopsy: A Guide to Modern Practice*, DOI 10.1007/978-3-319-27022-7_2

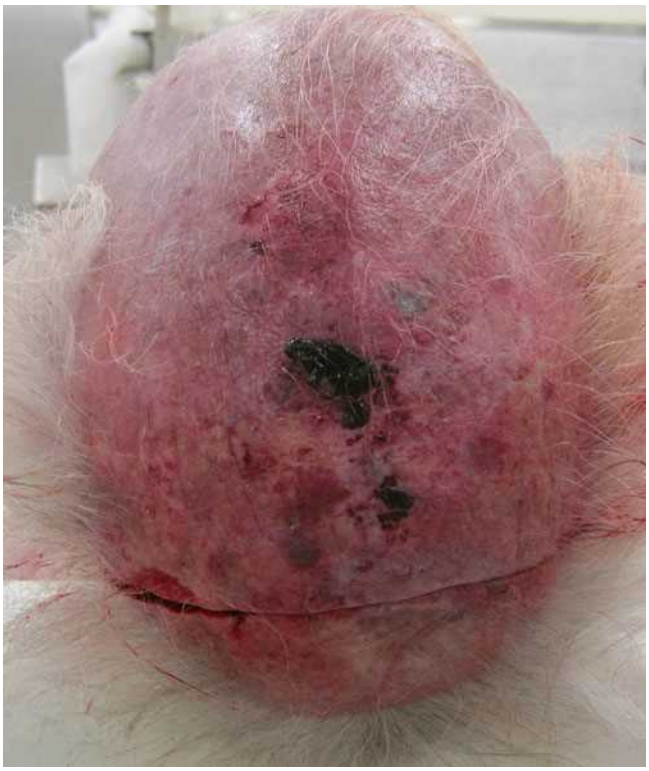

**Fig. 2.1** When there is not much hair, scalp injuries are visible. It is important to palpate the skull to identify fractures or surgical implants

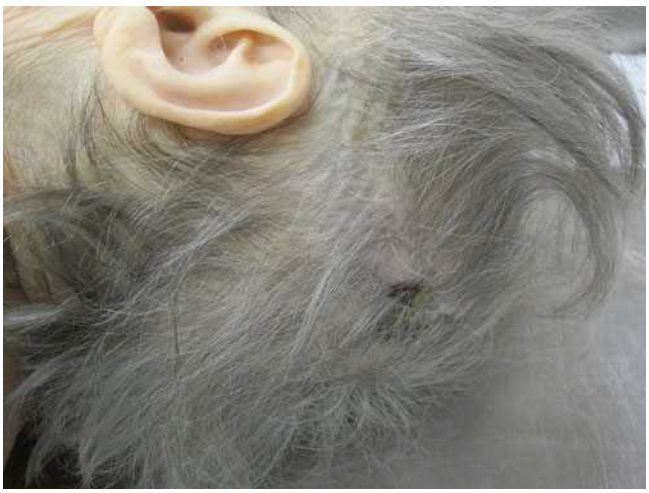

**Fig. 2.2** Examination of the head. Careful examination can reveal features of prior neurosurgery. Here palpable and visible in the scalp is a metal mesh support used to maintain skull integrity after significant injury. The old craniotomy scar was hidden under the hair

**Fig. 2.3** Staple closure over surgical incision following neurosurgery. This wound is fresh and congealed blood is seen around the staple line

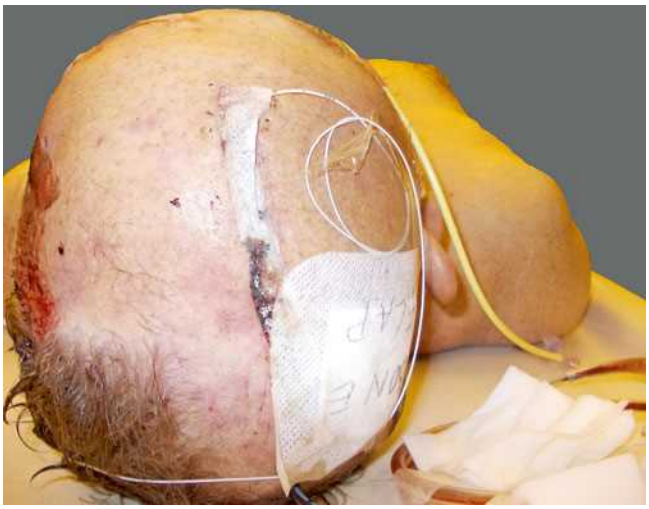

**Fig. 2.4** This is an intracranial pressure monitor and ventricular catheter. Part of the wound is visible and healing. The dressings are removed but the catheter remains in place

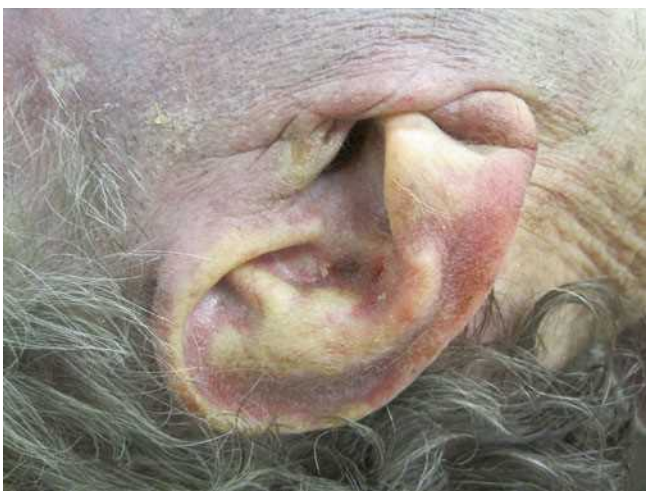

**Fig. 2.5** Diagonal earlobe creases are considered a potential indicator of coronary artery disease [15, 16] and cardiovascular causes of death [16, 17]

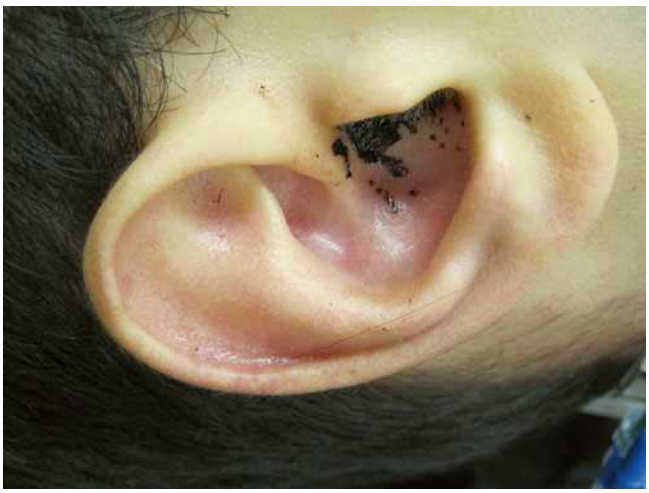

**Fig. 2.6** Cerebrospinal fluid and/or blood in the external auditory meatus/ear canal often indicates a basal skull fracture. However, one must check that the blood has not come from a facial or scalp wound

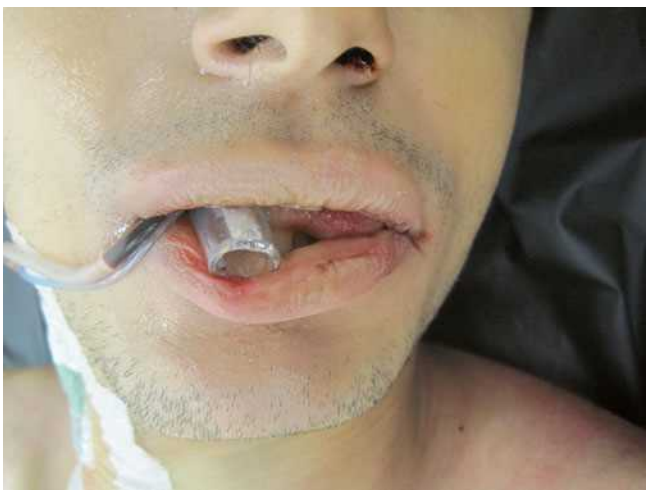

**Fig. 2.7** Endotracheal tube. The end of the tube has been trimmed to aid evisceration. Note that the narrow, clear inflating tube remains intact to help prevent the tube from becoming dislodged

**Fig. 2.8** Face trauma. Injury resulting from high-velocity accidents leads to loss of skin, soft tissue, and skeletal integrity

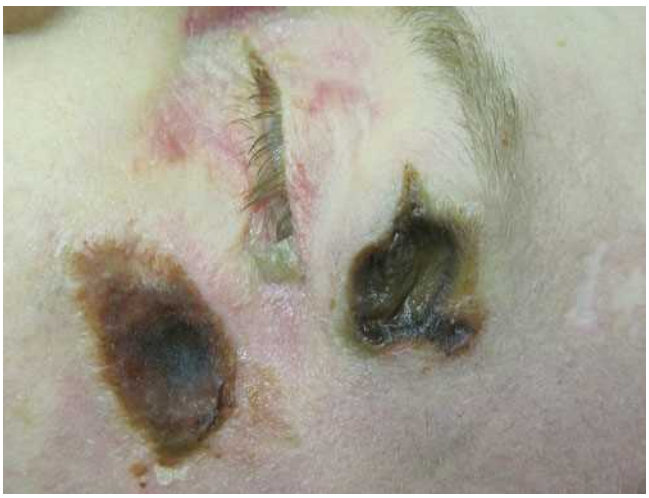

**Fig. 2.9** Post-mortem injury to the face. The face may show signs of injury. These pressure areas over bony prominences occurred after death. One notes that the edge of the area lacks a vital skin reaction, confirming post-mortem injury

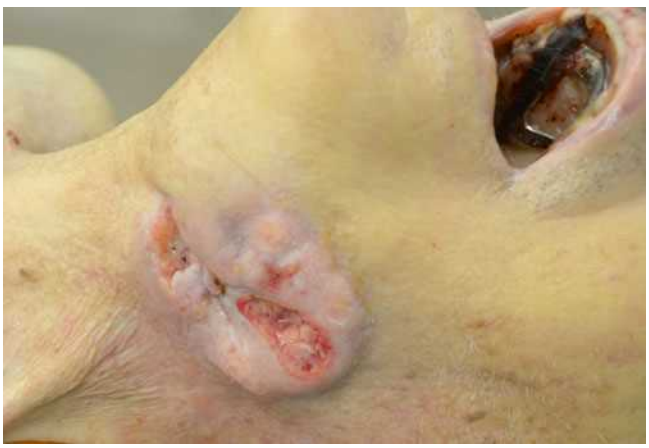

**Fig. 2.10** The skin is examined for tumours: the site, size, and appearance are recorded. This is a recurrent salivary gland neoplasm showing local recurrence affecting the tissues at the jawline

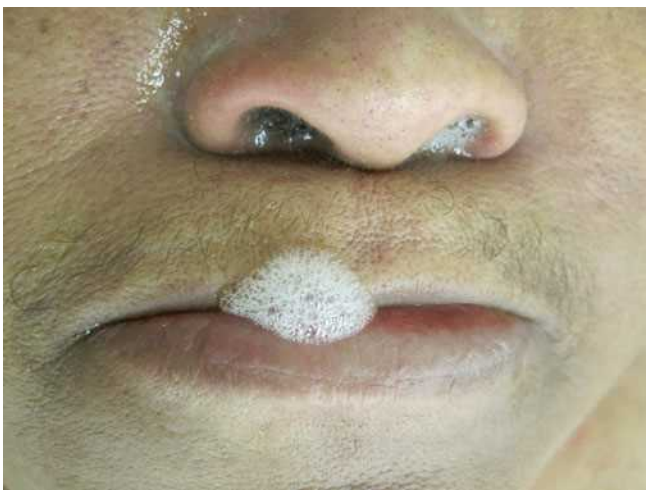

**Fig. 2.11** There is frothy fluid in the nose and mouth of this patient, who died of acute left ventricular failure. With drowning, a frothy 'plume' is also often seen in the oropharynx. There may be some blood staining

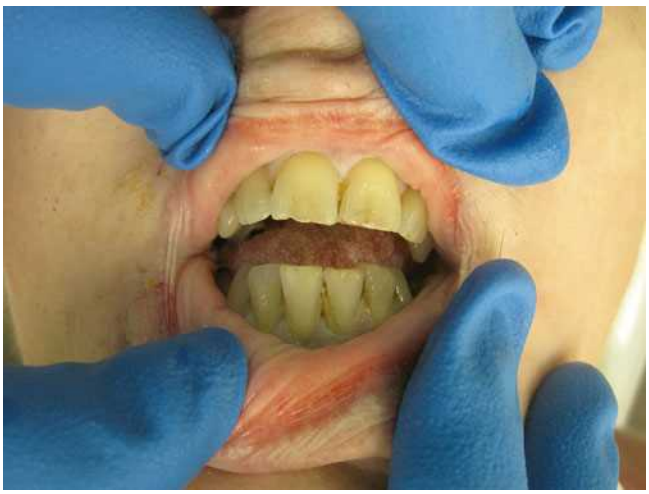

**Fig. 2.12** The presence and condition of teeth or dentures is recorded. Loose-fitting dentures may be a feature of weight loss

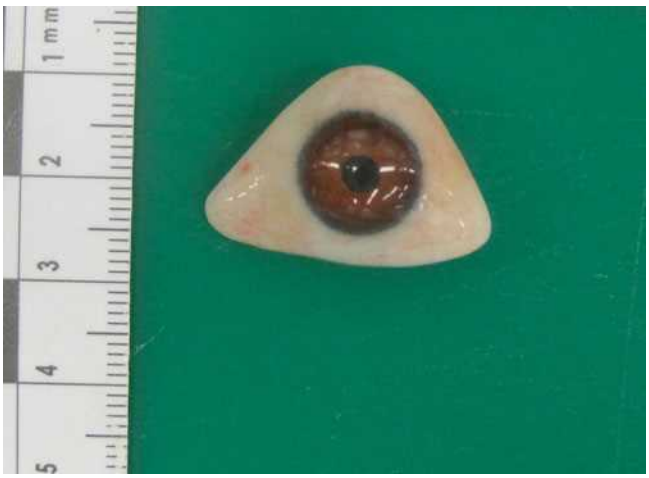

**Fig. 2.15** False eye. It is not necessary to remove the prosthesis, but it is important to identify this, if present

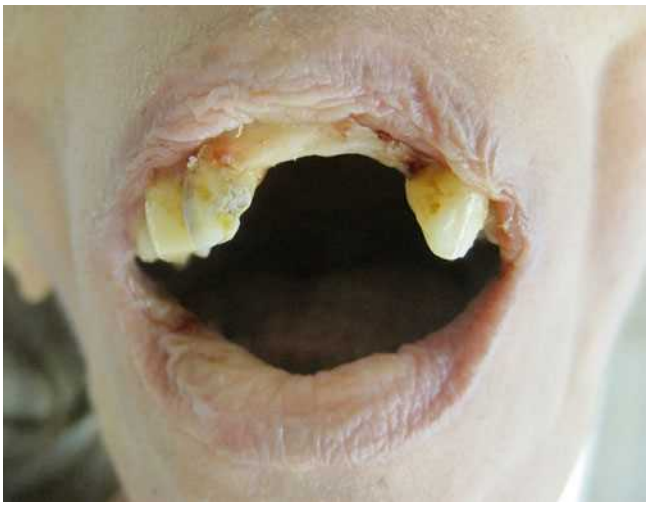

**Fig. 2.13** Severe dental caries are a source of bacteraemia and can cause generalised sepsis and endocarditis

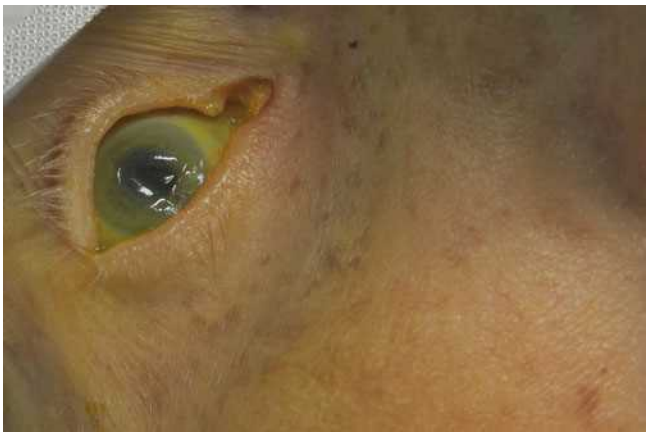

**Fig. 2.16** Jaundice of the sclera. Yellow discolouration of the sclera due to jaundice

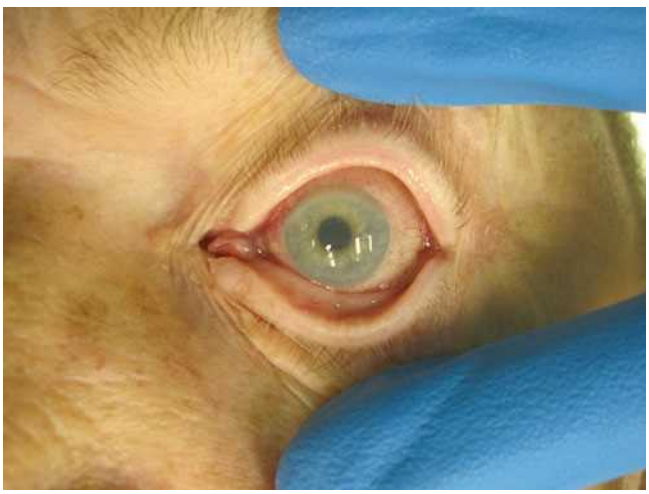

**Fig. 2.14** The skin around the eye and the iris, conjunctiva, and sclera are examined for haemorrhages or jaundice. The eyelids need to be partly retracted

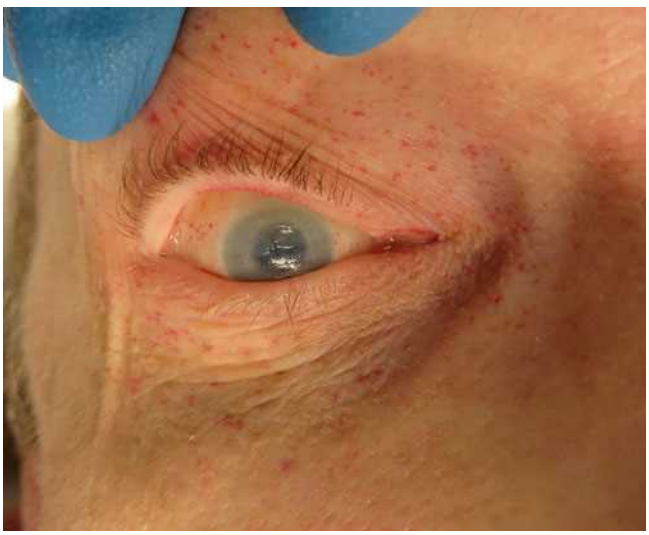

**Fig. 2.17** The rupture of scleral small capillaries may cause petechial haemorrhages, which are associated with venous overpressure and may reflect severe coughing or asphyxia due to respiratory disease or cardiac arrest

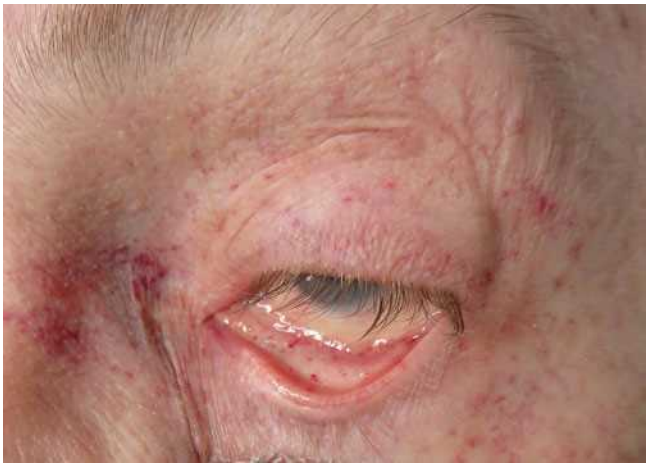

**Fig. 2.18** The eyelids are retracted to examine the conjunctiva, which in this case reveals petechial haemorrhages

**Fig. 2.19** Noose-type ligatures around the neck must be documented before removal, with examination of the ligature mark after the noose is removed (See also Chap. 12)

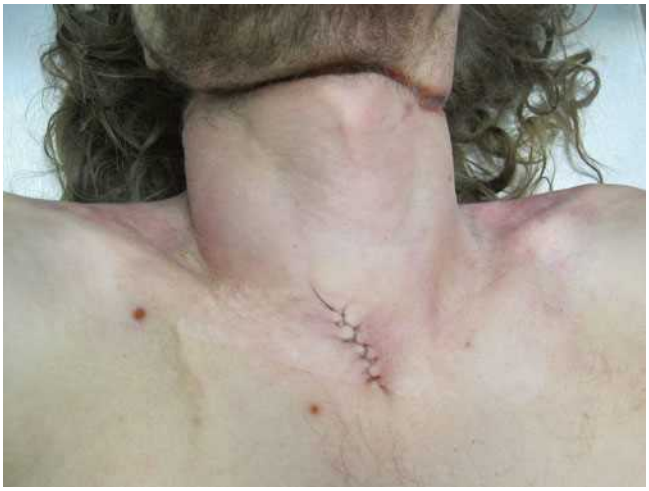

**Fig. 2.20** Ligature mark and embalming incision. Following removal, the marks made in the high neck by the ligature in a case of hanging can be examined. This image also shows a sutured low neck incision from embalming

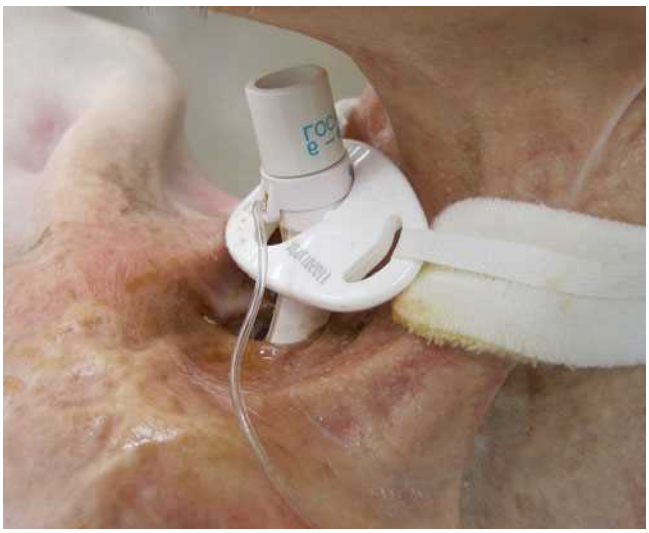

**Fig. 2.21** Tracheotomy tube. These tubes have an outer cannula with an inflatable cuff and an inner cannula that can be removed for cleaning. The device is often removed prior to evisceration, making sure to examine the tube and distal end for obstruction (mucus, inflammatory matter, tumour, etc.)

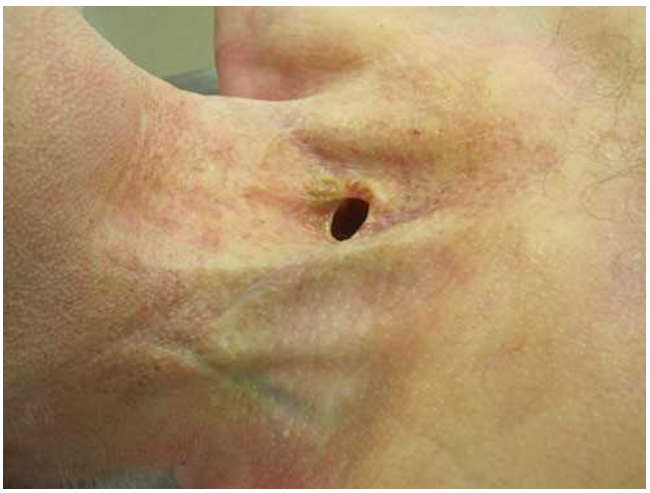

**Fig. 2.22** A surgical airway (tracheostomy) may be seen in patients for a wide number of underlying conditions, both acute and chronic. Fistula formation between the posterior tracheal wall and the oesophagus can occur in longstanding tracheostomies

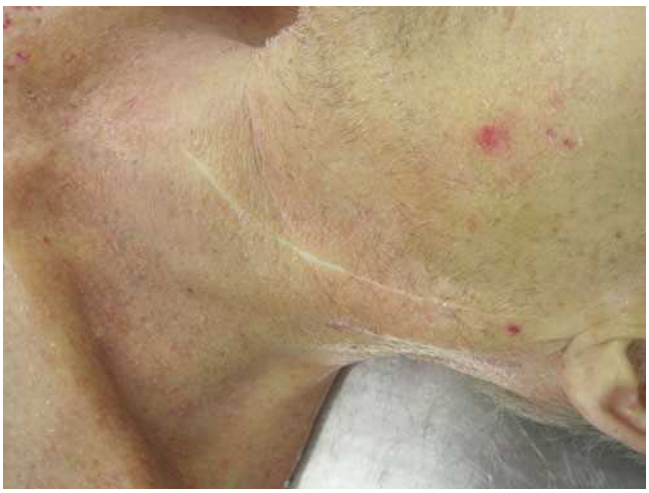

**Fig. 2.24** Surgical scarring in the anterior triangle of the neck, due to carotid atheromatous disease surgery

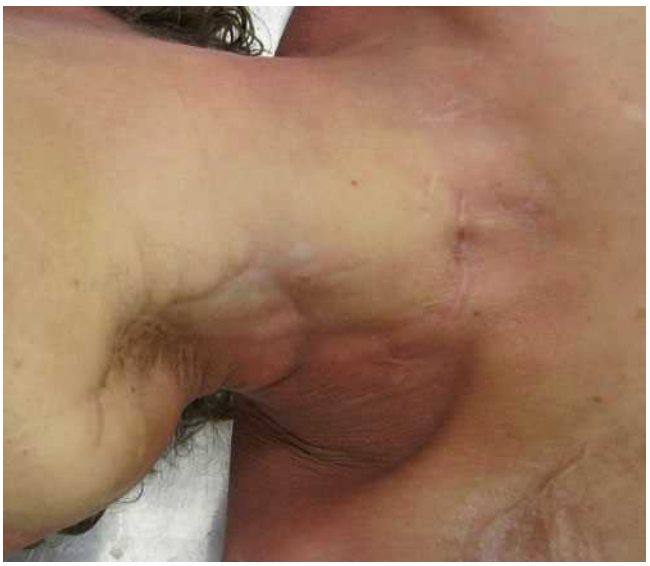

**Fig. 2.23** The large area of surgical scarring with underlying loss of tissue is due to a radical neck dissection for head and neck cancer. Note the healed tracheostomy scar in the midline

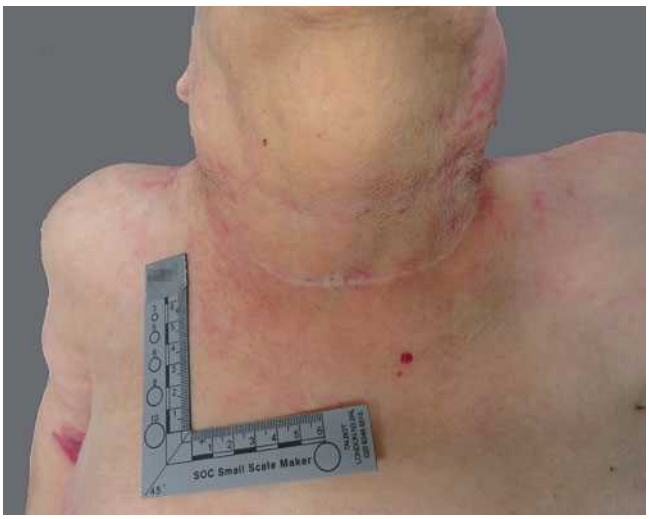


**Fig. 2.25** A transverse scar at the base of the neck is often due to thyroid surgery

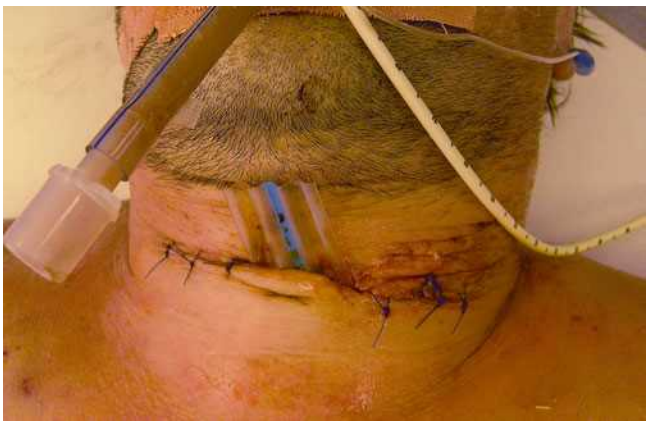

**Fig. 2.26** Self-inflicted neck wounds. Several initial incised wounds and a deeper cut are seen here on the neck, alongside evidence of surgical intervention. A corrugated drain is present, as well as sutures. An endotracheal and nasogastric tube are also present

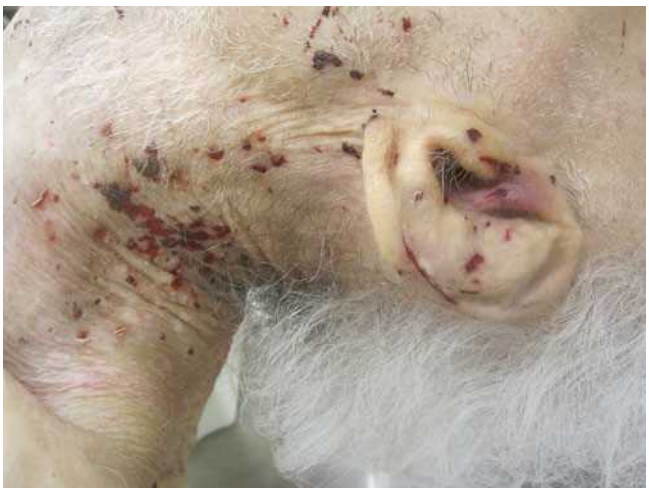

**Fig. 2.27** Scratch marks may reflect a confused state of delirium or dementia, pruritus, or skin infestation

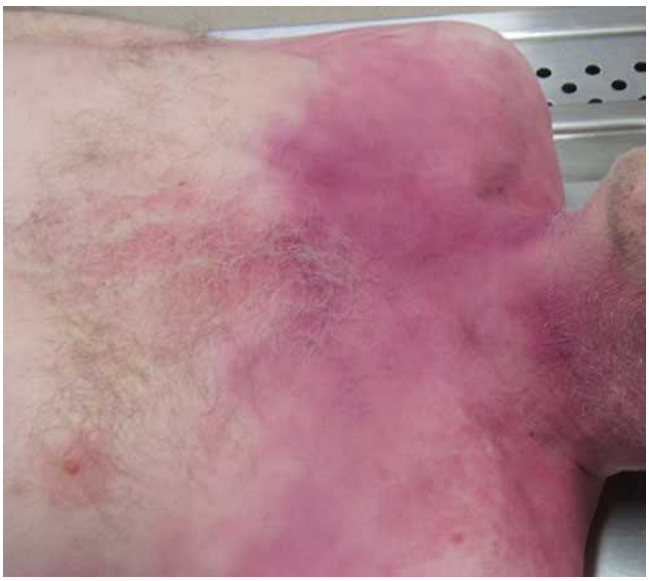

**Fig. 2.28** Carbon monoxide poisoning without burn injury. The cherry red discolouration of carbon monoxide poisoning is most pronounced in the neck and upper chest of this person, indicating that he was alive during the time when the fire was active nearby

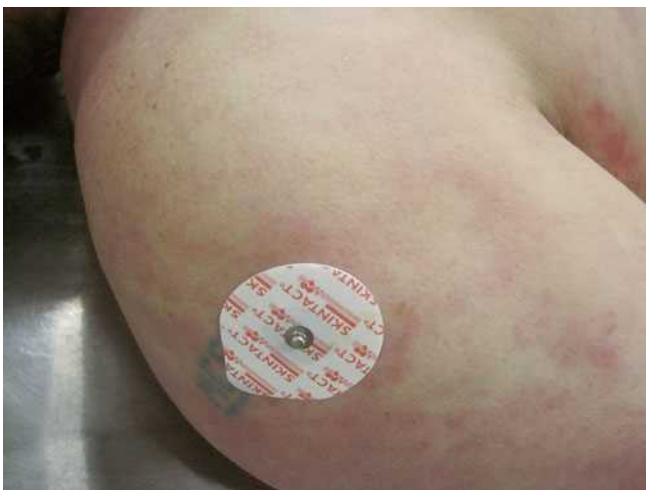


**Fig. 2.29** ECG stickers are often placed on all four limbs and around the chest. They should be removed and discarded at the start of the autopsy. This sticker is overlying a tattoo

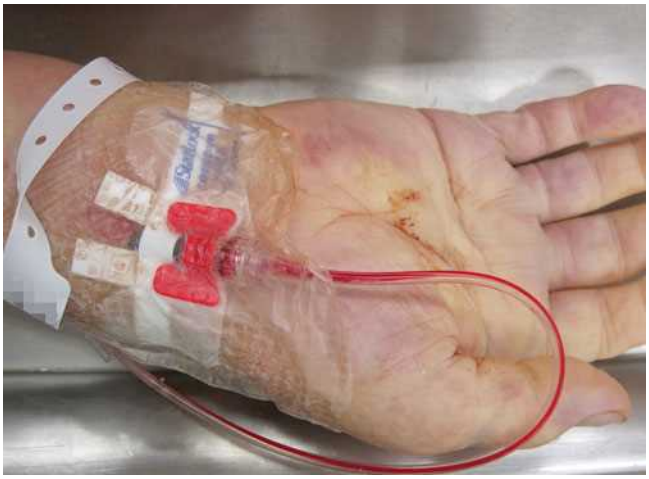

**Fig. 2.30** Arterial lines can be placed in any artery. The catheter tube has a red stripe along the surface to indicate to clinical staff that it is an arterial line. Superficial lines are removed and discarded prior to evisceration, if the pathologist is satisfied that the placement was correct

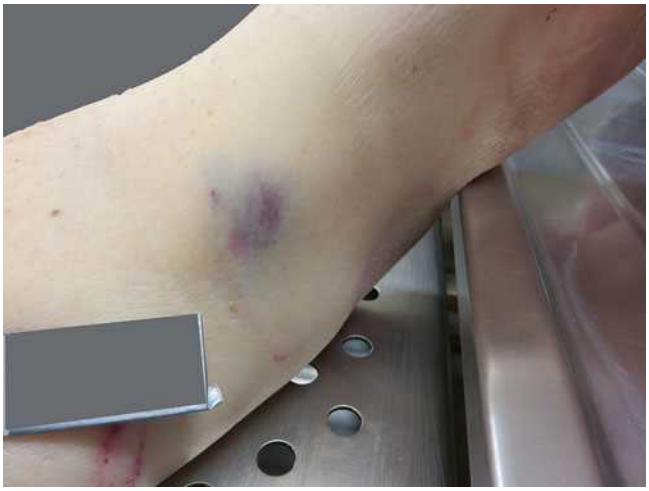

**Fig. 2.32** Venepuncture marks are often associated with localised bruising

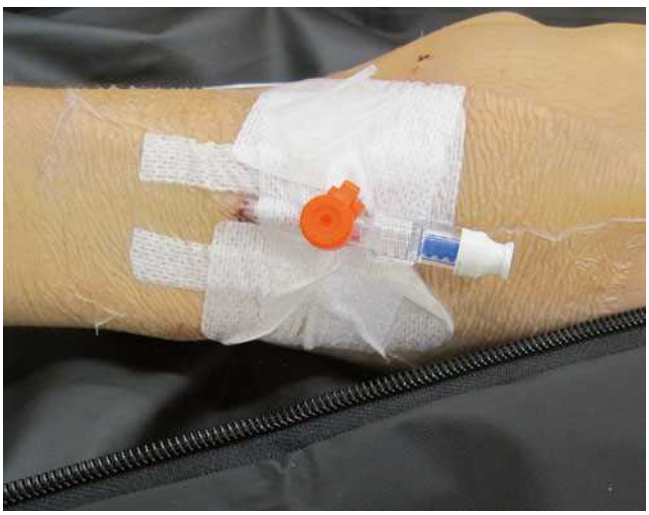

**Fig. 2.31** The intravenous cannula is often found in the upper limb. If the patient has spent some time in hospital, there may be previous sites showing bruising and/or swelling

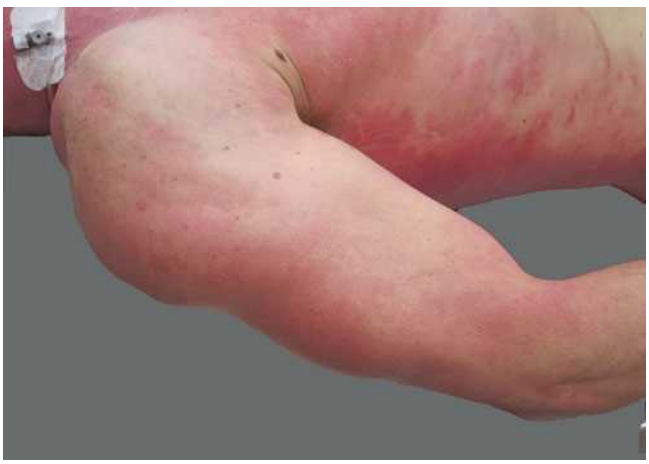

**Fig. 2.33** The limbs are good for assessing physical build. Increased muscle bulk may be due to general athleticism, or potentially to anabolic steroid use. A venous line is noted in the neck, used during the attempted resuscitation

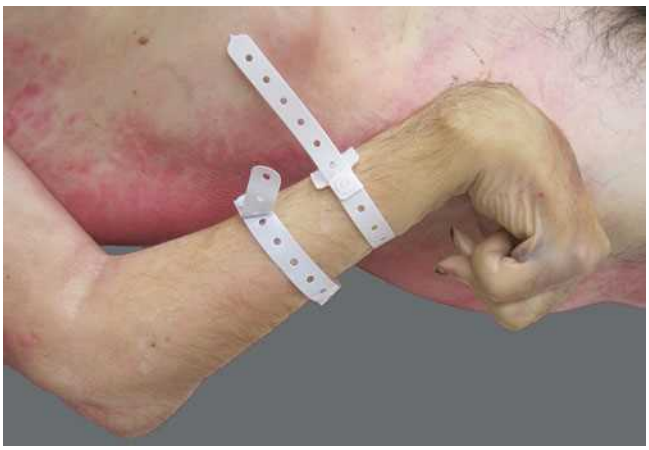

**Fig. 2.34** Neurological disease may be assessed externally, with this abnormal limb posture often seen following a cerebrovascular event, resulting in an upper motor neuron lesion

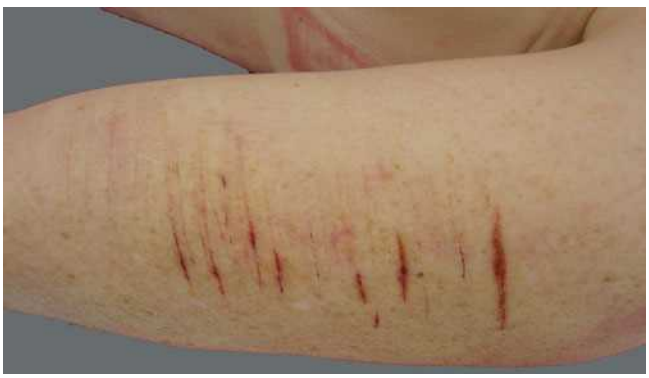

**Fig. 2.35** Multiple, superficial, parallel incisions in different states of healing are indicative of self-harming. They can be found anywhere, but the upper limbs, thighs, abdomen, and breasts are more common. Other types of injury also may be self-inflicted, such as burns or insertion of foreign objects such as broken glass

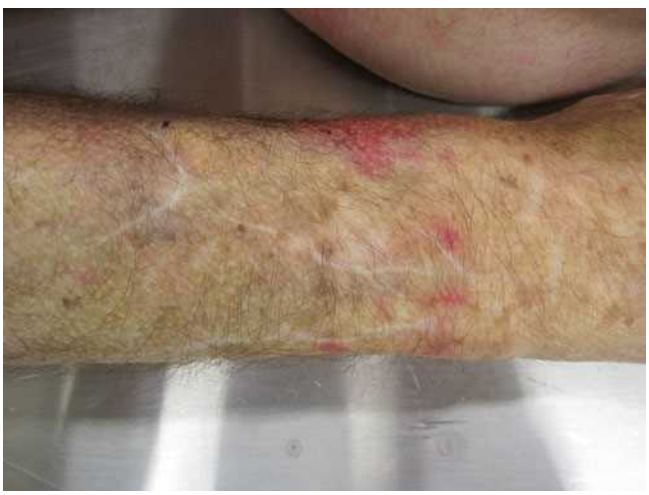

**Fig. 2.36** Old injuries, with irregularly distributed scars of previous traumatic injury

**Fig. 2.37** Assumptions regarding the nature of jewellery should be avoided; it is best to describe a yellow metal ring rather than a gold ring

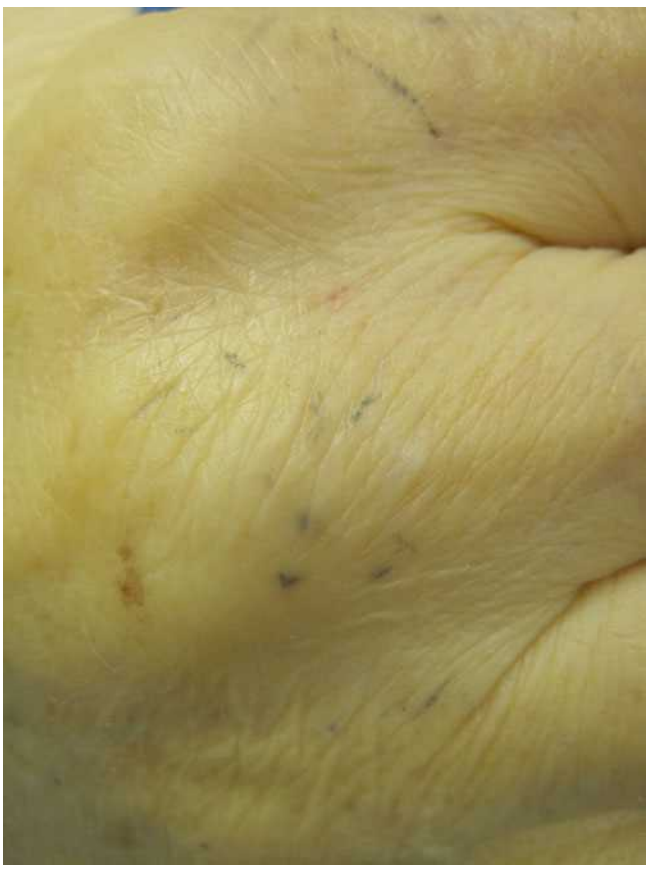

**Fig. 2.38** Coal tattoo. Irregular pigmentation may be occupation-related, such as this example of coal tattooing on the hands of a retired coal miner. This finding should make you consider the possibility of occupational lung disease

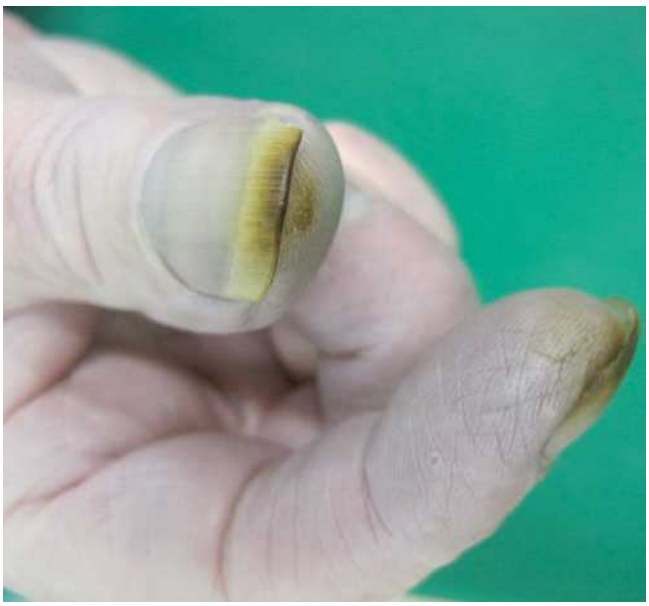

**Fig. 2.39** Cigarette smoking. Not all cigarette smokers get tar-stained fingers, but their presence usually indicates heavy smoking

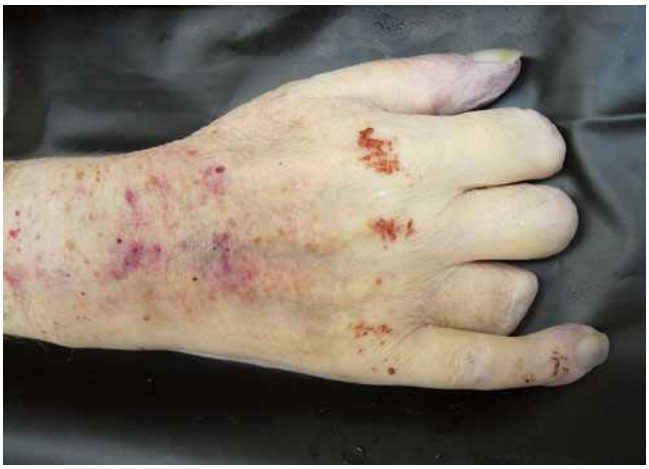

**Fig. 2.40** The hands are common sites for injury, which may be incidental or due to the circumstances surrounding death. This degree of damage reflected a workplace accident many years previously

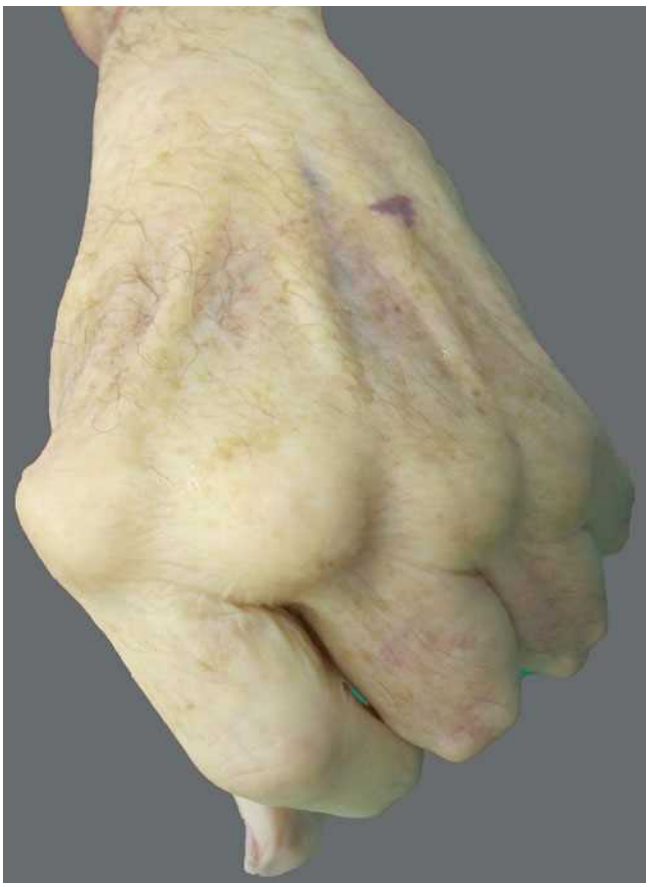

**Fig. 2.41** Rheumatoid arthritis causes bone and joint destruction, which in turn leads to specific joint deformity. There is also wasting of the small muscles of the hand

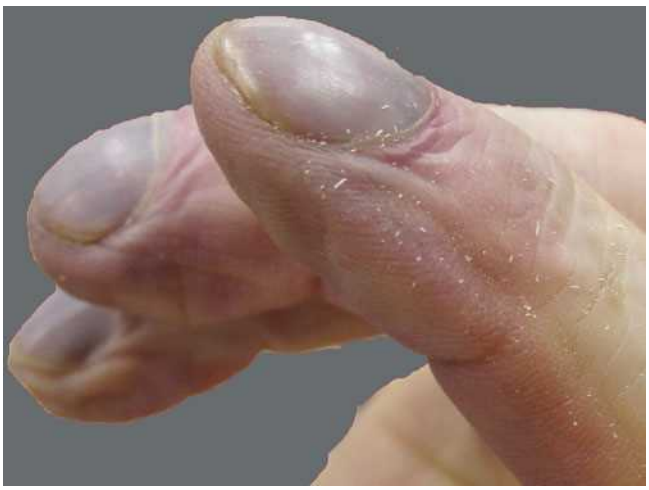

**Fig. 2.42** Digital clubbing may be idiopathic or associated with a variety of clinical conditions including bronchiectasis, lung cancer, liver cirrhosis, congenital heart disease, inflammatory bowel disease, and others

**Fig. 2.43** The nails often have a dusky appearance after death, which should not be over-interpreted as peripheral cyanosis

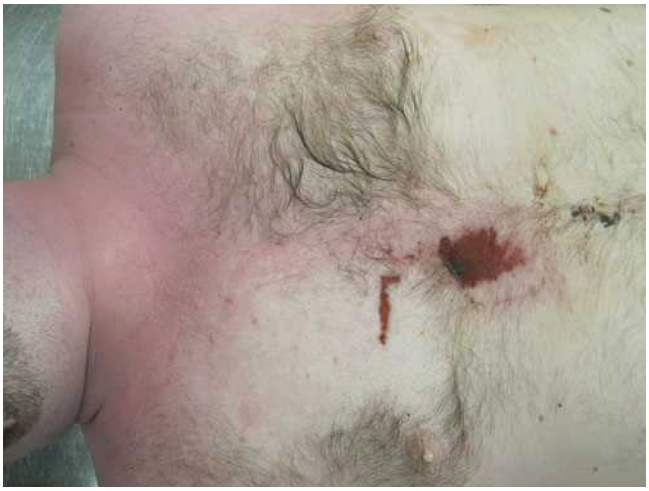

**Fig. 2.45** Marks left by cardiopulmonary resuscitation (CPR) are not unusual. Chest hair is shaved to improve electrode conduction. The defibrillator pads are extremely sticky and can cause superficial skin loss when removed. By the time of autopsy, these areas of skin loss will have the appearance of an abrasion with parchment texture

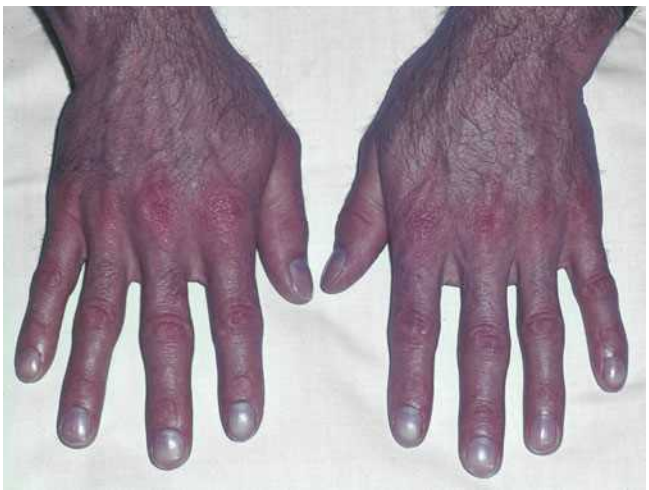

**Fig. 2.44** This ante-mortem image depicts argyria. This unusual blue/grey discolouration of the skin is due to exposure to silver-containing preparations

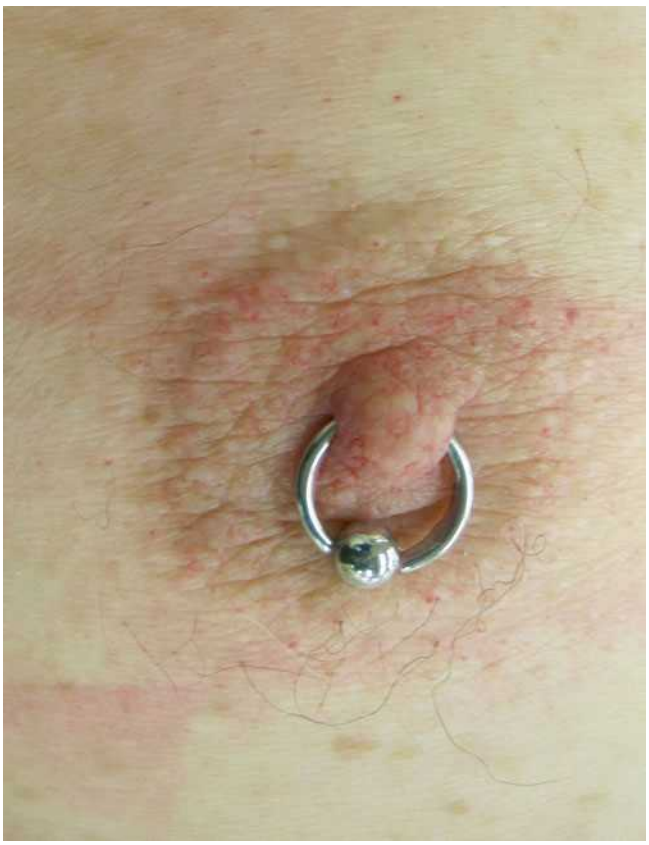

**Fig. 2.46** Various rings, studs, and implantable jewellery may be noted as part of the external examination. These should be documented and left in situ (unless otherwise requested by the relatives). They can be useful for identification purposes

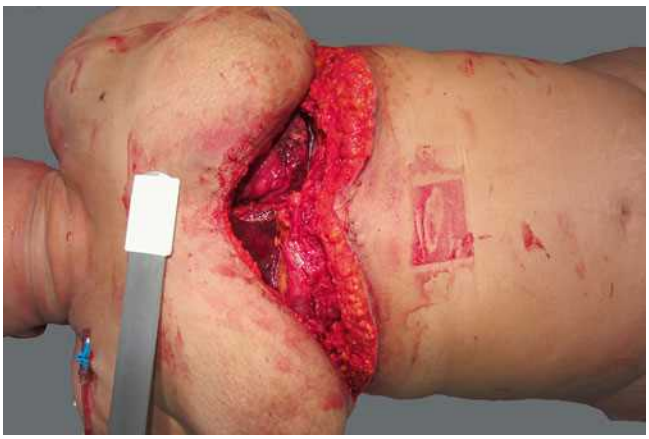

**Fig. 2.47** Emergency thoracotomy. Normally done only in an emergency trauma setting, usually for penetrating (and rarely blunt force) chest trauma, an emergency thoracotomy gives access to the chest to enable control of reversible causes of cardiac arrest (haemorrhage, cardiac tamponade). The sutures should be removed and the incision examined. (This is a 'clam-shell' incision.) Subsequent examination of internal organs will need to differentiate iatrogenic versus traumatic wounds

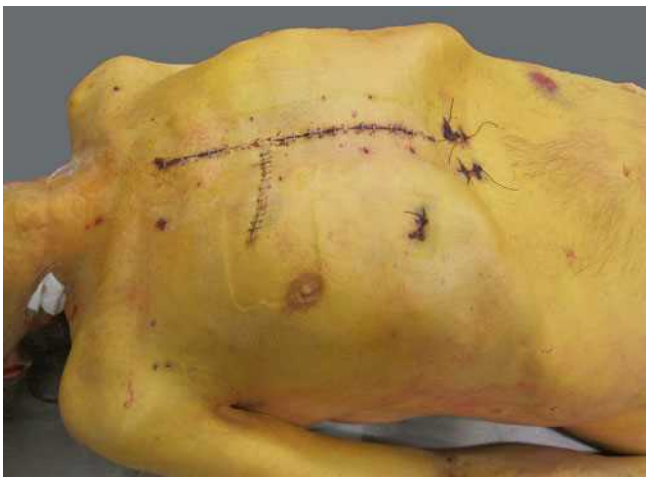

**Fig. 2.48** Elective thoracotomy. These are fresh surgical incisions of cardiothoracic surgery. The lateral horizontal incision is used for a minimally invasive approach, which appears in this case to have been converted into a central sternotomy approach. The sutured incisions are port or catheter sites. Note also the severe jaundice of the skin

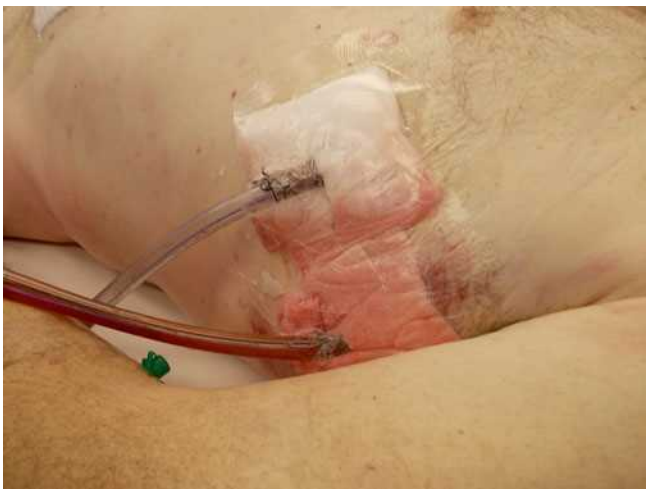

**Fig. 2.49** Chest drain. Large intrathoracic drains (with protective dressings) should remain in situ until the internal placement has been confirmed. The volume and nature of fluid in any drain bag should be documented. The dressings are removed to examine the skin surrounding the incisions. Surgical drains are often sutured in place, and care must be taken not to tear the skin during removal

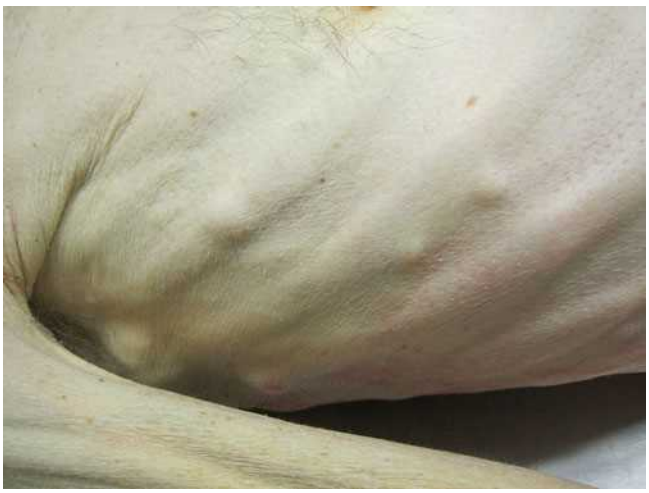

**Fig. 2.50** Many malignancies metastasise to the skin and subcutaneous tissues. These are deposits of malignant mesothelioma

**Fig. 2.51** The general state of nutrition can be assessed by looking at the abdomen. This patient is emaciated (cachectic). Note the green skin discolouration, which is an early sign of decomposition

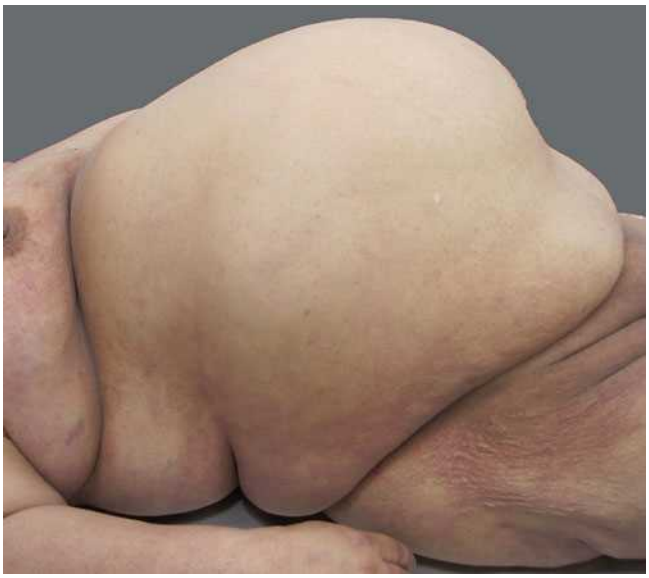

**Fig. 2.52** Abdominal distention may be due to fat, fluid, faeces, fetus, or gas. In this case it was clearly shown to be fat

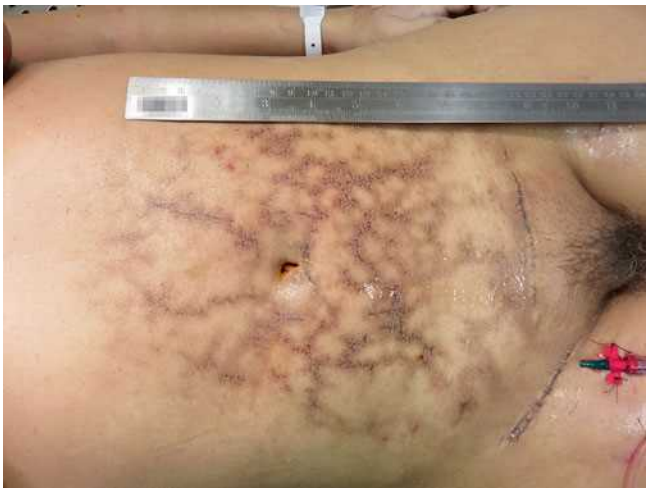

**Fig. 2.54** Erythema ab igne. Prolonged exposure of the skin to heat (hot water bottle, electric fire, laptop, etc.) can lead to the development of reticulated erythema, hyperpigmentation, scaling, and telangiectasia in the affected area. This patient had chronic abdominal pain due to endometriosis, relieved using a hot water bottle. Note the recently healed suprapubic Pfannenstiel scar and the fresh right groin incision closed by staples

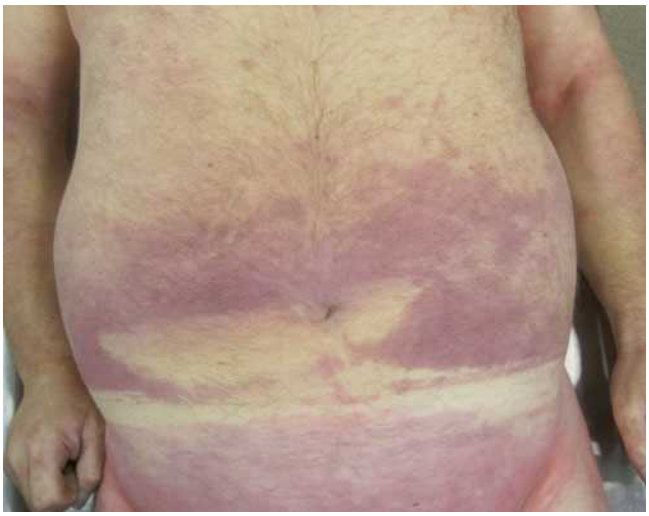

**Fig. 2.53** In this case of hanging, the post-mortem lividity/blood collection is inferiorly distributed over the abdomen and legs. Note also the negative imprint from some clothing

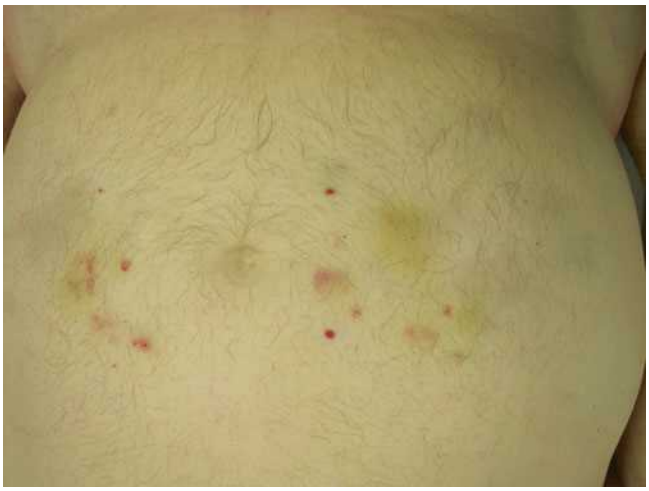

**Fig. 2.55** Subcutaneous injection sites. Low molecular weight heparin (LMWH) is delivered as a subcutaneous injection into the abdominal tissues. Hospital patients are at increased risk of deep vein thrombosis and receive LMWH as a risk-reducing measure, albeit with some minor bruising at the sites of drug delivery

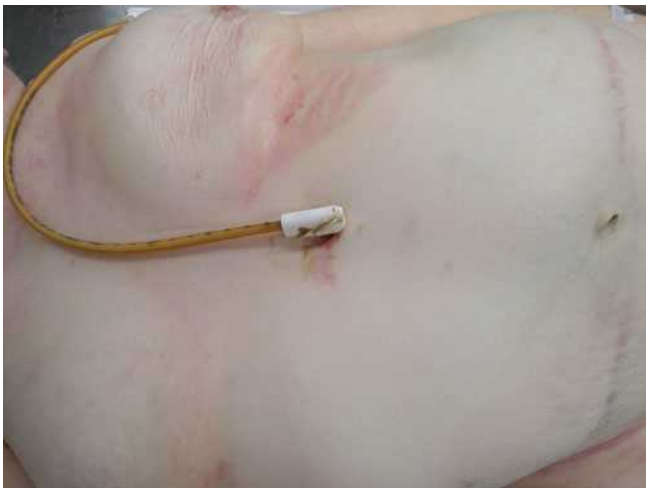

**Fig. 2.56** PEG device. Percutaneous endoscopic gastrostomy (PEG) devices enable enteral nutrition. There is a balloon cuff on the internal side of the device, which prevents the tube from falling out

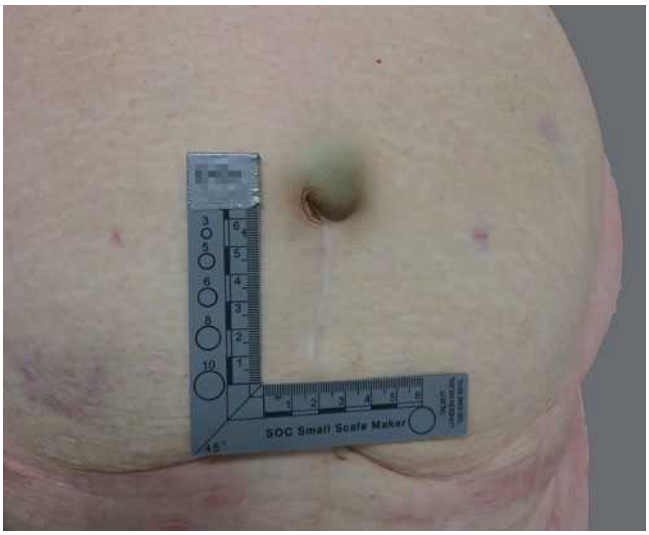


**Fig. 2.58** Previous surgery with incisional hernia. There is an area of discolouration and distention at the superior end of this old laparotomy scar, suggesting incarcerated hernia

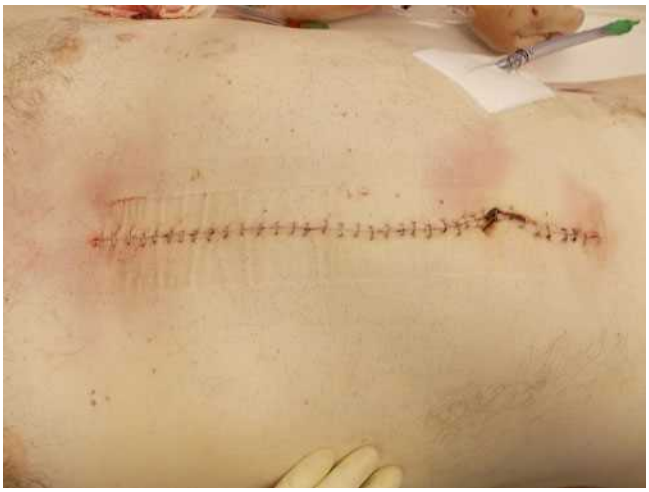

**Fig. 2.57** Dressings over wounds should be removed and the condition of the skin (healthy/inflamed/healing/etc.) should be considered. The length of the scar should be documented. The type of closure (here stapled) merits comment

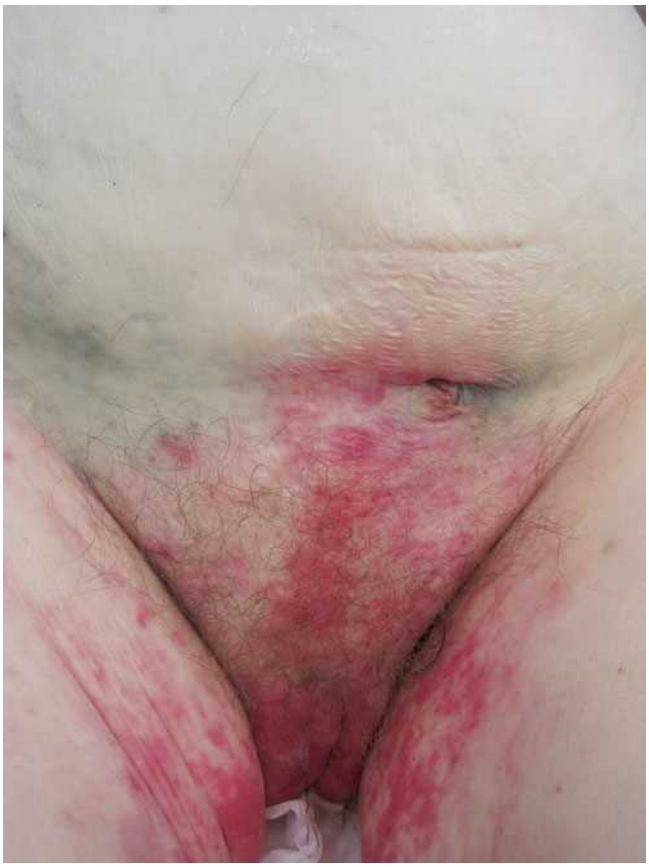

**Fig. 2.59** There is extensive reddening of the skin at the site of this suprapubic catheter. Urine spillage and local bacterial sepsis causes skin irritation, similar to 'nappy rash' seen in infants

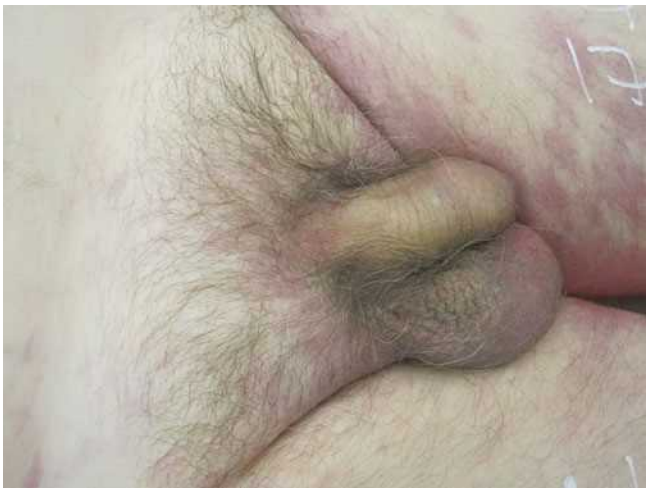

**Fig. 2.60** Male genitalia. The penis is lifted to examine the scrotum and testes. Scrotal oedema is seen in heart failure and fluid overload. One should also check for genital tattoos or piercings

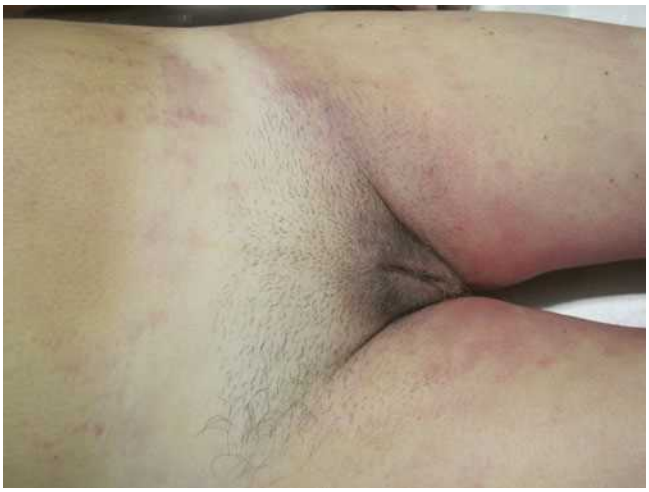

**Fig. 2.62** Examine the vulva for gross pathology, piercings, and tattoos. In this case, the pubic hair has been shaved. Some pathologists comment on the distribution and nature of body hair of both sexes

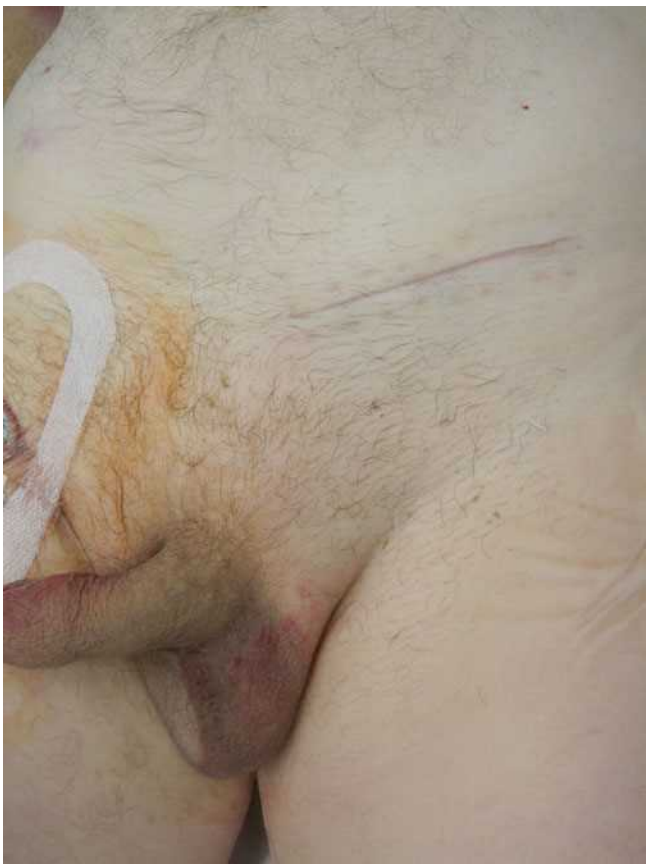

**Fig. 2.61** An inguinal scar provides a clue to previous orchiectomy. In this case, the loss of volume in the scrotum is visible, but palpation of the scrotum will confirm whether the testes are present and normal. One should remember that prosthetic testicles can be implanted. One should also be looking for hydrocele and tumours

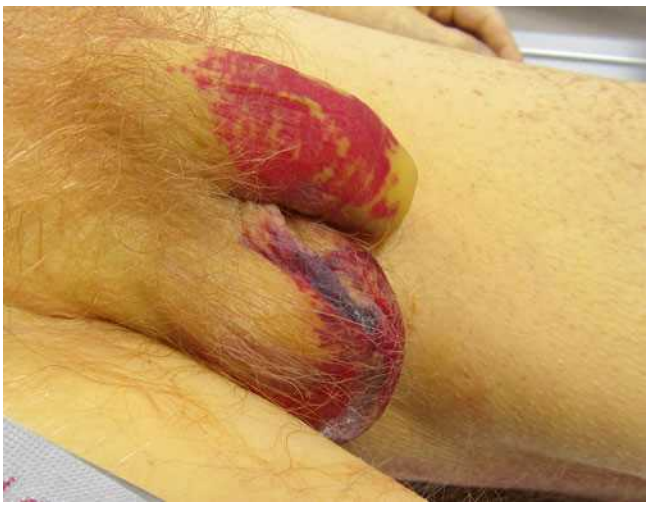

**Fig. 2.63** Genital ecchymosis. Consideration must be given to genital bruising. Generalised and diffuse bruising may be due to trauma, intraperitoneal haemorrhage, or surgery. In this case, the bruising is irregular yet localised, reflecting thrombocytopenia due to a haematological malignancy. Note also the severe jaundice in the skin, which makes the red colour stand out

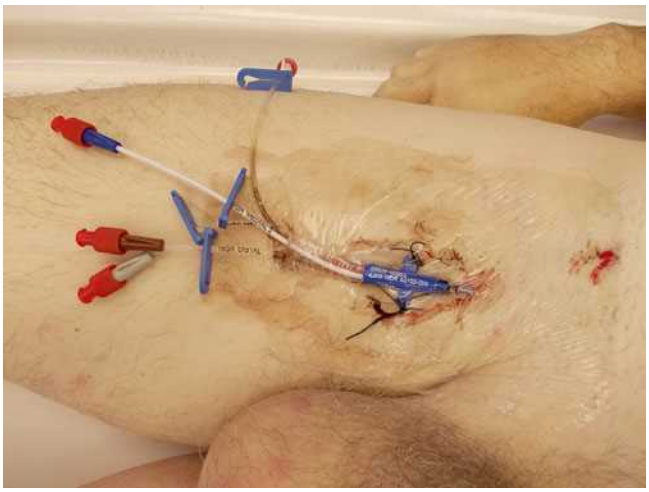

**Fig. 2.64** This is a groin-sited intravenous central line used for fluid delivery. It should be remembered that needle puncture marks may be from emergency care (as in this case) or from intravenous drug use. One should also check the groin for hernias, but unless very large, these are unlikely to be palpable in a prone dead body. One should always palpate for enlarged inguinal lymph nodes

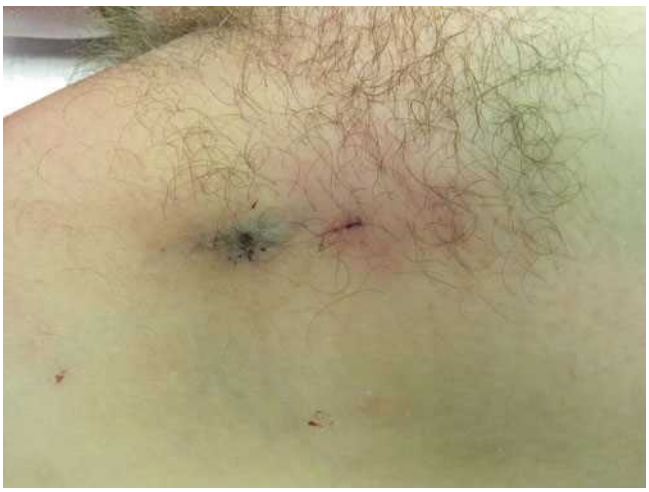

**Fig. 2.65** The groin in an intravenous drug user often shows evidence of repeated injections in the same site, with inflammation and bruising. Late changes will involve sinus formation

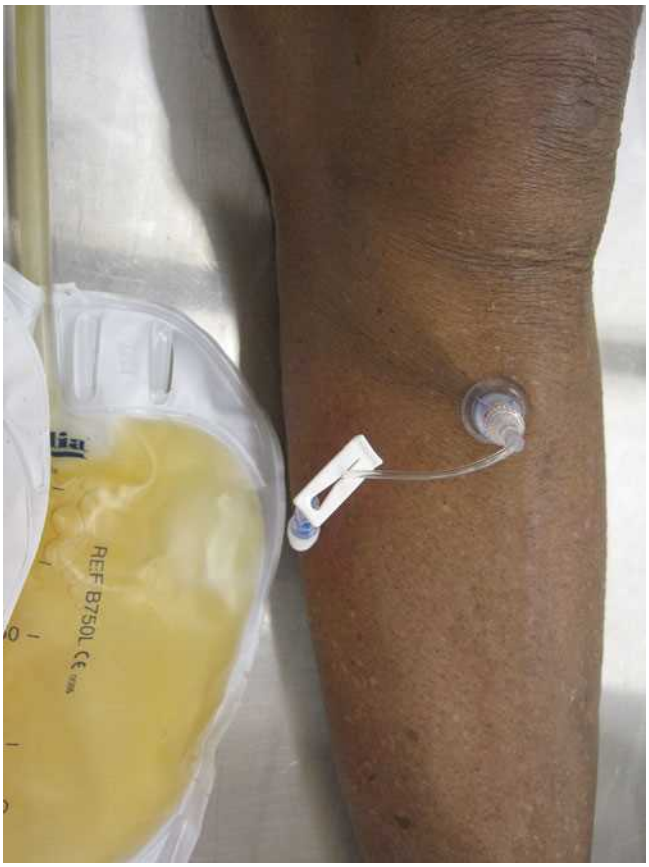


**Fig. 2.66** The intraosseous infusion route is commonly used in paediatric patients but also can be seen in acute/trauma adult settings [18]. A urine collection bag is seen adjacent

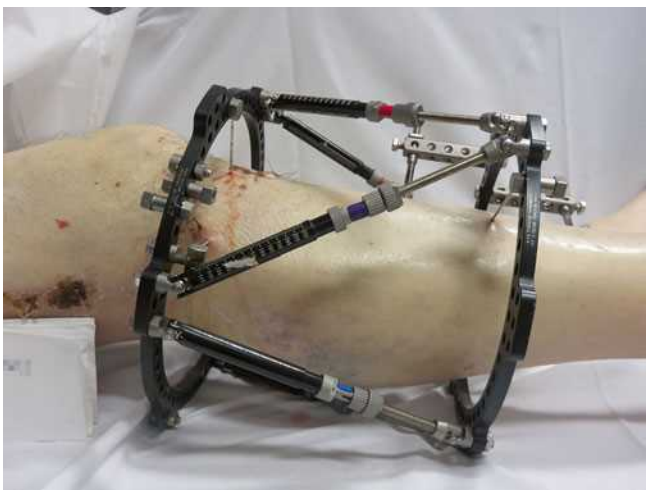

**Fig. 2.67** External fixation devices need to be removed by the appropriate technical department in the hospital before the body is released for burial or cremation

**Fig. 2.68** Tourniquet. This limb tourniquet was applied at the scene of a road traffic accident following a traumatic below-knee amputation

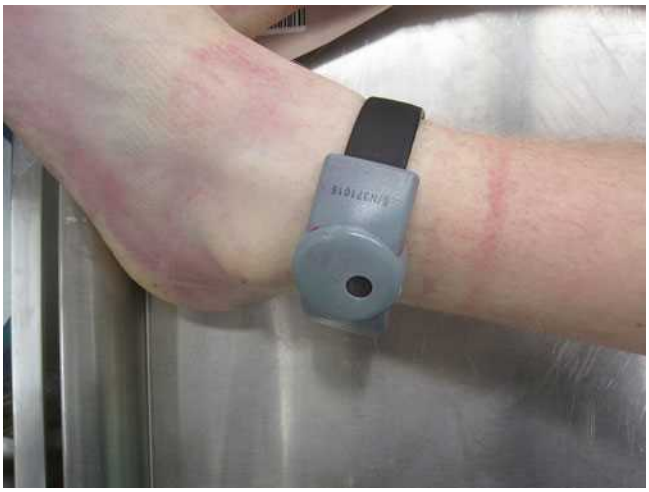

**Fig. 2.69** Monitoring (offender) tag. This is an electronic tag used to provide information on the geographic position of convicted persons present in the community and subject to control orders

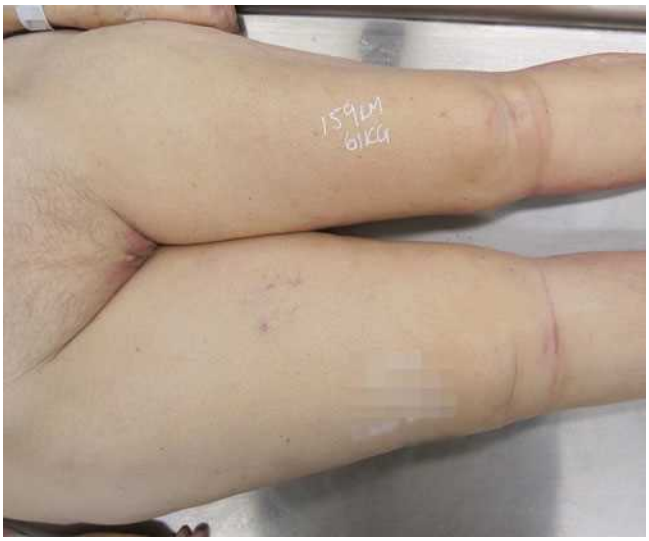


**Fig. 2.70** Leg symmetry. The right side appears bigger than the left. If legs are asymmetrical, one should ideally measure mid-thigh or calf circumference (10 cm below tibial tuberosity). A difference of 3 cm or more raises the possibility of deep vein thrombosis and potentially pulmonary thromboembolism

**Fig. 2.71** Limb deformity may be due to congenital or acquired musculoskeletal or neurological diseases. This is an open compound fracture

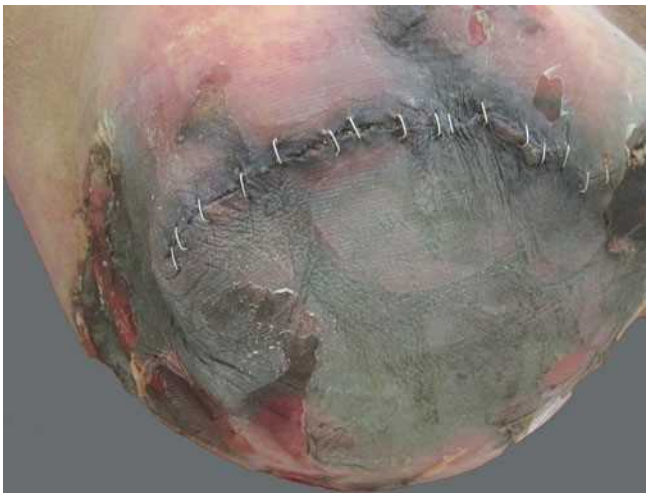

**Fig. 2.72** It is advisable to note the level of any amputation and comment on the condition of the stump. This is a fresh surgical wound (staples are still in place) showing signs of local tissue necrosis, suggesting sepsis (possibly necrotising fasciitis) and/or critical vascularity

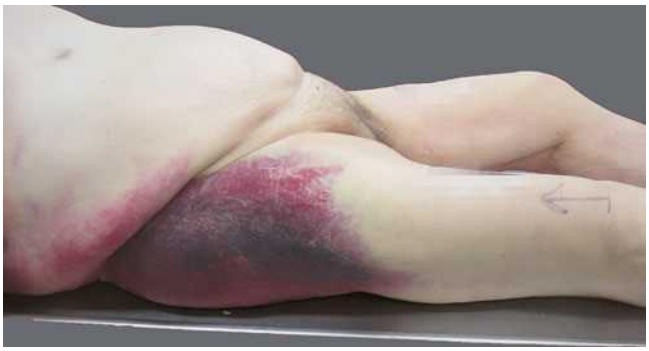

**Fig. 2.74** In the post-mortem room, as in life, a shortened and externally rotated lower limb provides a clue to a fractured neck of femur. In this case, there is massive haemorrhage around the hip, which has leaked into the subcutaneous tissues. Internal haemorrhages can progress after death as the blood pools in the dependent areas. Note the arrow drawn on the right leg by the surgical team to identify the correct site for planned surgery. Care must be taken when examining fracture sites internally, as there may be sharp bony edges

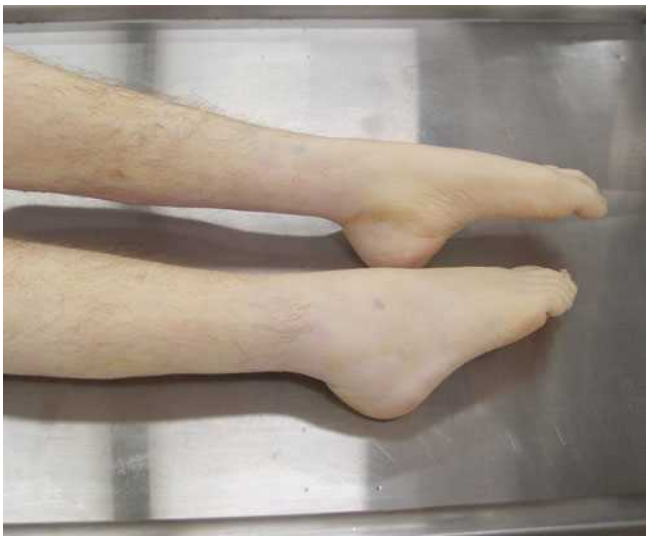

**Fig. 2.73** A spinal cord injury has led to bilateral (spastic-style) extension of the feet and muscle wasting

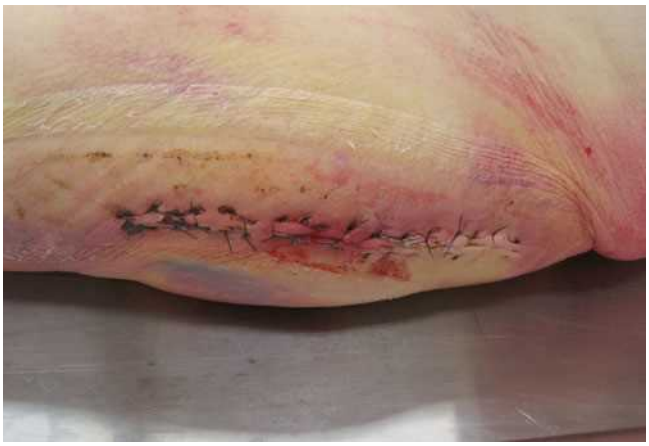

**Fig. 2.75** This surgical wound shows features of erythema and oedema. Releasing the sutures revealed purulent fluid, which should be collected in a sterile container and sent to microbiology. A wound swab is acceptable, but a volume of pus is preferred

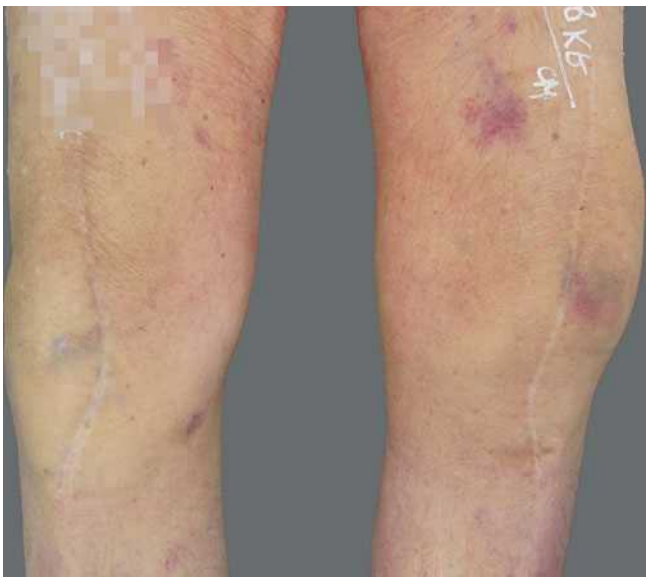

**Fig. 2.76** Joint replacement surgery of the hip and knee is often bilateral. Here there are also chronic leg deformities from osteoarthritis

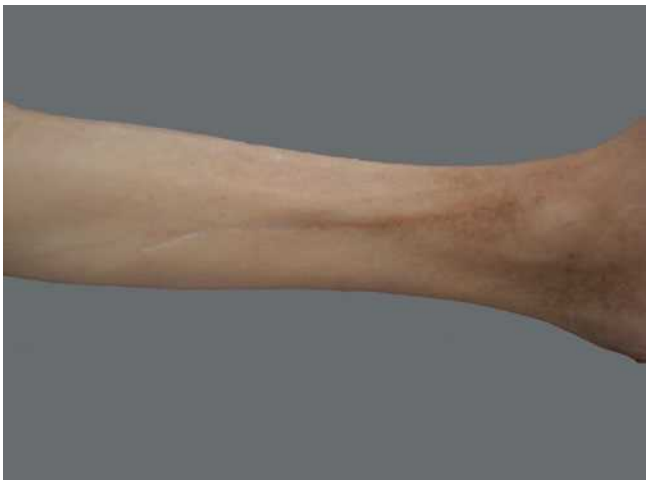

**Fig. 2.77** Finding a sternotomy scar should prompt you to look at the legs to identify vein graft harvest sites for coronary bypass surgery

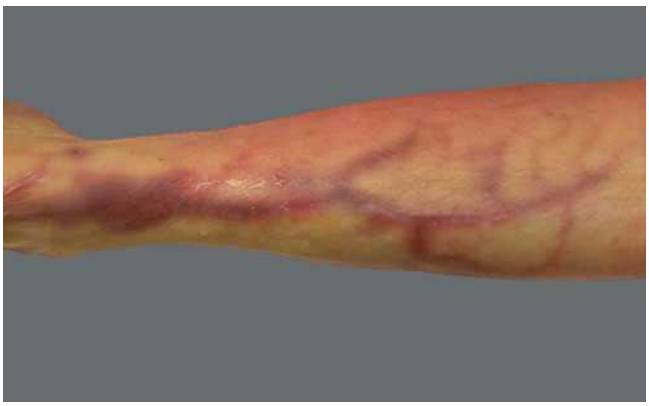

**Fig. 2.78** This pronounced marbling is due to bacterial load in the circulatory system immediately before death, with subsequent intravascular erythrocyte haemolysis

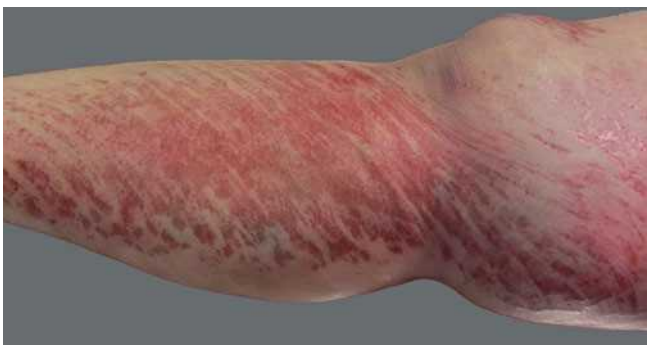

**Fig. 2.79** Superficial skin abrasions sustained in a road traffic collision. The direction of force can be established by looking at this injury. Skin abrasions may occur during the accident, but also might occur afterwards, when the body is moved

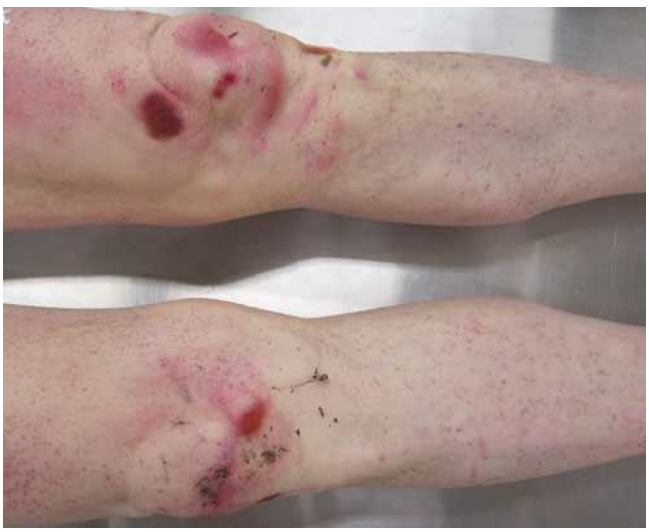

**Fig. 2.80** Multiple injuries over the knees at varying stages of healing may suggest chronic or repeated bouts of intoxication (*e.g.*, alcohol) and frequent falls

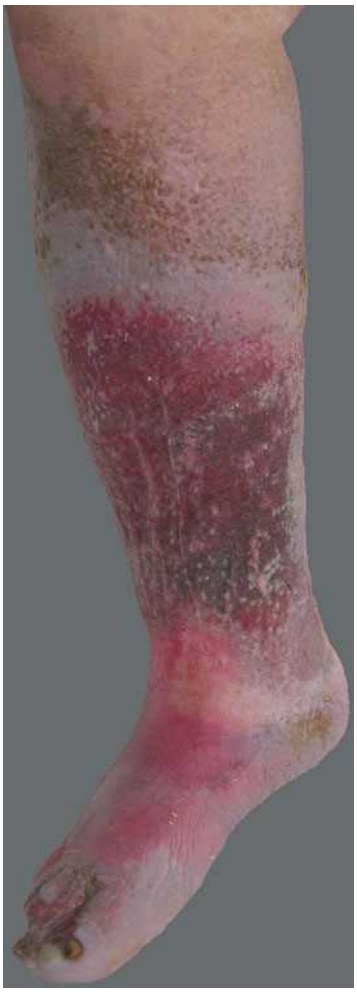

**Fig. 2.81** Chronic venous obstruction and lymphoedema. This may reflect obstruction to the venous and/or lymphatic outflow from the leg. It may be associated with ulceration

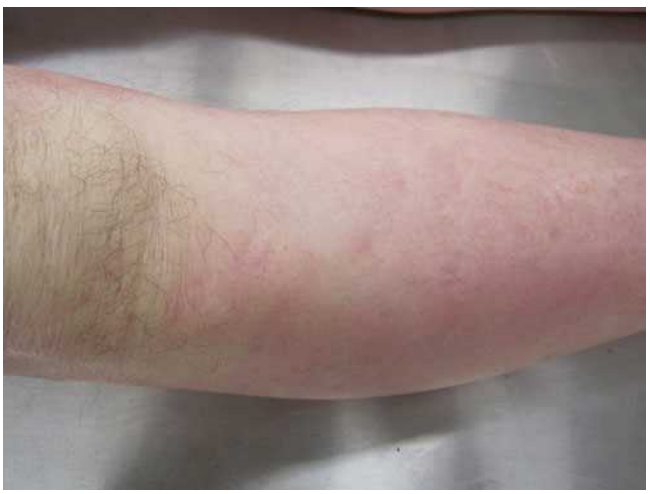

**Fig. 2.82** Peripheral vascular disease can lead to natural hair loss in the lower leg, often with diminished soft tissue and muscle bulk

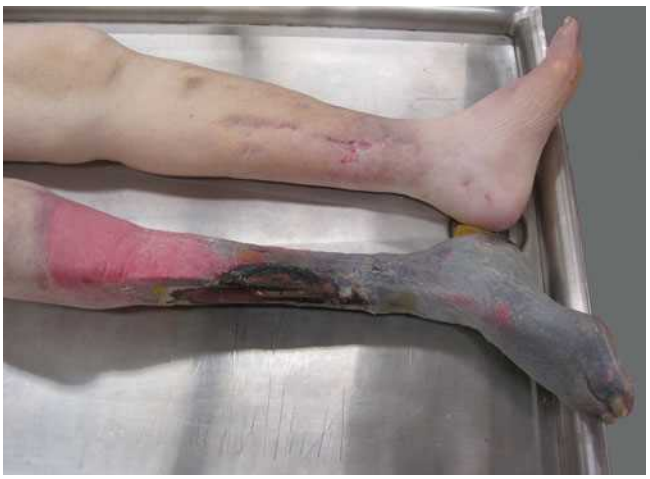

**Fig. 2.83** Severe peripheral vascular disease in a patient with poorly controlled diabetes. Note that the left leg is hairless and ulceration is present. The right leg shows extensive gangrene with ulceration through to the tibia. The back of the right heel is discoloured at the site of a pressure ulcer

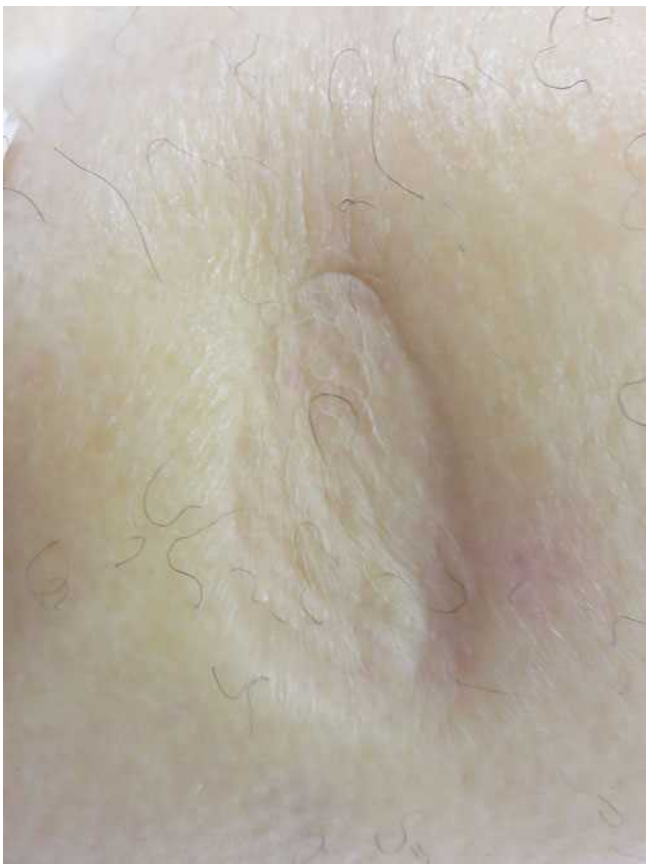

**Fig. 2.84** Pitting pedal oedema. Apply firm digital pressure (for at least 10 seconds) over a distal bony prominence to assess for pitting oedema

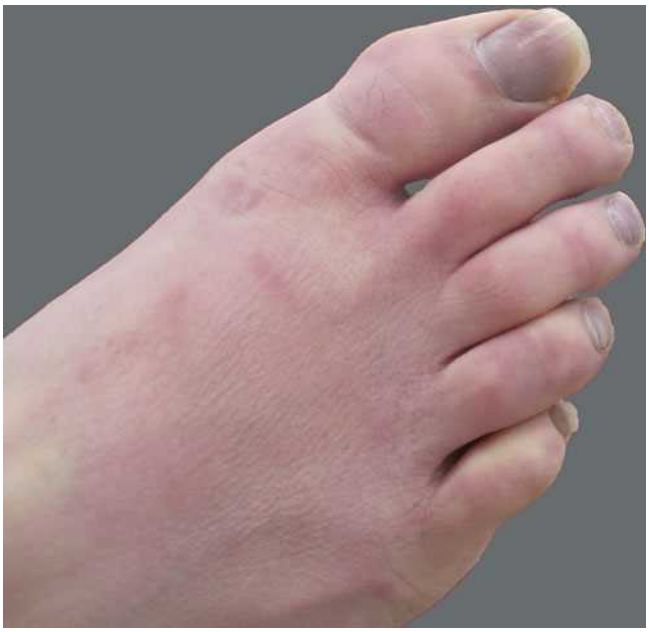

**Fig. 2.85** Although feet may appear unremarkable (as above), one should always look for missing toes, positional deformity (neurological, musculoskeletal), arthritis effects (rheumatoid or osteoarthritis, etc.), scabies, and needle puncture marks (*e.g.*, intravenous drug use). If there is a known history of diabetes, then one should check the feet carefully for signs of injury, ulceration, and/or infection

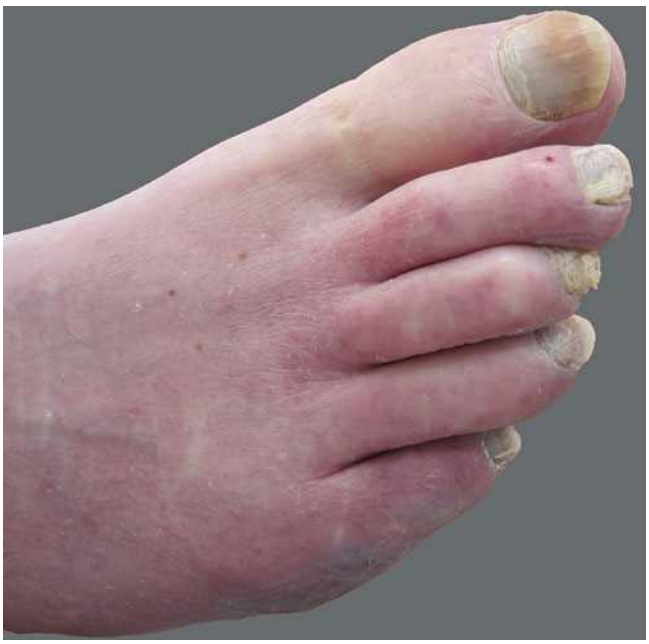

**Fig. 2.86** Fungal infection of the toenails is not uncommon in the adult population, but it can be associated with diabetes mellitus and with arterial and venous insufficiency

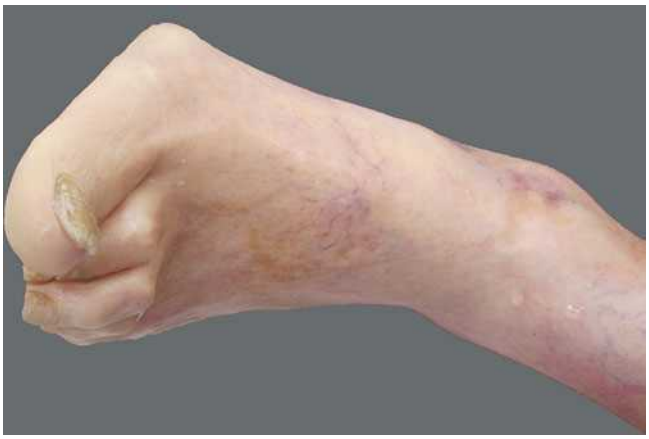

**Fig. 2.87** The small joints in the feet may show deformities of arthritis

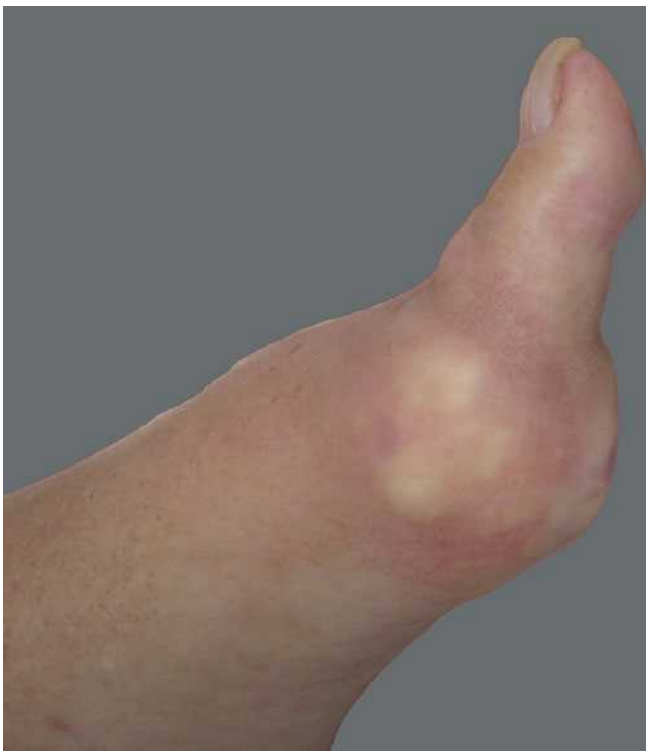

**Fig. 2.88** Gout. These subcutaneous nodules are gouty tophi in the classic disease site

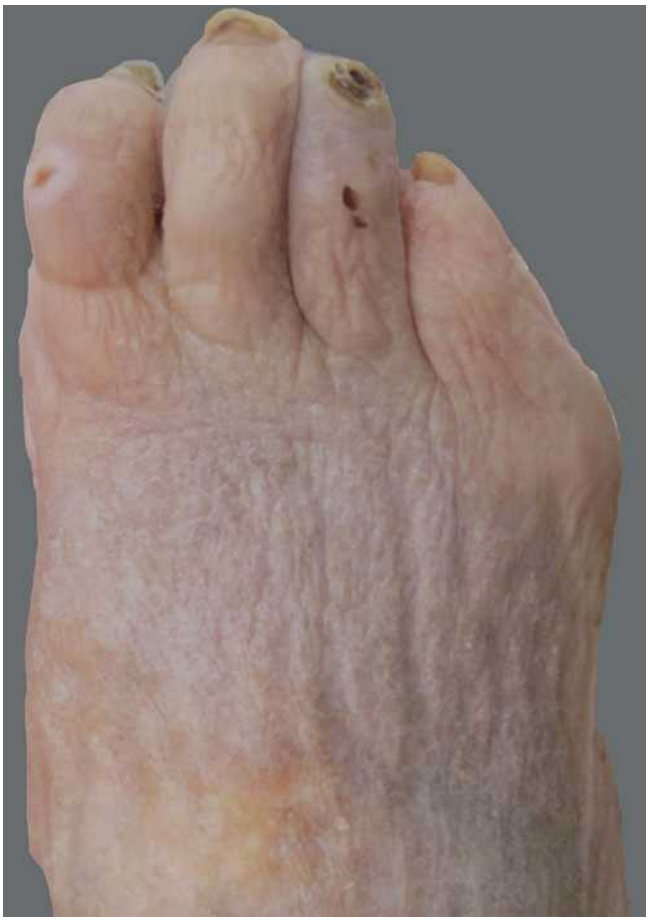

**Fig. 2.89** Diabetic foot. The big toe has been previously amputated, perhaps because of trauma or infection. There is toenail disease due to fungal infection. Ulceration is noted over the second toe, in keeping with diabetic neuropathy

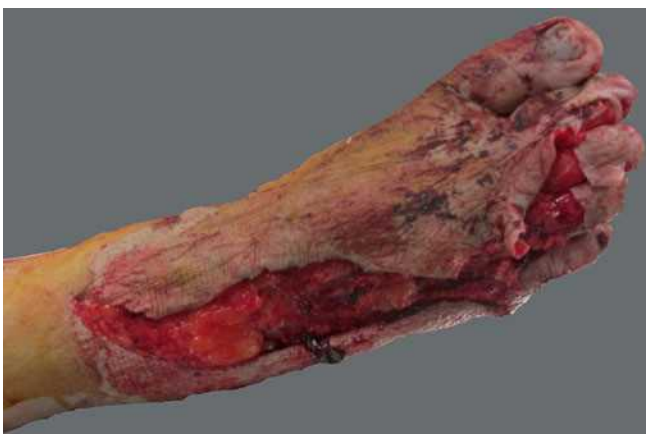

**Fig. 2.90** There is partial degloving of the skin overlying the foot and ankle, several of the toes were broken, and one was partially amputated

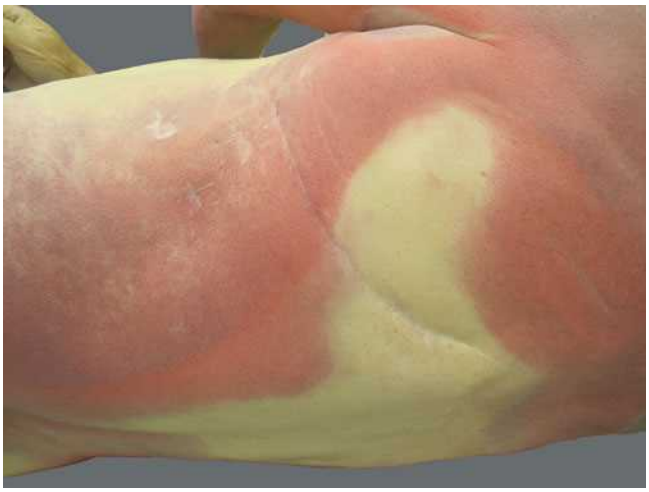

**Fig. 2.91** Thoracotomy scar. It is all too easy to look at the body only from the front or back. Old lateral-aspect thoracotomy scars can be overlooked. Note the separate smaller scars from previous chest drains

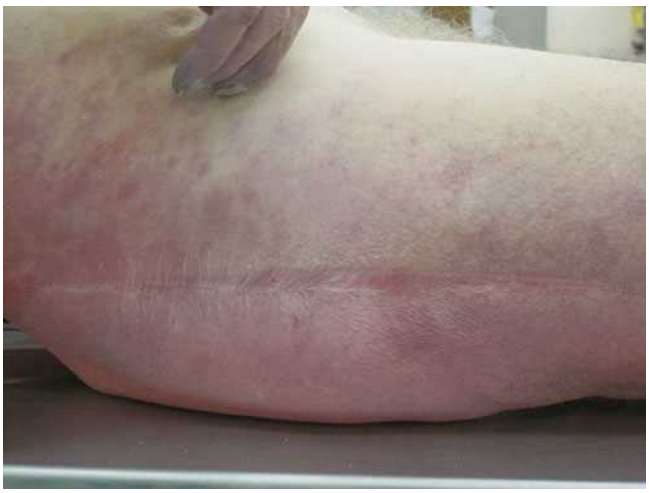

**Fig. 2.92** Hip replacement scar may be missed unless one looks at the sides of the body. Old hip replacement scars can be faded and difficult to appreciate

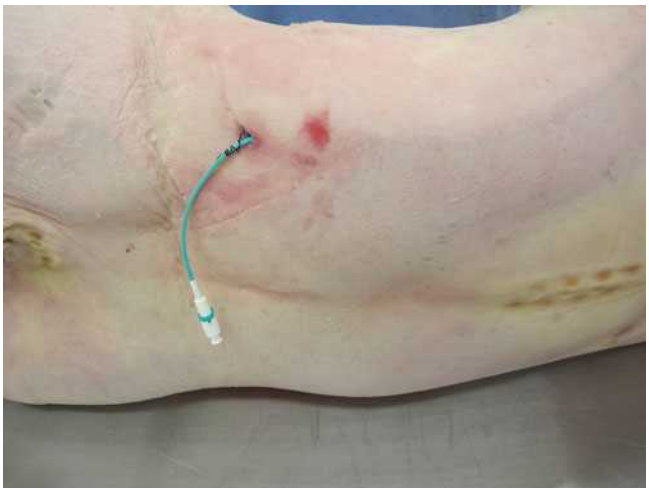

**Fig. 2.93** Direct drainage from the kidney pelvis via a nephrostomy catheter is seen. The tube is left in situ until the internal placement is confirmed. Note the pressure area skin damage over the spine and sacrum in this patient

**Fig. 2.95** Turning the body to the side. This body has been rotated onto the left side. It shows extensive burn injury affecting the hand (top right corner). This injury is seen in association with the cherry red discolouration of carbon monoxide poisoning (in the area where the skin surface has been lost). There is widespread soot deposition on the attached skin surfaces

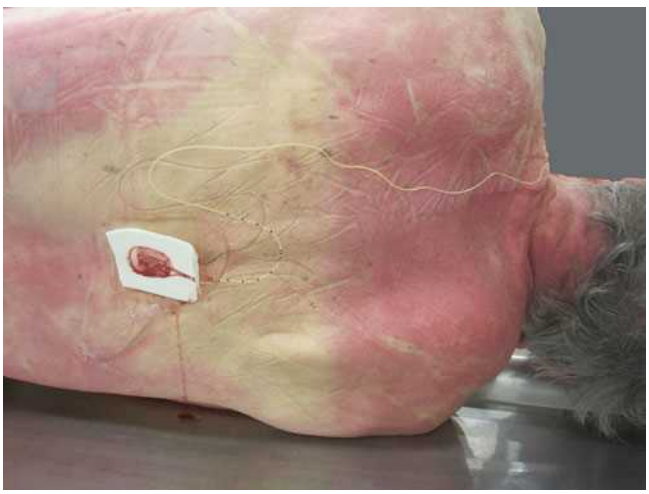

**Fig. 2.94** The dressing is removed to examine the insertion point of an epidural catheter

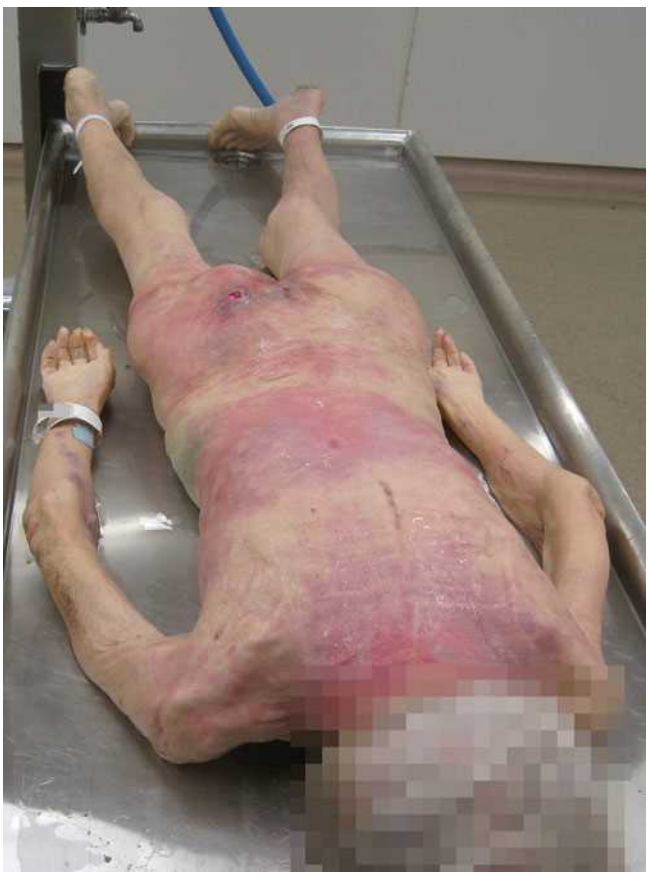

**Fig. 2.96** Prone examination. The body can be turned onto the front (prone) to get a good view of all the posterior tissues, especially if photography is needed. (Note the pressure areas over the buttock and sacrum, which were photographed.) However, as above, turning the body by 90° (right or left side) often suffices and is simpler

## Practical Realities of the External Examination

Any relevant external features are usually documented on a body diagram, using a combination of text, drawing, and shorthand symbols. The site and appearance of each feature is noted, along with relevant measurements. The diagram and descriptive information serve as memory aids for the final report and act as contemporaneous evidence of findings at the time of examination. Some may find it preferable to contemporaneously dictate the external examination using a voice recorder.

Photography is important, as it provides a permanent record for future reference. Photographing every detail with a scale rule is not necessary in a general autopsy case. All photographs should include the case reference number (either in the image or as coding digitally) and should be taken in accordance with good medical practise guidelines and local legal/consent realities. Taking photographs on personal smartphones is not advisable.

## The Examination in Stages

The external examination is akin to how one approaches external examination of a patient in a clinical setting; each organ system is given consideration. External stigmata of many diseases may remain after death, but some do not. Some features (*e.g.*, cyanosis) no longer hold relevance in the post-mortem setting. Palpation and articulation of the body is also necessary, akin to the clinical examination.

It does not matter in which order the examination is completed, but it is recommended that the approach is logical and is done in a standard fashion so that nothing is missed. The anterior, posterior, and lateral aspects are examined from head to toe.

Findings fall under broad headings: demographics (apparent age, ethnicity, sex, height, weight), descriptors (eye colour, hair colour, nutritional state, signs of neglect of personal hygiene), items attached to the body (clothing, jewellery, medical and surgical devices, ligatures, etc.), surgical and nonsurgical modifications (hair extensions, breast implants, etc.), body adornments (tattoos, piercings, etc.), signs of natural disease, signs of injury (new and old), signs of decomposition.

The documents written at the time of the external examination should be kept as part of the autopsy record according to local and national guidelines. In the UK, they are ideally kept for a period of between 8 and 30 years [3–5]. All medico-legal autopsy reports in the UK belong to the HM Coroner (or Procurator Fiscal), and permission is required to share them with any interested person, including family or medical staff.

## General Observations in All Reports

Identification of the body (see Chap. 1) is confirmed by using identification bands and case paperwork. A note is made of the height, weight, apparent age (some diseases cause premature aging), habitus (*i.e.*, general nutritional state), sex, and ethnicity.

The sex is recorded according to the external genitalia, with appropriate adjustment made in the final report to account for transgender status. Ethnicity is recorded either according to skin tone and bone structure (white, brown, black) or ethnicity (Caucasian, African, African-Caribbean, Indian subcontinent, Oriental, South American Indian, Polynesian, Inuit, etc.). If one is uncertain, describe the skin and hair, and be prepared to ask the family about the family tree [6].

In a mortuary setting, most of the deceased are already naked. If clothing is present, each item should be recorded prior to removal. Pockets should be examined using forceps, so as to avoid inadvertent sharps injury. Assumptions regarding the nature of jewellery should be avoided. For example, refer to a yellow metal ring with a clear stone, rather than a gold ring with a diamond.

The position of the patient may be relevant and the distribution of post-mortem hypostasis should be noted. Comment on mild, moderate, or pronounced rigor mortis. Fluids around the body are documented.

For repatriated bodies, there may have been an autopsy in the country of death and the external examination will reflect previous embalming and evisceration.

## Medical and Surgical Intervention Devices

The purpose of recording medical and surgical devices is to confirm the interventions, exclude iatrogenic injury, and seek information about disease treatment and management. (See also Chap. 11.)

Medical devices are documented by site and name. If the device has a specific registration code, this too is noted. Advances in medical technology may mean that the pathologist is faced with an unfamiliar device. Photography for future reference and review of the clinical/medical record should be considered.

Devices are commonly related to medical emergencies (ECG stickers), surgical treatment (drains), and organ monitoring (urinary catheters). However, there are also devices for chronic disease management, treatment, and drug delivery (Table 2.1). One is advised to record the drug name, dose, and location of drug delivery devices such as transdermal patches. Remember that some medical devices such as haemodialysis fistulas, Hickman lines, cardiac pacemakers, and intrathecal shunts may be visible below the skin or pal-

**Table 2.1** Examples of medical and surgical devices found during the external examination

| Type of device | Examples |
|---|---|
| Implantable devices | Pacemaker |
| | ICD (implantable cardiac defibrillator) |
| | Contraceptive |
| | Insulin pump |
| | Intrathecal shunt |
| | AV haemodialysis shunt |
| Drug/nutrition delivery devices | Transdermal patch |
| | Intravenous line |
| | Hickman line |
| | NG tube |
| | PEG tube |
| Organ monitoring/support devices | Arterial line |
| | Urinary catheter (indwelling/suprapubic) |
| | ET tube |
| | Tracheostomy |
| | Intracranial pressure inducer |
| Surgical devices | Chest drains |
| | Surgical drains |
| | Colostomy, urostomy, ileostomy bags |
| | Nephrostomy line |
| Emergency treatment | ECG stickers |
| | Defibrillator pads |
| | Limb tourniquets |

*AV* arteriovenous, *ET* endotracheal, *ICD* implantable cardioverter defibrillator, *NG* nasogastric, *PEG* percutaneous endoscopic gastrostomy

pable. If a cardiac pacemaker is known to be present, then it is important to clarify whether it is a defibrillator (ICD) device, as these devices must be deactivated prior to evisceration because there is a finite risk of electrical shock to the prosecutor [7]. Orthopedic external fixation devices may require specialist removal.

Bandages, dressings, and plaster casts must be removed to enable examination of any underlying wound. Interventional devices (*e.g.*, endotracheal tube, nasogastric tube, chest drain, peritoneal drain, surgical drain) remain in situ until the internal location is confirmed, but they may be cut flush with the skin to facilitate examination. Intravascular lines can also be cut flush to the skin if necessary, to aid assessment after evisceration. Superficial devices are removed and may be safely discarded if they are not required as evidence.

The volume and colour of fluid in drain bags should be recorded before it is discarded. The skin adjacent to devices or wounds is examined for signs of infection, haemorrhage, surgical emphysema, and damage. If there are features of infection, one may consider whether a microbiology swab is appropriate (although handling of the body post-mortem and skin commensals often provide a mixed flora result).

The common sites of venous access are the dorsum of the hand, anterior wrist, antecubital fossa, anterior neck, and groin, but any vessel may have been cannulated in patients with limited venous access. Interosseous needles are usually left in situ and are more common in patients from an emergency setting. One should always look for needle puncture marks (which might suggest illicit drugs), bruising, thrombophlebitis, and localized accumulation of fluid in subcutaneous tissues.

Medical devices may have been removed recently, and marks on the skin provide clues or indeed may cause concern; for example, deep bruising over the anterior triangle of the neck (from a central line) should not be misinterpreted as a sign of assault.

## Cardiopulmonary Resuscitation

Marks left by cardiopulmonary resuscitation (CPR) are not unusual. In order to be effective, CPR is a physically forceful procedure. It may leave marks externally and internally. Chest hair is shaved in order to improve electrical conduction. The defibrillator pads are extremely sticky and can cause superficial skin loss when removed. By the time of autopsy, the areas of skin loss will have the appearance of an abrasion with parchment texture. The ribs fracture during chest compressions. Palpation of the anterior chest wall may

demonstrate loss of ribcage integrity, leaving the internal examination to clarify which ribs are fractured. There may be numerous needle puncture marks due to attempted venous and arterial sampling. The location and dimensions of skin abrasions and the location of ECG stickers and so forth are recorded, but in the report are often summarised as 'features consistent with CPR'.

## Scars

Scars are identifying marks that may be a result of surgery, minor trauma, tribal marking, or self-harm. The site, size, and state of the skin affected (fresh, healing, healed) is documented for each mark, and the surrounding skin is examined for signs of infection or surgical emphysema. Scarring that does not fit with the history provided or that is unusual in type or distribution should raise the possibility of injury that is not accidental or self-inflicted. Further information should be obtained before proceeding.

Scars have particular characteristics that allow presumption of origin. Incised surgical wounds heal by primary intention and leave a narrow, linear scar. Those that heal by secondary intention have a wider profile. The pathologist should be aware of the common surgical approaches in major surgery and therefore be able to predict which internal organs may be missing or repaired. One should pay close attention to the lateral aspects of the body so as not to miss nephrectomy, hip surgery, and/or thoracotomy scars. The type of wound closure and the state of healing is documented, with or without photography.

Autopsy practitioners should be aware that surgical techniques and approaches have changed over time and continue to do so. Thus large laparotomy scars have been replaced by laparoscopic processes, so one must diligently hunt out faint 10- to 20-mm port scars across the abdomen.

Stretch marks and skin creases can be problematic; palpation of the mark and finding fibrous subcutaneous tissue should help determine a true scar. Old, healed scars may show hypertrophic or keloid changes.

Scars from old minor trauma are usually less regular in outline and can be found almost anywhere on the body. Self-inflicted injury, such as repeated cutting of the skin, leads to multiple linear scars that are usually located on the arms and legs, but the breast and abdomen are not unusual sites. The scars are likely to be clustered together and arranged in parallel lines. It may be difficult to determine the exact number. One approach is to record the location of the scars, the longest and shortest measurements, and the nature of the arrangement.

## Identifying Marks

The main purpose of recording identifying marks is for identification, but they also serve as clues to previous operations or procedures (*e.g.*, radiotherapy marking) or health risks (e.g., street-art cosmetic tattoos, with a potential risk of blood-borne viruses). One should document the type of identifying mark (Table 2.2), its location, description, size, and state of healing (fresh, healing, healed), remembering to look for signs of infection.

One should always consider identifiers with cultural or society realities. There are some good texts on this material [8] dealing with body modifications and piercings and cultural body modifications. One advises caution about commenting on specific details of artistic adornments or nonsurgical modifications. Stating that there is a tattoo on the left forearm measuring up to 30 mm is unlikely to cause problems, but incorrectly interpreting text or figurative drawings may lead to confusion, irritation, anger, or distress for the family.

**Table 2.2** Examples of identifying marks

| Type of marks | Examples |
|---|---|
| Adornments | Tattoos (artistic, related to radiation therapy, related to occupation; record text, style, position of tattoo) |
| | Piercings |
| | Scarification |
| | Branding |
| Nonsurgical modifications | Cosmetics |
| | Hair treatments/extensions |
| | Nail extensions |
| Surgical modifications | Implants |
| | Prostheses |
| | Gender reassignment |
| Scars | Surgical |
| | Old injury |
| | Self-harm |

## Indicators of Internal Disease and General Health

The skin provides information about the age, general health, nutrition status, and mobility of the deceased, primary dermatological diseases, and systemic illness. As the largest provider of information during the external examination, it is considered separately in this section.

In older individuals, the skin is thinner, more fragile, and more likely to be damaged, even during handling after death. Signs of aging include an increase in seborrheic keratosis, loss of elasticity, and Campbell de Morgan spots (cherry haemangiomas).

General and nutritional health, care, and neglect may be reflected in the general appearance, condition, and cleanliness of the skin. Pressure ulcers over bony prominences and the sacrum (due to immobility and occasionally to limited nursing care) are described by site and degree of skin breakdown. (Ideally take a photograph with a scale rule.) Poorly controlled diabetes mellitus may cause nonhealing arterial or neuropathic ulcers in the lower limbs, which have a characteristic punched-out appearance. Both diabetes and severe peripheral vascular disease may result in hair loss over the lower limb, gangrene, or distal surgical amputations.

Features that might indicate potential neglect of the deceased should be considered carefully before any further examination takes place, particularly if the individual has been in social, nursing, or hospital care facilities. In such cases, forensic/specialist practitioners may need to be involved.

The site, size, location and description of primary skin pathology such as individual skin tumours (whether benign or malignant) should be recorded. Multiple skin tumours may be associated with underlying genetic disease (*e.g.*, neurofibromatosis) or immunosuppression (*e.g.*, renal transplant patients). In some cases, the changes clearly indicate a potential cause of death (*e.g.*, malignant melanoma). Chronic inflammatory diseases of the skin such as psoriasis and eczema are usually not life-threatening but should prompt one to look for the effects of chronic steroid use. These conditions may point towards an 'Addisonian-type' crisis as a mode of death. Clearly, any generalised skin inflammatory process will debilitate an individual, and in cases of infection, a rash may suggest fatal bacteraemia. (The rash of meningococcal sepsis, for example, remains after death.)

A variety of colour changes are useful clues as to internal disease. As in life, bright yellow discolouration of jaundice is an indicator of raised bilirubin, consistent with red cell lysis, biliary obstruction, and/or liver failure. It should be noted that the classic skin stigmata of chronic liver disease such as palmar erythema and spider naevi fade when the circulation ceases, but abdominal varices and clubbing will persist.

Pallor is a good indicator of anaemia in the living, but it is a less specific sign after death. Gross pallor may nevertheless be due to massive internal haemorrhage.

In situations of carbon monoxide poisoning, the skin takes on a cherry-red colour due to carboxyhaemoglobin formation.

The blue discolouration of cyanosis, seen ante-mortem in chronic respiratory illnesses, is less specific after death [9]. Indeed, the peripheries are often noted to have a dusky hue in those both with and without chronic lung disease. Other colour clues include focal skin discolouration, such as tar staining on fingers in those who are heavy tobacco smokers, or coal tattoos affecting miners and a few other occupations.

The distribution and quality of petechial haemorrhages in the skin should always be assessed. Those resulting from asphyxia are likely to be distributed in the face and neck, whereas those from disorders causing thrombocytopenia have an irregular distribution across the body and are likely to be accompanied by larger purpura changes. In addition, the petechial haemorrhages seen within areas of dependent lividity have a coarse appearance. Disorders of coagulation (liver failure, haematological malignancy) may lead to widespread purpuric bruising.

Autoimmune diseases may cause scarring and skin deformity (*e.g.*, scleroderma). If the external examination raises the possibility of an infectious disease, it is important to notify other people working with you so that appropriate safety measures can be taken.

## Schematic Examination Guides the Assessment of Internal Pathology

Each part of the body is carefully examined for external features of disease (Table 2.3). Pathologists ideally should be familiar with many of the external stigmata of systemic diseases. This book cannot cover all possibilities; other texts [10] and a clinical examination textbook should be consulted where necessary [11]. Site-specific or device-specific medical or surgical intervention related to underlying disease processes should be simultaneously considered.

Positive findings immediately guide the internal examination, and relevant supporting internal pathology should be sought to corroborate external findings. For example, an abdominal surgical scar should make one consider not only visceral gastrointestinal surgery but also hernia repair. In the same way, information from the clinical history should be corroborated with the external examination; a clinical diagnosis of pulmonary embolism, for instance, should direct one to carefully examine the symmetry of the lower limbs. Documenting important negative findings is also prudent, based on what is expected when one reviews the clinical ante-mortem data.

**Table 2.3** Considerations at specific body sites during the external examination[a]

| Body site | Considerations during examination |
|---|---|
| Skin | Assess age, general health, nutrition, and cleanliness |
| | Look for signs of immobility or neglect. Describe the site and size of primary skin pathology (tumours, rashes, etc.) |
| | Look for signs of systemic diseases: autoimmune, infectious, haematological, etc. Identify signs pointing to specific illnesses: *e.g.*, chronic liver disease, peripheral vascular disease, diabetes mellitus |
| Hair | Colour, length, hair loss |
| Scalp | Look and feel for injury |
| Face | Note characteristics of congenital syndromes. Feel integrity of the facial skeleton. Look for xanthelasma |
| Ears | Look for fluid in the external auditory canal, earlobe creases (associated with cardiovascular disease), postauricular bruising |
| Eyes | Surrounding tissues: periorbital oedema, panda eye bruising |
| | Iris: colour (if difficult to decide, use pale *vs* dark) |
| | Sclera: colour, petechial haemorrhages |
| | Conjunctiva: colour, congestion (commonly seen in cardiac arrests), petechial haemorrhages (invert the lower lids) |
| | Cornea: cataract, corneal arcus (significant if <40 year), eyeliner/tache noire (discolouration occurring when eyelids are left open after death) |
| | Pupils: symmetry no longer relevant after death |
| Nose | Integrity of the nasal septum (can disintegrate with heavy cocaine use) |
| Lips | Angular stomatitis (vitamin deficiency) |
| Tongue | Injury (epilepsy), glossitis (vitamin deficiency), candida (immunosuppression, steroid use) |
| Teeth | The presence and condition of teeth/dentures |
| Gums | Gingivitis, hypertrophy (phenytoin treatment) |
| Mouth | Note fluid around or in the mouth. Look for foreign body, enlarged tonsils, and aphthous ulcers (inflammatory bowel disease). Check for a high arched palate (Marfan's) |
| Neck | Feel for masses: lymph nodes, thyroid gland, cysts, tumours |
| | Check trachea: central (deviated in pneumothorax), tracheostomy |
| Shoulders, clavicles | Articulate to reveal closed fracture/dislocations |
| Upper and lower arms | Compare sides, assessing symmetry and colour (upper limb DVT, limb ischaemia). Palpate axillary lymph nodes. Examine elbow for rheumatoid nodules. This is common site for intravenous access; check for thrombophlebitis and localised fluid accumulation from 'tissued' cannulas. Palpate scars to reveal AV fistulas of renal dialysis |
| Hands, fingers | Check for missing digits, muscle wasting (may be neurological or musculoskeletal). Assess bony deformities of rheumatoid and osteoarthritis. Examine interdigital web spaces for scabies and needle puncture marks in intravenous drug user (IVDU) cases. Feel for Dupuytren's contracture (liver disease) |
| Nails | Nail signs remain after death: leukonychia (hypoalbuminemia), koilonychia (iron deficiency), splinter haemorrhages (infective endocarditis, manual work), pitting (psoriasis), paronychia, onychomycosis, digital clubbing |
| Chest | Assess shape: barrel chest (chronic lung disease), pigeon chest (congenital). Palpate left upper chest for pacemaker. Compress ribs to elicit fractures. Check lateral chest for old chest drain scars |
| Breast | Palpation may reveal breast masses, implants, or gynecomastia. Look for skin changes associated with breast disease. Lift breast to check for scars or skin infection |
| Abdomen | Distention may be due to fat, fluid, faeces, fetus, or gas. Lift the abdominal apron to examine skin creases for infection, obscured medical paraphernalia, and injury. Look for bruising caused by common subcutaneous injections (insulin, low molecular weight heparin). Examine each quadrant carefully, looking for scars and hernias (epigastric, umbilical, inguinal, incisional). Note surgical drains, recording volume and appearance of fluid in drain bags. Removal of stoma bags is necessary; check that a replacement bag is available for reconstruction post autopsy. Cullen's sign (umbilical/periumbilical bruising) is a rare sign of retroperitoneal haemorrhage (ectopic pregnancy, acute pancreatitis, etc.) |

**Table 2.3** (continued)

| Body site | Considerations during examination |
|---|---|
| Groin | Needle puncture marks may be from emergency care or intravenous drug use (IVDU), deep sinus formation is due to chronic intravenous drug use. Check for hernias (unlikely to be palpable in a prone dead body unless very large). Palpate for inguinal lymph nodes. This is a common site for interventional radiological access, so be alert for iatrogenic injury |
| External genitalia | Lift the penis to examine the scrotum and testes |
| | Scrotal oedema is seen in heart failure and fluid overload. Palpate the scrotum to identify both testes (prosthetic testicles can be implanted after orchiectomy), swellings (hydrocele), and tumours. Examine penis and vulva for gross pathology, piercings, and tattoos |
| | If you are considering a speculum examination of the vagina, are you the correct person to carry out the autopsy? |
| Upper and lower legs | Limb deformity may be due to congenital or acquired musculoskeletal or neurological diseases. Note the level of any amputation and comment on the condition of the stump |
| | Examine over joints for surgical scars. If legs are asymmetrical, measure calf circumference (10 cm below tibial tuberosity); a difference of ≥3 cm raises possibility of DVT. Comment on surface changes: unilateral skin mottling due to critical limb ischaemia remains after death; venous insufficiency leads to hair loss, venous eczema/stasis dermatitis, hyperpigmentation, oedema, varicose veins, and/or cellulitis. Distinguish between arterial, venous, and pressure ulcers. Apply firm pressure over a distal bony prominence to assess for pitting oedema. Moving proximally, repeat to find most proximal level (remembering that there will be dependent pooling of plasma on the posterior surface) |
| Feet, toes | Check for missing toes, positional deformity (neurological, musculoskeletal). Look for changes related to rheumatoid and osteoarthritis. Examine interdigital web spaces for scabies and needle puncture marks in IVDU cases. In diabetics, check the feet carefully for signs of injury and infection |
| Toenails | Note if the toenails are extremely long; this can lead to falls in the elderly. Long nails can also damage the skin and have been implicated in variable haemorrhage and/or local skin sepsis |
| Lateral surfaces | Grey-Turner's sign (bruising over the flank) is due to retroperitoneal blood (*e.g.*, acute pancreatitis). Bruising in this area also can be due to trauma (fight or accident) and may not have been noticed by the police. Scars from nephrectomy or thoracotomy are found at this location |
| Posterior surfaces | A visual assessment of the back will reveal spinal deformity, surgical scars, and intrathecal devices. Attention should be paid to the sacral pressure areas, particularly in bedbound and elderly people. Pressure areas are measured and the depth of epidermal damage is noted. The posterior surfaces of the limbs should be examined for injury and tattoos |
| Anus | Haemorrhoids and skin tags are not uncommon. There may be faecal matter or malaena around the anus. Remember to be alert for injury |

*AV* arteriovenous, *DVT* deep vein thrombosis, *IVDU* intravenous drug use
[a]Injuries, identifying marks, and devices are noted for all locations

Generalised features such as muscle wasting may indicate malnutrition or musculoskeletal or neurological disease. There are many causes of pitting peripheral oedema. The lymph nodes in the neck, axilla, and groin are palpated to identify widespread or focal lymphadenopathy.

A constellation of findings may provide good supporting evidence for a specific internal or systemic pathological process, such as a distended abdomen with incarcerated and discoloured incisional hernia, which is in keeping with bowel obstruction and fatal toxaemia. Similarly, a constellation of findings may point to a narrow differential, as when shiny, hairless skin on the lower leg with distal ulceration, gangrene, and previous toe amputation point to chronic peripheral vascular disease and diabetes mellitus.

## Trauma and Injury

Traumatic injury can result in direct, indirect, or delayed death. The pathologist may be examining injuries from a recent accident or one that happened in the past. It is important to record findings accurately (Table 2.4), and photography is extremely useful in this instance. The types of injuries and their relevance to the cause of death is discussed in Chap. 12.

**Table 2.4** Terminology and images of wounds and fractures

| Injury | Definition |
|---|---|
| Abrasion | Superficial graze or scratch |
| Bruise | Result of blunt trauma without skin breakage, causing discolouration through various stages: red →blue →green →yellow →brown |
| Laceration | Result of blunt trauma causing the skin to tear |
| Incision | A wound caused by a sharp edge |
| Closed fracture | Bone does not protrude through skin |
| Open fracture | Bone protrudes through skin |
| Amputation | Limb or digit missing; may be traumatic or surgical |

Bony injury may be visible or may be revealed by palpation or articulation of the limbs, thoracic cage, pelvis, and scalp. The identification of any closed fractures should be communicated to the person eviscerating the body so as to guard against injury from sharp-edged bony fragments. Injury sustained once blood flow has ceased will lack a surrounding skin reaction, helping to differentiate between pre-mortem and post-mortem injuries.

Recognising that the general autopsy make take place with an incomplete or limited history of the events leading up to death, it is vital to be alert for findings that do not quite fit the story given. Very careful consideration is paid to the mechanism of any traumatic injury and how it relates to the clinical history provided.

Specifically, the external examination is to ensure that the correct post-mortem examination is carried out. Unexpected or unusual findings should be discussed with police or legal representatives or relevant medical staff. It is better to pause an examination in order to gather more information than to go ahead with the evisceration and potentially destroy evidence.

## External Examination as Part of the Radiological Autopsy

Post-mortem imaging is being increasingly used as an adjunct to autopsy examination (see Chap. 13). This evolving specialist technique has demonstrated good results in assessing traumatic injury [12]. Accessing hospital pre-mortem emergency scans may be also feasible in some settings. A forensic text should be consulted for in-depth descriptions of traumatic injuries and their common mechanisms [9].

## Post-mortem Changes and Decomposition

Post-mortem changes and decomposition will be the least familiar external features to the uninitiated pathologist. Importantly, the pathologist must guard against over-interpreting decomposition and post-mortem damage as injury related to the death. Therefore, an understanding of the basic physiological changes in anatomical tissues after death and an overview of the macroscopic appearances is required.

The features may be documented in some detail at the time of external examination but are often summarised carefully in the final report so as to give accurate information to the reader whilst minimising unnecessary distress to the bereaved, should they read the report.

The complexity of organic matter decomposition is beyond the scope of this brief text, as it is affected by a multitude of environmental and endogenous conditions. The literature refers to various sequential stages of decomposition (Table 2.5), but there is usually a degree of heterogeneity in external decomposition.

Once circulation ceases, the blood and plasma fall to the dependent areas of the body (hypostasis). Haemoglobin breakdown gives a red-blue hue to the skin (lividity), and plasma pooling leads to dependent oedema. Small pinpoint, coarse haemorrhages may be seen within the post-mortem hypostasis, and these should not be over-interpreted as petechial haemorrhages of asphyxiation, which are finer and more discrete. Any item exerting pressure on the skin at the time of death will leave a negative imprint in the area of lividity.

Large, superficial, fragile skin blisters are due the pooled plasma moving into subcutaneous and epidermal tissues. These blisters may break or coalesce, leading to small or large areas of skin slippage. Areas of superficial epidermal loss become dry and attain a parchment-like quality. These areas may resemble superficial burns, but confusion is unlikely when the clinical history and circumstances of death are considered.

The actions of endogenous cellular mechanisms (autolysis) and microbial proliferation (putrefaction) gradually break down cellular tissue components. Liquefaction of mucosal linings happens rapidly, and brown fluid seen in or around the mouth and nasopharynx should not be over-interpreted as evidence of upper gastrointestinal haemorrhage. Haematemesis is not usually a subtle finding, and there will be internal findings to support the diagnosis.

**Table 2.5** Progression of putrefactive decomposition in a temperate climate

| Days since death | Changes observed |
|---|---|
| 0–1 | Dependent post-mortem hypostasis |
| | Development of rigor mortis |
| 1–2 | Green discolouration of the anterior abdominal wall |
| 2–3 | Abdominal bloating begins |
| 3–4 | Venous marbling begins |
| 5–6 | Abdominal bloating established |
| | Skin slippage and blister formation |
| 14 | Marked bloating of the abdomen and scrotum |
| 21 | General softening of the tissues |
| | Bulging eyes |
| 28 | Generalised blackening of the skin |
| | Skin liquefaction |

Modified from Table 20.1 from Burton [14]; with permission

Green discolouration of the skin is usually an early sign of internal decomposition. Red blood cells leak into the subcutaneous tissues and bacteria convert haemoglobin into sulphaemoglobin. Because the caecum contains a high concentration of gut flora and is located relatively near to the skin surface, this process commonly begins in the right lower quadrant of the abdomen and spreads across the abdomen and flanks. Venous marbling is due to decomposition of haemoglobin within blood vessels and gas assisting the travel of sulphaemoglobin through vessels and lymphatics [13]. The result is a striking appearance of superficial vessels against the usual skin tone of the deceased, giving a marbled appearance, which can be very pronounced in a very short time in those who die in circumstances of overwhelming sepsis. Gas formation also leads to abdominal bloating, and the internal soft tissues appear 'bubbly' (similar to surgical emphysema seen in vivo).

Sometimes bodies are remarkably well-preserved, even if they have come to the attention of the authorities some time after death. This state may be due to mummification, as occurs in dry or cold environments, or to the formation of apodicere (a waxlike organic substance formed by the anaerobic bacterial hydrolysis of fat), more common in wet environments.

In general, external tissues soften and become black as they putrefy. Maggot infestation begins in natural orifices but spreads throughout the body. Maggots flee from light and are best assessed when the body bag is first opened. Care is taken to ensure that all maggots that escape from the post-mortem table are cleared up, so as to prevent fly infestations within the mortuary. Whilst entomology is a central topic of interest in forensic dramas, it is very rarely relevant to the general autopsy. One only needs to comment on the presence of eggs, maggots, or flies; it is not necessary to document the stages of pupation or the number of adult insects.

After putrefaction, all that remains of the deceased is musculoskeletal or skeletal tissue. If the body is not yet identified, a specialist orthodontic examination is required. Even with very little human remains, a careful external examination with appropriate sampling is required.

In most cases, as a general rule, the pathologist needs only to comment on early or late decomposition changes and to document specific findings such as maggot infestation or bite marks left by domestic, wild, or marine animals (Figs. 2.97, 2.98, 2.99, and 2.100). It is unlikely that the pathologist would be asked to provide a time of death based on decomposition; such cases should be handled by those with experience in decomposition changes.

**Fig. 2.97** Decomposition is affected by environmental factors such as temperature, but also microbes, organisms, and insects. This is fungal bloom over the head and neck of a man found hanged

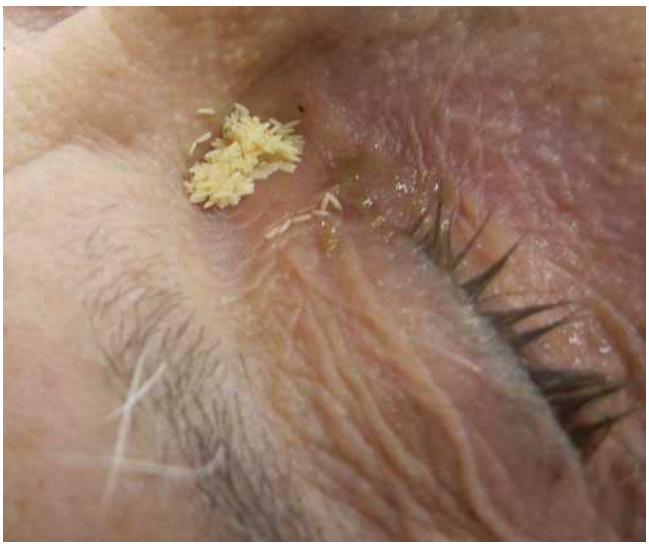

**Fig. 2.99** Early decomposition. The presence of a body in the open often leads to flies laying eggs at mucocutaneous junctions early—in this case, at the eye. These eggs will hatch after a short period and the maggots will degrade the decomposing flesh, as in Fig. 2.100

**Fig. 2.98** Animal marks. Domestic, wild, or marine animals may scavenge tissue from the dead. In this case, a domestic pet was responsible for the loss of tissue over the face

**Fig. 2.100** Infestation and decomposition. The external tissues soften and become brown-black as they putrefy. Maggot infestation is seen initially in natural orifices but will spread throughout the body. Maggots flee from light and are best assessed when the body bag is first opened. Small holes are seen on the surface of the body where the maggots have burrowed into deeper tissues

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# Evisceration

3

Maxine S. Coe and S. Kim Suvarna

## The Process of Opening the Torso and Removing the Tissues

The process of evisceration involves the removal of the organs from the mouth down to the anus with preservation of anatomical organ and tissue relationships. The process is performed in stages, allowing the consideration of various pathological constructs, such as the presence of pneumothorax, examination of the cavities for fluid collections, and direct inspection of the tissues for haemorrhage or other focal lesions.

The process also allows staged reduction of the tissue volume, as the majority of the bowel tissues are removed prior to the thoracic, upper gastrointestinal tract, retroperitoneal, and pelvic contents. The images in this chapter detail the stages in the recommended dissection pathway, although it is accepted that many practitioners have their own variations and/or protocols. The technique presented is recommended as suitable for most autopsies.

Following inspection of the cavities, the body may be prepared for reconstruction whilst dissection of the main organs continues.

There are many different permutations for organ removal, some advising removal of tissues in single block format and others suggesting smaller blocks. Although it is possible to extract the mouth/neck and thoracic tissues in one piece, extract the abdominal bowel tissues, resect the retroperitoneal content down to the pelvic rim, and remove the pelvic organs in sequential and separate parts, doing so will necessarily alter anatomical relationships and is not advocated for standard autopsy purposes. One should always dissect tissues in a manner likely to maximize the data to be collected and to assist sampling.

Finally, it should be remembered that high-risk infection cases may merit limited tissue handling or minimal dissection and direct tissue sampling once the torso has been opened.

## Samples and Investigations to be Taken During Evisceration

The advised dissection protocol provides the opportunity to consider pathology as it becomes visible, and permits the operator to adapt the dissection according to the needs of the case and/or the pathology under consideration.

The role of digital photography cannot be overemphasized in this regard, as initial views and then dissected images of pathology processes are a vital adjunct to both standard pathology and medico-legal cases. They also serve as a valuable resource for teaching.

It is possible to gather microbiology, toxicology, and other tissue samples at various stages during the dissection. Benefits include direct vision collection of the sample and a minimal manipulation field as the samples are retrieved.

This illustrated dissection protocol (Figs. 3.1, 3.2, 3.3, 3.4, 3.5, 3.6, 3.7, 3.8, 3.9, 3.10, 3.11, 3.12, 3.13, 3.14, 3.15, 3.16, 3.17, 3.18, 3.19, 3.20, 3.21, 3.22, 3.23, 3.24, 3.25, 3.26, 3.27, 3.28, 3.29, 3.30, 3.31, 3.32, 3.33, 3.34, 3.35, 3.36, 3.37, 3.38, 3.39, 3.40, 3.41, 3.42, 3.43, 3.44, 3.45, 3.46, 3.47, 3.48, 3.49, 3.50, and 3.51) also allows direct inspect of the inner aspect of the torso and thoracic walls. Later access to the vertebral column (for bone/marrow samples) is also possible. It is relatively easy to progress onwards to the spinal cord following removal of the vertebral column, psoas musculature, and liver tissues if toxicology samples are required (*e.g.*, decomposed cases). The process also allows one to see the undersurface of the palate and tissues of the postnasal space.

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**Fig. 3.1** The evisceration process starts with a block being placed under the shoulders of the individual so that the head is pulled backwards by gravity into a hyperextended posture. This allows direct inspection of the anterior neck tissues and identification of any scars, which are often hidden within skin creases at this site

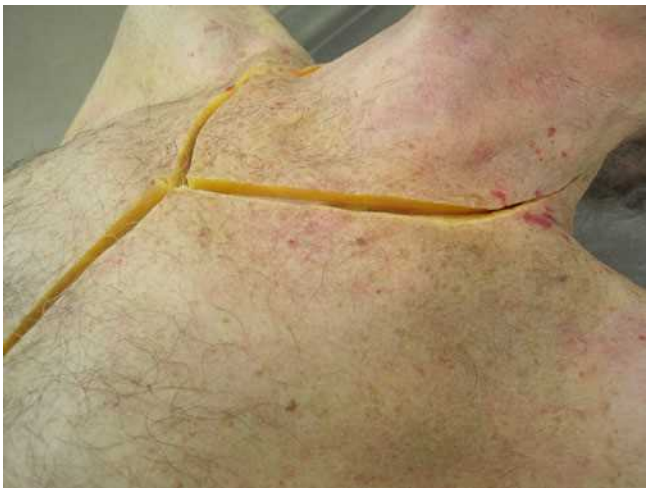

**Fig. 3.3** The cuts from either side can join in a V-shaped interface at the manubrium level and then pass as a single cut along the front of the chest and downwards towards the pubis/low abdomen centrally

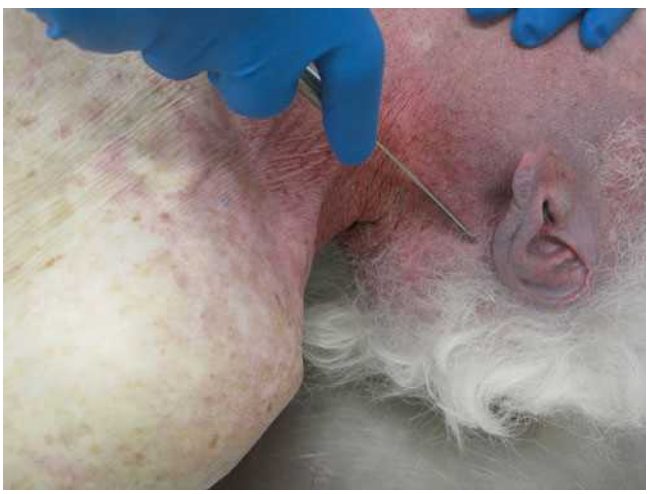

**Fig. 3.2** An incision is passed from the mastoid process downwards and forwards, alongside the edge of the sternomastoid muscle and down across the neck. The cut should be made as perpendicular to the skin as possible, although it is recognized that the neck is a complicated three-dimensional structure

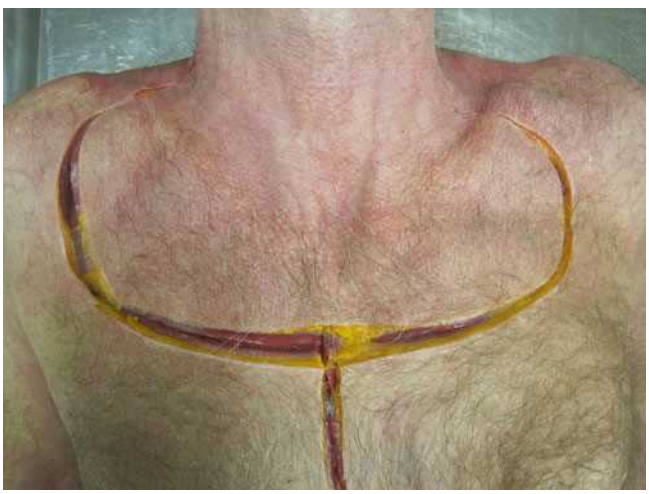

**Fig. 3.4** An alternative method for dissection involves the cuts from the mastoid processes running behind the sternomastoid musculature and then swinging in a U-shaped fashion across the front of the chest. These cuts then continue down the midline of the central chest and abdomen

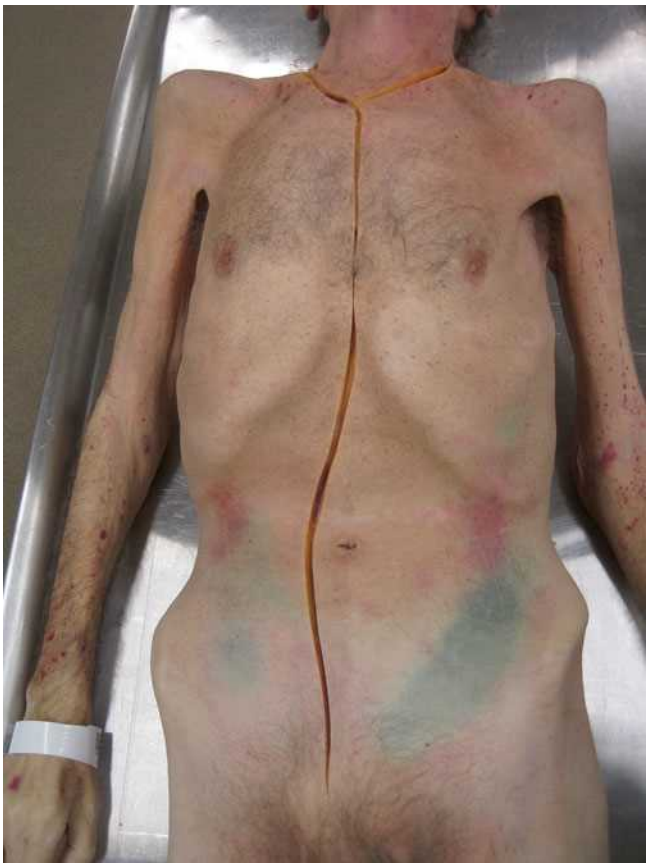

**Fig. 3.5** The midline dissection, usually by knife, passes down to the suprapubic region, avoiding the umbilicus

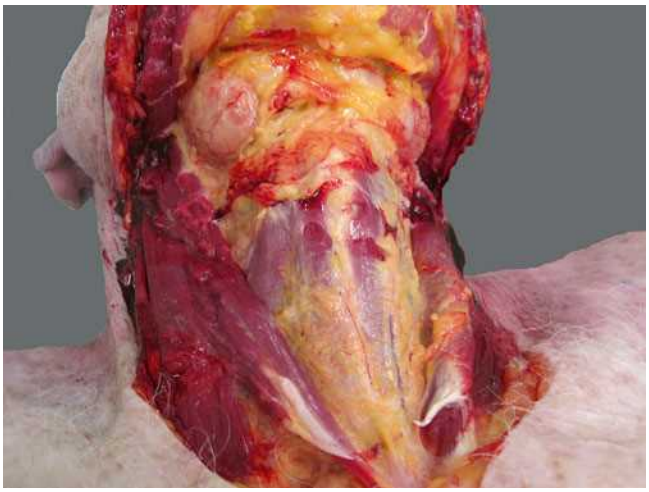

**Fig. 3.7** In this case, the V-shaped dissection has allowed the soft tissues of the skin and fatty subcutis to be dissected free of the local neck structures. These are lifted upwards towards the jaw. The dissection in this plane of tissue should continue up to the edge of the mandible, thereby exposing the strap muscles around the hyoid bone as well as the submandibular salivary gland tissues superiorly. At this point, one can consider whether to dissect the strap musculature sequentially, in order to consider damage to the hyoid bone/larynx (*e.g.*, in medico-legal or assault cases). It is also possible to approach the thyroid from the front, although dissection of the thyroid and related tissues often is left until later (see Chap. 8)

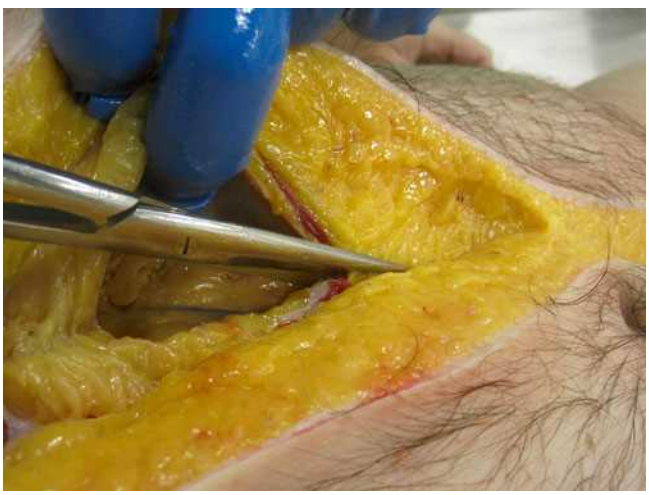

**Fig. 3.6** One can open directly into the abdominal cavity at this point with scissors, allowing direct inspection of the abdominal cavity

**Fig. 3.8** The tissues of the thorax are incised directly onto the sternum, with lateral undermining of the muscles and other soft tissues of the chest. These are reflected further laterally to expose the intercostal musculature and ribs. This dissection plane also allows identification of any pacemaker or other devices that penetrate into the vasculature or soft tissues around the chest. In this case, haemorrhage and fracturing of the ribs is evident focally

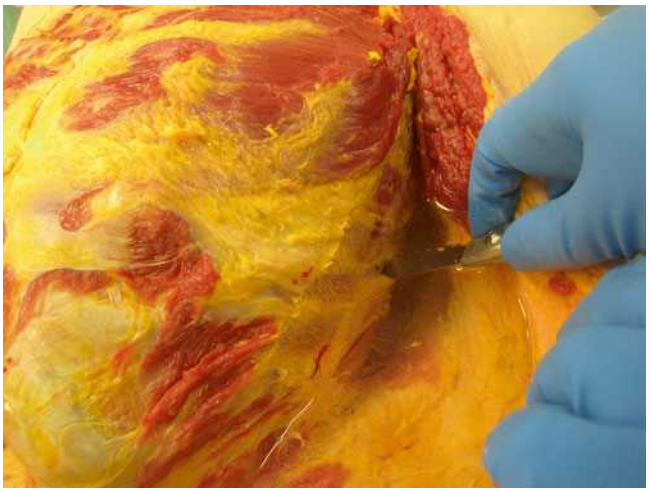

**Fig. 3.9** To test for pneumothorax, before the body is further opened, one dissects around the chest wall and reflects the soft tissue, thereby creating a pocket, which may be filled with water. The next step is to insert a knife or scissors into the intercostal musculature between the ribs, below the water level, penetrating into the pleural space. It is possible to facilitate the examination by turning the knife or scissors through one quarter turn and gently pressing on the front of the chest to check for air. Tension pneumothorax will be evident as a rush of gas being released at this point. Small, nonsignificant pneumothoraces may not be identified by this technique, but a normal pleural cavity will characteristically suck inwards some of the fluid without bubbles. One should also be aware that pushing a knife or scissors deeply into the chest will inevitably damage any underlying lung tissue

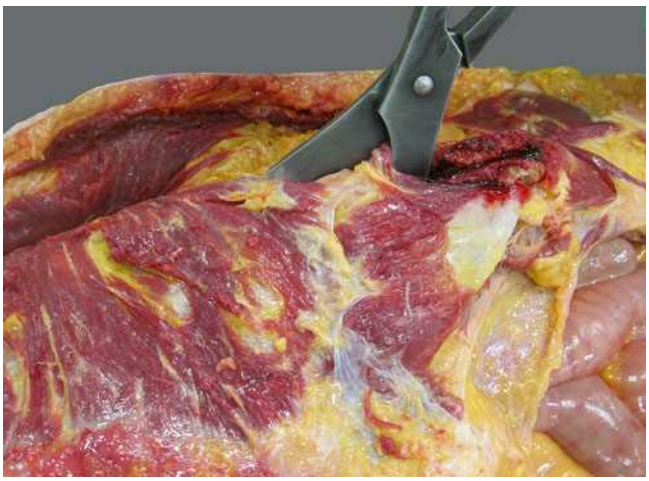

**Fig. 3.10** The chest wall is removed by inserting bone shears into the chest at the lower edge of the rib cage and then cutting across the musculature and bone tissues along the lateral aspect of the chest and towards the manubrioclavicular junction

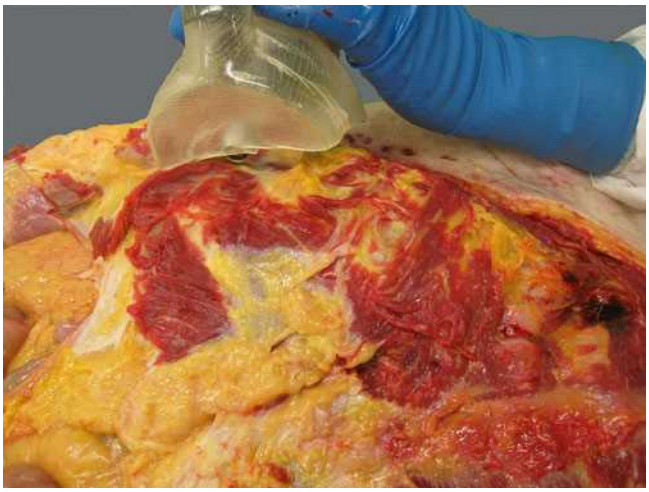

**Fig. 3.11** It is also possible to use a mechanical or hand saw to cut through rib tissues, though rib shears are quick and efficient. Note that the saw is fitted with an extractor hood to reduce bone dust aerosolisation

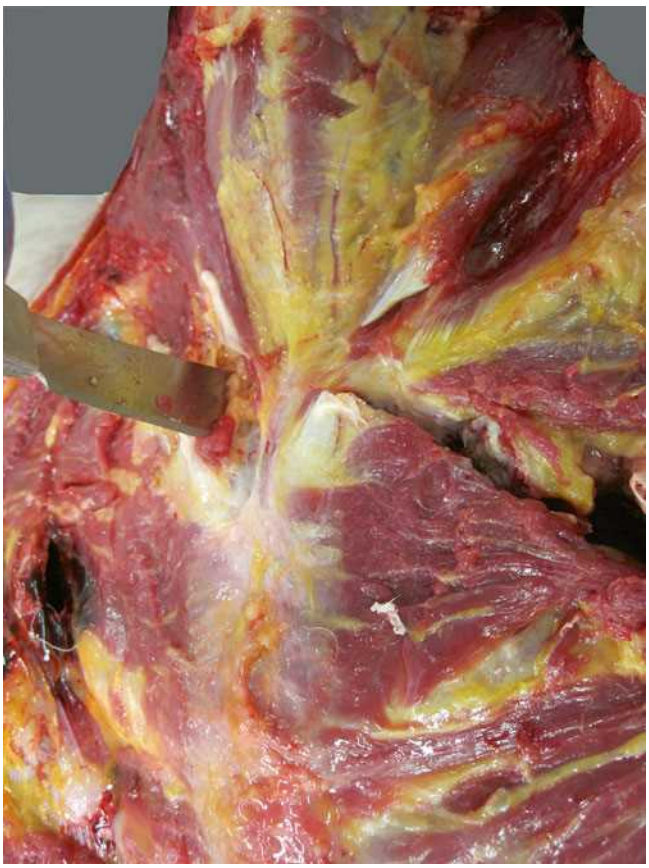

**Fig. 3.12** The removal of the chest bony tissues and front of the rib case initially requires the manubrioclavicular joint to be freed from its attachments. This joint can be identified by moving the ipsilateral shoulder slightly and feeling for the joint space at the manubrioclavicular junction. In this process, a knife blade is inserted vertically downwards and moved around the joint, allowing this tissue to be freed. In this image, the cutting of the ribs and that at the manubrioclavicular junction join up

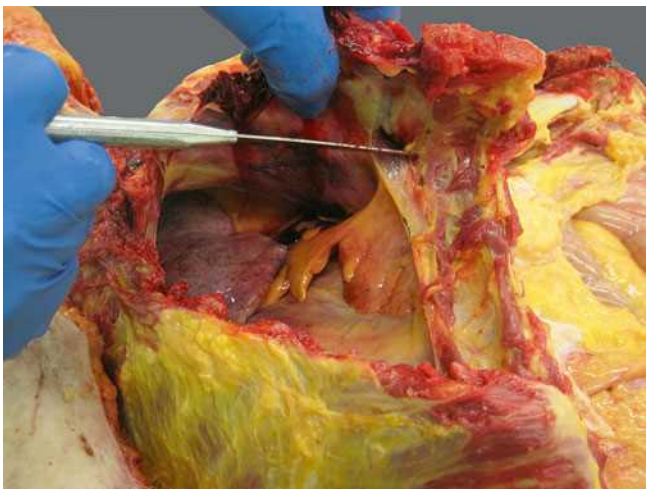

**Fig. 3.13** The lateral aspect of the rib cage is now lifted upwards, allowing a knife to be used to free up the mediastinal content tissues from the inner aspect of the chest contents. If the individual has undergone coronary bypass surgery, attention should be paid to the origin of the left internal mammary artery (LIMA), which is often used as part of the revascularisation technique. If relevant, a suture placed around the end of the artery may be useful in the subsequent examination of cardiac tissue (See Chap. 4.)

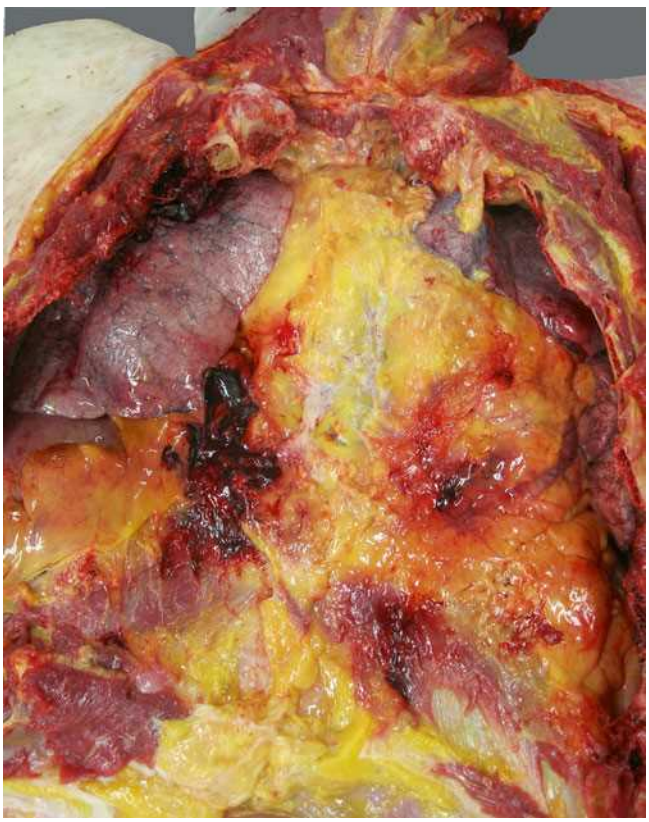

**Fig. 3.14** The rib cage has now been removed from the front of the body, exposing the lungs, heart, and upper abdominal vasculature. The pleural cavities should be inspected for fluid collections and sepsis. The pericardial tissue should be looked at from the external/outer aspect, and may be opened at this point. If there is a fluid collection, then it may be appropriate to aspirate the content. This image also shows some blood related to rib fractures incurred during attempted resuscitation

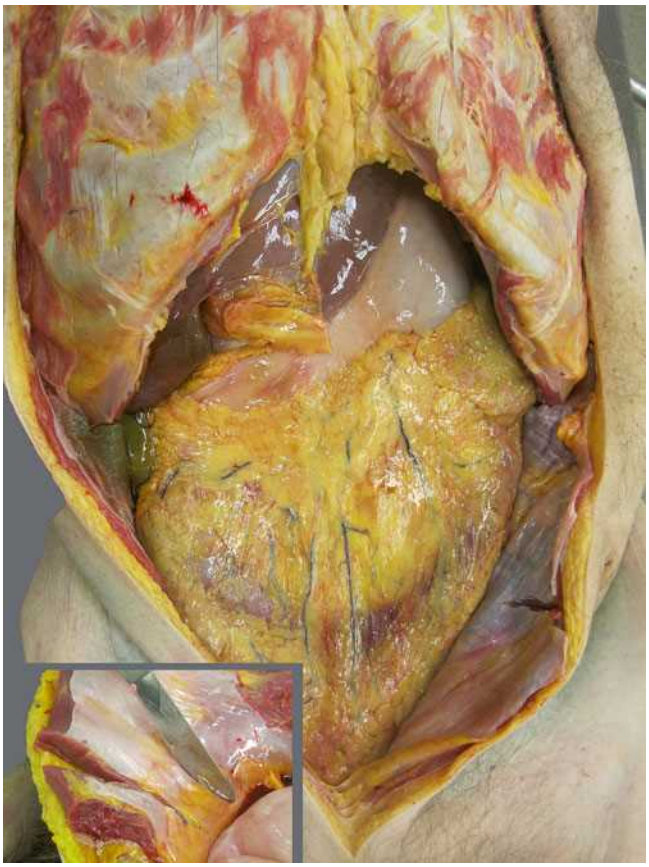

**Fig. 3.15** The abdominal tissues have been opened using the cut running along the front of the abdomen, which has been extended into the peritoneal cavity. The abdominal incision edges have been pulled to the sides, exposing the liver and small bowel. The small bowel is often seen to have a moderate amount of fatty tissue. The lower left inset image shows transverse cuts into the lower abdominal wall, which facilitate laying the abdominal wall outwards

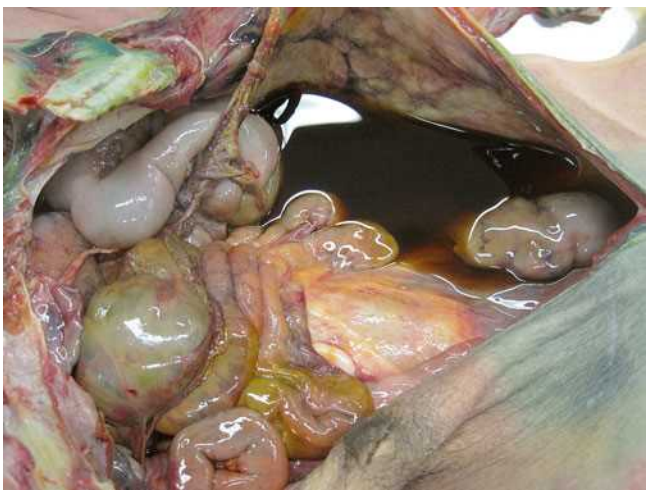

**Fig. 3.16** The examination of the abdominal content may often identify variable fluid collections. These may be serous (*i.e.*, clear yellow), haemorrhagic, or turbid/purulent. This case shows some altered blood on a serous background. Fluid for cytological or microbiological tests can be sampled at this point, before any contamination by further dissection. The color and consistency of the fluid may give an indication as to likely intra-abdominal pathology. In all cases, the total volume of fluid should be assessed

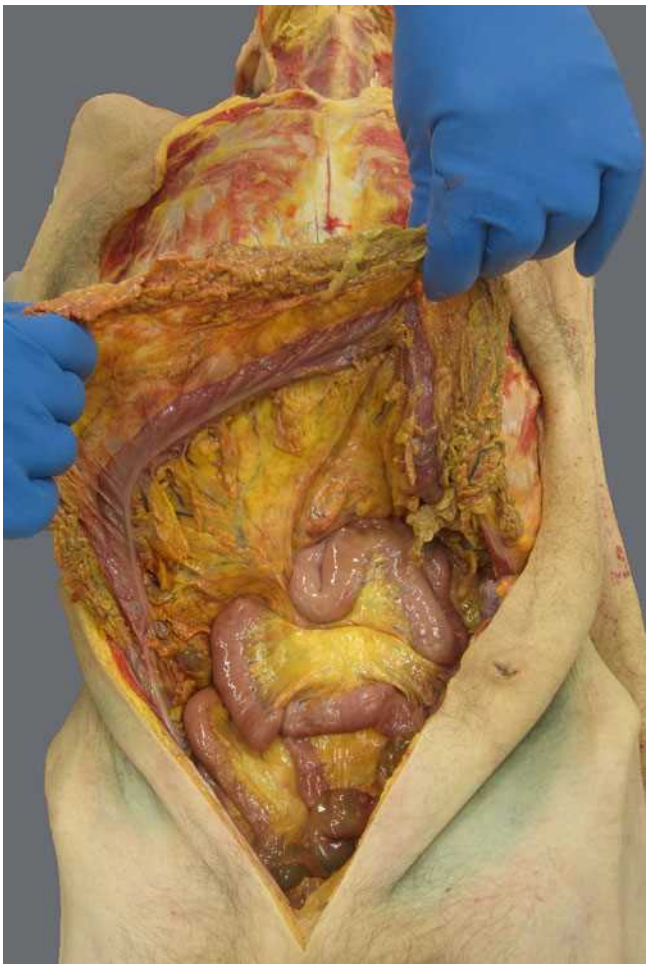

**Fig. 3.17** In most cases, the abdominal tissue content appears broadly normal with regard to the contents and architecture. To continue the examination, the proximal jejunal and distal duodenal tissues are identified by pulling the transverse colon upwards towards the head. After this manipulation, most of the small bowel is moved gently towards the right side, exposing the root of the mesentery

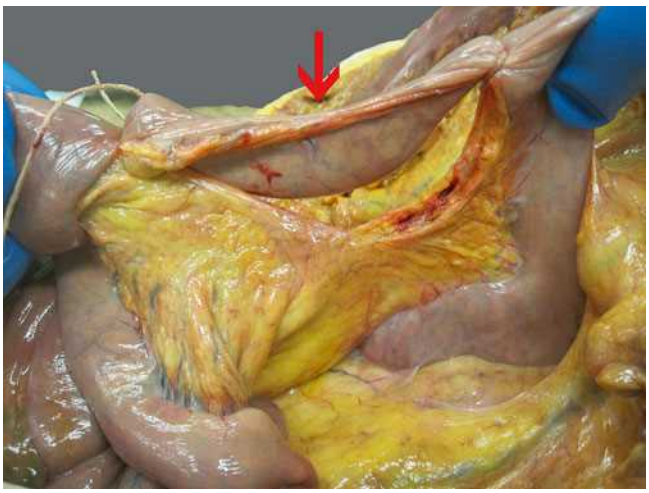

**Fig. 3.18** By making a single cut into the proximal jejunal mesentery, one can insert ties that will prevent bowel content leakage. One then cuts across the centre of this small bowel zone (*red arrow*)

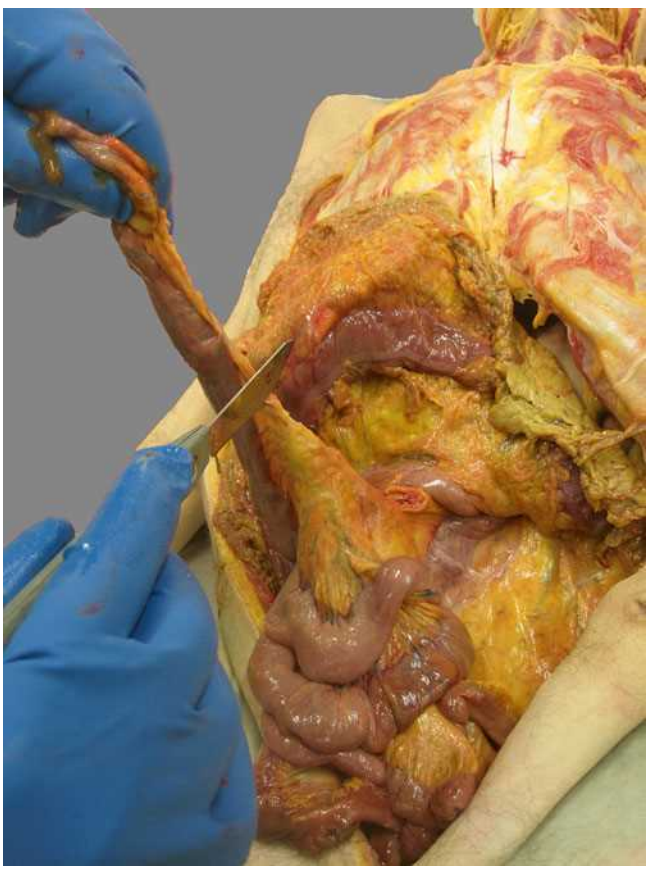

**Fig. 3.19** The small bowel (from jejunum to distal ileum) is dissected free by cutting through the mesentery adjacent to the edge of the small bowel. The cut allows the bowel to be removed proximal to distal. The bowel is lifted outwards and out of the body cavity

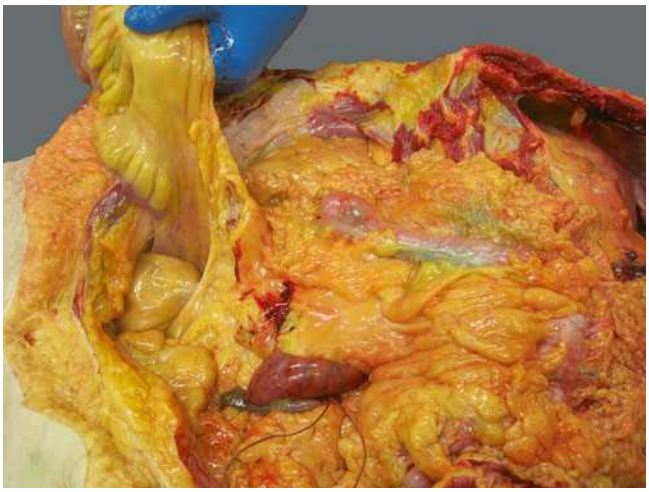

**Fig. 3.20** Alternatively, one can dissect the small bowel tissues by cutting close to the vascular origin. This process allows review of the vasculature in relation to the bowel tissues

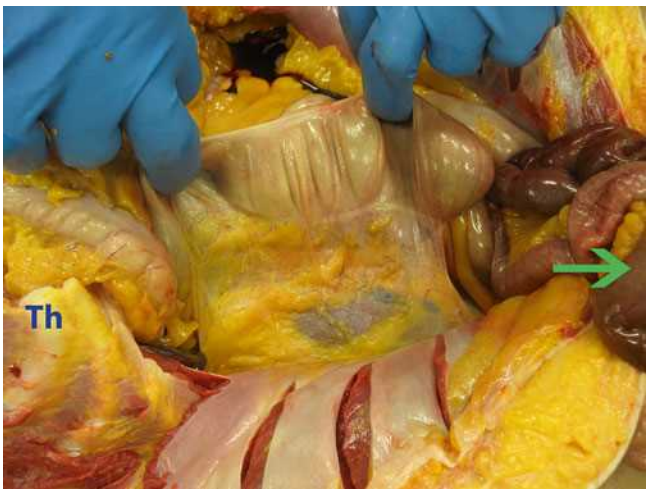


**Fig. 3.22** Looking from the right side of the opened abdomen, with the small bowel pulled downwards towards the feet (in the line of the *green arrow*), and with the part of the already opened thoracic rib cage (Th) in this view; one pulls the right side colon tissues medially and frees the bowel gently. This blunt dissection progresses up to the hepatic flexure

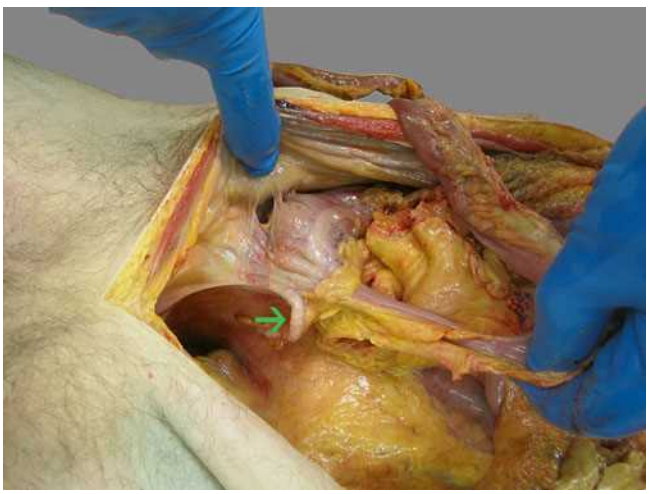

**Fig. 3.21** It is to be remembered that the cecum and ascending colon are retroperitoneal. When one has dissected down to the level of the terminal ileum, it is necessary to pull gently and bluntly dissect (with fingers/scissors) into the retroperitoneal compartment around the cecum, freeing the proximal colon tissues. At this point, the appendix (*arrow*) should be directly inspected to confirm normality and absence of inflammation

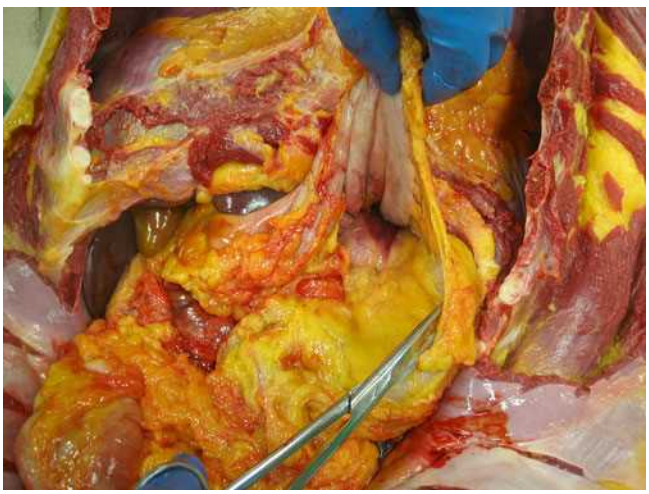

**Fig. 3.23** The cut passes across the top of the lesser sac using scissors, separating the stomach and transverse colon tissues. One can then look onto the anterior surface of the pancreas, to check for inflammation. The colonic tissues are then mobilized onwards, around the splenic flexure and down the descending colon in a similar manner to that on the right side, with the retroperitoneal descending colon tissues being mobilized downwards, towards the sigmoid

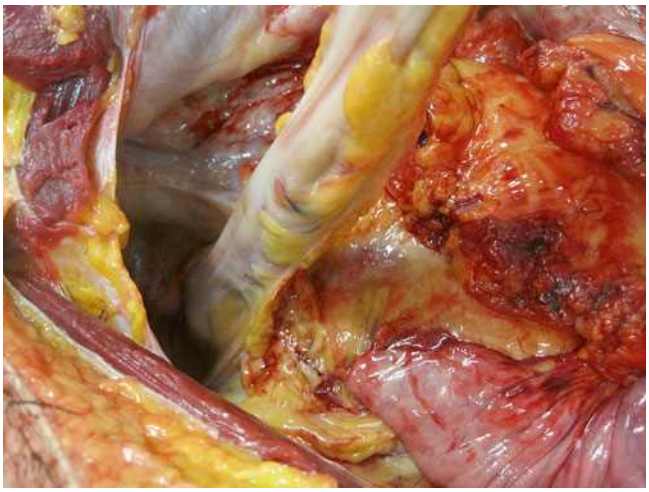

**Fig. 3.24** The pelvic tissues and sigmoid colon are freed and pulled upwards, freeing the sigmoid colon at the mesenteric root. One may then cut off the colon tissues down to the upper rectum level, with or without tying of the bowel at this point. All the bowel tissues are now removed and put to one side. Some may require further dissection. (See Chap. 5)

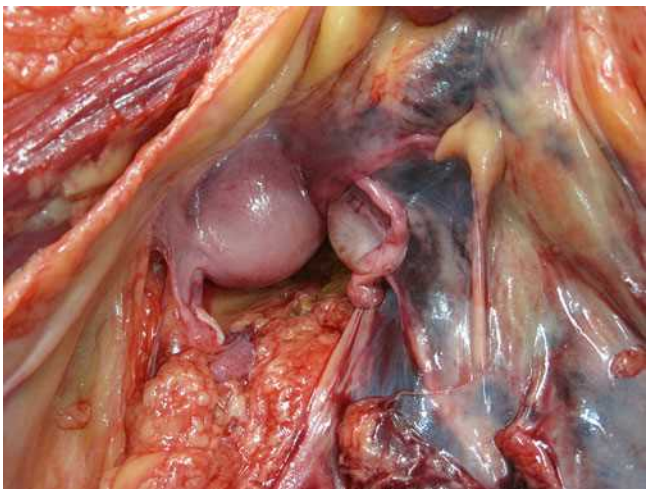

**Fig. 3.25** Once the colon and proximal tissues are removed, one can inspect the internal genital organs in females. In this case, the superior aspect of the uterus, the tubes and ovaries can be seen

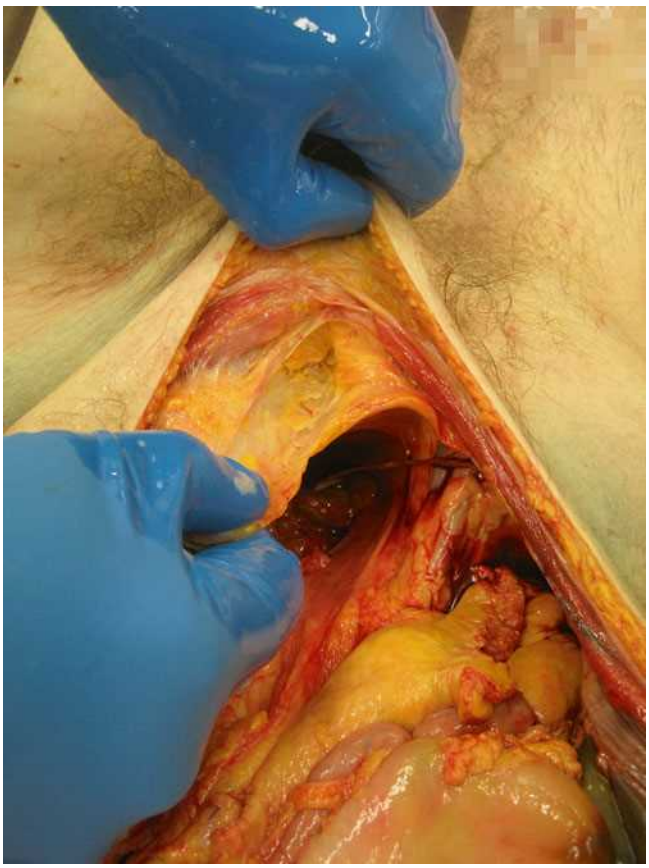

**Fig. 3.26** The pelvic tissues are removed with the bladder and rectal parenchyma intact. In starting this maneuver, the soft tissue plane in front of the bladder is identified at the distal end of the original abdominal incision

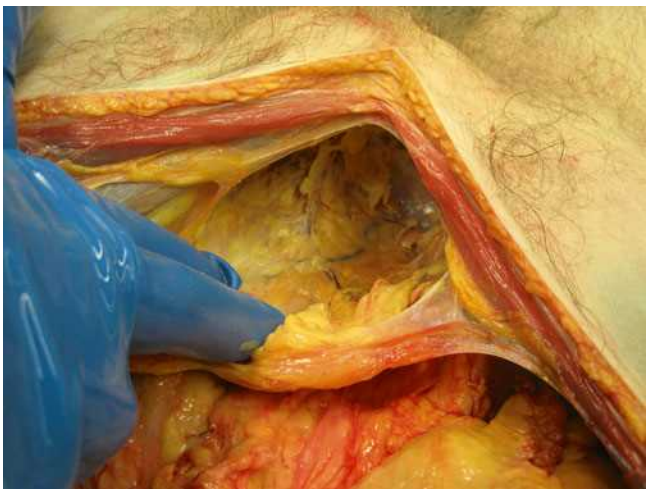

**Fig. 3.27** Using finger dissection down around the bladder neck, one creates a plane between the inner aspect of the pelvis anteriorly and the bladder

**Fig. 3.28** The finger dissection passes downwards and around the bladder neck/prostate (in men) or around the bladder/cervix (in women). The fingers from both hands continue to bluntly dissect the soft tissues around the pelvic floor until one reaches behind the rectum. The fingers (from both hands) should now be able to be in direct contact with each other, in front of the sacral bony tissues. The next manoeuvre is to pull this entire group of tissues upwards, thereby increasing the separation of the rectum/bladder from the pelvis. Performing this technique means that one should be able to mobilize the bladder, rectum, and internal genital tissues (prostate and seminal vesicles in males; uterus/cervix, tubes, and ovaries in females) without having disturbed the relationship between the tissues

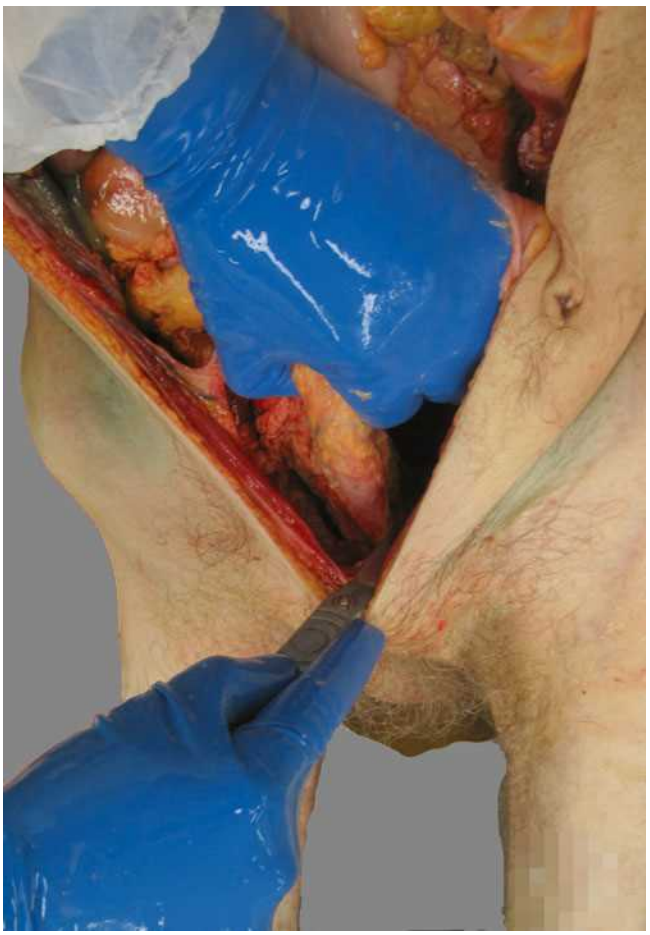

**Fig. 3.29** Although it is difficult to look into the pelvis directly, one now must run a knife behind the pubic bone, making a cut transversely across the pelvic floor tissues to free the bladder (with prostate in men) and internal genital organs (in women), and transversely across the rectum—ideally in one motion

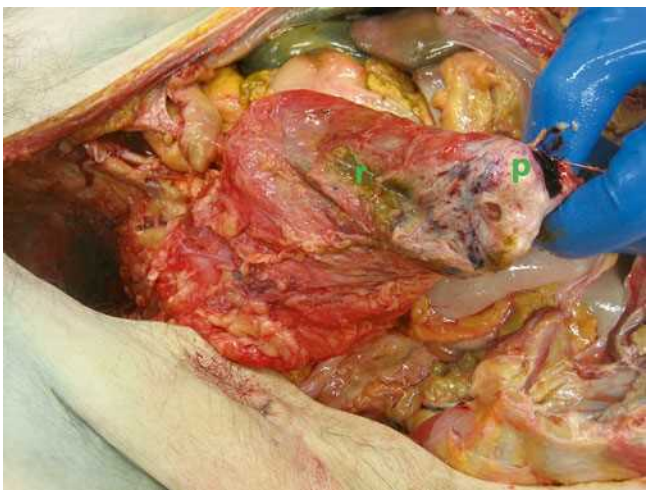


**Fig. 3.30** The anteriorly freed tissues are then pulled upwards and cranially with blunt dissection across the pelvic bony tissues towards the posterior pelvic brim. The cut end of the prostate (*p*) and the rectum (*r*) are seen in this view

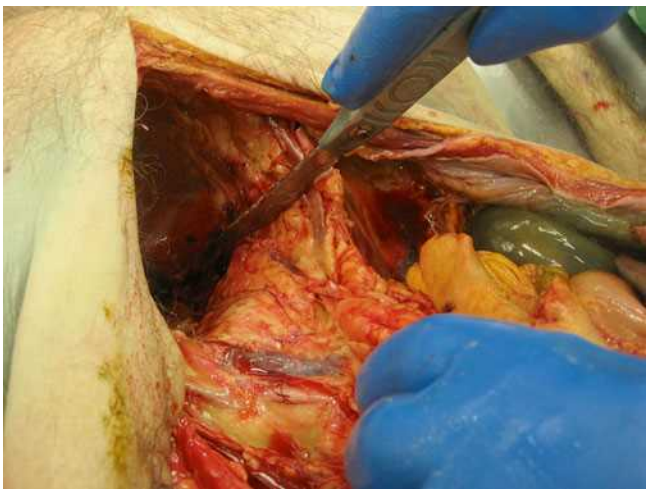

**Fig. 3.31** Having pulled the pelvic tissues upwards in a cranial direction, an oblique cut is made through the soft tissues at 45° at the pelvic brim. This cut transects the common iliac artery and vein, but leaves the medially placed ureter intact

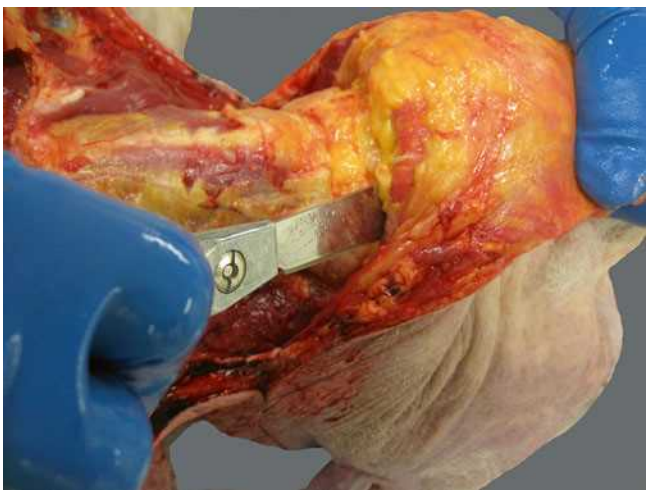

**Fig. 3.32** The mouth tissues are dissected by passing a knife around the inner aspect of the mandible, thereby freeing up the tongue and related salivary glands. Cutting the soft tissues and vasculature around this site may cause some degree of blood loss into the operating field, but this blood often can be mopped up to allow clear inspection of the next set of cuts

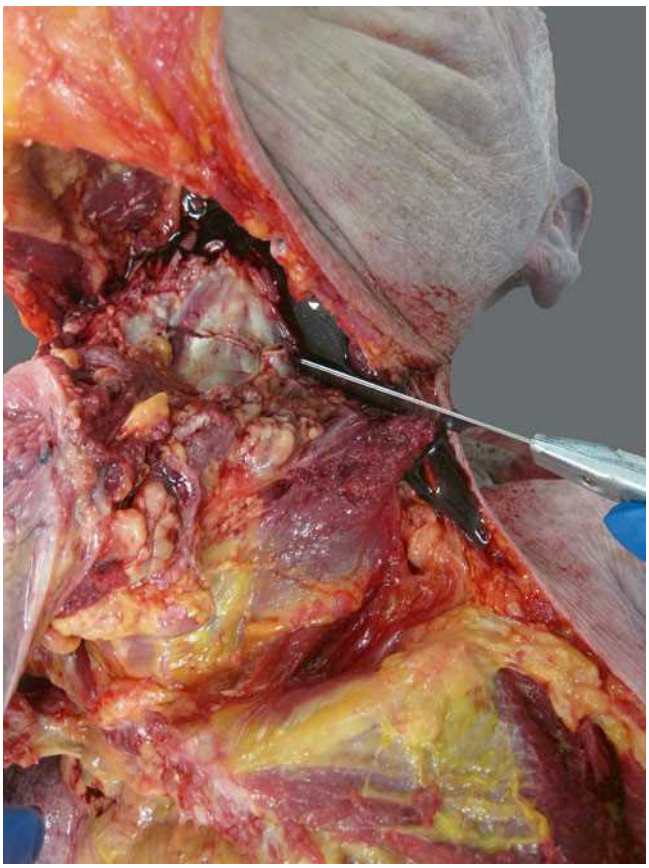

**Fig. 3.33** The next step is a transverse cut made across the back of the pharynx, which will then divide/transect the carotid vasculature above the carotid bulb. It will also cut across the internal jugular vein and around onto the soft tissues adjacent to the cervical spine


**Fig. 3.34** The mouth tissues are pulled downwards and away from the inner jaw tissues. (The mandible is marked *M*.) The epiglottis and larynx can be inspected for obstructive pathology

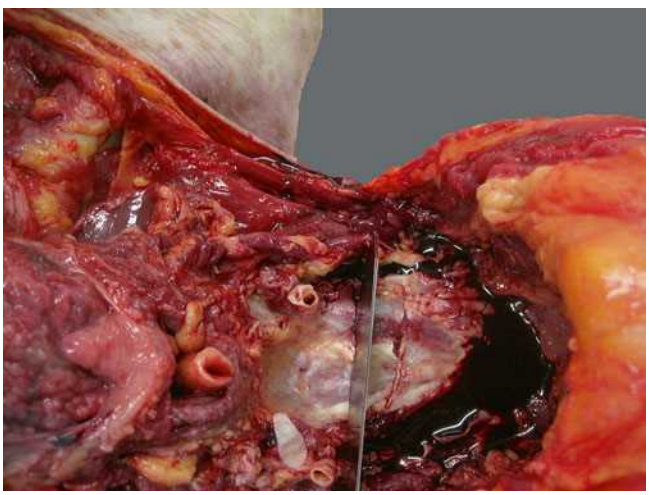

**Fig. 3.35** The cut on both sides should be made down onto the bony tissues, to guarantee preservation of these structures, which will be considered later. The process should be undertaken on both sides equally. One should look for atheromatous disease at the cut ends of the carotid vasculature

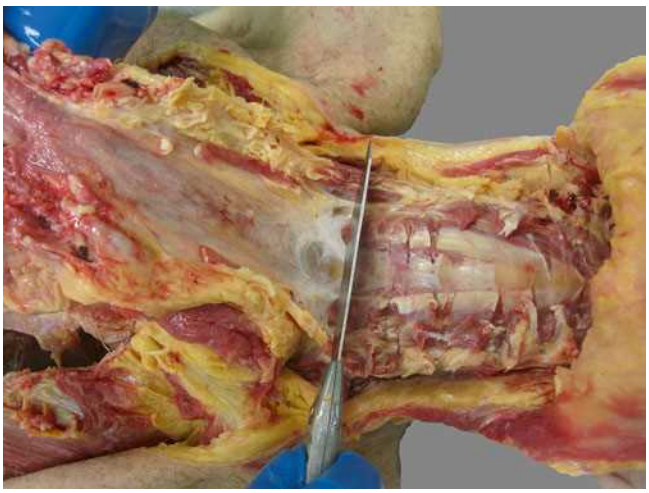

**Fig. 3.36** To free the neck tissues requires the transverse oropharyngeal cut to be extended downwards on both sides and laterally at the side of the vertebral column. Cutting directly onto the lateral aspect bony tissues of the vertebral bodies means that all relevant medially placed and important neck structures will be kept intact

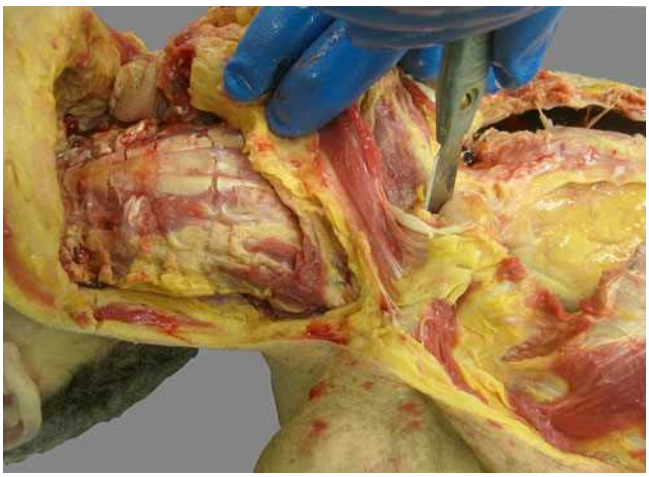

**Fig. 3.37** The soft tissues at the thoracic inlet are now cut by running the knife around the inner aspect of the first rib. All the neck structures now will be freely mobile

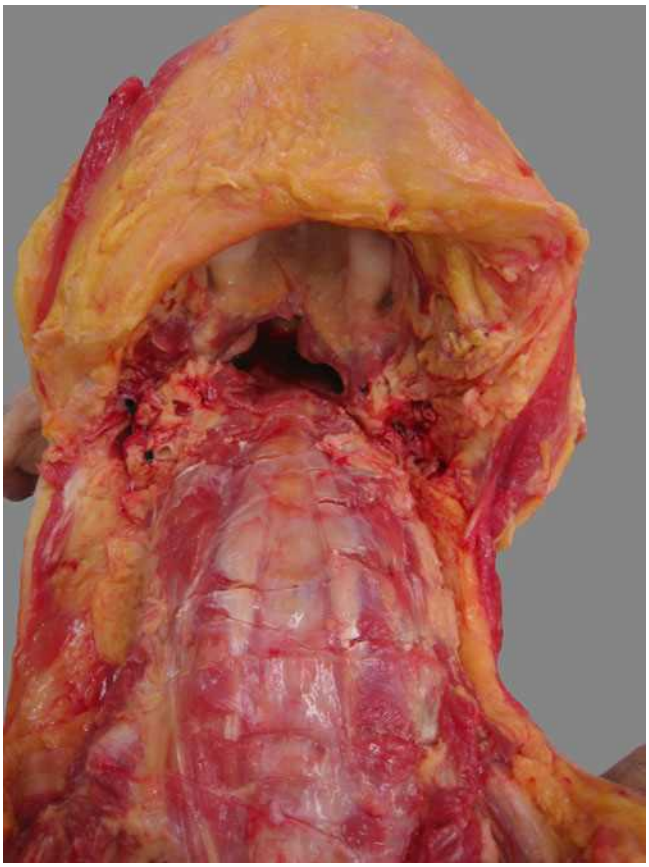

**Fig. 3.38** The neck structures have been removed and one can look upwards into the mouth, up to the hard/soft palate

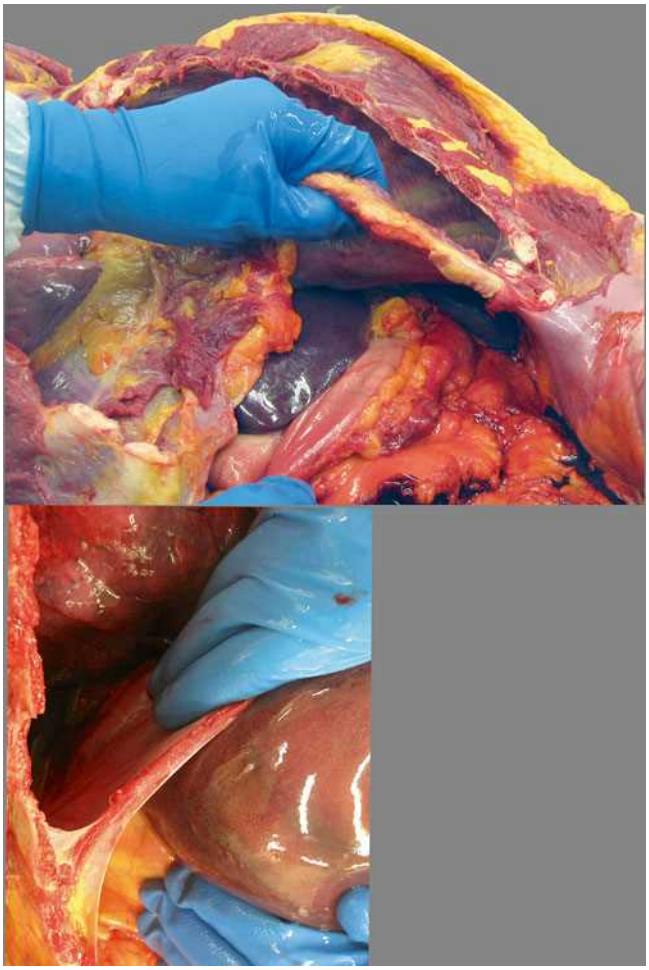

**Fig. 3.39** The diaphragm tissues are freed by running a knife around the inner aspect of the lower rib cage, and around towards the lateral aspect of the vertebral bodies on both sides. In these two images, the diaphragm is being identified and pulled taut before the cut is made. On the left side (*upper*), one protects the spleen and kidney on the left side by pulling this tissue upwards and away from the knife. The same approach applies on the right (*lower*) for the liver and kidney

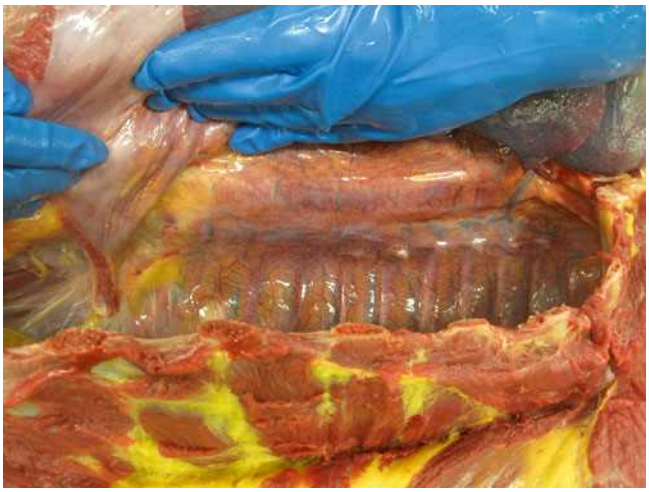

**Fig. 3.40** To avoid any tearing of inflexible thoracic content (*e.g.*, atheromatous aorta) during the next phase, one pulls the thoracic content to one side and cuts along the lateral aspect of the vertebral bodies. Note how the diaphragm has been cut around the edge and onto the vertebral column

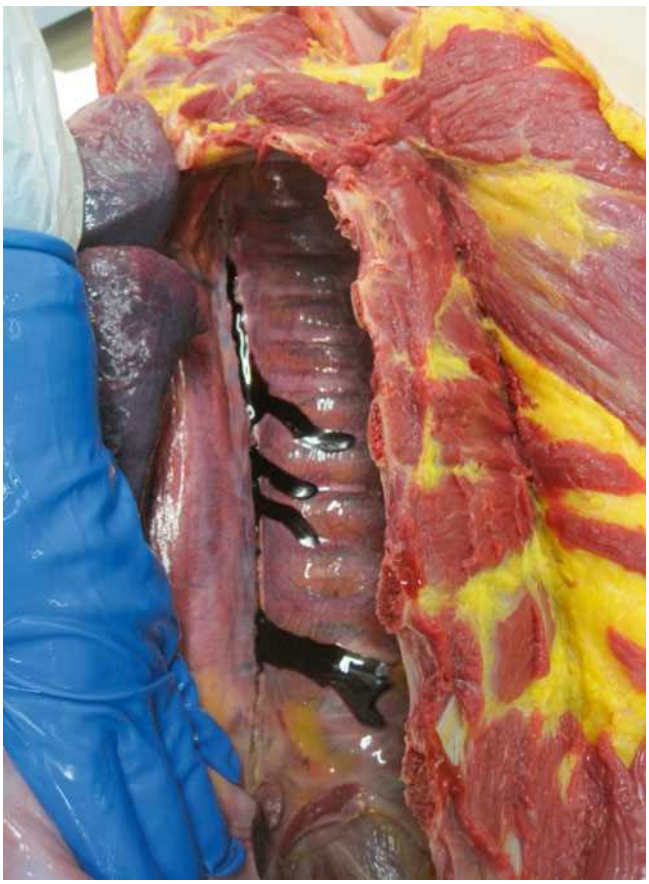

**Fig. 3.41** This image shows the long cut made onto the vertebral tissues

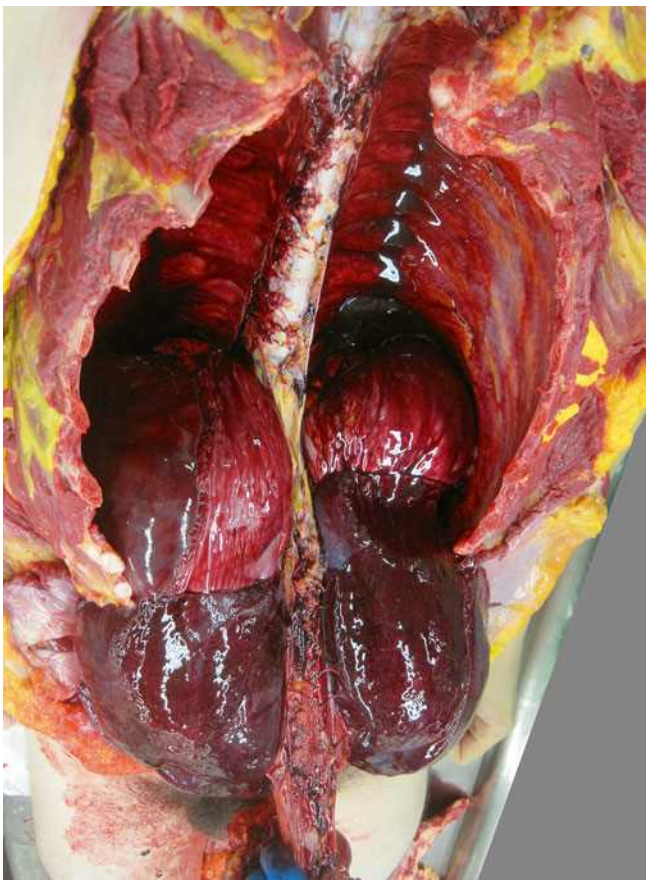

**Fig. 3.42** Grasping the neck content firmly, one now pulls the thoracic tissues downwards towards the pelvis. There should be minimal resistance, as the tissues are free at the thoracic inlet and around the vertebral column, but some additional cuts may be needed to ease the tissues out

**Fig. 3.43** The tissues can be pulled further downwards, towards the pelvis. This is often facilitated by an earlier light incision into the psoas muscle on both sides. This cut continues down towards the pelvic brim, where it will intersect with the prior dissection of the pelvic tissues and iliac vessels. Again, in some cases, some cuts around the vertebral column are required to assist the tissue extraction. Now all the thoracic, abdominal, and retroperitoneal tissues can be removed in one piece

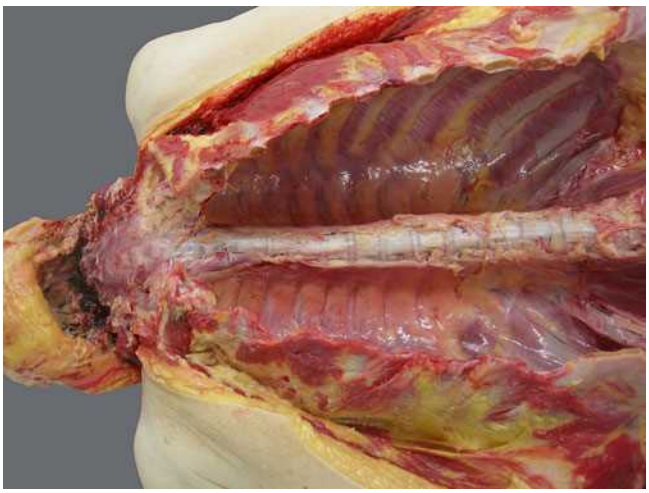

**Fig. 3.44** Once the viscera from the thorax, abdomen, and retroperitoneum have been removed, it is possible to inspect the back of the torso. This view allows one to confirm the normality of the pleura and the integrity of the vertebral column and to assess the state of the musculature and fat. The same examination should follow for the lower vertebral column and pelvis. Note how the examination is enhanced by having cleaned the inner tissues of body fluids

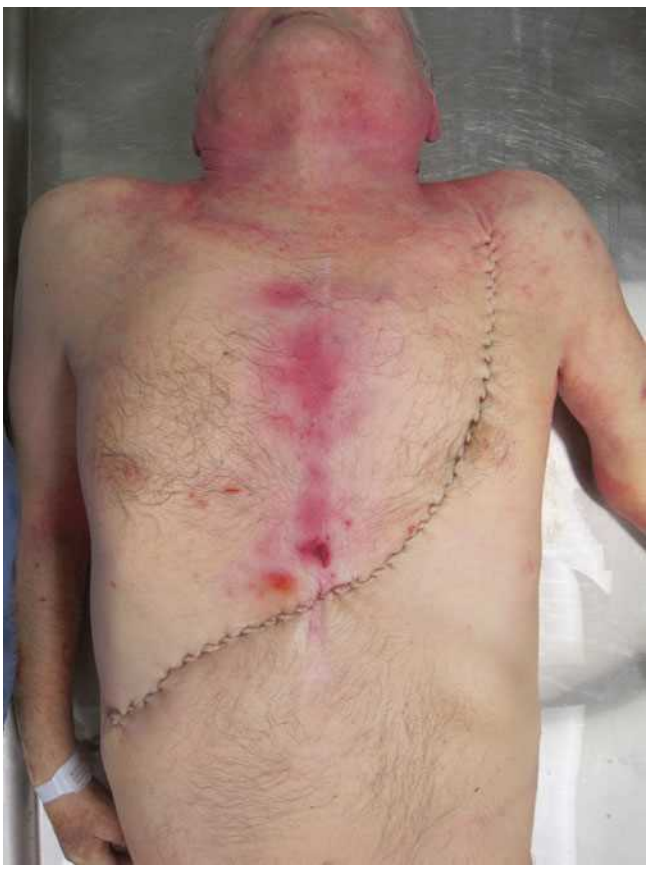

**Fig. 3.45** Limited autopsy examination is increasingly common, given the range and abilities of ante-mortem investigations. This case was chest-limited but permitted access to the abdomen. By running the incision downwards and obliquely across the chest, the incision was hidden but good access to the chest was still available to remove the heart, lungs, and mediastinal content. Opening directly through the diaphragms also allowed sampling of the liver and kidneys

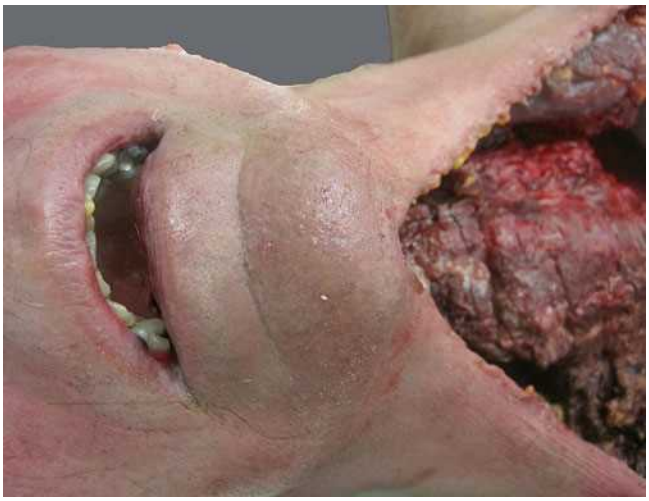

**Fig. 3.46** This second autopsy case shows a particularly poor evisceration incision that ran up to the mandibular tissues and chin in the midline. This should be avoided if possible, as it is very difficult to achieve a good cosmetic reconstruction result after the autopsy

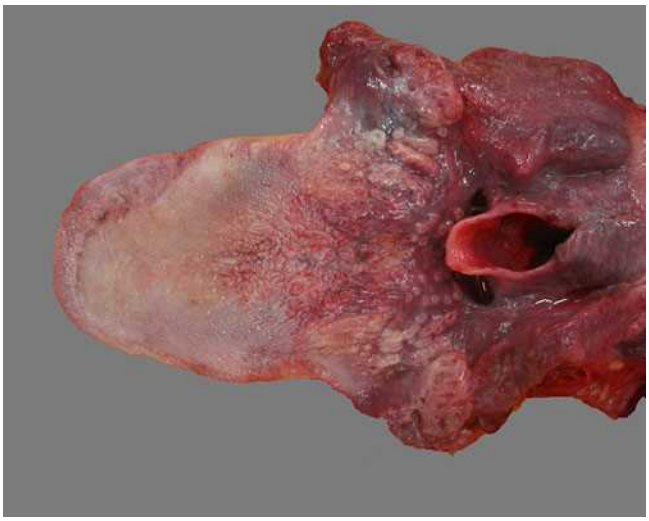

**Fig. 3.48** The cause of death may also be demonstrated early during evisceration by simple observation of the tissues. This case clearly showed a tooth pattern on the tongue, in keeping with tongue-biting. This individual was known to have epilepsy, and this evidence points toward an epilepsy-related death

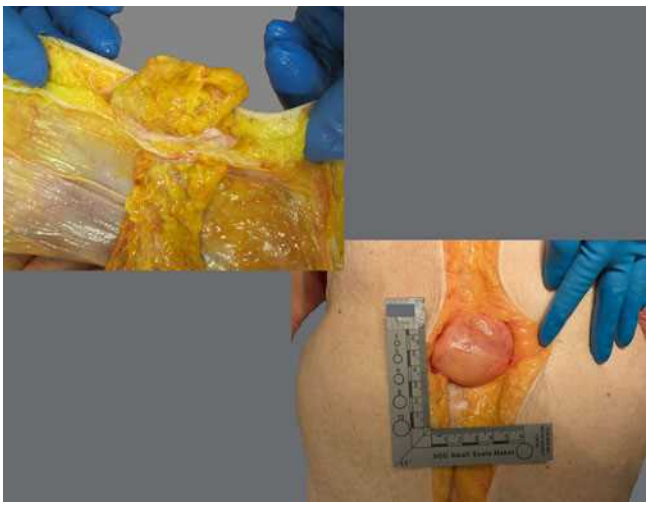

**Fig. 3.47** At the time of opening the abdomen, one may identify hernias at various points. They are common around the groin, where the abdominal wall is weak, but following surgery, soft tissues of a mesenteric nature (*upper left*) or potentially bowel (*lower right*) can protrude into defects in the abdominal wall lining in some cases despite surgical closure by sutures

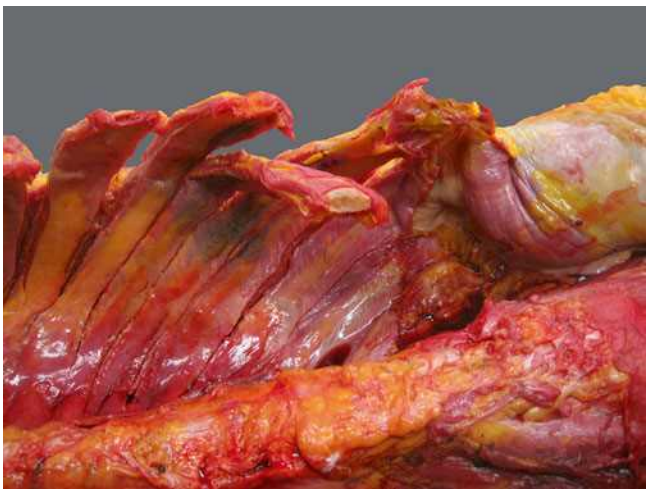

**Fig. 3.49** During the evisceration, fractures of the ribs may become obvious. There is bruising locally, in keeping with these fractures being ante-mortem. They are best demonstrated by incision through the intercostal muscles

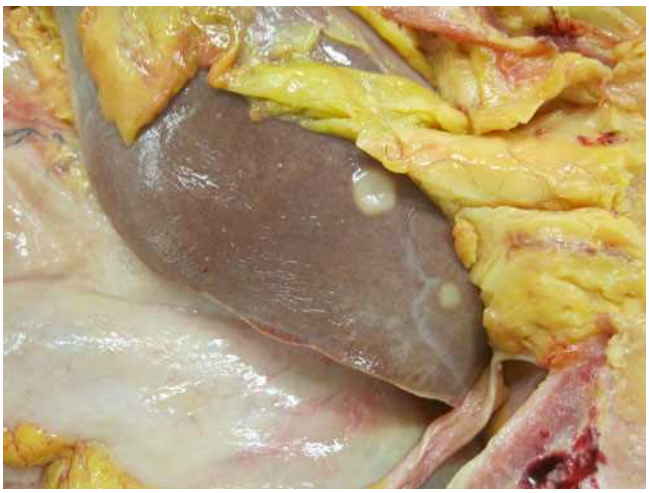

**Fig. 3.50** During evisceration, the cause of death may become apparent. Here, the liver appears slightly enlarged and has several foci of cream tumor on the surface. This points towards carcinomatosis, subsequently determined to be disseminated lung cancer

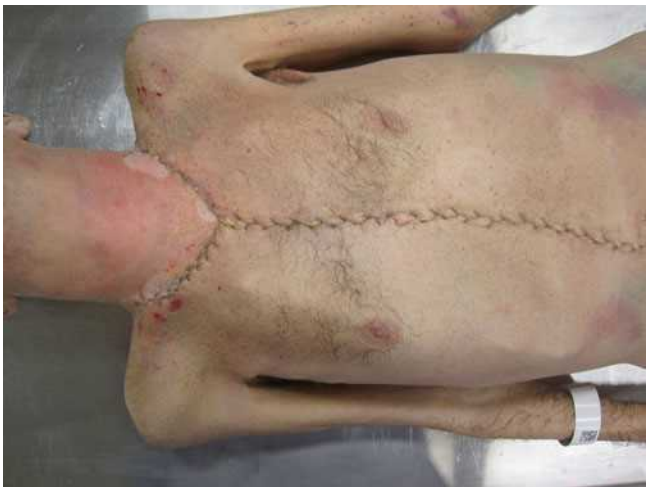

**Fig. 3.51** Following evisceration, tissue examination, and the taking of samples, the body is reconstituted with all the tissue contents placed into bags within the thorax/abdomen cavity. This includes the central nervous system, as leakage from cranial reconstitution would not be acceptable. The tissues can be split into bowel and other tissues to limit the rate of tissue degradation if a second autopsy is likely. The evisceration incisions should be closed by twine in a secure manner so that skin integrity is complete. Thereafter, the body should be washed and dried. It is then returned to the refridgerator

## Suggested Reading

Burton JL, Rutty G. The hospital autopsy. 3rd ed. London: Hodder Arnold; 2010. p. 115–35.

Knight B. The post-mortem technician's handbook. Oxford: Blackwell Scientific Publications; 1984.

Ludwig J. Autopsy practice. 3rd ed. Totowa: Humana Press; 2002.

Royal Institute of Public Health and Hygiene. A handbook of mortuary practice and safety: for anatomical pathology technicians. London: Royal Institute of Public Health and Hygiene; 1991.

# Thorax: Heart, Lungs, Mediastinum, and Pleura

4

S. Kim Suvarna

## General Principles of Examining the Thorax

At the start, it is important to emphasize that a thorough knowledge of normal anatomy, physiology, and general pathology is required if one is to understand any thoracic autopsy findings. This background allows the examination to progress with understanding of any pathology, mapped against the patient's clinical data and history.

To fully appreciate cardiac and lung disease, it is important to separate the tissues carefully and sequentially, looking for disease as one proceeds. It is fair to say that the heart and lungs can best be regarded as a single unit, in that the heart has two halves with the lungs operating between the right and left side cardiac chambers.

The first part of this chapter (Figs. 4.1, 4.2, 4.3, 4.4, 4.5, 4.6, 4.7, 4.8, 4.9, 4.10, 4.11, 4.12, 4.13, 4.14, 4.15, 4.16, 4.17, 4.18) allows one to inspect the thoracic parenchymal units and to then start to break them down into smaller pieces. In this way, certain disorders (pulmonary embolism, congenital variations, and upper respiratory infections) can be spotted early in the case examination. Thereafter, the chapter looks specifically at the lungs and heart tissues.

Photography is paramount in thoracic cases, and ideally should be used during the entirety of the dissection process, recording different aspects of the heart tissues. Histological sampling is also important, alongside microbiology assessment.

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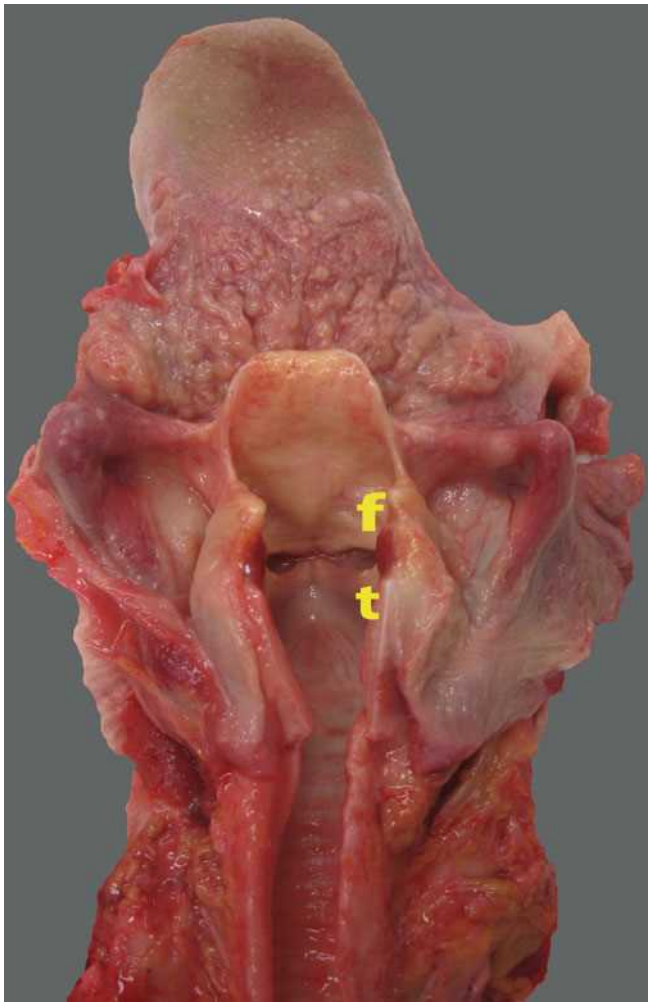


**Fig. 4.1** Examination of the thoracic block of tissues should ideally start with the upper airway tissues. Placing tissues in a posterior-anterior position (*i.e.*, inspecting from the back), one can see the posterior wall of the trachea and laryngeal tissues. The epiglottis is noted proximally, and the tongue also may be inspected at this point. The airway should be checked for oedema, secretions, foreign matter or food debris, and tumours. The laryngeal cartilages are opened, using scissors, along the midline and the vocal cords (*t* true, *f* false) are seen in their correct position. The proximal tracheal tissues are now available for inspection

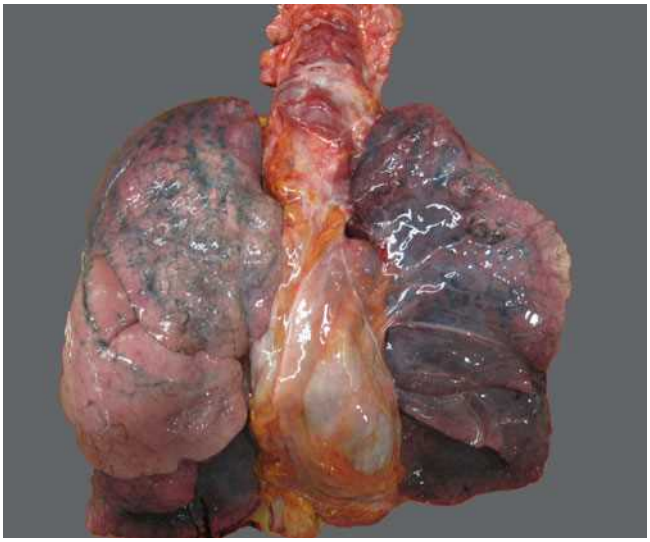

**Fig. 4.2** The thoracic contents are seen from the front, having been removed from the body. The heart, within the pericardial sac, is centrally placed, with the lungs either side. The general architecture should be checked, with attention to congenital variations and external pathology

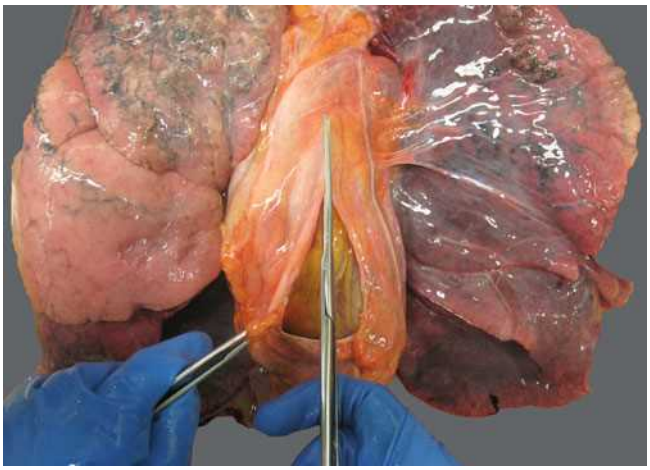

**Fig. 4.3** The pericardium is opened, making sure that any fluid content is assessed (volume/type). If there is any fluid, then samples may be taken for microbiology and cytology

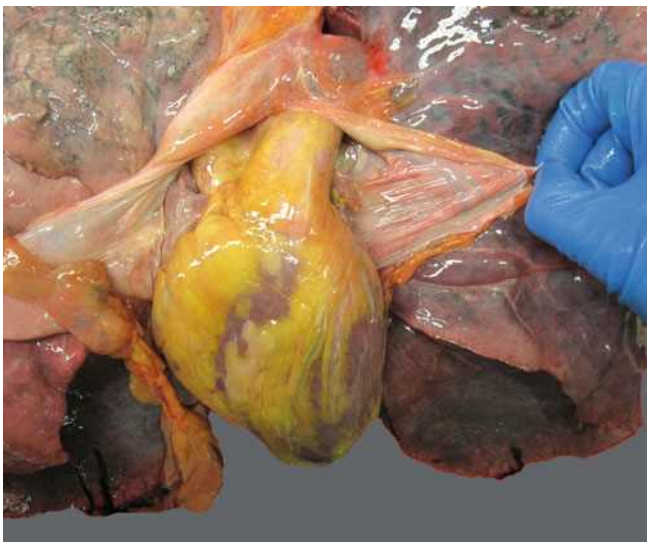

**Fig. 4.4** The internal aspect of the pericardium should be inspected for possible tumour foci and fibrinous deposits. This inspection also allows a view of the heart, again allowing one to assess isomerism and other congenital cardiac variants

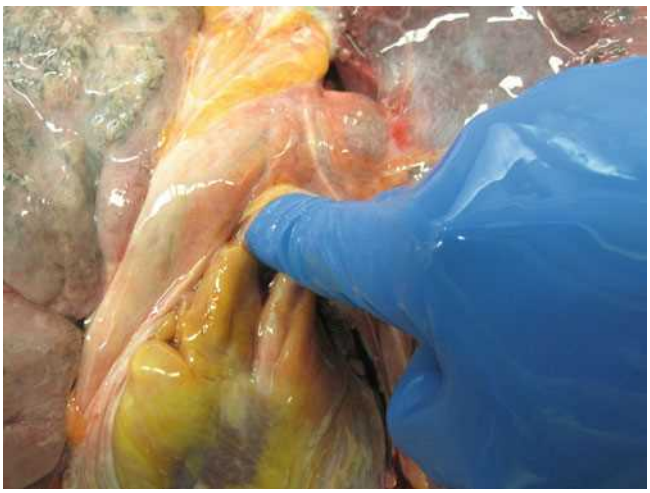

**Fig. 4.6** A single finger may be introduced into the pulmonary artery to palpate into the pulmonary artery branches on either side. Pulmonary emboli often can be felt before being visualized in this technique, and they may be pushed slightly onto the artery to avoid their loss when the lungs are separated from the mediastinal content

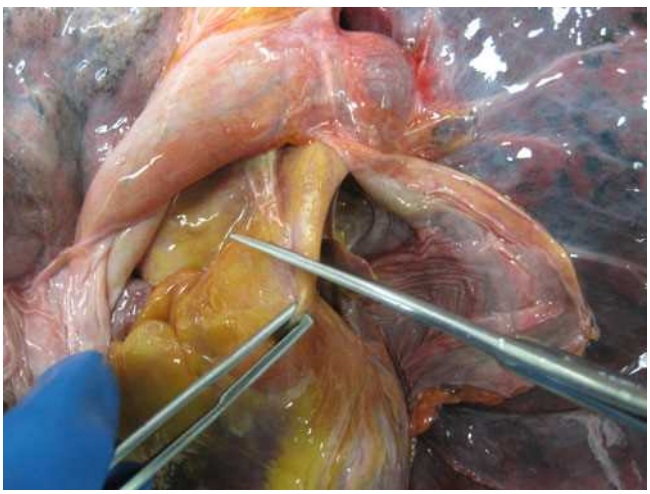

**Fig. 4.5** The pulmonary artery is put under slight tension and the vessel is opened anteriorly using scissors, to permit examination of the vessel for large pulmonary emboli

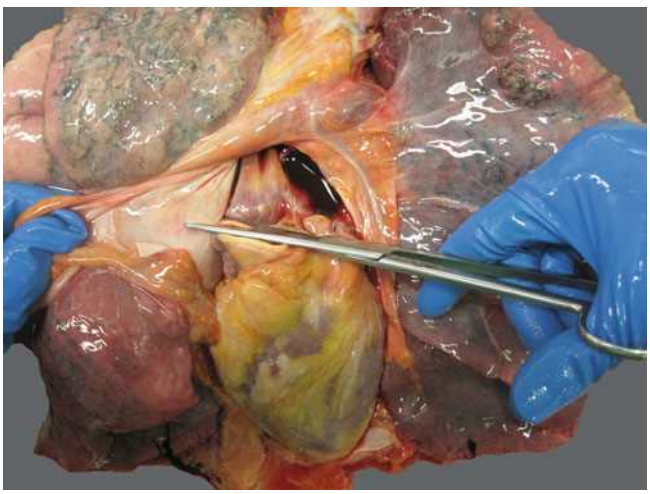

**Fig. 4.7** After the examination of the pulmonary artery, the heart may be removed from the block of tissue by completing the cut across the pulmonary artery trunk and aorta in sequence

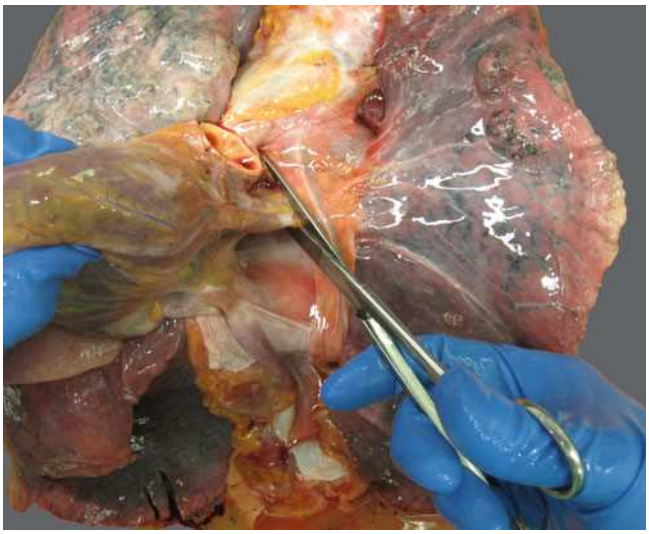

**Fig. 4.8** Next, the heart is lifted upwards and towards the right side. The dissection proceeds with cutting across the pulmonary veins. These are transected, ideally with scissors, above the left atrial roof and posterior wall

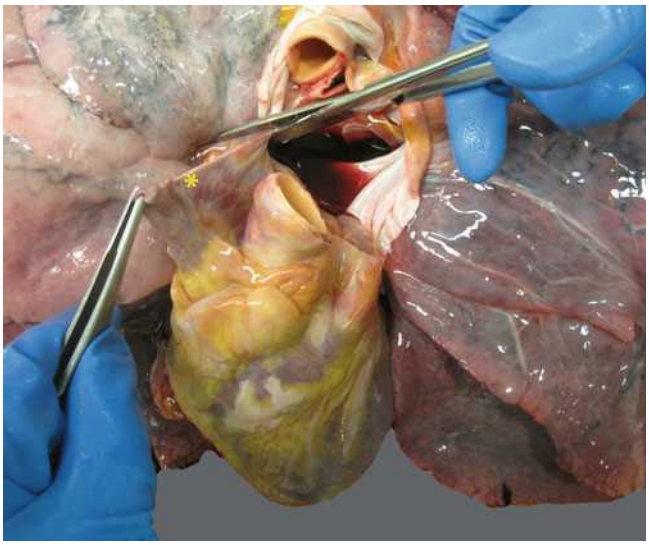

**Fig. 4.10** The superior vena cava (SVC) must be cut above the sinoatrial node (SAN). The SAN is at the interface of the SVC and the right atrium anteriorly (*asterisk*). By gently putting the SVC under tension, one can ensure that the cut across the SVC is at least 10 mm above the SAN

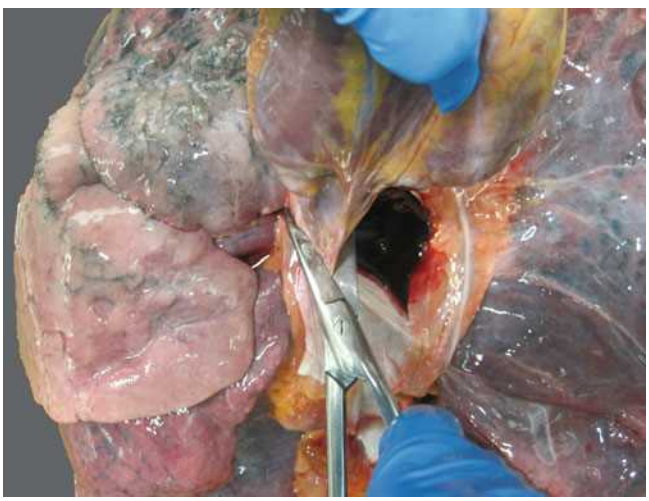

**Fig. 4.9** Lifting the heart upwards (towards the head) allows the inferior vena cava to be cut where it enters the right atrium

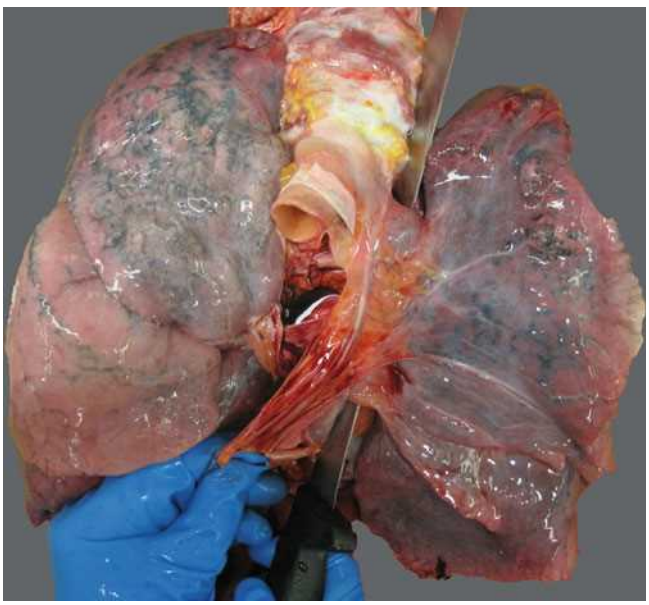

**Fig. 4.11** The next step is to separate the lungs. In this case, the left lung is resected first, by placing a long knife under the left lung hilum. It is safest to put the knife in position with the blunt side upmost and then rotate the blade so the cutting edge is upward when the knife is in position. The lung should be removed with one slice, rather than a sawing motion

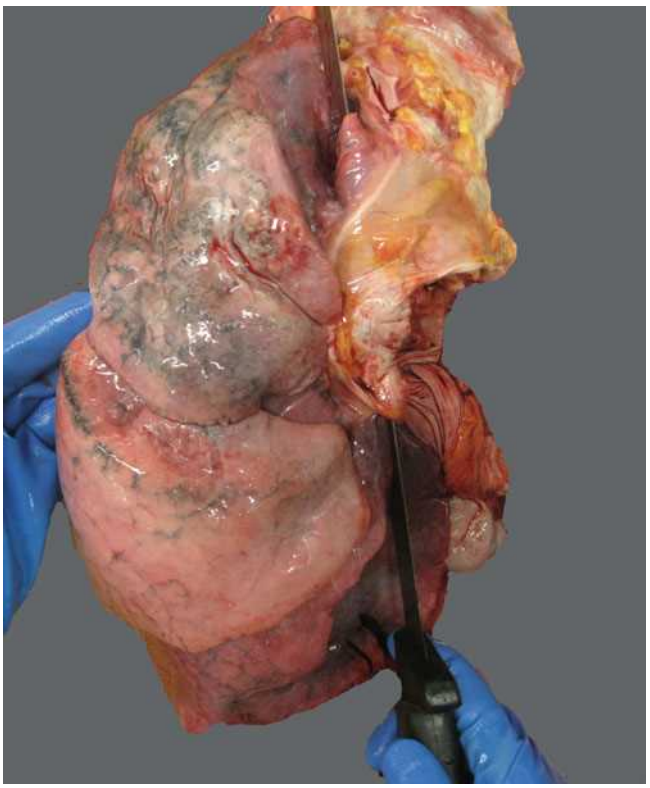

**Fig. 4.12** The right lung is now removed by repeating the process at the right lung hilum

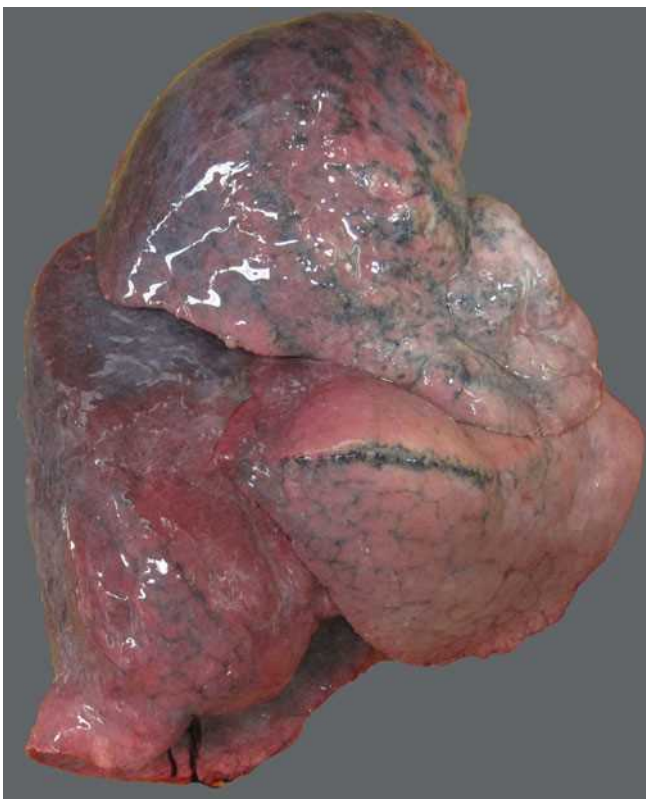

**Fig. 4.13** The right lung is seen from the lateral aspect, with an unremarkable pleural surface. Note the three lobes

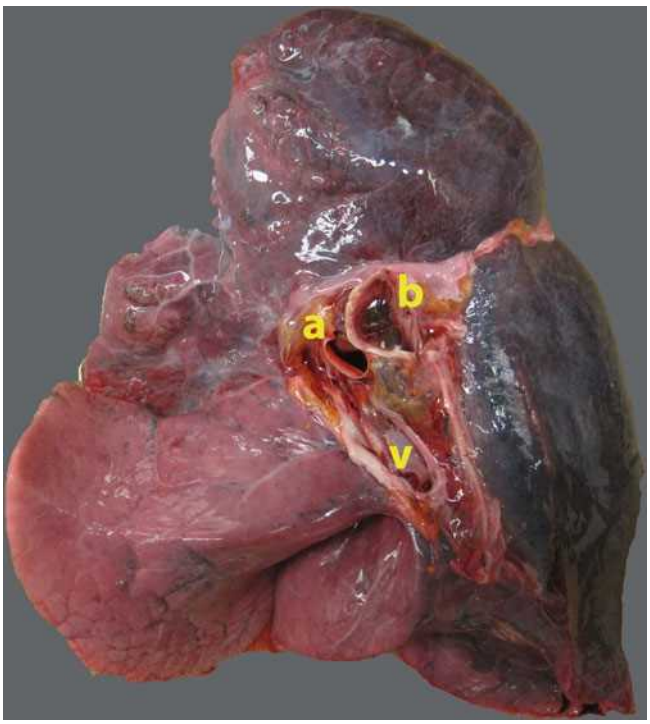


**Fig. 4.14** The mediastinal view of the right lung shows the normal bronchus (*b*) and vasculature (*a* artery, *v* vein)

**Fig. 4.15** The external surface of the left lung lateral aspect is seen; the two lobes show a normal pleural surface

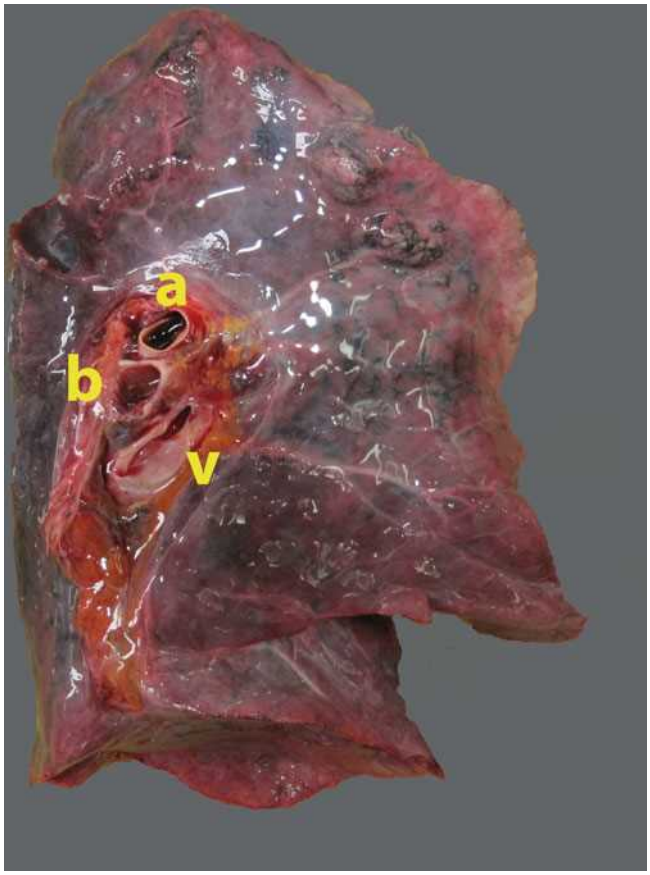


**Fig. 4.16** The mediastinal view shows the normal bronchus (*b*) and vasculature (*a* artery, *v* vein)

**Fig. 4.17** The mediastinal view of the left lung shows part of the pericardium attached, highlighting the proximity of the lung to the heart tissues

**Fig. 4.18** Modern urban life rarely leaves individuals in a low-pollution environment. Consequently, many lungs show some anthracotic (*black*) dust accumulation, generally reflecting pollution from fossil fuels and general dusts

## The Lung Tissues: Acute and Infective Processes

Examination of the lung tissues (Figs. 4.19, 4.20, 4.21, 4.22, 4.23, 4.24, 4.25, 4.26, 4.27, 4.28, 4.29, 4.30, 4.31, 4.32, 4.33, 4.34, 4.35, 4.36, 4.37, 4.38, 4.39, 4.40, 4.41, 4.42, 4.43, 4.44, 4.45, 4.46, 4.47, 4.48, 4.49, 4.50, 4.51, 4.52, 4.53, 4.54, 4.55, 4.56, 4.57, 4.58, 4.59, 4.60, 4.61, 4.62, 4.63, 4.64, 4.65, 4.66, 4.67, 4.68, 4.69, 4.70, 4.71, 4.72, 4.73, 4.74, 4.75, 4.76, 4.77, 4.78, 4.79, 4.80, 4.81, 4.82, 4.83, 4.84, 4.85, 4.86, 4.87, 4.88, 4.89, 4.90, 4.91, 4.92, 4.93, 4.94, 4.95, 4.96, 4.97, 4.98, 4.99, 4.100, 4.101, 4.102, 4.103, 4.104, 4.105, 4.106, 4.107, 4.108, 4.109, 4.110, 4.111, 4.112, 4.113, 4.114, 4.115, 4.116, 4.117, 4.118, 4.119, 4.120, 4.121, 4.122, 4.123, 4.124, 4.125, 4.126, 4.127, 4.128, 4.129, 4.130, 4.131, 4.132, 4.133, 4.134, 4.135, 4.136, 4.137, 4.138) is primarily concerned with initially confirming normality and then by seeking out any pathology issues. The lung tissues must be considered carefully, with attention to the vasculature as well as the airways. Consideration of the pleural membranes and chest wall may also be necessary if one is to fully appreciate pulmonary function.

Common culprits in terms of pathology include embolic disorders, with large emboli being common lesions in the autopsy room. If large, they are almost instantly fatal.

Lung infections (standard community-acquired pneumonic conditions as well as atypical bacterial, fungal, and viral diseases) are common lesions in the autopsy room. With any infective process, samples should be taken for microbiological assessment and histology.

The placement of chest drains, septic and haemorrhagic complications from surgical intervention, and the impact of fractures are important aspects of assessing the thoracic tissues. The role of photography is highlighted.

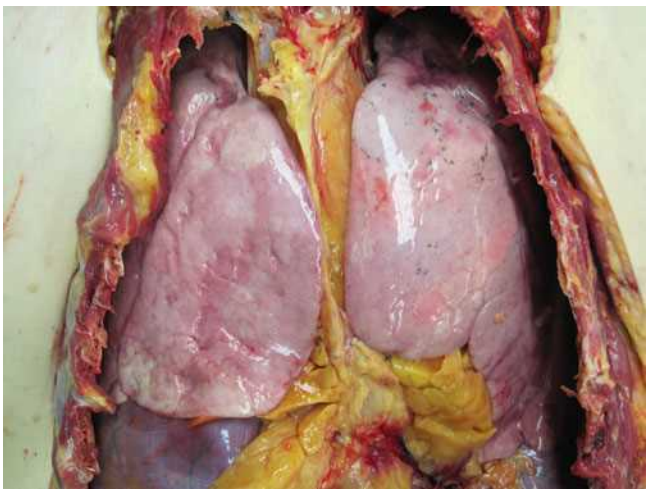

**Fig. 4.19** These lungs are relatively clear of dust; they are from an individual who lived in a very rural setting without significant soot staining

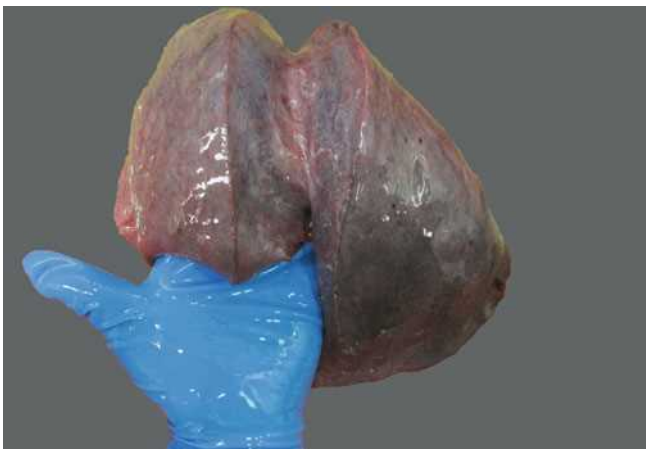

**Fig. 4.20** The internal aspect of the lung tissues is commenced with the vasculature. The examination of the pulmonary vasculature is best appreciated by exposing the interlobar fissure, best accomplished by holding the lung at the hilum and letting the lobes fall on either side of the hand

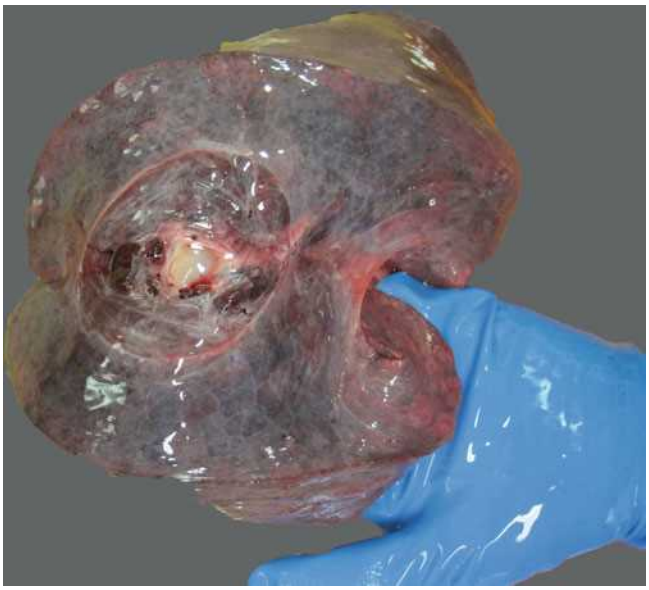

**Fig. 4.21** An incision is made into the interlobar fissure over the pulmonary artery, opening the bifurcation point of the pulmonary artery. The lung tissues fall either side of the cut, if one holds the lung centrally from below, at the hilum

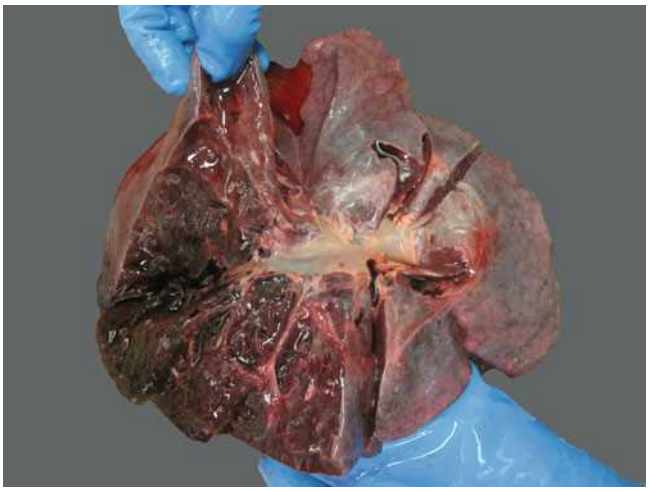

**Fig. 4.23** The arteries should be explored outwards towards the periphery of the lung tissue, not simply 10–20 mm from the centre of the lung

**Fig. 4.22** By gently inserting scissors into the vessels and only then cutting, the pulmonary arteries can be carefully exposed along the lengths of the vessels towards the lung edge. One should consider the extent of any atheromatous change and the presence of thromboemboli or other focal lesions

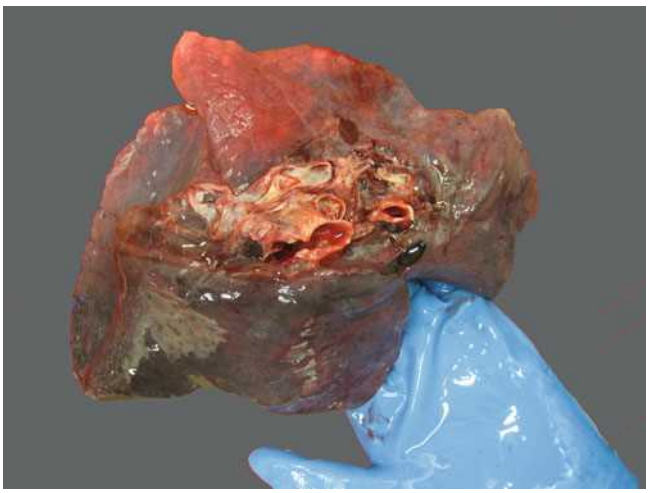

**Fig. 4.24** The lung tissues are now turned over to show the mediastinal aspect uppermost. The bronchi are inspected initially, looking for obstructing tumours or foreign material

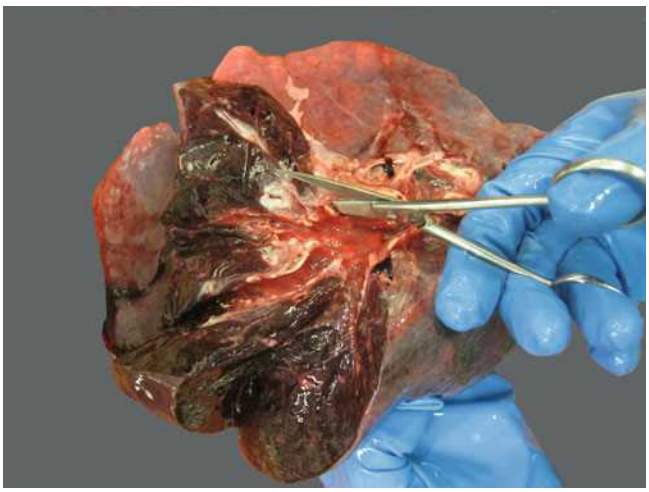

**Fig. 4.25** In a similar manner, the next step involves the scissors being gently inserted into the bronchi, with cuts being made onwards and outwards towards the periphery of the lung tissue

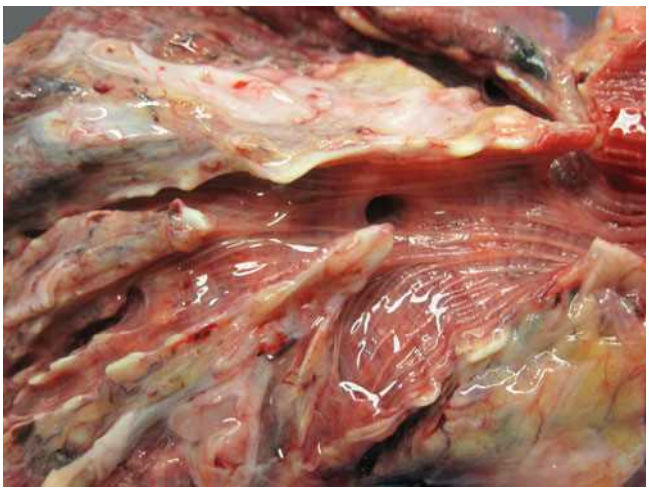

**Fig. 4.27** The airways (in health) generally are seen to have a ridged or corrugated surface quality, which assists drainage of secretions

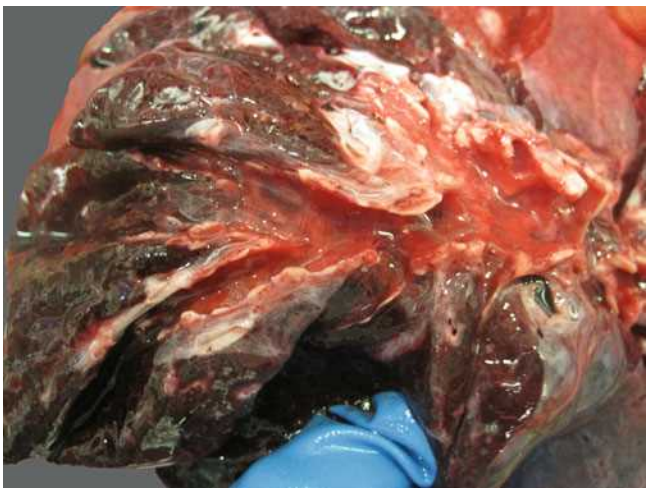

**Fig. 4.26** The surfaces on the bronchial tree should be inspected with regard to sepsis, tumour, or obstructive lesion. The airways shown are clear of tumour and inflammatory exudate, but there is a degree of bronchial congestion, raising the possibility of some bronchitis

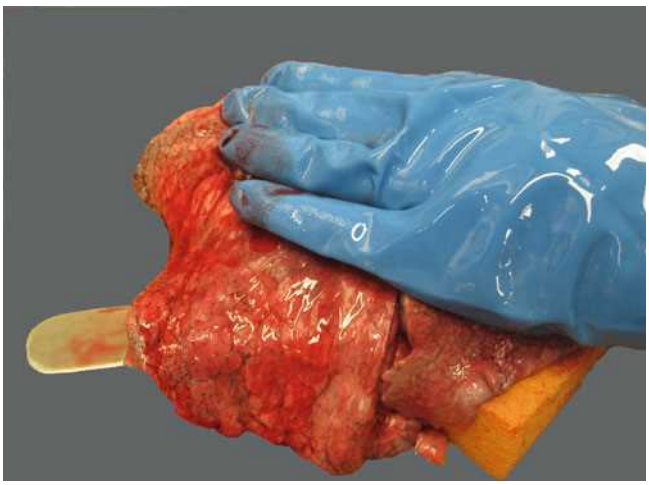

**Fig. 4.28** One can slice lung along its long (parasagittal) axis in order to display the tissues. This method is quicker, but it clearly cannot fully assess the bronchi and vasculature. It is particularly useful, however, when considering interstitial and diffuse lung disease

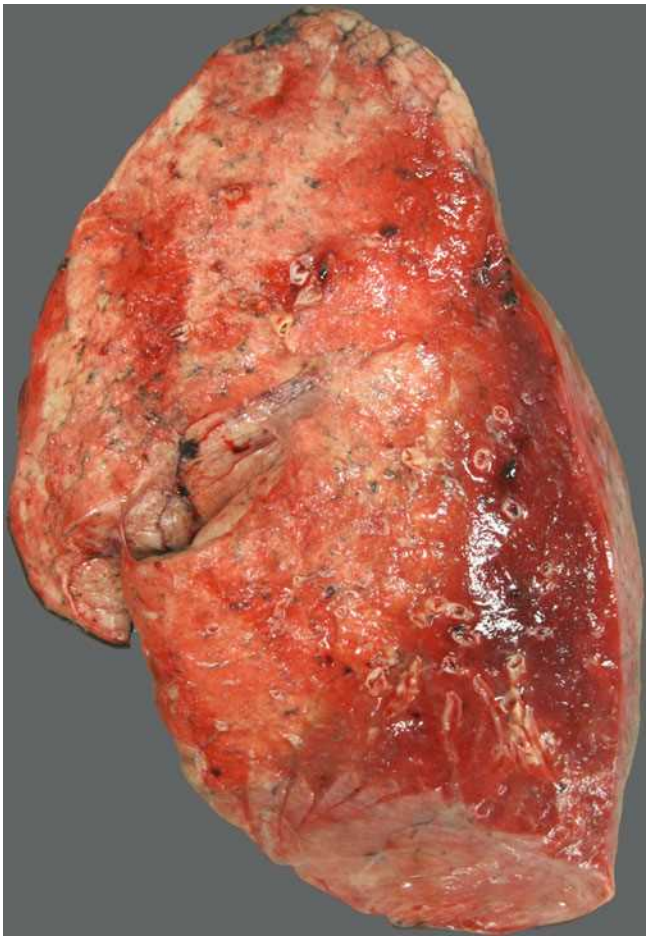

**Fig. 4.29** The cut surface of the lung seen in parasagittal section. The lung tissues appear largely normal, but hypostasis affects the lungs in the same way as it does the rest of the body. Blood has accumulated in the posterior and dependent part of the lung following death. This technique, apart from being quicker, does allow a more global view of the parenchyma

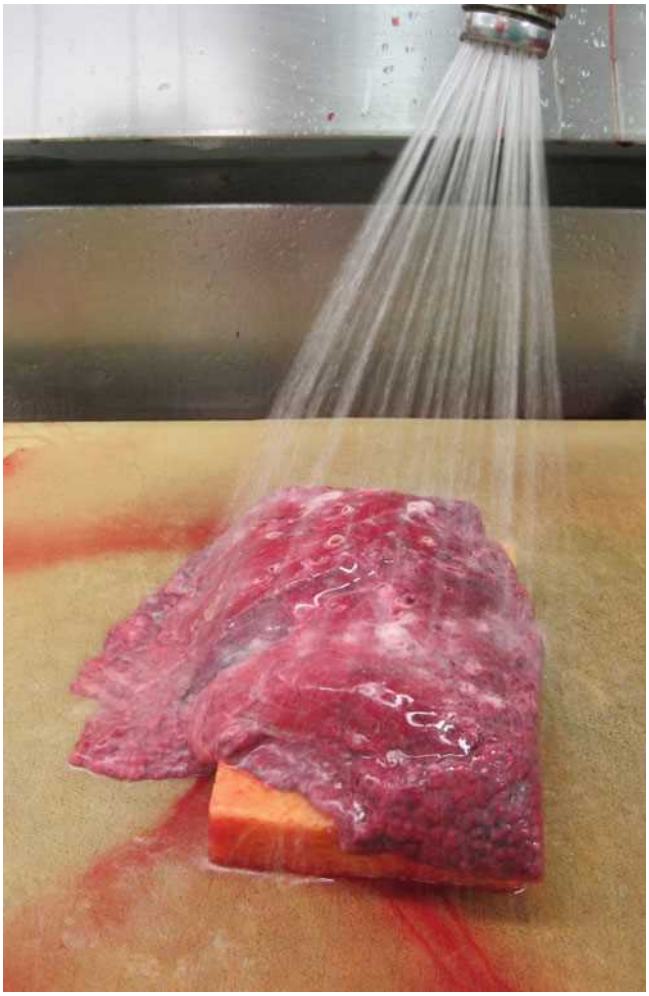

**Fig. 4.30** Lung tissues contain a lot of blood and often are rich in secretions, so lung detail may be obscured macroscopically. One solution is to gently spray water onto the cut surface for about 3–5 min

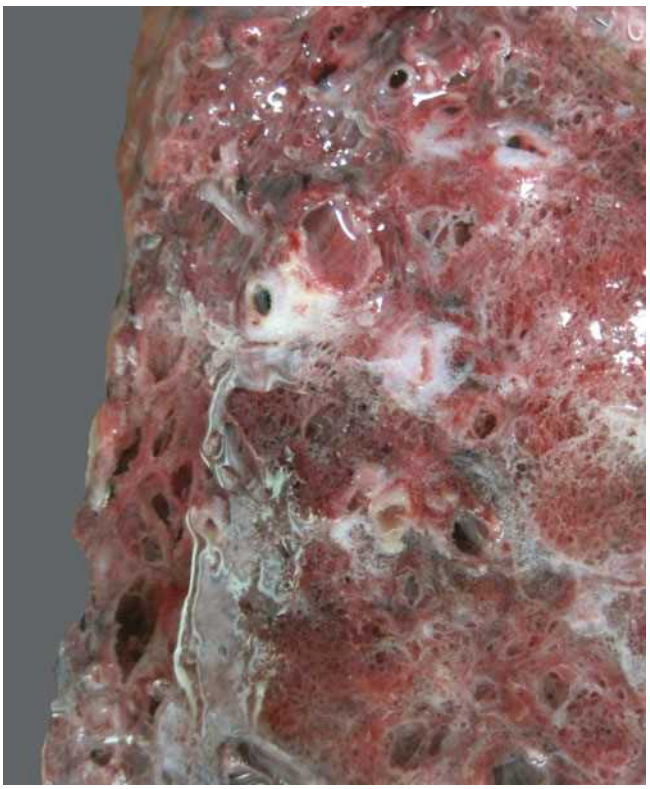

**Fig. 4.31** After gentle washing, more detail is apparent in the washed lung

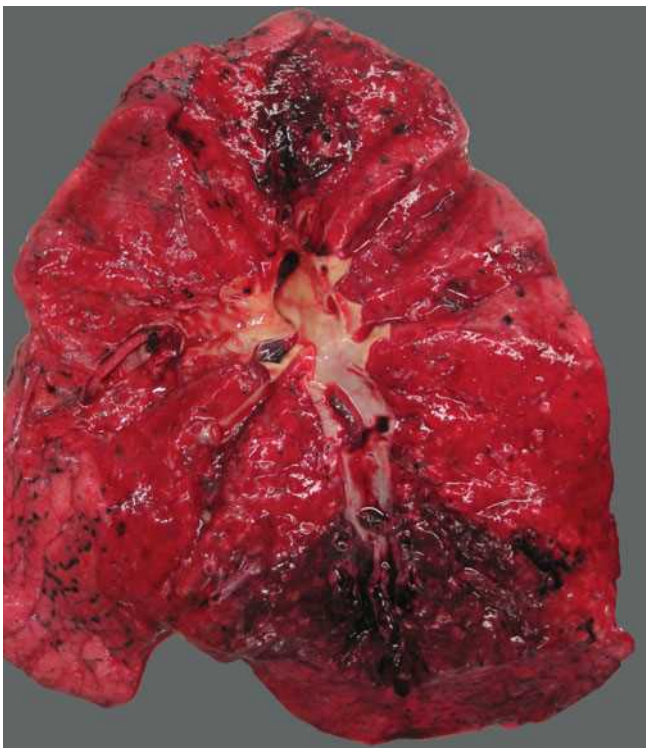

**Fig. 4.33** The lung tissues have been opened from the blood-vessel aspect. There are two zones of haemorrhagic infarction towards the top and bottom of the lung fields. Some adherent thrombus is also seen attached to one of the segmental pulmonary arteries

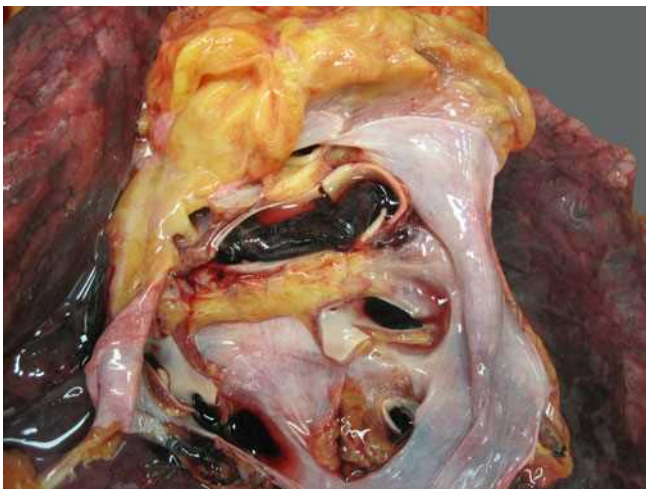

**Fig. 4.32** Shown is a saddle embolus, clearly occluding the branch of the pulmonary artery and causing sudden death. This is evident before the lungs are removed, but after the heart has been separated from the mediastinal content

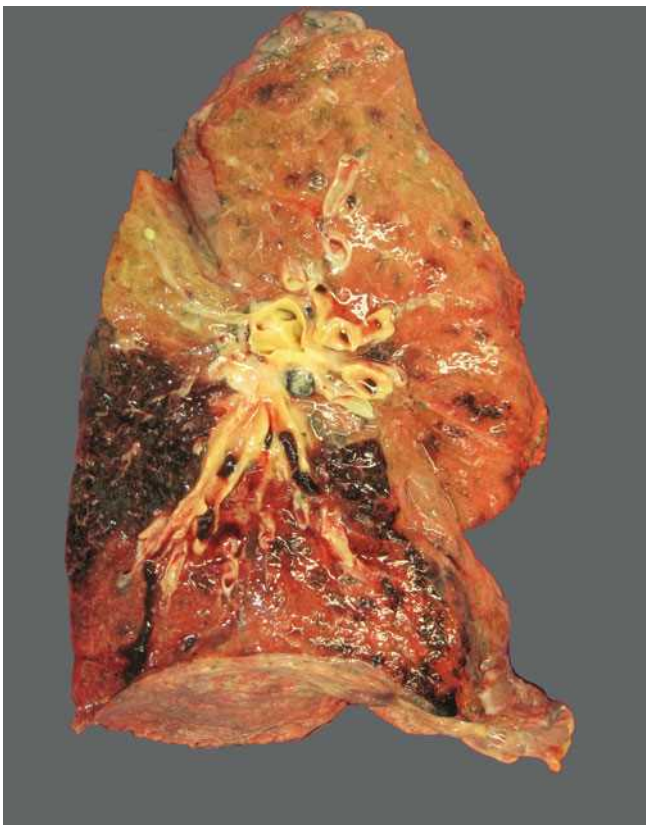

**Fig. 4.34** The classic wedge-shaped pulmonary infarct is seen after a few days in a case of pulmonary embolism. Note the partially adherent thrombotic material in the proximal artery. The local viable lung showed consolidation and septic changes histologically

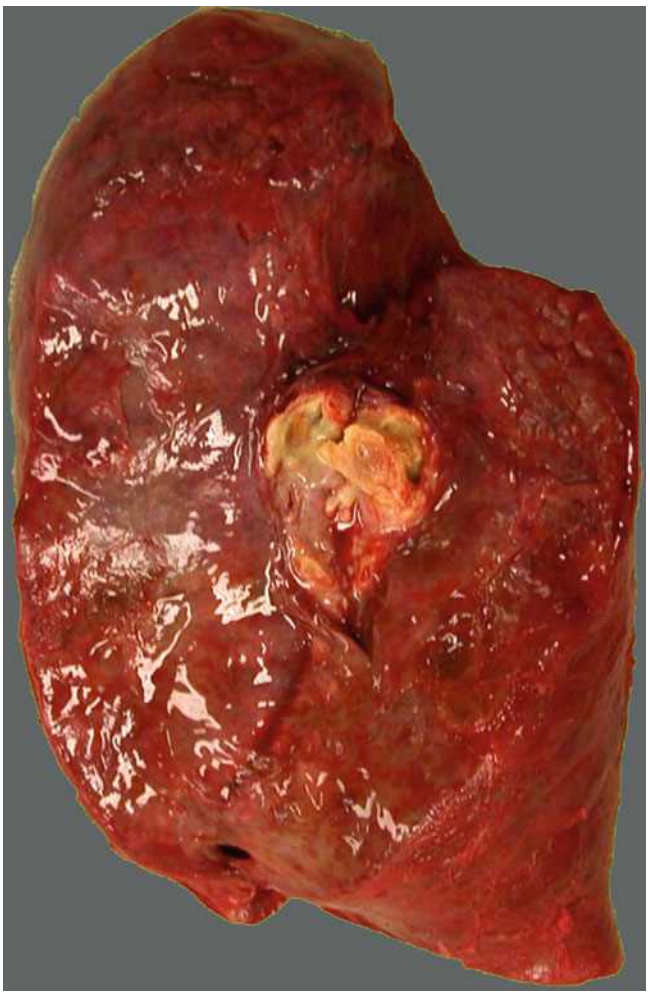

**Fig. 4.35** Thrombotic material can be complicated by septic findings. Bacterial infection can localize within dead tissue or thrombus at the root of the lung

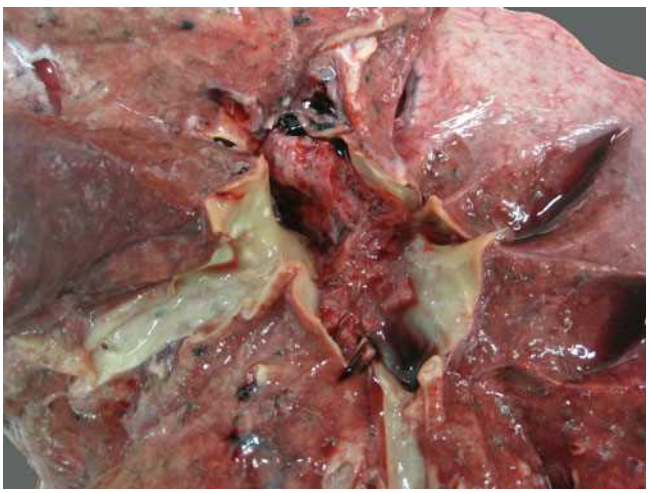

**Fig. 4.36** Thromboembolic matter may reflect longstanding thrombotic events within the venous circulation. This very firm and pale thrombus was found in a patient who collapsed suddenly and who had a chronic history of recurrent deep vein thrombotic change. This thrombus had likely formed in the large veins many weeks beforehand

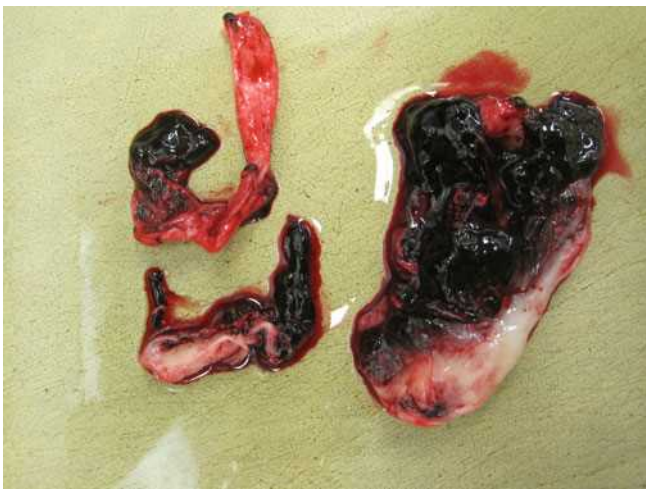

**Fig. 4.37** Post-mortem coagulum, in the form of semisolid protein and blood cells, is often seen at autopsy. The coagulum is layered (owing to post-mortem settling out of erythrocytes and other cells) and generally quite soft; it should not be confused with thrombus

**Fig. 4.38** Thromboembolic matter is seen with the venous radicle configuration, which allows one to map the thrombi (by virtue of their size) to the likely source within the venous circulation

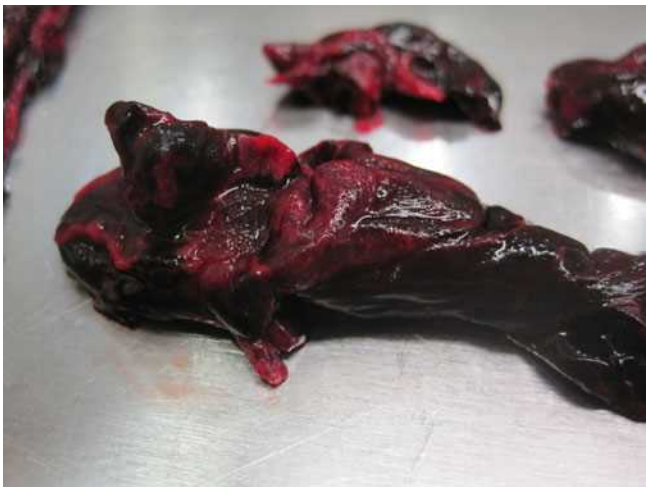

**Fig. 4.39** Close up of venous thromboembolic material shows irregularities in keeping with valve projections in the venous circulation at the sites where the thrombus is formed

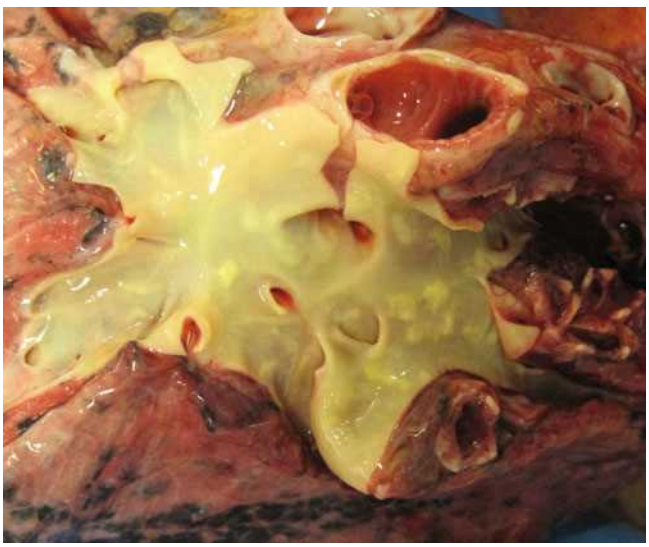

**Fig. 4.40** The pulmonary artery in this case shows some atheroma deposits. If these are present through into the deeper segmental arteries then the atheroma may indicate pulmonary hypertension. However, a few streaks of atheroma solely at the root of the pulmonary artery do not prove pulmonary hypertension. Additional evidence of pulmonary hypertension will require histological examination of the alveolated lung vasculature

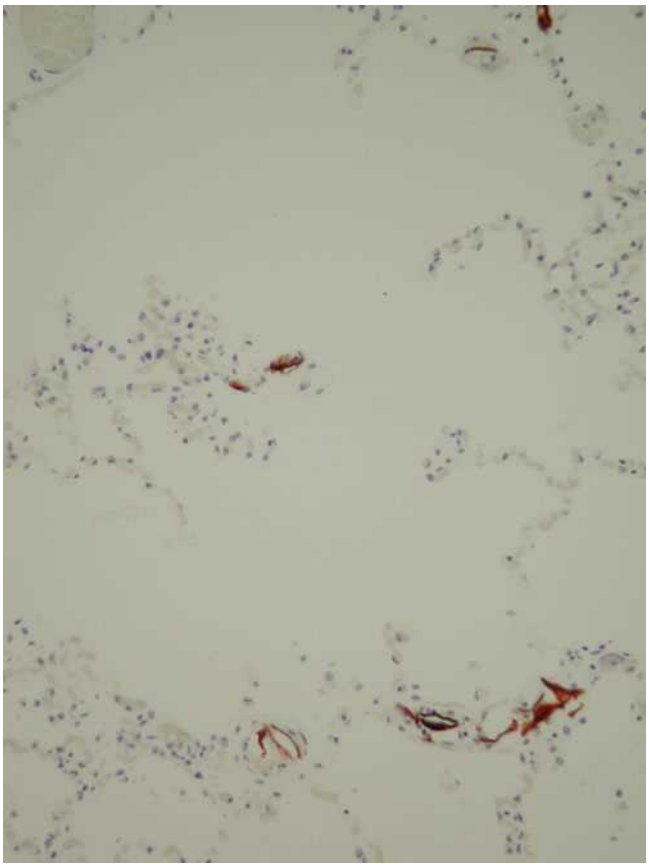

**Fig. 4.41** Histology of lung tissues, using high molecular weight cytokeratin immunohistology, clearly defines fetal epithelial cells (*stained brown*) from amniotic fluid embolism. These cells are present within the capillary vasculature of the lung tissues

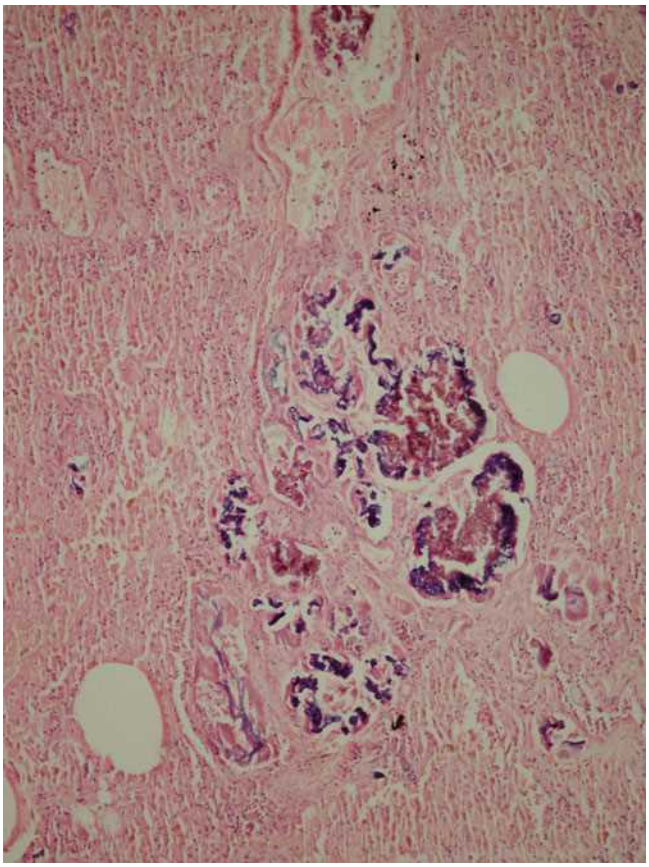

**Fig. 4.42** In cases of intravenous drug use, irregular foreign material concretions are often seen in and around the pulmonary vasculature. The somewhat corrugated and partially calcified debris seen in this view reflects fragments of gelatin-type matrix from partially dissolved drug capsule matrix, which was directly injected into the venous circulation some time previously

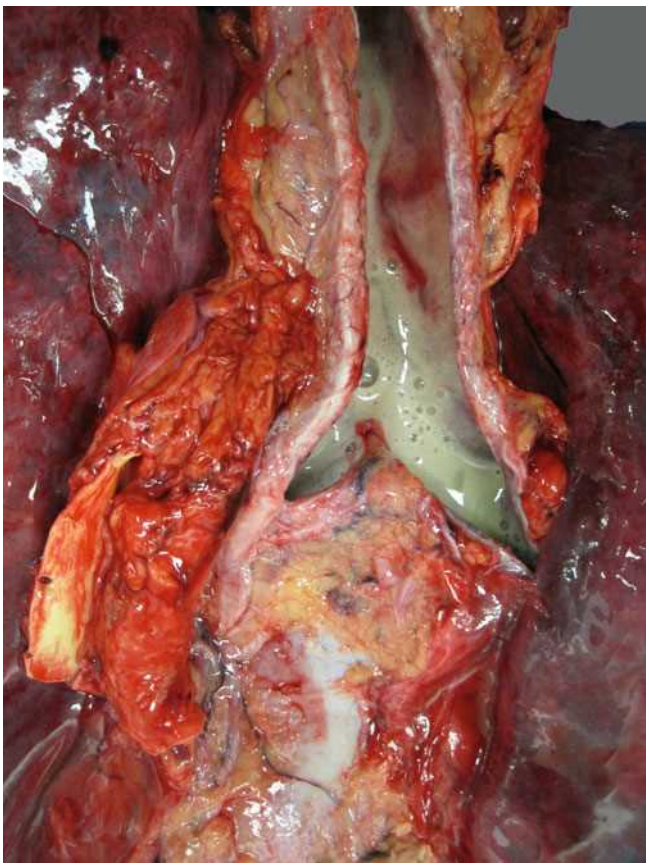

**Fig. 4.44** Bronchopneumonia is seen in the distal trachea at the bifurcation into the lobar bronchi. The mucopurulent secretions are clearly thick and tenacious; they largely obstruct both major airways. This obstruction is a potent cause of death, particularly in the elderly and debilitated

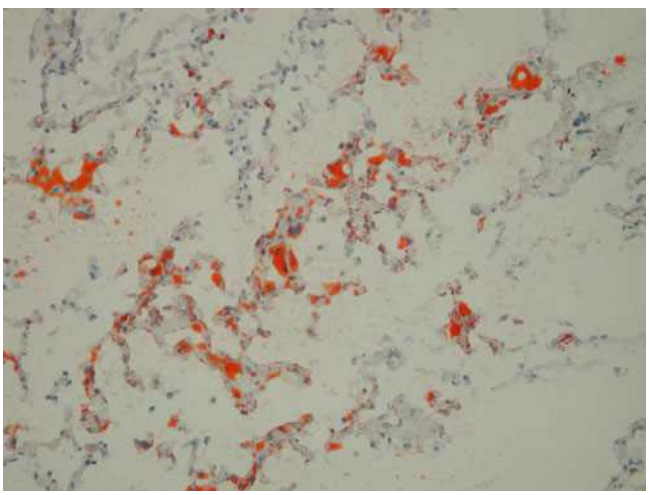

**Fig. 4.43** Frozen section of fresh tissues from the autopsy, followed by Oil Red O staining, serves to highlight fat fragments in a case of fat embolism. It is not possible to use this technique after formalin fixation and paraffin processing, as the fat droplets are removed during tissue processing

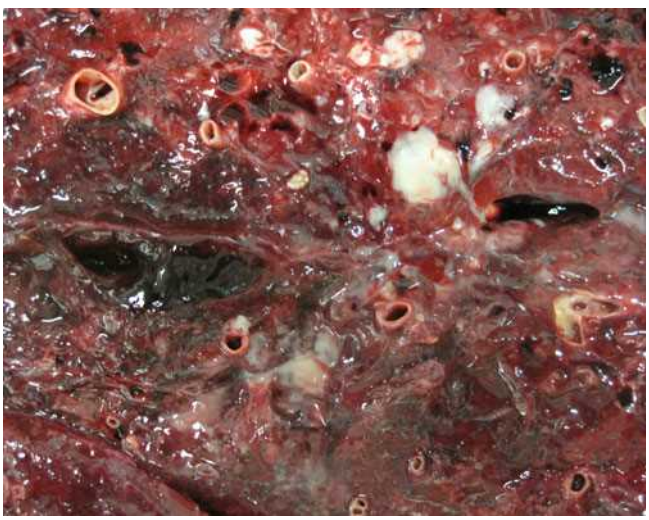

**Fig. 4.45** The cut surface of lobar and bronchopneumonia often shows consolidation, with mucopurulent secretions involving the airways

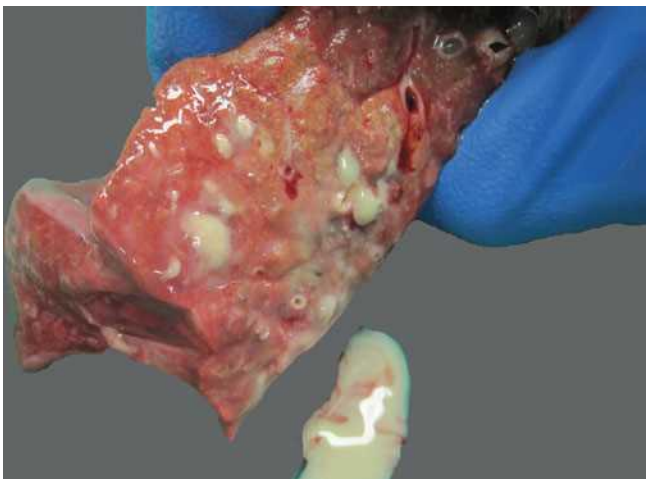

**Fig. 4.46** With gentle pressure, the lung tissues will exude pus from the cut surface, confirming the macroscopic interpretation and allowing sampling of the infected material

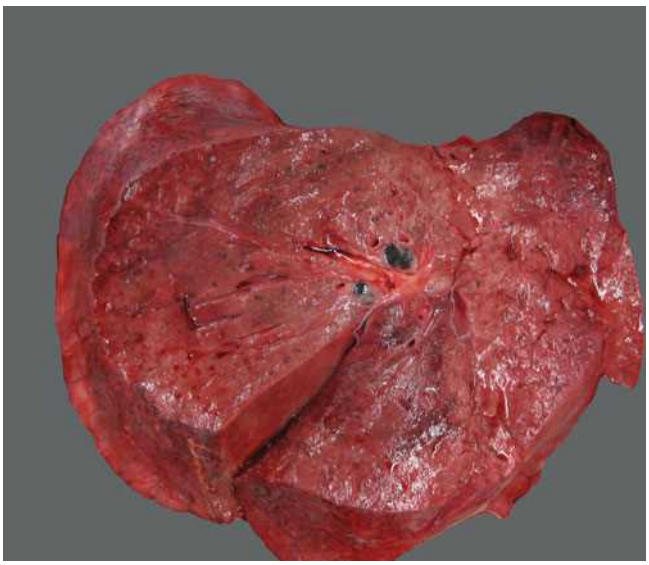

**Fig. 4.48** Consolidation of the lung tissue is often appreciated as solid-appearing lung, but it also can be appreciated as palpably firm tissues

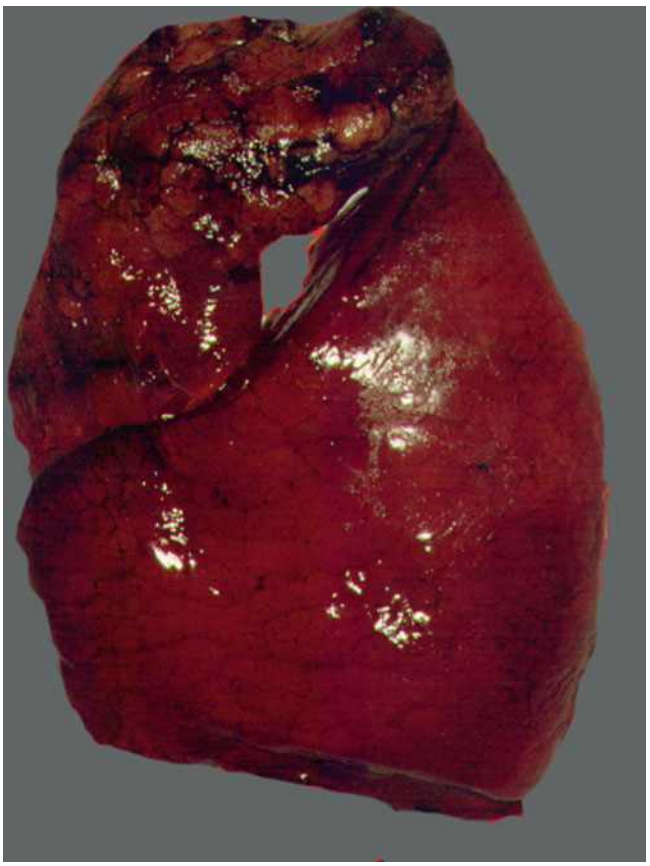

**Fig. 4.47** Lobar pneumonia, with a classic engorged and consolidated lobe of lung, is seen; compare the adjacent upper lobe

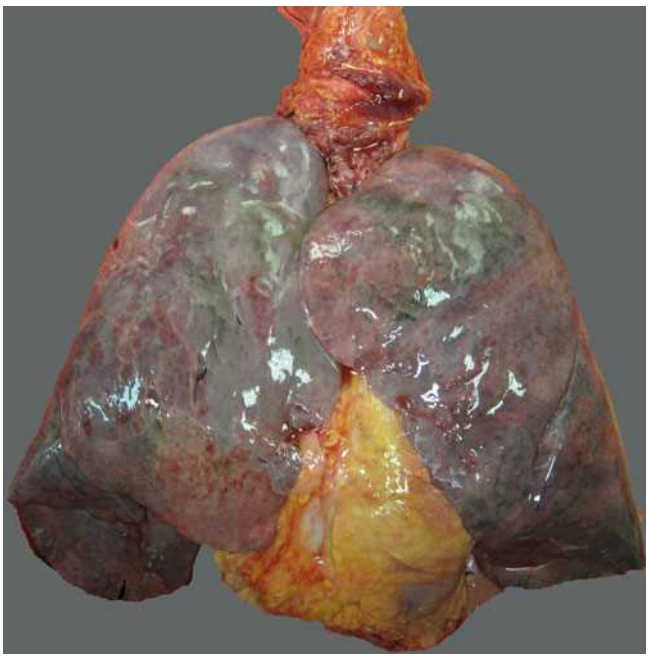

**Fig. 4.49** In this case of influenza pneumonia with bilateral lung consolidation, the distended lungs project across the front of the mediastinum

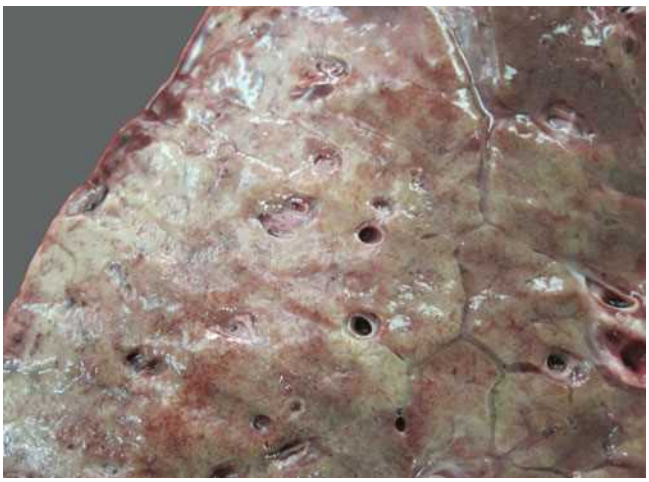

**Fig. 4.50** Influenza may produce diffuse lung consolidation. This H1N1 influenza case shows extreme consolidation of the lung tissues, with grey hepatisation. Histologically, a range of inflammatory cells are often present, together with fibrin and spilled erythrocytes

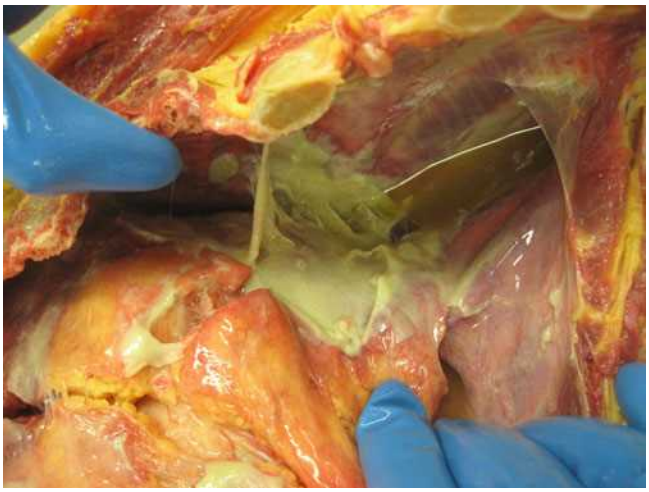

**Fig. 4.51** Septic collections are common in the pleural cavity and are often best examined immediately after the chest has been opened. A septic collection is present at the base of this hemithorax, which contains both turbid fluid and clearly purulent material at the costophrenic angle

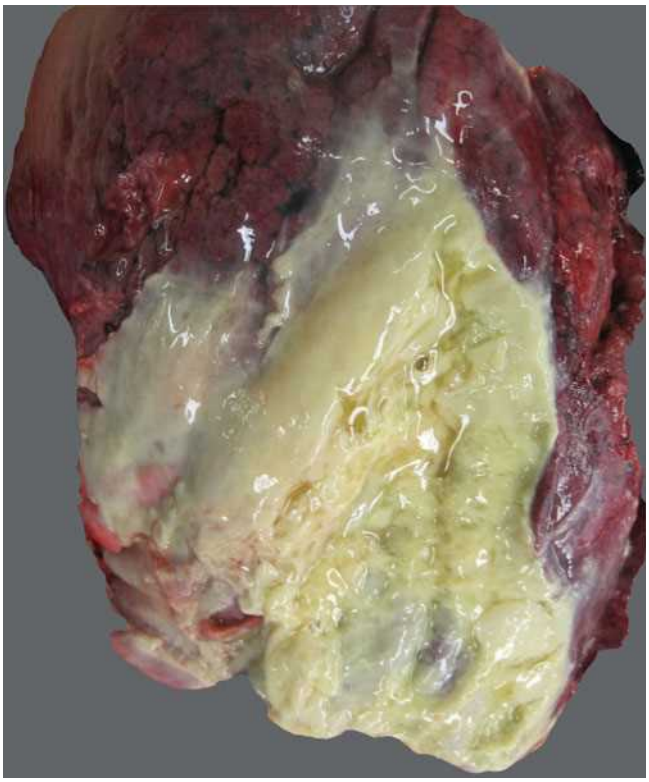

**Fig. 4.52** Empyema can be tenacious and can encase lung tissues. This area of subacute empyema was in the process of organising, with semi-solid and gelatinous/mucoid components being evident. Swab samples should be taken to consider the nature of the infective agent. In addition, histology of the underlying lung and infected substrate is useful

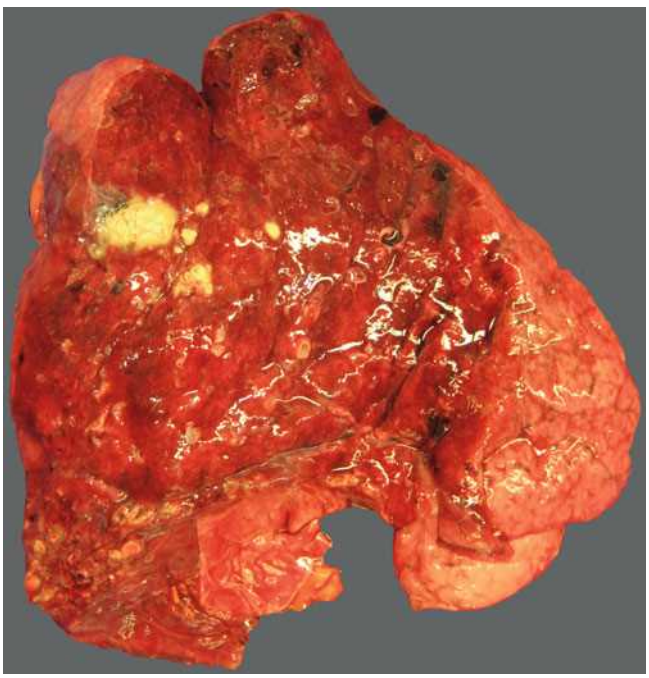

**Fig. 4.53** Peripheral lung tissue is seen in a case of old tuberculosis. There is a focus of fibrosis and old granulomatous change in the lung parenchyma, with smaller nodules of similar inflammation adjacent. No active parenchymal sepsis is present, and the appearances are in keeping with previous tuberculosis

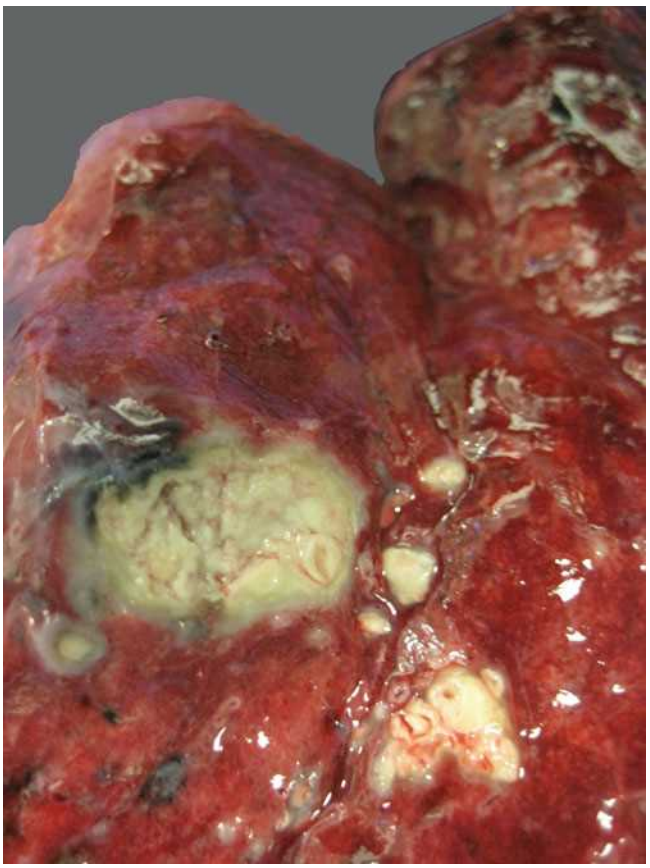

**Fig. 4.54** Higher magnification of the Ghon focus, highlighting old caseous necrosis and local fibrosis

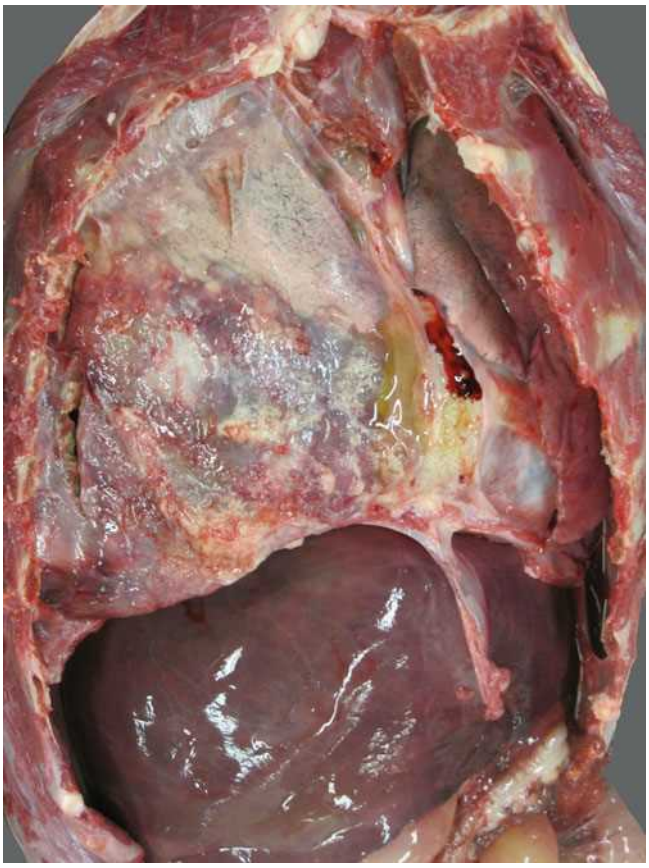

**Fig. 4.55** The chest has been opened in another tuberculosis case, revealing largely consolidated lung tissue that was tightly bound to the chest wall

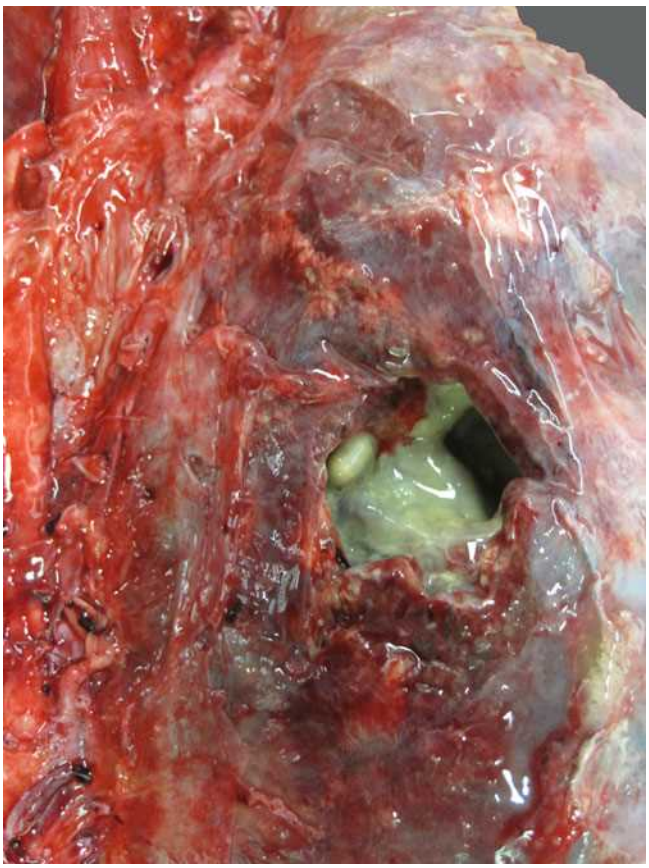

**Fig. 4.56** Further dissection of this case has revealed a cavitated area, which should be regarded as tuberculosis until proven otherwise. Standard cultures as well as specialist culture for mycobacteria and fungi should follow

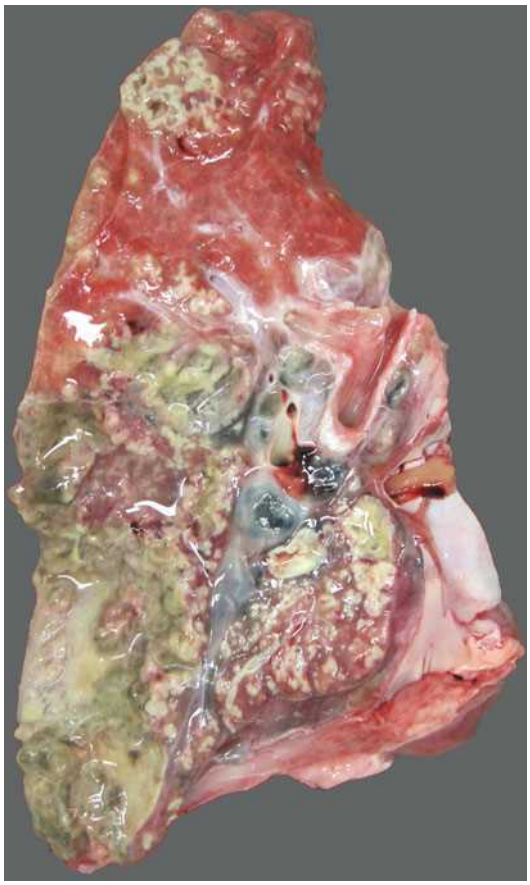

**Fig. 4.57** This case of tuberculosis with overwhelming lung sepsis shows necrotising pneumonia, consolidation, and destruction of the pulmonary parenchyma

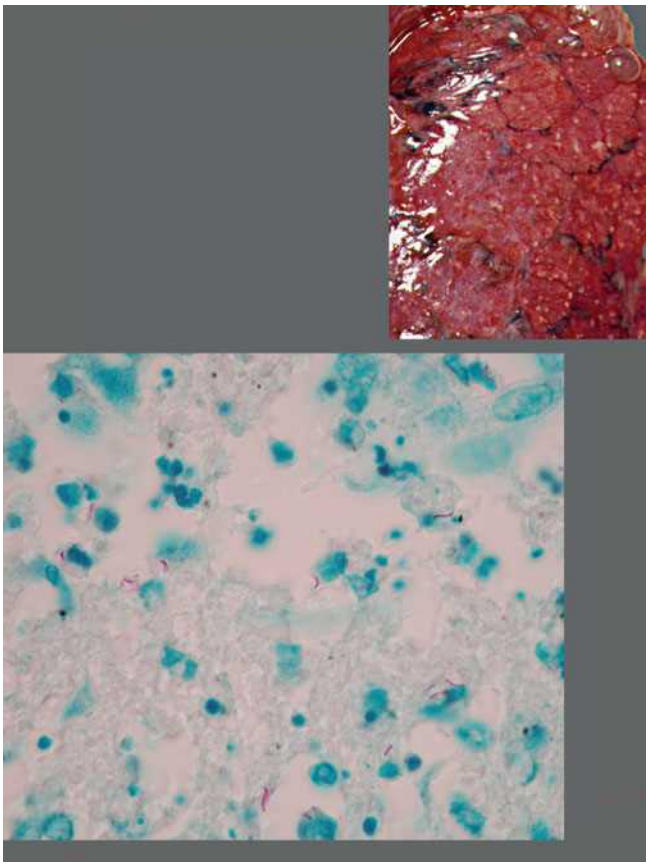

**Fig. 4.58** Miliary (millet seed–like) tuberculosis is seen, with fine, small flecks of yellow discolouration diffusely across the lung parenchyma. Acid-fast bacilli are seen with characteristic elongated, red-stained forms in a Ziehl-Neelsen stain

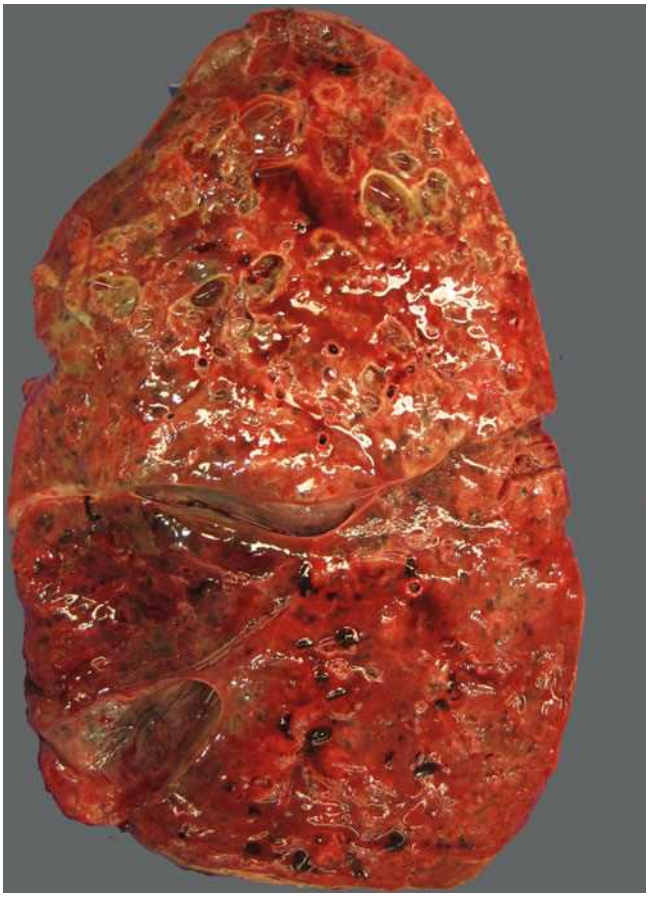

**Fig. 4.59** Bronchiectasis can be lobe-limited or diffuse. Dilatation of many of the airways is seen, often with some collapse of the parenchymal tissues and persistent sepsis. In this case, the lung is markedly infected, and swabs or lung tissue should be taken to assess microbiological content

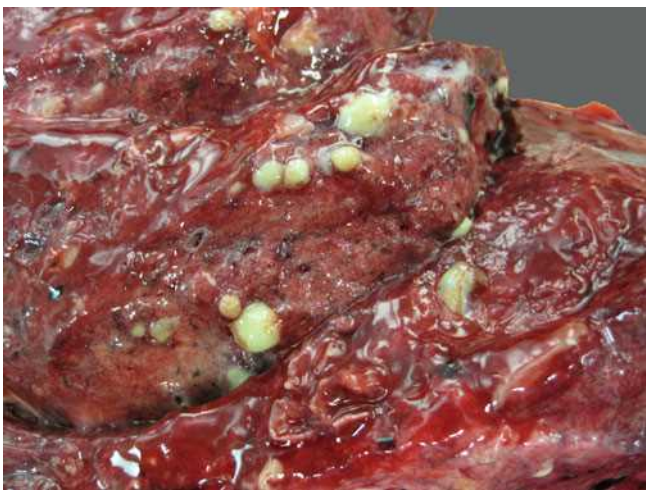

**Fig. 4.60** Bronchiectasis is often complicated by persistent sepsis with significantly pathogenic bacteria. This young individual, suffering with cystic fibrosis, shows marked bronchopneumonia associated with bronchiectasis. The degree of bronchiectasis is masked by the pronounced infected secretions

**Fig. 4.61** This lobe-limited zone of bronchiectasis had developed into a persistent zone of sepsis with necrotizing pneumonia and finally abscess formation

**Fig. 4.62** Close inspection of the cut surface of this lung in a case of mild bronchiectasis shows somewhat fibrotic-walled airways, which are dilated and congested. In addition, there is local, patchy emphysema and some cystic change and interstitial scarring. Many cases of lung disease do not show a single, pure format

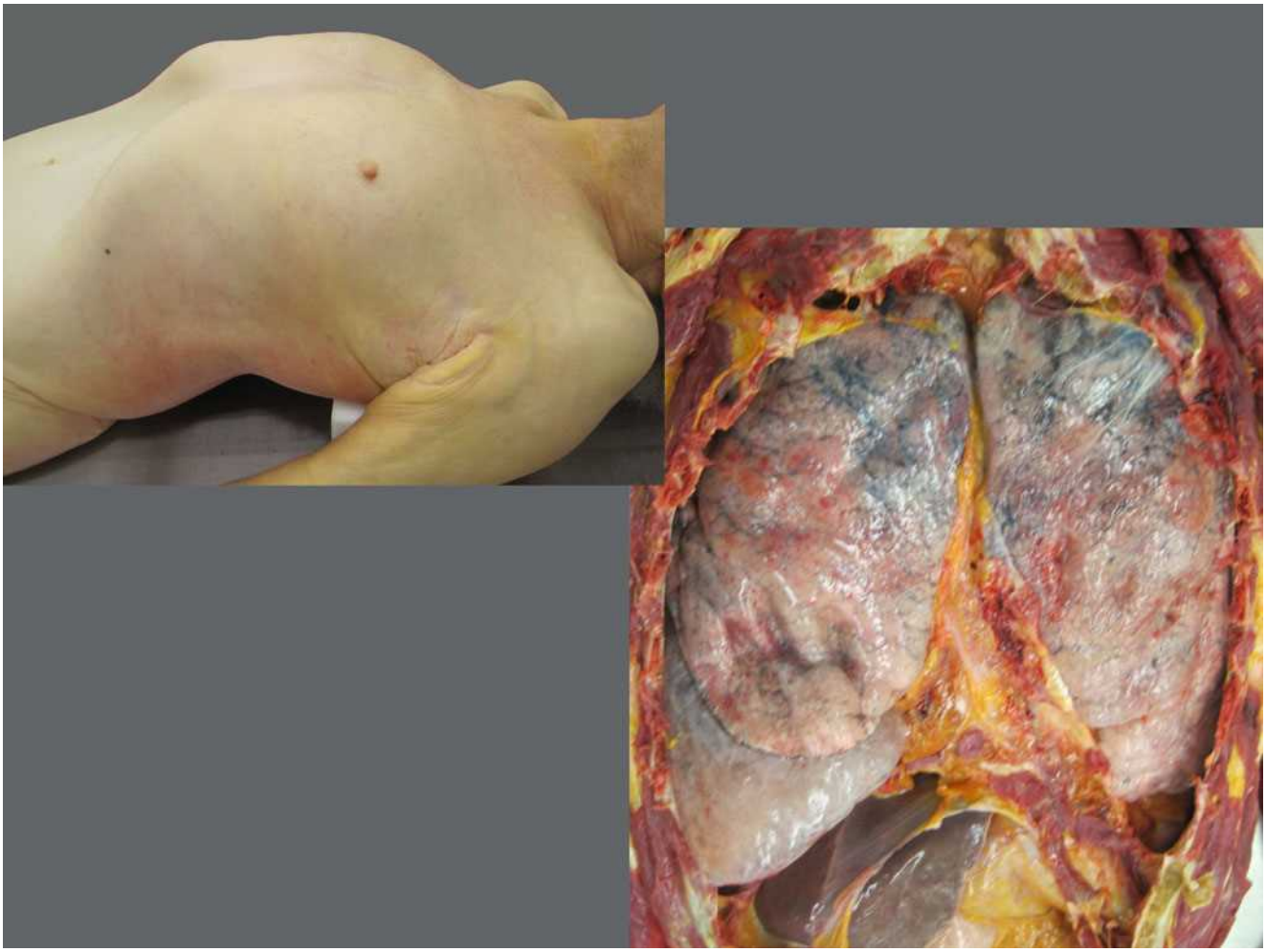

**Fig. 4.63** The external examination of the chest of a patient with emphysema (*upper left image*) often starts with the appreciation of a 'barrel chest' reflecting pulmonary hyperinflation. The *lower right image* shows a case of emphysema with lung hyperinflation filling the chest cavities and extending across the anterior mediastinal soft tissues. Bullous change is noted peripherally at the base of the right lung

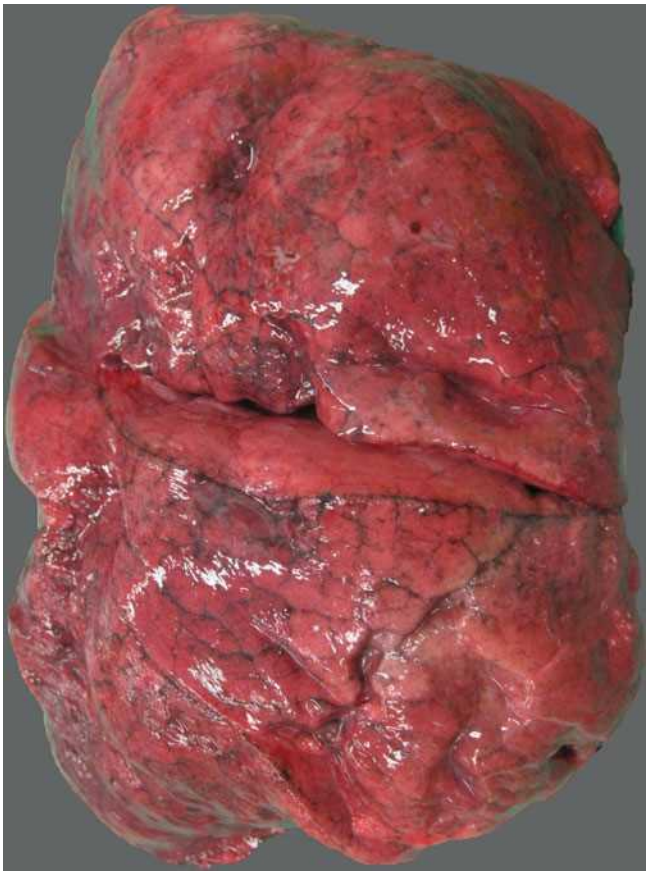

**Fig. 4.64** Emphysematous lungs often remain inflated despite dissection from the mediastinal content

**Fig. 4.65** End-stage pulmonary emphysema is seen with widespread bullous change affecting the tissues of both the upper and lower lobes

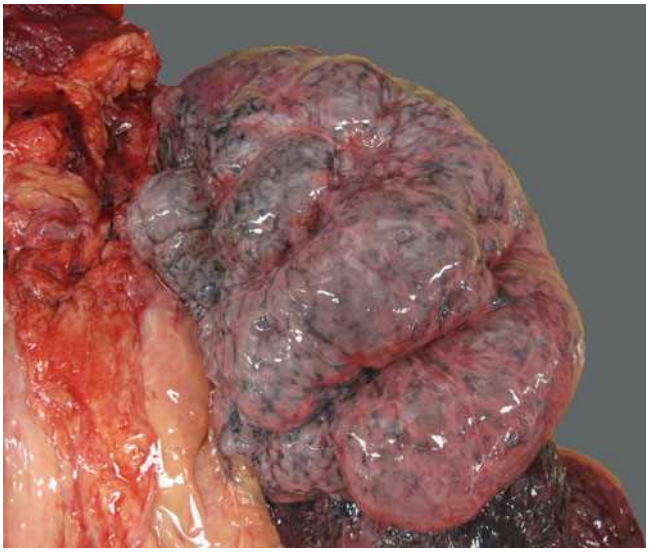

**Fig. 4.66** Large bullae are often evident when lungs are examined; an assessment of the overall size is useful in the report

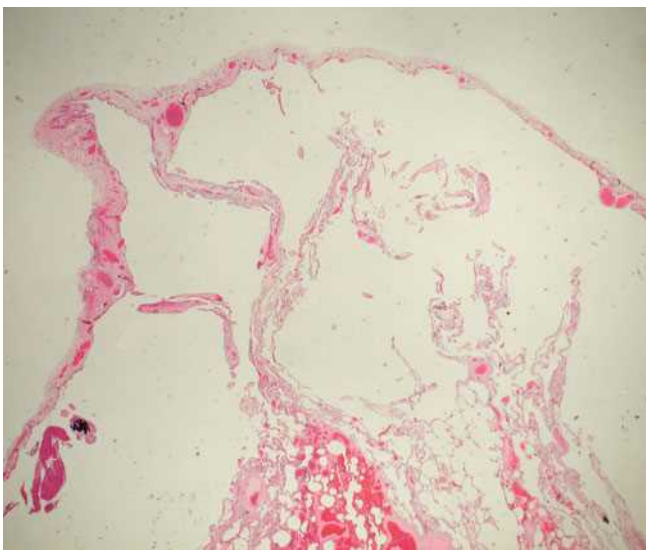

**Fig. 4.67** Histological examination of emphysematous tissue reveals the characteristic alveolar wall loss and airspace dilatation

**Fig. 4.68** This Gough and Wentworth section shows evidence of widespread emphysematous change. This historical technique is rarely used now, given the significant advances of radiology, including high-resolution CT scanning (HRCT)

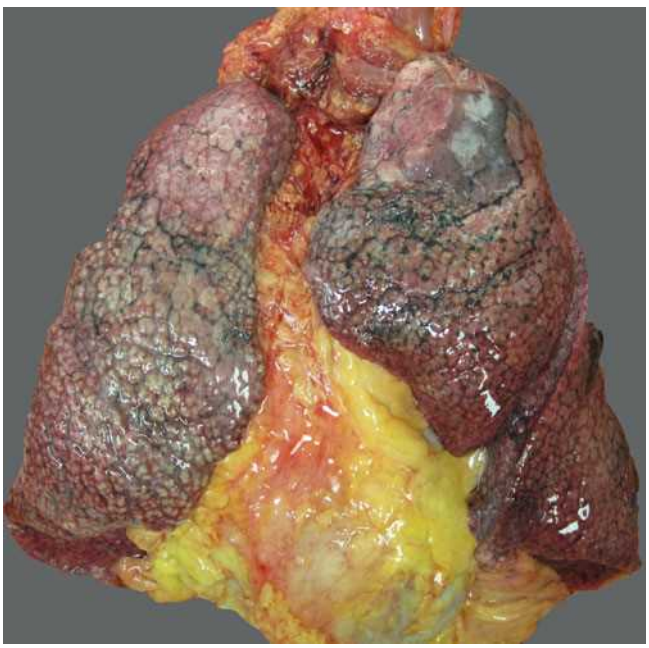

**Fig. 4.69** The lung parenchyma shows firm lungs with a somewhat bosselated pleural surface quality, akin to morocco leather. The tissues are palpably firm

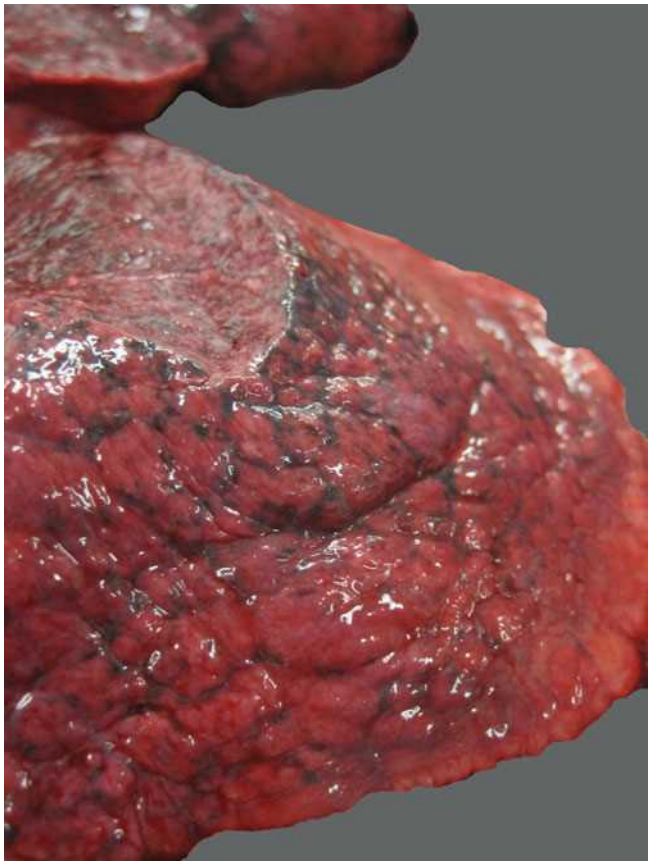

**Fig. 4.70** Lung tissues with interstitial fibrosis often maintain their architecture even after slicing into the lung tissue at the periphery

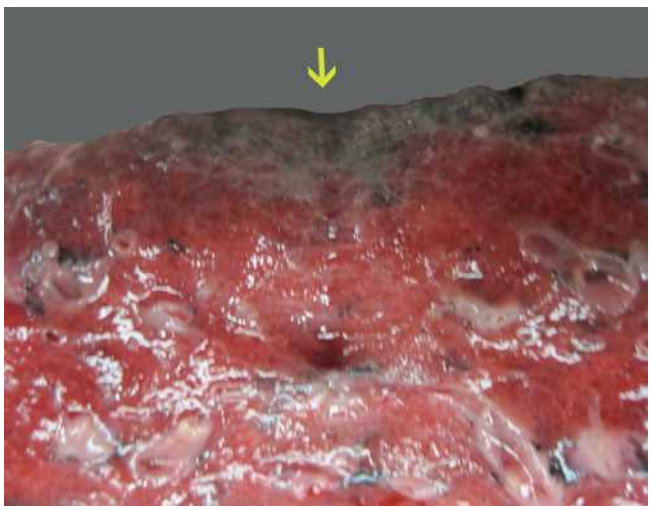

**Fig. 4.71** Usual interstitial pneumonia (UIP) is often the format for idiopathic lung fibrosis, also known as cryptogenic fibrosing alveolitis (CFA). The peripheral fibrosis is markedly grey (*arrow*). This area has been highlighted by washing the lung tissue under running water

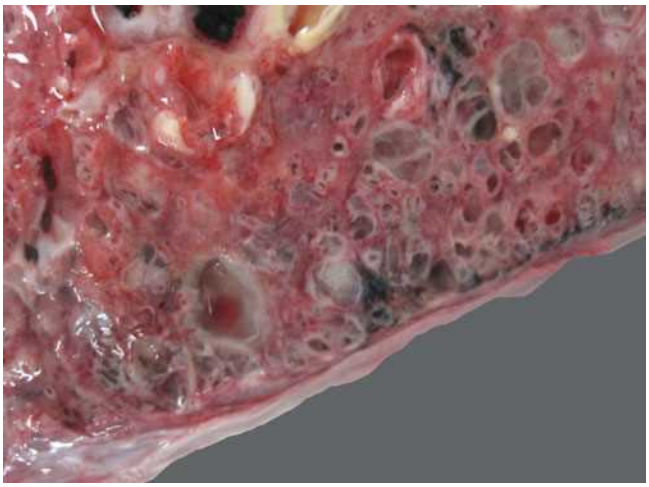

**Fig. 4.72** This lung tissue has been washed carefully. It shows pronounced loss of the normal tissues and airspace dilatation/retraction artefact, manifest as peripheral cystic change

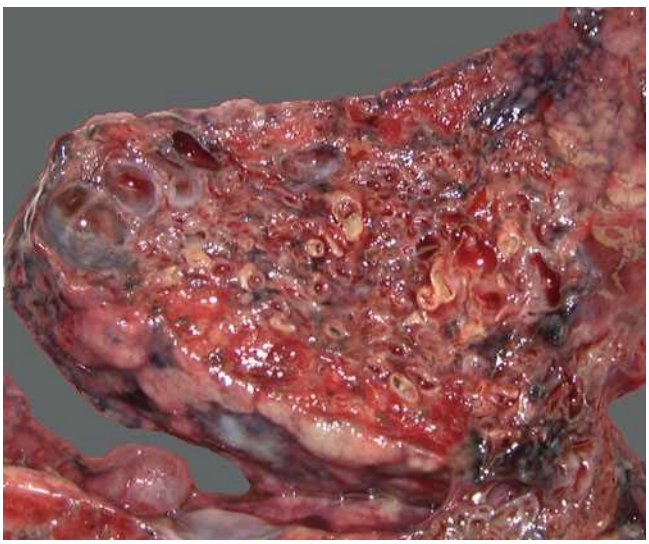

**Fig. 4.73** Lung tissues with extensive or end-stage fibrosis generally all look similar, with honeycomb change, fluid collections in cystic spaces, and some sepsis. This case of lung disease had a background of exposure to metal dust and fumes. From the macroscopic perspective, it looks no different from naturally occurring lung fibrosis

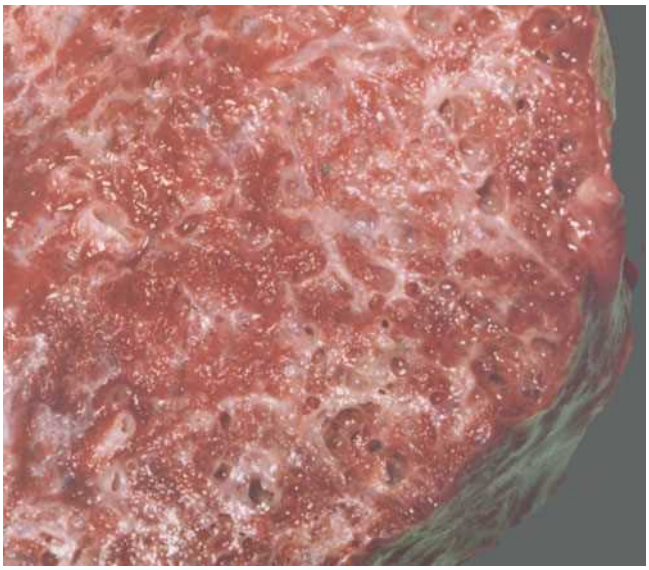

**Fig. 4.74** A surgical sample is seen in a case of Langerhans' cell histiocytosis (LCH). The cut surface of the lung has widespread cystic and fibrotic change characteristic of this condition. LCH is often similar morphologically to lymphangioleiomyomatosis (LAM)

**Fig. 4.75** The fibrotic and cystic disease features of lymphangioleiomyomatosis (LAM) are seen in this lung tissue, which has been fixed and then sectioned. In many ways, the appearance is akin to Langerhans' cell histiocytosis (LCH)

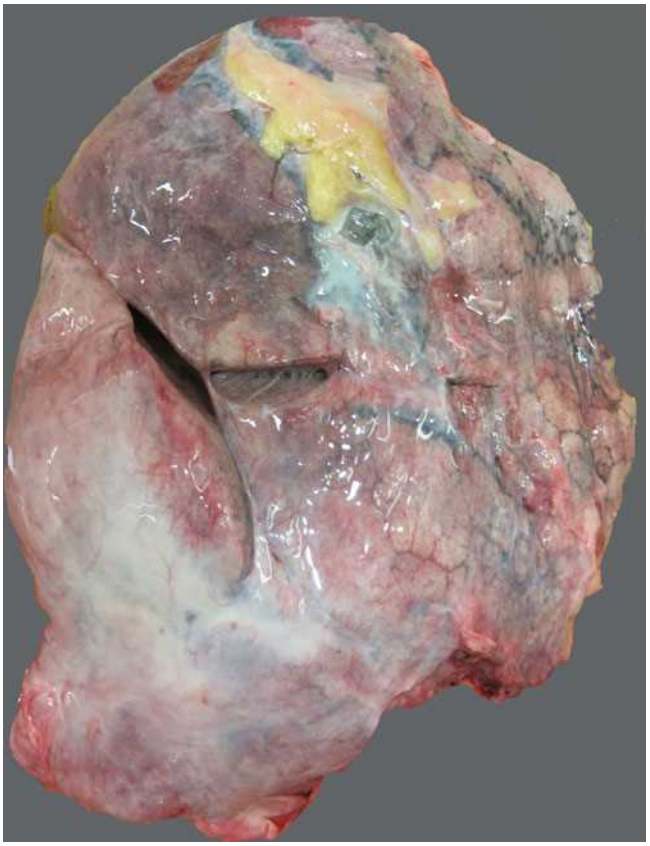

**Fig. 4.76** In some cases of asbestos exposure, persistent pleural effusions can cause protein deposition, which produces a slow accumulation of fibrous matrix around the dependent parts of the visceral pleural surface. This sometimes manifests as diffuse pleural fibrosis, one of the components of asbestos-related disease

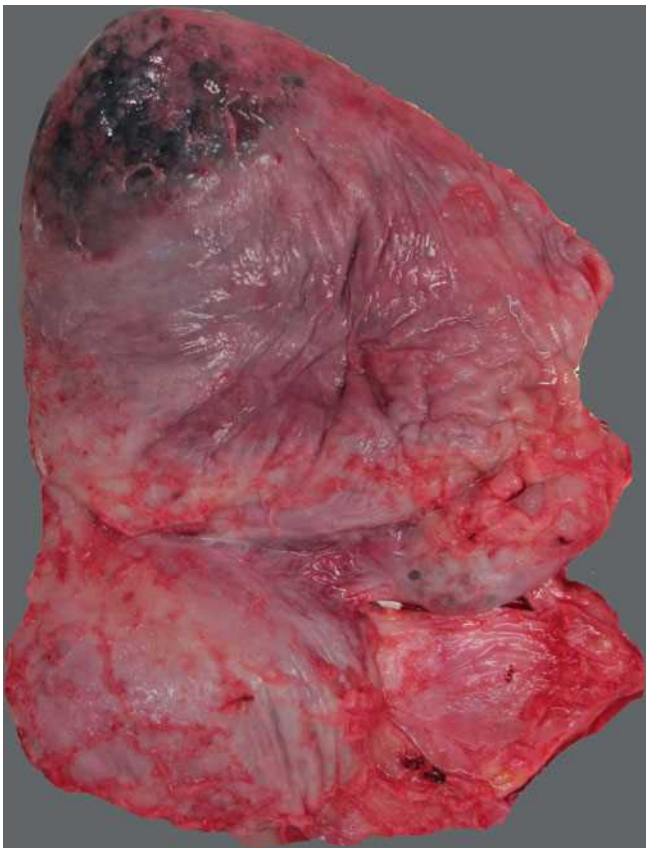

**Fig. 4.77** Diffuse pleural fibrosis in a case of asbestos disease. This should not be confused with mesothelioma or other pleural neoplasia

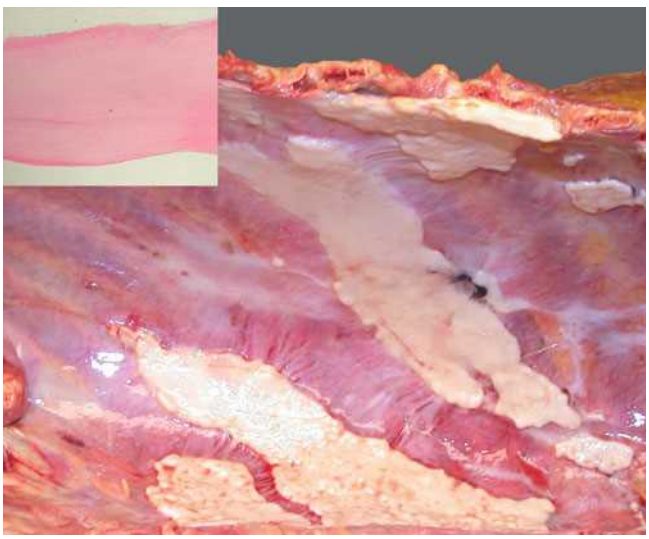

**Fig. 4.78** Benign pleural plaques are dense zones of fibrous tissue fixed onto the pleural surface. They are often parietal tissue biased, but may be widespread on the visceral pleural surfaces. The plaques may be calcified. The *inset* shows the histological appearance of dense hyaline collagen, with a somewhat 'basketweave' quality. Asbestos is linked with formation of pleural plaques, but they can occur with mild or trivial exposures. The presence of plaques does not automatically imply significant exposure to asbestos fibres

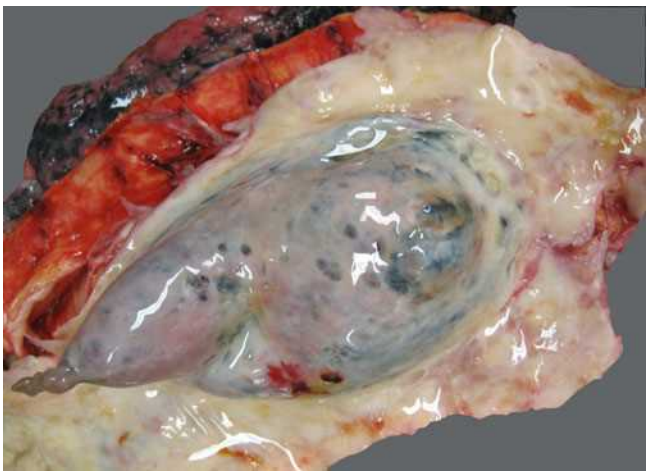

**Fig. 4.79** Pleural mesothelioma commonly surrounds and compresses lung tissue. This case had a persistent effusion, with tumour widely infiltrating the chest wall. This lung was firmly encased within a dense, fibrous tumour capsule. The tumour has compressed the lung to roughly one third of its original volume

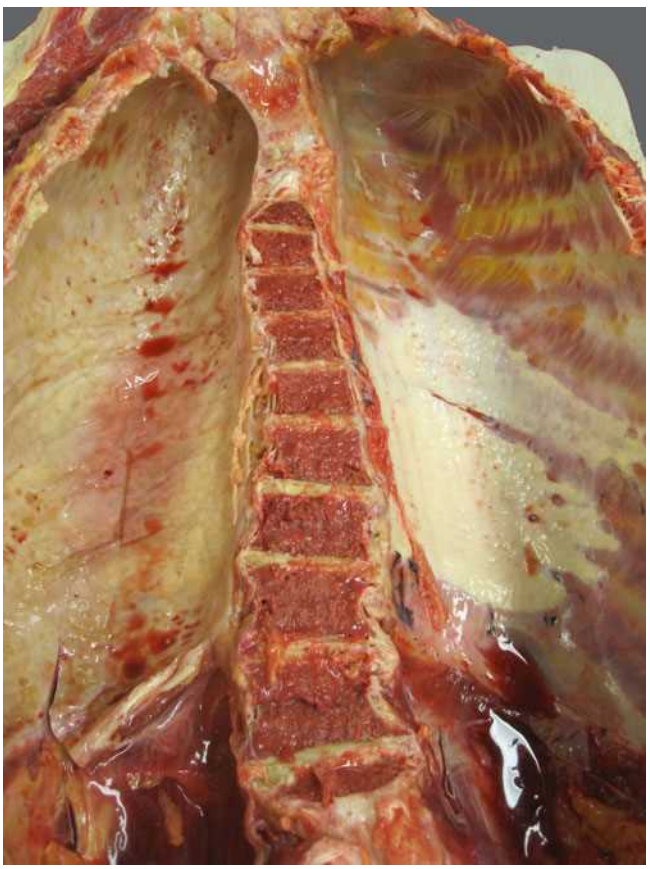

**Fig. 4.80** The opened chest in a case of mesothelioma (*right hemithorax*) is seen with the *left hemithorax* containing pleural plaques. Confirmation of plaques is important in cases of asbestos disease

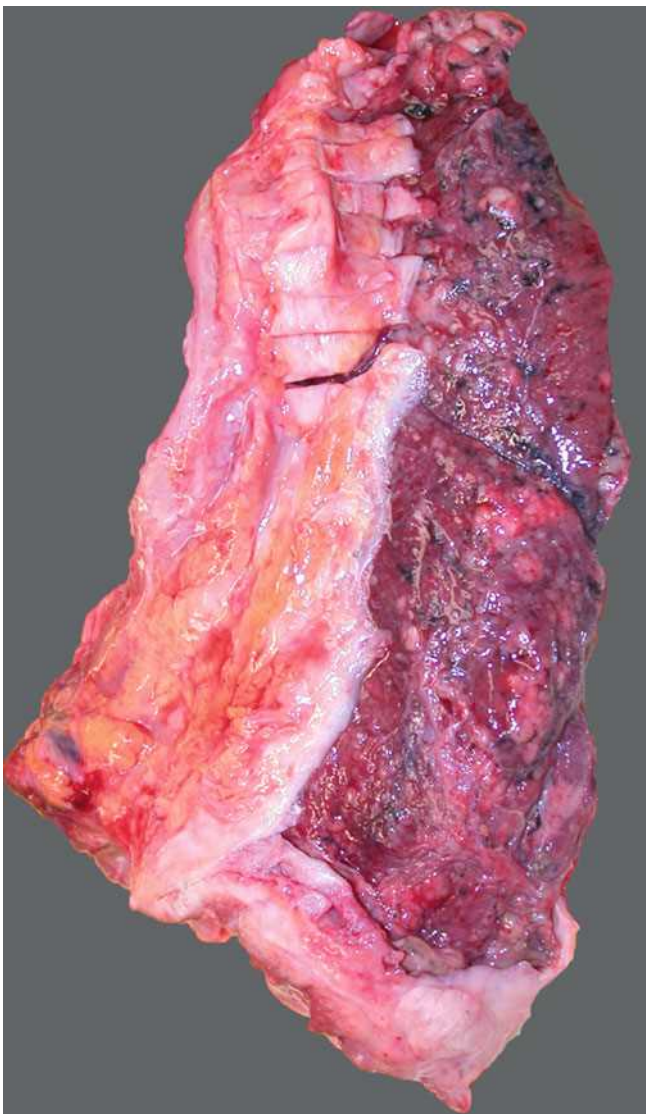

**Fig. 4.81** Mesothelioma tissue is seen running around the lung parenchyma, tightly binding together the tumour, chest wall, and lung tissue. The lung itself is compressed and atelectatic, with nodularity representing tumour infiltrating in a lymphangitis-like fashion throughout the tissues

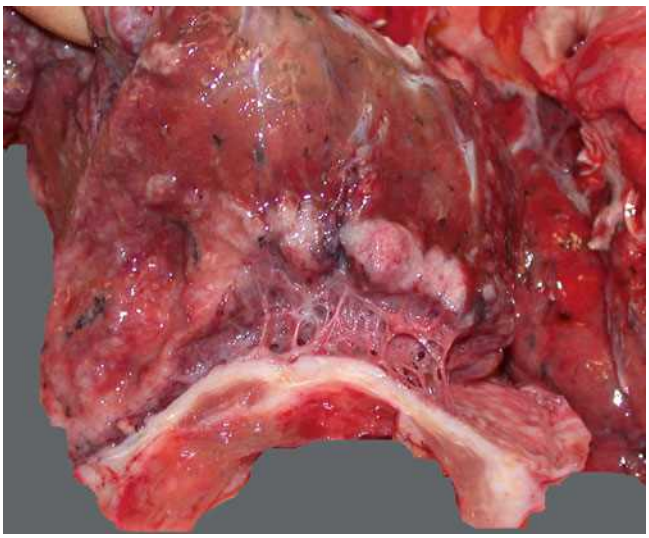

**Fig. 4.82** Mesothelioma characteristically produces a rind of tumour on the outer aspect of the lung, involving the visceral pleura. Tumour foci are also seen here eroding into the lung tissue, with bands of tumour linking the visceral and parietal pleural tissue

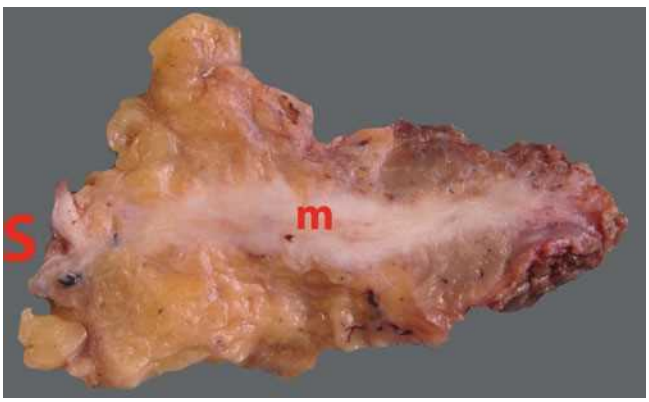


**Fig. 4.83** Mesothelioma (grey tissue *m*) may often extend out along thoracotomy tracts to involve chest wall tissues and skin surface. This is a perpendicular section of chest wall tissues highlighting the path of the mesothelioma towards the skin surface (*S*). For this reason, radiotherapy is often given to the port site of thoracoscopic intervention

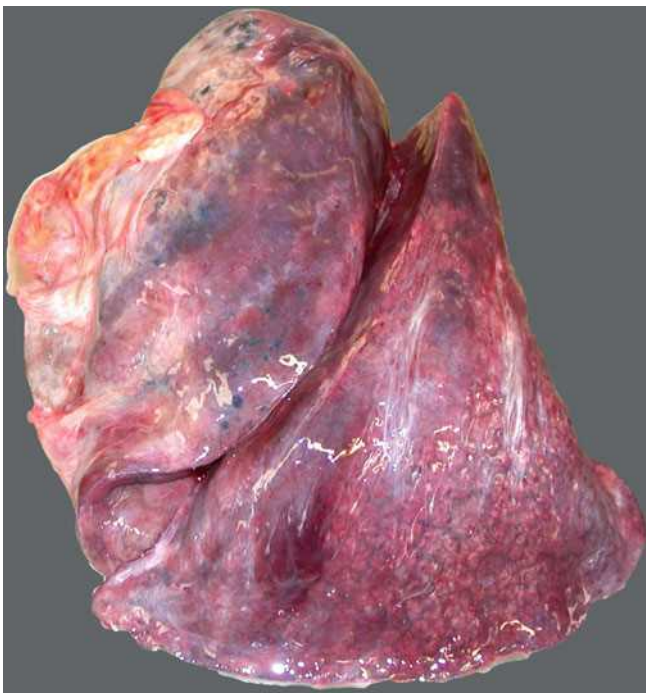

**Fig. 4.84** Pulmonary fibrosis due to asbestos appears morphologically similar to pulmonary fibrosis or interstitial lung disease from other causes

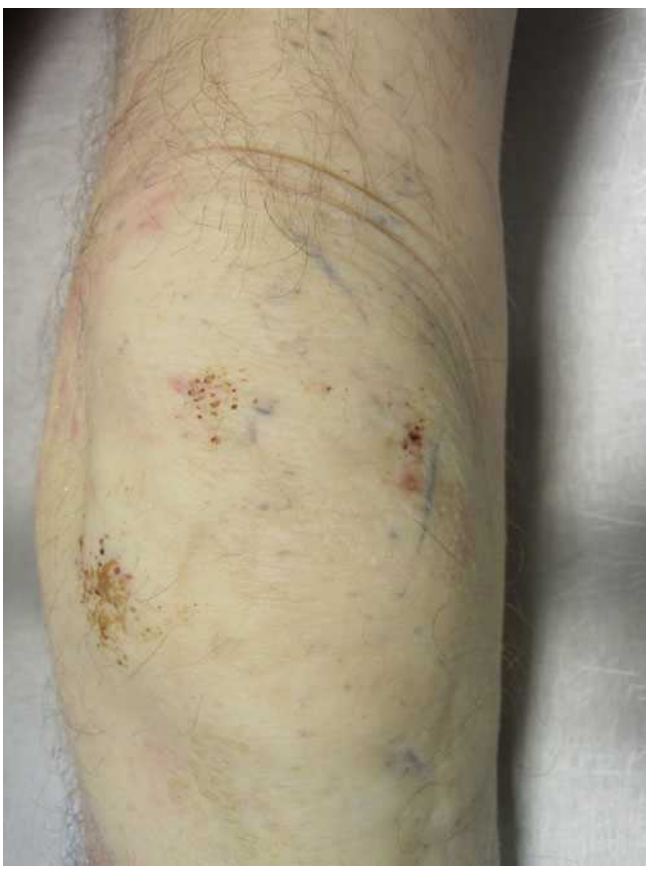

**Fig. 4.85** Aside from considering the lung tissue in cases of coal workers' disease, it is important to look for coal-dust tattoos, which are usually present on the hands, elbows, shoulders, and knees. This view of the knee shows irregular, black discolouration of the dermal tissues in keeping with coal dust 'inoculation' into the dermal tissues during underground coal mining

**Fig. 4.86** Coal workers' lungs can be variably affected by the dust. Extreme forms have heavy anthracosis and distortion of the lung tissue

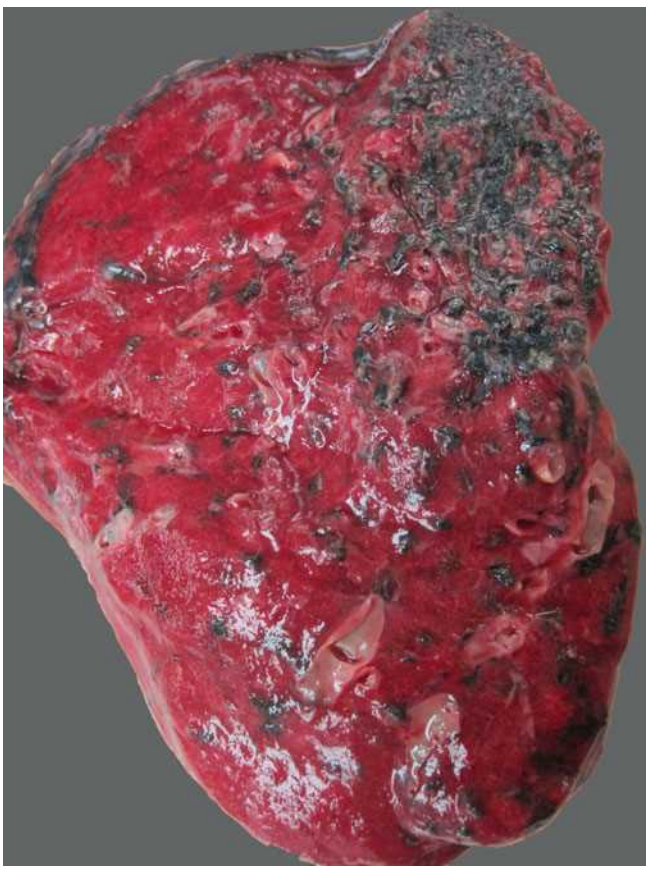

**Fig. 4.87** The lung parenchyma in a coal worker shows gradation of nodularity, with a zone of complex nodularity bordering on progressive massive fibrosis in the upper part of the lung. Small nodularity with anthracosis is present in the lower part of the lung tissues

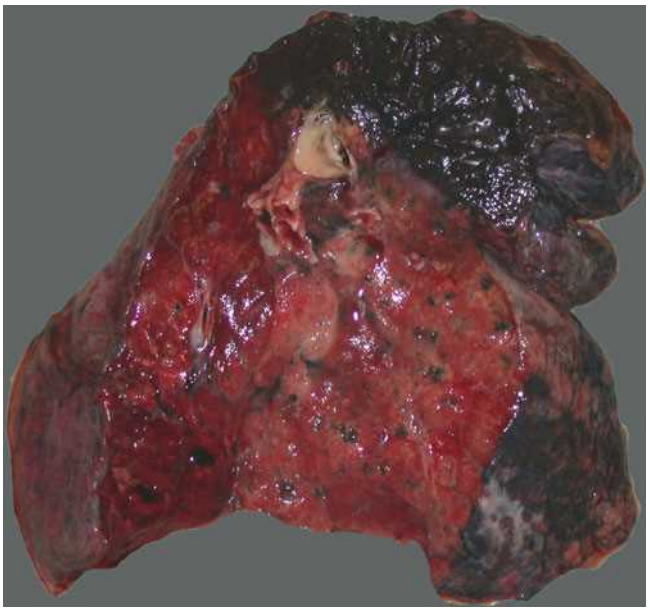

**Fig. 4.88** This is a case of progressive massive fibrosis within the upper lobe tissues

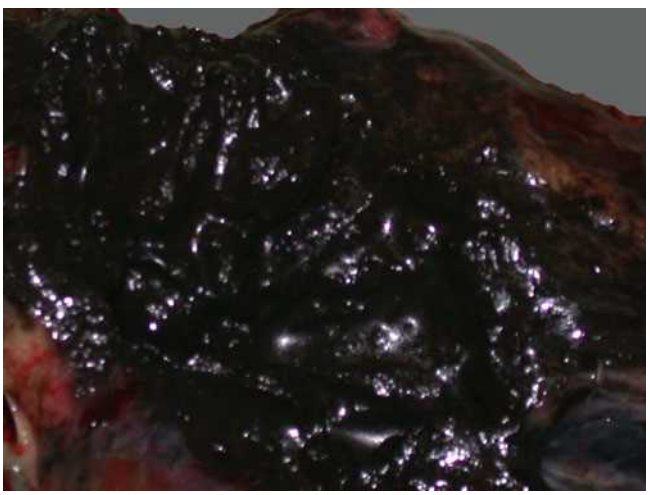

**Fig. 4.89** Progressive massive fibrosis, seen close up, reveals the solid and rather gritty mass replacing lung parenchyma. The cut surface often has no substructure; after being sectioned, it may ooze black fluid

**Fig. 4.90** Classically, Gough-Wentworth lung slides were used to assess the broad detail of lung parenchyma, but this technique is no longer commonly used. The heavy dust accumulation in this case is mainly associated with emphysema

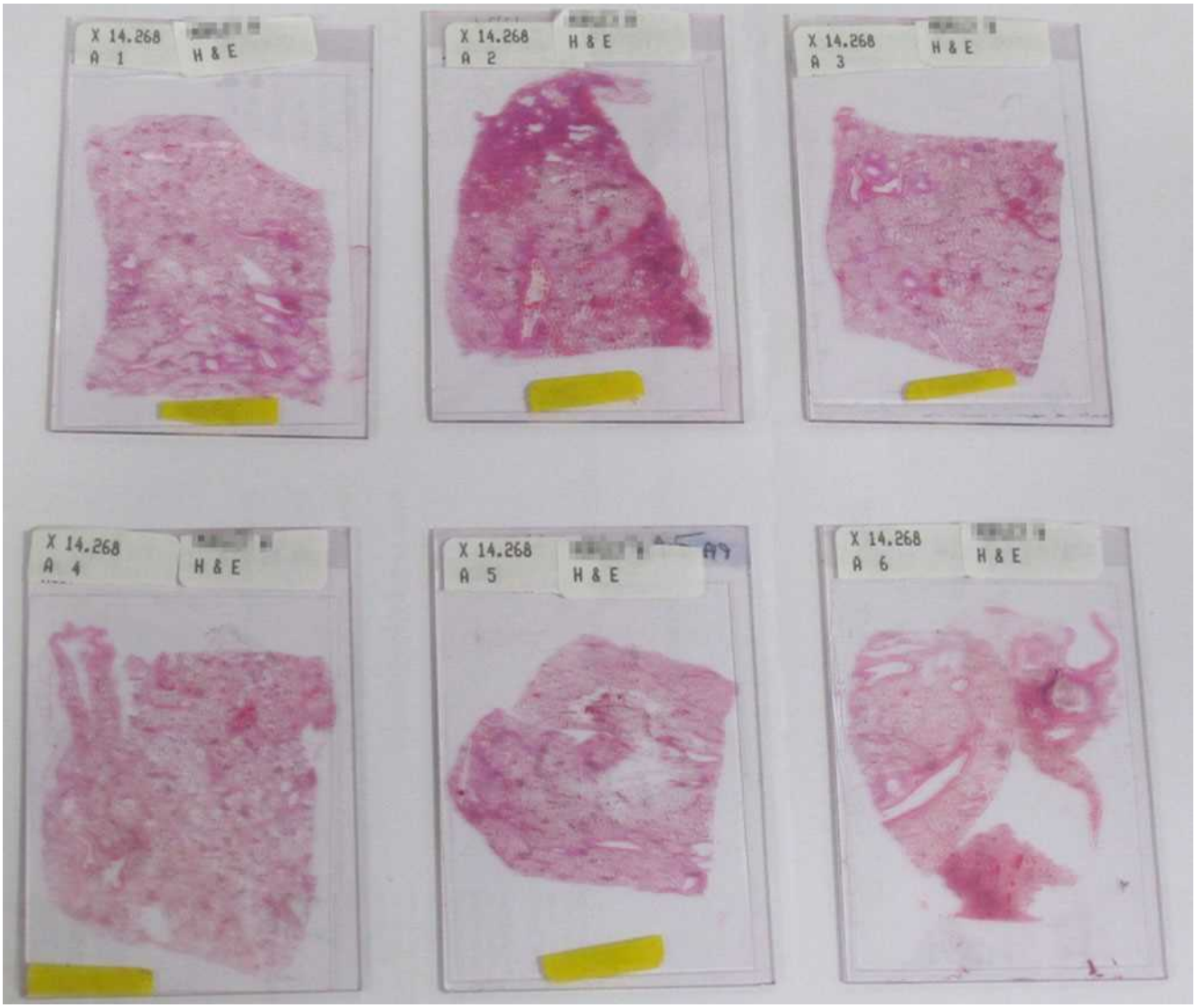


**Fig. 4.91** The modern technique to assess lungs in a manner comparable to Gough-Wentworth slides uses large blocks of paraffin-embedded tissues (*e.g.*, akin to colorectal cancer blocks)

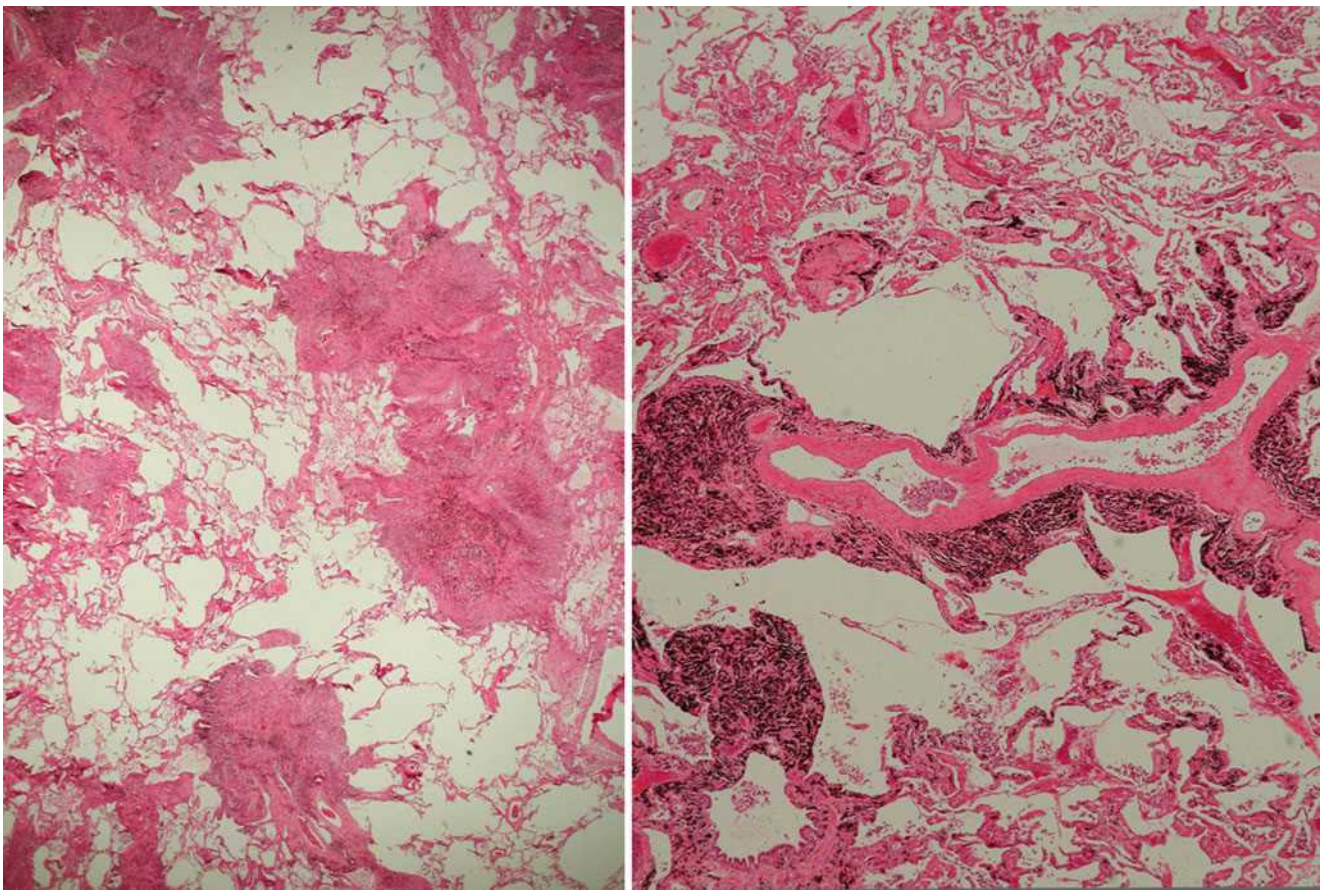

**Fig. 4.92** Histological review of lung parenchyma allows assessment of the degree of dust deposition and any nodularity. *Left* the size of any nodules should be assessed; those more than 10 mm in diameter are often functionally more important and are judged complicated coal workers' pneumoconiosis. *Right* review of this lung shows emphysematous phenomena with dust accumulation in a centriacinar quality (dust-associated emphysema)

**Fig. 4.93** The periphery of the lung tissues and some nodes centrally show an irregular grey/cream discolouration with nodularity. This was a case of silicotic change in the lung tissues, with the zones affected showing dense fibrosis and the characteristic stellate, irregular scars of this mineral dust accumulation

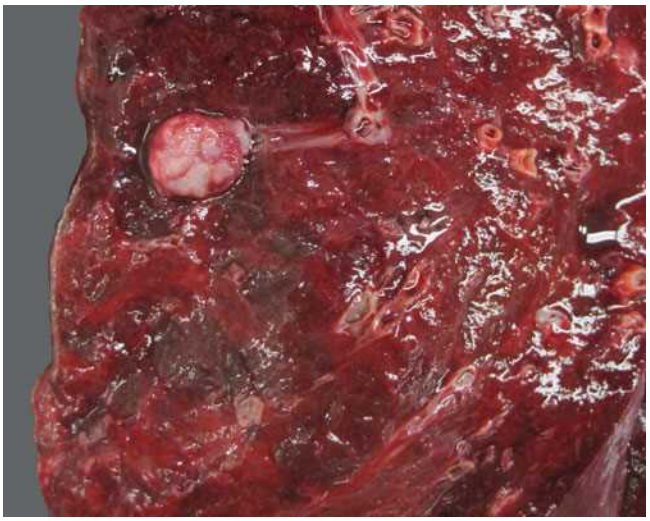

**Fig. 4.94** Benign tumours occasionally can be found in lung tissues. This slice of lung shows a rounded hamartoma with unremarkable lung tissues adjacent

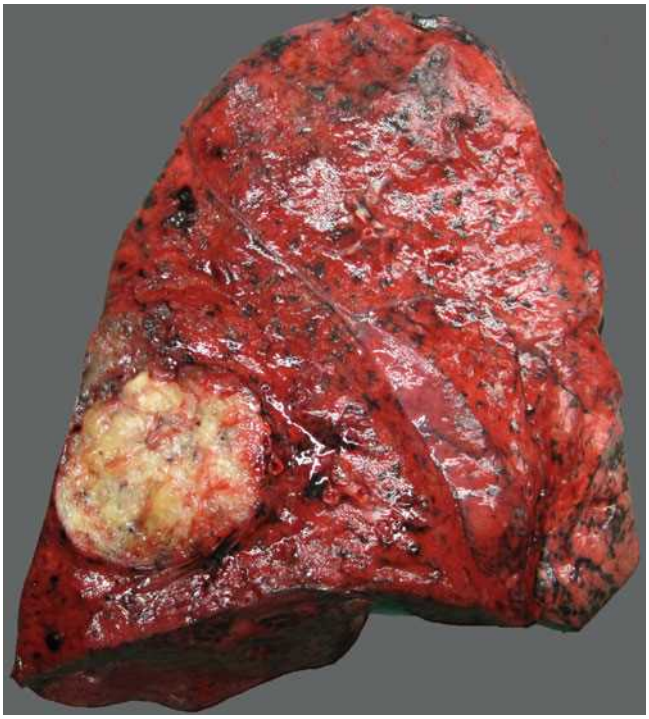

**Fig. 4.95** Metastatic tumours are commonly found in the lung tissues. These tumour foci usually have a more rounded profile with compression of the local tissues and little in the way of background lung alteration unless there is airway obstruction. This was a case of disseminated colonic adenocarcinoma

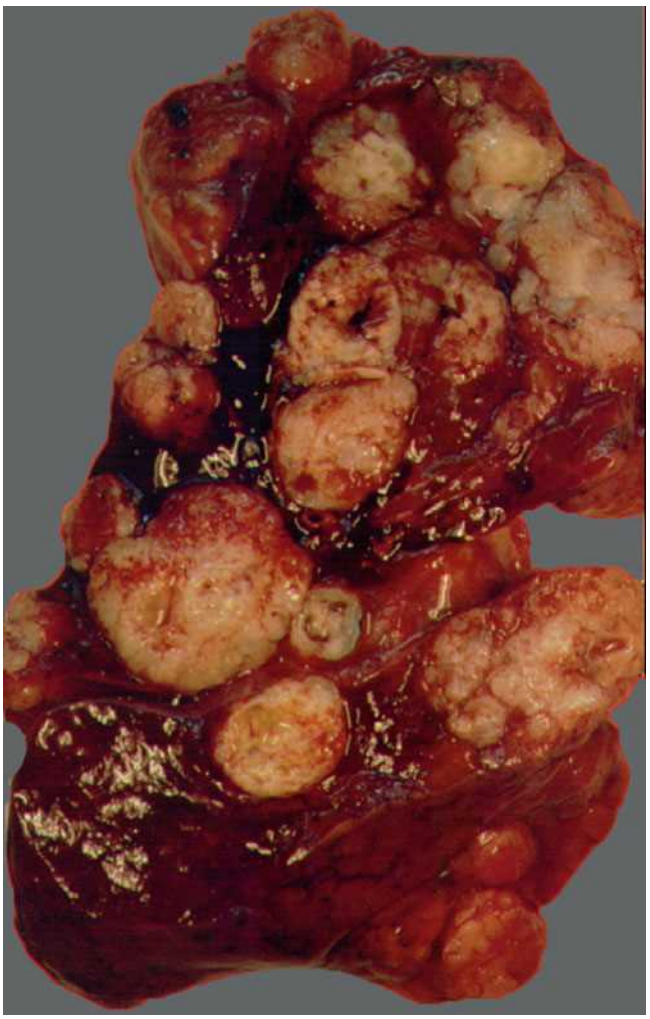

**Fig. 4.96** Diffuse pulmonary metastases are often seen in sarcomatoid tumours. This is an example of metastatic chondrosarcoma

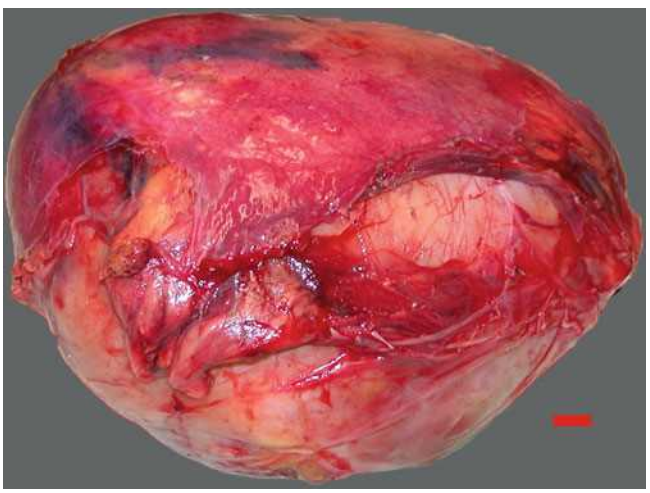

**Fig. 4.97** Soft tissue tumours—occasionally of large size, as in this case of liposarcoma—can be found interacting with the lung within the thoracic cavity (*Bar marker* 10 mm)

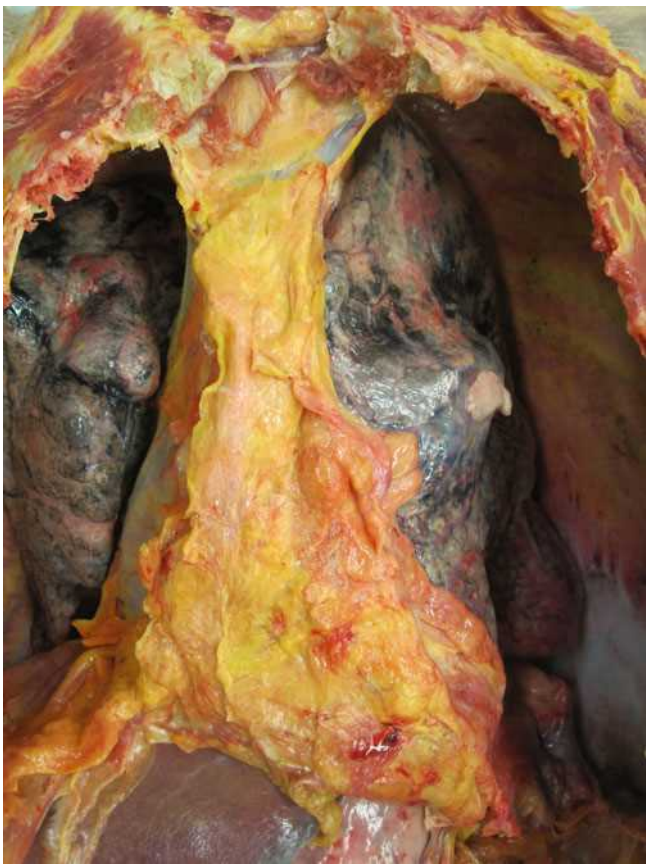

**Fig. 4.98** Lung tumours are often primary carcinomas. This left upper lobe tumour clearly stands out from the adjacent lung tissues and is immediately apparent after opening the chest

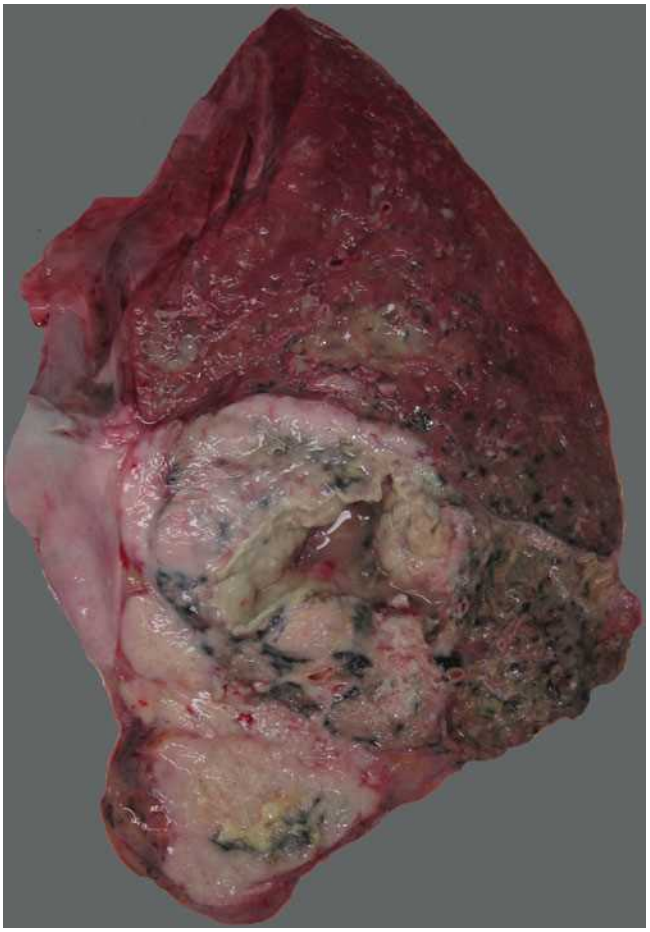

**Fig. 4.99** Lung cancer is often associated with local or distal chest infection and collapse of the lung tissues, making precise assessment of the boundaries complex. This case shows a lower lobe tumour mass with central cavitation in this longitudinal section. This was a small-cell pulmonary carcinoma

**Fig. 4.100** Small-cell carcinoma rapidly disseminates to local nodes and the mediastinum, in this view compressing the pulmonary vasculature. The tumour has surrounded and overrun a lymph node

**Fig. 4.101** A primary lung cancer is seen, with a relatively rounded periphery and a beige/black cut surface. The tumour crosses the interlobar fissure. It is often tempting to speculate as to the histological subtype (in this case, squamous carcinoma), but histology is required for confirmation

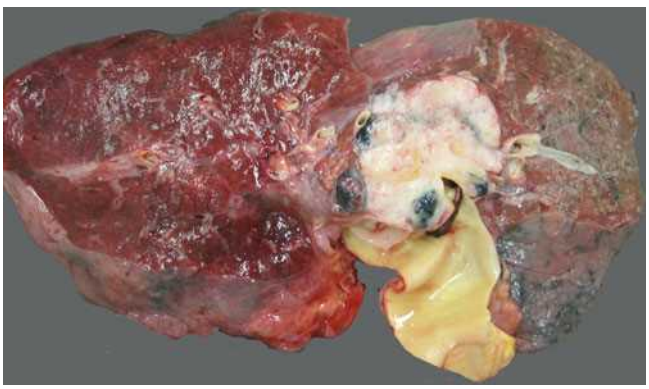

**Fig. 4.102** The lung tissues have a central grey/cream tumour, clearly directly infiltrating the local nodes and interacting with the pulmonary vasculature in a case of primary squamous carcinoma of the lung

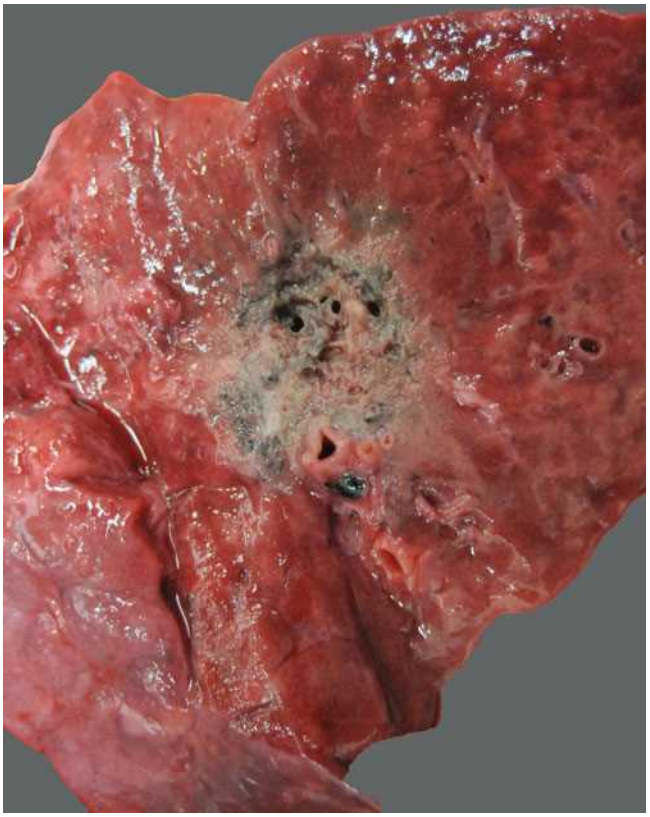

**Fig. 4.103** A pulmonary adenocarcinoma is seen with some central degeneration and an irregular/infiltrating peripheral boundary. Local congestion and pneumonic change is present. Some central cystic degeneration is noted

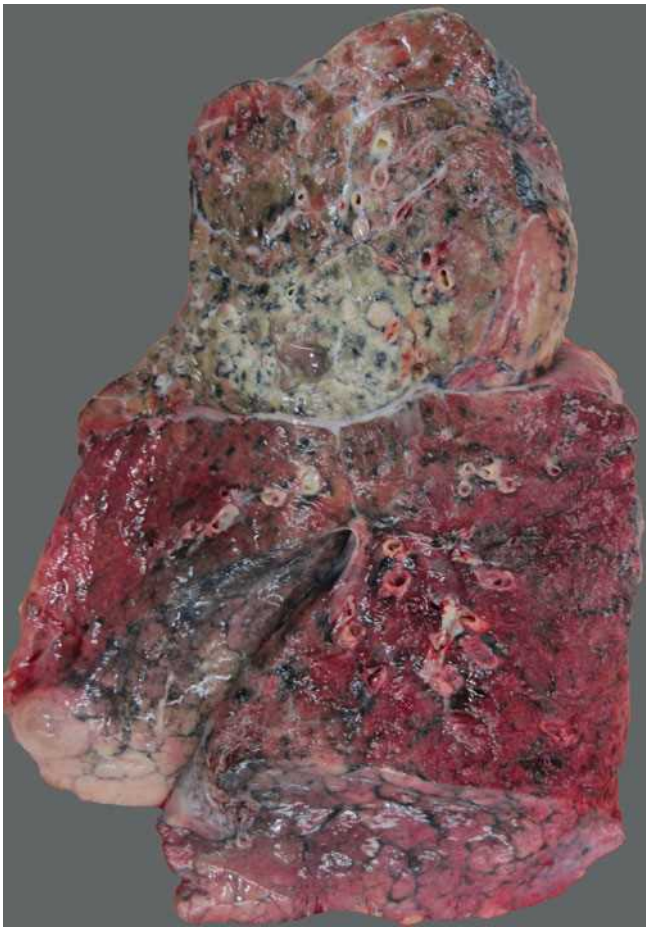

**Fig. 4.104** Primary pulmonary adenocarcinoma is seen with infiltration of the interlobar fissure and upper lobe obstructive pneumonia

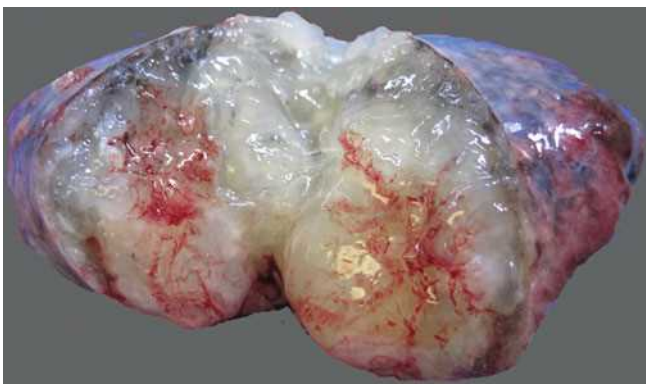

**Fig. 4.105** The cut surface of some tumours is characteristic, such as this extensive mucoid matrix in a case of mucinous adenocarcinoma

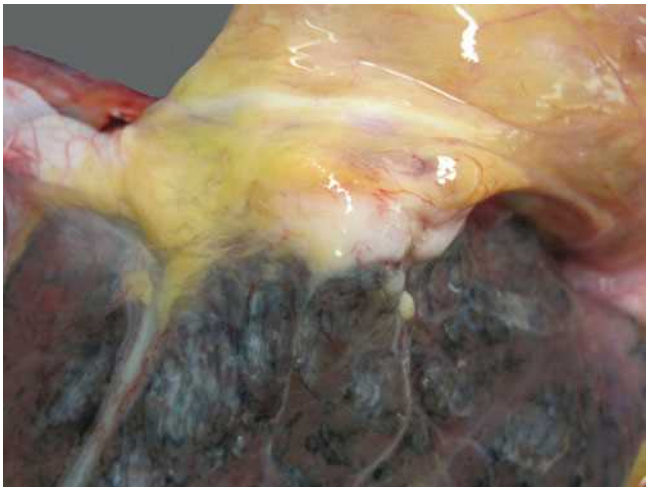

**Fig. 4.106** Metastatic tumour is seen in the soft tissues around the hilar root at autopsy. Some grey discolouration of the surface pleural parenchyma is adjacent, representing lymphangitis

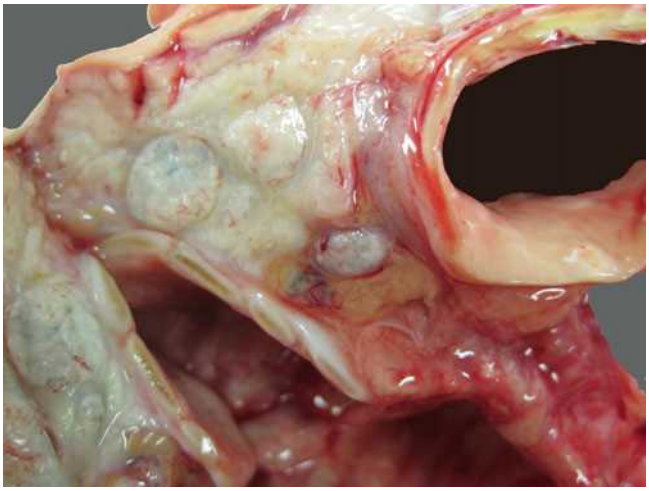

**Fig. 4.108** A close-up view of the mediastinal content shows cream tumour widely occupying the mediastinal soft tissues and directly replacing local lymph nodes

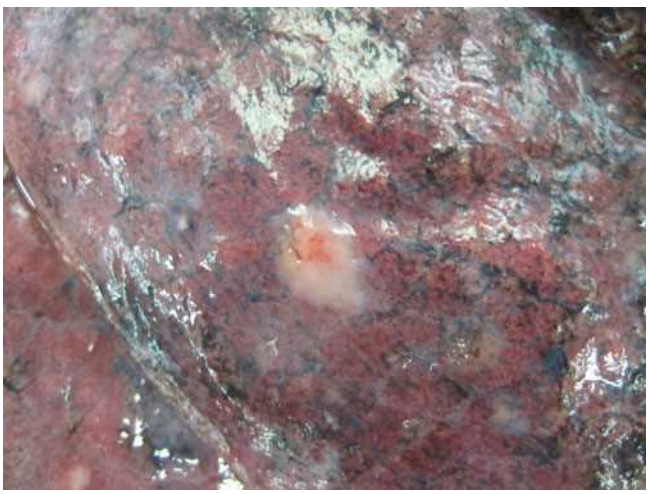

**Fig. 4.107** The pleural surface is seen to be studded by small plaques of beige tissue with a somewhat lacy network of similar tissue running around the surface. This is the characteristic appearance of lymphangitis carcinomatosa involving the pleural surface

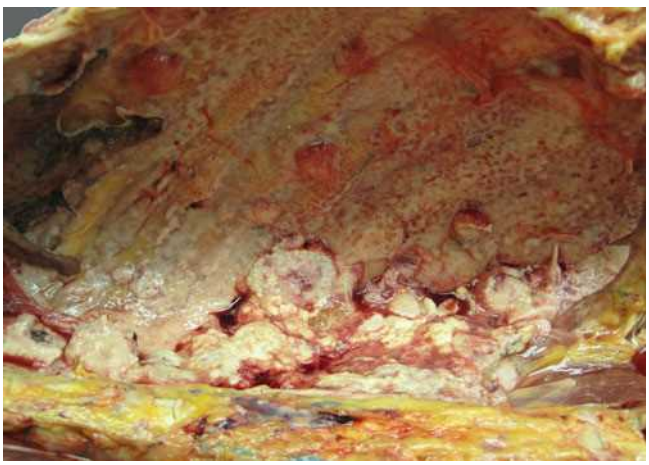

**Fig. 4.109** Some lung cancers spread around the chest cavity, mimicking mesothelioma. This was a nonkeratinising squamous carcinoma. The tumour is seen coating the parietal pleural tissues with nodularity

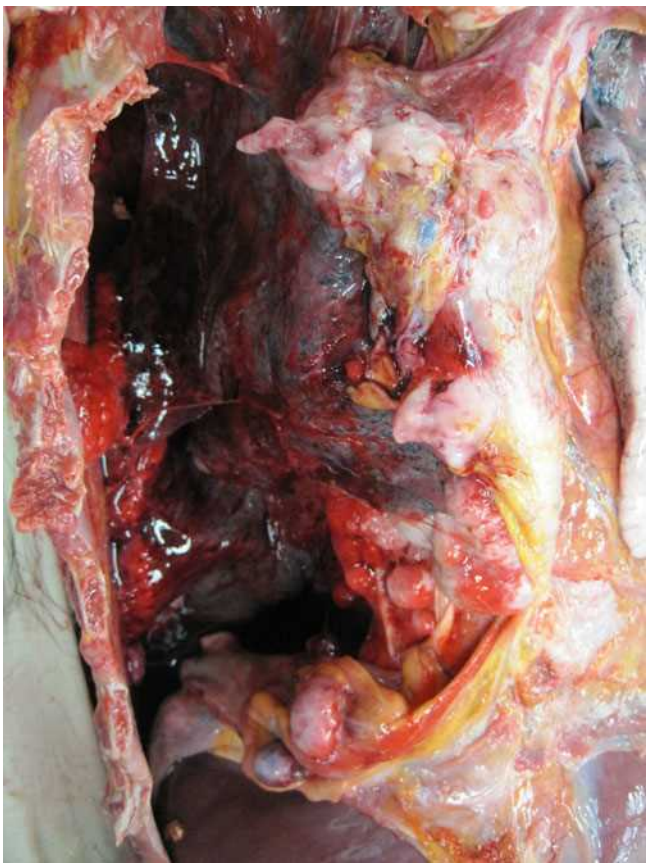

**Fig. 4.110** Disseminated sarcomatoid neoplasia can be seen in a variety of forms, often mimicking mesothelioma. The right hemithorax, in a case of spindle cell carcinoma, here shows widespread nodularity and distortion with some local haemorrhage

**Fig. 4.111** The inner aspect of the hemithorax should always be considered, after removal of the heart, lungs, and mediastinal content. This chest wall shows the normal association of fat in relation to the musculature and ribs, with some anthracotic dust pigmentation (in this case reflecting coal dust exposure). The pleural surface is noted to be smooth, glistening, and without any fluid accumulation

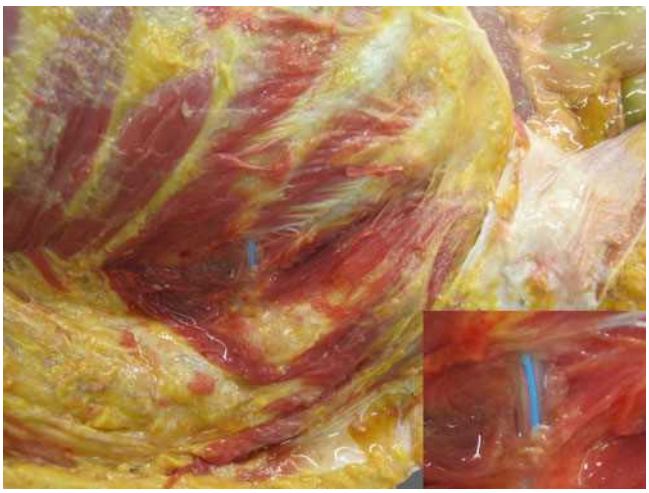

**Fig. 4.112** In cases of chest disease with drains, assessment of the body requires identification of the drainage tube and assessment of its position within the hemithorax. Drains should not be pulled from the body until the assessment has taken place. In many cases, the drains are cut flush with the skin, and then pushed a small distance inwards. This allows dissection of the skin as soft tissues around the chest in the normal manner and preservation of the tube position. Here a chest drain is seen in the low lateral thorax, having been placed under radiology guidance. The *low right inset* shows higher magnification. The drain is highlighted by a *bright blue band* within the plastic tube

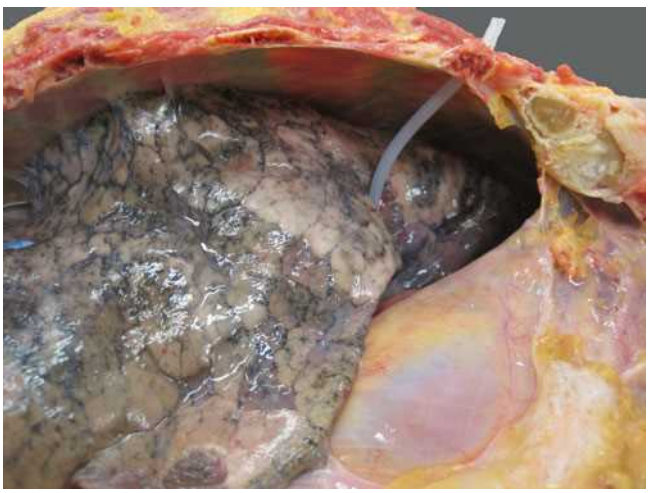

**Fig. 4.113** A correctly positioned chest drain here is seen to push into the chest wall and lie in the interlobar fissure rather than on the periphery of the lung tissues. Local adhesions are present, binding the visceral and parietal pleural tissues together

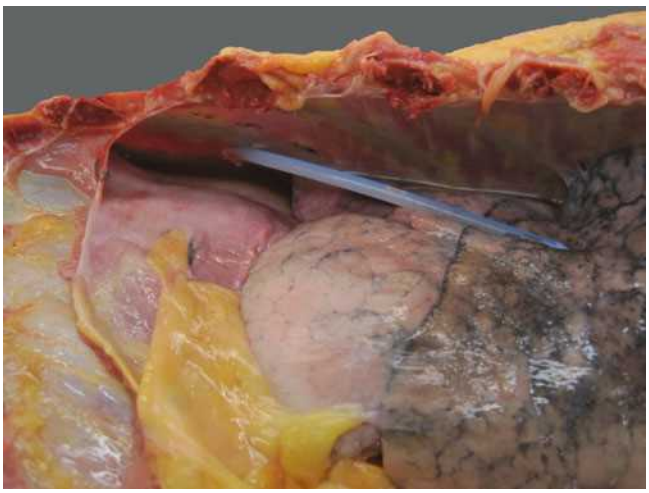

**Fig. 4.114** A correctly positioned chest drainage tube is seen entering the low thorax and running up onto the outside aspect of the lung tissues. This targeted chest drainage was placed under radiological observation. Some fluid is still present behind the drain

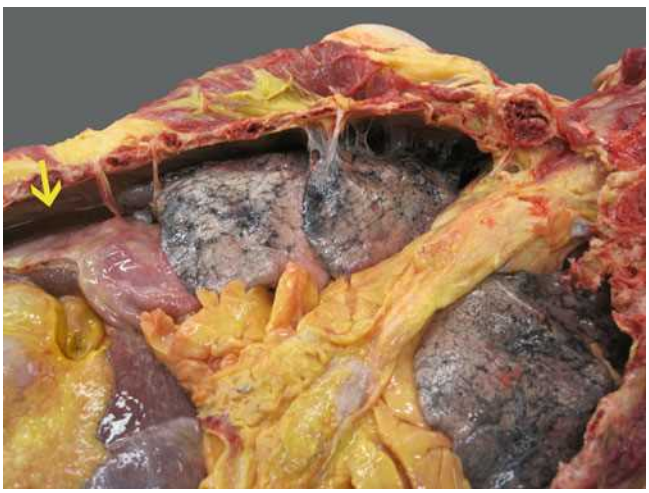

**Fig. 4.115** After removal of the chest plate, adhesions and fluid collections should be assessed. It is possible to culture fluid collections if present, even small effusions (*arrow*), but some caution is needed, as post-mortem contamination of static fluids is not uncommon

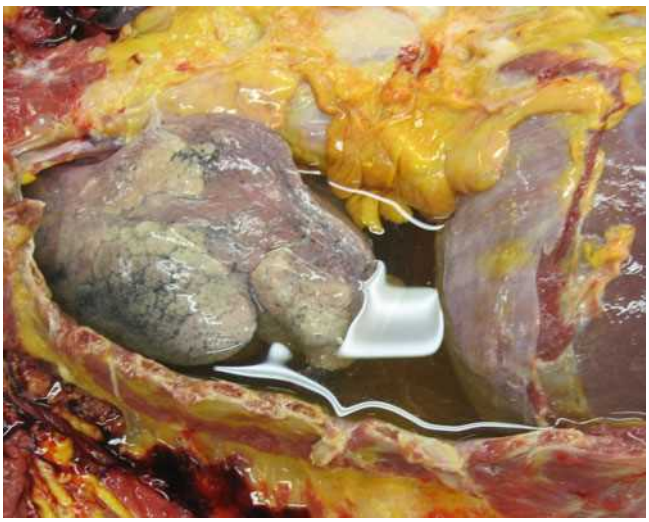

**Fig. 4.116** Large pleural effusions often have a physiological impact in life. Their volume and character must be considered. Inspection of any fluid collection requires assessment of both the volume of fluid and the nature of the collection. Such collections should be described in general terms (*e.g.*, clear yellow fluid, turbid brown fluid)

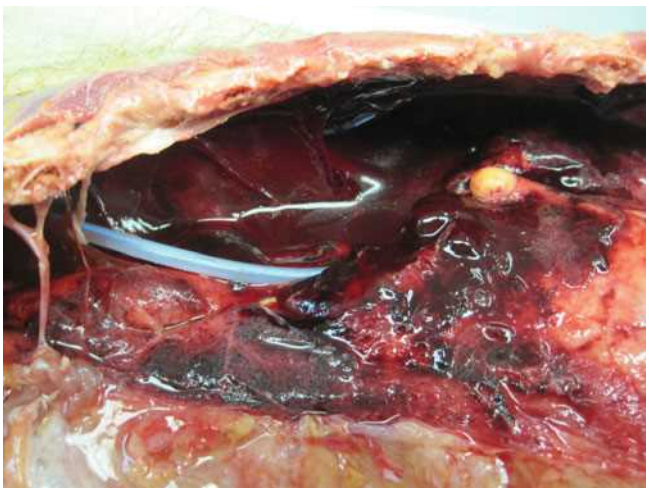

**Fig. 4.117** Any chest drain device inserted into the thoracic cavity should always be assessed for position and for complications. In this case, massive haemorrhage was incurred due to direct lung trauma

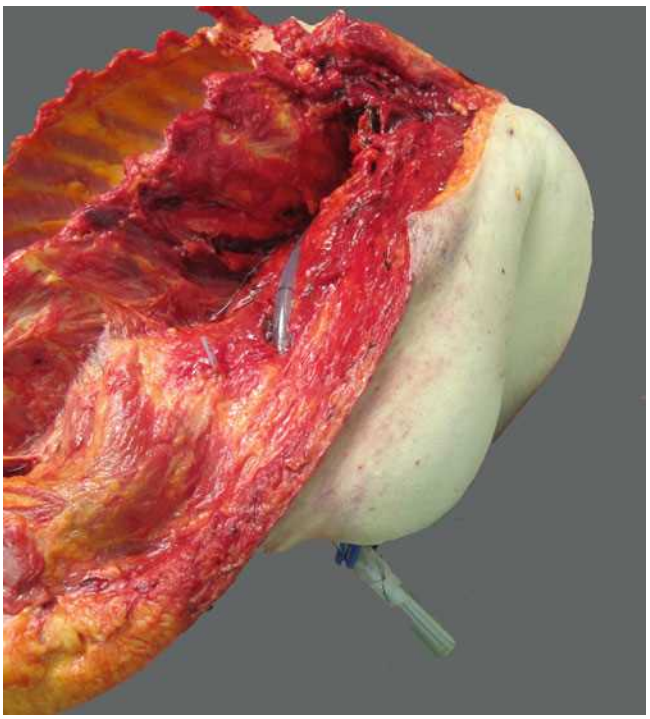

**Fig. 4.118** This chest drain did not enter the pleural space, having been placed into the chest/axillary soft tissues. It clearly would not have improved the patient's situation and may be a factor in the cause of death

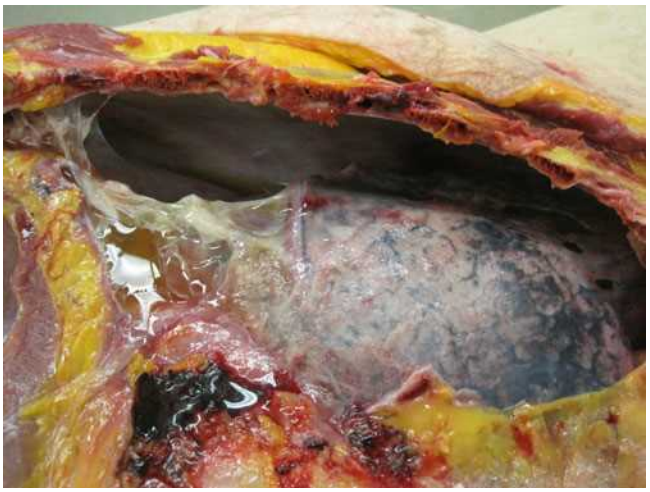

**Fig. 4.119** Lung adhesions to the chest wall can become loculated. The encysted pools of effusion fluid potentially provide an ideal septic focus. Assessment of adhesions is important in cases of lung disease. Adhesions may reflect both reactive/reparative phenomena and tumour infiltration of the pleural cavity. The fibrous adhesions and some fluid in this case reflected persistent chest sepsis

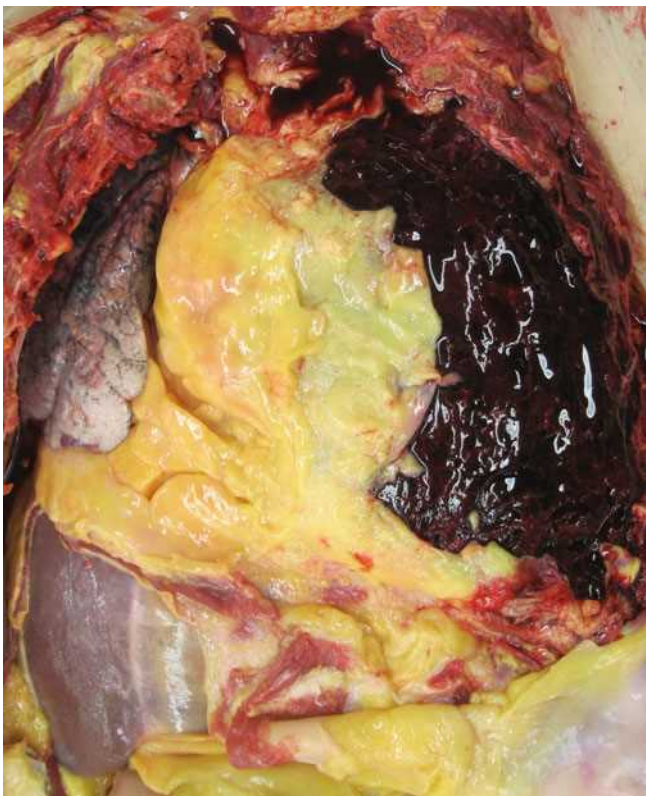

**Fig. 4.120** Haemorrhage into the pleural cavity may also occur from tumours. The necrosis of a tumour involving the pleural cavity, in a patient with anticoagulant treatment, has resulted in a massive haemothorax, which was pertinent to the death of this individual

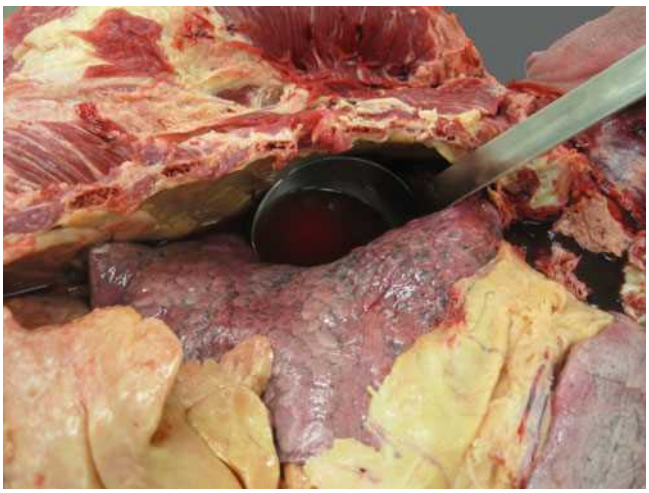

**Fig. 4.121** Fluid collections can be assessed by ladling out the content into measuring jugs or by direct mechanical aspiration into fluid drainage devices

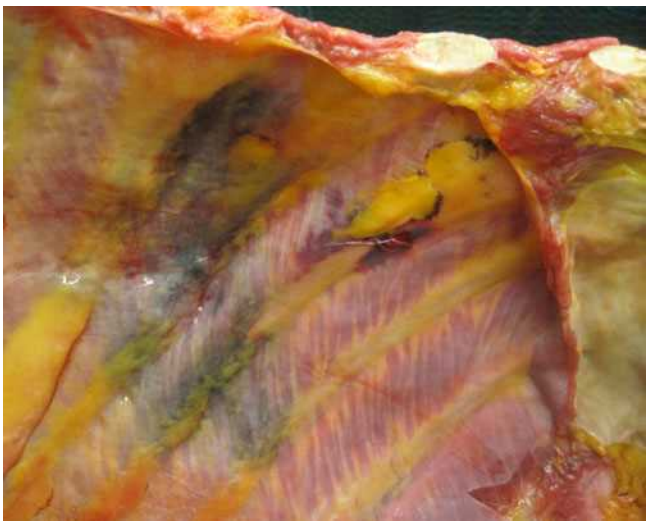

**Fig. 4.122** Undisplaced fractures of the ribs are often apparent with local bruising, even if there is no penetration of the pleural cavity by the fracture itself

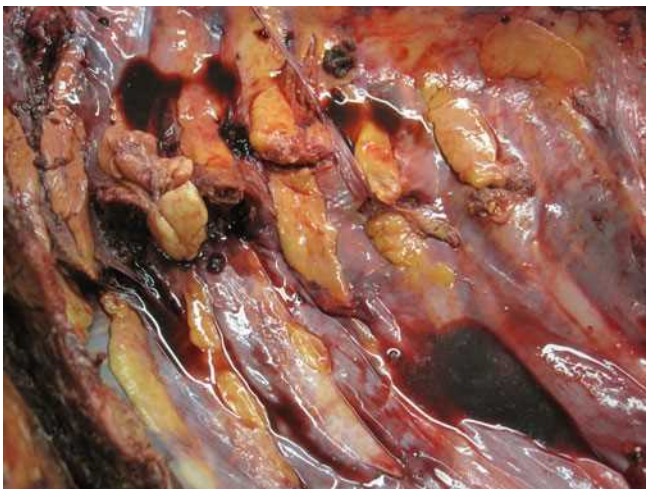

**Fig. 4.123** Multiple fractures are seen in this chest, with clear evidence of local haemorrhage into the soft tissues and pleural compartment. If the ribs are broken on both sides, then the chest wall integrity is lost and it becomes a 'flail' chest

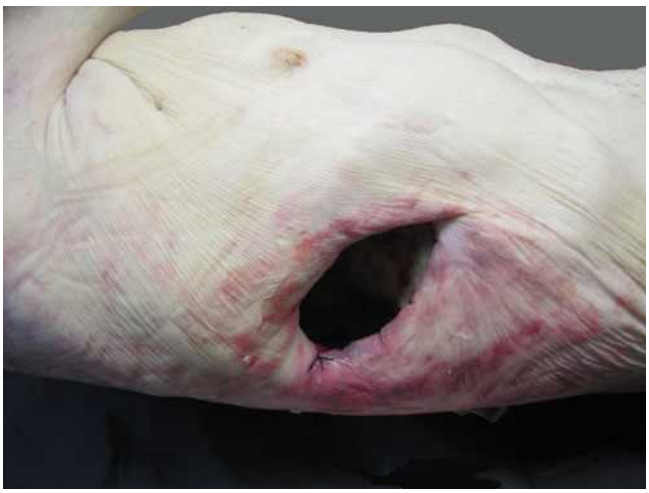

**Fig. 4.124** This large thoracostomy was required for chronic sepsis following pneumonectomy. The persistent production and accumulation of septic material in the right hemithorax required a large drainage route. The object of such surgery is to permit complete drainage and treatment of any septic focus, with subsequent chest closure (if possible)

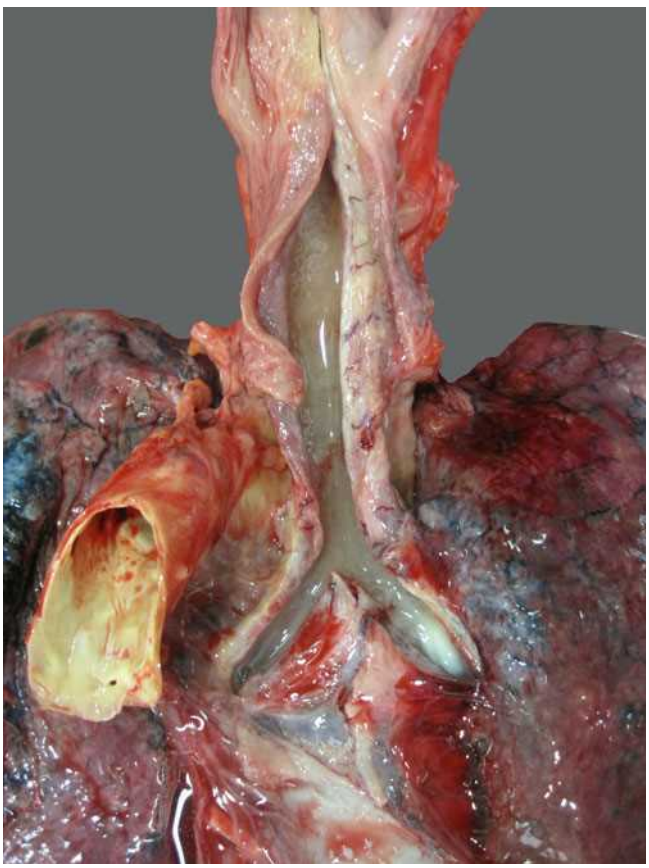

**Fig. 4.125** Significant allergic reactions (anaphylactic shock) are classically associated with extensive tenacious mucoid secretions within the airways. These are best appreciated early in the thoracic dissection, as the trachea is opened with the cut extended towards the lobar bronchi. This is not to be confused with bronchopneumonia

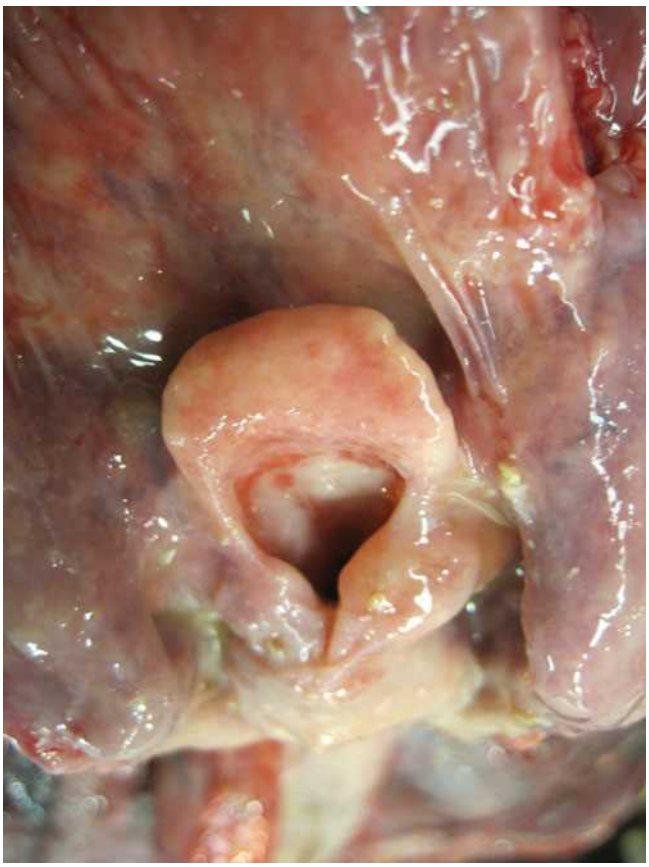

**Fig. 4.126** Acute anaphylaxis is seen with laryngeal oedema in a case of drug-related allergy. The airway was closed, causing the death of this patient in a case of penicillin allergy

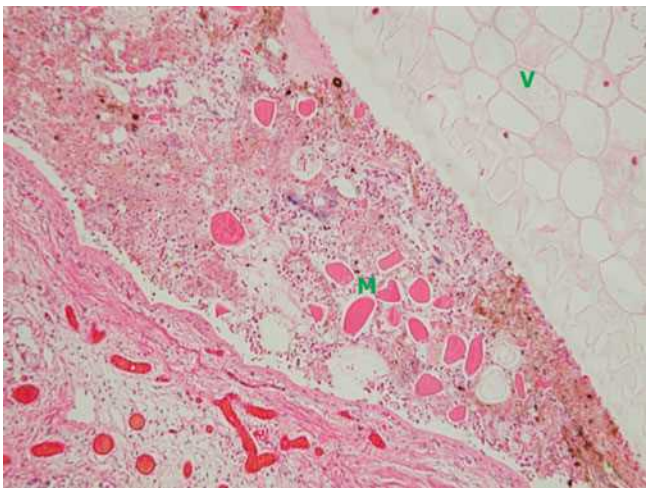


**Fig. 4.127** Aspirated material is often best appreciated in the small airways by histology. Small fragments of meat (*M*) are present within irregular mucoid and degenerate matrix. Some vegetable (*V*) material is seen adjacent

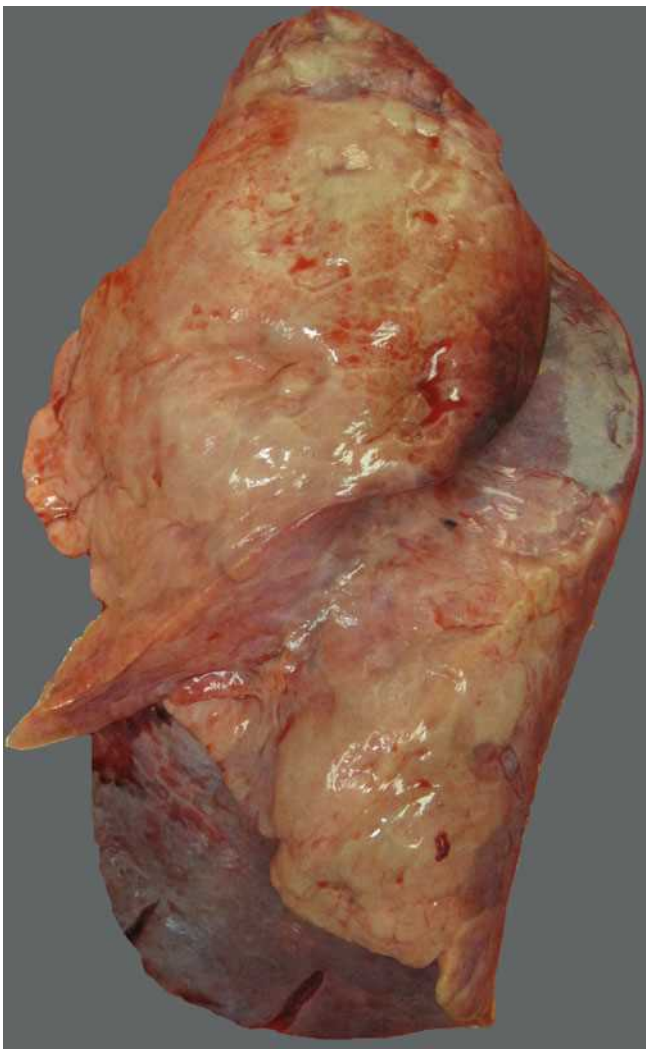

**Fig. 4.128** In a case of acute asthma, hyperinflated lungs are often seen, remaining in their inflated form even after separation from the mediastinal content

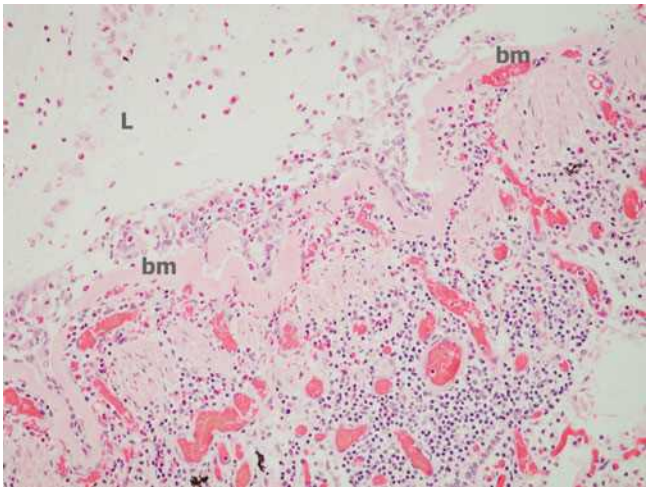


**Fig. 4.129** Histological examination in a case of allergic lung reactions shows epithelial denudation with inflammatory cells and degenerate cellular material being seen within the bronchial lumen (*L*). The basement membrane (*bm*) is slightly corrugated but is clearly thickened and eosinophilic. The adjacent connective tissue shows pronounced vascular engorgement with a moderate inflammatory cell component containing many eosinophils

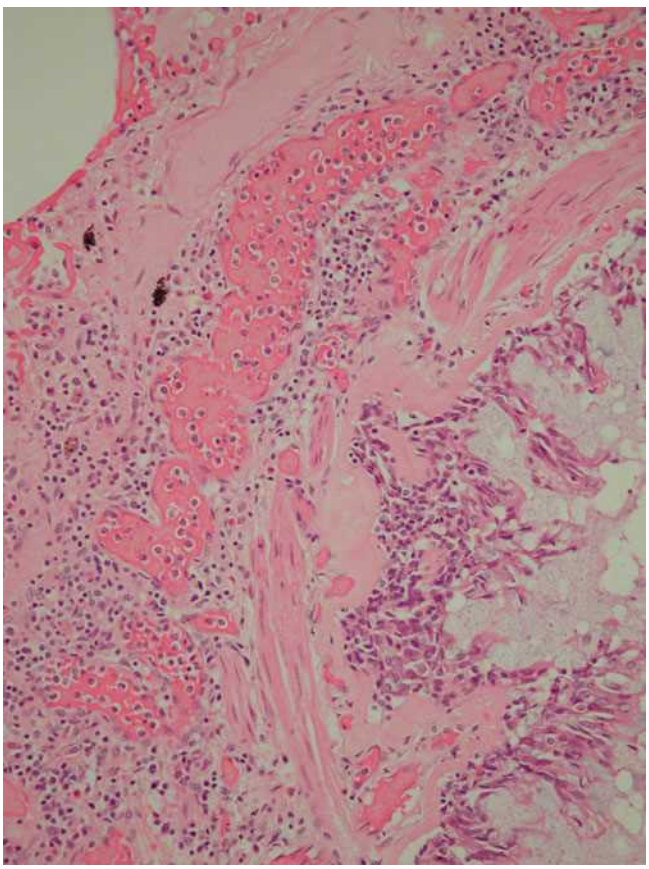

**Fig. 4.130** The proximal airways in asthma cases often show hyperplasia of the epithelium with excess mucus secretion. There is widespread local inflammation with many eosinophils

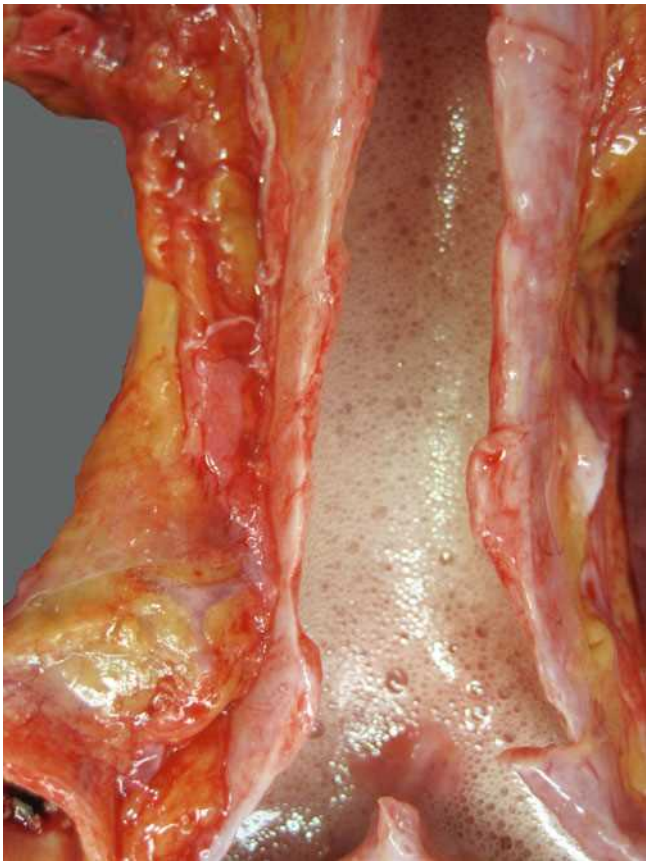

**Fig. 4.131** In drowning, a plume of frothy material often extends from the mouth and nose, but this may be lost by the time of the autopsy. Foamy fluid can also be seen within the trachea, supporting this interpretation of drowning

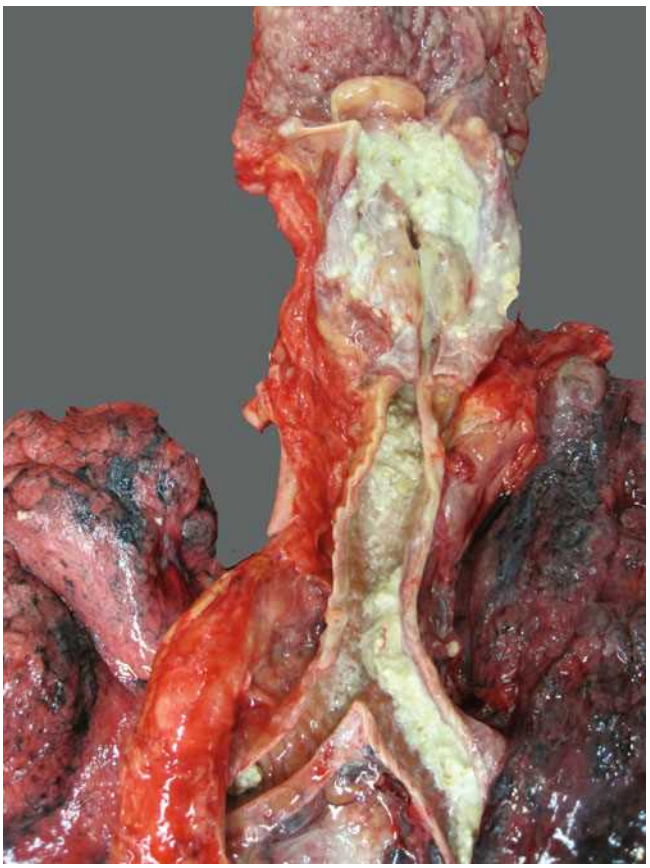

**Fig. 4.132** This thoracic dissection, opening the trachea and bronchi as well as the larynx, shows a case of massive food aspiration, clearly occluding the proximal airway and causing death. Photographic evidence is of particular use in such cases with a medicolegal reality

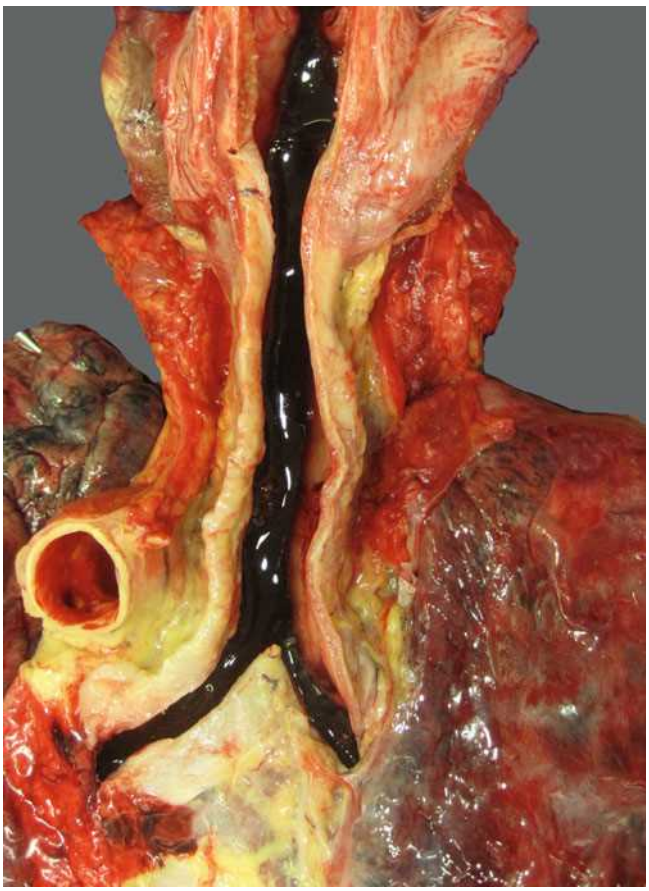

**Fig. 4.134** Massive haemorrhage into the large airways has occluded the trachea, in a case of carcinomatous intrapulmonary haemorrhage due to bronchial artery rupture directly into the airway of one lobe

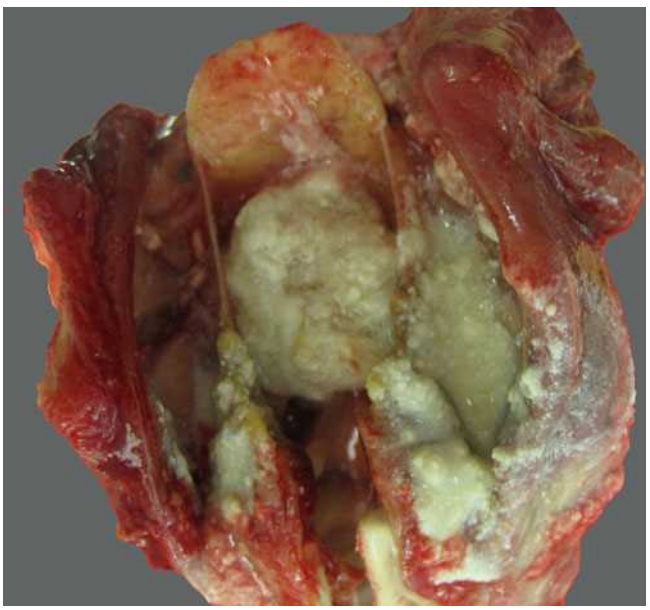

**Fig. 4.133** A close-up view of the larynx with food material blocking the airway

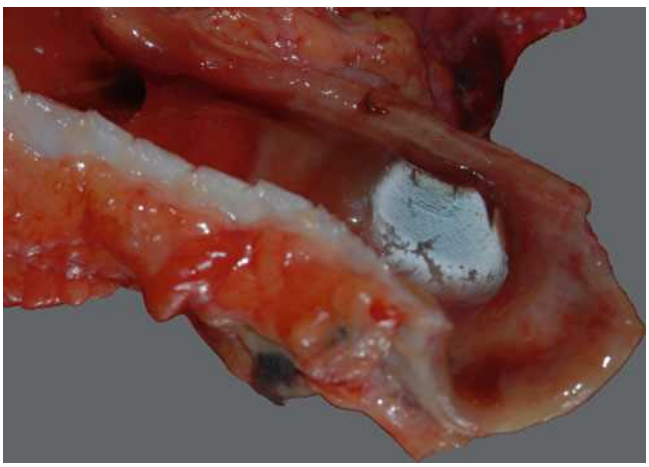

**Fig. 4.135** An incidental, nonpathological feature seen in some autopsy cases is post-mortem growth of a fungal colony within the airways

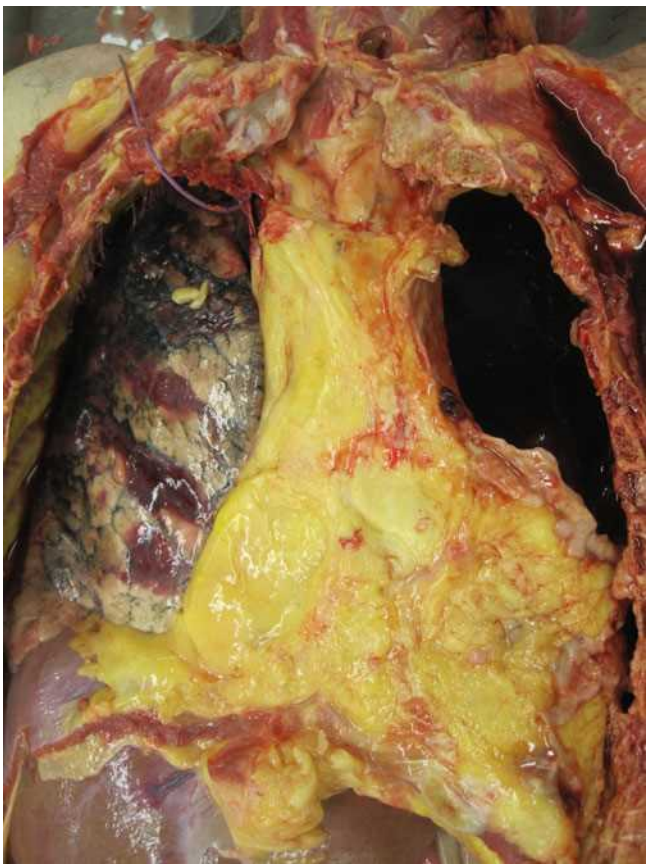

**Fig. 4.136** Pneumonectomy, with complete removal of the tissues of the whole lung, leaves a potential space in the hemithorax. Part of this space is filled by expansion of the other lung and movement of the mediastinal content, but the chest space initially is normally occupied by fluid, with subsequent organization. This person died shortly after the surgical procedure, with evidence of significant haemorrhage into the chest space. This case involved systemic sepsis following the pneumonectomy

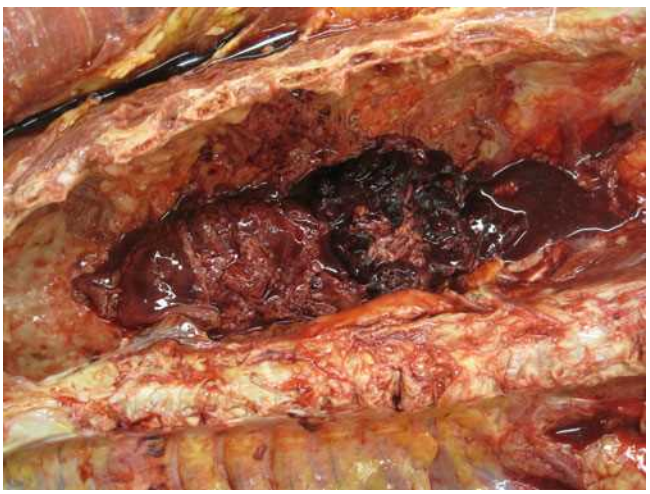

**Fig. 4.137** Later stages of pneumonectomy (without sepsis) involve the organization of the proteinaceous and blood-stained material. A protein coagulum is seen on the posterior aspect of the chest. Histologically, this characteristically has a moderate mixed inflammatory infiltrate within it, although one can check for recurrent carcinoma

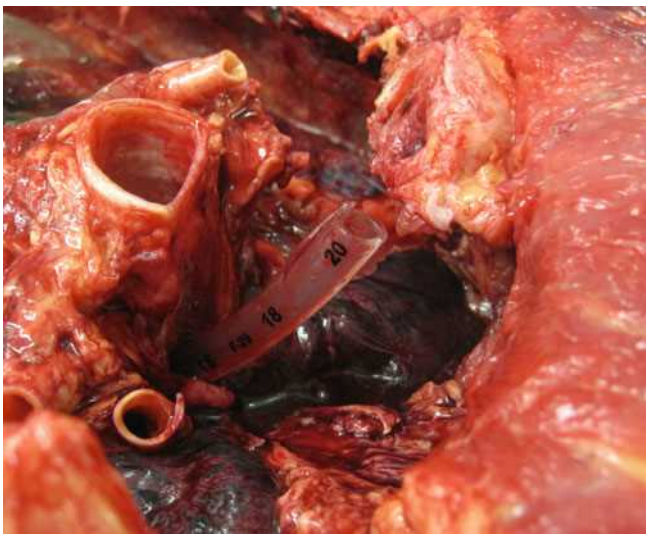


**Fig. 4.138** Assessment of the pulmonary tissues will always involve the position of devices. In this case, the endotracheal tube is incorrectly positioned in the esophagus, behind the trachea. This positioning clearly would have a significant impact on the chances that the patient will survive a cardiorespiratory arrest

## Degenerative Lung Conditions

The chapter deals first with chronic obstructive pulmonary disease, a group that includes asthma, bronchiectasis, chronic bronchitis, and emphysema in variable format. Both macroscopic and histological assessment may be pertinent in fully appreciating the relevance to the cause of death.

The lung can be also involved by diffuse fibrosing processes of primary or secondary type. Many of these conditions have an autoimmune background, although dust-related diseases (pneumoconiosis) also are important, particularly with exposure to coal dust, silica, or asbestos.

## Thoracic Neoplasia

Lung cancer is a major cause of morbidity and mortality, with these tumours being directly involved in the cause of death in many individuals. Secondary effects from chronic sepsis in the lungs are also well recognized, with disseminated disease being particularly important. Lastly, issues with regard to the pleura are important.

## Gross Cardiac Pathology

The cardiac tissues are best assessed in terms of the gross anatomy, the coronary arteries, the myocardial tissue itself, and the valves. Given the architectural complexity of this dynamic organ, it is vital to examine the tissues in a stepwise fashion, as the disorders liable to be seen in the autopsy room merit judgement in turn. This order guides the observer to congenital lesions, degenerative and inflammatory disorders, infections and inflammation, and the cardiomyopathies (Figs. 4.139, 4.140, 4.141, 4.142, 4.143, 4.144, 4.145, 4.146, 4.147, 4.148, 4.149, 4.150, 4.151, 4.152, 4.153, 4.154, 4.155, 4.156, 4.157, 4.158, 4.159, 4.160, 4.161, 4.162, 4.163, 4.164, 4.165, 4.166, 4.167, 4.168, 4.169, 4.170, 4.171, 4.172, 4.173, 4.174, 4.175, 4.176, 4.177, 4.178, 4.179, 4.180, 4.181, 4.182, 4.183, 4.184, 4.185, 4.186, 4.187, 4.188, 4.189, 4.190, 4.191, 4.192, 4.193, 4.194, 4.195, 4.196, 4.197, 4.198, 4.199, 4.200, 4.201, 4.202, 4.203, 4.204, 4.205, 4.206, 4.207, 4.208, 4.209, 4.210, 4.211, 4.212, 4.213, 4.214, 4.215, 4.216, 4.217, 4.218, 4.219, 4.220, 4.221, 4.222, 4.223, 4.224, 4.225, 4.226, 4.227, 4.228, 4.229, 4.230, 4.231, 4.232, 4.233, 4.234, 4.235, 4.236, 4.237, 4.238, 4.239, 4.240, 4.241, 4.242, 4.243, 4.244, 4.245, 4.246, 4.247, 4.248, 4.249, 4.250, 4.251, 4.252, 4.253, 4.254, 4.255, 4.256, 4.257, 4.258, 4.259, 4.260, 4.261, 4.262, 4.263, 4.264, 4.265, 4.266, 4.267, 4.268, 4.269, 4.270, 4.271, 4.272, 4.273, 4.274, 4.275, 4.276, 4.277, 4.278, 4.279, 4.280, 4.281, 4.282, 4.283, 4.284, 4.285, 4.286, 4.287, 4.288, 4.289, 4.290, 4.291, 4.292, 4.293, 4.294, 4.295, 4.296, 4.297, 4.298, 4.299, 4.300, 4.301, 4.302, 4.303, 4.304, 4.305, 4.306, 4.307, 4.308, 4.309, 4.310, 4.311, 4.312, 4.313, 4.314, 4.315, and 4.316).

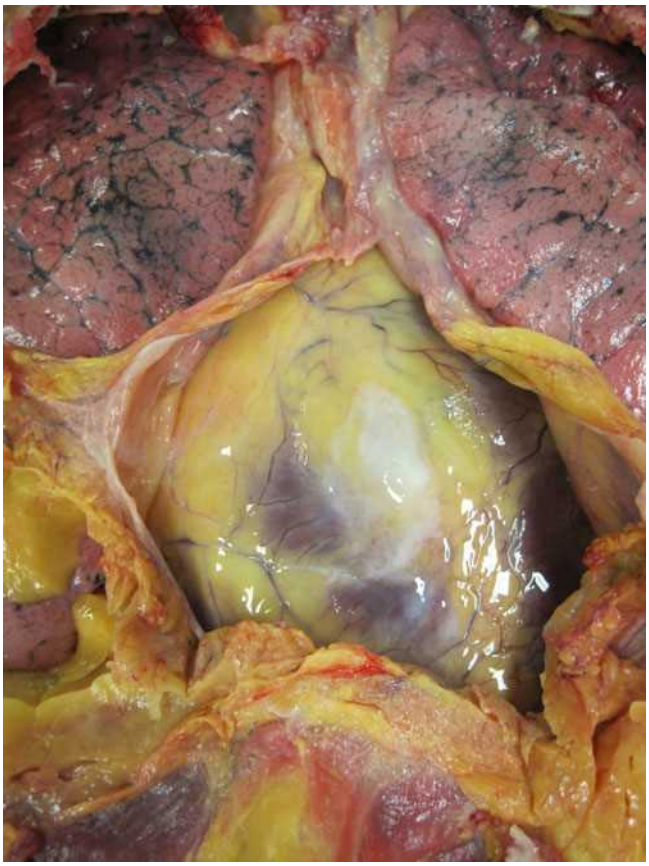

**Fig. 4.139** The cardiac tissues initially may be examined in situ, just after the chest plate has been removed. In this view, the pericardium has been opened, with the local thoracic tissues still in situ. The heart is seen in its normal position, with some fibrous tissue on the anterior epicardial surface. This fibrous tissue plaque is a normal feature, reflecting the epicardial surface rubbing against the inner aspect of the anterior chest wall tissues

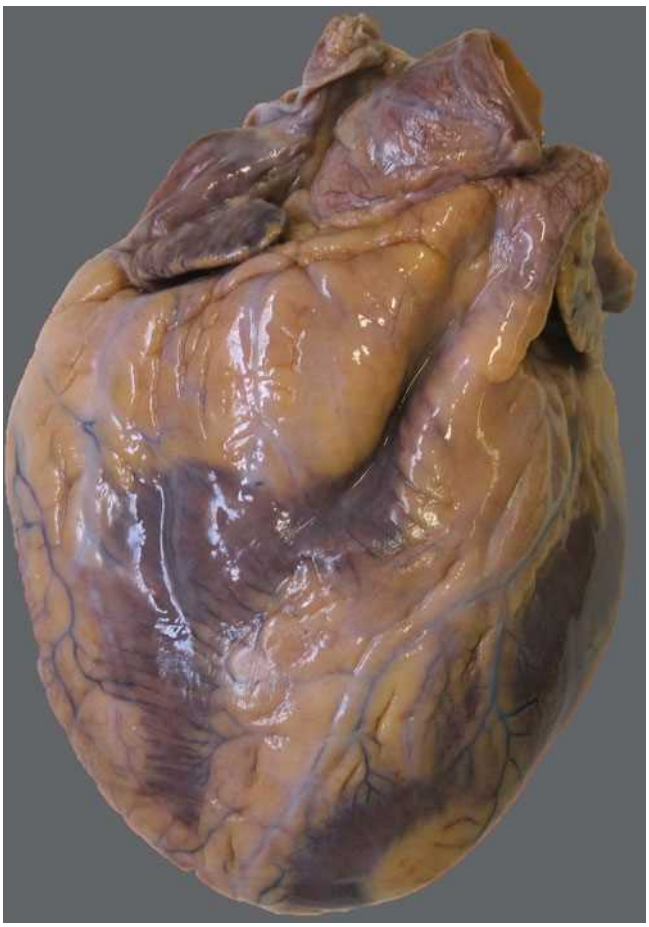

**Fig. 4.140** This adult heart (fixed after extraction in this case) is seen from the anterior aspect. The auricular appendages can be seen, with the normal position of the great vessels. Also seen is the normal coronary tributary pattern and the usual amount of fat adjacent to the main coronary arteries and veins

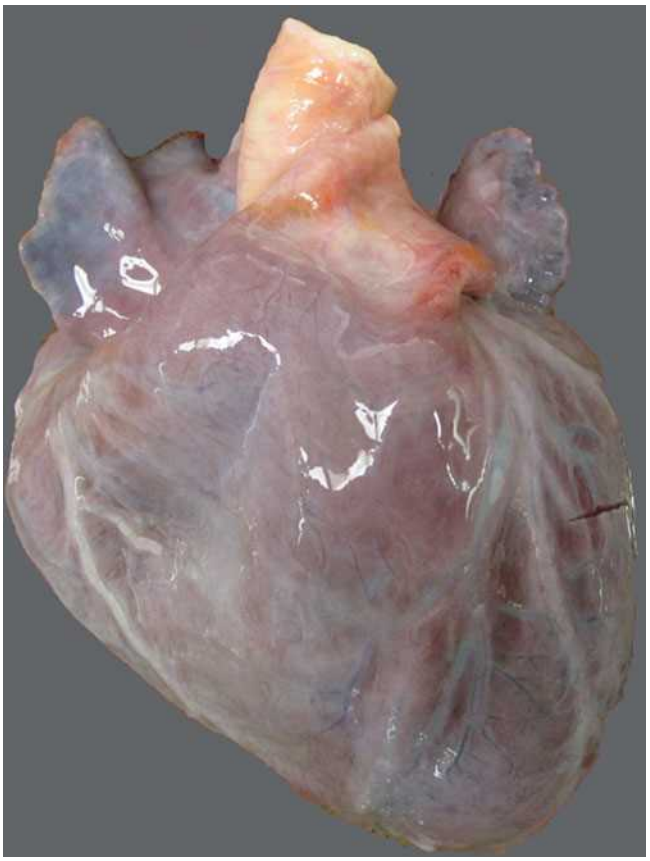

**Fig. 4.141** This adult heart has no appreciable epicardial fat. This is a rare occurrence in Western society. The individual was of light build and was very undernourished. It is unusual to see no fatty tissue around the course of the coronary artery tissues on an adult heart

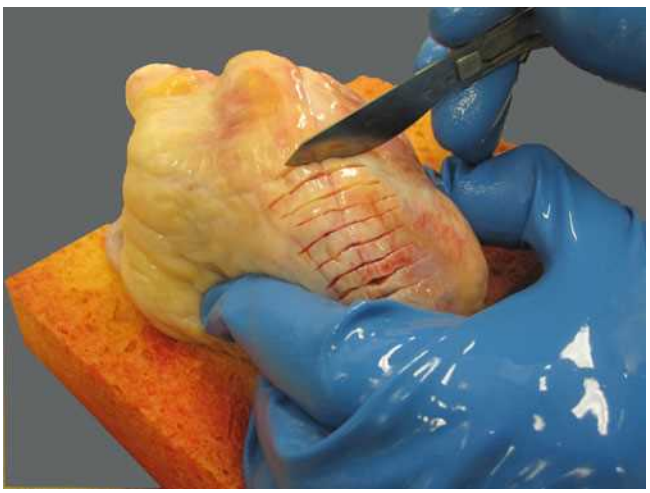

**Fig. 4.142** The coronary arteries may be sectioned at intervals of 3–5 mm, with direct inspection of the lumens. This process is ideally performed with a sharp scalpel blade. Areas of high-grade atheroma, thrombosis, or both may be identified and scored (generally as a percentage occlusion remark, such as 50 % narrowed). The tissues are partly stabilized by holding the tissue on a sponge

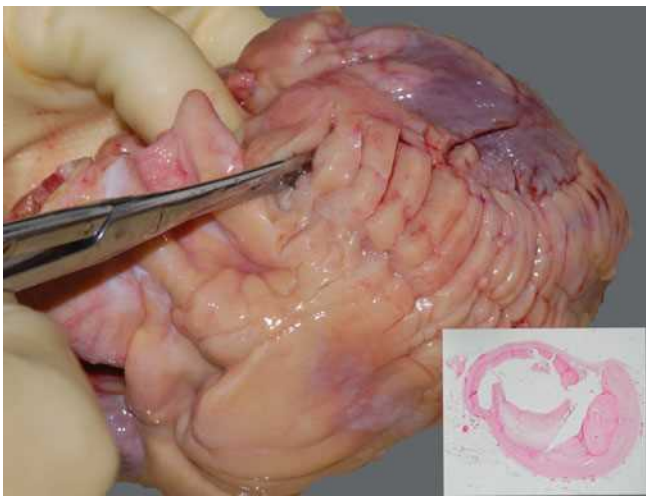

**Fig. 4.143** Heavily calcified coronary arteries are difficult to cut with scalpels. They may be cut with scissors, but one must appreciate that the scissors may partly fragment any calcified plaque disease, as seen in subsequent histology (*inset*)

**Fig. 4.144** Some coronary arteries can be very heavily calcified, significantly hindering examination of the vessels, but if they are dissected intact (en bloc), the arteries can be fixed (48 h) and then decalcified (48 h)

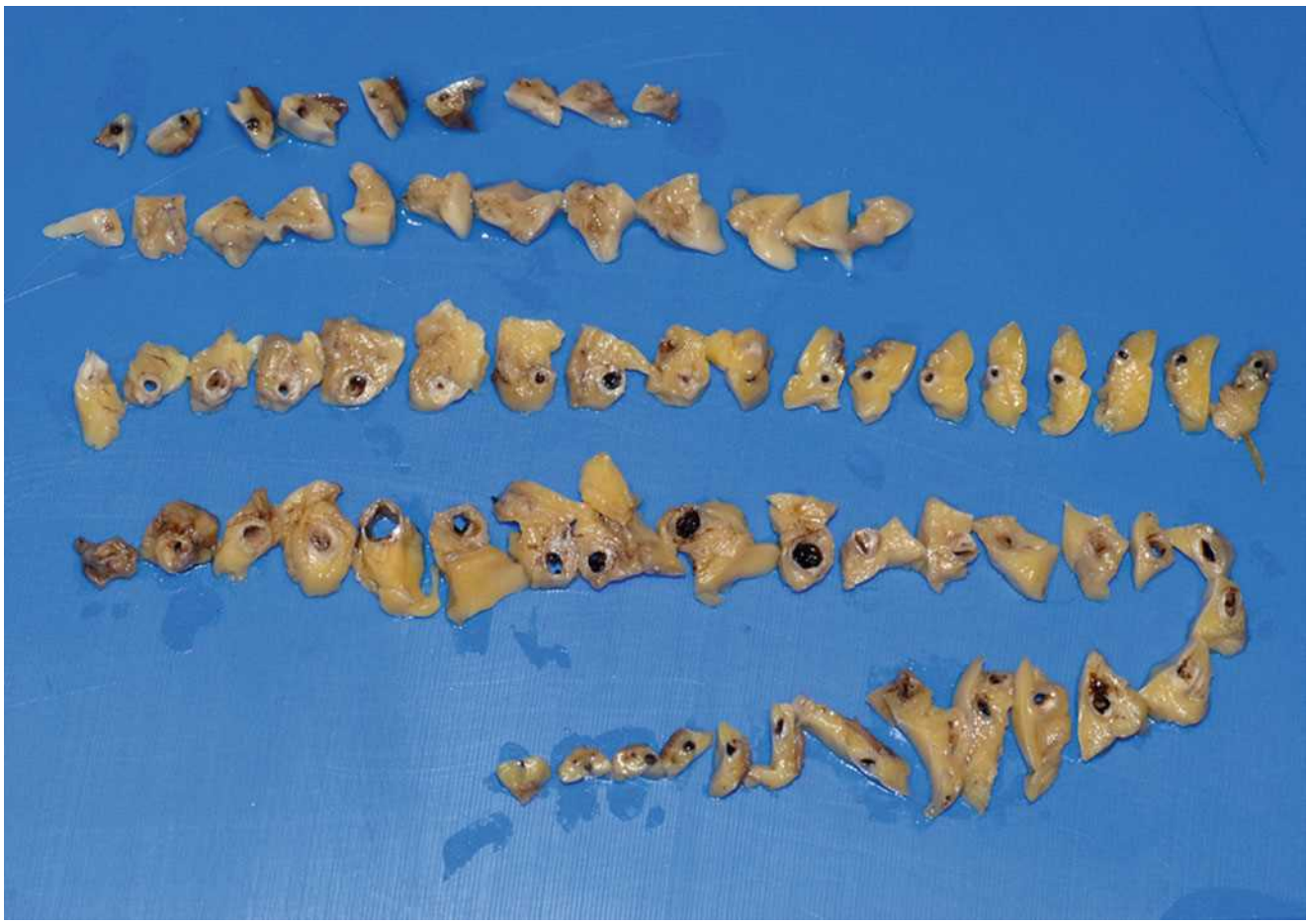

**Fig. 4.145** After decalcification, the three main arteries, as intact vessels, are easy to transversely slice and examine in sequential format. Areas of heavy plaque disease may be selected for photography and/or histology (Adapted with permission from Suvarna SK. National guidelines for adult autopsy cardiac dissection and diagnosis–are they achievable? A personal view. Histopathology. 2008;53:97–112)

**Fig. 4.146** The coronary arteries can be mounted into cassettes in small groups, allowing precise understanding of the degree of atheroma as each slice is a step along the coronary artery course

**Fig. 4.147** The heart (following examination of the coronary arteries) is sectioned across the ventricles (usually in three slices), with the final cut at mid-ventricular level. The *inset* shows the view of the slice of ventricular tissues. The right ventricle is positioned on the left side of the image, with anterior tissues towards the base of the image (From Suvarna (2013))

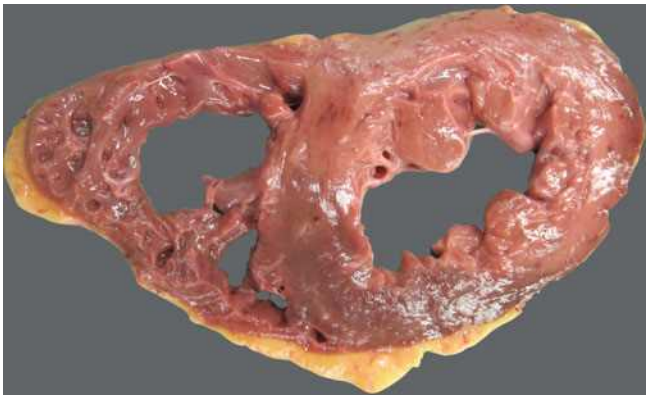

**Fig. 4.148** The transverse sections of myocardium at mid-ventricular level provide information about the right and left ventricle walls and the septum, in relation to the chamber architecture. In this case, the colour of the otherwise normal ventricular tissues mapped to the position in which the individual was found (lying on the front/right side), with hypostasis being more marked in this position

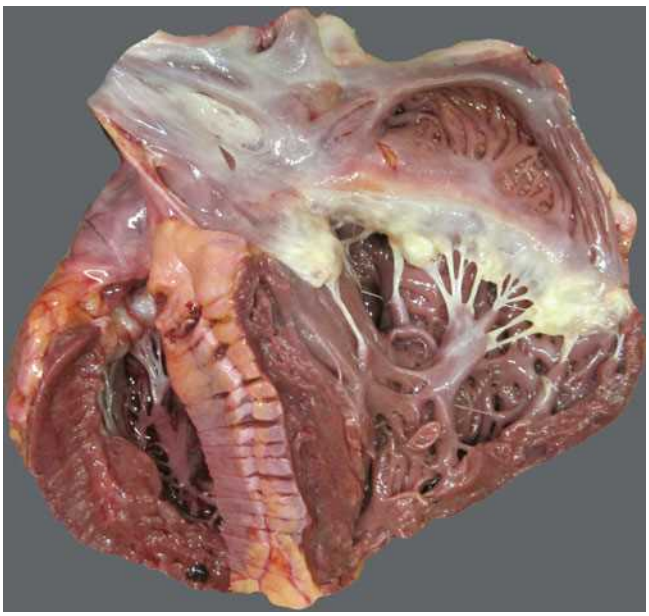

**Fig. 4.149** The right atrium and ventricle in this intact heart have been opened by a cut running along the back of the right atrium (between the superior and inferior vena cava) and then across the tricuspid valve. The cut passes downwards into the right ventricle, about 10 mm to the side of the posterior interventricular artery. The three cusps of the tricuspid valve are seen; the anterior aspect of the atrium/auricle is notably trabeculated. This appendage can be inspected without further dissection. The right ventricle trabeculations are moderately coarse. The papillary muscles are seen with the chordae running upwards to the tricuspid valve tissues

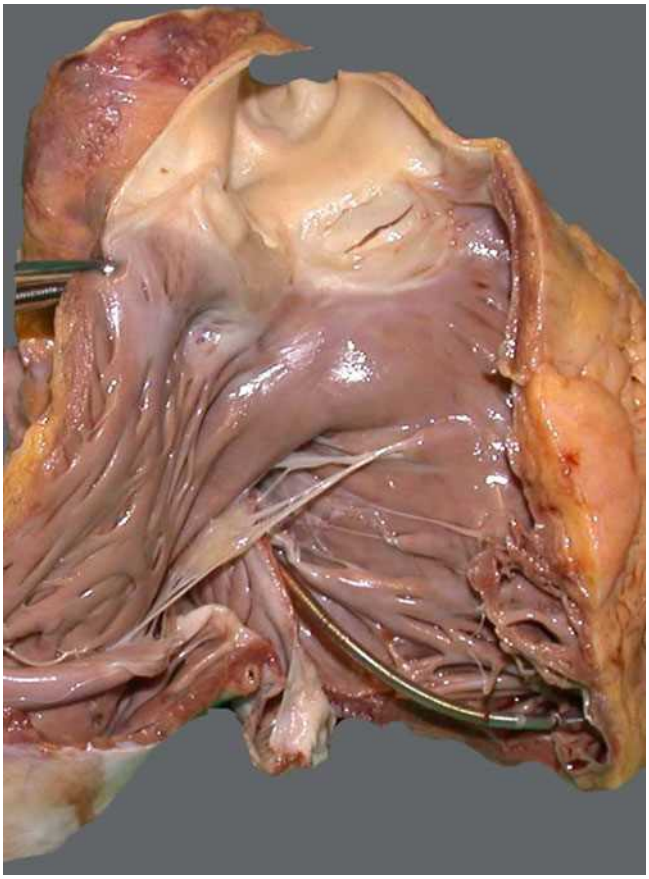

**Fig. 4.150** Turning the heart onto the back allows one to cut along the anterior aspect of the right ventricle, across the pulmonary valve and into the pulmonary artery. This demonstrates the outflow tract of the right ventricle with three cusps of the pulmonary valve showing a normal semilunar architecture. Note the absence of coronary ostia. The outflow tract to the right ventricle is smooth, contrasting with the rest of the right ventricle tissue. An incidental pacemaker lead is seen running to its normal position in the tip of the right ventricle

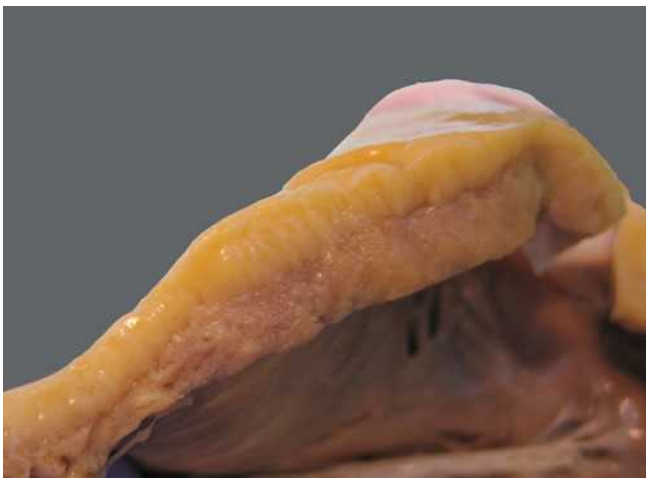

**Fig. 4.151** Fatty tissue on the pericardial aspect of the right ventricle outflow tract is normal, but variable. In this view, the amount of fat is very pronounced, but not associated with wall thinning or overt fibrosis. This is a normal variant, seen in the general population; it is more common in females and the elderly. It should not be mistaken for arrhythmogenic right ventricular cardiomyopathy

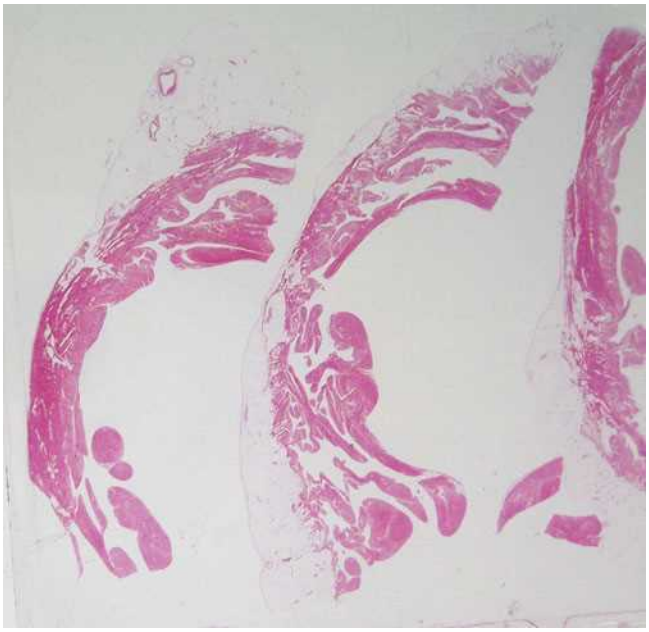

**Fig. 4.152** Histological examination of the right ventricle tissues may confirm the presence of a large amount of fat. Without loss of muscular tissues and fibrosis, this should not be taken as indicating arrhythmogenic right ventricular cardiomyopathy

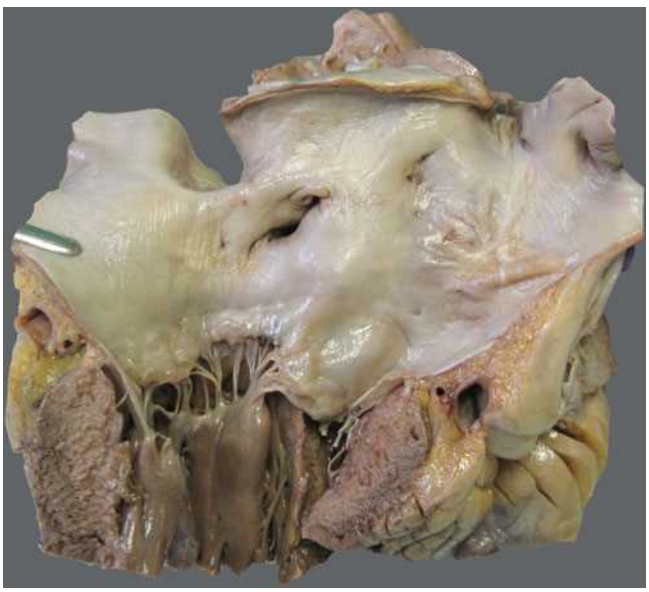

**Fig. 4.153** The left atrial and upper left ventricular tissues opened from the back in this view, with the mid and apical ventricular tissues having been removed beforehand by the three-slice technique. This view requires the orifices of the pulmonary veins to be opened at the atrium roof. Next a cut passes from the right inferior vein orifice along the posterior wall of the left atrium, down across the mitral valve. The cut is extended down the back of the left ventricle (about 10 mm to the side of the posterior interventricular artery). The overall view shows that the left atrium is smooth compared with the right side. The auricle is usually smaller than that on the right side, but it can be inspected directly without further cuts being made. The left atrial view also shows the closed foramen ovale. The twin-leaflet mitral valve is thin, pliable, and attached to thin chordae and the papillary muscles

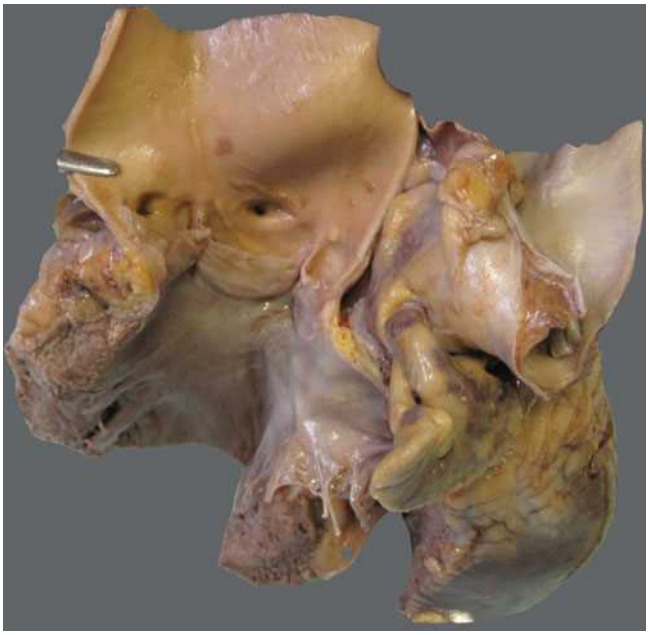

**Fig. 4.154** Turning the heart onto the back allows one to run a cut alongside the course of the left anterior descending artery, up towards the bifurcation of the left main stem, into the left anterior descending and circumflex arteries. At this point, the cut turns inwards and towards the aortic root, passing transversely across the origin of the circumflex artery. In this view, the outflow tract of the left ventricle has been opened across the aortic valve. This shows that two cusps of the aortic valve are associated with coronary artery ostia. The third (noncoronary) cusp is seen to be positioned adjacent. The left ventricular outflow tract is notably smooth, as compared with the remainder of the trabeculated chamber. The left side trabeculations are generally finer than those on the right side. Note that there is no encroachment of the muscular part of the ventricular septum into the outflow canal

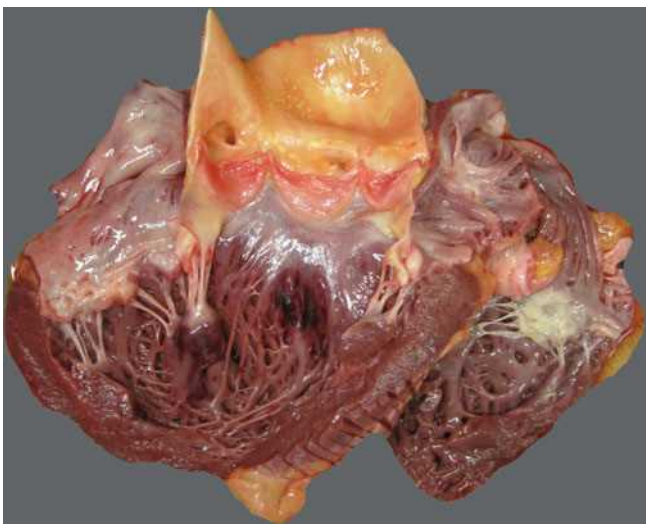

**Fig. 4.155** The alternative opening of the tissues of the left ventricle is with a cut across the mitral valve and thereby into the aortic root. This cut naturally distorts the mitral valve by dividing it into two parts, but it is less complex than the cut passing alongside the left anterior descending artery, as described above. It may have some additional value in simplifying the cuts needed for the Fulton weights assessment (See Figs. 4.274 and 4.275)

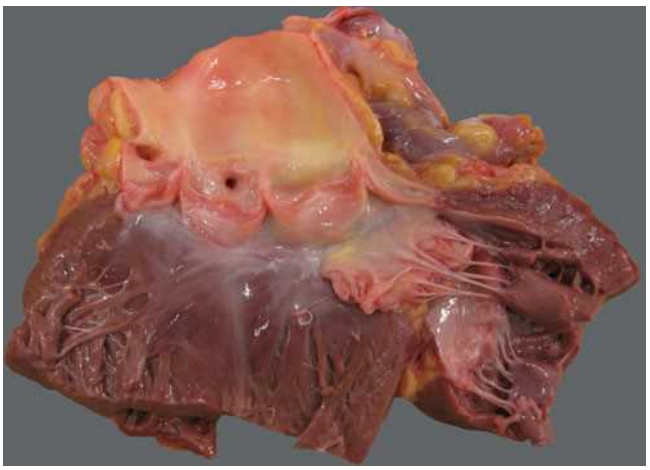

**Fig. 4.156** The aortic root shows the normal coronary artery origins. There are three cusps: the *left* main stem, *right* coronary, and noncoronary cusps (from *left to right*). One should note the close arrangement of the aortic and mitral valve, explaining why disorders of one often affect the other. Note also how the chordae interact with the underside of the mitral valve, not just the edge. The ventricular trabeculations are finer than those of the right ventricle

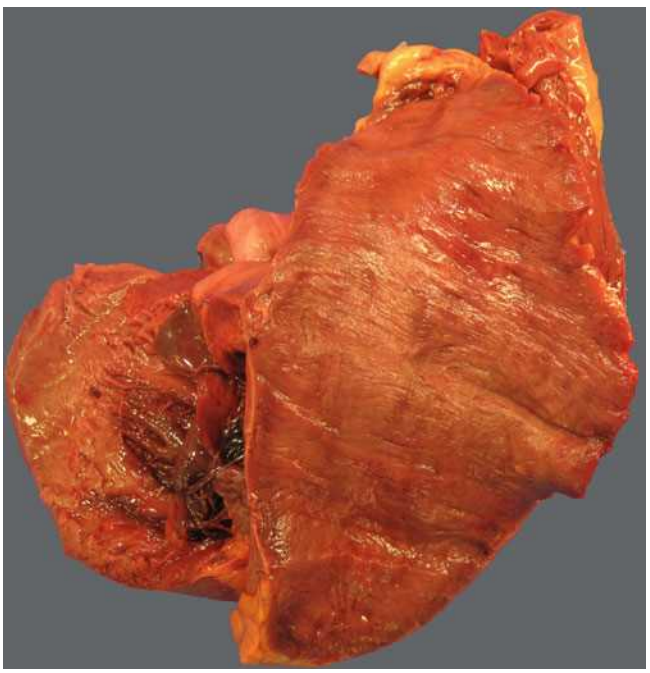

**Fig. 4.157** Some practitioners slice the lateral wall and septum of the left ventricle along the midmural plane. This approach exposes a large amount of muscular tissue but distorts one's appreciation of the relationship of the wall and chambers. It is not recommended

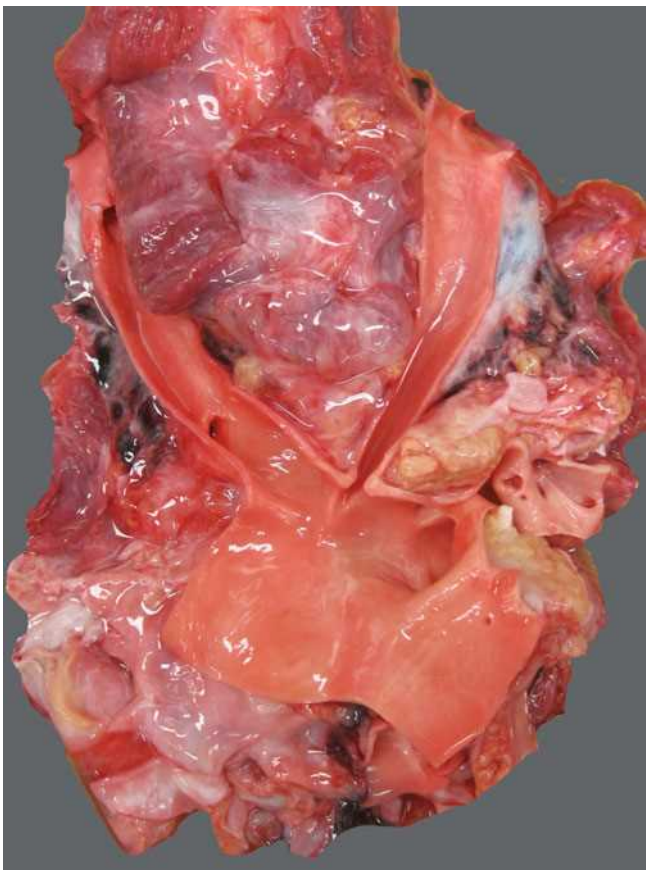

**Fig. 4.158** The aortic arch has been opened, revealing the origin of the innominate artery, branching to the right subclavian and right common carotid artery. Next is the left common carotid artery, and beyond this is the origin of the left subclavian artery. Note the absence of atheroma

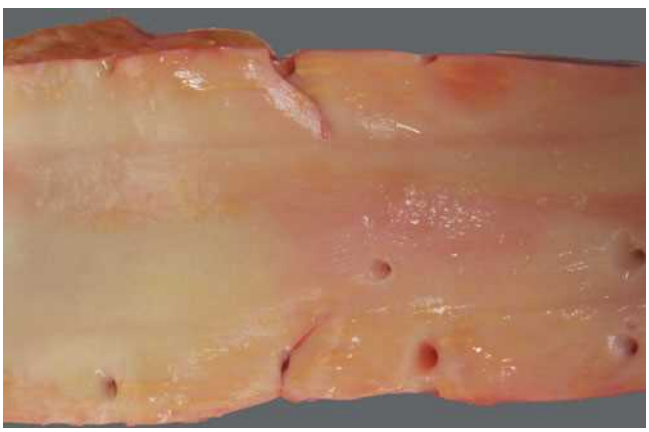

**Fig. 4.159** The normal thoracic aorta is characterized by a smooth, yellow intimal aspect, patent arteries, and the absence of atheroma, dissection, or ulceration. This intimal appearance is normal for all major arteries

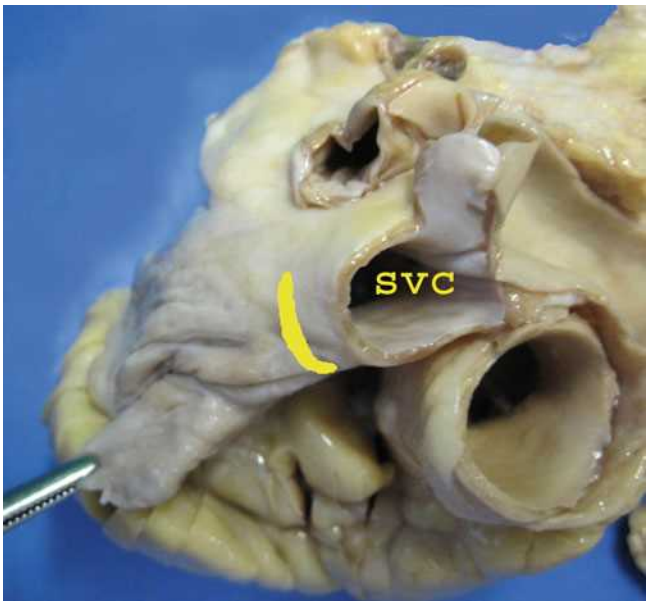


**Fig. 4.160** The sinoatrial node is not apparent by naked eye inspection. With the heart seen from above the right atrium, its position (*yellow*) is found at the tip of the right atrium adjacent to the superior vena cava (SVC), (From Suvarna (2013))

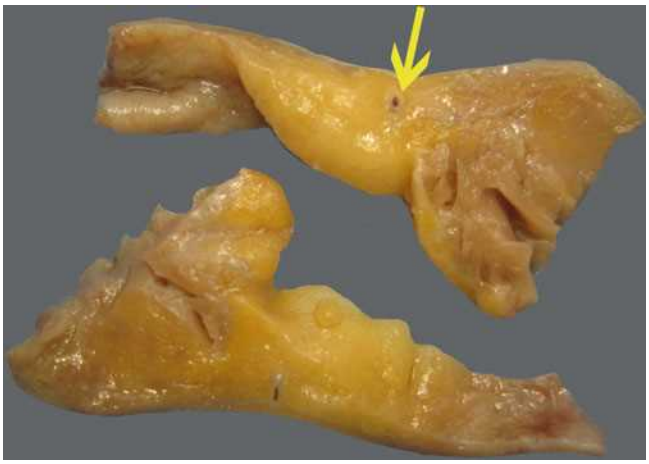

**Fig. 4.161** Close-up of the sectioned tissues at the top of the right atrium shows the small sinoatrial artery (*arrow*) at the interface between the atrial parenchyma and the superior vena cava. This is the landmark for the sinoatrial node, although the node itself cannot be discerned macroscopically

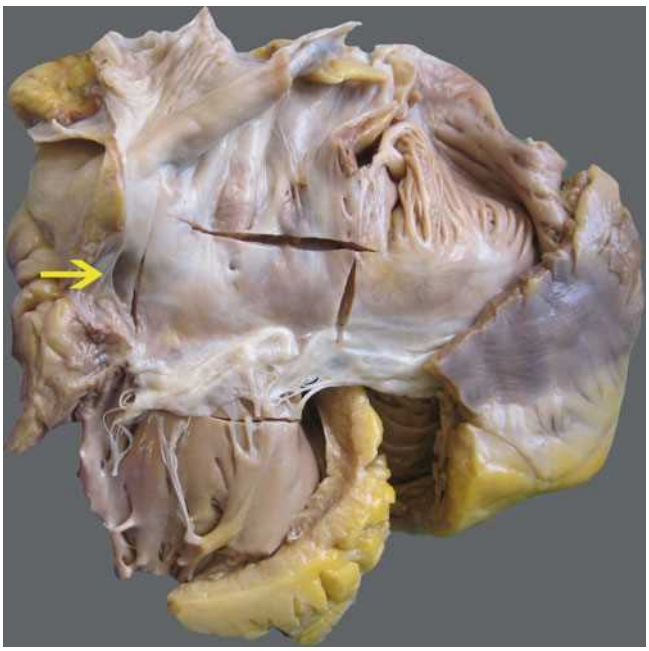

**Fig. 4.162** A view of the right atrium and ventricle in a fixed heart. The coronary sinus is seen immediately to the left of the view (*arrow*). Four superficial cuts have been made into the tissues, to define where one will then make deeper cuts. The deep cuts will allow removal of the centre of the heart parenchyma. The block of tissue will incorporate part of the tricuspid valve at the base, the membranous septum in the centre, and tissues from the atrium

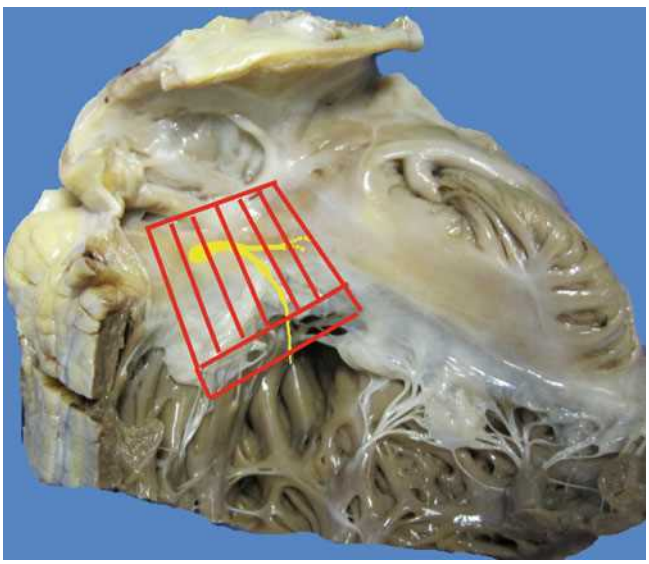

**Fig. 4.163** Further sectioning of this *square block* of tissue involves initially removing a transverse slice from the base (*i.e.*, the septal tissues, with both the right and left side being evident). One then sections the remainder in a north–south direction, passing from the left to right side. Taking pieces at intervals of 2–3 mm allows the slices to go straight into cassettes. This protocol allows atrial, membranous septum and ventricular tissues to be assessed. It also allows histological evaluation of the atrioventricular node, the His bundle, and bundle branches (From Suvarna 2013)

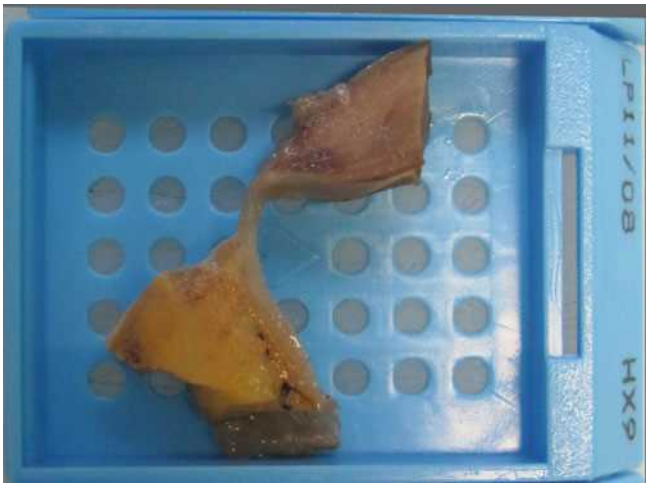


**Fig. 4.164** As an illustration, one section of the atrioventricular node/His bundle system has been cut and placed in a cassette so that architectural integrity is maintained. The thin and pliable membranous septum spans between the fatty atrial tissues and the upper interventricular muscular parenchyma. It should be noted that the fat of the atrial septum (*lower left corner*) may be prominent, but it should not be confused with a lipoma or lipomatous hypertrophy of the septum, which usually shows a rounded tumour phenotype

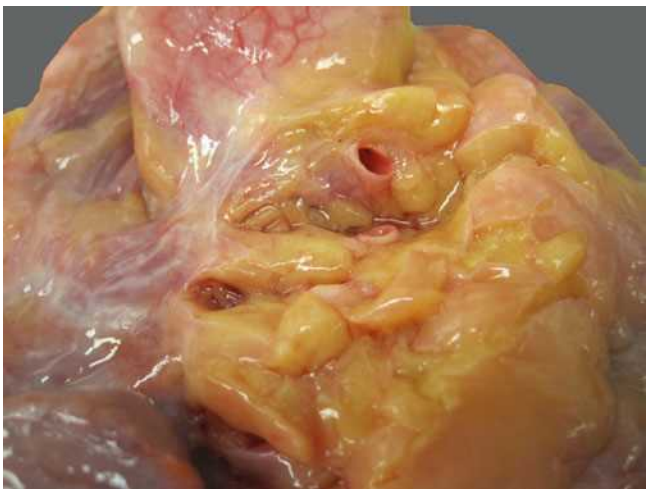

**Fig. 4.165** This view of the proximal right coronary artery, next to the aorta, shows no appreciable atheromatous disease

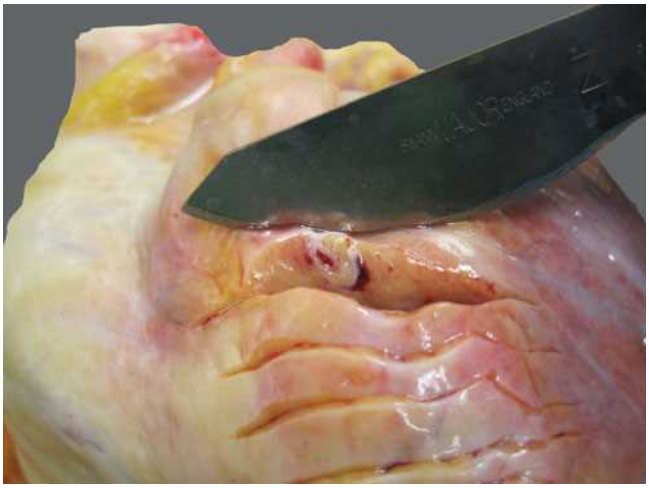

**Fig. 4.166** The serially sectioned coronary vessels in most adults seen at autopsy have some atheromatous changes. As one progresses across the arterial tree with this slicing technique, it is important to examine the cut surface of the arteries and make a judgment about the degree of narrowing

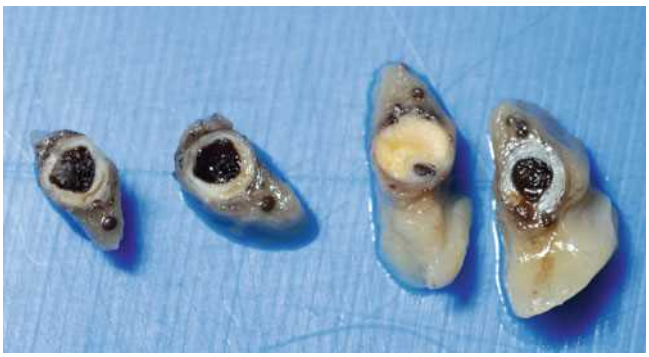

**Fig. 4.167** Sequentially sliced coronary artery tissue often shows considerable variation in the degree of stenosis. This artery shows a focal, high-grade lesion with thrombotic change of an acute type in the segments adjacent

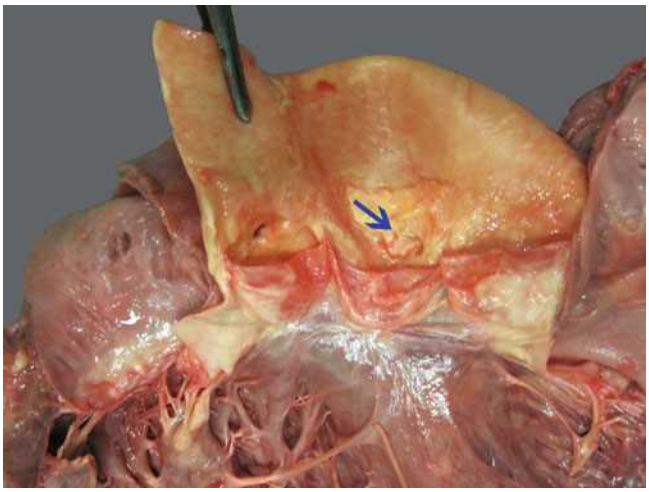

**Fig. 4.168** The coronary arteries may have only mild or moderate disease in some cases of myocardial infarction. Review of the coronary ostia (in this case, the right coronary ostium) will occasionally point to the culprit lesion (atheromatous plaque, *arrow*) that has occluded blood flow along the entire length of the artery

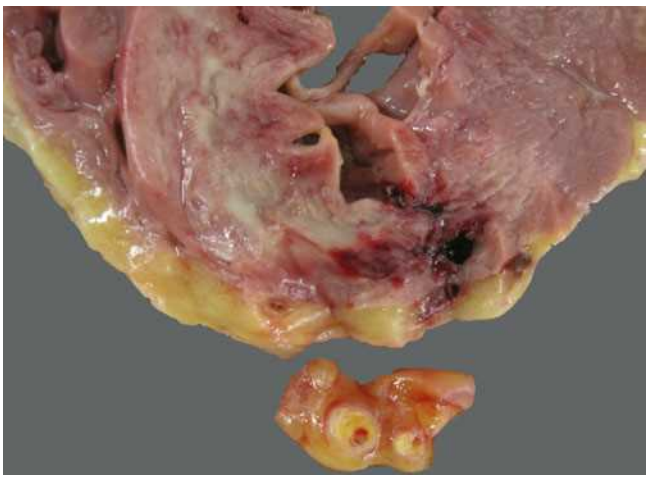

**Fig. 4.169** A high-grade coronary atheroma may be complicated by thrombosis. The consequence of thrombosis of main branches of the coronary artery is zonal myocardial infarction. This case of myocardial infarction was linked to the thrombosis of the left anterior descending artery, seen adjacent

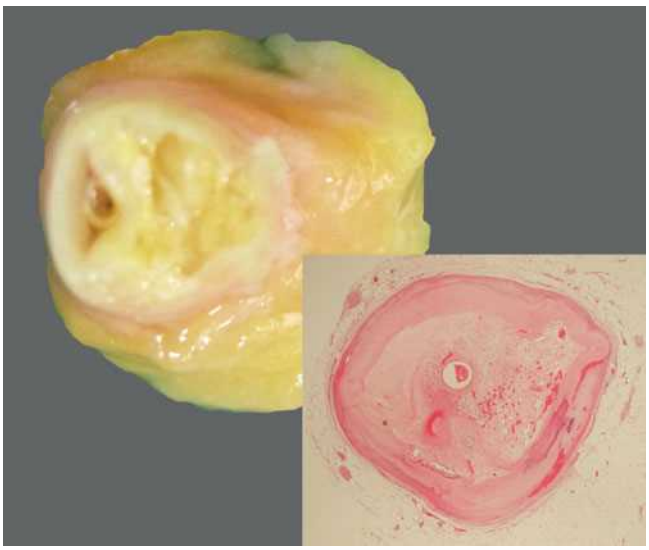

**Fig. 4.170** High-grade stenosis (seen in histological and macroscopic views) is commonly encountered in cases of sudden death and clearly points to a causal link, but one should not forget that the individual had this degree of disease in the days and months beforehand, given the slow rate of disease progression. Histology of a high-grade stenosis lesion often provides the best understanding of the disease status

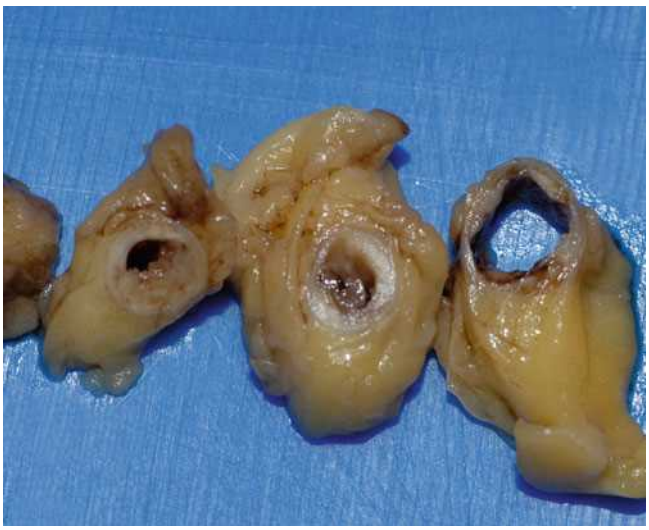

**Fig. 4.171** Atheromatous disease may be complicated by thrombus that persists. It often has a grey-brown granular or solid quality. There may yet be a microscopic lumen through this area of narrowing, explaining why complete occlusion is not seen in the adjacent segments

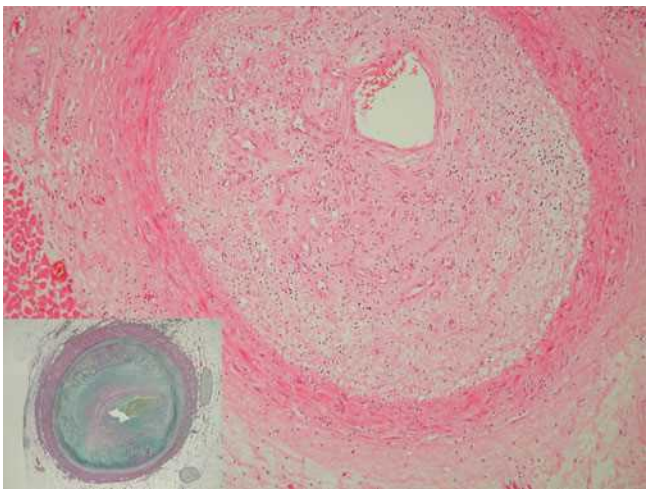

**Fig. 4.172** Not all coronary narrowing is atheromatous. This case of myocardial ischaemic damage was caused by antibody-mediated rejection causing high-grade circumferential triple vessel narrowing of the arterial vasculature in a cardiac transplant. Abundant lymphoid cells need not be present. The standard histology can be augmented by a combined elastic van Gieson and Alcian blue stain (*bottom left*), highlighting the oedematous stromal background

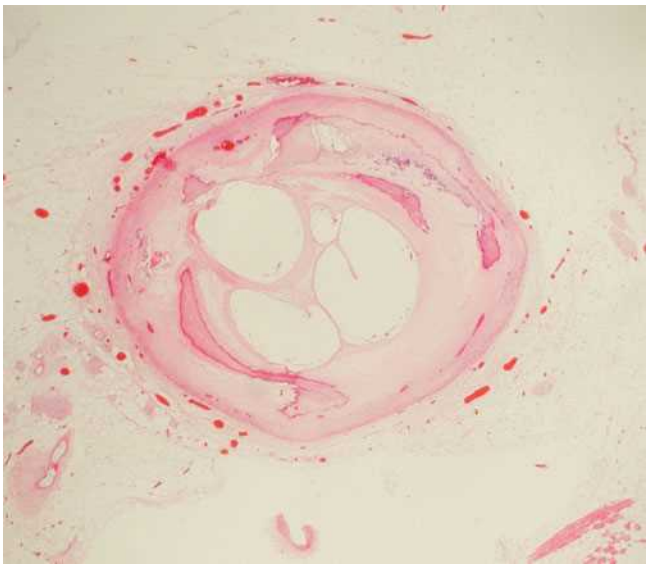

**Fig. 4.173** Histological review of high-grade coronary artery disease confirms the presence of a recanalised artery lumen following a previous partial thrombotic event

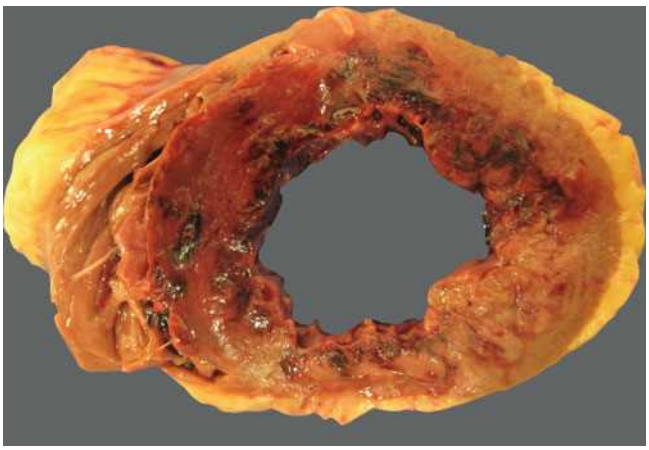

**Fig. 4.174** Myocardial infarction is often zonal in type, representing just one coronary artery territory, but hypotension and widespread atheromatous disease tends to produce significant subendocardial infarction. In this case, the subendocardial infarction is almost global/circumferential. The demarcation between infarcted tissue and viable peripheral parenchyma is relatively easy to see but has been complicated by some haemorrhage, reflecting revascularization drug therapy. There is some chamber dilatation in keeping with cardiac failure

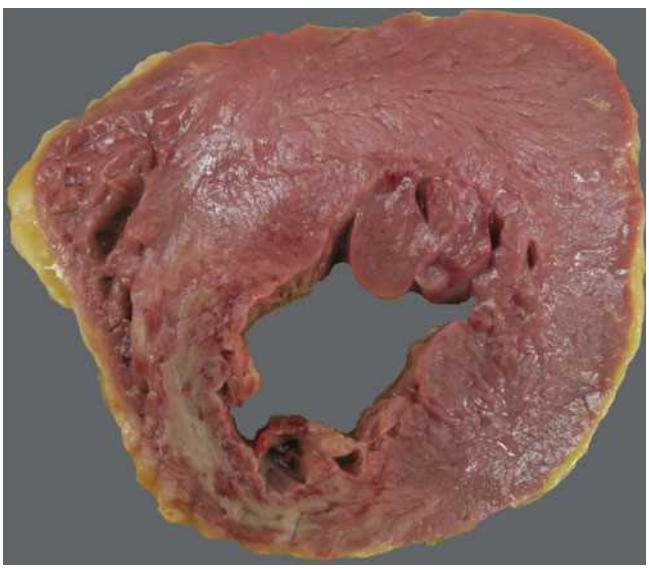

**Fig. 4.175** This hypertrophied left ventricle has an anterior myocardial infarction visible. The yellow-grey discolouration indicates infarction 2–3 days prior to death. The anterior wall is thinned; this thinning may indicate previous (several months or more) infarction at this site

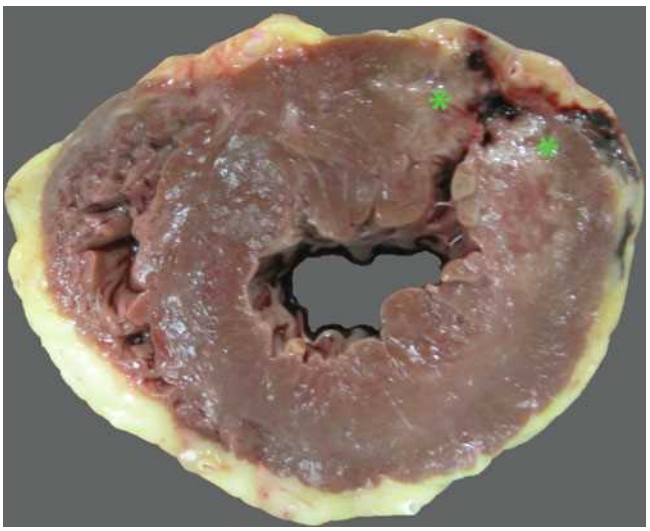

**Fig. 4.176** Acute myocardial infarction of zonal type is capable of rupture after about 4–6 days, spilling blood into the pericardium (cardiac tamponade). At autopsy, this manifests as pale, necrotic tissue (*asterisk*) with a central haemorrhagic tract running through the ventricular wall

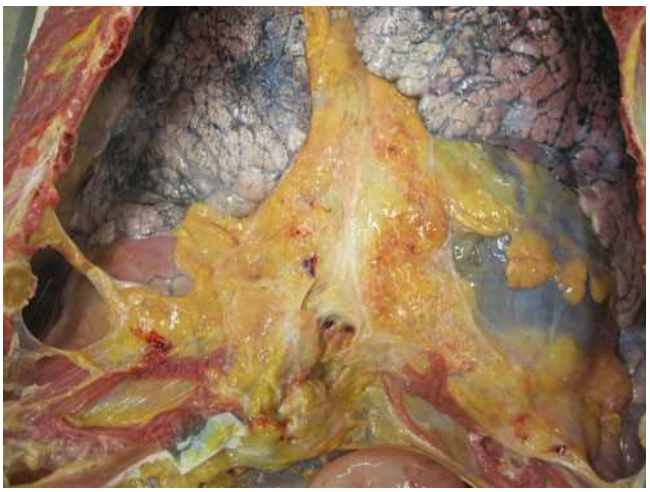

**Fig. 4.177** A 'blue bag' in the case of acute cardiac tamponade. The pericardial sac is distended by a large amount of blood and clot. The features are evident after the chest wall has been removed at the start of evisceration

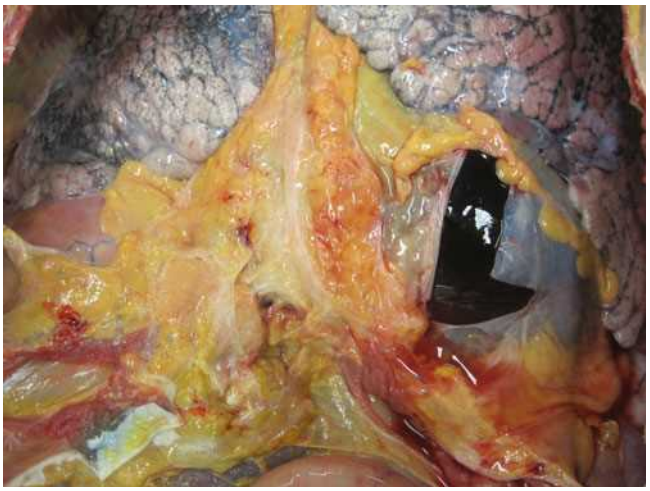

**Fig. 4.178** The cardiac tamponade case has been opened to reveal the blood and clot that caused the tamponade surrounding the heart. The infarcted heart tissue is not visible until the blood and clot has been removed

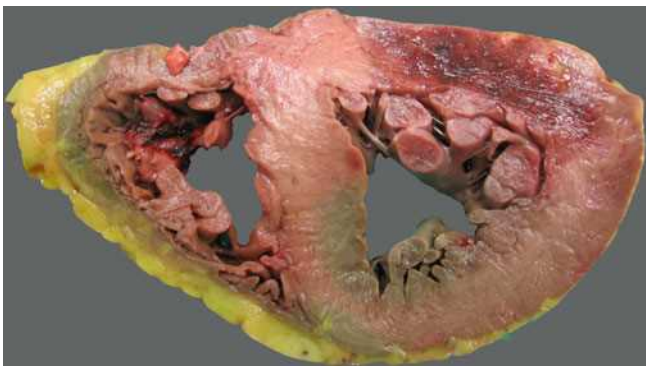

**Fig. 4.179** Myocardial infarction is rarely left without treatment, commonly thrombolytic and/or other revascularization therapy. This therapy may cause secondary haemorrhage within the infarcted myocardium, as seen in the posterior left ventricle in this case

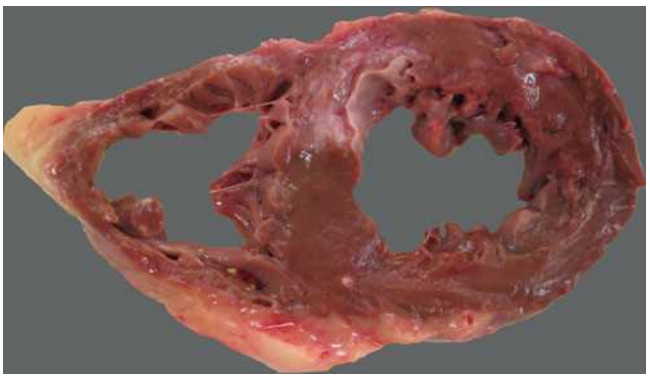

**Fig. 4.180** Scarring fibrosis in the posterior septum and back wall of the left ventricle is indicative of previous (now healed) infarction damage

**Fig. 4.181** Aneurysmal dilatation of the left ventricle is common in cases of extensive infarction. The secondary mural fibrosis and thinning allows the aneurysm to expand

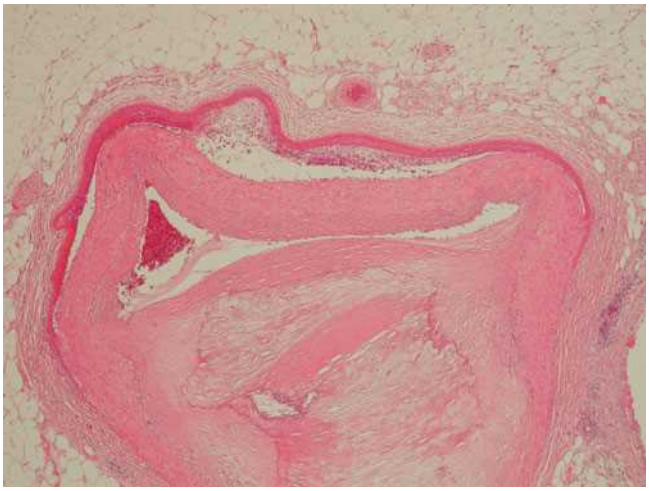

**Fig. 4.183** Acute coronary occlusion may follow coronary dissection. The histology is vital in confirming that the true lumen is occluded by the false lumen (running in the outer media), which fills with blood and blocks the normal luminal blood flow. Background atheromatous disease is present

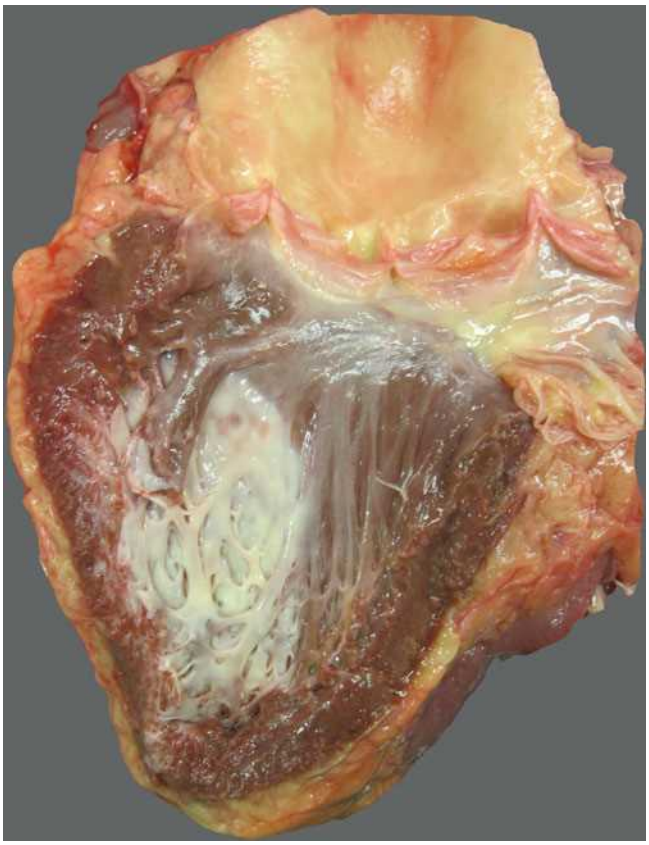

**Fig. 4.182** A well-defined zone of infarction, with pronounced endocardial fibrosis, is seen in the low septal tissues. No acute changes are seen. This may cause a functional aneurysm bulging into the right ventricle or a risk for sudden dysrhythmic death

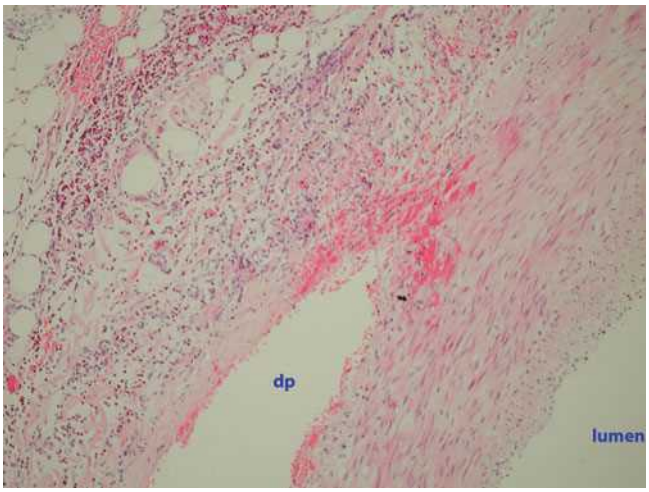


**Fig. 4.184** Coronary artery dissection can occur as a consequence of localised vasculitis. This case had a pronounced eosinophilic component. The true *lumen* is seen in the bottom right of the image, with the dissection plane (*dp*) running across the tissue obliquely. There is some debate as to whether the eosinophils are a consequence of the dissecting artery or the cause for the dissection

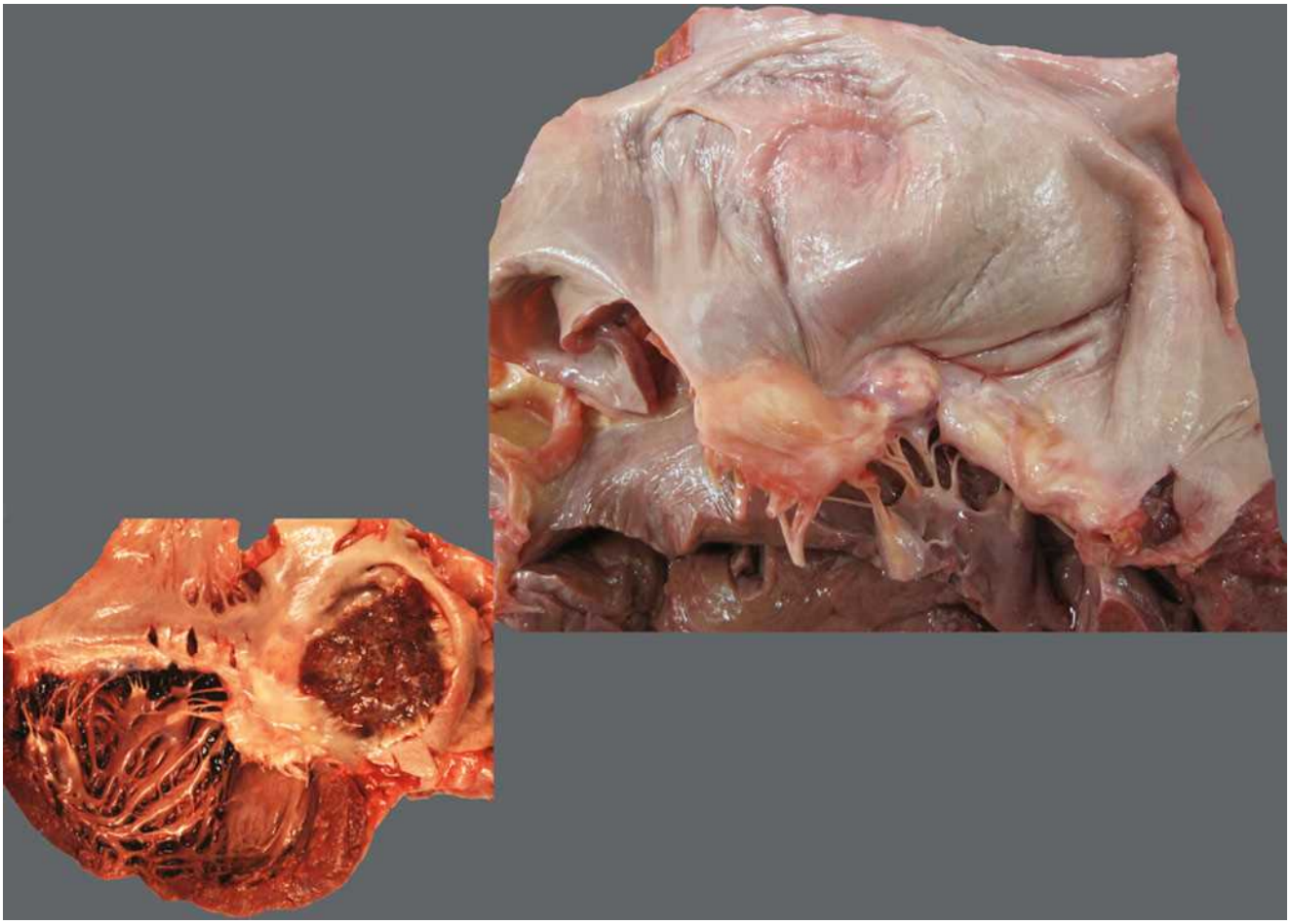

**Fig. 4.185** Mitral valve stenosis can be difficult to define at autopsy. A particularly enlarged or ballooned left atrium (*upper right*) often is supportive evidence for this process, but requires mapping to ante-mortem clinical data. A dilated atrium is often a potent driver for thrombosis associated with atrial fibrillation (*lower left*) and a risk for systemic embolization

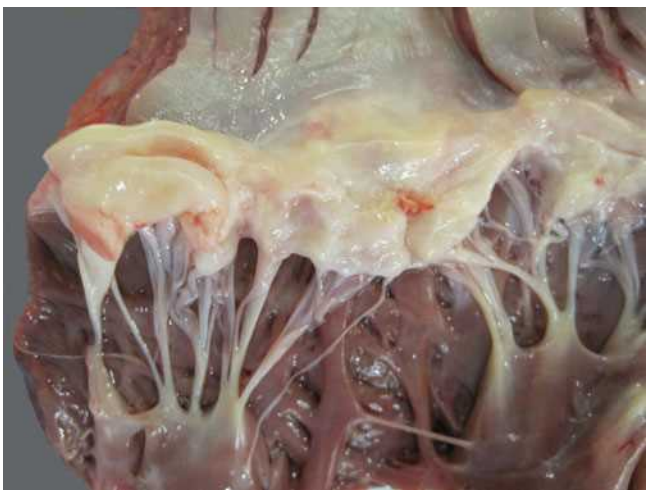

**Fig. 4.186** A floppy mitral valve is seen. It demonstrates the thickened and mucoid quality of the valve tissue, with similar changes affecting the chordae tendineae

**Fig. 4.187** A floppy mitral valve is seen with classic ballooning, 'parachute-type' deformity of the free margins

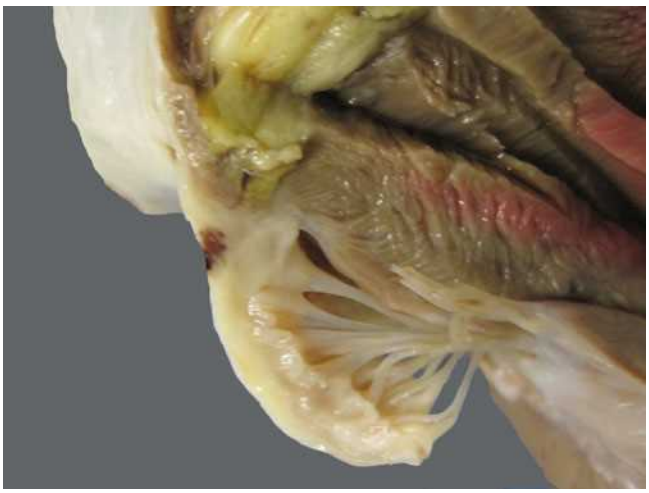

**Fig. 4.188** Section through a markedly thickened and mucoid floppy valve. Note how the chordae extend from the free margin of the valve as well as from various points of attachment along the undersurfaces of the valve

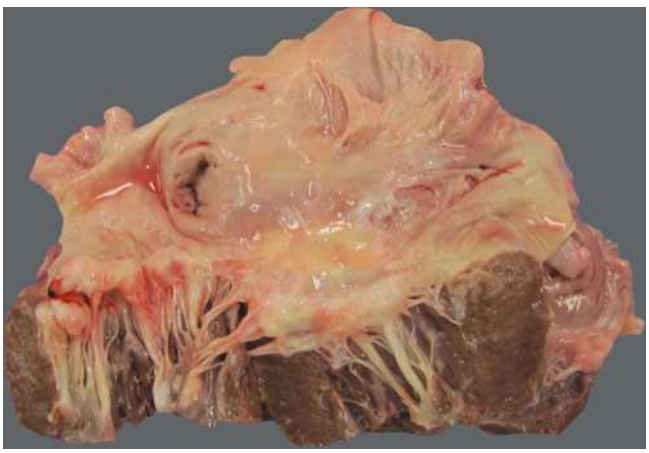

**Fig. 4.189** Rheumatic mitral valve disease is still a common feature in the autopsy room. Thickening of the chordae and irregularity, distortion, and thickening of the valve cusps are common on the atrioventricular valves. Thickening and distortion of the semilunar valves in aortic and pulmonary roots also can be seen. It is important to consider whether there is a functional deficit of valve function in terms of opening and closure. One should also diligently search for features of infective endocarditis

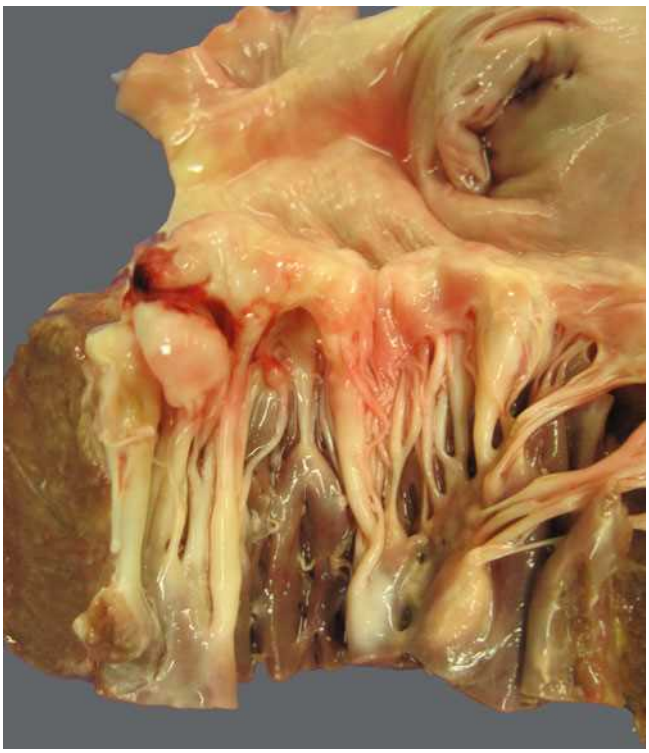

**Fig. 4.190** Close-up magnification of the thickened chordae in a case of mitral valve stenosis secondary to rheumatic fever

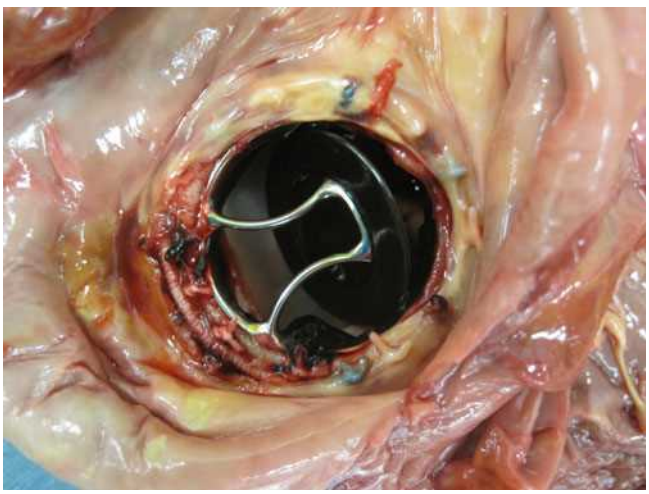

**Fig. 4.191** In this case, mitral valve disease has been treated by metal valve replacement. The valve opens and closes with the central disc tilting back and forth. This type of device is no longer used but is still occasionally encountered in the autopsy room

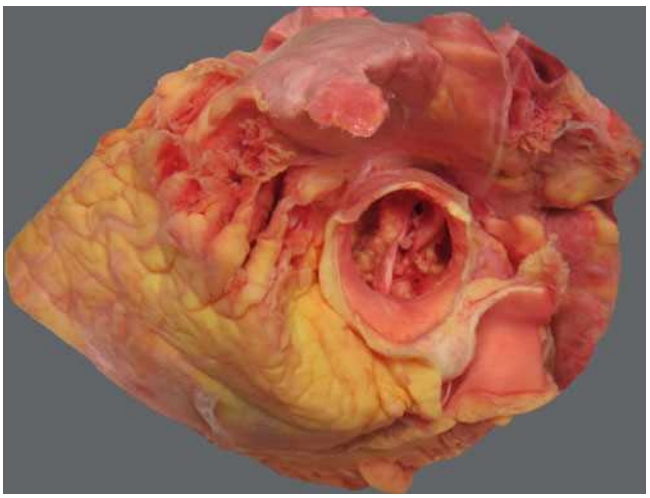

**Fig. 4.192** The aortic root, seen from above, shows a tightly stenotic, bicuspid aortic valve. Nodular dystrophic calcification is present in the valve cusps. This case was photographed having been placed under water to aid and support the natural positioning of the cardiac tissues

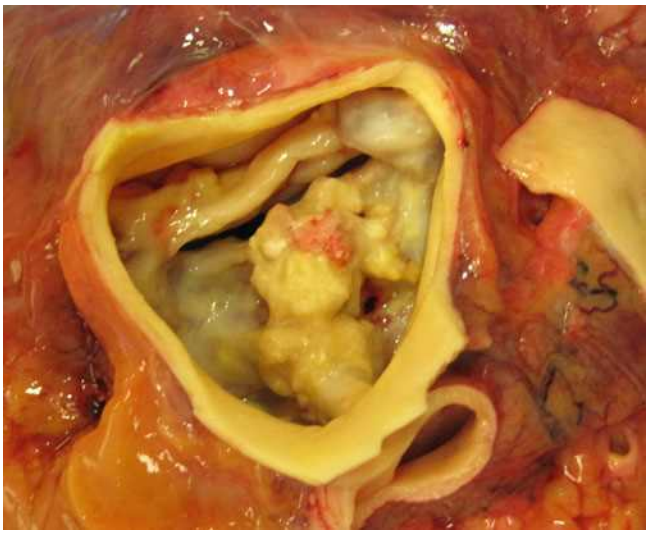

**Fig. 4.193** A pseudobicuspid aortic valve is seen with heavy nodular calcification. One of the commissures is fused with nodular calcific deposits. The net result is tight occlusion of the valve orifice. No vegetations are seen on the valve tissue

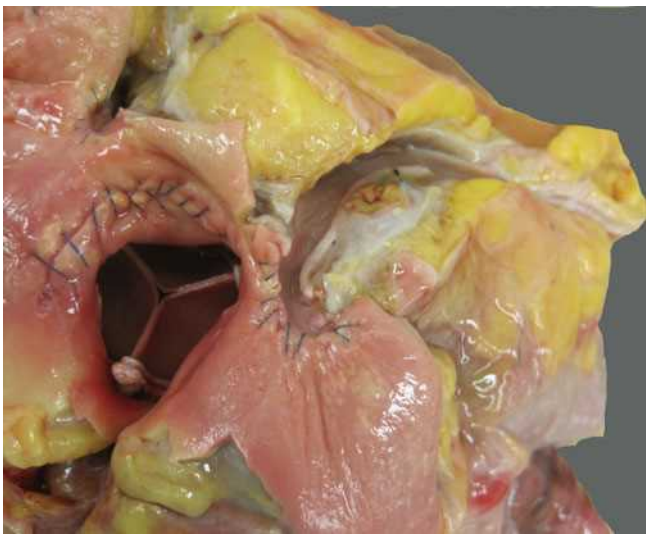

**Fig. 4.194** Replacement of the calcified aortic valve has been accomplished by a tissue graft with vein bypass surgery. Exploring this reality at autopsy requires consideration of the surgery performed and then careful incisions to expose the operative solutions. The valve must be shown to be correctly positioned and with sepsis/thrombus. The vein graft should be free from thrombus. The suture lines from the surgery can be inspected for leakage

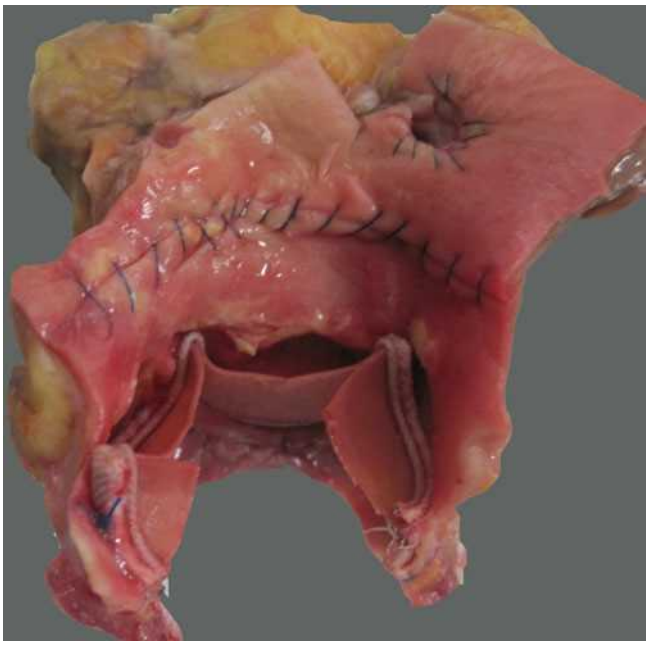

**Fig. 4.195** The tissue graft has been removed, with local cardiac tissues. The valve ring is then cut open with metal pliers, as scissors will not be able to cut through the metal support rings. The valve is forced open to demonstrate the inner leaflets

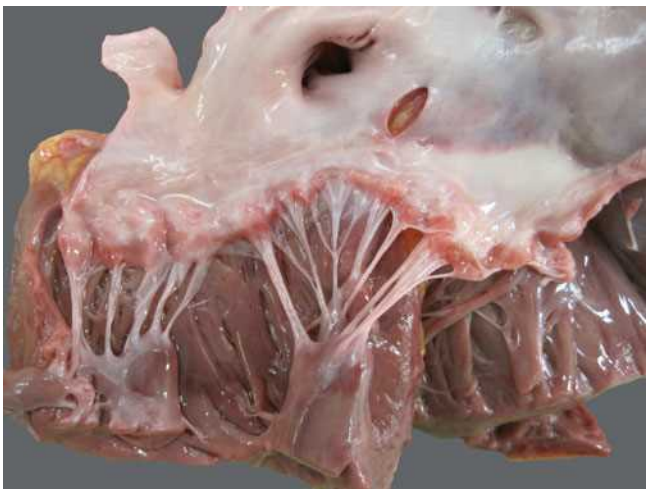

**Fig. 4.196** Nonbacterial (noninfective) thrombotic endocarditis [NBTE] is often seen as a rather fleshy line of thrombotic matter at the free margin of the valves. It is not associated with valve tissue destruction and often appears to run in a complete ring around the mitral valve, as in this case. Note the preservation of the chordae. Histology and microbiology assessment to confirm the aseptic quality of the vegetations should follow

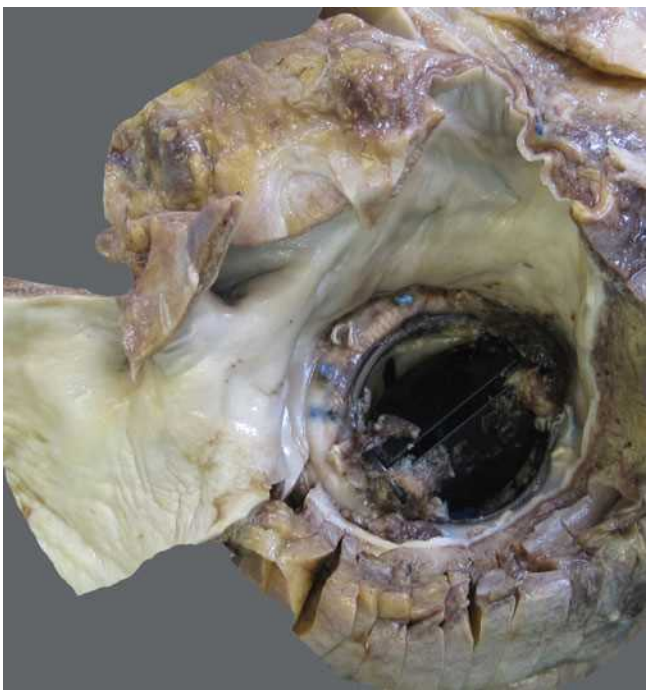

**Fig. 4.197** A valve may show a marked degree of thrombus, often occluding valve leaflet excursion. Such tissues should be reserved for microbiological study and for histology in order to consider the possibility of infective endocarditis. This case proved to be streptococcal infective endocarditis

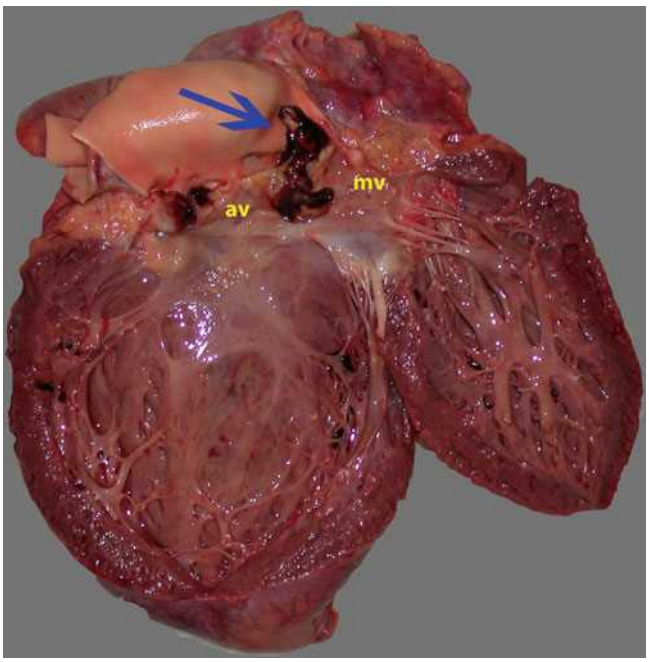


**Fig. 4.198** Naturally occurring infective endocarditis (*arrow*) can involve any valve, although the mitral and aortic valves are most affected. This case of infective endocarditis reflected streptococcal sepsis, settling upon the aortic valve. No specific underlying valve defect was identified, but often the severe tissue destruction prevents accurate assessment of the possible drivers for infective endocarditis. Note the proximity of the aortic valve (*av*) disease to the mitral valve (*mv*) parenchyma, explaining why dysfunction of two valves can occur readily

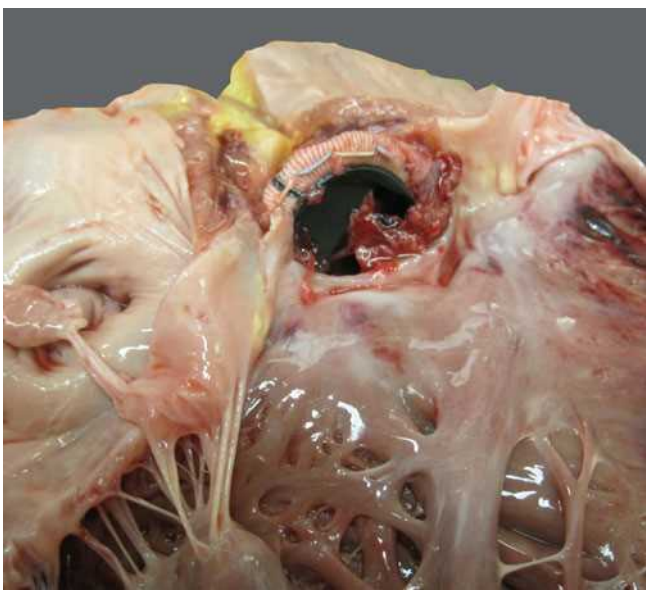

**Fig. 4.199** A mitral valve prosthesis (twin-leaflet metal device) is seen with irregular vegetation around the rim of the valve and partially interacting with the valve mechanism. Histological identification of bacterial colonies confirmed infective endocarditis

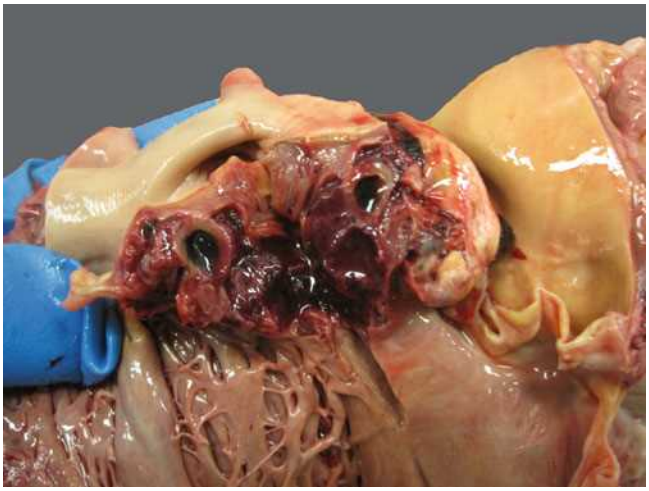

**Fig. 4.200** The degree of tissue destruction in cases of infective endocarditis can be underappreciated when looking at the ventricular aspect of the valve. Slicing into the septic focus can expose significant haemorrhagic tissue destruction with cystic loculi, as in this case. Such tissue destruction often is associated with virulent bacteria, in this case staphylococci

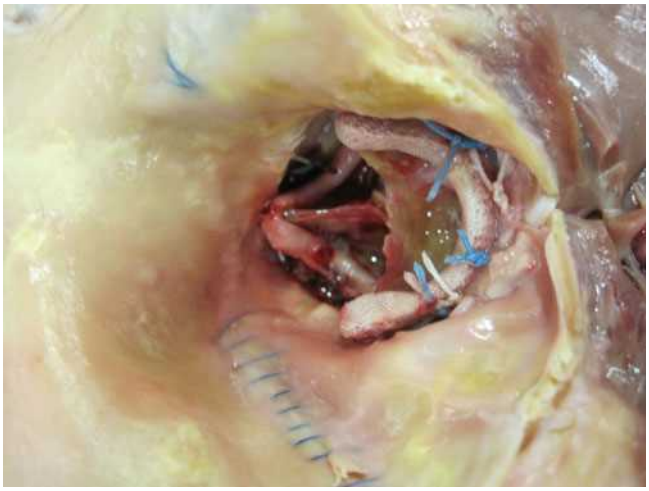

**Fig. 4.202** One of the problems with tissue valve prostheses is that the leaflets degrade with time to become progressively less competent. These valves were thickened, discoloured, and partly fragmented

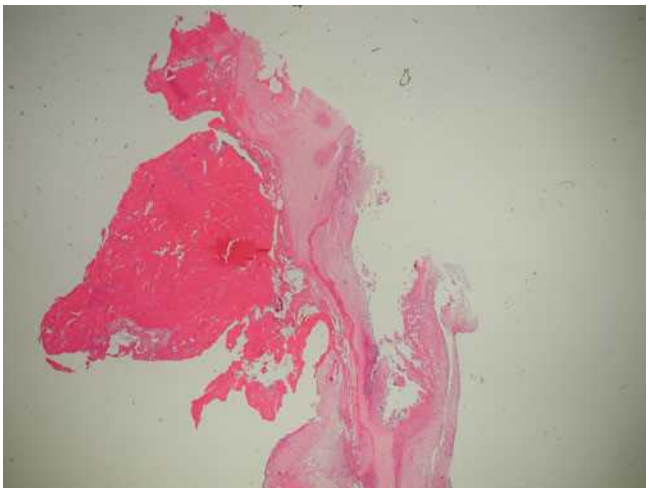

**Fig. 4.201** Histology of infective endocarditis shows adherent thrombus and valve destruction

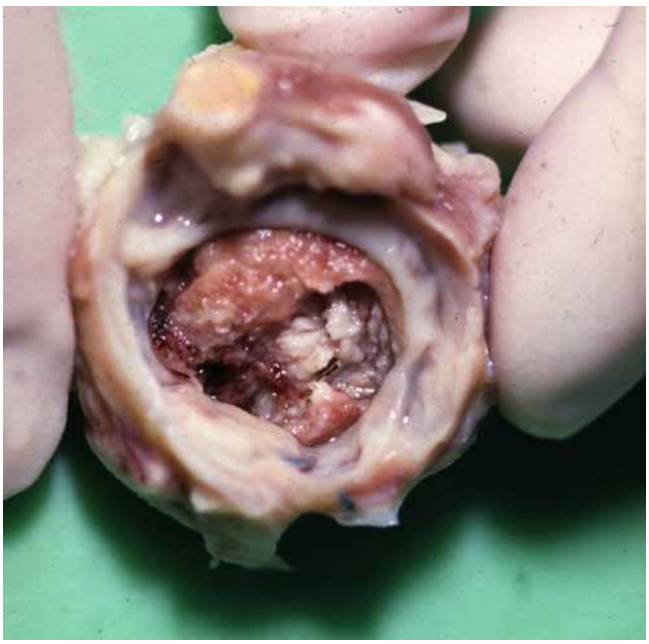

**Fig. 4.203** Large amounts of thrombus forming on the valve prosthesis will occlude leaflet movement and put the individual at risk of infective endocarditis

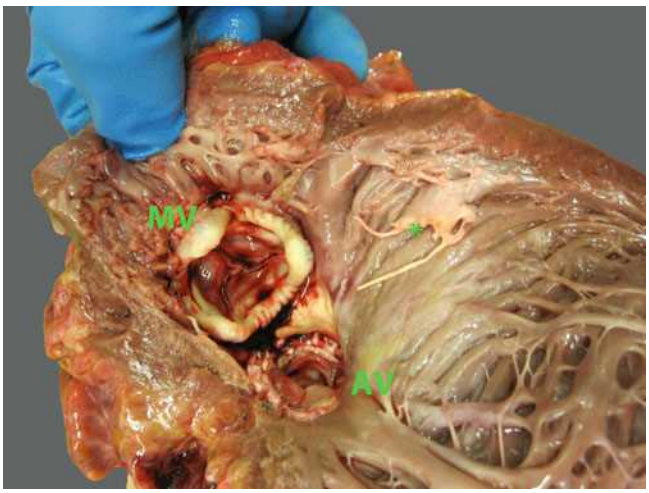


**Fig. 4.204** Two tissue prostheses are seen replacing the mitral valve (*MV*) and aortic valve (*AV*). Both sides of the tissue prostheses can be seen in this view. Opening the ventricular tissues is hampered by the prostheses, blocking cuts made in the standard perpendicular fashion across the valves. One alternative is to cut around the ventricle at a high level and open the tissues outwards. This allows direct visualization of the valves and their movement (in effect looking upwards). Residual mitral valve chordal and papillary muscle elements (*asterisk*) are present

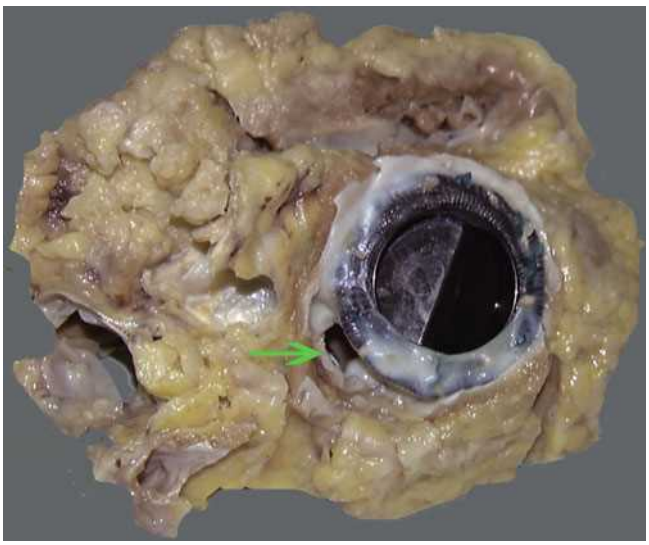

**Fig. 4.206** Demonstration of paravalvular leak is often difficult in the autopsy room, particularly as the valve tissues are often fibrosed within somewhat distorted tissues. In this case, the entirety of the left atrium has been dissected free, exposing the mitral valve prosthesis and a leakage point (*arrow*) causing valvular inefficiency

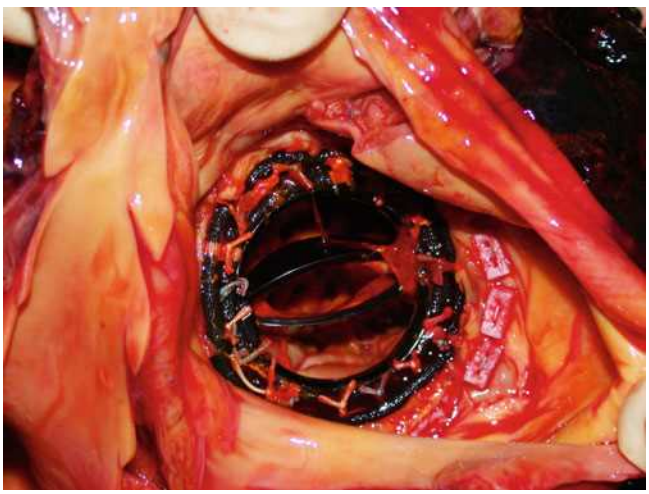

**Fig. 4.205** The valve replacement here is a twin-leaflet tilting device. At autopsy, the leaflets should be checked to see if they open and close normally. As always, check for infective endocarditis and/or thrombus

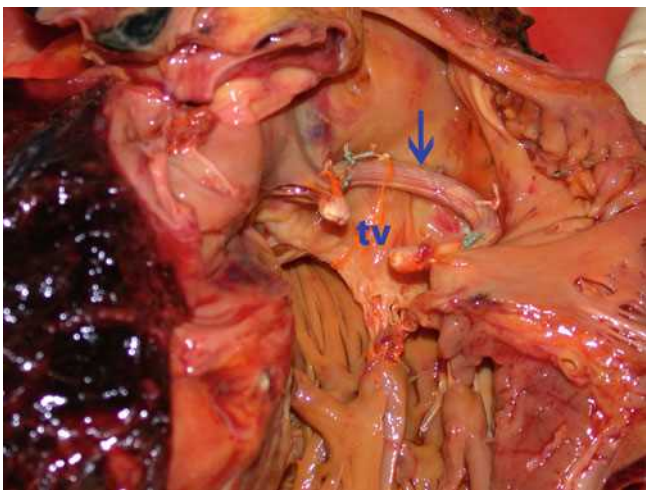


**Fig. 4.207** The right atrium and ventricle have been opened from the posterior aspect to reveal a C-shaped annuloplasty device (*arrow*) at the root of the tricuspid valve (*tv*). The incompetent valve has been assisted to close normally, with the annuloplasty drawing the valve ring inwards (Adapted with permission from Suvarna SK. National guidelines for adult autopsy cardiac dissection and diagnosis–are they achievable? A personal view. Histopathology. 2008;53:97–112)

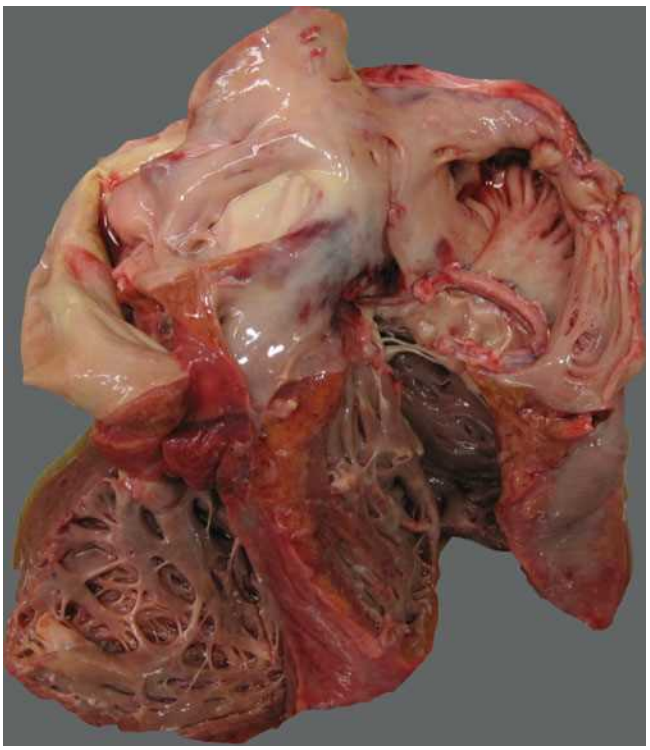

**Fig. 4.208** Another annuloplasty is seen with local septal haemorrhage. This patient had also undergone a mitral valve replacement, which was accomplished by a transeptal approach

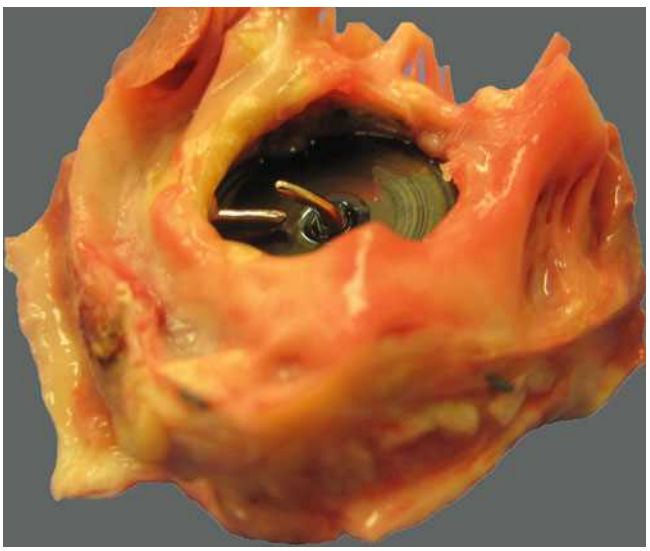

**Fig. 4.209** Pannus is a fibrous tissue overgrowth that runs from the local paravalvular soft tissues onto the surface of the valve mechanism. If significant, it can partially or largely obstruct valve function and cause progressive cardiac failure

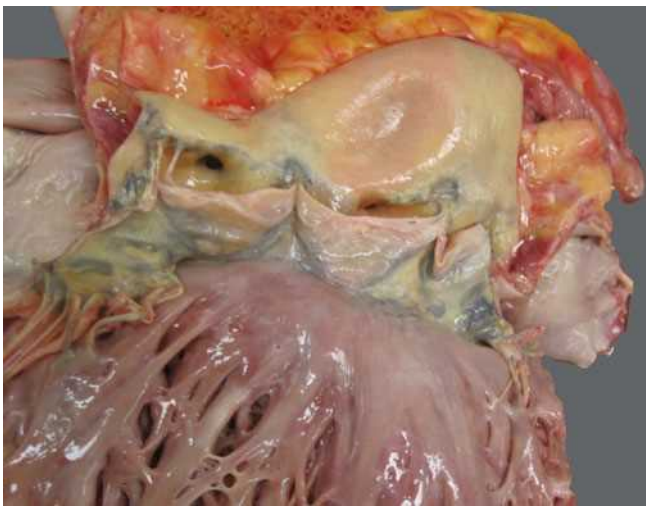

**Fig. 4.210** Discoloured aortic valve tissues are seen in a case of ochronosis. The impact on valve function generally is minimal

**Fig. 4.211** A selection of metal cardiac valves, showing different designs over the years. The early types were ball-in-cage devices (*top*), which were associated with red cell haemolysis and thrombotic issues. Several tilting-disk devices are also shown (*left* and *bottom*). The favoured type of metal device (*right*) has twin metal leaflets set within a metal frame

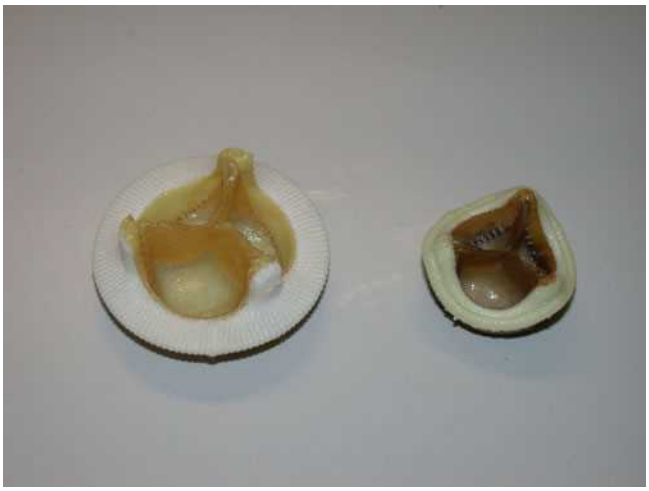

**Fig. 4.212** Two tissue prostheses of different sizes. The tissue valve prosthesis (*bottom*) generally comprises a plastic/fabric ring and strut mechanism, which allows the xenograft or homograft tissue valve to be hung on the support

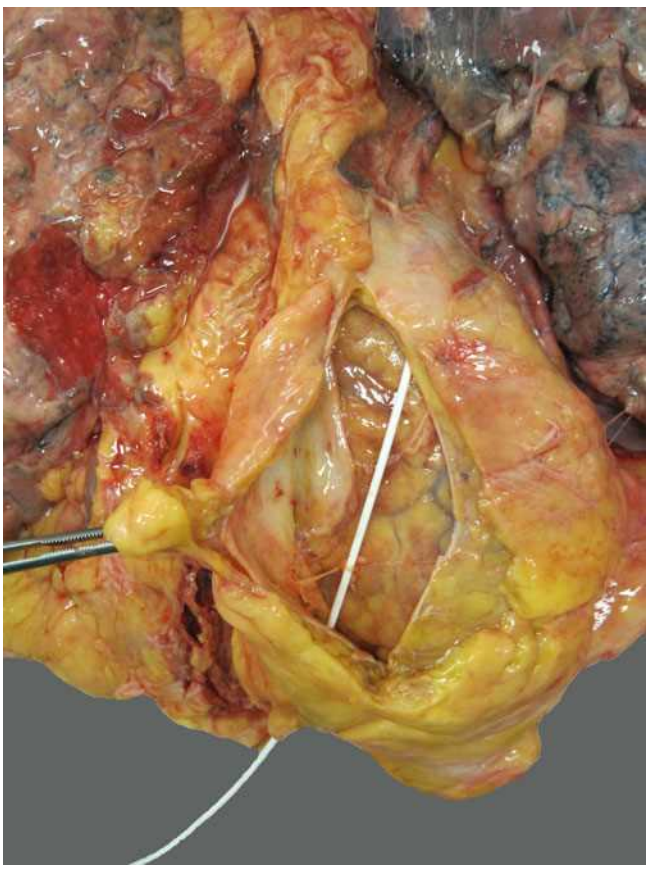

**Fig. 4.213** The following set of images details the examination of a postoperative case (coronary bypass surgery and aortic valve surgery), in which the surgery complicates the tissue analysis. Part of the dissection will inevitably be altered, but part follows the usual dissection protocol. At the start, dissection of the pericardium demonstrates the correctly positioned pericardiocentesis drain, without damage to the underlying myocardium

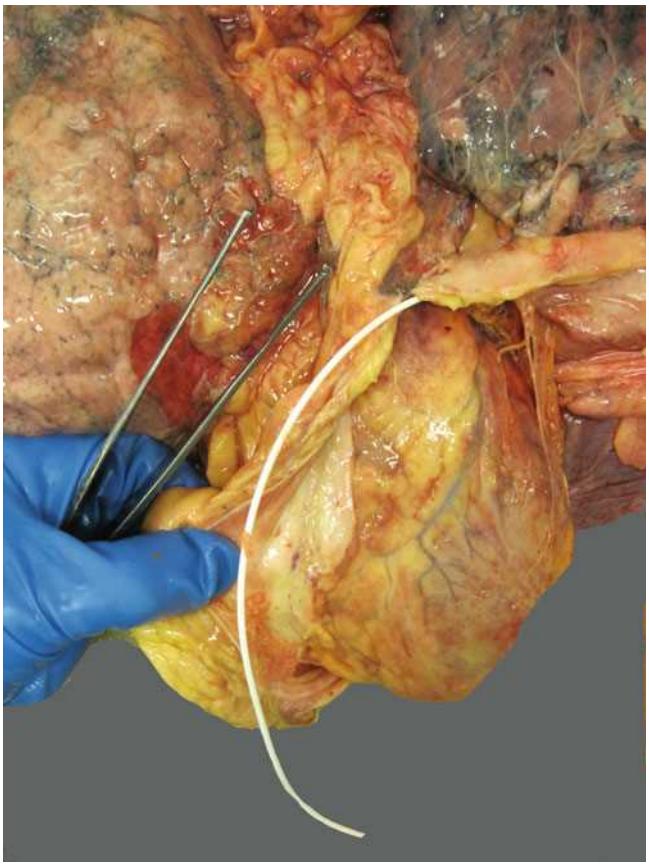

**Fig. 4.214** The pericardial tissues are dissected free of the specimen

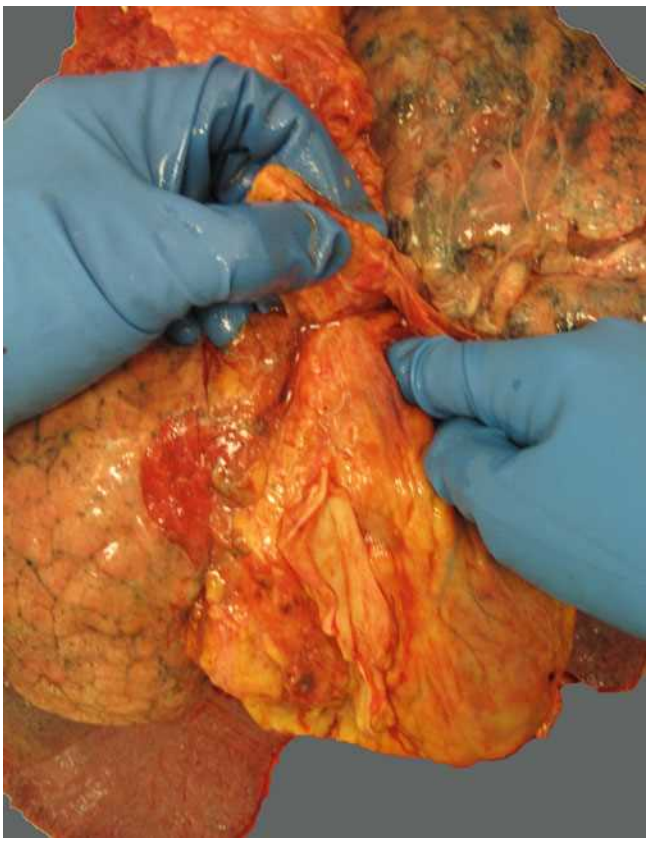

**Fig. 4.215** The pulmonary artery is incised, and fingertip palpation for pulmonary embolism follows. This procedure may be complicated by the nature of the cardiac surgery, which prevents easy manipulation of the large vessels and upper heart tissues

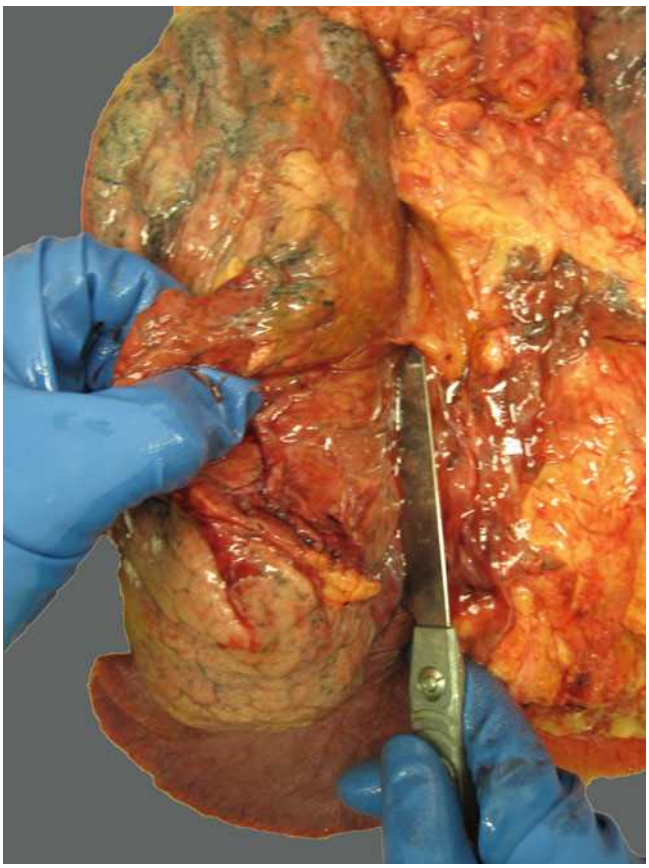

**Fig. 4.216** It is recommended that the two lungs are removed at this point by direct transection of the pulmonary hilum on either side. The heart tissues are then available for study, paralleling the normal approach

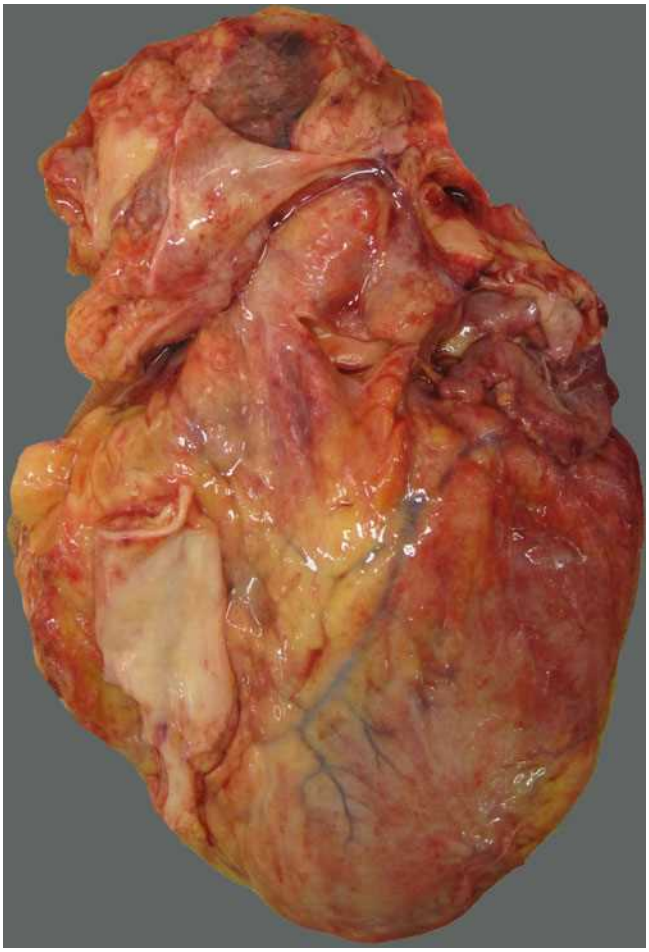

**Fig. 4.217** The freely dissected cardiac tissues are now available for inspection. The front of the heart should be considered first

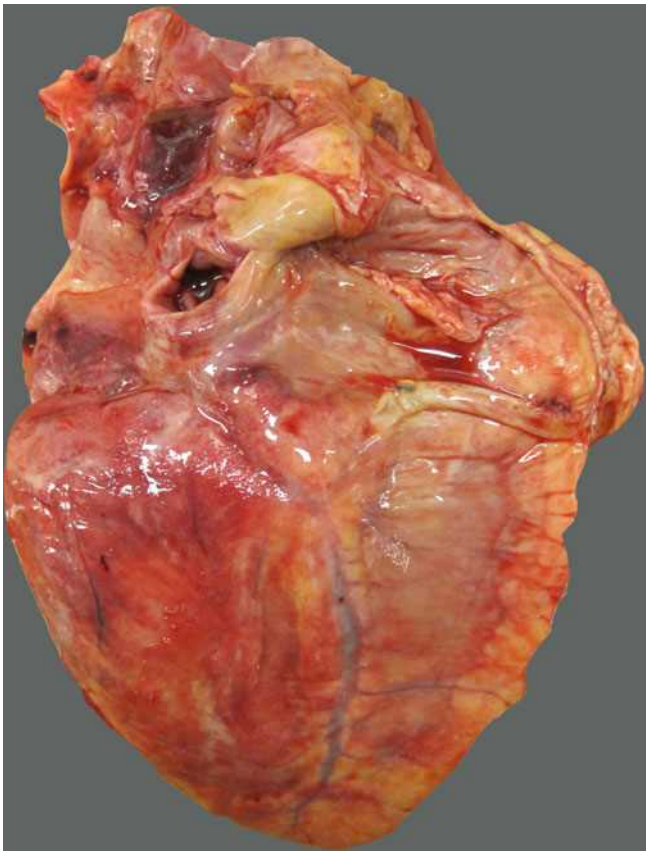

**Fig. 4.218** The tissues at the back of the heart are likewise to be examined. Surgical suture lines should be considered. The insertion points of the coronary grafts should be also sought

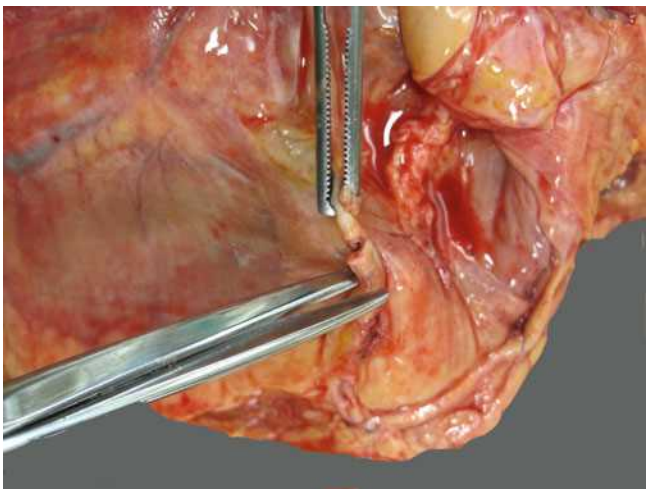

**Fig. 4.219** Partial transection of the vein graft immediately above the insertion allows direct inspection and consideration of possible thrombus in the vessel at this point

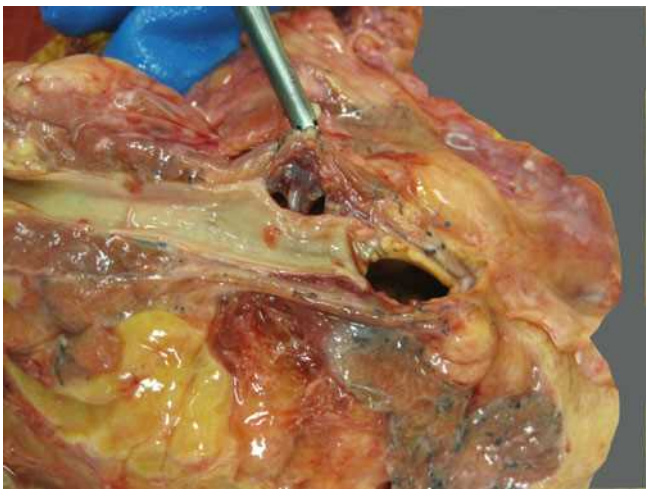

**Fig. 4.221** Both ends of the graft were clear of thrombus. The scissor dissection runs upwards into the aortic root

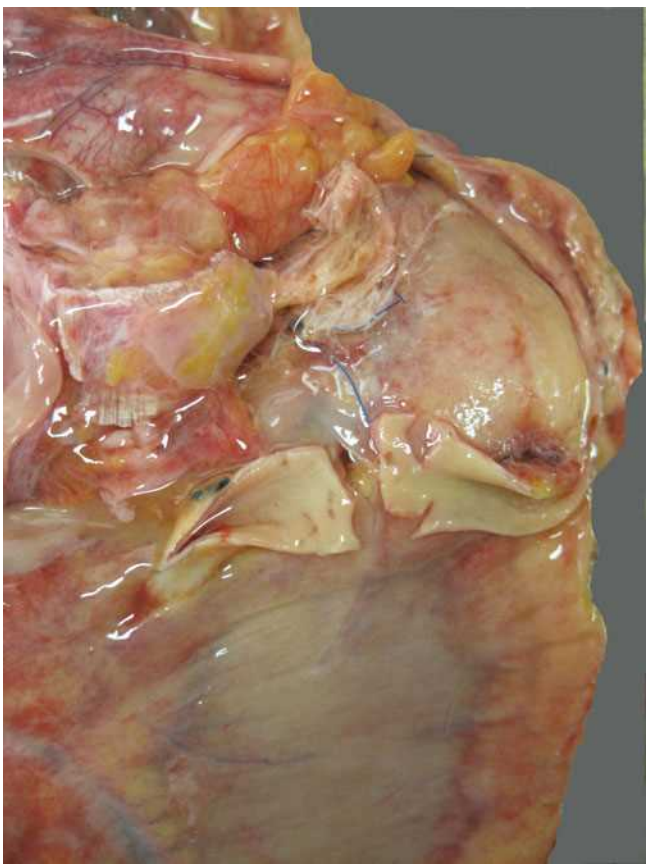

**Fig. 4.220** If there is no thrombus, then the origin can likewise be partially transected and inspected. If there is no thrombotic obstruction at either end, then the artery can be opened longitudinally using the scissors along its length

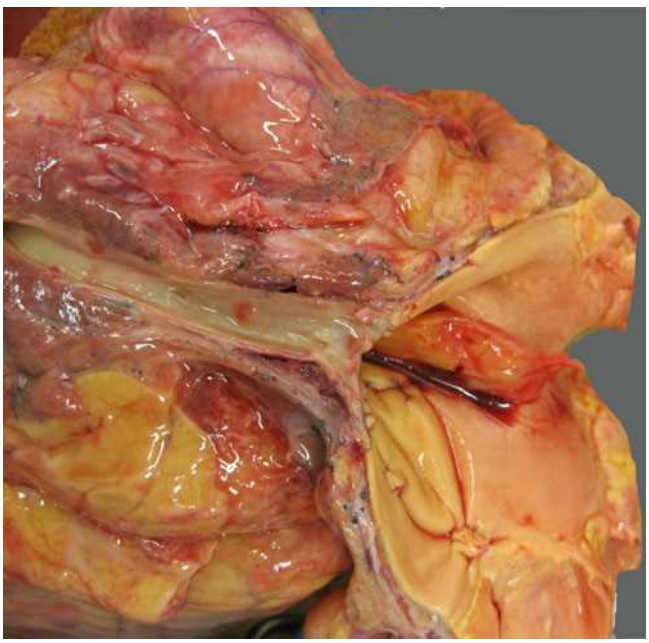

**Fig. 4.222** The ostium of the vein graft shows no atheroma or stenosis. It is always advisable to look inside the aorta as one makes this cut, to be sure that thrombus is not being pushed out of the way

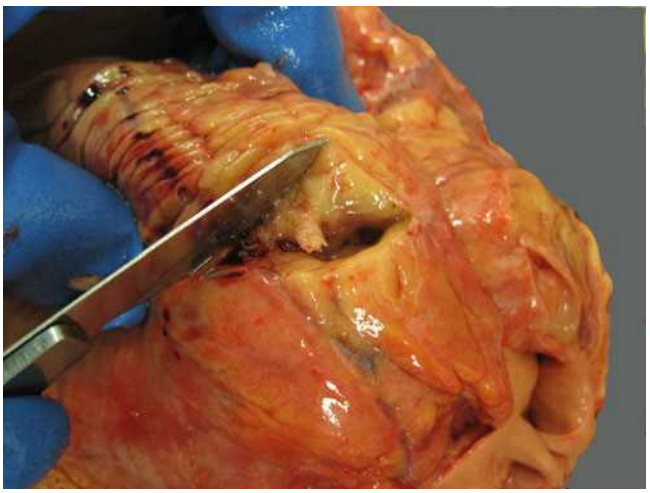

**Fig. 4.223** The native coronary arteries are examined in the normal fashion, after the grafts have been examined. Heavily calcified vessels (seen here) may need to be taken off, decalcified, and then examined later (in sequence) by macroscopy and histology

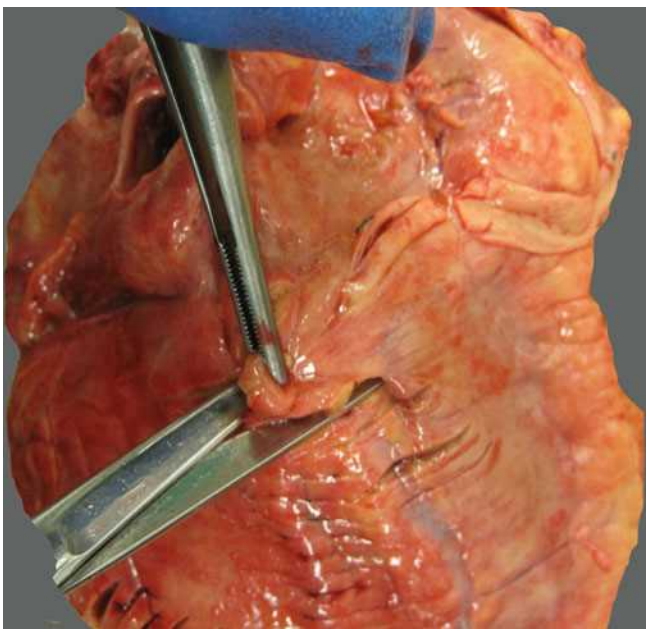

**Fig. 4.224** If there is a suspicion or evidence of thrombus obstructing the origin or anastomosis point, the section of tissue must be taken out intact for histology (after fixation and decalcification)

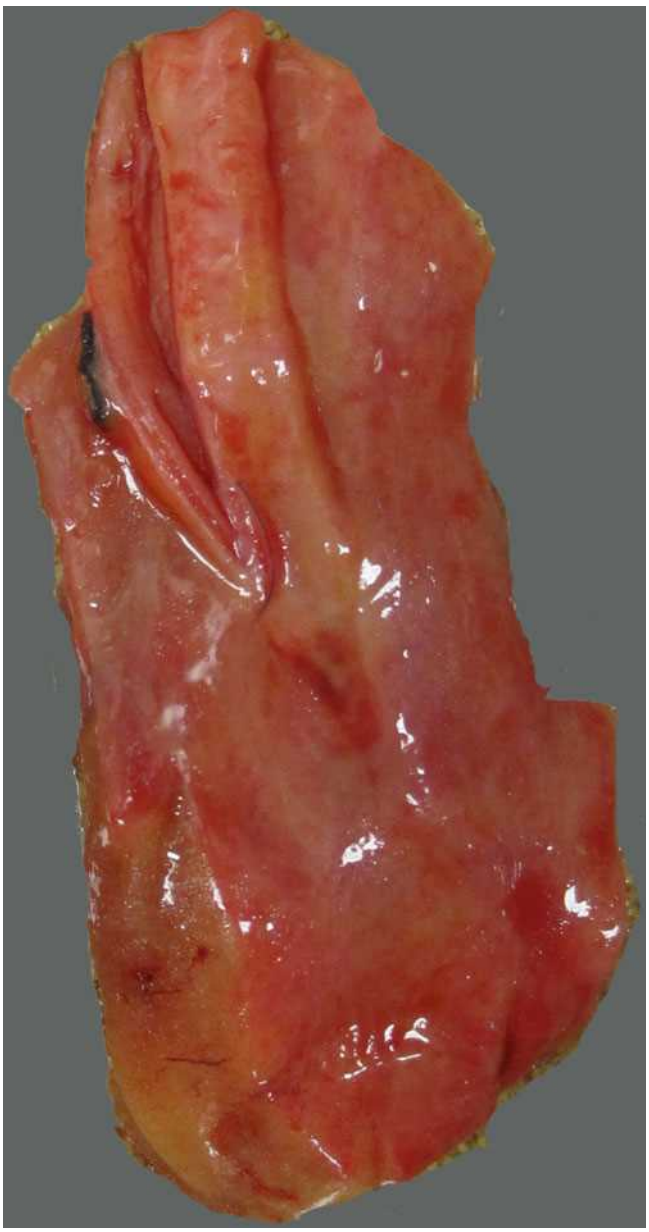

**Fig. 4.225** The insertion point (magnified) of the graft should be fixed whole. It is recommended that the tissue is decalcified (48 h). After this, the tissue may be sliced transversely and processed for histology

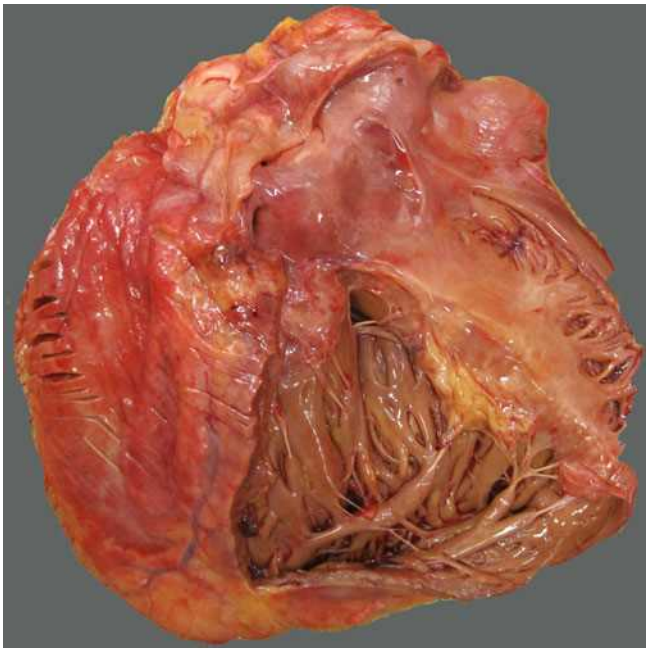

**Fig. 4.226** Whilst it is possible to follow the transverse slicing of the ventricular tissues in cases of previous cardiac surgery, occasionally it can be beneficial to leave the specimen intact and open the back of the right ventricle down towards the apex

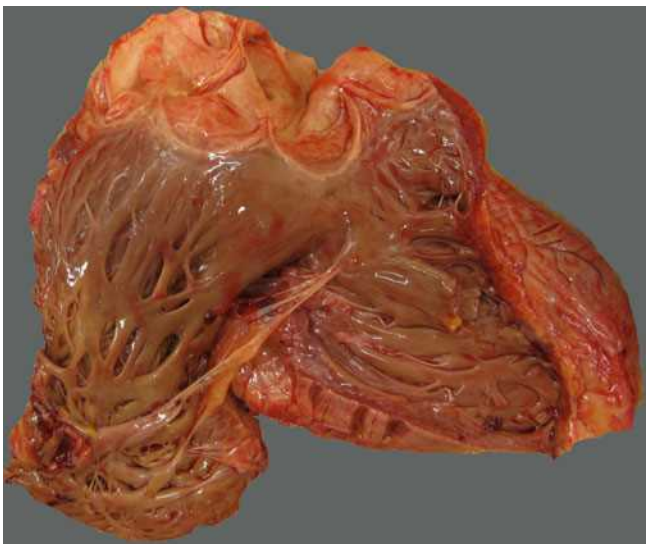

**Fig. 4.227** The cut is then extended around onto the front of the heart and out through the pulmonary valve into the pulmonary artery root. The internal architecture of the right ventricle is now fully available for study

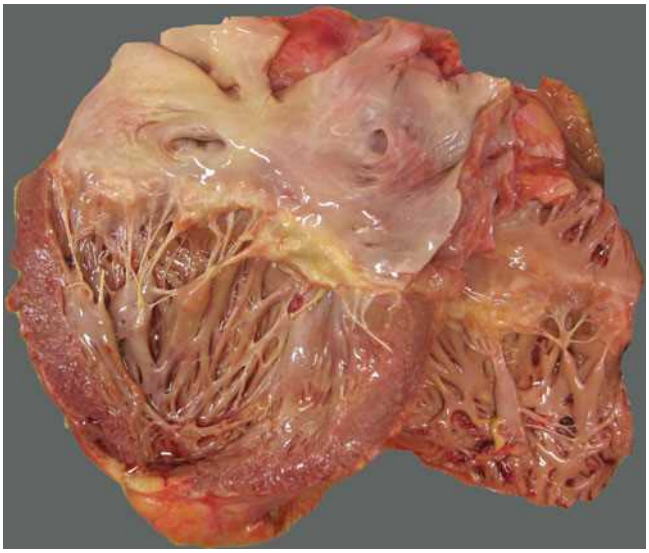

**Fig. 4.228** The left atrium and ventricle are opened by opening the back of the mitral valve and then down along the left ventricular posterior free wall. Inspection of the atrial and ventricular tissues and valvular apparatus is accomplished with this technique

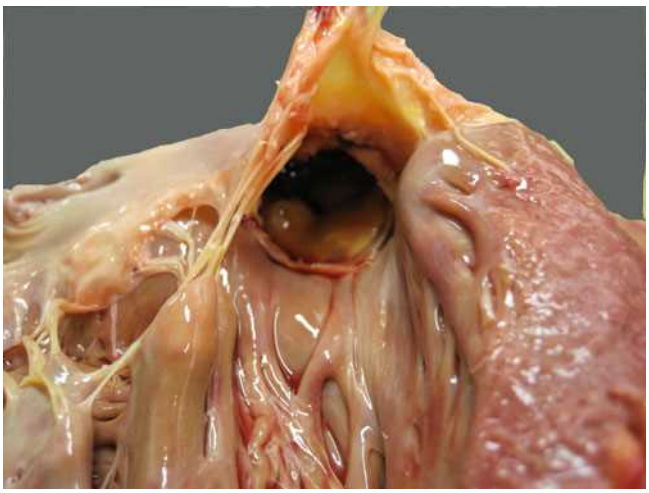

**Fig. 4.229** In this case, an aortic valve replacement is evident. It can be inspected initially from below by lifting the mitral valve tissues upwards, thereby allowing direct inspection of the valve parenchyma

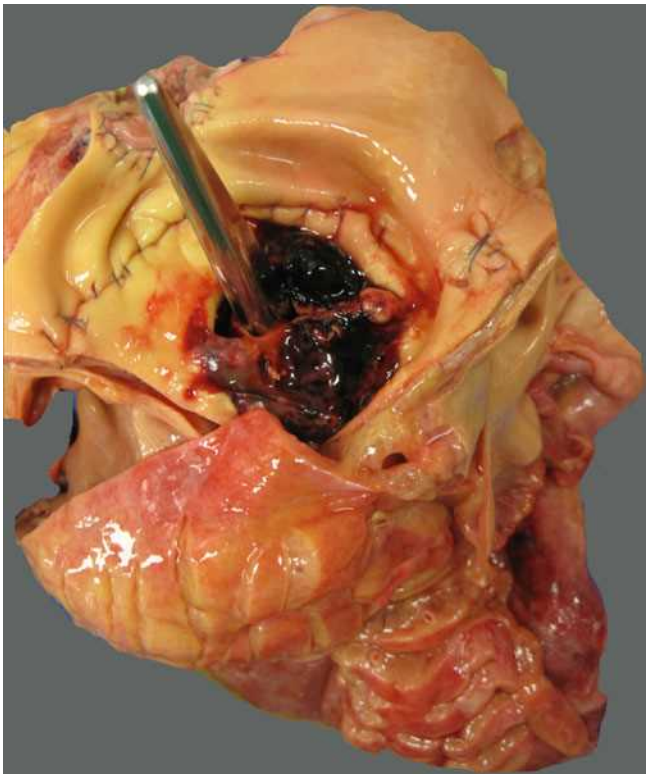

**Fig. 4.230** The valve patency can be assessed gently, probing the valve prosthesis by a small finger or closed scissors. One should be careful not to dislodge any vegetations or adherent thrombus

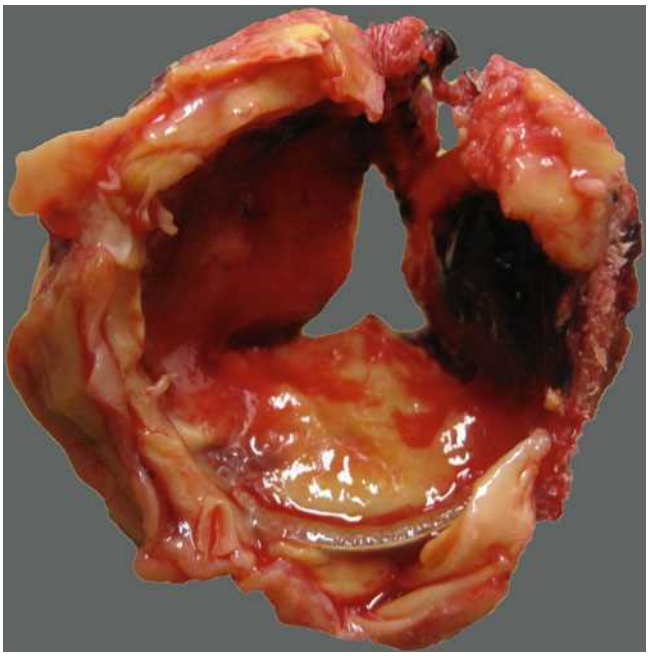

**Fig. 4.231** The valve xenograft has now been dissected free and can be closely inspected from above and below

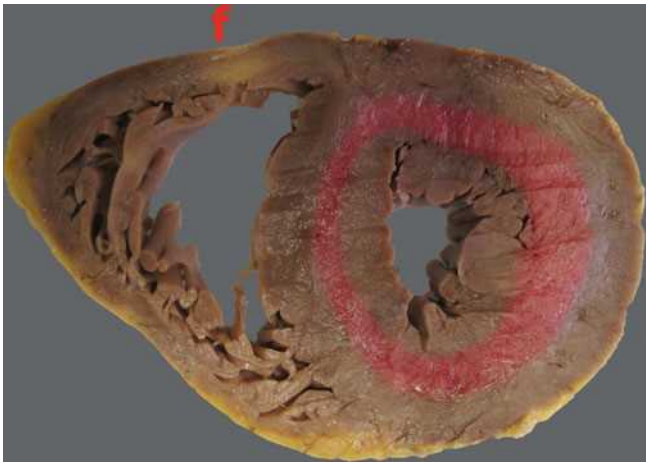


**Fig. 4.232** The macroscopic appearance of arrhythmogenic right ventricular cardiomyopathy is often first suspected using the transverse ventricular slice, which highlights an unusual accumulation of fat (*f*) and a large amount fatty tissue in the wall of the right ventricle

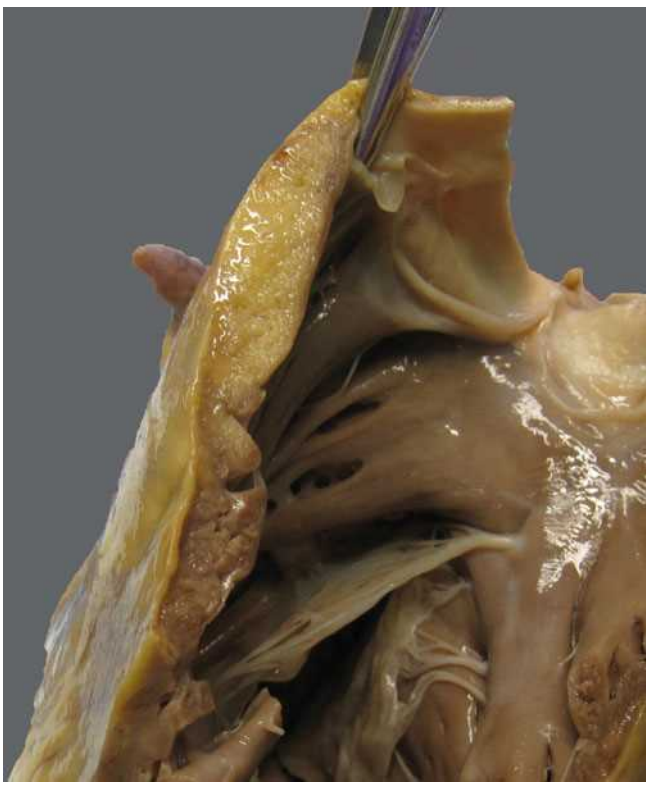

**Fig. 4.233** Arrhythmogenic right ventricular cardiomyopathy, showing pronounced fatty tissue replacement in the right ventricular outflow tract. This tissue is often most marked in the outflow tract

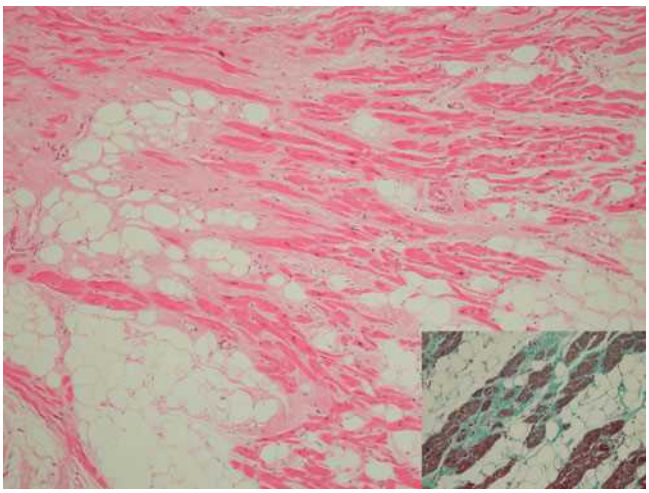

**Fig. 4.234** The histology of arrhythmogenic right ventricular cardiomyopathy shows pronounced fatty and fibrous tissue replacement of the cardiac myocytes. The inset at lower right shows the same reality using the Masson trichrome stain

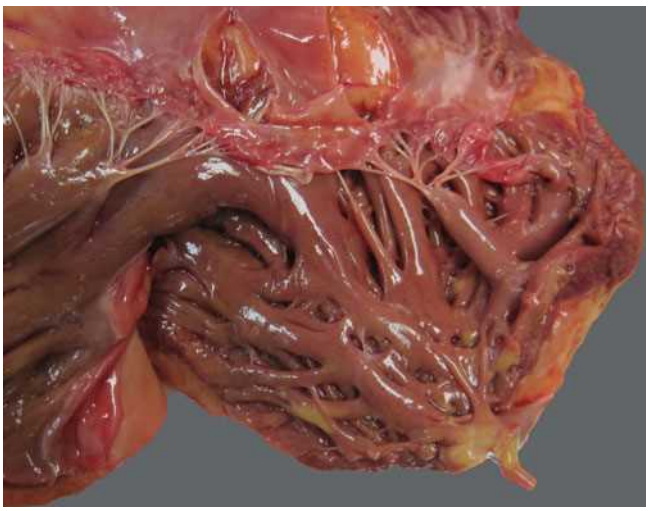

**Fig. 4.236** The so-called 'tabby-cat' heart is characterized by yellow flecks of fatty tissue, particularly on the endocardial aspect of the ventricular parenchyma. This is a normal reflection of increased age, with a female sex bias. It is very mild in most cases. When marked, it can provoke consideration of arrhythmogenic right ventricular cardiomyopathy

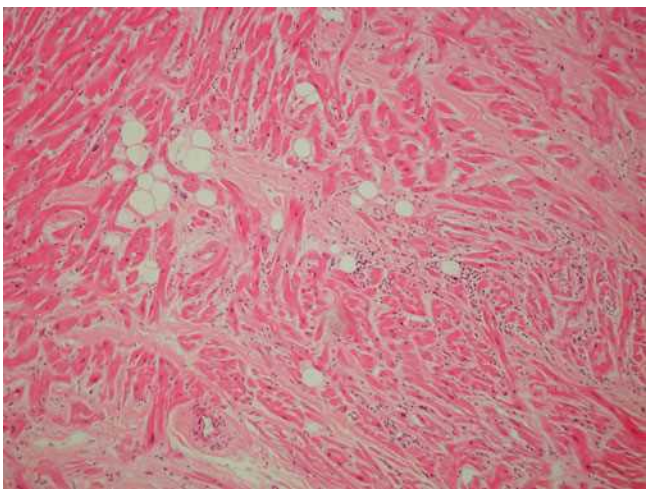

**Fig. 4.235** It must be remembered that finding some fat and fibrous tissue in cardiac tissues is not automatically indicative of arrhythmogenic right ventricular cardiomyopathy. This case showed some non-specific features in a patient dying from metastatic neoplasia

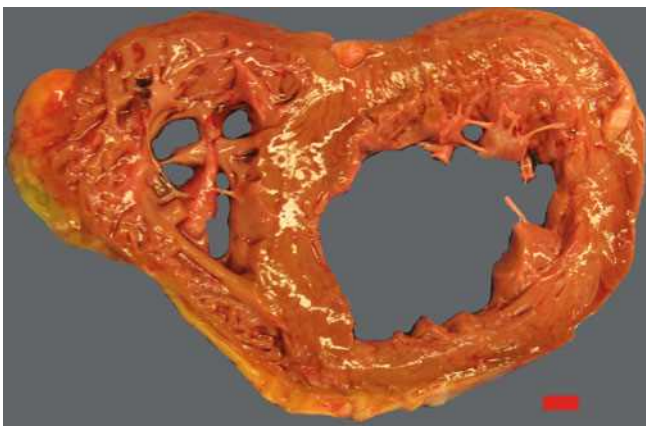

**Fig. 4.237** Dilated cardiomyopathy is best perceived from the ventricular transverse slice, in which the transverse diameters of the right and left ventricles can be appreciated. It is possible to define a variety of different transverse diameters depending on the plane of measurement taken, but the largest should be used

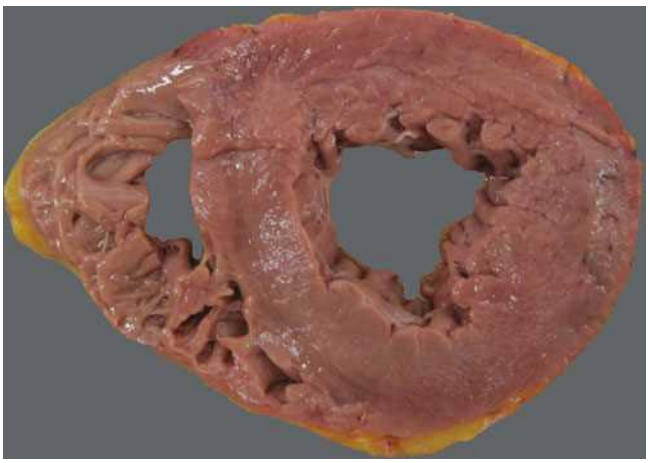

**Fig. 4.238** Peripartum cardiomyopathy can occur in the peripartum and immediate postpartum period. This rare condition can be fatal owing to rapidly progressive dysfunction of the heart. The macroscopic and histological features are not specific. Correlation against the ante-mortem and maternal record is required, along with consideration of the family history. Exclusion of alternative causes of cardiomyopathy is paramount

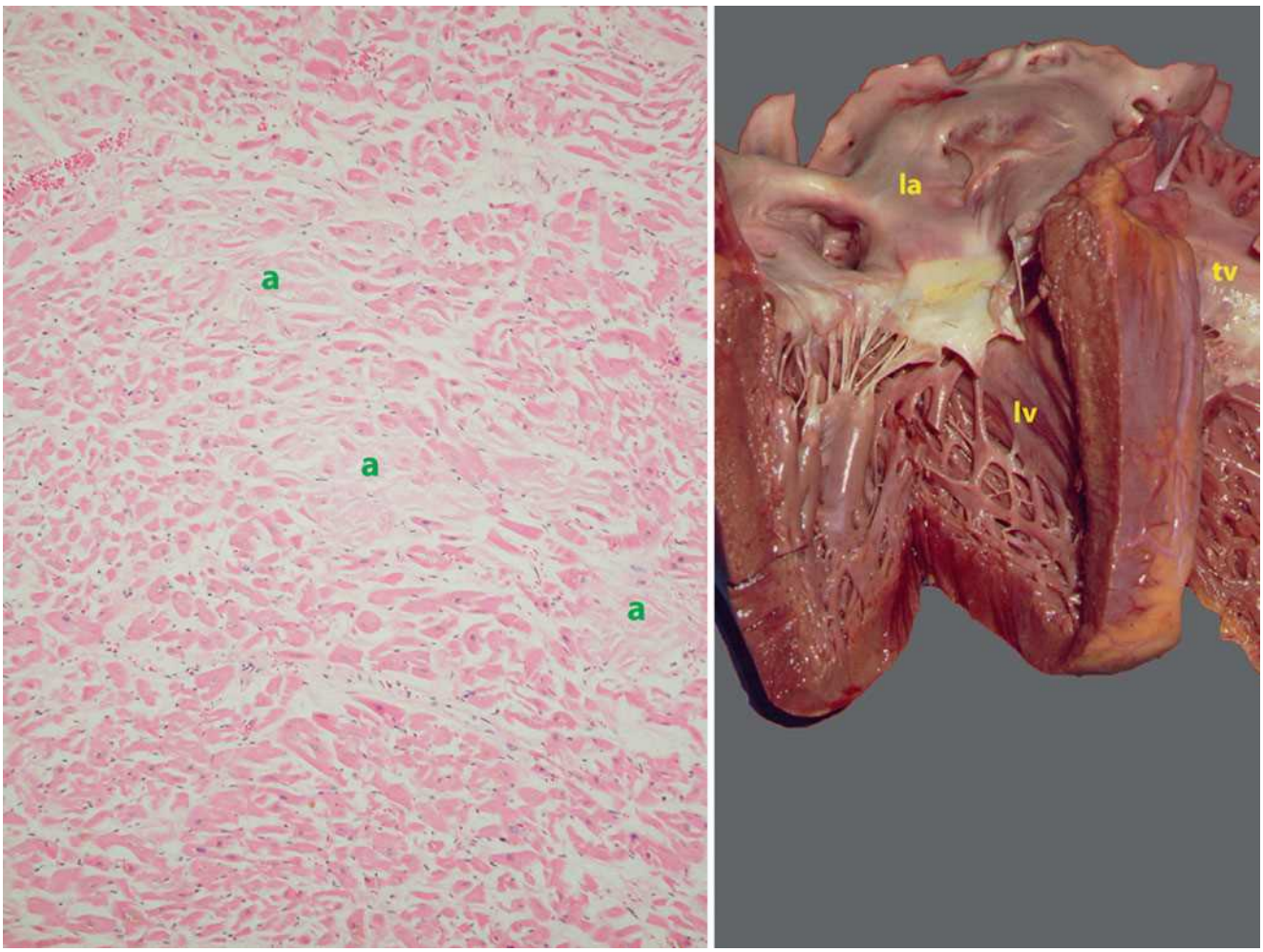


**Fig. 4.239** *Right* cardiac amyloid often may be seen with enlarged, thick, and stiff ventricle tissues (*la* left atrium, *lv* left ventricle, *tv* tricuspid valve). This fresh specimen shows the rigidity of the ventricular parenchyma, despite being sliced open. *Left* histologically, cardiac amyloid can be subtle or marked. The degree of eosinophilic matrix infiltrating around cardiac myocytes, in the interstitium (*a*), and (in other cases) around blood vessels varies. It is often suspected on standard haematoxylin and eosin (H&E) staining, but it is best visualized using special stains (Congo or Sirius Red), or potentially electron microscopy

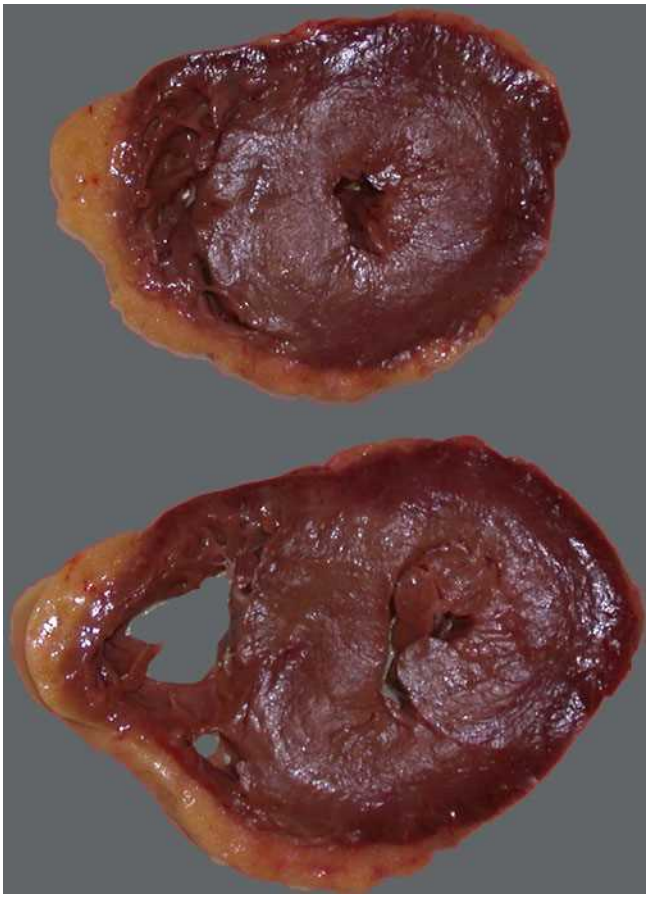

**Fig. 4.240** Hypertrophic cardiomyopathy is often suspected by virtue of the macroscopic features. Transverse sections across the right and left ventricle may show the asymmetry that is suggestive of this condition

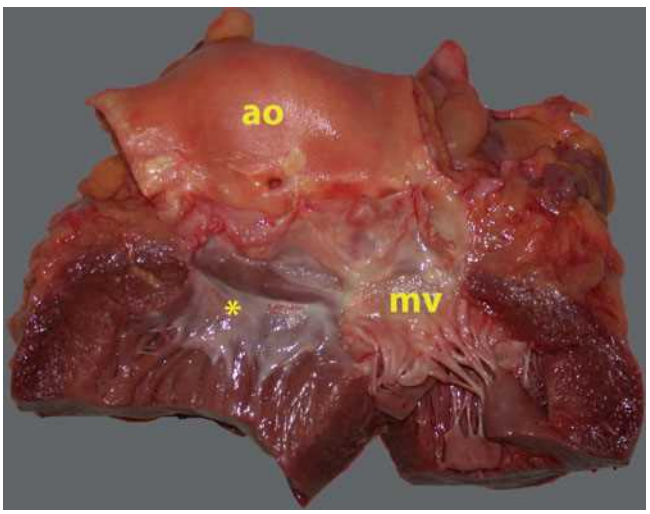


**Fig. 4.241** Hypertrophic cardiomyopathy often shows an impact lesion (*asterisk*) on the endocardial surface adjacent to the anterior mitral valve (*mv*) leaflet. This is often the mirror image of the valve tissue itself, representing localised trauma to the endocardial tissues as the septal tissues are struck by the valve leaflet at the maximal excursion point. The aorta (*ao*) is seen above the left ventricular outflow tract

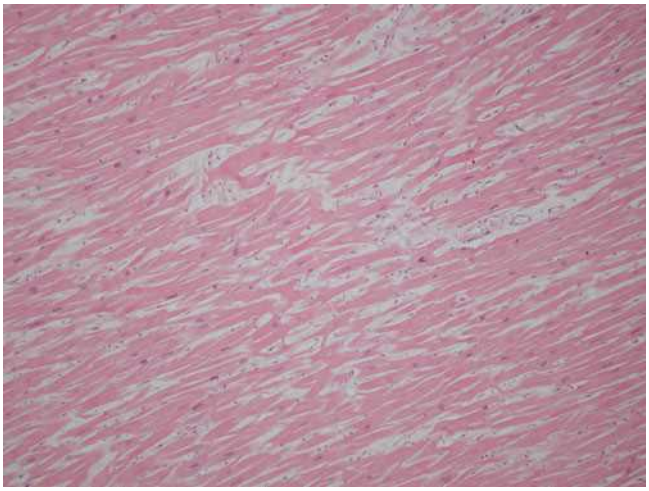

**Fig. 4.242** Hypertrophic cardiomyopathy shows characteristic myocyte disarray histologically. A minor degree of disarray is often seen at the confluence of the right and left ventricle free walls, but finding disarray of this quality in most blocks diffusely across the myocardial parenchyma points towards hypertrophic cardiomyopathy

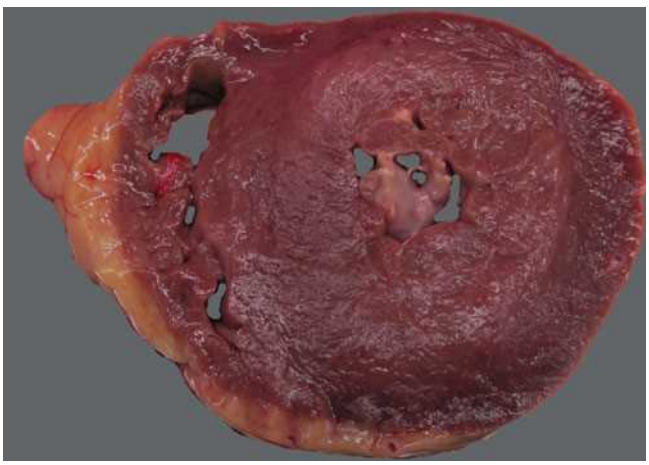

**Fig. 4.243** Hypertension may produce significant left ventricle muscle bulking and mimics hypertrophic cardiomyopathy, but the hypertrophy is concentric. There will be little or no myocyte disarray histologically, but there does appear to be familial linkage in some cases. Idiopathic left ventricular hypertrophy might be considered if there is no evidence of hypertension

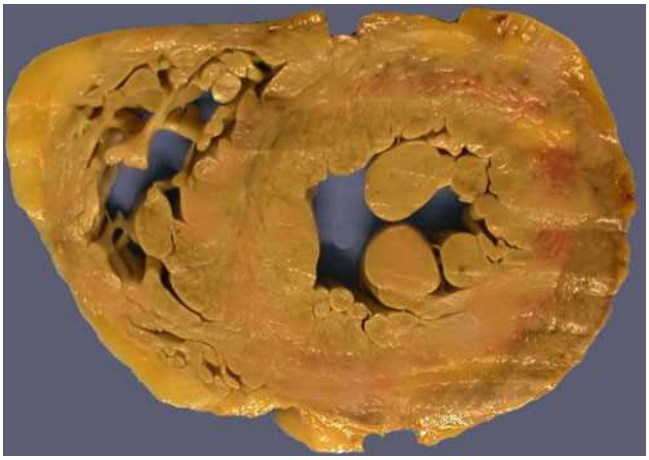

**Fig. 4.244** A transverse cut across the ventricular tissues in a case of Anderson-Fabry cardiomyopathy shows pronounced mural hypertrophy. This condition may mimic hypertrophic cardiomyopathy (Adapted from Keeling L, Suvarna SK, Hughes DA. Female Anderson-Fabry disease mimicking hypertrophic cardiomyopathy. J Clin Pathol. 2012;65:377–8; with permission)

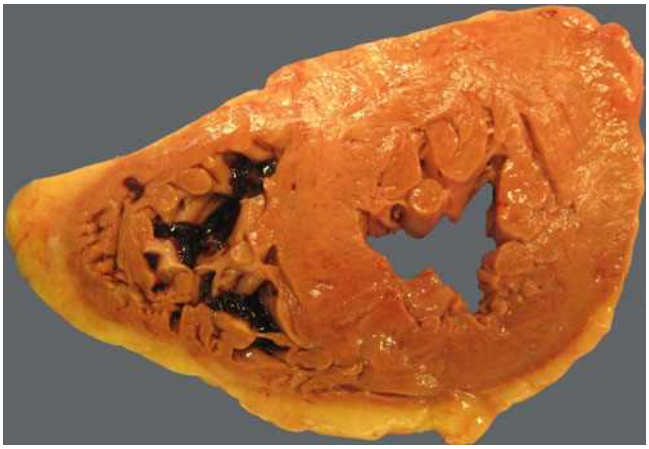

**Fig. 4.246** Cases of inherited channelopathy, manifesting as unexpected sudden death, occasionally may appear in the autopsy room. The gross examination and histology are unremarkable. Ancillary tests (microbiology, toxicology, and serology) are negative. This case, in which the individual died suddenly while at rest, involved a known Brugada-type mutation, emphasising the need for reserving splenic or other tissue for subsequent DNA analysis

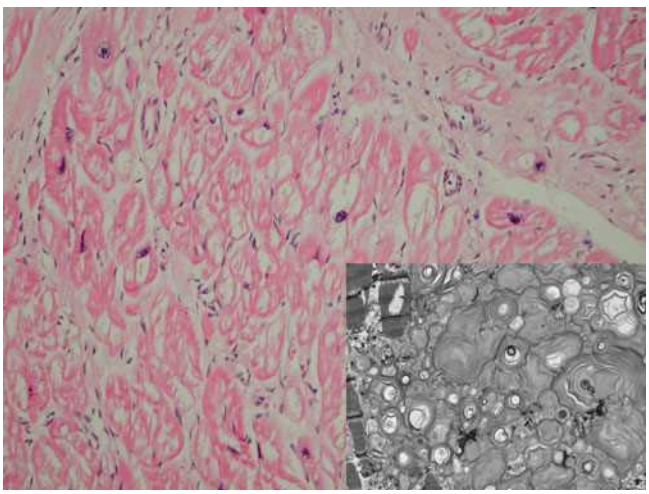

**Fig. 4.245** Light microscopy of the Anderson-Fabry heart shows some fibrosis and variably vacuolated cardiac myocytes. Electron microscopy (*inset*) reveals the classic laminated inclusions often best seen in lysosomes of glycolipid material

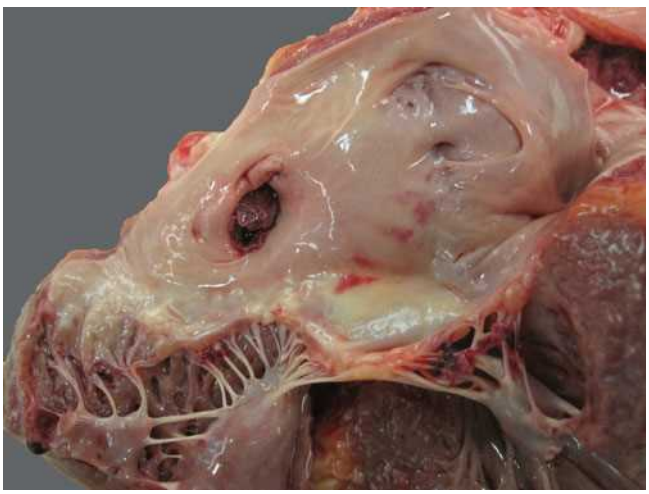

**Fig. 4.247** Many cardiomyopathic conditions—and indeed secondary degenerative disorders of the myocardium—are often associated with atrial fibrillation. Thrombus in the appendages can be seen in such circumstances and provides a risk for systemic artery embolization

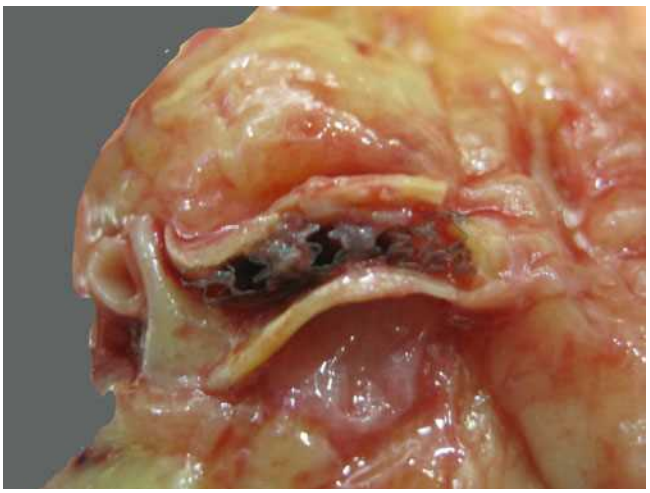

**Fig. 4.248** Coronary artery stents can thrombose, particularly in the time immediately after implantation. This deroofed stent has no significant thrombus. This technique is good for demonstration purposes but is rather fiddly and likely to damage the wire framework

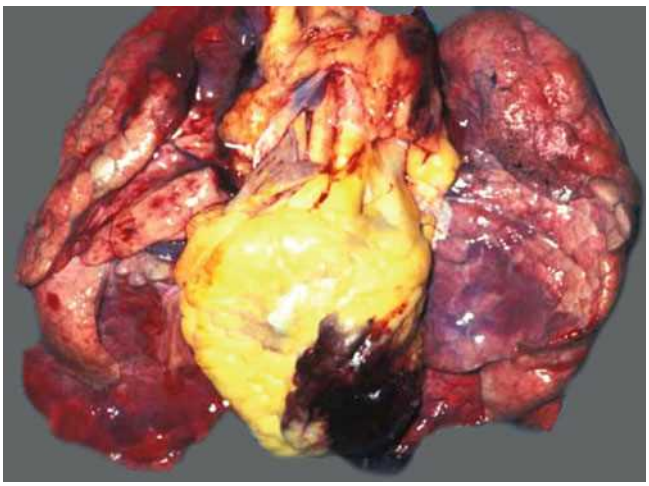

**Fig. 4.250** Coronary stenting can cause rupture of the artery with secondary local haemorrhage and cardiac tamponade resulting in death. This case shows distal left anterior descending artery damage in a case of coronary artery disease stenting (From Suvarna SK, editor. Cardiac pathology: a guide to current practice. London: Springer-Verlag; 2013; with permission)


**Fig. 4.249** Extraction of the stent in one piece allows confirmation of thrombus within the stent. An additional technique for assessing the stent sometimes is to pass fluid along the course of the coronary artery and confirm the lack of distal flow along the artery (From Suvarna SK, editor. Cardiac pathology: a guide to current practice. London: Springer-Verlag; 2013; with permission)

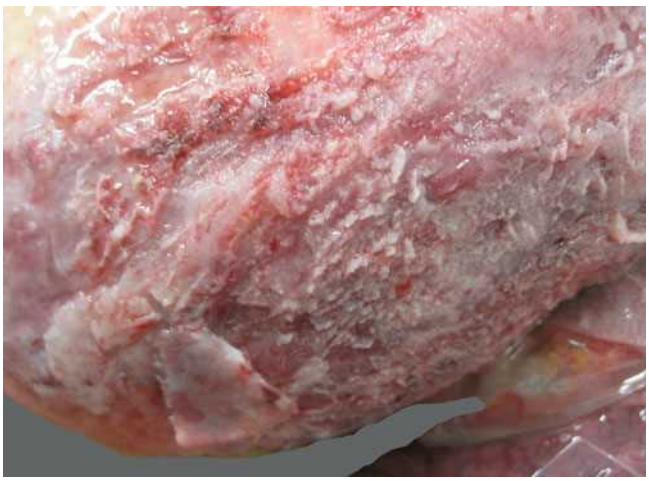

**Fig. 4.251** Extensive pericardial roughening due to fibrin deposition is seen soon after valve replacement

**Fig. 4.252** Markedly heavy haemorrhagic pericarditis is seen shortly after complications following cardiac surgery. This pericarditis may have a tamponade effect and provide a locus for sepsis. In this case, the organising clot matrix completely obliterates the external view of the heart tissue. Carefully teasing this material away from the heart surface allows standard cardiac tissue examination to follow

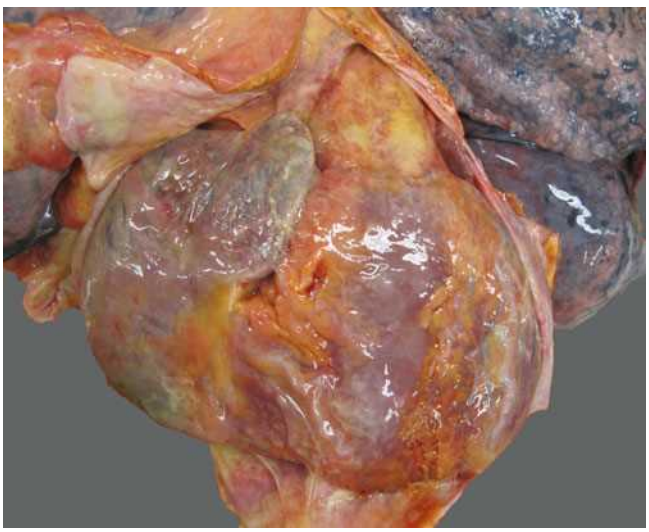

**Fig. 4.253** Chronic fibrotic pericarditic change following coronary bypass surgery, performed about 1 year previously. The pericardial sac is bound to the epicardial surface. Note that the sac is adherent to the heart surface but can be separated with patience

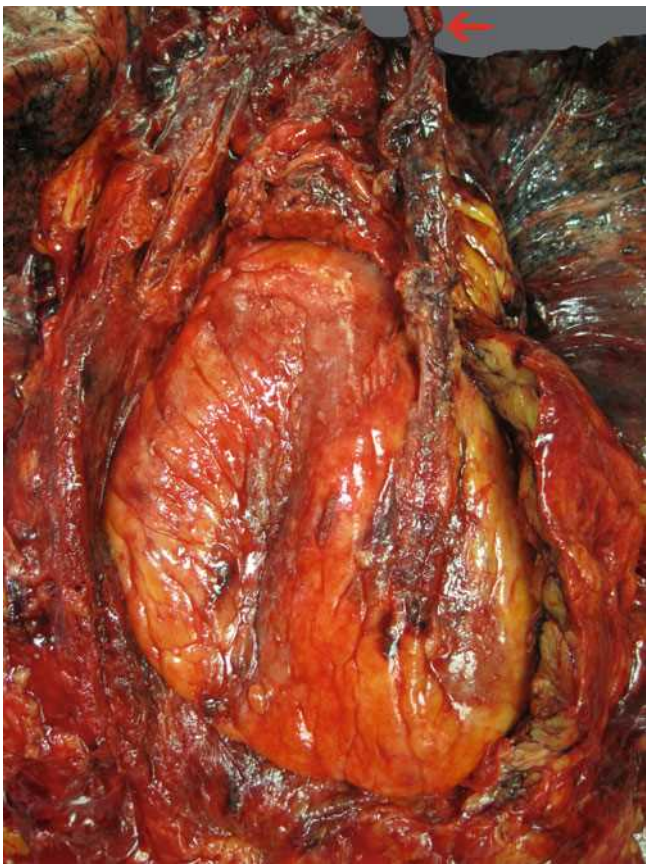

**Fig. 4.254** Examining the heart in a death shortly after surgery often requires inspection of the connections in situ. This case demonstrates the left internal mammary artery (LIMA) branch (*arrow*), running onto the left anterior descending artery from the undersurface of the sternum/chest wall. If one is to assess the patency of the bypass artery tissue, careful dissection of the ribs and sternum is required

**Fig. 4.255** This heart has had bypass surgery using vein grafts harvested from the leg. The patient had complications almost immediately after surgery, and fresh haemorrhage in the soft tissues is noted. Blue pacing wires are also present focally

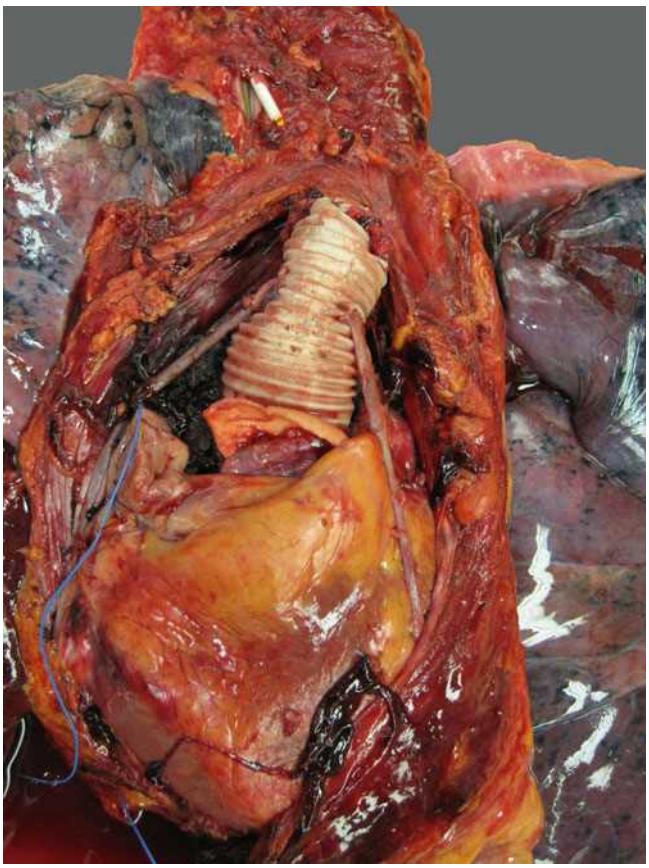

**Fig. 4.256** Coronary bypass surgery has taken place, with an aortic valve/root replacement. Examination of the tissues before manipulation and evisceration is vital for full assessment of the vascular modifications

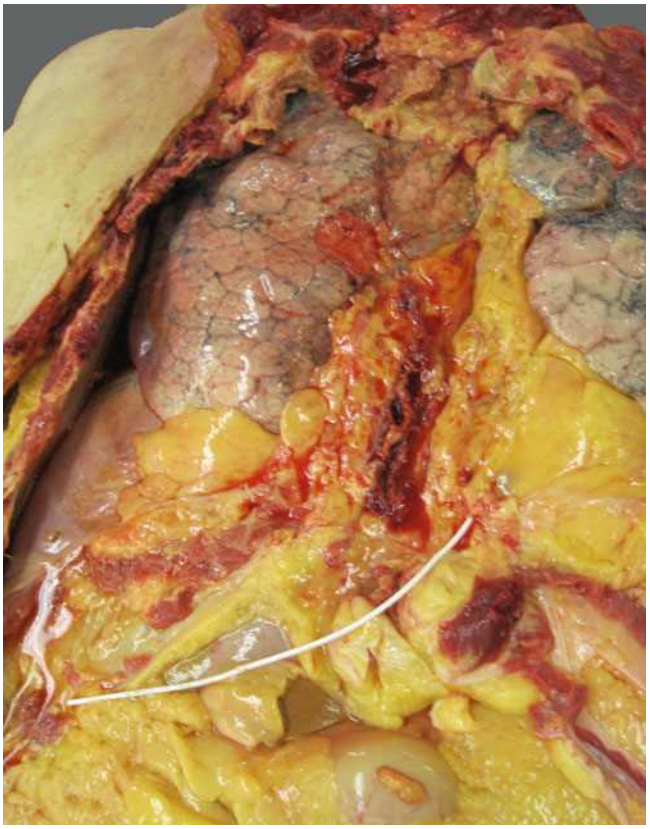

**Fig. 4.257** Pericardial collections may need drainage, if there is tamponade. Various routes can be used to perform the drainage, but inserting such lines is not without risk. This image shows an anterior subxiphisternal approach. Tracing a pericardial drain is important if there is any possibility that the drain insertion has adversely affected the heart or other tissues. In such circumstances, it is recommended to cut the drain flush with the skin and push the device slightly inwards; then the drainage line is approached from inside the body in order to guarantee the correct alignment and position of any drainage tubes

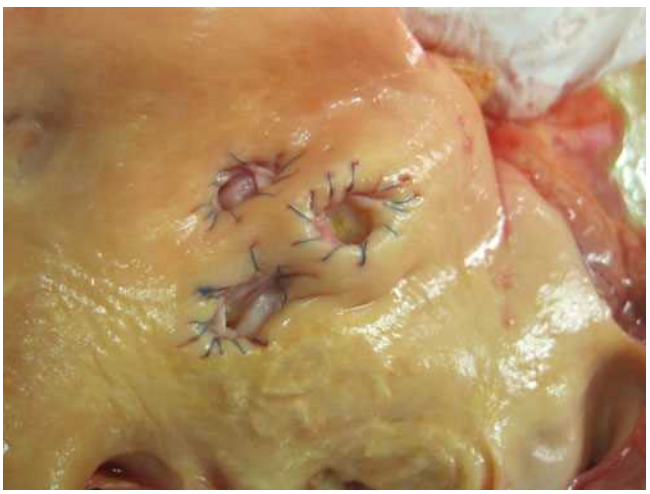

**Fig. 4.258** The origin of vein bypass grafts in the aorta is seen with three closely placed button holes

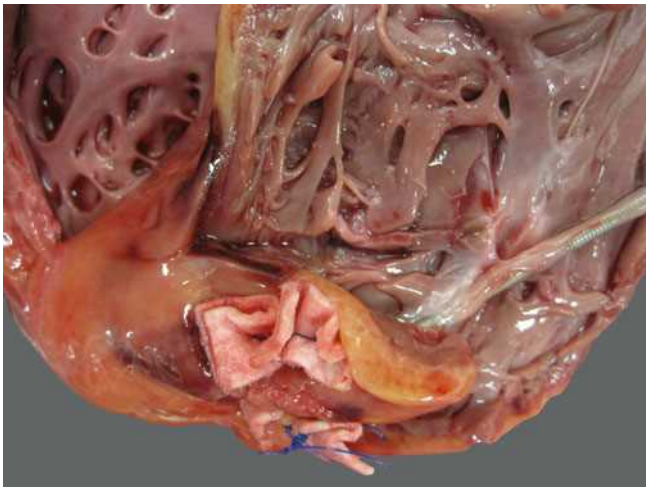

**Fig. 4.259** This is a view of the tip of the right ventricle, which has required open surgical repair with pledgets following an attempt to remove the right ventricle lead. Tearing of the right ventricle at this point caused cardiac tamponade. Although the haemorrhage was stemmed to a large degree, the patient died during the procedure. Close examination of the position of the pledgets and careful probing of the site of perforation should occur in order to test whether any residual leak was present. It is stressed that over-pressure by the probe on the right ventricle wall will always create a false dissection tract, so only gentle probing should be used

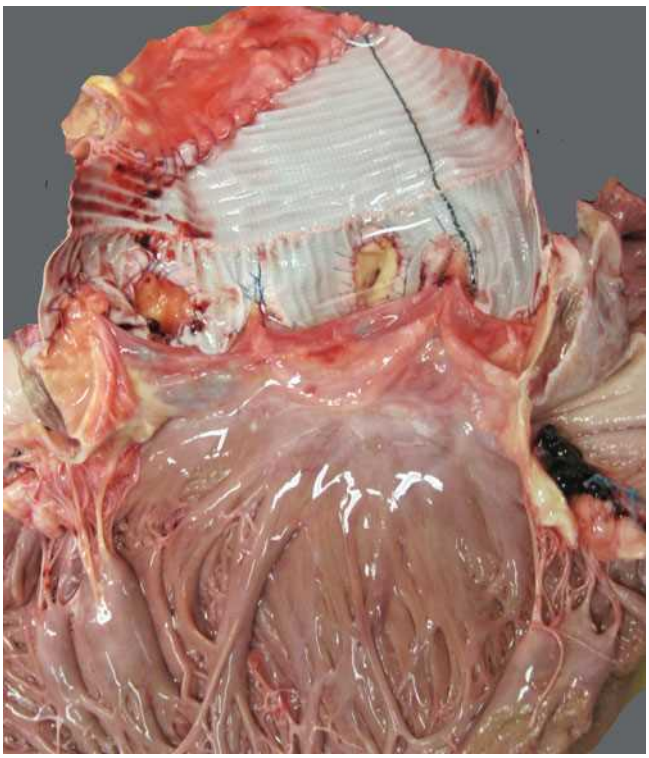

**Fig. 4.260** An aortic root graft is seen with button-holes for the coronary arteries. The Dacron root graft has intact suture lines. This patient died from complications outside the heart shortly after surgery. Autopsy examinations in such cases require careful consideration of the surgical materials, to exclude leak points and sepsis

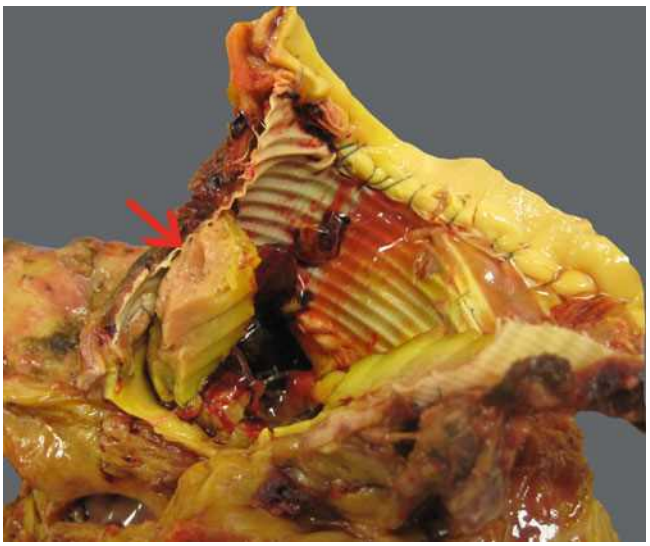

**Fig. 4.261** This aortic root graft shows an infected yellow thrombus (*arrow*) on the luminal aspect of the Dacron graft

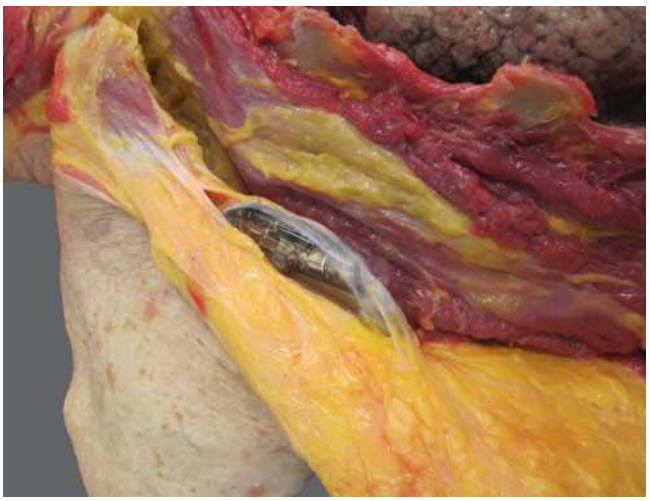

**Fig. 4.263** A pacemaker is seen in its fibrous walled 'pocket' on the upper chest. There is no evidence of sepsis


**Fig. 4.262** Pacemakers have changed in design, shape, and complexity over many years. A variety of pacemakers are seen in this view, for comparison with an old design (*top left*). By Modern devices are relatively thin and light. Most are designed simply to check for gaps in the cardiac cycle and fill in the necessary electrical stimulus, if missing. Examination of the pacemaker will show the device, manufacturer details, and a serial number. This information can be particularly useful in identifying a decomposed body. The leads emerge from one point on the device and pass onwards to the heart. At autopsy, most cases have the generator unit (box) cut free from the wires to allow easy extraction

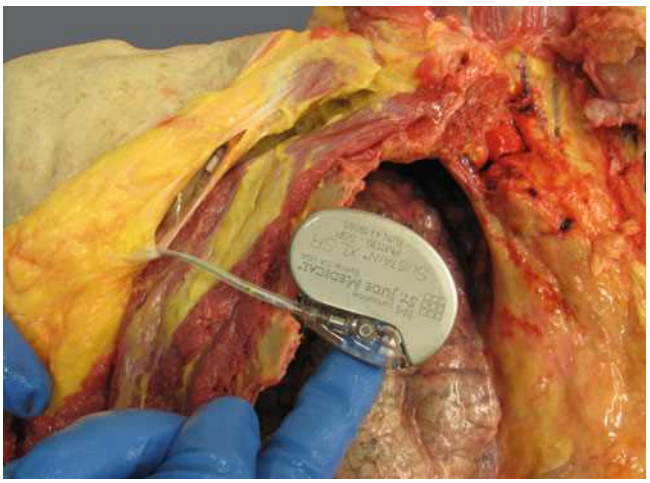

**Fig. 4.264** The pacemaker generator unit has been pulled free, still with the lead attached. Pacemaker devices can come with one, two, or three leads. Most autopsy dissections simply cut the leads about 20–40 mm from the generator (box). It is important to remember that defibrillator pacemakers (ICDs) must be deactivated before wires are cut, in order to prevent accidental discharge of the device affecting mortuary staff

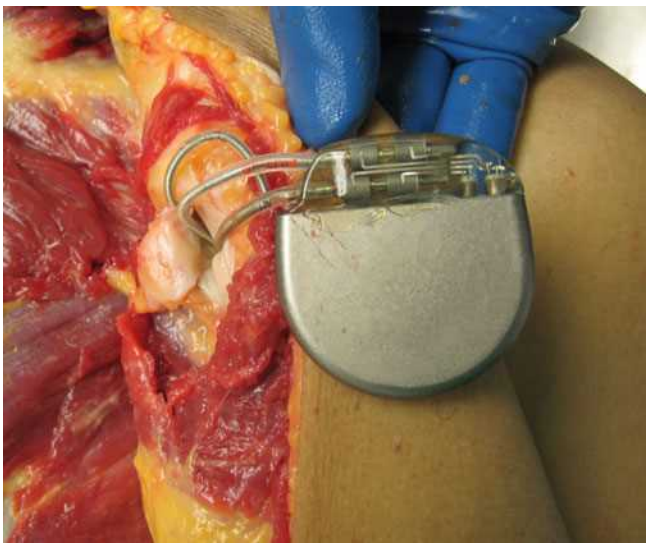

**Fig. 4.265** A pacemaker unit ideally should be inspected in situ before any wires are cut. In this case, the soft tissues have been opened to show the pacemaker pocket to be free of infection. The pacemaker generator unit is seen with the three leads attached

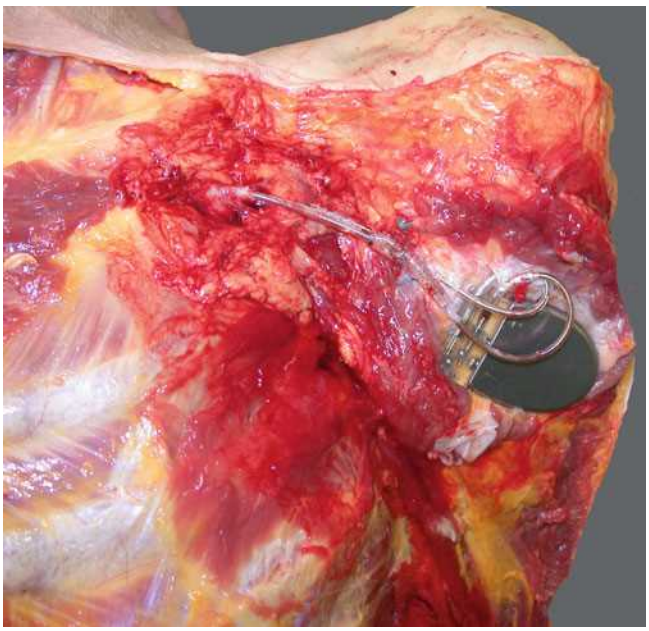

**Fig. 4.266** There is marked fibrosis around the leads of this pacemaker, in keeping with the pacemaker insertion previously. The wires pass normally towards the venous system and may be dissected out without transecting any wires. If one wishes to dissect pacemakers in this fashion, it may be helpful to cut across the clavicle in order to maintain wire integrity before removing the sternum and rib sections

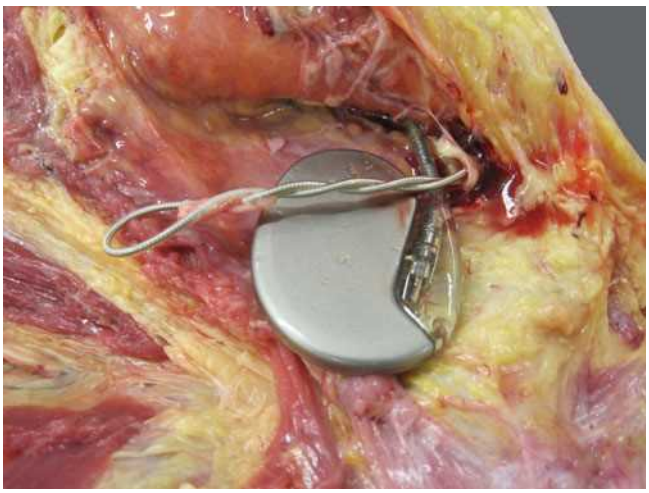

**Fig. 4.267** Extensive twisting of pacemaker leads can be found in some individuals. It can occur naturally, but twisted wires may represent a 'pacemaker twiddler', an individual who fidgets with the pacemaker unit under the skin. The wires become twisted, even to the point of dislodging the electrodes from the cardiac attachment site. More importantly, in this case there was a light exudate present, which points towards possible active sepsis at the site. The exudate should be swabbed for microbiology, and histology of the pocket should follow

**Fig. 4.268** A temporary (*lower*) and permanent (*upper*) pacemaker lead electrode. The temporary wire has two electrical connection points, seen by the metal parts; it can be withdrawn with ease from the patient. The permanent pacemaker has a screw insertion point to help anchor it to the endocardial substrate. It is noted that the insulation in this case has been partially pulled back during electrode handling, exposing the inner aspect of the lead

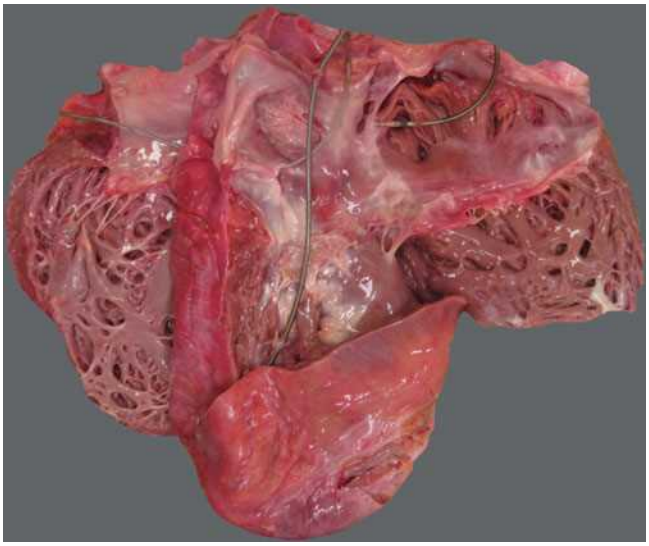

**Fig. 4.269** A three-lead cardiac pacemaker device was present in this case. One lead proceeds to the right atrium. Another passes across the tricuspid valve and runs into the apex of the right ventricle. A third lead is noted to enter the coronary sinus and to run around the great cardiac vein to be attached over the tissues of the anterior left ventricle

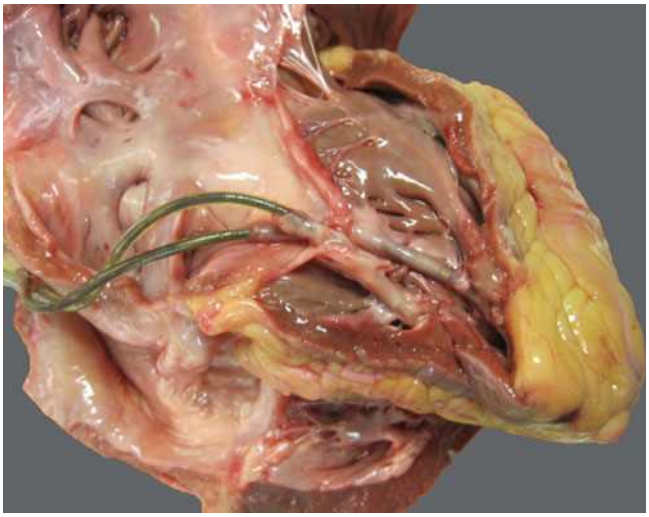

**Fig. 4.271** Pacemaker generator units do need replacement with time, and on occasion the lead will need replacement, but sometimes it can be difficult to remove some pacemaker leads. Specialised tools to extract the leads exist, but some leads are simply left in situ. A second pacemaker lead has been inserted in this case, with the electrode embedded in the tip of the right ventricle. The first lead is noted to have extensive fibrosis binding it to the cardiac tissues

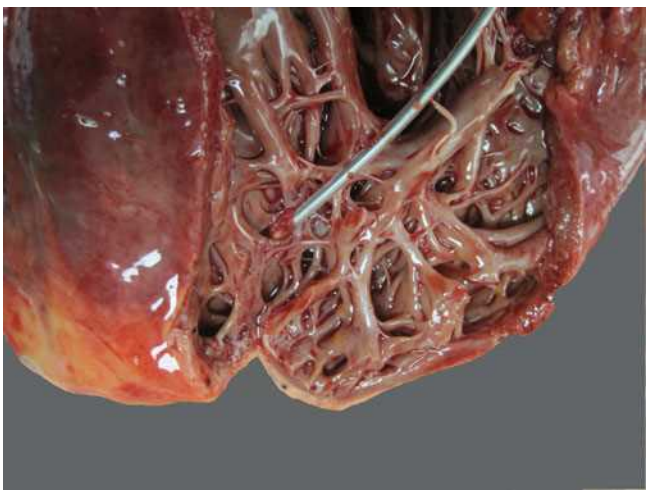

**Fig. 4.270** The cardiac pacemaker lead is seen correctly embedded in the tip of the right ventricle

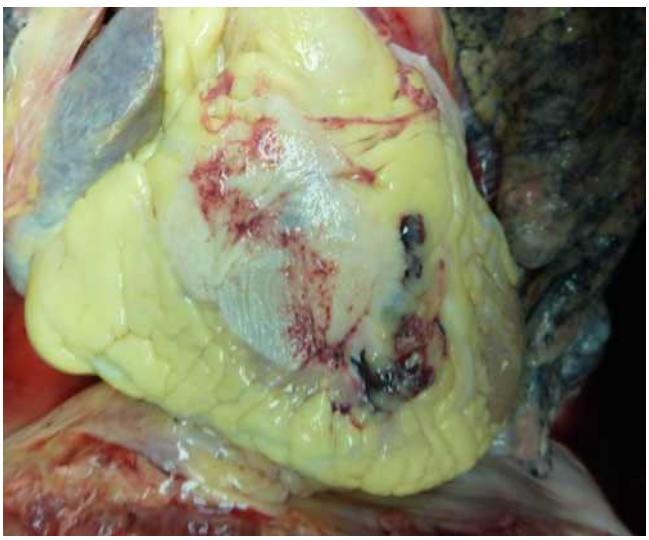

**Fig. 4.272** Placement of a temporary cardiac pacemaker wire lead can be complicated by right ventricular perforation. This case had clear evidence of cardiac tamponade with evidence of haemorrhage and a dissection path close to the apex of the right ventricle

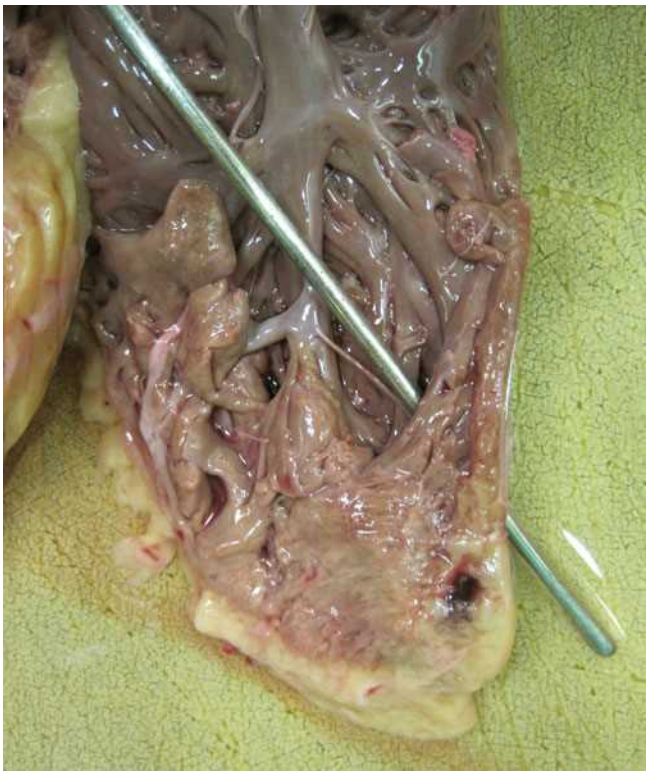

**Fig. 4.273** Another case of temporary pacemaker lead penetration of the right ventricle wall. Transverse slicing of the ventricle should not take place, but dissection along the flow of blood is maintained with the heart intact, thereby preserving the wire connections. Here, the autopsy probe highlights the perforation pathway with some soft tissue haemorrhage adjacent. Right ventricular perforation is probably more common than appreciated, but because the right ventricle is a relatively low-pressure chamber, the perforation can seal relatively promptly with minimal blood loss

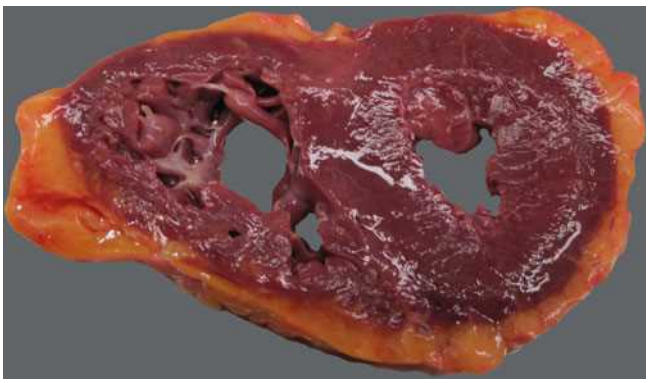

**Fig. 4.274** Cardiac hypertrophy can occur in cases of lung disease, with the increased mass mainly affecting the right ventricle. The comparison ratio of the right ventricle weight against that of the left ventricle and septum (Fulton index) provides an estimate of the degree of right ventricle overload


**Fig. 4.275** The Fulton method uses the heart after dissection and coronary examination. Here, the dissection lays open the heart with the mitral valve bisected, thereby making this weighing protocol easier. The right ventricle (*RV* without fat) is dissected free and compared with the left ventricle and septum (*LV+S* without fat). A ratio of 2.3–3.3:1 for the LV + S: RV is normal

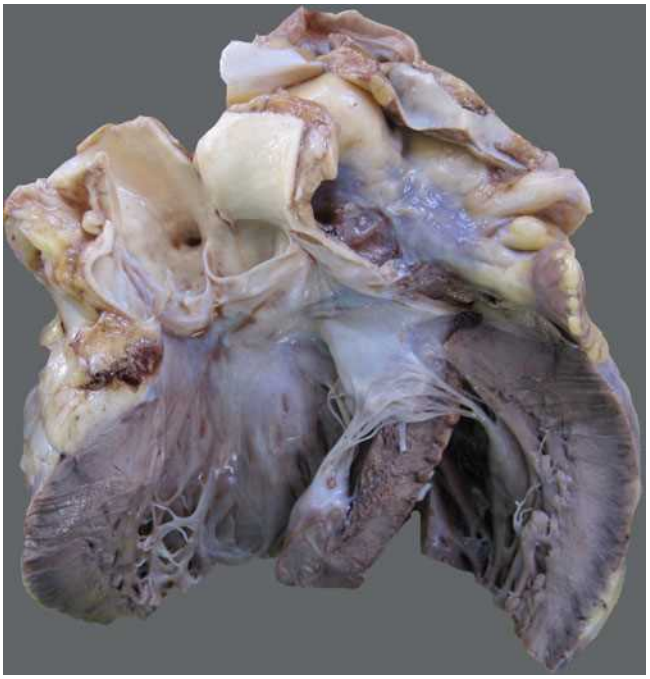

**Fig. 4.276** A fixed heart specimen is seen with a coronary artery anomaly, with only one coronary ostium being present. This underwent bifurcation into the left anterior descending artery and right coronary artery branch, which continued around the back of the heart to supply the posterior wall and ultimately the lateral wall of the left ventricle. At this point, there was left ventricular fibrosis in a patchy fashion, indicative of ischemic heart disease, explaining the sudden death of this individual

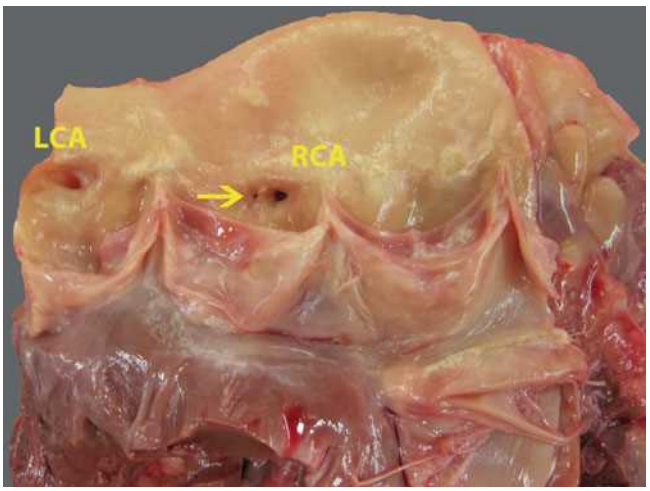


**Fig. 4.277** Conus artery. The aortic valve tissues have the normal arrangement of the origins of the right coronary artery (*RCA*) and left coronary artery (*LCA*). In addition, there is a small artery (*arrow*) next to the RCA. This is the conus artery—a normal variant, not a congenital coronary anomaly

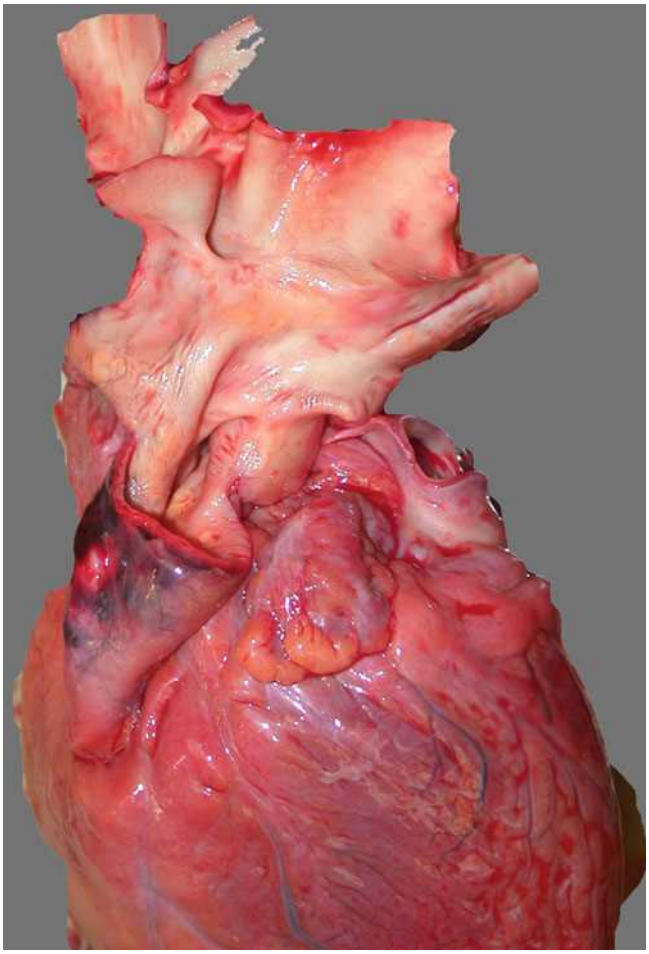

**Fig. 4.278** This is a complex dissection showing both aortic and pulmonary artery tissues that have been opened anteriorly to demonstrate the small area of connection between the two circulation vessels. This is an example of patent ductus arteriosus, which has not been complicated by infective endocarditis

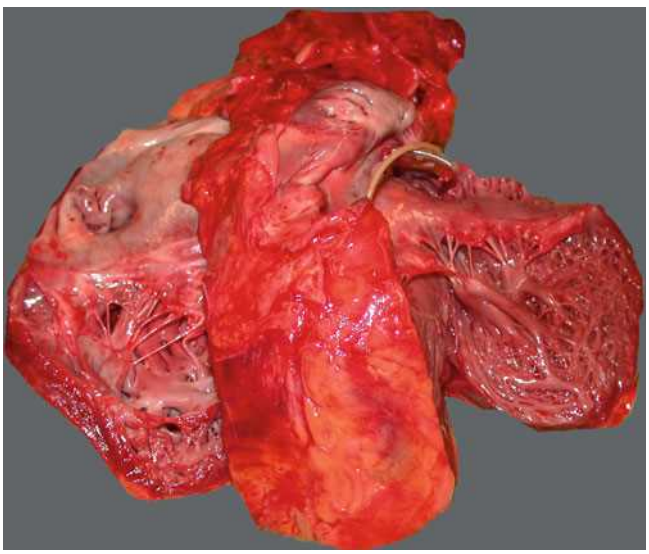

**Fig. 4.279** The increase in successful congenital heart surgery means that cases often live to adulthood. This individual underwent an operation to treat transposition of the great arteries (TGA), in which the morphological right ventricle is on the left and vice versa (looking from the back). The clue is the trabeculation pattern

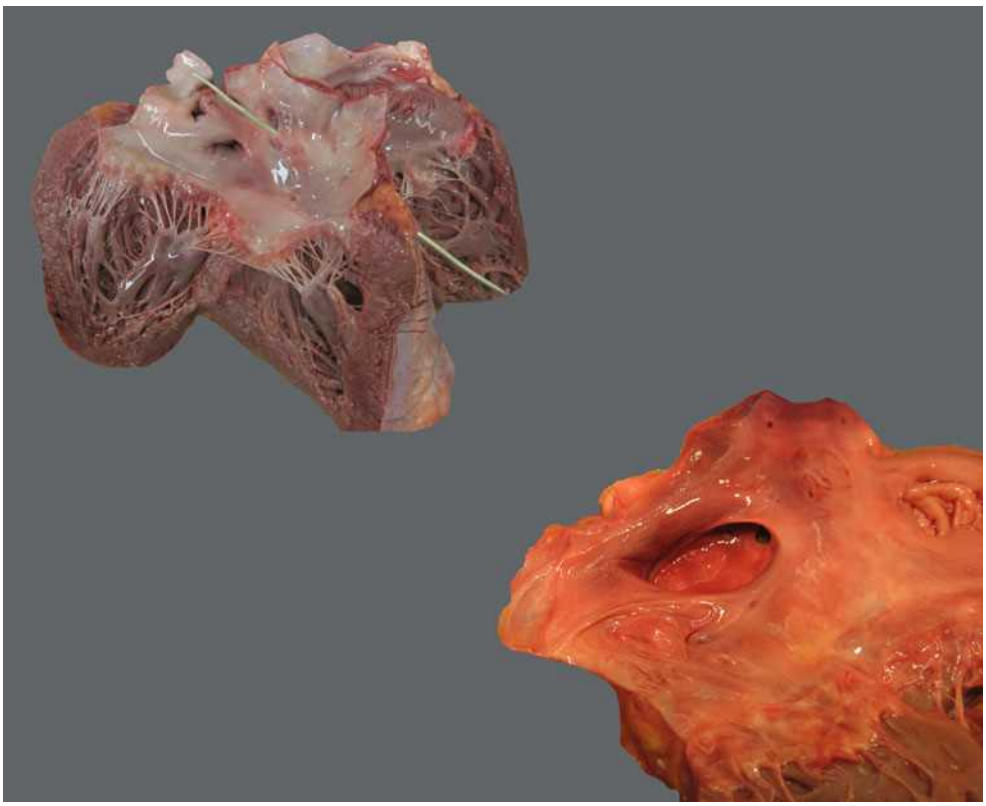

**Fig. 4.280** A probe-patent foramen ovale (*top left*) is not an uncommon feature, occurring in 15–20 % of normal subjects. It generally has no functional significance, although there is a small risk of infective endocarditis. This view shows a probe passed from the right atrium into the left, highlighting the patency of the septal wall. If the natural closure of the foramen ovale has not been complete (*bottom right*), a persistently open rounded defect can be found anteriorly. This defect may be relatively small, but it can have significant flow across it, capable of causing shunting that could end up with functional haemodynamic significance

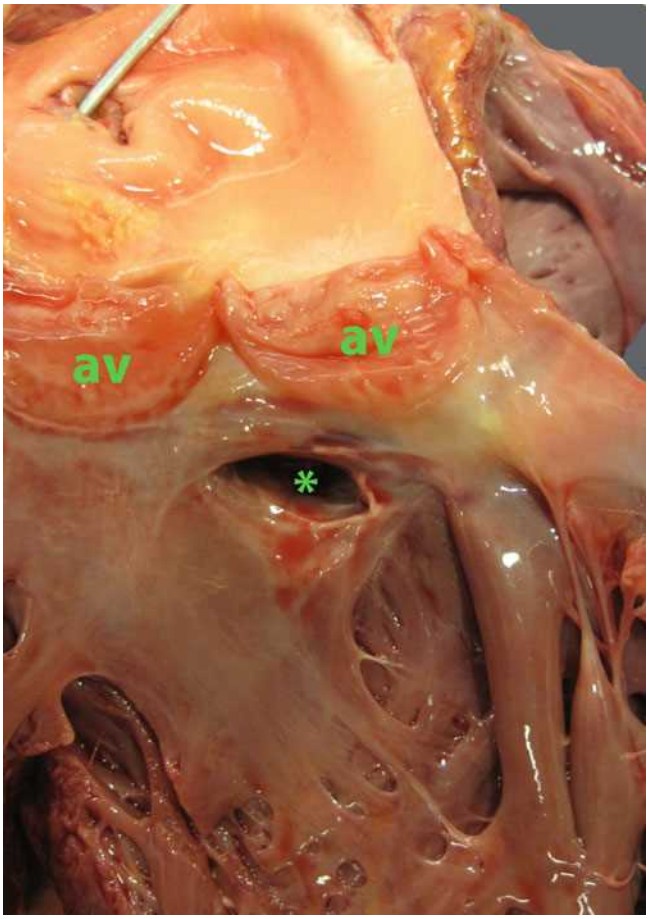


**Fig. 4.281** A ventricular septal defect (*asterisk*) is seen centrally, below the aortic valve cusps (*av*). No vegetation is present. A probe is seen entering the aortic root fistula created between the aorta and pulmonary artery in this treated case of heart disease. Sudden deaths in the second or third decades are common in such cases, even with apparent functional restoration of the congenital lesion

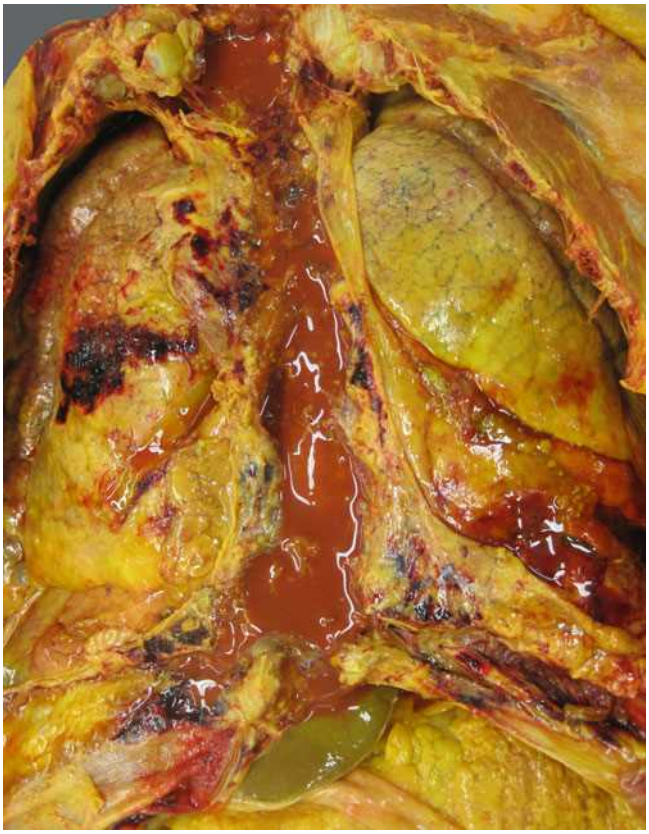

**Fig. 4.282** Infections following surgery for coronary and/or valve surgery are generally bacterial, particularly staphylococcal. Atypical infections, such as *Aspergillus*, can nevertheless complicate various surgical techniques. This case shows purulent collections around the site of prior surgery. Heavy jaundice was also present in this case because of right-sided cardiac failure

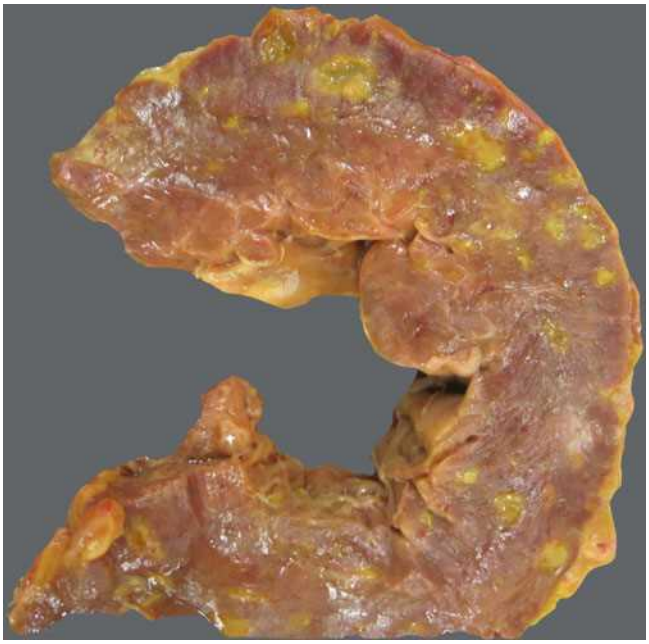

**Fig. 4.283** Seeding of septic organisms into the coronary tissues causes a florid septic myocarditis. The *Aspergillus* infection in Fig. 4.282 was responsible for the fatal suppurative myocarditis

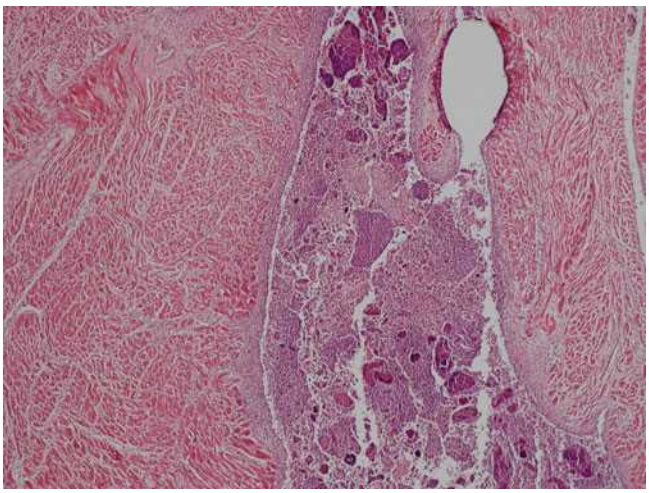

**Fig. 4.284** Perimortem showering of staphylococcal organisms into the coronary circulation (in a case of aortic valve infective endocarditis) has caused a patchy bacterial myocarditis

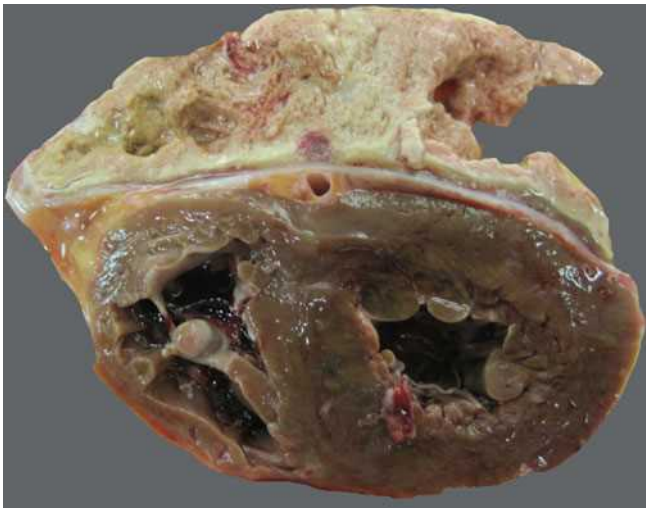

**Fig. 4.285** Rheumatoid arthritis can cause a significant pericarditic reaction. In this view, the right and left ventricles are attached to a florid, necrobiotic, granulomatous reaction, which is limited to the pericardial tissue. The remainder of the inflammatory reaction has been dissected free of the specimen, but clearly this inflammatory/fibrotic process caused significant tamponade

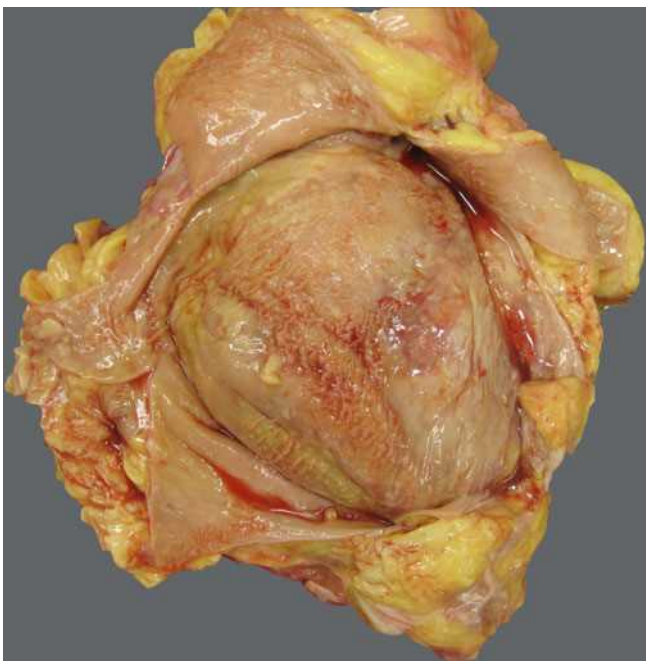

**Fig. 4.286** The profound pericarditis in this case has heavy fibrin deposition. The differential diagnosis would be disseminated neoplasia

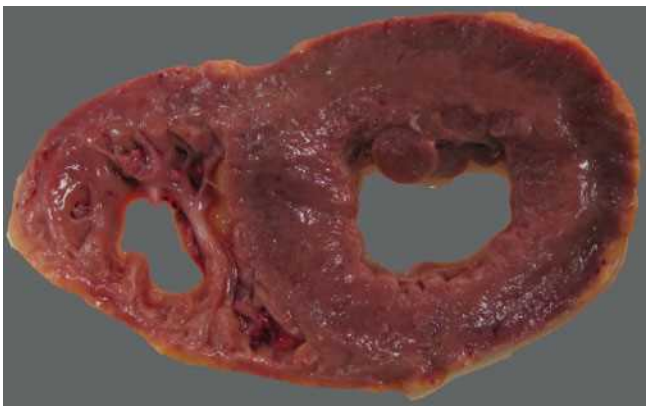

**Fig. 4.287** The macroscopic features of myocarditis are often not evident, although histology will provide the diagnosis. This case of acute lymphocytic myocarditis shows some slight congestion of the myocardial parenchyma in transverse section but no clues as to the diagnosis. This case reflected viral infection

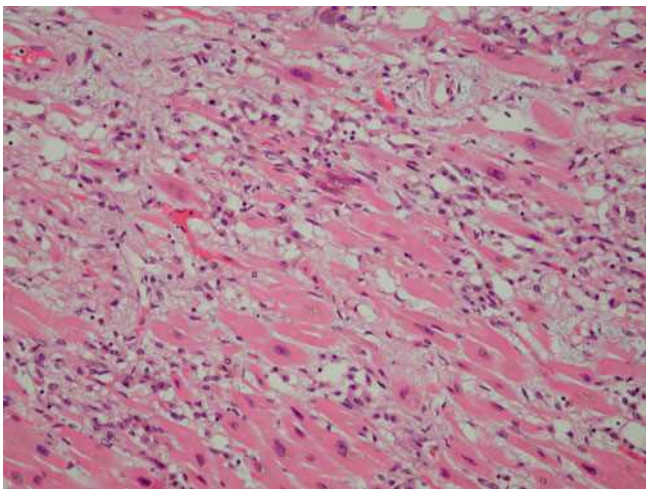

**Fig. 4.289** Marked lymphocytic myocarditis with widespread disruption and damage to individual cardiac myocytes is seen in this histological section

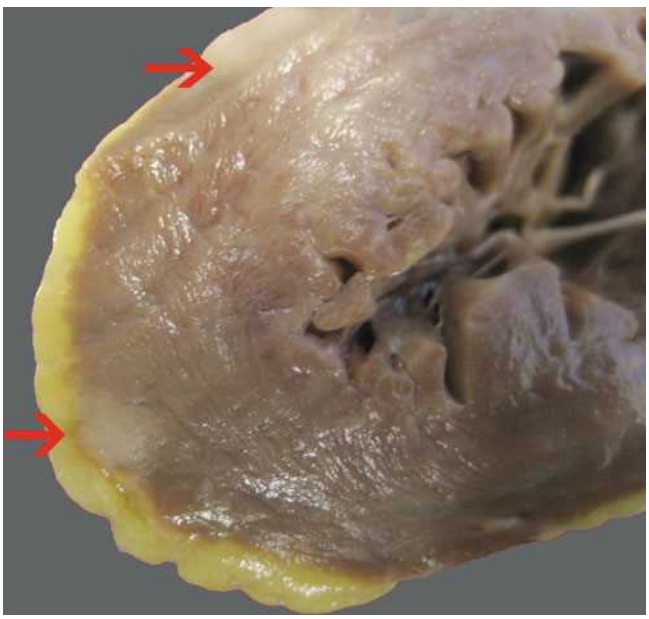

**Fig. 4.288** Giant cell myocarditis in a primary presentation with sudden death is seen with flecks of congestion and fibrosis (*arrows*) in the left ventricle wall

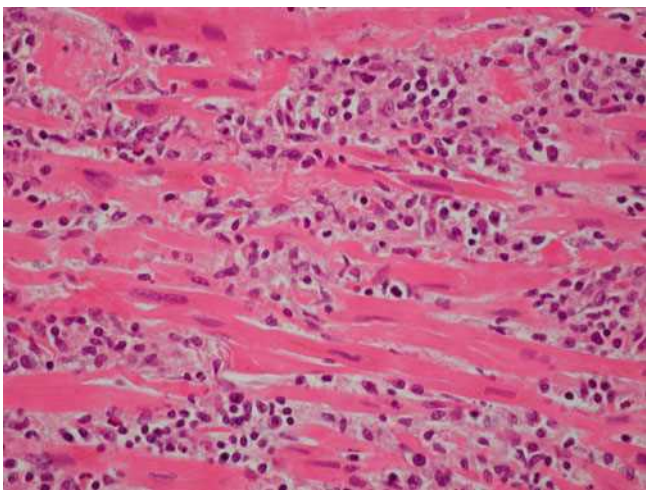

**Fig. 4.290** Myocarditis can be variable in density within the heart tissues. There is infiltration of the interstitium by a variety of inflammatory cells, with separation and lysis of individual cardiac myocytes

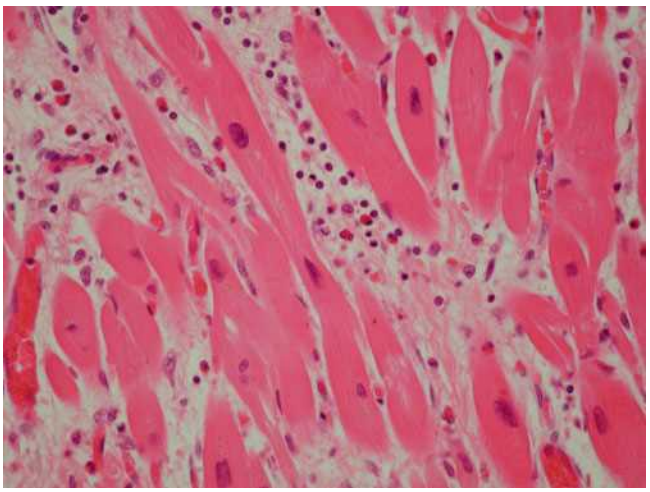

**Fig. 4.291** Classically, lymphocytic myocarditis (associated with viral infections) can cause death, but this case shows a florid, eosinophil-rich myocarditis, which should prompt consideration of atypical infections and allergic phenomena

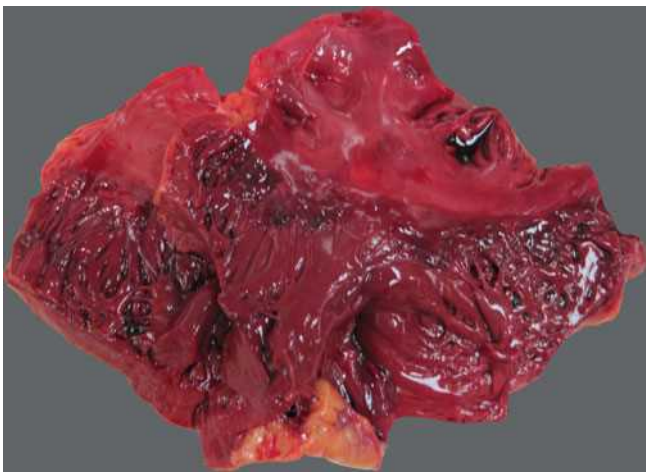

**Fig. 4.293** Terminal bacteremia may cause significant haemolysis. Whilst not specific, the finding of brown staining of the endocardial and arterial intimal surfaces may suggest infection by a group A streptococcal or equivalent species

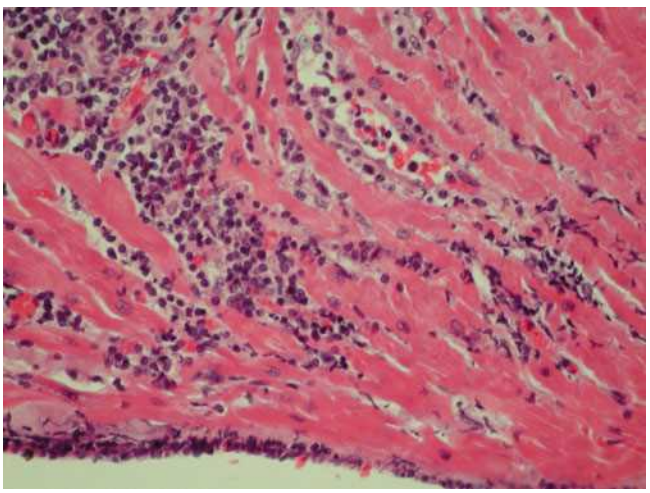

**Fig. 4.292** To an extent, acute cellular cardiac rejection is a form of myocarditis. Significant graft infiltration by activated T cells will cause myocardial damage and potentially sudden death

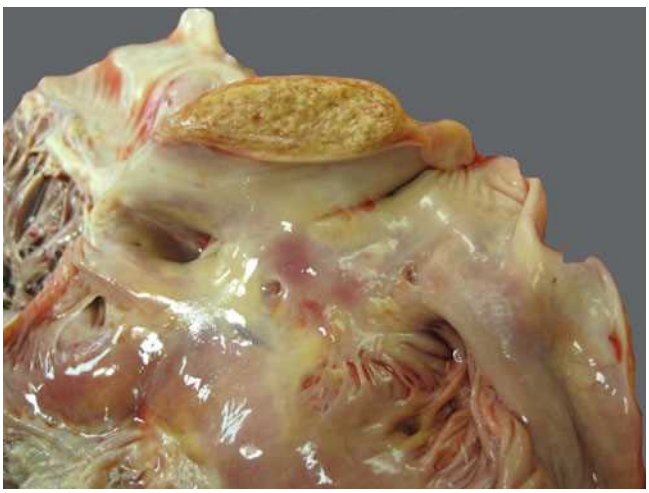

**Fig. 4.294** Pronounced fatty tissue in the wall of the interatrial septum can be seen in cases of atrial lipoma and atrial lipomatous hypertrophy. This lipoma was composed entirely of adipocytes histologically, but the cells were largely of brown fat morphology and it was designated a cardiac hibernoma

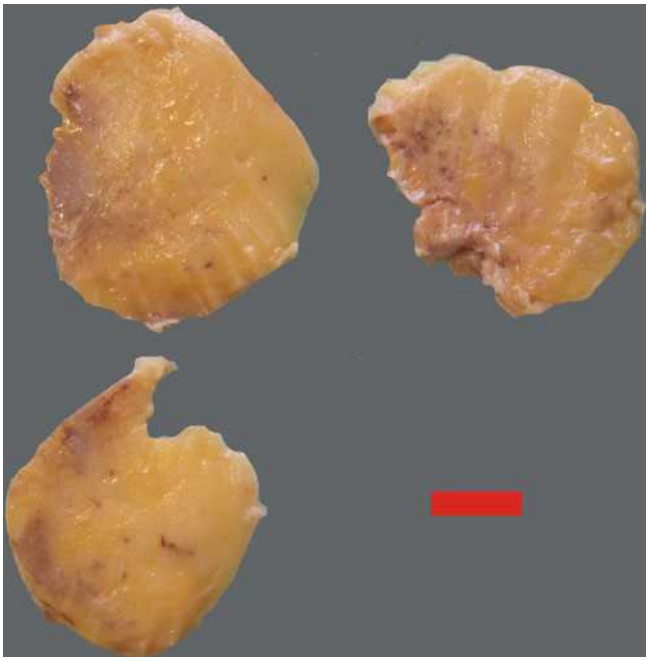

**Fig. 4.295** This surgical specimen shows a standard area of lipomatous hypertrophy of the interatrial septum, which has been resected successfully. The fatty tissue appears uniform for most of the cut surface, although clearly there is more obvious myocardial parenchyma seen peripherally at several points (*Marker* 10 mm)

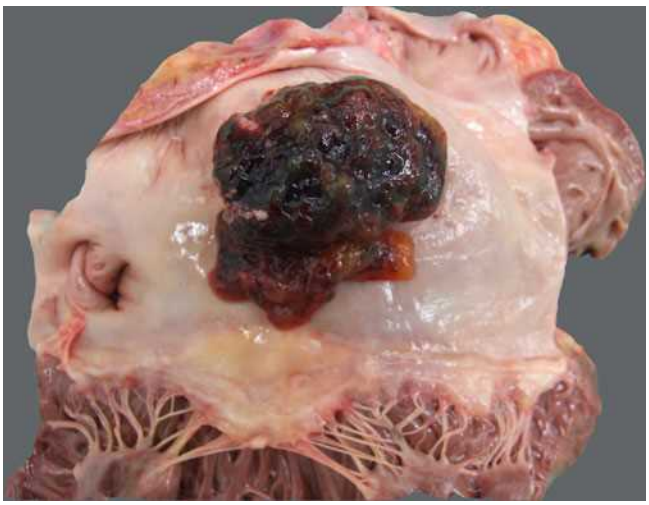

**Fig. 4.296** A cardiac myxoma within the left atrium can be an unexpected finding in the autopsy room. These tumours classically occur on the intreratrial septum, although they may be associated with other sites within the heart, including across the valves, within the ventricles, and occasionally in multiple sites. They are characterized by a soft, jellylike quality, which in this case has been coloured by intra-myxomatous parenchymal haemorrhage. Myxomas can be the source of systemic emboli, and appropriate confirmatory histology may be required (at other sites)

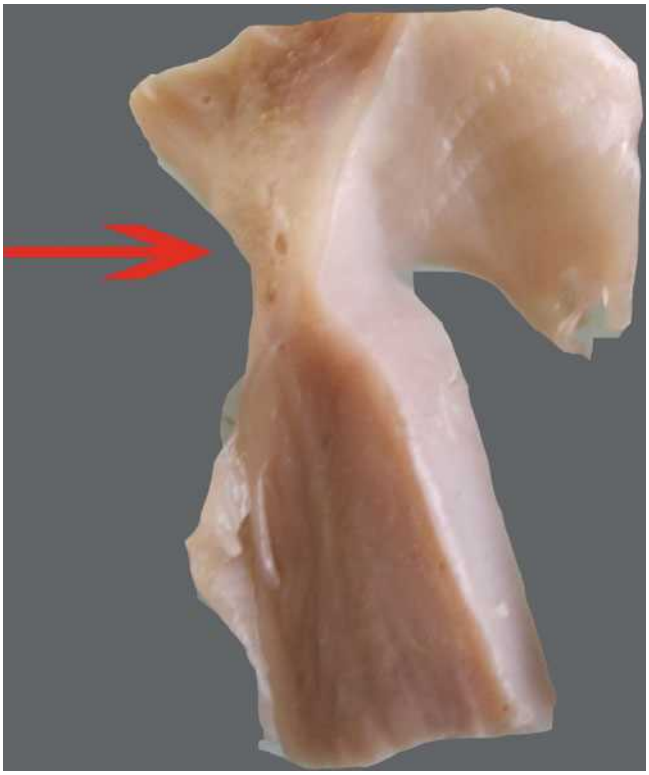

**Fig. 4.297** This close-up view of the membranous septum of a young child shows the characteristic small, cystic spaces of the cystic atrioventricular node tumour (*arrow*). This is a rare cause of complete heart block and potentially sudden cardiac death, both in childhood and in adulthood. It is characterized by epithelial-lined spaces with variable cystic content. It has no malignant potential, but it may be functionally malignant by virtue of posing a risk for sudden cardiac dysrhythmias or death

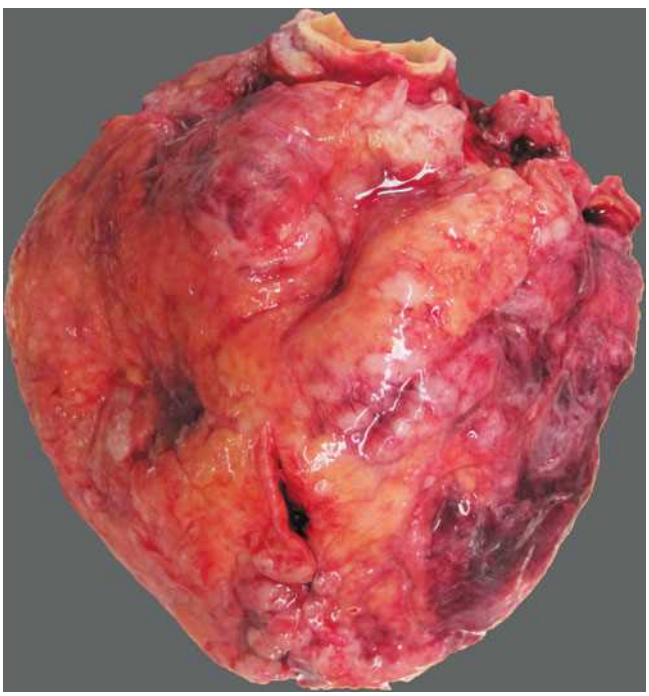

**Fig. 4.298** Mesothelioma of the pericardial sac is a recognised condition associated with asbestos exposure. It will behave exactly the same as pleural mesothelioma, but often it produces a more rapid decline and death, given the involvement of the cardiac tissues

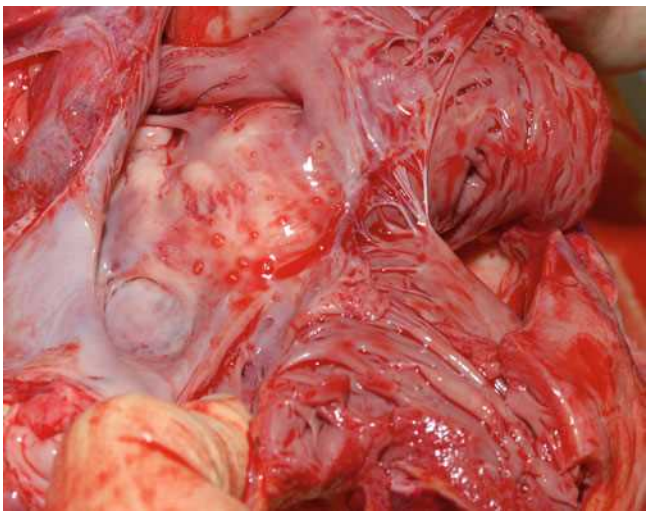

**Fig. 4.299** A close-up view of the free wall of the right ventricle, showing a large tumour mass within the central part of the heart. This was a case of high-grade non-Hodgkin lymphoma. The proximity of the tumour to the conduction system explains the sudden dysrhythmic death

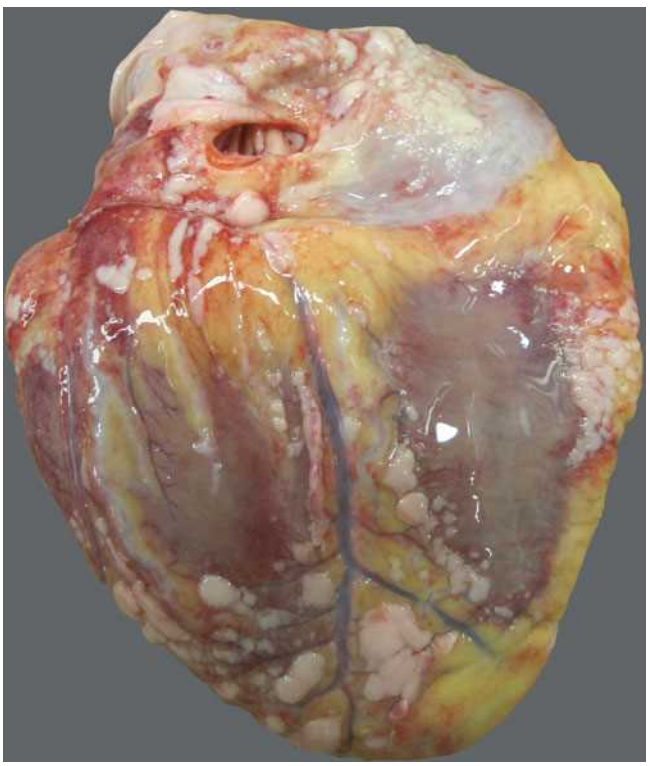

**Fig. 4.300** The heart may be involved by metastatic disease on the epicardial surface. Widespread pulmonary carcinomatous metastases are most marked around the coronary tissues

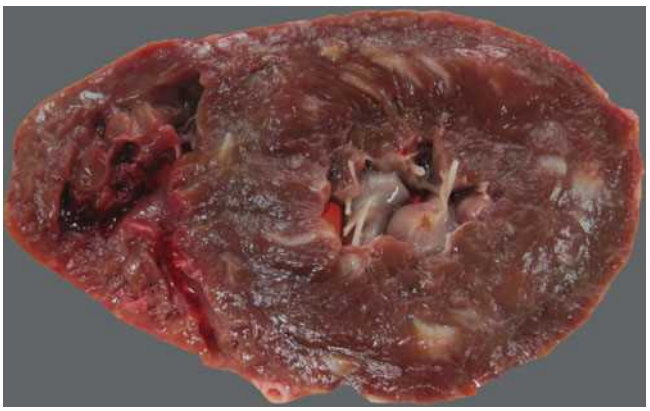

**Fig. 4.301** The heart can be a site for metastasis in many diseases. The left ventricle wall (seen in transverse section) has multiple flecks of cream/grey tumour scattered throughout, in some places appearing to run alongside the branches of the penetrating arteries

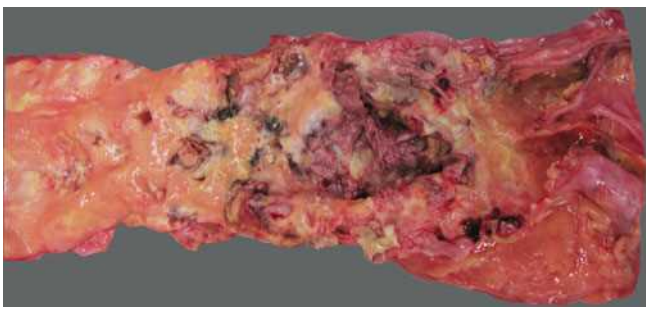

**Fig. 4.302** The lower aorta is seen with extensive, ulcerated atheroma. This is a particularly vulnerable site with regard to fibrolipid plaque disease and aneurysmal change. It is noted that the more proximal part of the aorta is less affected

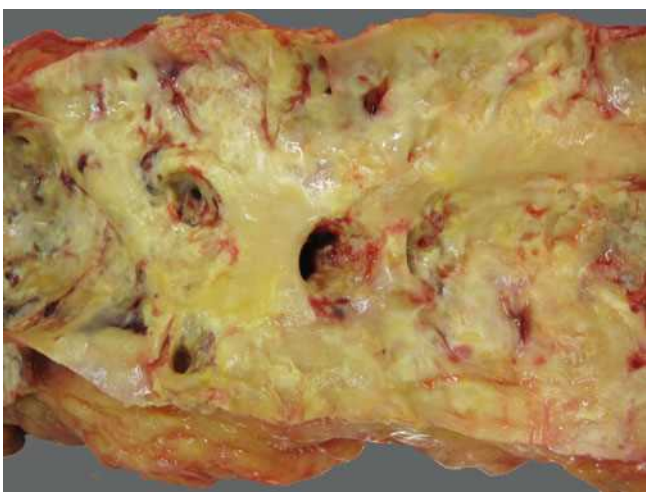

**Fig. 4.303** Part of the abdominal aorta is seen with significant narrowing of the main artery branches that run off to supply the abdominal viscera and retroperitoneal tissues

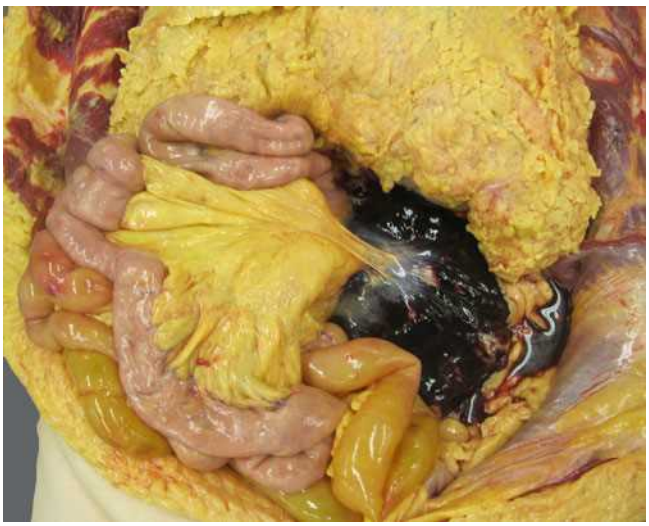

**Fig. 4.304** Opening the abdomen shows a zone of massive haemorrhage in the retroperitoneal compartment from a burst aortic aneurysm

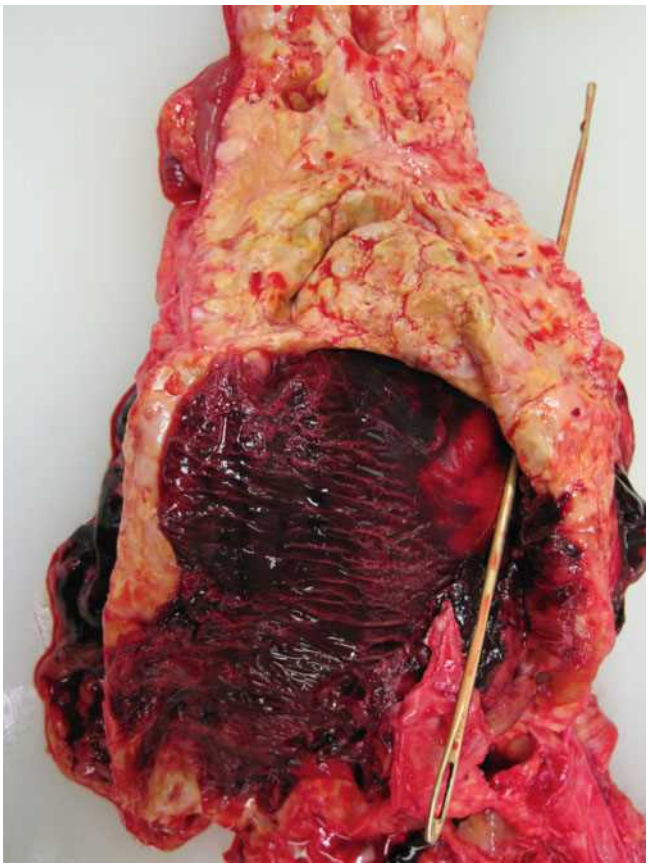

**Fig. 4.305** An aortic aneurysm is seen with dilatation of the vessel and marked thrombus on the endoluminal aspect. The vessel had ruptured. The leak point is marked by a probe

**Fig. 4.306** Laminated thrombus is seen with the imprint of a synthetic graft running centrally. This patient had had a large aneurysm at risk of rupture, prompting endovascular repair

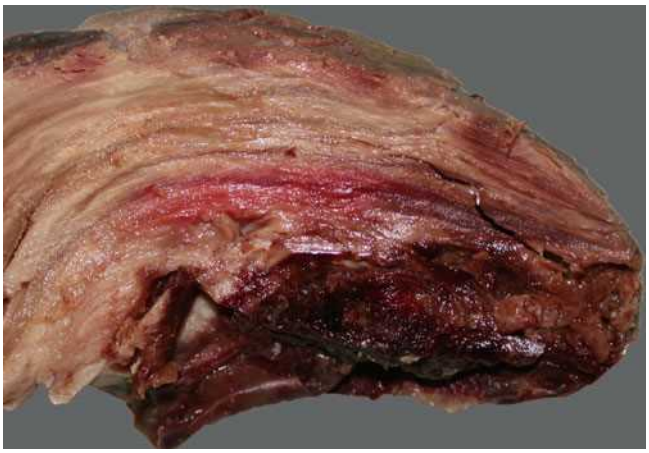

**Fig. 4.307** The thrombus from an aneurysm has been removed. The sectioned material shows the lines of Zahn, comprising laminated thrombus with admixed blood components

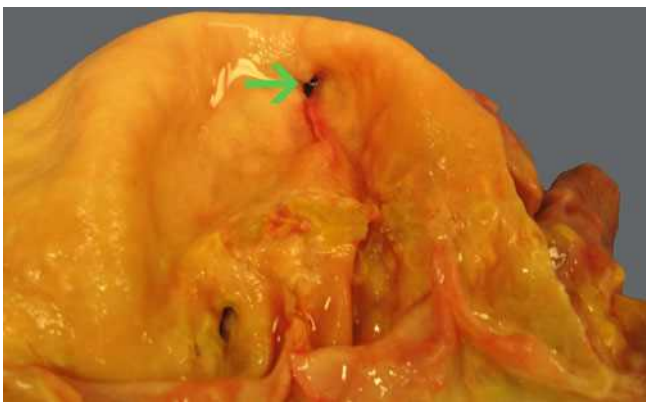

**Fig. 4.308** The proximal aorta is seen with the aortic leaflets at the bottom of the view. One coronary ostium is present. The aorta appears largely normal and without high-grade atheromatous disease, but an irregular laceration/splitting of the intima and media gave rise to aortic dissection and cardiac tamponade. The aortic dissection (*arrow*) can be relatively small in terms of the entry point size but pronounced in terms of the amount of blood loss

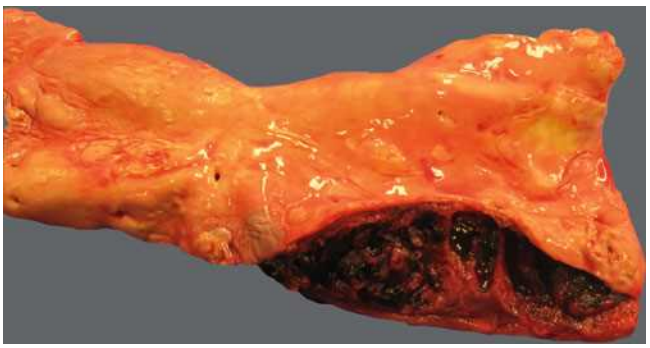

**Fig. 4.309** An aortic dissection is present in the descending thoracic aorta. The intimal and medial tissues have been pushed forward into the lumen, and blood and fibrin are running in the outer media and within the adventitial tissues. This process will dissect and shear the vertebral arteries and others, causing ischaemic damage to various tissues

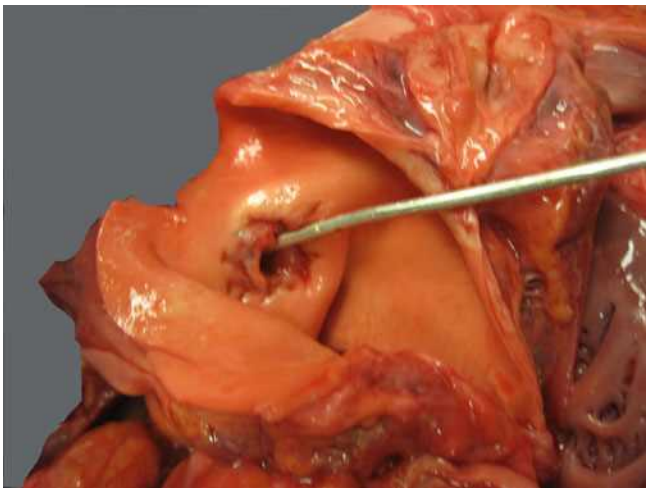

**Fig. 4.310** Congenital disease of major significance is occasionally treated with a surgical fistula to offload the circulation problems. This view shows a connection between the aorta and pulmonary artery

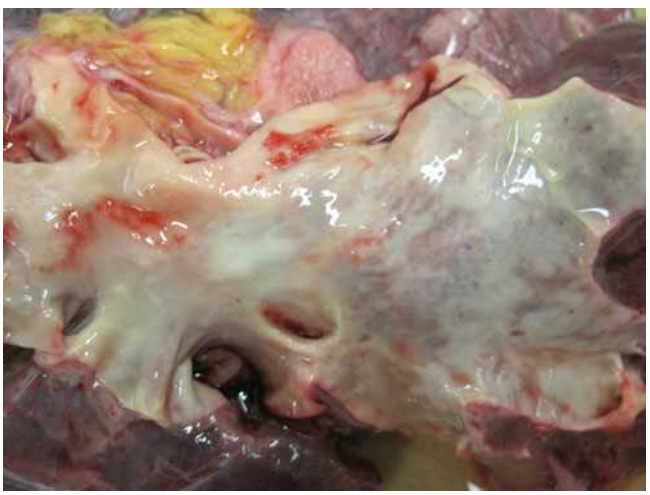

**Fig. 4.311** This is a view of the opened pulmonary artery. The intimal surface appears irregular and variably thickened and fibrotic. This patient had surgery for recurrent and significant pulmonary embolism approximately 6 months before death. The surgery had involved opening the pulmonary artery and extracting partially adherent clot material directly, thereby causing intimal scarring

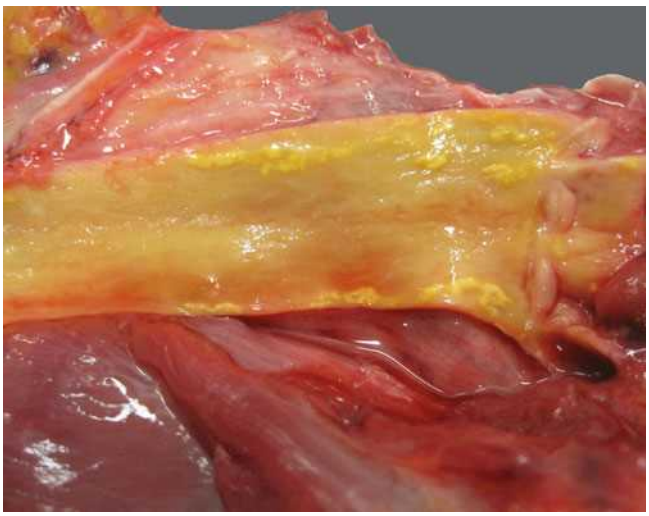

**Fig. 4.312** This carotid artery bifurcation shows early-stage atheromatous disease with small fatty plaques. There is no occlusive disease

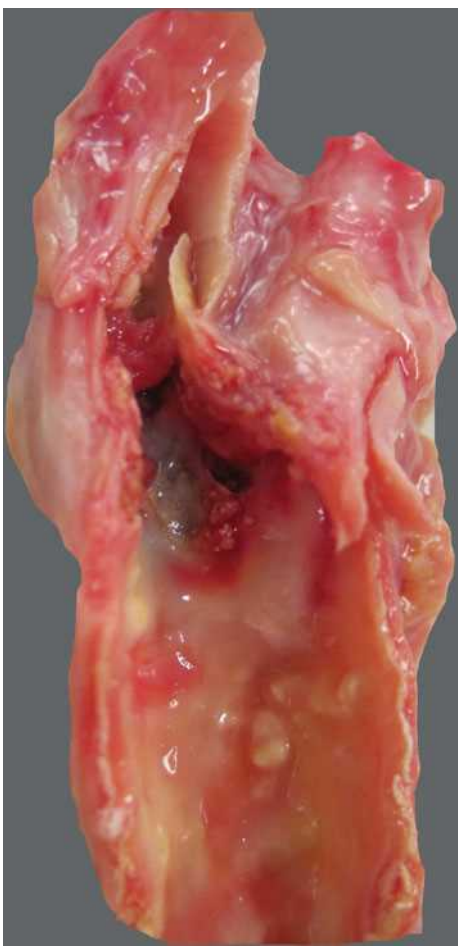

**Fig. 4.313** High-grade atheromatous disease of the carotid bifurcation is relatively common. It may occlude the internal carotid artery and cause thromboembolic disease with secondary ischaemic and infarctive consequences in the brain, but it is often initially clinically silent in many patients

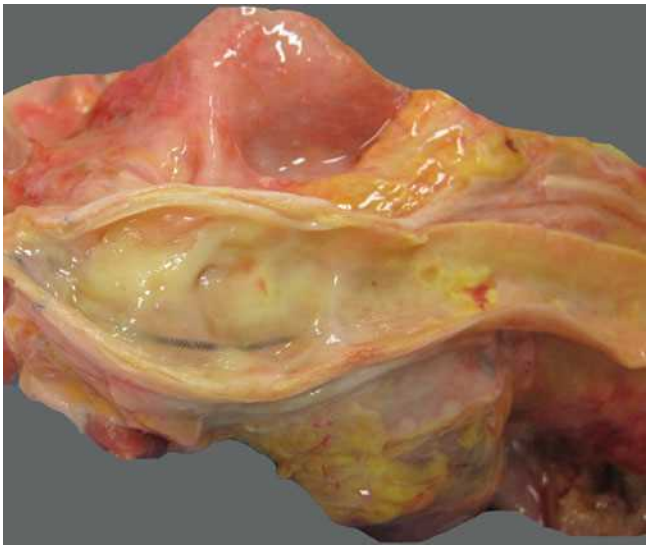

**Fig. 4.314** Vascular grafts can be used at many sites in the arterial tree. Here is a largely incorporated Dacron graft at the carotid bifurcation. The blue marker in the synthetic matrix is seen running through the graft site. There is no obstruction, and the intimal surface is smooth and flat

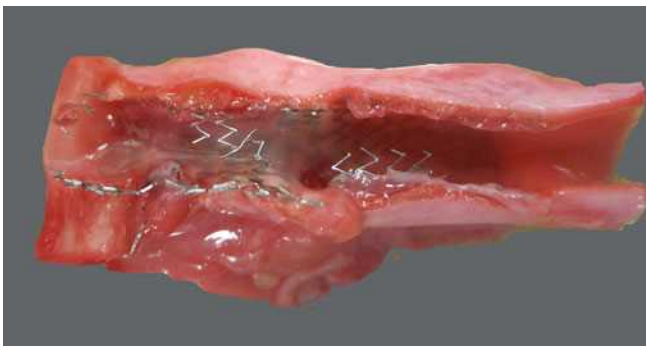

**Fig. 4.315** High-grade stenosis is often treated by surgical excision of the plaque tissue (endarterectomy). Some individuals are not be suitable for open operation because of comorbidities. In such circumstances, a wire stent can be used to maintain the artery patency. This artery has been opened at autopsy using scissors, showing the metal from the stent. The endoluminal tissues appear smooth and without thrombus, indicating remodeling of the artery surface, in keeping with a good outcome for this endovascular repair

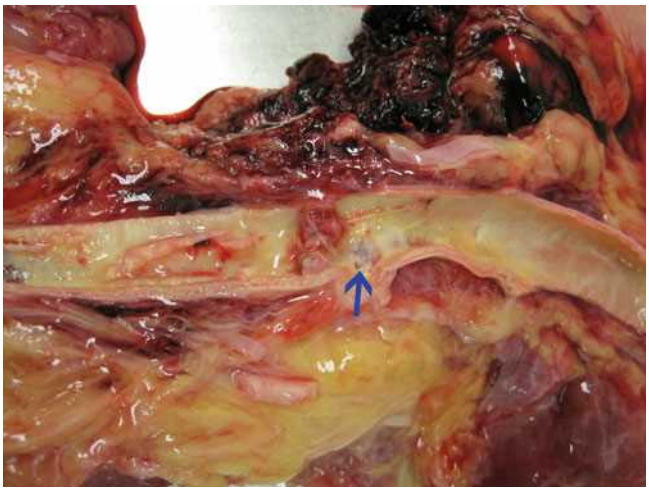

**Fig. 4.316** Significant haemorrhage can occur from peripheral artery sites during angioplasty procedures. This external iliac artery underwent angioplasty. The grey fibrin plug (*arrow*), containing some suture material, is often difficult to discern. It appears that a dissection extended after the angioplasty procedure, causing rupture of the artery at this point. The intima is noted to have peeled off in part, and there is haemorrhage into the local soft tissues

## Ischemic Heart Disease

Ischemic heart disease, manifesting as coronary narrowing of varying degrees, possibly augmented by coronary thrombosis or dissection, is the primary player in terms of the cause of death in the developed world. Findings often can provide a cause of death just by macroscopy, but reservation of tissues for histology may also be relevant. Any devices placed for correction of arterial narrowings (*e.g.*, stents) should be assessed.

The impact of myocardial ischaemia is seen in the form of acute, subacute, and chronic myocardial infarction realities. Many of these have direct relevance to the cause of death, being potent drivers for cardiac dysrhythmias, as well as causing "pump failure". Sampling of these tissues may assist dating of the ischaemic events, but it must be recognised that modern therapeutics with revascularisation techniques and medicines does alter the basic pathophysiology of this natural disease.

## Valvular Heart Disease

This group of disorders is principally degenerative in type, with associated stenosing and incompetent processes. Documentation of the ring circumferences of the valves may be of assistance alongside measurements of transverse chamber diameters and mural thicknesses. The presence of any thrombotic or septic process in relation to a valve is an important data item and likely pertinent to the cause of death.

Valve prostheses, both tissue and metal, are increasingly seen in autopsy practice. The same rules apply with regard to assessing valve function; careful inspection of the leaflet should be undertaken in order to test valve function. Any thrombus should be reserved for microbiology and histology to exclude sepsis.

## Inherited and Congenital Heart Disease: The Adult Reality, GUCH, and Cardiomyopathies

Congenital cardiac diseases (*i.e.*, those one is born with) fall into two broad groups: structural anomalies (*e.g.*, septal defects, vascular or chamber anomalies) and cardiomyopathies (*e.g.*, hypertrophic or arrhythmogenic cardiomyopathies).

The 'bad' end of structural congenital heart disease tends to kill in infancy, but adult survivors of structural lesions (whether treated or undiscovered) may find their way to the autopsy table—often to the surprise of surviving family members. Those surviving surgical therapies are often described as having "grown-up congenital heart disease" (GUCH). Clearly, this text cannot be exhaustive in terms of presenting these diseases, but some common culprit lesions are shown.

The cardiomyopathies are a complex and diverse group of diseases, often with a good family history. They are generally broken into those of primary nature (gene-related disorders) and secondary processes. Whilst some maintain that cardiomyopathy should be simply regarded as primary diseases of the heart (*e.g.*, hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy, mitochondrial cardiomyopathy), it is realistic to see that other disease processes also contribute to cardiac hypertrophy, dilatation, and distortion. Thus, amyloid heart disease and hypertensive heart disease may produce a 'myopathic' quality. The value of histology cannot be underestimated in this regard. It is also possible to perform electron microscopy, even from paraffin-embedded material, if required. In general, any case with even slight suspicion of an inherited disorder should have full histology, possibly with ultrastructural analysis. Glutaraldehyde is the ideal fixative in such cases, but formalin will suffice.

Among the common lesions are myogenic mutations, cytoskeleton mutations, and storage disorders. Many are relatively common in terms of the general population, including hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy, and dilated cardiomyopathy. Characteristic macroscopic and histological features may be readily seen, or their appearance may be unremarkable, requiring comprehensive specialist tests to be undertaken.

The issue of channelopathies is complex. These often present in the autopsy room as sudden deaths with no positive findings, and they are histologically unremarkable. If such a disorder is considered to be present, the autopsy practitioner is advised to perform multiple tests, as described by specialist texts.

Suspected congenital myopathies, particularly of the children's type, may require fresh tissue to be snap frozen for enzyme assessment, although this procedure is usually not needed. It is also important to remember that a 2 cm cube of spleen should be reserved in cases of cardiomyopathy, in order to assess any genetic mutations. The high nuclear content of the spleen makes this tissue ideal for the purposes, although technically any tissue could be used.

Often, cases are examined and then referred to specialist centres for further commentary and analysis. Should one be faced with an adolescent with congenital heart disease, whether surgery has been performed or not, the standard rules of dissection will apply, along with photography and histological sampling.

## Infections and Inflammatory Conditions

Inflammatory conditions in the heart generally produce significant clinical effects with rapid-onset cardiac failure or sudden death. The presence of myocarditis should be specifically sought, with macroscopic analysis, histological sampling, and reservation of tissue samples (at least a 1 cm cube) for genetic and viral studies. Lymphocytic, eosinophilic, and granulomatous disorders are the most common forms of myocarditis. Direct sepsis from local infections may also be apparent, occasionally seeding from infected valves or being part of a more systemic bacteraemia.

## Cardiac Tumours

Cardiac tumours are comparatively rare, but disseminated neoplasia involving the heart is not uncommon; breast, thoracic, and melanomatous tumours are well documented. Primary neoplasms of the heart, aside from mesothelioma of the pericardium, generally reflect cardiac myxomas. Rare cardiac tumours such as lymphomas or sarcomas are unlikely to be encountered in routine practice unless one works close to a cardiac unit. Such tumours merit photography and histological sampling in order to confirm macroscopic features and to assist possible referral of the case to a specialist centre.

## Technology and the Heart

The heart has become a common site for interventions such as vascular devices (*e.g.*, stents) and valve prostheses. Other devices of importance include Dacron structural grafts and septal closure devices.

Perhaps the most commonly employed device, which is regularly seen in autopsy practice, is the pacemaker. Single-lead, double-lead, and triple-lead pacemakers are increasingly common and used to treat a variety of cardiac disorders. The pacemaker needs to be considered as a unit, and if there is any suspicion of pacemaker dysfunction, then the pacemaker generator unit (box), the lead (wire from the box onwards), and the electrode (the tip of the wire) must be dissected without pieces being cut apart. In short, the device needs to be retained whole for detailed inspection. Examination of the pacemaker site may be relevant in terms of sepsis, and it is possible to send the pacemaker away for testing of the programme, battery life, and functionality.

The other common technology involving the heart is valve prostheses. These have evolved markedly over the past 40 years, with many poor designs. Modern replacement valves are implanted by open surgery or endovascular routes. They can radically improve cardiac function in the elderly, but anticoagulation may be required. Complications of sepsis are particularly important.

## The Large Arteries

Whilst much of this vasculature exists outside the thorax, it is considered specifically in this chapter. Vascular catastrophes, principally atheromatous in type, are commonplace and must be assessed and recorded accurately.

Figures illustrate the dissection of the thoracic tissues and the pathological processes that are commonly found in the lungs, heart, and great vessels.

## Suggested Reading

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# Gastrointestinal and Hepatobiliary System

5

Stuart C. Brown and S. Kim Suvarna

## Introduction

The examination of the gastrointestinal organs is a major component of the autopsy. It takes in the examination of a large number of organs, and if done thoroughly can be time-consuming. Often, pathology cannot be demonstrated in the gastrointestinal organs, and sometimes it may be appropriate to perform an abridged examination. It may not be necessary in all autopsies to fully open the intestine, for example. If there is a history suggestive of gastrointestinal disease, however, or if a cause of death is not immediately apparent in other organs, a thorough examination of the gastrointestinal organs is recommended.

Death caused by disease of the gastrointestinal system is not uncommon: 13.8 % of the deaths recorded in the UK in 2013 were classified as gastrointestinal deaths. The largest group of these are cancer deaths, with the most common primaries being the colon, pancreas, esophagus, liver, and stomach.

Non-neoplastic diseases account for about one third of all gastrointestinal deaths, with liver disease, particularly alcoholic liver disease, being very common. Alcoholic liver disease is one of the most common causes of death in younger patients.

Gastrointestinal deaths are most common in middle age. They account for nearly a quarter of deaths in those in their fifties because of a high prevalence of both alcoholic liver disease and malignancy in that group.

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## External Examination

The examination of the gastrointestinal system begins with the external examination of the body.

The abdomen should be inspected for signs of natural disease and markers of medical intervention such as a scar, stomas or drain sites. The eyes may show evidence of liver and biliary tract disease, and fingernail clubbing may be seen in cases of chronic idiopathic inflammatory bowel disease and liver disease.

External examination is not complete without an inspection of the anus, which may show evidence of malignant or inflammatory disease.

## Internal In-Situ Examination

The abdominal cavity is entered in the manner described in Chap. 3, taking care not to damage the internal organs. On entering the abdominal cavity, one may notice fluid. There usually should be no more than about 20 mL. The omentum can be examined and then retracted to reveal the intestines and appendix beneath. Major pathology like intestinal dilatation, ischemia, volvulus, intussusception, or adhesional scarring is usually better appreciated at this point, before evisceration.

The intestines may be manipulated to examine the mesenteries and peritoneum, as well as the bowels. The omentum can be gently retracted downwards to visualise the inferior aspect of the liver, the gallbladder, the spleen, and the stomach. If indicated, the lesser sac can be inspected in situ by cutting through the greater omentum where it joins the greater curve of the stomach.

The remainder of the examination will assume that the evisceration has been carried out in the method described in Chap. 3. If intestinal ischemia is encountered with in situ examination, however, the usual technique of evisceration may be altered in order to consider the mesenteric vessels and preserve their relationship to the bowel.



S.K. Suvarna (ed.), *Atlas of Adult Autopsy: A Guide to Modern Practice*, DOI 10.1007/978-3-319-27022-7_5

## Dissection of the Tissues in Sequential Format

### Omentum, Mesenteries, and Peritoneum

The omentum is an apron of fatty tissue that is attached to the greater curve of the stomach and duodenum and hangs down freely to float over the intestines. The volume of adipose tissue varies between individuals and may be great. Histologically it is characterized by lobulated adipose tissue with a mesothelial lining. The small intestine and the transverse and sigmoid colon have a mesentery that also carries a variable volume of adipose tissue. The peritoneal lining envelopes the intestine and lines the abdominal wall, imparting a glistening appearance. Histologically it is characterized by mesothelium overlying a thin layer of loose connective tissue. The omentum and peritoneum are usually inspected in situ, but the mesenteries can also be inspected in detail after evisceration.

### Esophagus

The esophagus is a tubular, muscular organ that extends from the pharynx to the stomach, traversing the diaphragm. It is approximately 20 cm in length. The normal mucosa of the esophagus is a grayish white with vertically orientated mucosal folds. Histologically it is characterized by nonkeratinized, stratified squamous epithelium with submucosal mucinous glands. It may be examined by opening it along its length or by inverting it by pulling it through the gastroesophageal junction with forceps.

### Stomach

The stomach is a muscle-lined, expanded tubular reservoir lying in the upper left quadrant of the abdomen. When full, it holds approximately a litre of food. Like many of the luminal digestive organs, the mucosa of the stomach often shows a marked degenerative appearance on histology, but it is usually possible to distinguish between the body with oxyntic epithelium and the pylorus with foveolar epithelium. The mucosa can be inspected by cutting along the greater curve. Care should be taken to preserve the stomach contents if indicated.

### Duodenum

The first part of the small intestine is broadly C-shaped and curves around the head of the pancreas, accepting the opening of the pancreatic ducts at the ampulla of Vater. On histology, it has a villous architecture with distinctive submucosal glands in its proximal portion. The duodenal mucosa can be examined by continuing the stomach incision through the pylorus. The ampulla may be identified by applying pressure to the gallbladder and observing the extrusion of bile through it.

### Pancreas

The pancreas normally has a creamy, salmon pink color, best appreciated on its anterior, peritonealized surface. It is a tongue of glandular tissue approximately 15 cm long, with a distinctive uncinate process that reflects back on itself, wrapping around the superior mesenteric vessels. It usually shows marked autolytic change on histology, though some residual acinar glandular and islet structure may be appreciated. The pancreas can be examined by serially slicing through its long axis or by opening it along its main duct, either from the tail or head end.

### Gallbladder and Biliary Tract

The gallbladder is a blind-ended reservoir for bile lying underneath the liver and measuring approximately 8 cm in length. It is usually thin-walled, with a mucosa pigmented green from the bile within. On opening, it is not uncommon to identify calculi. Because of the spiral valve (of Heister), it is difficult to continue the gallbladder incision into the cystic duct. The rest of the biliary tract can be opened at the free edge of the lesser omentum if indicated, and explored up and down. The common bile duct is normally no greater than 10 mm in maximum diameter.

### Liver

The liver is a large organ that occupies much of the right upper quadrant of the abdomen. From the front it is broadly triangular and divided into two lobes by the falciform ligament. Histologically it tends to be relatively well preserved compared with the luminal structures, showing characteristic acini and portal tracts. It should be cut into sections no greater than 10 mm in width to maximize the chance of identifying small lesions internally.

### Small Intestine and Appendix

These tissues are usually presented for examination in a separate block of tissue containing the small intestine from

the duodenojejunal flexure onwards, the appendix, and the colon. The small intestine is up to 7 m long and no greater than 3 cm in maximum diameter. Grossly the mucosa is pink with transverse plicae circulares. Histologically it is characterized by a villous architecture, without submucosal glands. The intestine may be cut along its length to examine the mucosa. Alternatively, small incisions may be made at intervals along its length in order to examine its contents.

The appendix is a narrow, blind-ended muscular tube with a mesentery. Its size may range from 2 to 20 cm, but it is usually about 10 cm long and about 1 cm in maximum diameter. It can be found by tracing proximally the transverse taenia coli on the serosal surface of the colon.

## Colon

The colon makes a clockwise circuit of the lower abdominal cavity, starting in the lower right quadrant. It is approximately 150 cm in length. The caecum may be up to 9 cm in diameter, but the colon distal to the hepatic flexure should be no more than 6 cm. Much of the colon is attached directly to the posterior abdominal wall, but the transverse and sigmoid parts have a mesentery. As in the small intestine, there should be a clear division between the fatty mesentery and the intestinal wall, with no encroachment of fat onto the serosa. Lobules of fat called *appendices epiploica* are typically seen on the transverse and sigmoid parts. The mucosa is normally featureless; histologically, it shows a characteristic "row of test tubes" appearance.

## Rectum and Anus

The rectum is usually separated from the colon on evisceration and is examined as part of an organ block with the other retroperitoneal organs. It is thin-walled, with a featureless mucosa, and is surrounded by a layer of fatty mesorectal tissue. It is usually about 15 cm long. The rectum is usually ligated at the level of the pelvic floor, leaving the perineal anus in situ in the body. The anus can be safely digitally examined after evisceration. Occasionally it may be appropriate to perform an exenteration-type excision of the anus in order to closely examine these tissues.

Figures 5.1, 5.2, 5.3, 5.4, 5.5, 5.6, 5.7, 5.8, 5.9, 5.10, 5.11, 5.12, 5.13, 5.14, 5.15, 5.16, 5.17, 5.18, 5.19, 5.20, 5.21, 5.22, 5.23, 5.24, 5.25, 5.26, 5.27, 5.28, 5.29, 5.30, 5.31, 5.32, 5.33, 5.34, 5.35, 5.36, 5.37, 5.38, 5.39, 5.40, 5.41, 5.42, 5.43, 5.44, 5.45, 5.46, 5.47, 5.48, 5.49, 5.50, 5.51, 5.52, 5.53, 5.54, 5.55, 5.56, 5.57, 5.58, 5.59, 5.60, 5.61, 5.62, 5.63, 5.64, 5.65, 5.66, 5.67, 5.68, 5.69, 5.70, 5.71, 5.72, 5.73, 5.74, 5.75, 5.76, 5.77, 5.78, 5.79, 5.80, 5.81, 5.82, 5.83, 5.84, 5.85, 5.86, 5.87, 5.88, 5.89, 5.90, 5.91, 5.92, 5.93, 5.94, 5.95, 5.96, 5.97, 5.98, 5.99, 5.100, 5.101, 5.102, 5.103, 5.104, 5.105, 5.106, 5.107, 5.108, 5.109, and 5.110 illustrate the dissection of the gastrointestinal and hepatobiliary systems and the pathological processes that are commonly found in the gastrointestinal organs.

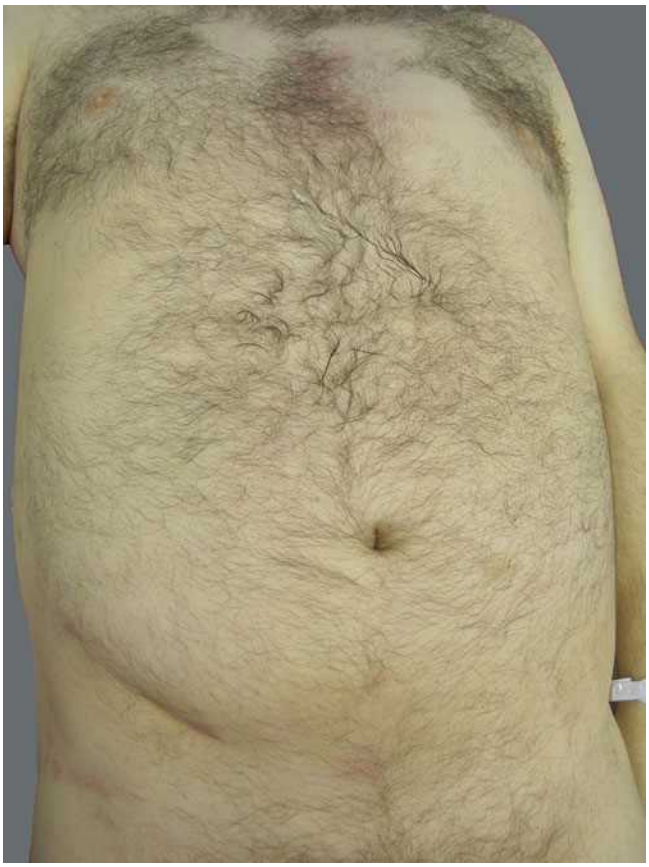

**Fig. 5.1** Even before internal examination, clues regarding gastrointestinal disease can be seen in the external appearance of the body. Here, there is an oblique scar in the right iliac fossa consistent with a history of appendectomy. Also note the areas where hair has been removed on the chest—a sign that ECG electrodes were used during resuscitation attempts

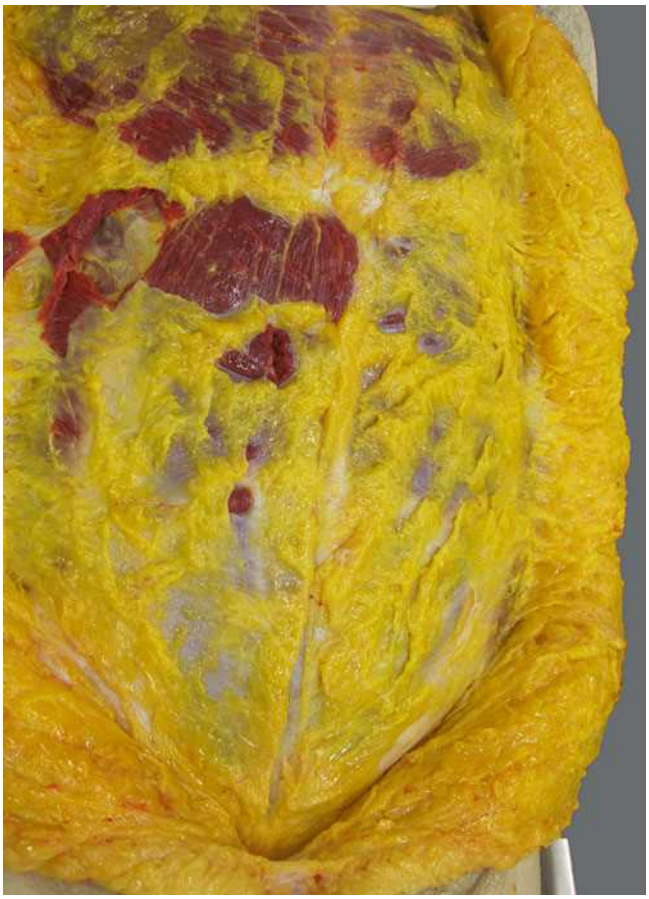

**Fig. 5.2** The adipose tissue of the abdominal wall can be removed to reveal the underlying muscle. This dissection is not a standard part of the autopsy, but it may help in looking for hernias and assessing abdominal wall injury

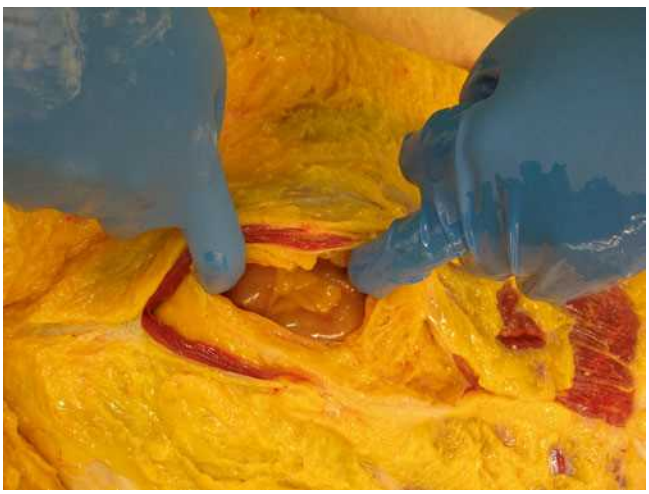

**Fig. 5.3** The abdominal cavity is opened in the upper midline between the rectus muscles. Beneath the muscles is usually a pad of fat that can be swept away with the finger to reveal the abdominal cavity, where immediate clues regarding abdominal disease (*eg*, peritonitis, neoplasia) may be seen

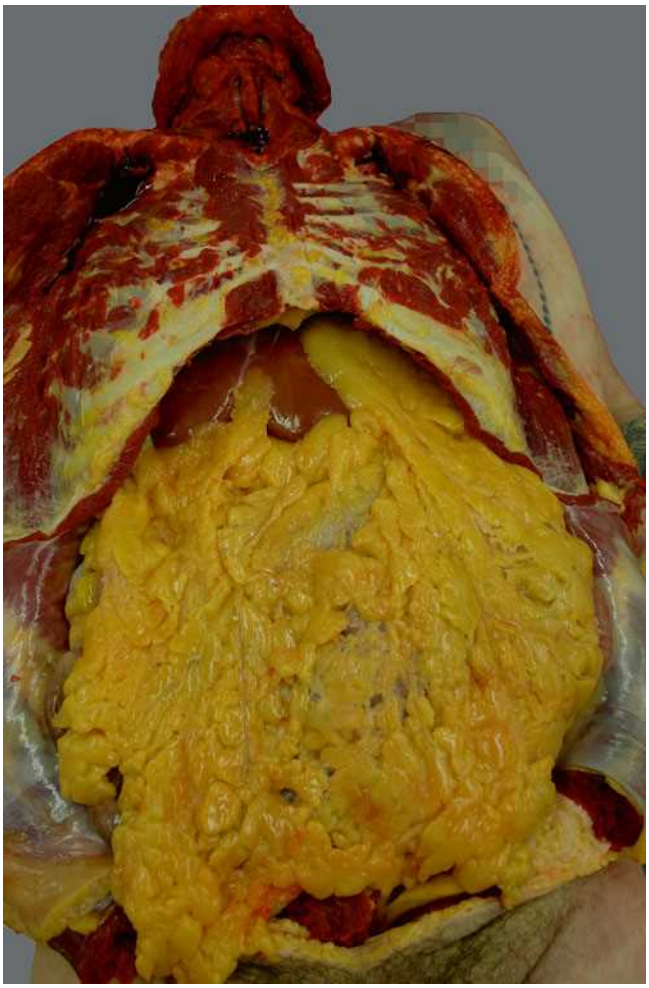

**Fig. 5.4** The omentum is often overlooked in terms of gastrointestinal disease. It provides an assessment of the nutritional status of the individual. The omentum is usually thin, but it may be bulky, with excess adipose tissue in those who have a raised body mass index. The omentum may become adherent to inflamed peritoneum, forming an abscess. The omentum, or part of it, may have been removed previously as part of surgical treatment for intra-abdominal neoplasia, usually of the ovary, endometrium, appendix, or colon

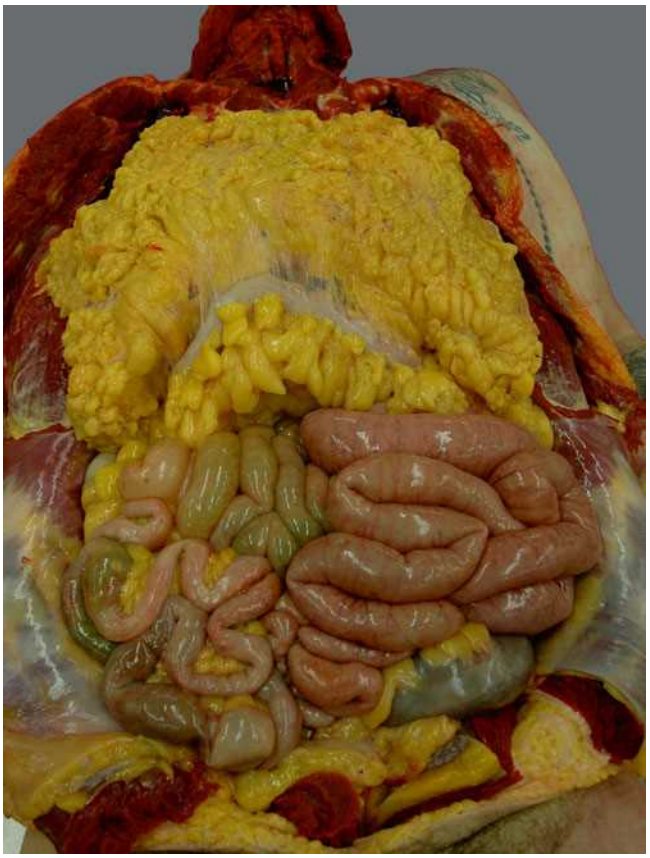

**Fig. 5.5** The assessment of the intestines starts even before evisceration. The intestines appear to zig-zag back and forward, winding their way down to the lower right quadrant. The proximal intestine appears larger than on the left side. This is the normal appearance, although the amount of gas distension of the bowel varies on a case-by-case basis

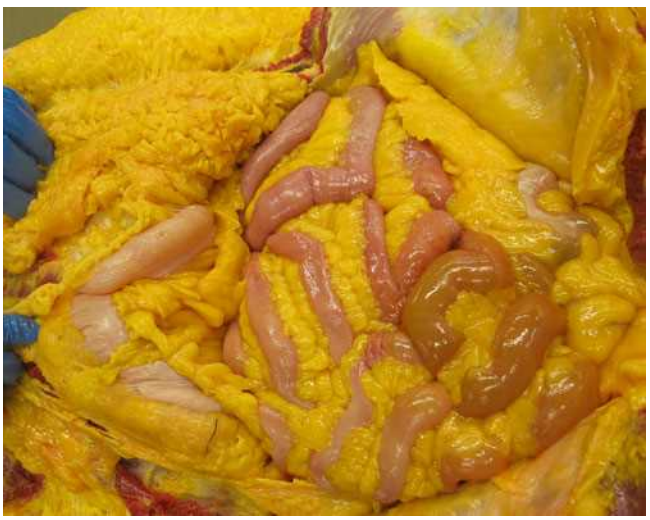

**Fig. 5.6** This abdominal cavity shows a marked amount of fat around the bowel mesenteries

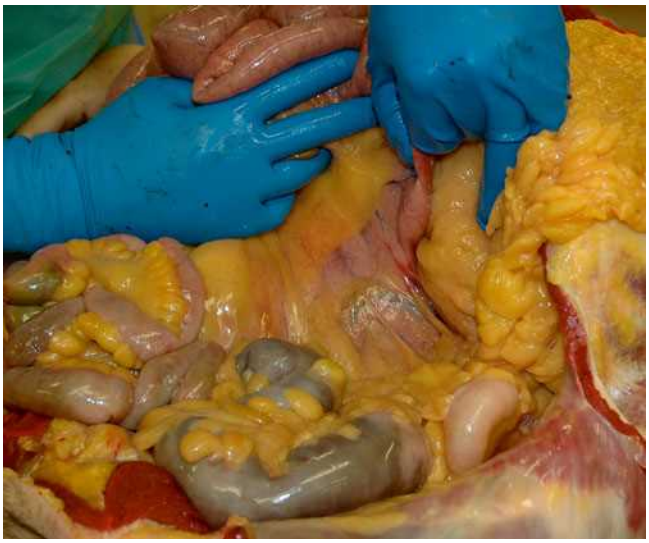

**Fig. 5.7** The duodenojejunal flexure is the landmark used for evisceration of the bowels. Before cutting into these tissues to start removing bowel, it is worth considering the stomach, pancreas, spleen, and local mesenteries for any overt disease

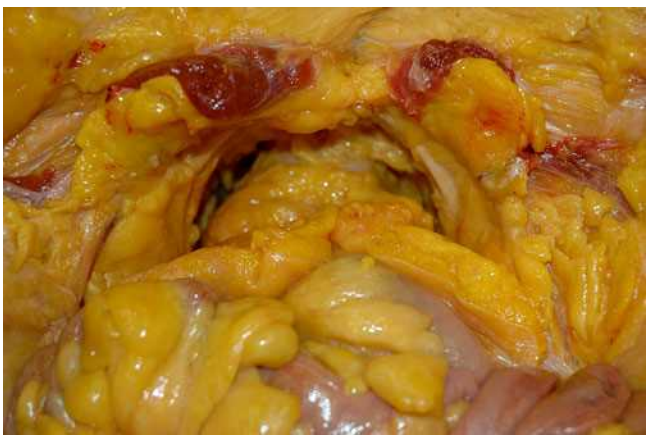

**Fig. 5.8** In the same way, it is worth considering other parts of the peritoneal cavity and bowels at this stage. This view (looking down into the pelvis with the pubic ramus at 12 o'clock) shows no pathology. Note that the mesorectal tissues have been dissected bluntly from the pelvic wall

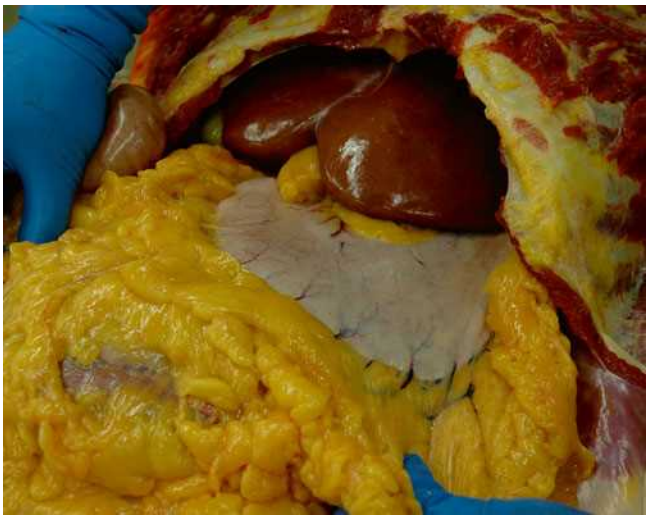

**Fig. 5.9** Limited views of the liver and gallbladder may be obtained by retracting the omentum and stomach downwards. One should note any fluid collection around the liver. The anterior surface of the stomach can be seen

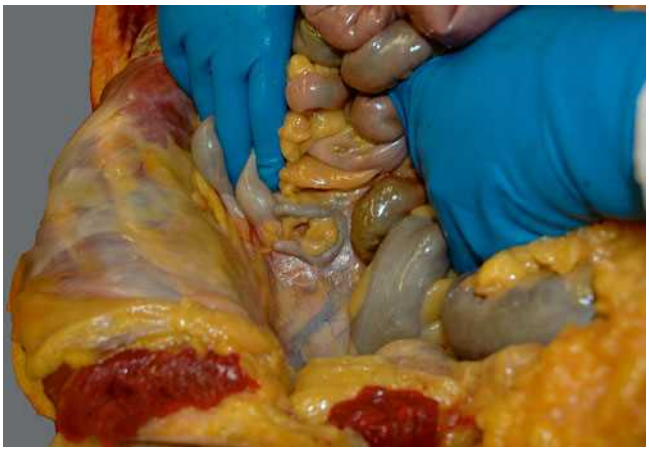

**Fig. 5.11** The appendix can be seen in the right lower quadrant of the abdomen after retracting the intestines and colon. In addition, at this stage with the small intestine reflected, the nonmesenteric/retroperitoneal parts of the bowel can be inspected. These include the paracolic gutters, which are often sites of fluid collection. These gutters may channel fluid from intra-abdominal sepsis

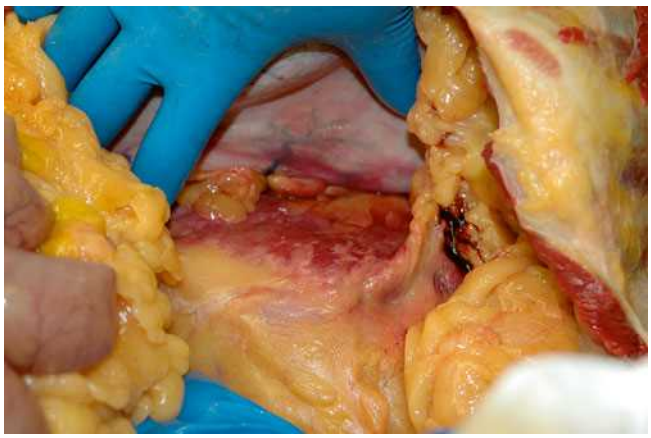

**Fig. 5.10** Opening the omentum below the stomach allows one to look into the lesser sac. The bowels have been pushed to the right to facilitate the view

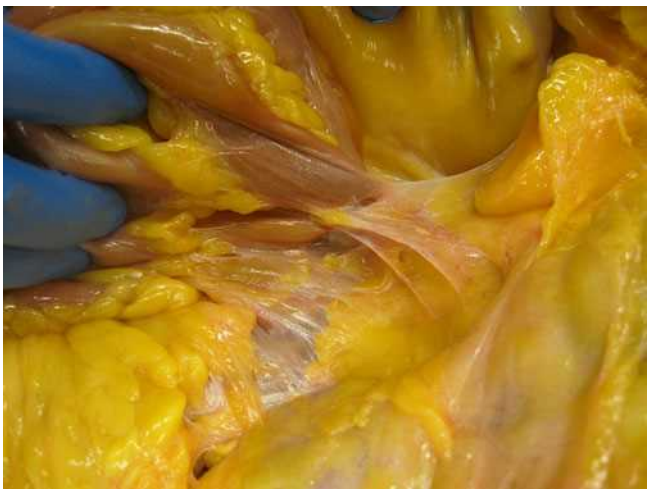

**Fig. 5.12** Normally the peritoneum is smooth, thin, and glistening. Where there has been previous inflammation or surgery, scarring can occur between organs and the abdominal wall. In this example, the appendix has been removed, and as a result the caecum is now stuck onto the abdominal wall

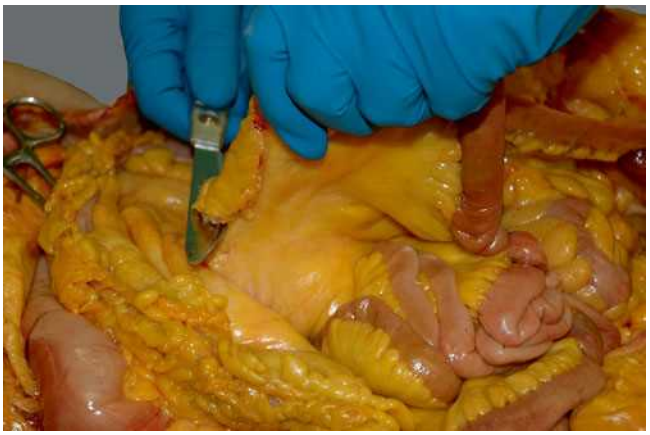

**Fig. 5.13** The small intestine may be removed by cutting through the mesentery, at the mesenteric root in this view. This approach has the advantage of keeping the vessels and bowel together as a single block, but it can be more difficult to open the small intestine or remove it from the mesentery later

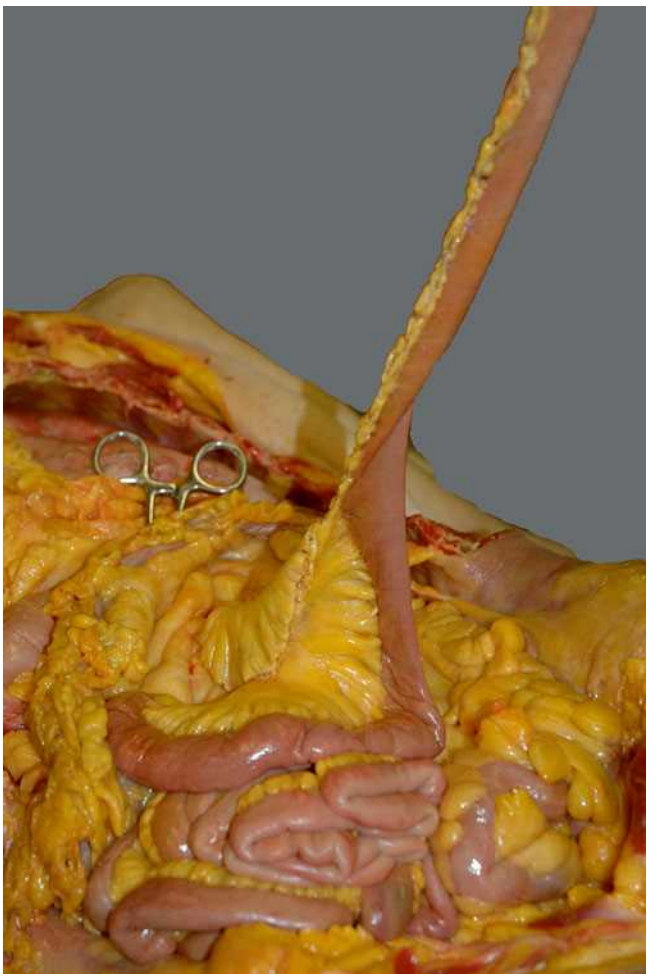

**Fig. 5.14** The bowel also can be removed by cutting along the inner/mesenteric aspect of the small and large bowel. Whichever method is used, the evisceration protocol (see Chap. 3) continues, with steady resection of the bowel from the duodenojejunal flexure down to the upper rectum. Attention is paid throughout, in order to palpate the bowels. Key elements include the identification of perforations and/or tumors

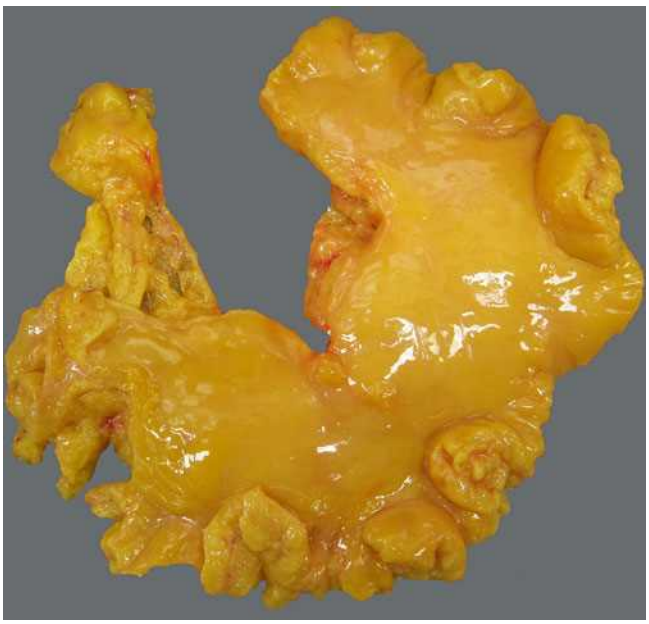

**Fig. 5.15** Dividing the mesentery from the intestine after it has been removed en bloc with the intestine may simplify its examination. The lost sense of continuity with end organs may make evaluation of bowel ischemia more complex, however

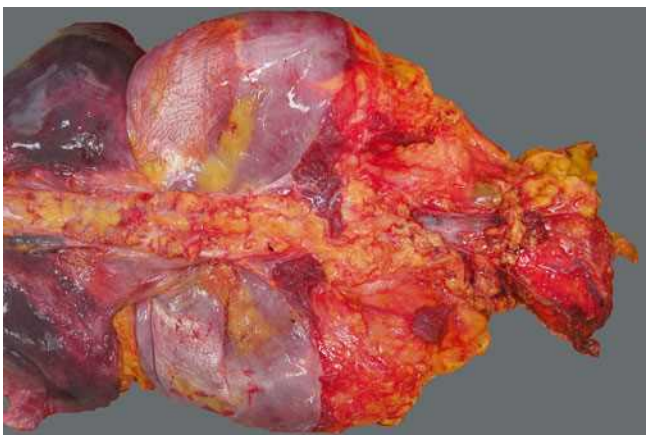

**Fig. 5.16** Complete evisceration has occurred (see Chap. 3), and one is left with abdominal/retroperitoneal tissues below the diaphragm and also tissues above this landmark. The diaphragms, kidneys, adrenals, bladder, and great vessels should be removed for later examination (see Chaps. 6 and 8)

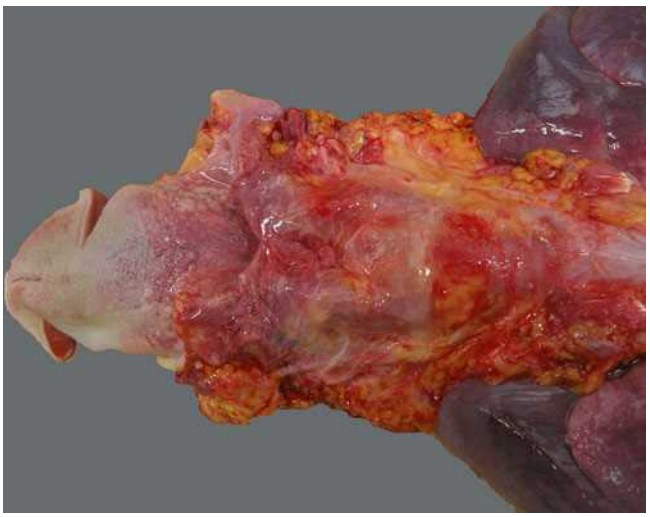

**Fig. 5.17** The proximal part of the esophagus can be identified here posterior to the larynx. One should always check the tissues of the oropharynx for disease. One is also able to inspect the tongue. Areas of squamous mucosal change to the tongue should be sampled for histology. In addition, one may consider sampling of salivary gland tissues and inspecting the rest of the mouth

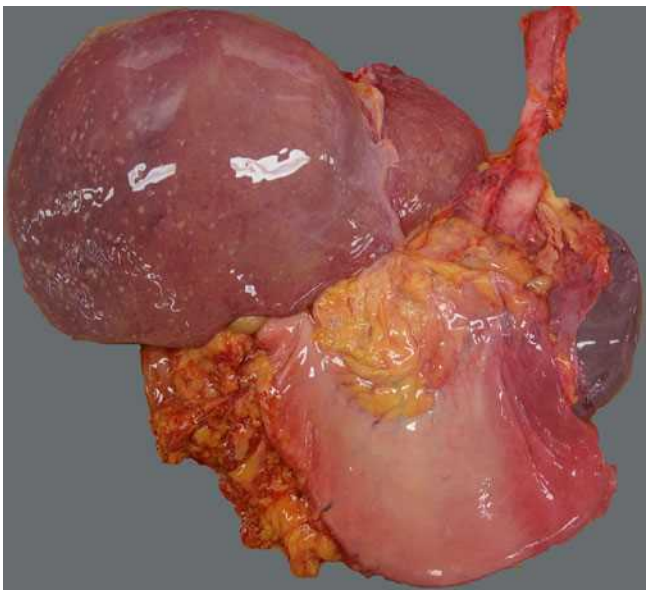

**Fig. 5.19** This view of the upper gastrointestinal organ block shows the esophagus, stomach, liver, and spleen anteriorly. The gallbladder, pancreas, and biliary tree are not seen in this view

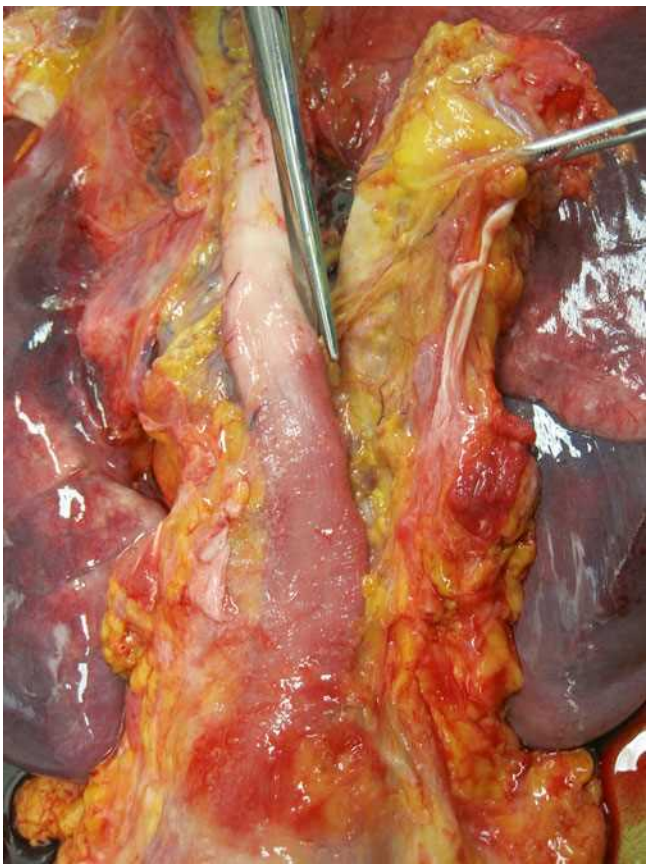

**Fig. 5.18** It is possible, at this juncture, to open the esophagus and examine the esophageal mucosa, but often, to simplify examination in continuity with the stomach, it can be freed and left intact with the stomach. It should be easy to find a plane of dissection between the esophagus and the other thoracic organs. One should take care to avoid opening the trachea and the pericardium

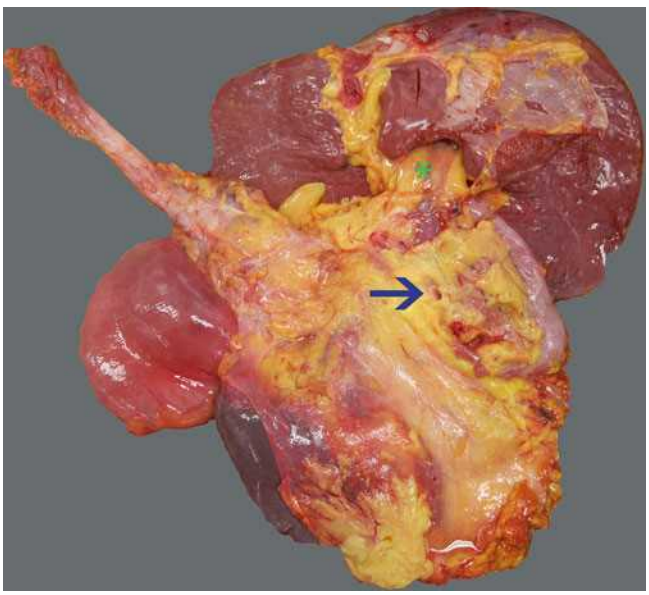

**Fig. 5.20** Posteriorly, much of the detail is obscured by retroperitoneal fat. The portal vasculature and biliary structures (*asterisk*) may be seen at the hilum of the liver. The portal vein should be available for inspection at this point, but the pancreas is still obscured. The superior mesenteric artery is seen open, heading underneath and anterior to the pancreatic uncinate process (*arrow*)

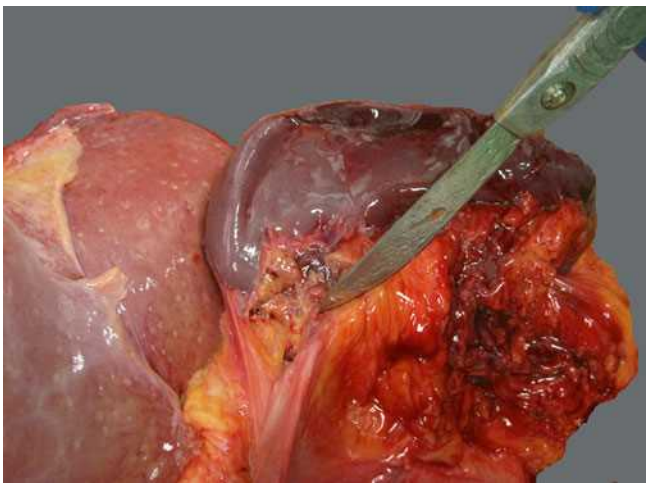

**Fig. 5.21** At this point, one may separate the spleen by cutting through the gastrosplenic ligament and splenic vessels. The biliary tract, pancreas, and luminal structures stay connected

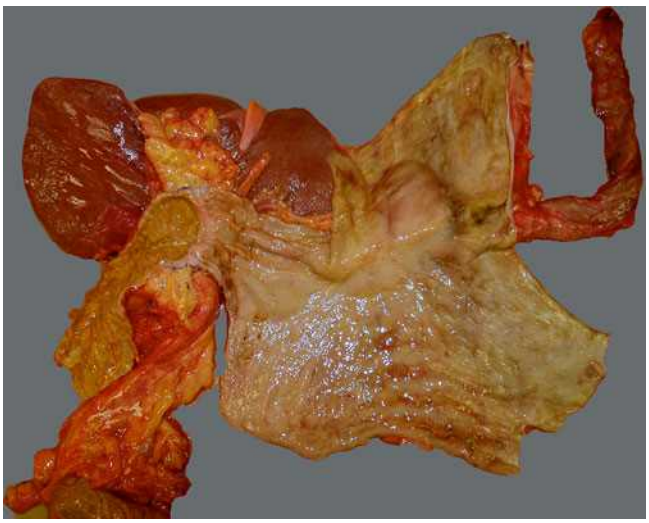

**Fig. 5.23** Alternatively, the stomach and duodenum may be opened (along the edge of the stomach greater curve to the pylorus, and then crossing to the small bowel outer aspect/greater curve). Opening them has revealed the normal appearance of the gastric and duodenal mucosa. The gastric rugae seen here are mucosal folds. Note the transverse folds and biliary staining in the duodenum. Ulcers, tumors, and points of bleeding are not present in this case. Note also that this dissection has not opened the esophagus (see Fig. 5.24)

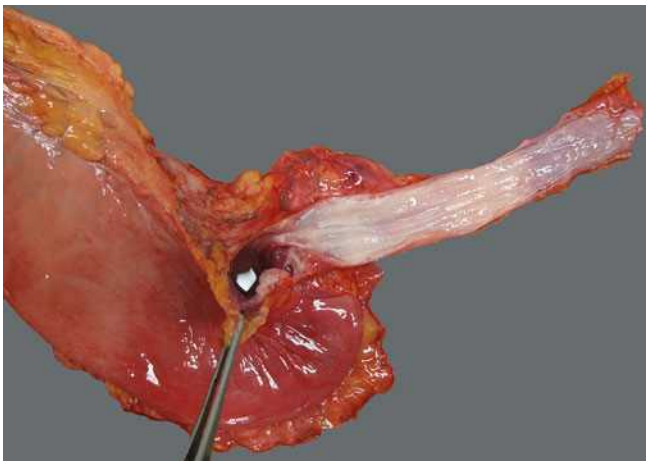

**Fig. 5.22** Here the esophagus has been opened along its length to show its normal appearance. Common pathological findings to look for are strictures, tumors, ulcers, and varices. Note that the stomach contains some fluid, which may be sampled if toxicology issues are pertinent

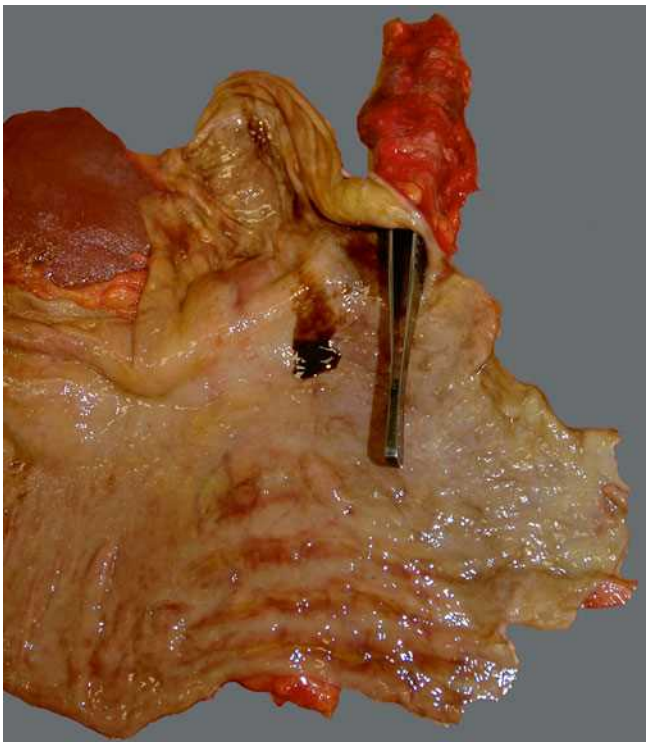

**Fig. 5.24** An alternative to cutting open the esophagus is to pull it inside out through the stomach, having previously tied off the proximal end. This technique may help to preserve engorged variceal vessels

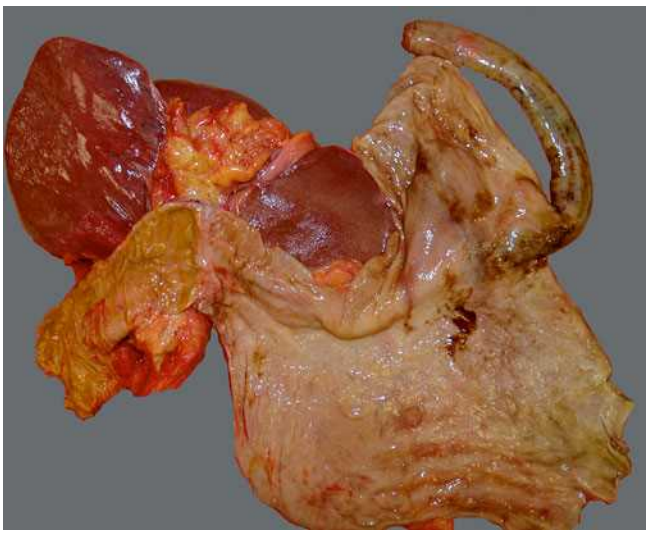

**Fig. 5.25** The inverted esophagus is seen with the local stomach mucosa

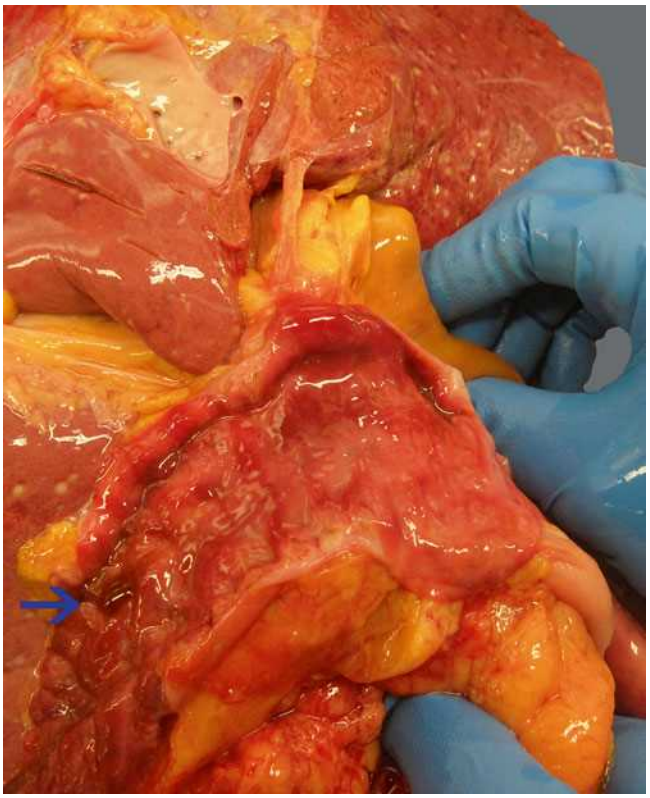

**Fig. 5.26** Gently squeezing the gallbladder often will cause bile to extrude through the ampulla, allowing it to be identified (*arrow*), but this cannot be relied on as an absolute test of obstruction or patency

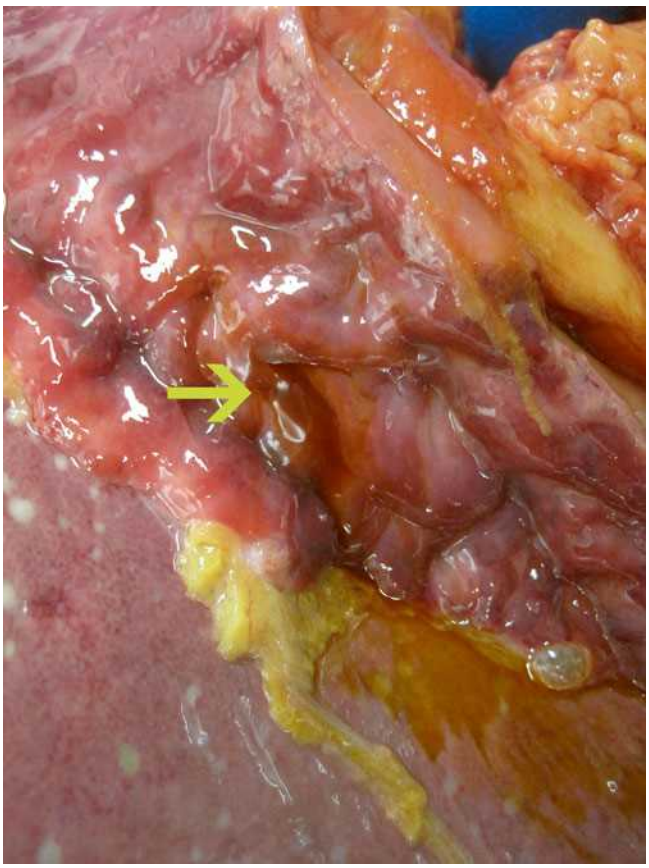

**Fig. 5.27** The ampulla shows the flow of bile (*arrow*). If one wishes, one can explore the bile and pancreatic ducts upwards, having gently placed a probe into the ampulla

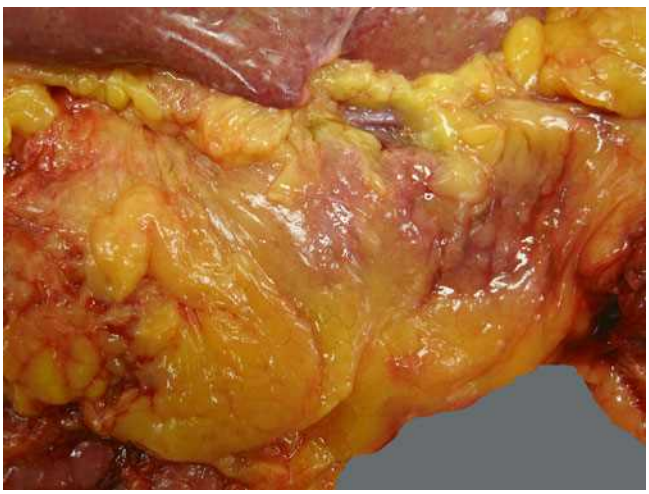

**Fig. 5.28** The normal appearance of the anterior surface of the pancreas can be seen. Note the slightly nodular surface appearance and intimate association with the retroperitoneal fat. The splenic artery and vein run along the superior aspect

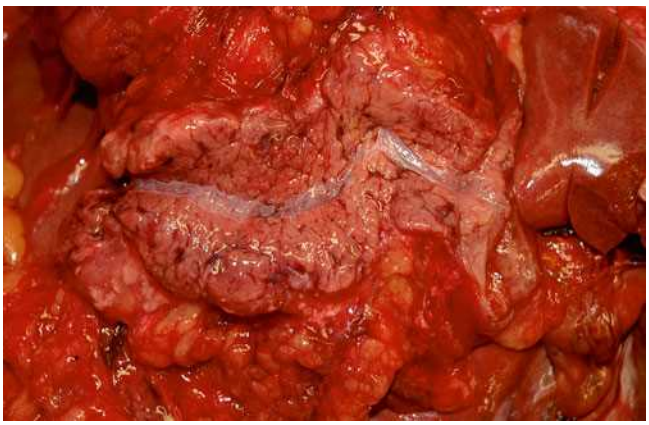

**Fig. 5.29** One can open the pancreatic duct along its length. Doing so should readily show any tumors or stones, or tumors within the adjacent acinar/endocrine tissues

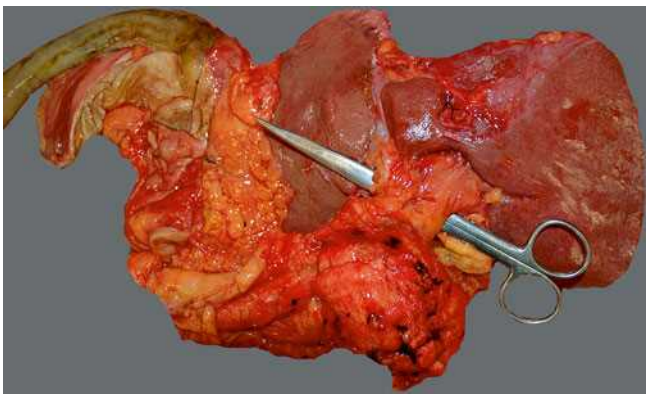

**Fig. 5.31** The portal vasculature with the adjacent pancreas, stomach, and spleen can be examined early if there are ante-mortem issues of obstruction. Here the portal vessels and common bile duct are supported by the scissors. A cut into the tissues at this point assesses these conduits. They may be followed along their length or in transverse format

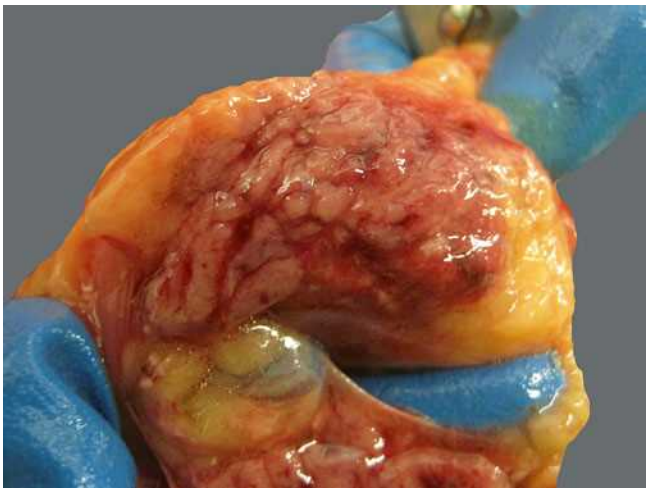

**Fig. 5.30** Alternatively, one may opt to serially section in transverse fashion (at 10-mm intervals) along the body of the pancreas, starting at the tail. This technique has the advantage of showing the parenchyma at multiple points (helpful in looking for neoplasia) and also shows whether there is any duct dilatation in cross-section format. This view shows a common issue with pancreatic parenchyma—softening and dissolution of the tissues

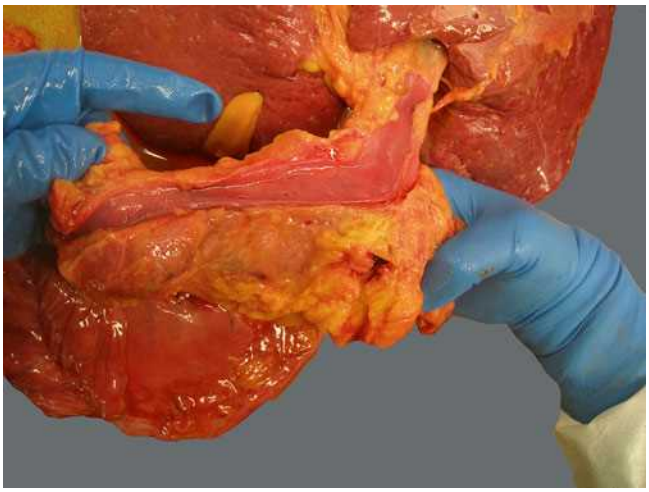

**Fig. 5.32** This view shows the opened portal vein and its tributary, the splenic vein, running up to the liver. The splenic and superior mesenteric veins may be probed or opened if desired at this point

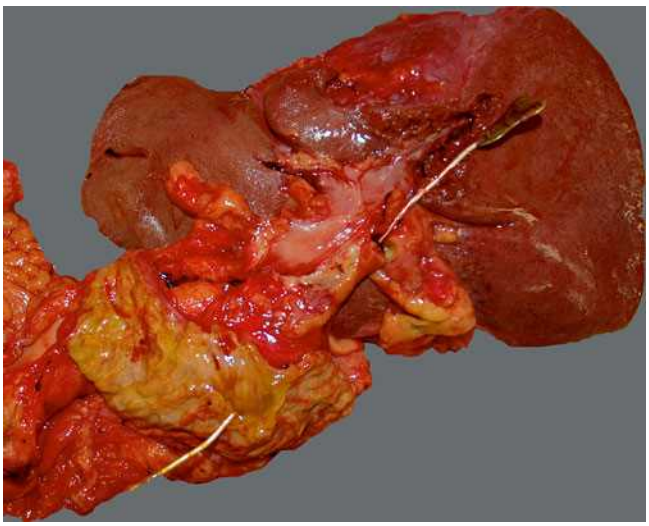

**Fig. 5.33** Here a probe has been inserted into the common bile duct, below the spiral valve of the gallbladder, and passed into the duodenum

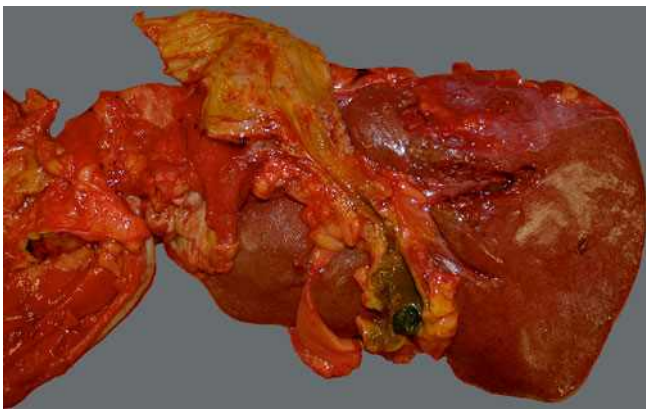

**Fig. 5.35** The gallbladder and cystic duct have been opened, continuous with the common bile duct. Note the black calculus in the gallbladder

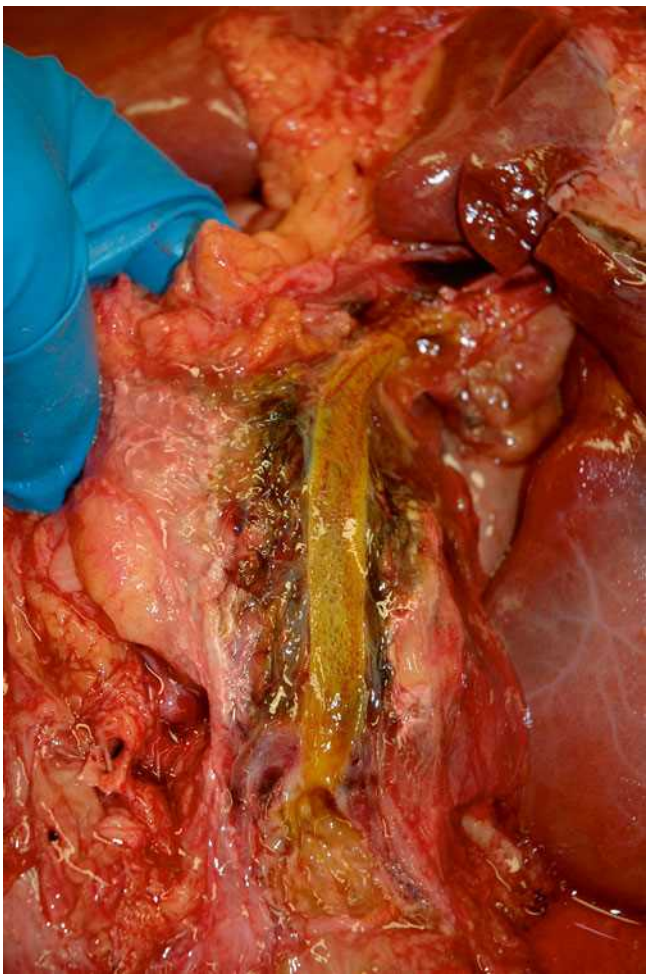

**Fig. 5.34** One may use the probe to guide opening along the duct, if desired. This view shows the green mucosal surface without obstructive lesion

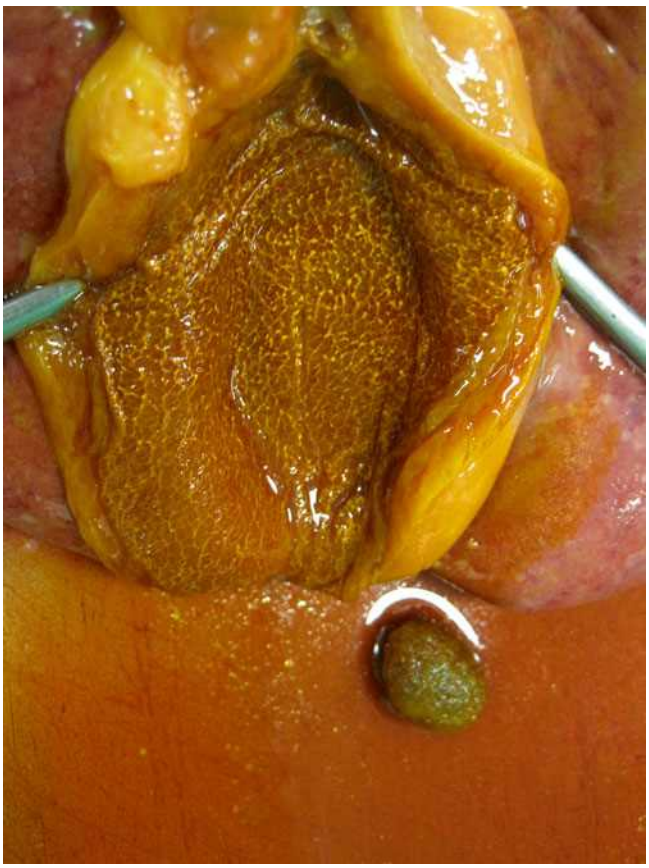

**Fig. 5.36** This gallbladder has been opened to reveal a calculus and mucosal cholesterosis. Note the yellow, reticulate pattern of the mucosa, which is caused by cholesterol within macrophages in the tips of villi in the mucosa. The gallbladder wall is also thickened

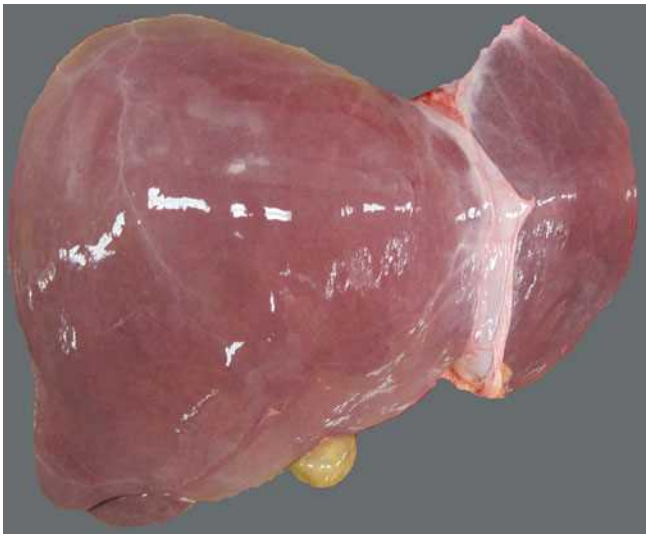

**Fig. 5.37** Here the external anterior appearance of the liver can be seen, with the left and right lobes clearly evident

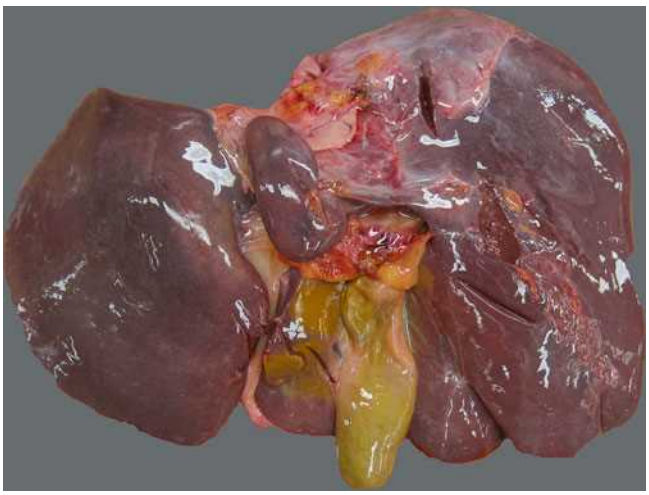

**Fig. 5.38** Posteroinferiorly, the liver shows the caudate lobe, which can be seen above the gallbladder, and the quadrate lobe, between the gallbladder and the ligamentum teres

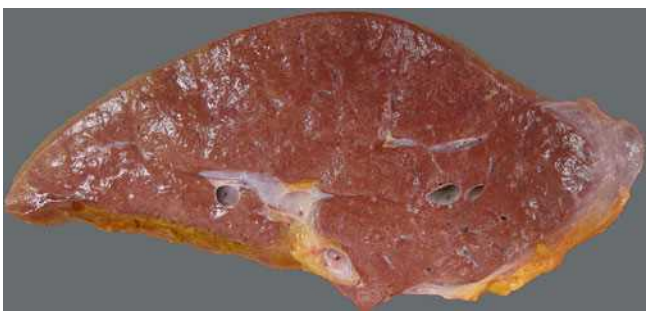

**Fig. 5.39** On section, the normal red-brown color and smooth surface of the liver can be seen. Conventionally, cuts are made 10 mm apart from right to left, to help identify tumors and fibrosing processes

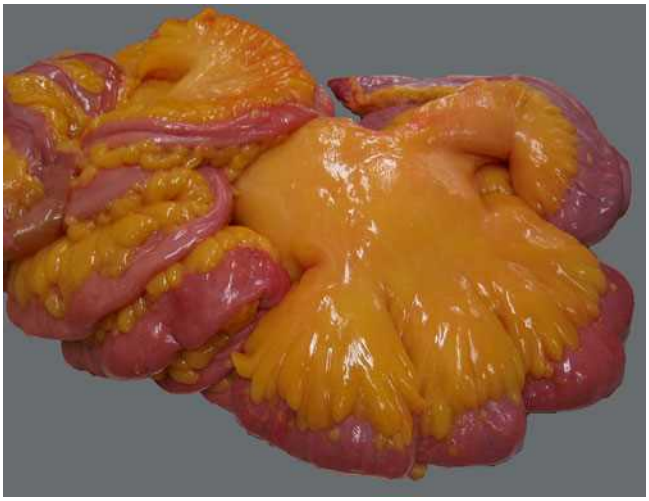

**Fig. 5.40** A section of small intestine is seen with local fatty mesentery. Here there is some encroachment of fat onto the serosa of the bowel, but this is within normal limits. The mesentery, like the omentum, can contain abundant adipose tissue

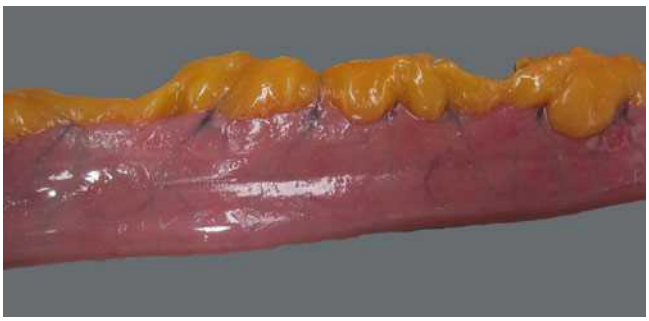

**Fig. 5.41** The small bowel (largely stripped of local mesentery) may be laid out relatively flat. Any masses of significance can be readily seen, often in association with obstructive phenomena. Note the normal, smooth serosal surface. Often, if the case pathology lies elsewhere, the bowel is examined no further, but there is an opportunity to open and wash out the bowel at this point

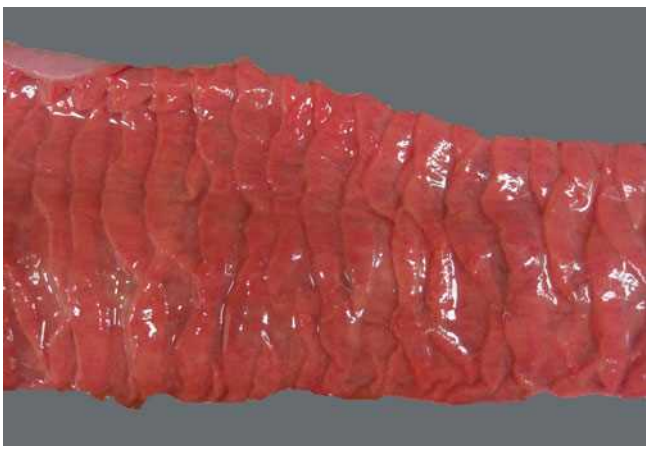

**Fig. 5.42** The tissues of the small intestine can be opened along their length. The intestinal mucosa does autolyse fairly quickly, but one often sees the normal circumferential folds. These folds are permanent and fixed, unlike the rugae of the stomach

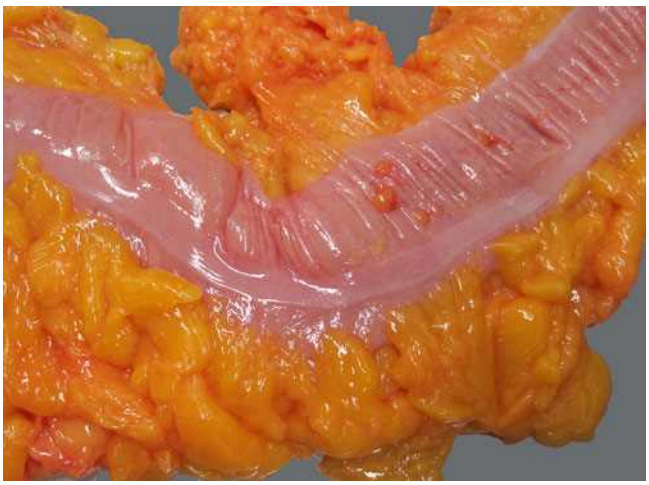

**Fig. 5.43** The large intestine can be similarly examined, first from the outside, revealing normal fat and serosal tissues. The longitudinal taeniae coli can be seen here on the surface of the sigmoid colon. Note the appendices epiploicae (fatty nodules). One should be aware that many individuals die with a variable amount of fecal matter within the lumen. Simple palpation and observation of obstruction may indicate pathology, but one should have a low threshold for opening the large bowel along its length

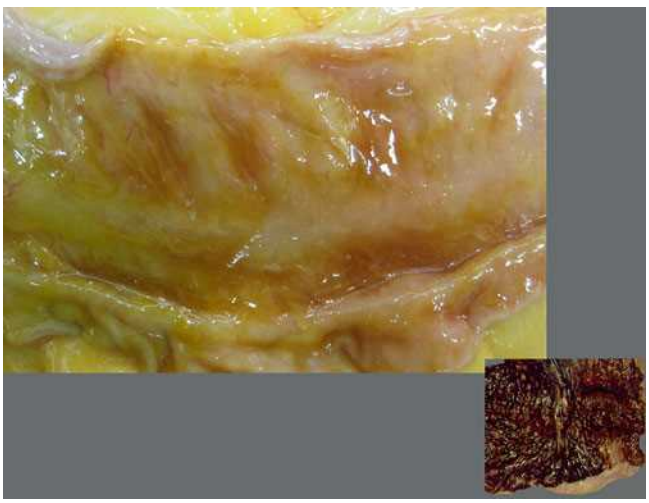

**Fig. 5.44** The large intestine mucosa is relatively featureless once it has been emptied of content and washed. Here it has been discolored minimally by the bowel content. Note the normal vascular markings. There may be extensive melanosis (brown discoloration), particularly in the elderly, due to lipofuscin deposition in the mucosa (*inset*)

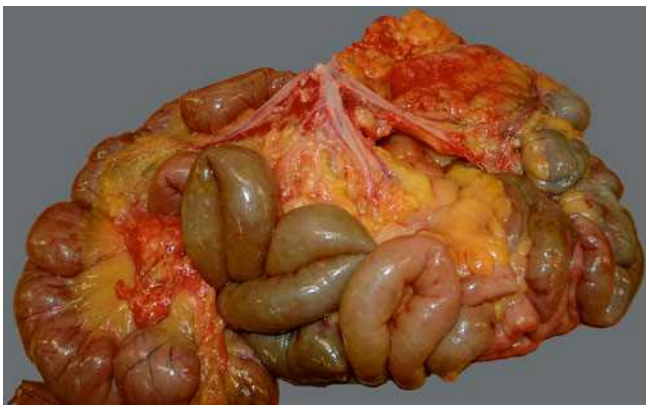

**Fig. 5.45** The superior mesenteric artery and its branches have been dissected within the omentum adjacent to the normal small intestine. Examining the vasculature in this way can help relate vascular lesions to end-organ changes

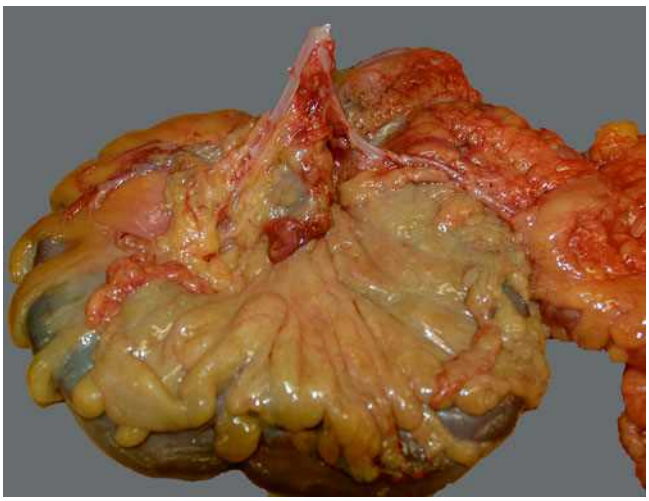

**Fig. 5.46** The inferior mesenteric artery is less frequently involved in bowel ischemia. Here it can be seen opened up along its branches in relation to a normal sigmoid colon

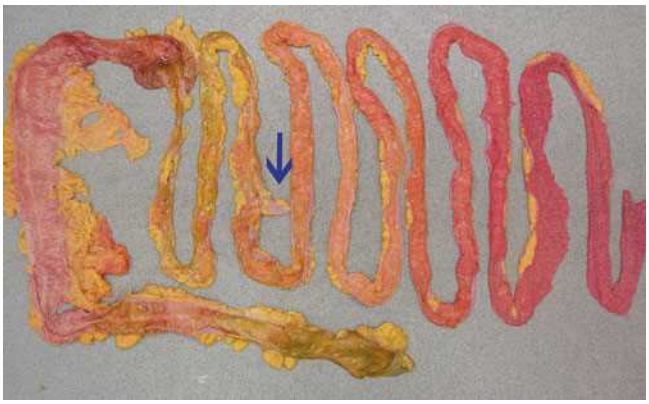

**Fig. 5.47** Displaying the whole intestine is difficult but useful, particularly if one wishes to demonstrate regional pathology (*e.g.*, inflammatory bowel disease, carcinomas, necrosis). Here the bowels were emptied, cleaned, and photographed (from a position well above the tissues). The mucosa is normal in this case, but an incidental Meckel's diverticulum (*arrow*) can be seen in the usual position, approximately 50 cm from the iliocecal valve

**Fig. 5.48** Peptic ulceration can occur at many points in the esophagus, often related to acid reflux, drug therapy, or both. The significance will depend upon whether bleeding has occurred from the ulcer bed, or if the ulcer interfaces with the mediastinal tissues. On occasion, a large abscess can form adjacent

**Fig. 5.49** This esophagus shows lower-zone scarring and sclerosis, resulting in a stricture. The origin may be acid reflux or it possibly may be neoplastic. Histology is important in reviewing such lesions

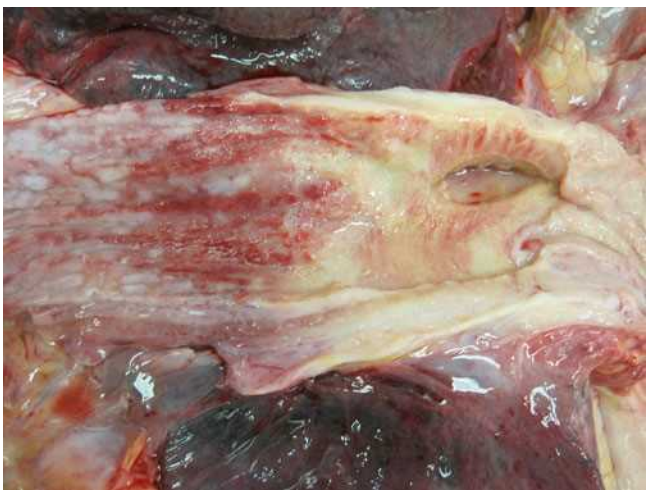

**Fig. 5.50** This view of the low esophagus shows a chronic ulcer just above the stomach inlet, with pronounced fibrosis locally. Immediately above the ulcer is a variegated grey/pink mucosal architecture, in keeping with Barrett's esophagus. The lower ulcer has a hard and sharp edge, pushing into the local soft tissues. There was no active bleeding or mediastinitis

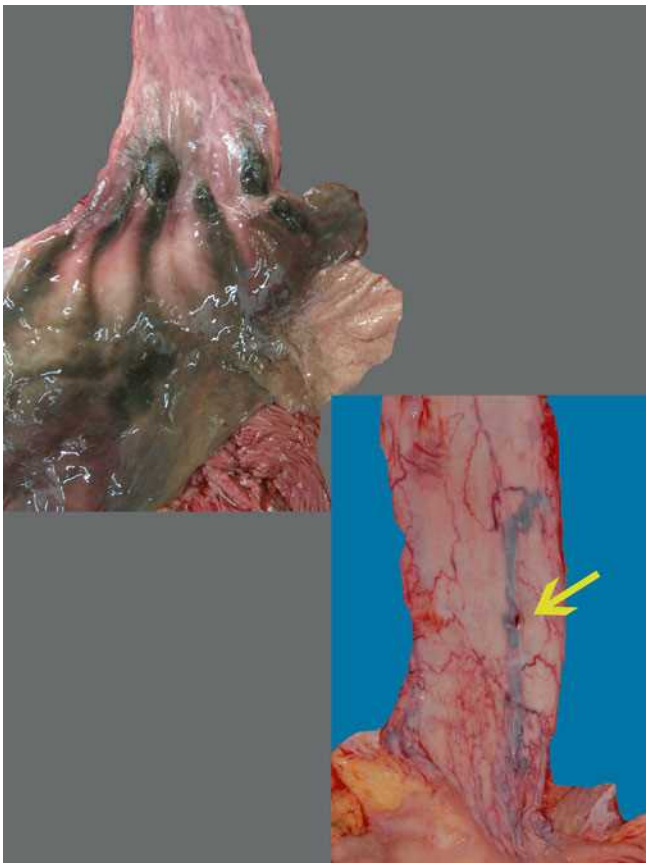

**Fig. 5.51** Esophageal varices may be seen with spontaneous thrombosis, or after sclerotherapy. In another case (*lower right inset*), death followed a single vessel bleed (*arrow*)

**Fig. 5.52** This esophageal tumor has perforated into the right-side thorax cavity. It was associated with direct involvement of the inflammatory process onto the adjacent lung tissue and with an active pleuritic reaction (seen with purulent material covering the diaphragm)

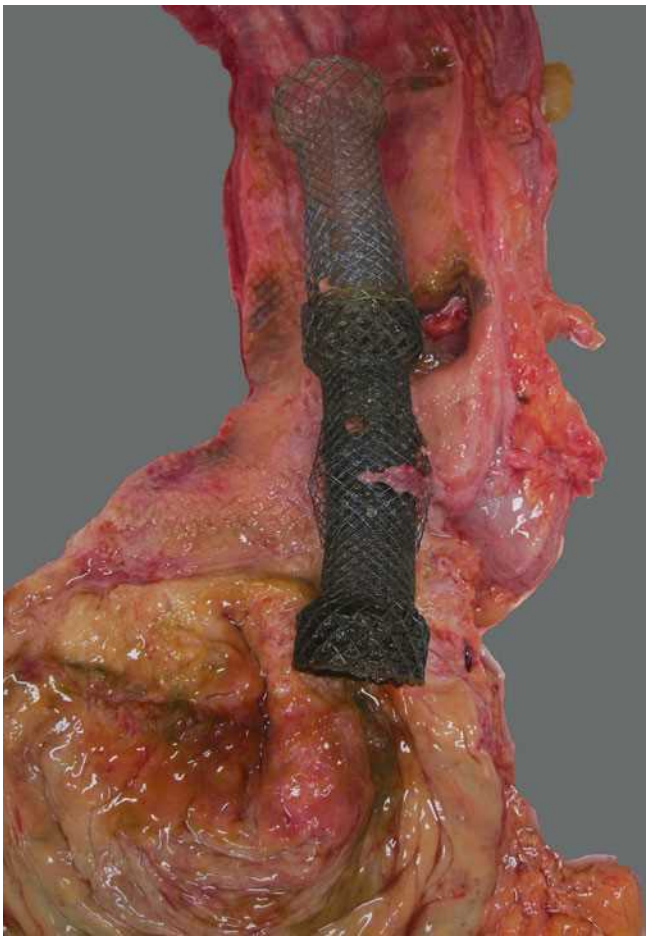

**Fig. 5.53** This esophageal carcinoma has been treated by means of metal stent insertion to preserve esophageal lumen integrity. The stents can be positioned in multiple segment format so that a longer zone of protection is offered. In this case, the junction point between two stents is associated with an area of ulceration. It is important to consider the stent integrity as well as the lumen when examining tumor tissues; careful dissection down onto the stent with a sharp scalpel is recommended

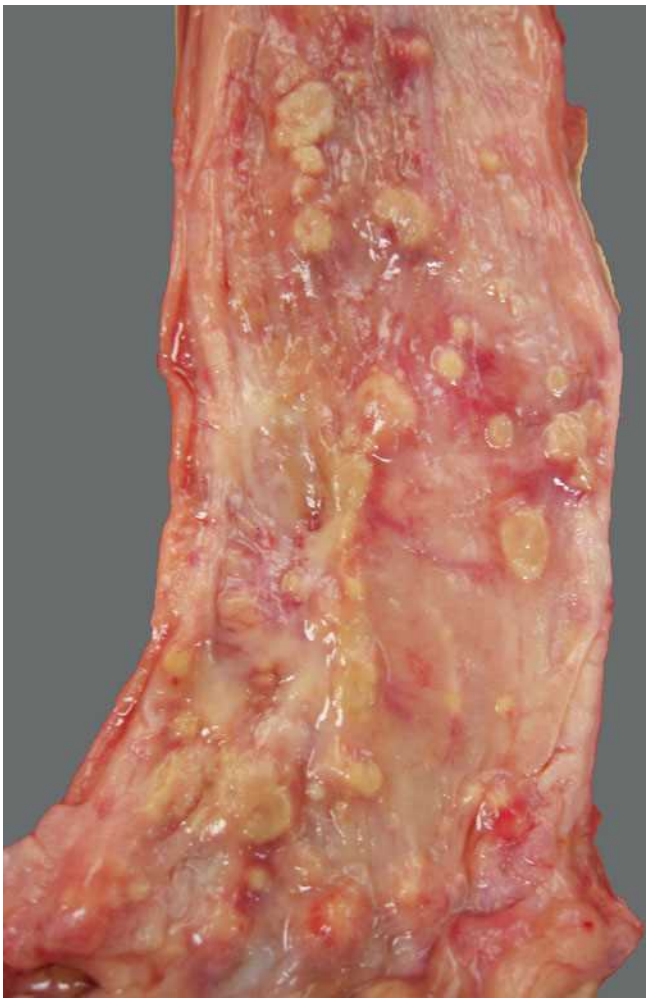

**Fig. 5.54** The esophageal tumor is seen to be spreading widely in lymphatics, with multiple nodules on the mucosal surface. This case was relatively early in stage, and the individual died from another disease

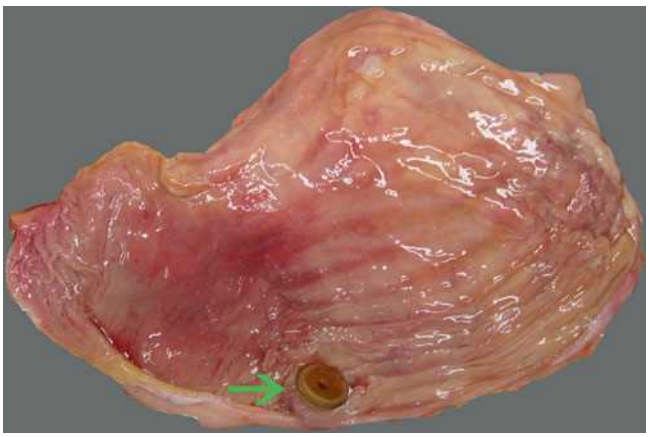

**Fig. 5.56** The inner aspect of a PEG tube (used for feeding purposes) is seen on the mucosal aspect of the stomach

**Fig. 5.57** Unusual items can be found in the stomach—in this case, a hearing aid in a demented patient

**Fig. 5.55** The stomach mucosal surface (*upper left*) shows widespread granular debris, in keeping with tablet residue. In suspected cases of drug overdose, the stomach contents (food and tablet residue) should be reserved and weighed and measured in terms of mass and volume, with a portion set aside for testing

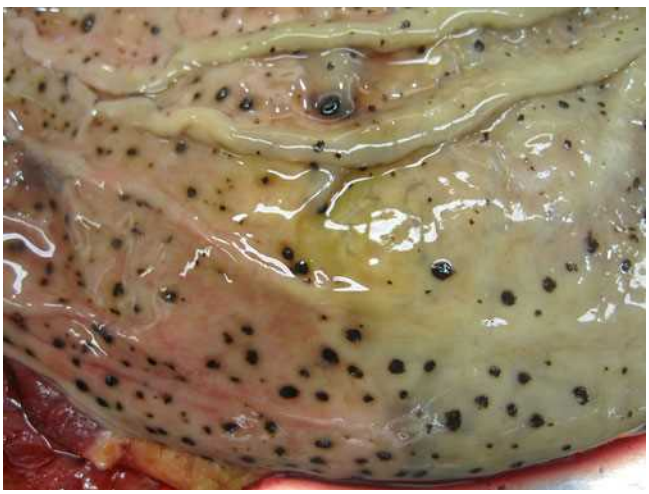

**Fig. 5.58** Multiple black mucosal patches are often seen on the stomach surface in terminal stress-related conditions. They represent microscopic areas of ante-mortem ulceration and haemorrhage

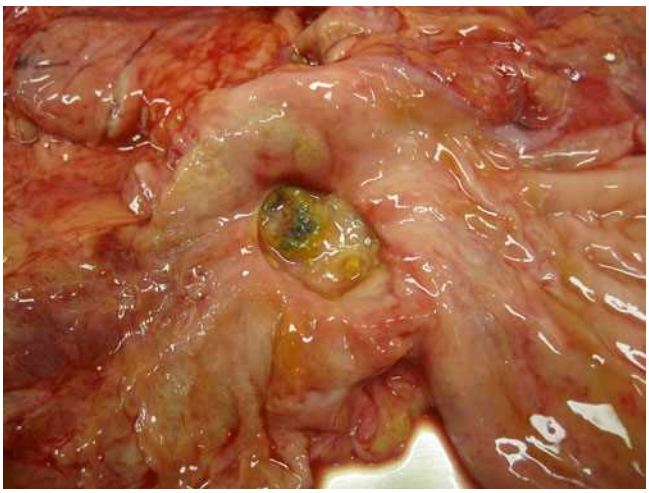

**Fig. 5.59** This benign gastric ulcer is seen to have a relatively sharply defined edge, together with black areas of discoloration around the base. These are blood vessels with some thrombus. They can be associated with significant haemorrhage

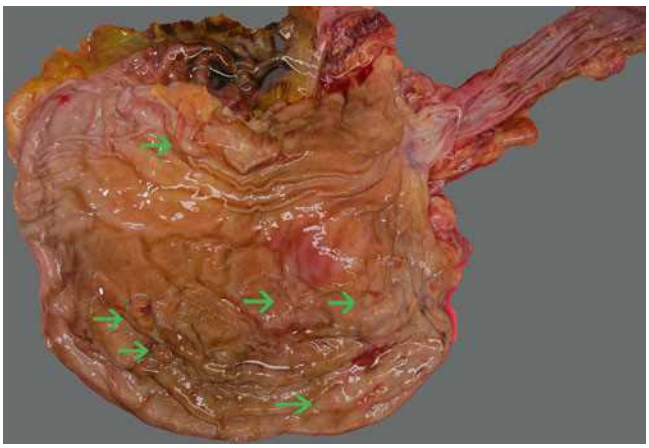

**Fig. 5.61** Multiple small nodules (*arrows*) are seen on the mucosal aspect of the stomach in this case of neuroendocrine cell hyperplasia with evolving neuroendocrine tumors

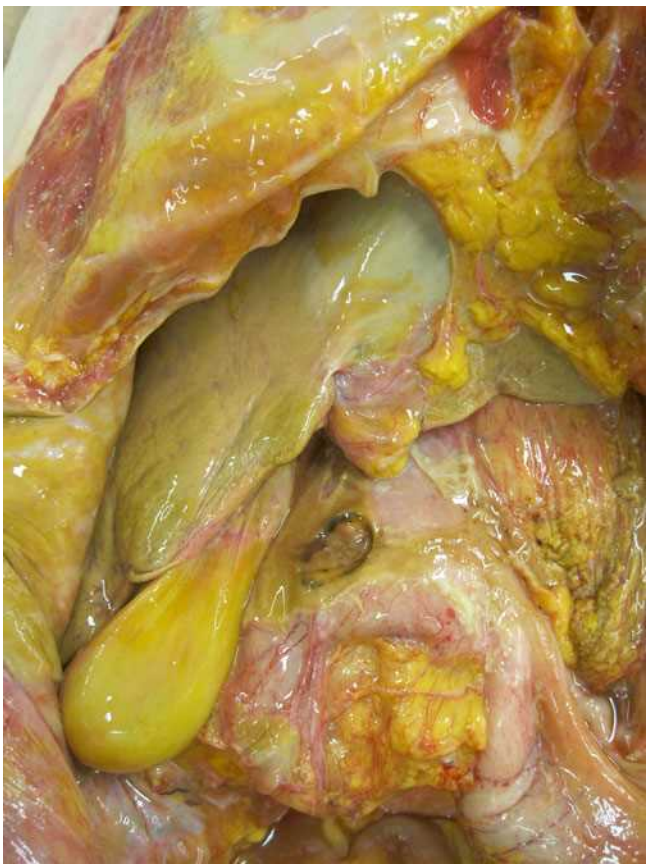

**Fig. 5.60** This view shows a pyloric peptic perforation with associated peritonitis in a case of chronic peptic ulceration. Around the edge of the ulcer, where it interfaces with the serosal surface, is a zone of black tissue in the form of coagulated blood and necrotic debris

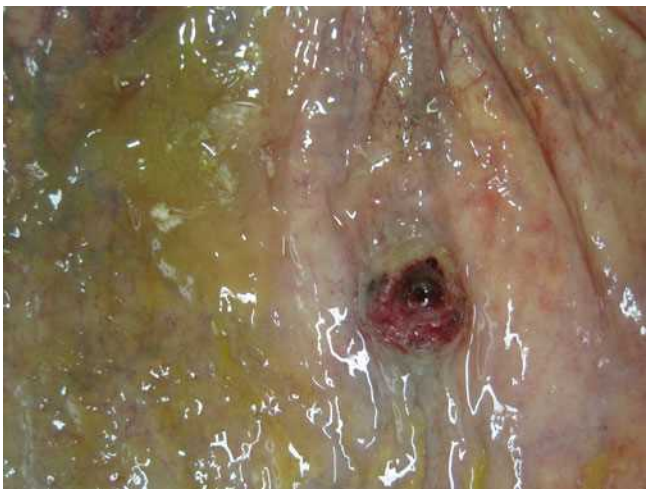

**Fig. 5.62** Stomach tumors vary in their architecture and appearance. Some are very bland and nodular, with relatively shallow ulcers. This stomach carcinoma showed notable haemorrhage and firmness in association with relatively little local mucosal infiltration

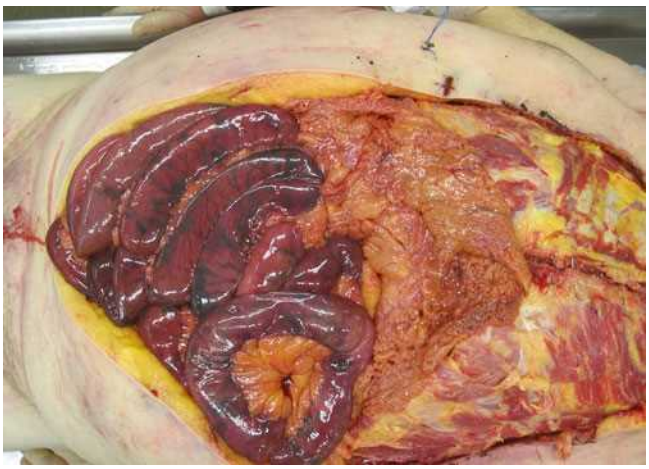

**Fig. 5.63** The small bowel is particularly vulnerable to hypoxia in cases of hypotension. This individual suffered a profound drop in blood pressure following myocardial infarction, resulting in secondary small bowel mucosal necrosis and haemorrhage. This situation worsens the hypotension by virtue of blood loss, fluid loss, and toxemia; it also may result in small bowel perforation and peritonitis

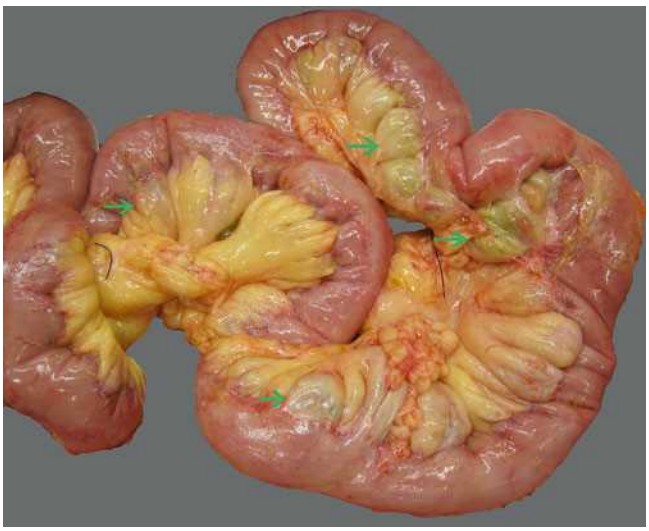

**Fig. 5.65** Jejunal diverticulosis is seen as thin-walled outpouchings (*arrows*) of the small bowel. This condition is often silent but may be associated with malabsorption conditions

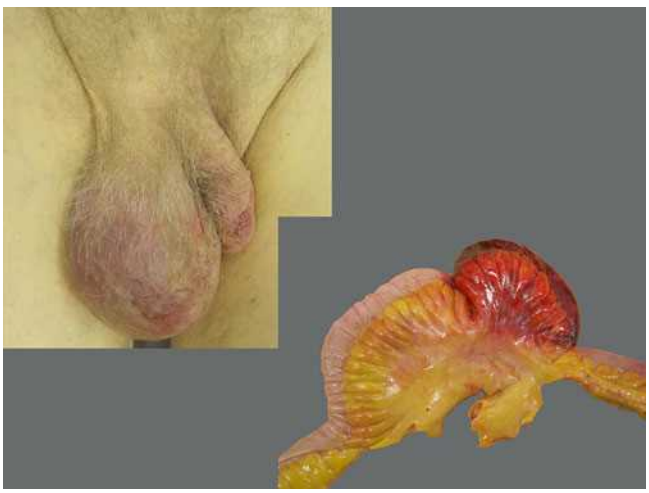

**Fig. 5.64** (**a**, **b**) This segment of small bowel had been incarcerated in a hernia sac, leading to mucosal necrosis with secondary toxemia and bacteremia

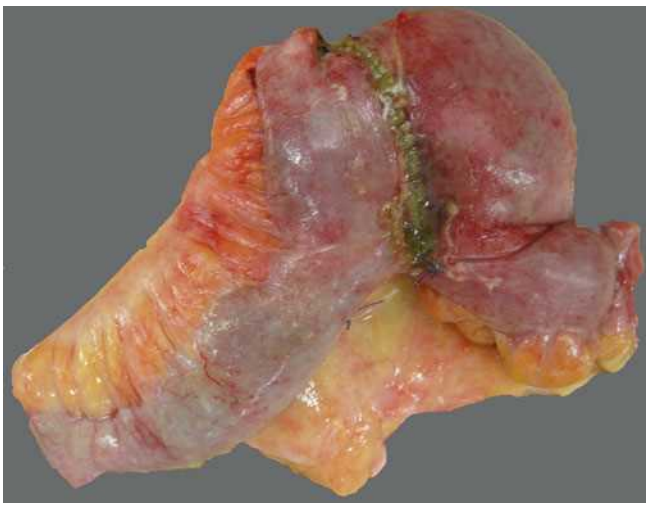

**Fig. 5.66** Inflammation and necrosis around a poorly vascularized anastomosis can lead to significant bacteremia/peritonitis and thereby death. The postoperative status of all gastrointestinal surgical cases needs to be checked for possible leakage and mural necrosis. Histology may also be of benefit in these cases

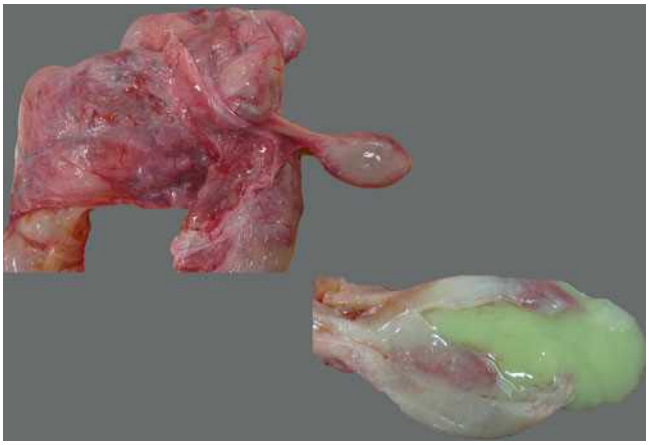

**Fig. 5.67** An acutely inflamed, but not ruptured, appendix is seen. The fluctuant and dilated tip of the appendix is noted to be discolored. When incised (*inset*), the same appendix leaked purulent content

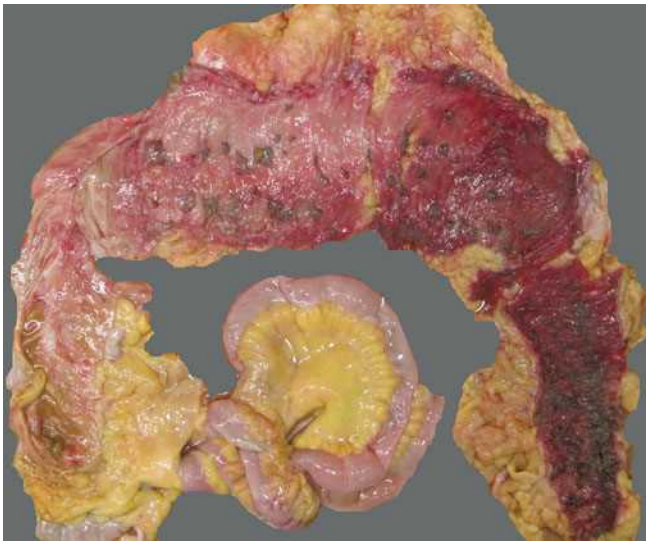

**Fig. 5.69** An acute-on-chronic case of ulcerative colitis, showing classic inflammation and ulceration, particularly towards the left side. Note that the small bowel is spared

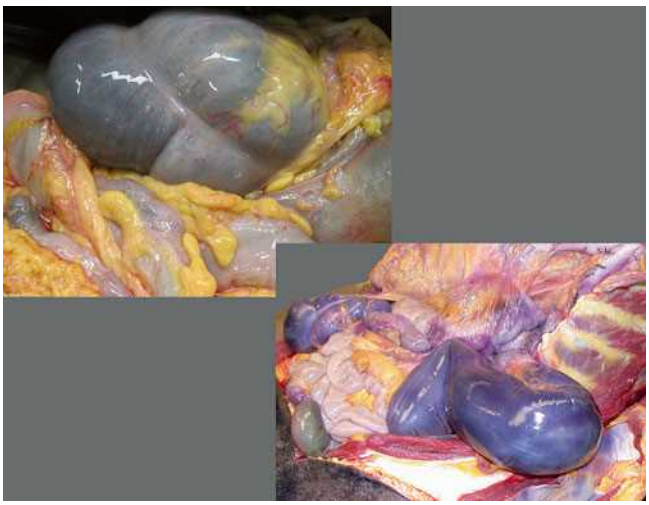

**Fig. 5.68** The color of bowel tissues may be markedly altered by contents, as in the case of an individual with pronounced dietary intake of beetroot (*lower right*). The dark gray and black color (*upper left*) was initially considered to be evidence of melena, but there was no gastrointestinal bleeding. These tissues were discolored by oral iron (ferrous sulfate) therapy

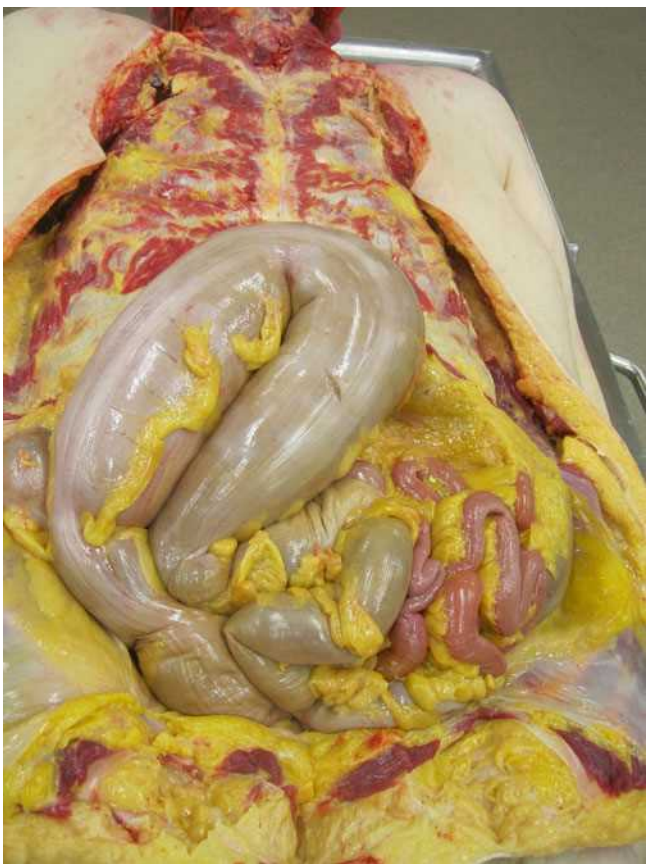

**Fig. 5.70** A sigmoid volvulus is present, with dilated, gas-filled content pushing the sigmoid tissues up and towards the liver. Associated twisting of the mesenteric tissues can result in not only bowel stasis or obstruction but also bowel necrosis and peritonitis

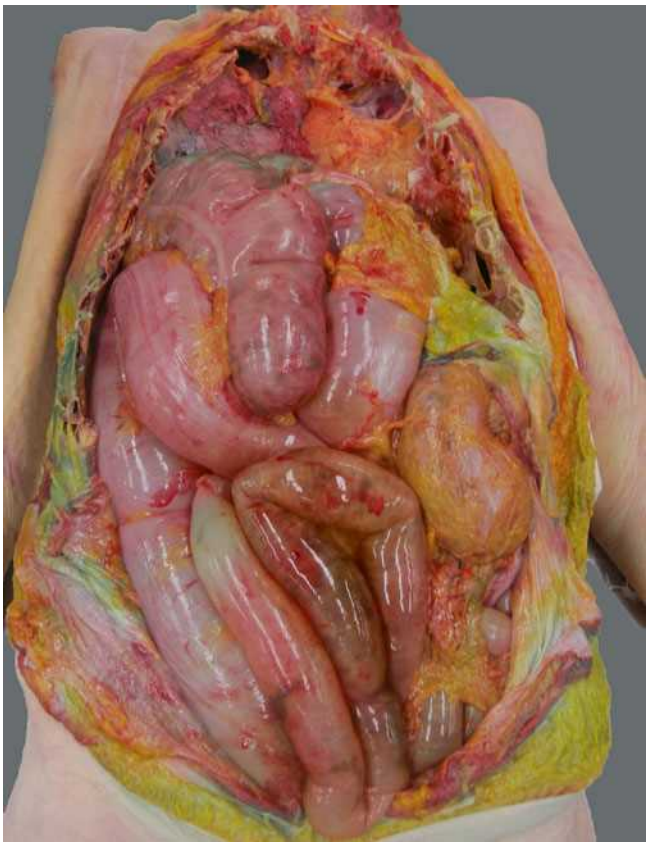

**Fig. 5.71** Postoperative complications may include significant ileus. This individual showed pronounced gaseous dilatation of the small and large bowel in association with intestinal pseudo-obstruction. Nevertheless, an anatomical or pathological reason for obstruction should be sought in all cases by opening of the bowel

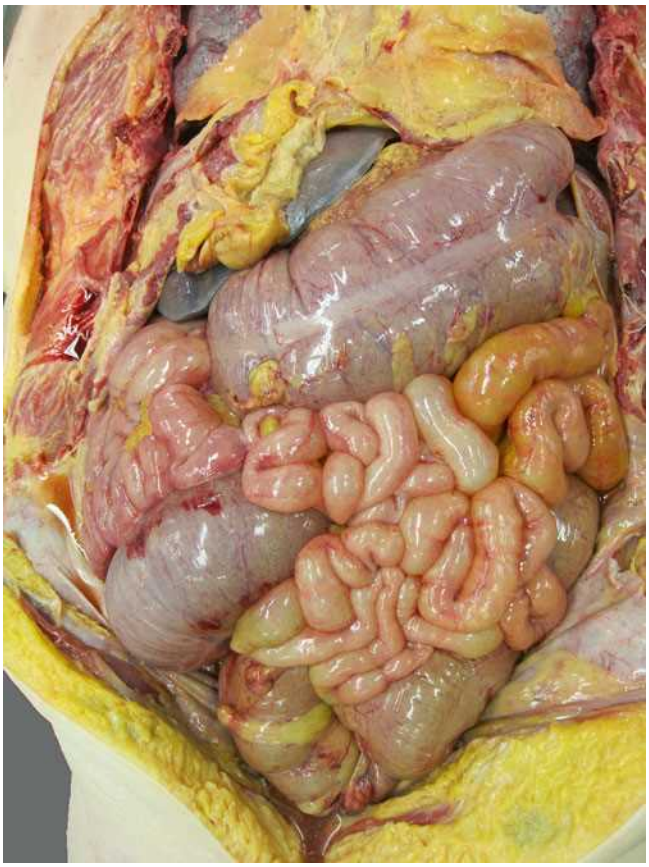

**Fig. 5.72** Large bowel obstruction is seen, suggesting a rectal/sigmoid obstruction lesion

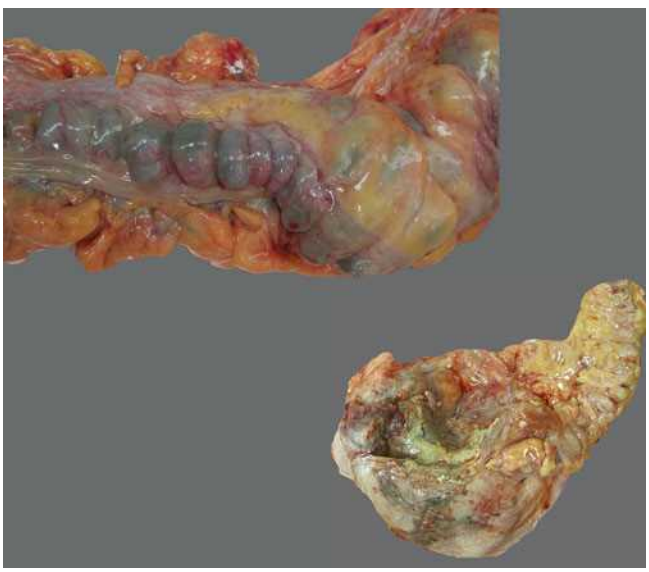

**Fig. 5.73** Diverticular disease of the colon (*upper left*) is seen with pronounced saccular dilatations on the serosal surface. These are common findings at autopsy and are rarely of significance unless there is rupture and associated peritonitis (*lower right*)

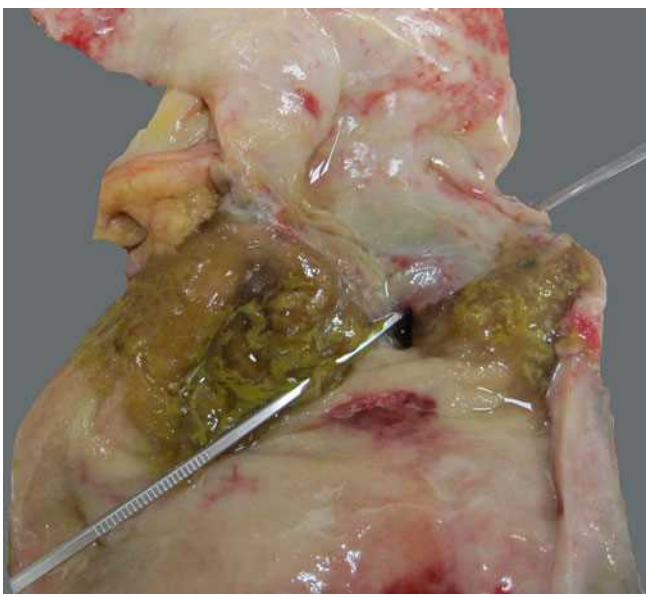

**Fig. 5.74** Focal large bowel ulceration and perforation in a case of vasculitis

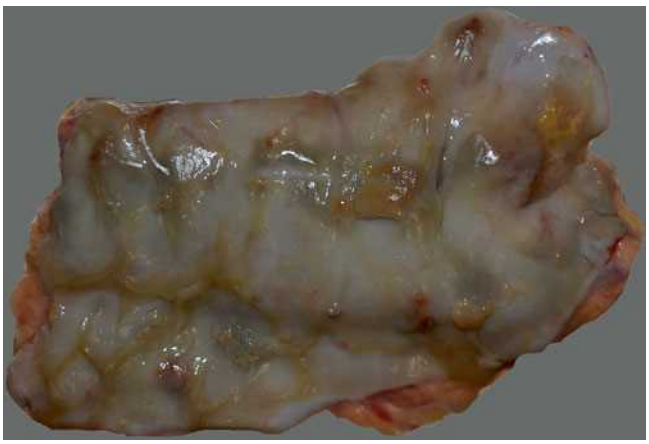

**Fig. 5.75** *Clostridium difficile* colitis. Small areas of ulceration, mucosal necrosis, and exudative change represent the pseudomembranes. At autopsy, they are often darkly stained by fecal material

**Fig. 5.76** Metastatic tumors to the bowel can be part of disseminated cancer cases. This image shows the serosa adjacent to the colon in a case of disseminated lung cancer

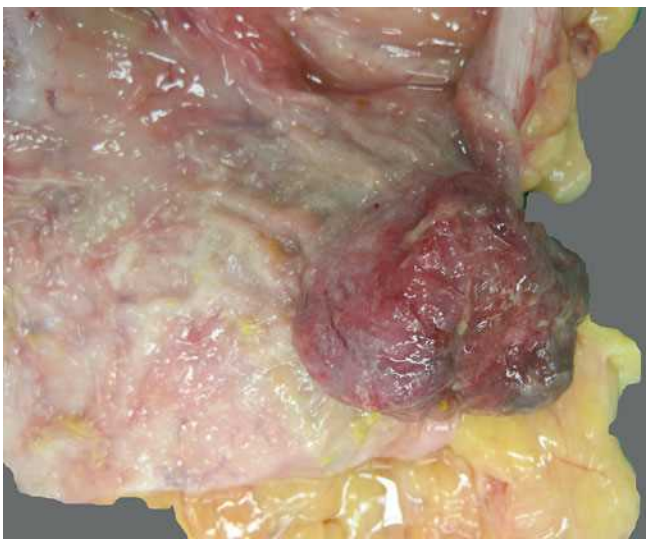

**Fig. 5.77** Colon adenomas are commonly seen at autopsy. They have no significance unless they are obstructive or bleeding

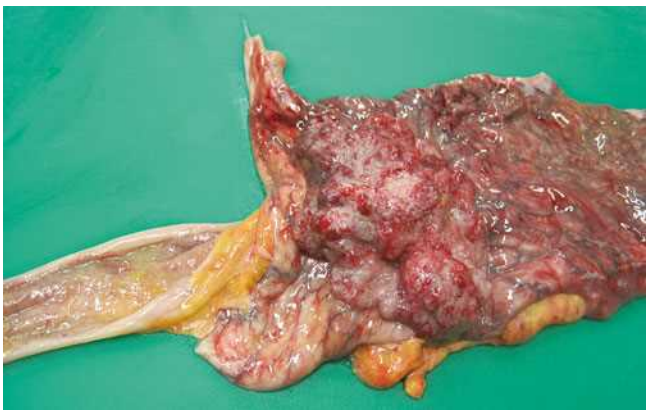

**Fig. 5.78** Carcinoma of the colon can be the cause of death in some individuals with bowel obstruction and/or perforation. Disseminated bowel cancer is relatively common in terms of autopsy analysis, but incidental findings of unsuspected adenocarcinoma can be identified by careful palpation of the bowel tissues during evisceration and opening the bowel at suspect sites

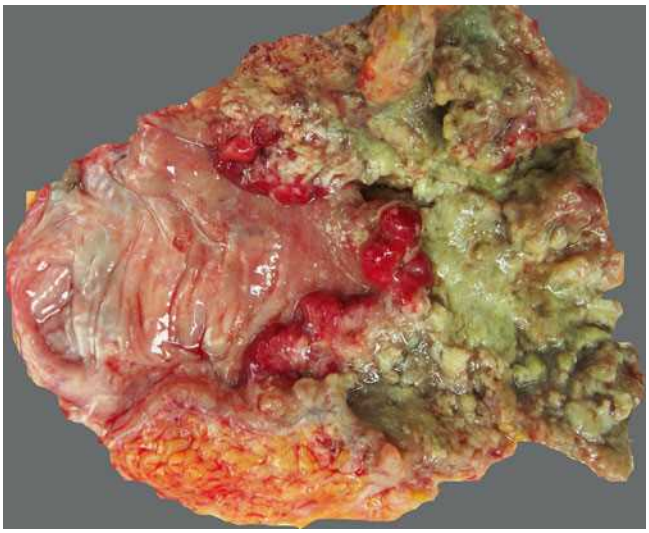

**Fig. 5.79** Some cases of advanced rectal cancer are occasionally found at autopsy. They need to be staged in the same manner as antemortem cases

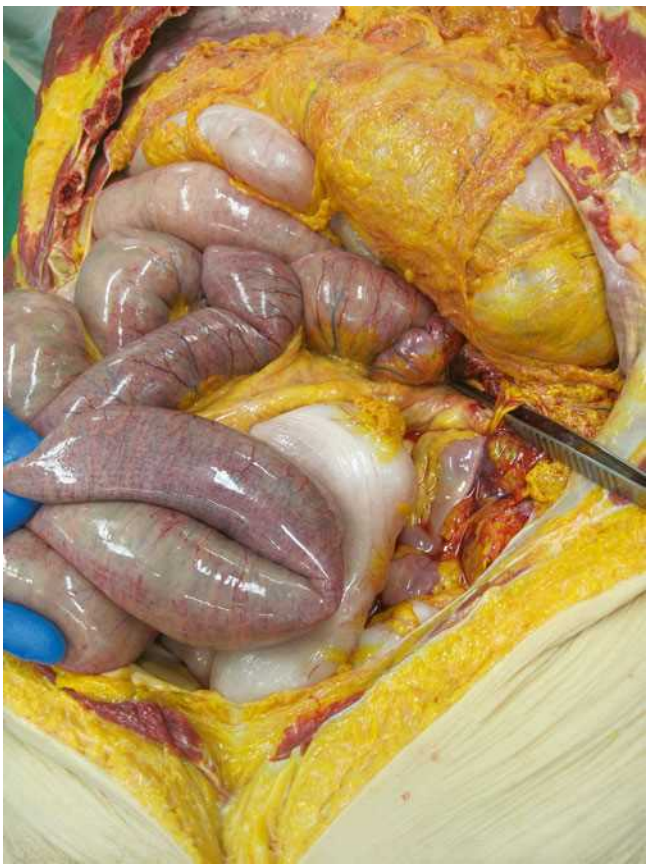

**Fig. 5.80** Fibrous adhesions and bands are often consequences of previous surgery or instrumentation of the abdominal cavity. On occasion, they can obstruct the bowel tissues

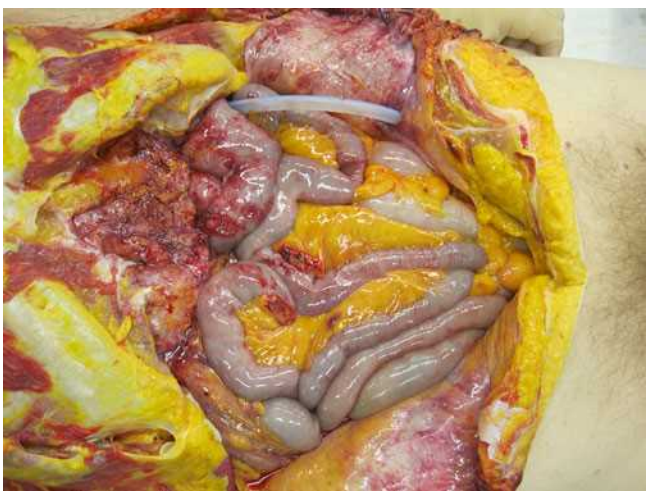

**Fig. 5.81** Postoperative changes in relation to abdominal surgery need to be explored carefully. Here, there is pronounced haemorrhage around the site of previous closure of a peptic ulcer by means of oversewing. One also needs to consider the placement of any drains (entering through the left low abdomen) and evaluate any collections, blood loss, or further pathology

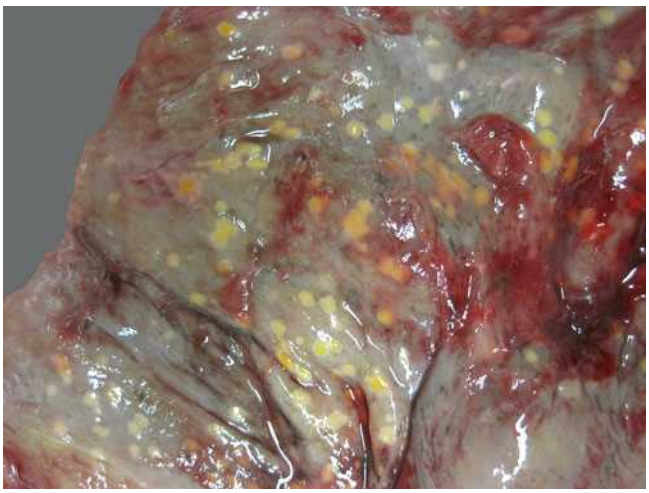

**Fig. 5.82** The peritoneal surface is seen to be inflamed and congested, with areas of fat necrosis, in this case of pancreatitis. These findings represent liberation of pancreatic enzymes into the peritoneal cavity, with patchy saponification of tissues

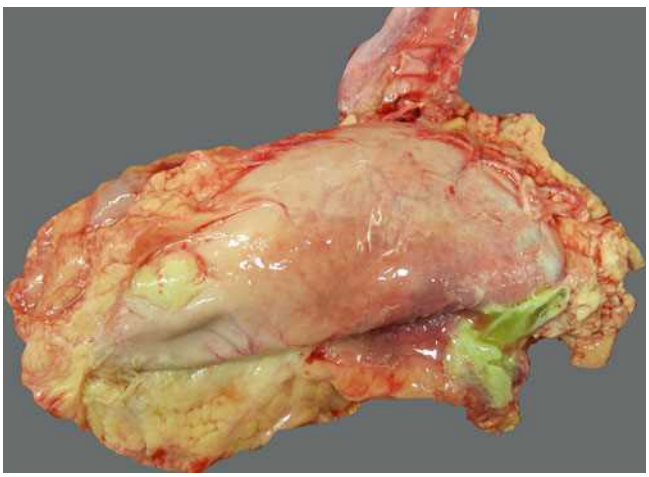

**Fig. 5.83** The stomach has been pushed upwards in this case, revealing focal, early acute pancreatitis

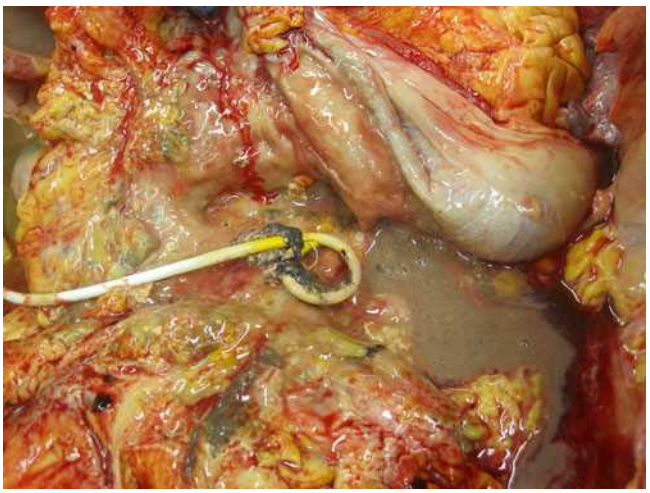

**Fig. 5.84** A large pancreatic pseudocyst with associated seminecrotic fatty residue is seen in a case of acute-on-chronic pancreatitis. A pigtail catheter was being used to drain out the collection via the anterior abdominal wall, but the patient was nevertheless overwhelmed by the pancreatitis

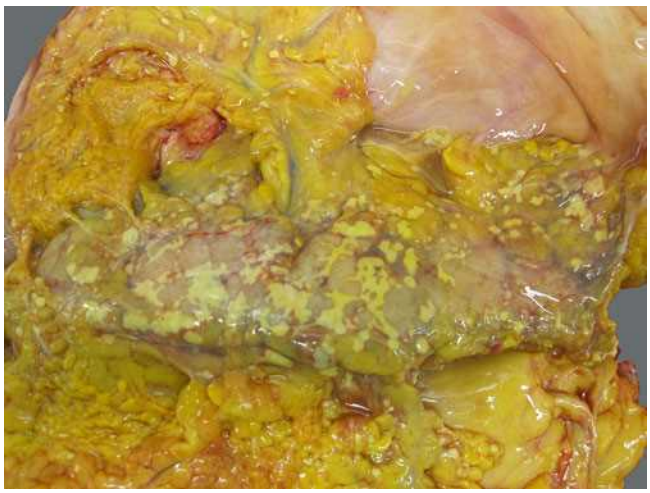

**Fig. 5.86** Pancreatitis occasionally can be suspected in cases of peritonitis. This mottled fat necrosis change was present only on the pancreatic serosal surface. No deep parenchymal disease was present

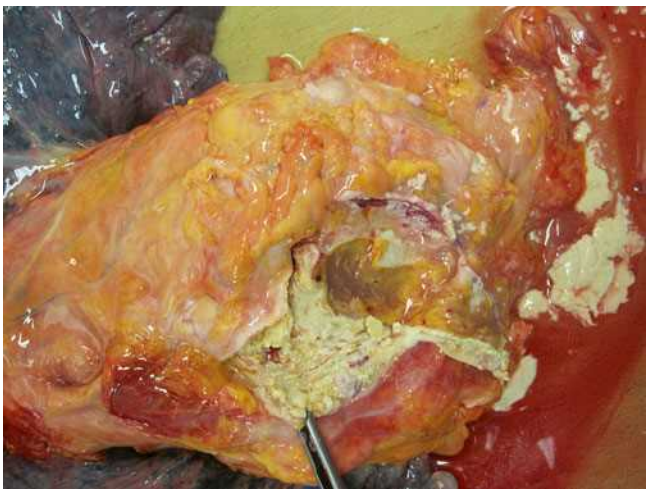

**Fig. 5.85** A pancreatic pseudocyst is seen with inflammatory content and marked local fat necrosis. The stomach has been lifted upwards and the omentum has been cut to facilitate the view of the pathology

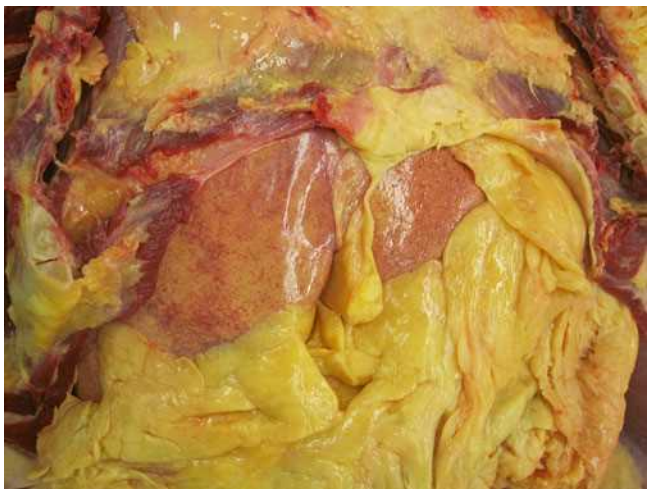

**Fig. 5.87** Fatty change in the liver is suggested by a yellow/orange discoloration of the liver parenchyma. The liver itself often is enlarged

**Fig. 5.88** Fatty livers tend to have a greasy cut surface, but in extreme cases the fatty tissue is capable of floating—here within a sink of water

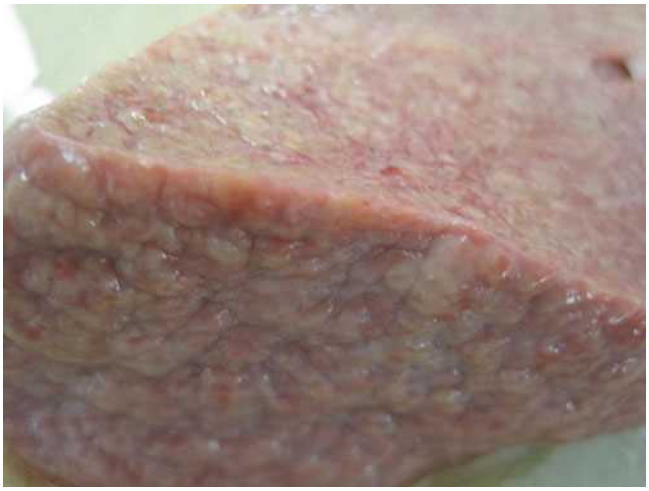

**Fig. 5.90** Liver cirrhosis is characterized by nodularity not only on the surface of the tissue but also extending into the deep parenchyma. Nodules vary in size from a few millimeters up to a centimeter or more

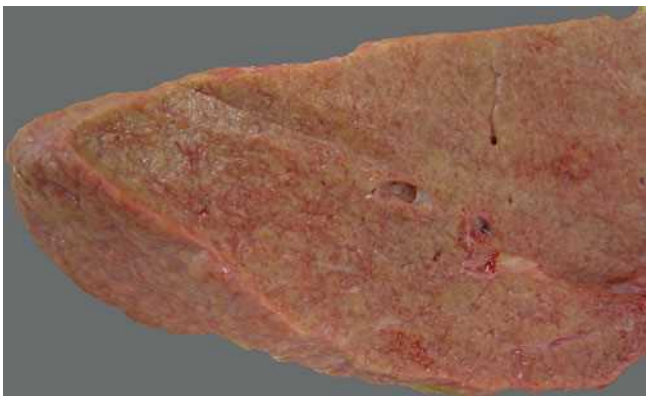

**Fig. 5.89** The liver can show pronounced fibrosis, bordering on micronodular cirrhosis. In such circumstances, any cuts made into the liver tend to hold their shape, and the cut surface of the liver also shows slight granularity. Histological review of such cases is important, as the full criteria for cirrhosis may not be present

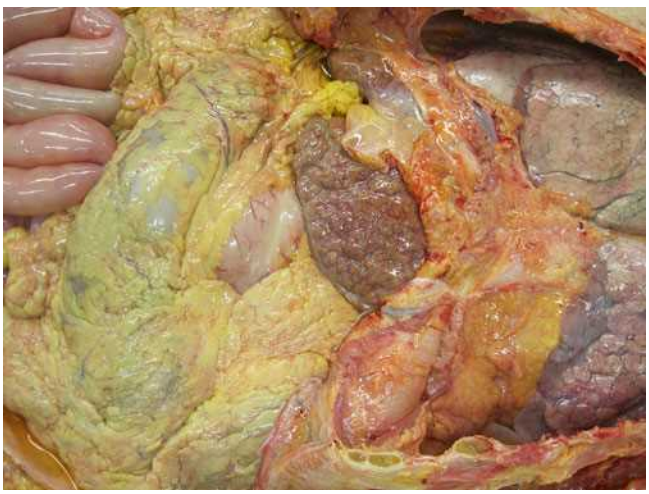

**Fig. 5.91** Liver cirrhosis often can be identified at the beginning of the autopsy examination, with the liver edge being clearly seen to have adjacent nodularity and fibrosis. One should be aware of the need to check for other lesions such as esophageal varices and other features of liver failure

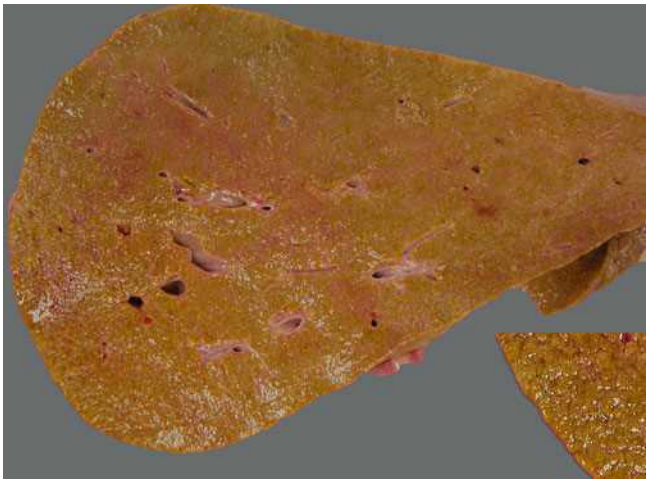

**Fig. 5.92** Micronodular cirrhosis is classically associated with excess alcohol intake but is not specific in this regard. Pronounced yellow/green color points to fatty change in surviving parenchyma and some bile stasis. The *inset* shows the fine nodularity

**Fig. 5.94** Cirrhosis is often accompanied by clear, yellow ascites, as a feature of altered blood flow through the liver and a hypoproteinemic state. If the fluid is opaque or turbid, one may suspect spontaneous bacterial peritonitis, which can significantly aggravate any case of cirrhosis and can be fatal

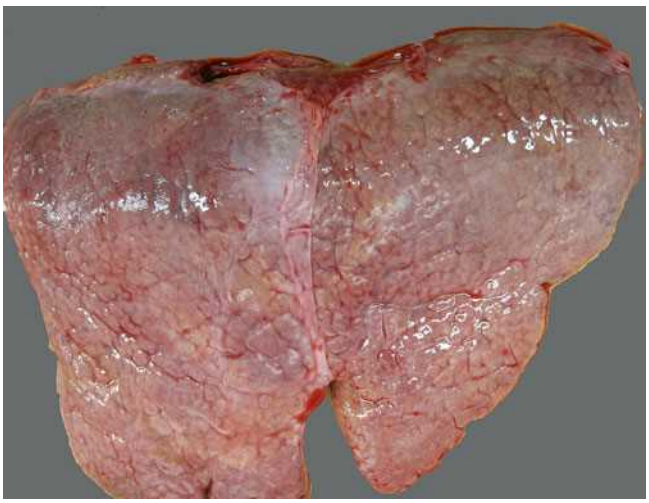

**Fig. 5.93** Macronodular cirrhosis may be encountered in many different pathologies that end with cirrhosis

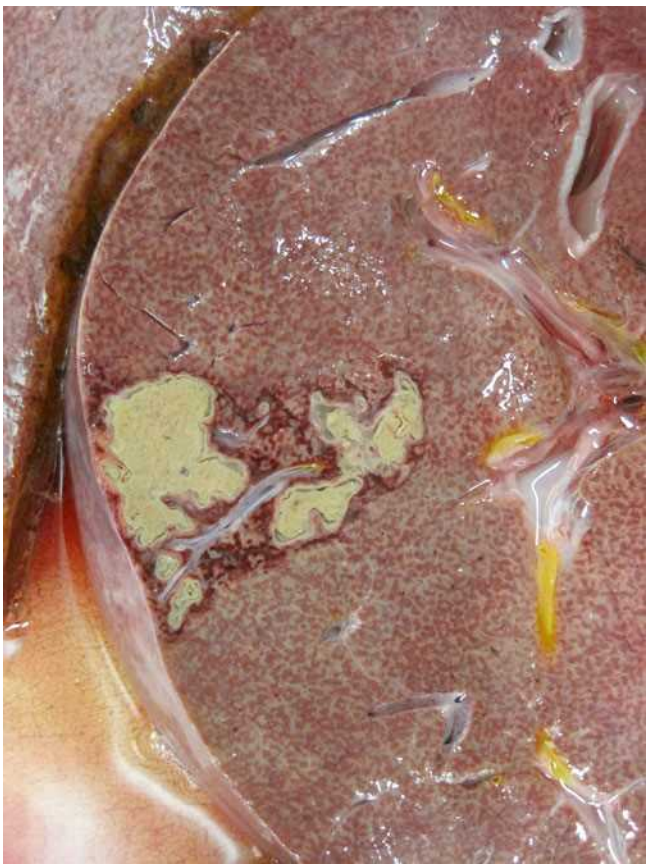

**Fig. 5.95** A wedge-shaped infarct is seen in a case of infective endocarditis. Yellow discoloration is present, with associated moderate congestion and haemorrhage

**Fig. 5.96** Exacerbations of liver function can be associated with coagulopathies. Central venous congestion and then thrombosis has occurred in this case, with an acute deterioration of liver function as a terminal event

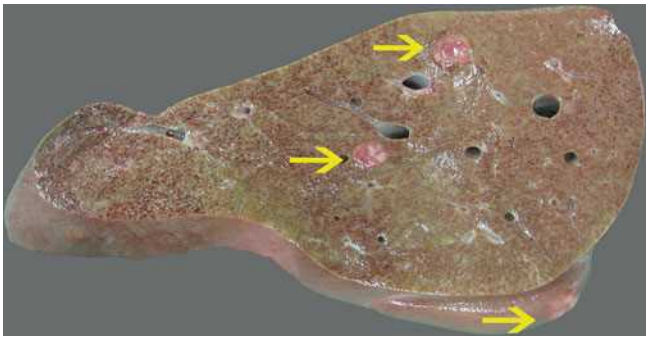

**Fig. 5.99** Metastatic disease to the liver is commonplace at autopsy. Multiple lesions (*arrows*) are present. A rigorous search for the primary should be undertaken, with consideration of the prior medical history. Sampling for histological confirmation is often required, unless the tumour was documented before death

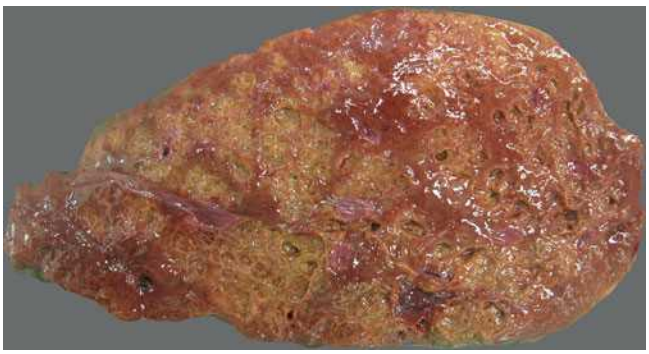

**Fig. 5.97** Decomposing bodies often present gas collections in the liver. This 'Swiss-cheese' effect, with softening/liquefaction change, may indicate terminal bacteremia or may just be part of general decomposition

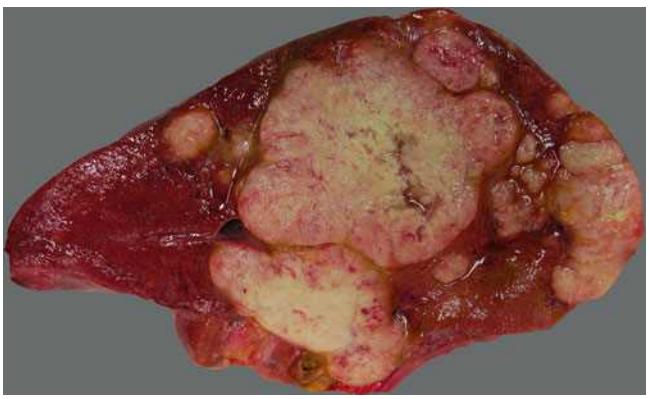

**Fig. 5.100** The degree of carcinomatous infiltration of the liver can vary. Indeed, widespread infiltration of the liver can be associated with normal liver function. This individual had squamous carcinoma of the cervix with widespread bone, liver, and lung metastases. The ultimate cause of death was in fact pulmonary embolism

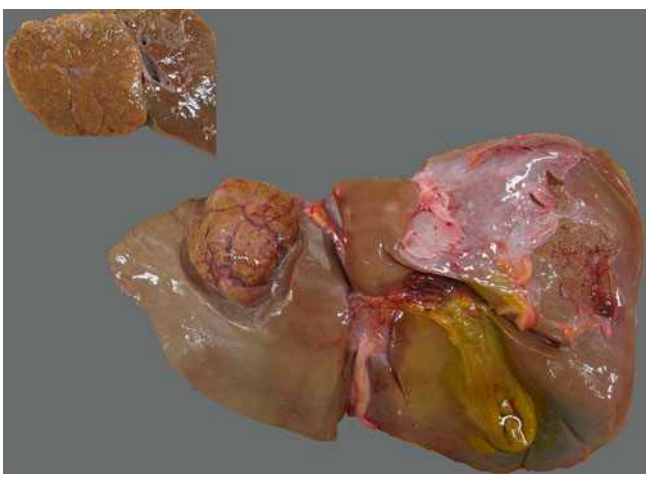

**Fig. 5.98** A rounded liver tumor is seen on the undersurface of the left lobe. This tumor has a characteristic bosselated and folding architecture. On section (*top left*), there is central fibrosis and distortion in keeping with a liver cell adenoma

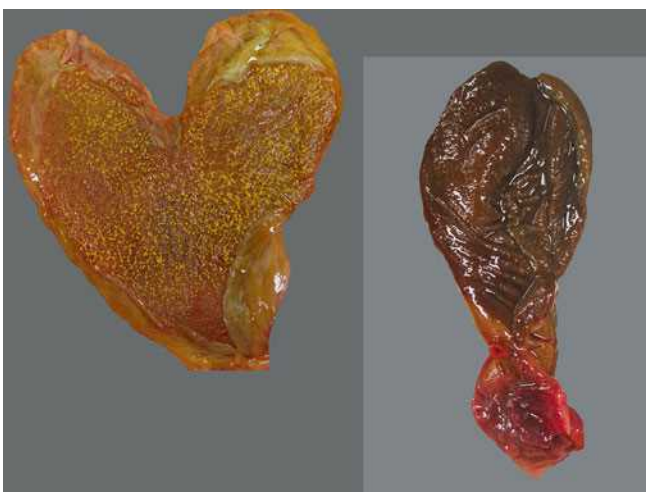

**Fig. 5.101** Cholesterosis of the gallbladder is characterized by small, yellow foci of discoloration on the mucosal surface. These foci represent macrophages with abundant fat and are commonly seen in association with gallstones. The features are contrasted with a normal gallbladder (*inset*)

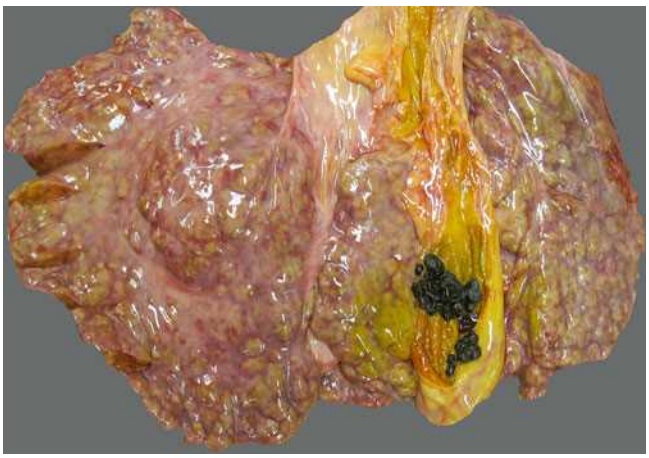

**Fig. 5.102** A case of liver cirrhosis is seen in association with gallstones. The gallstones were incidental to the background liver disease, as is the case in many autopsy examinations

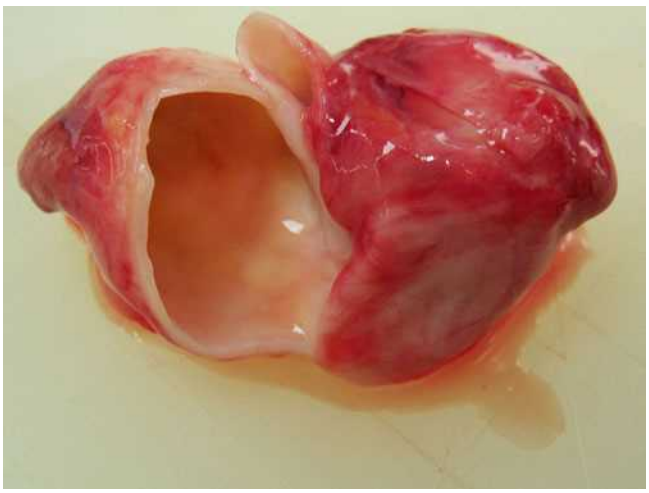

**Fig. 5.104** Gallstones impacted at the neck of the gallbladder may cause chronic changes that end with a mucocele. The gallbladder content will be clear fluid

**Fig. 5.103** At top left, the gallbladder is clearly visible at the point of opening the abdomen; it is filled with a single, rounded stone, and there is local fibrosis. The adjacent liver is noted to be finely granular and cirrhotic; these findings can represent a hepatic inflammatory or degenerative condition, or gallstone disease may have previously obstructed the bile ducts. At lower right, the image shows a rounded and pigmented stone in the gallbladder. Note the pale pink/beige mucosal surface in this case of chronic cholecystitis

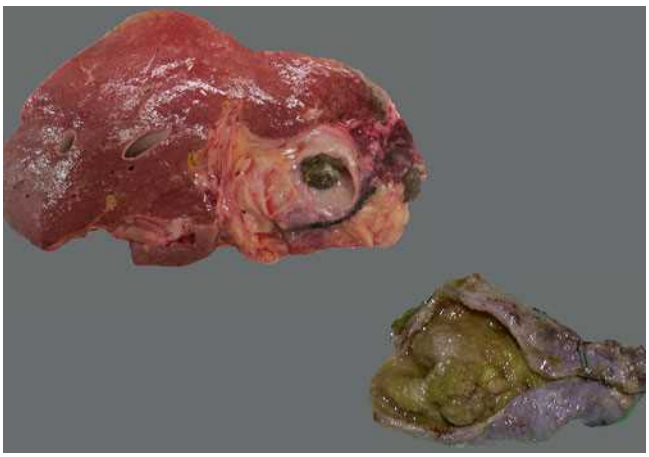

**Fig. 5.105** Gallbladder carcinoma often is associated with gallstone disease. The autopsy may demonstrate widespread tumor infiltration of the liver, but on occasion (*top left*), the gallbladder simply will show more marked fibrosis and firmness around the gallbladder bed. At lower right, the image shows a surgical pathology (fixed) gallbladder with mucosal irregularities in a case of adenomatous disease transforming to carcinoma

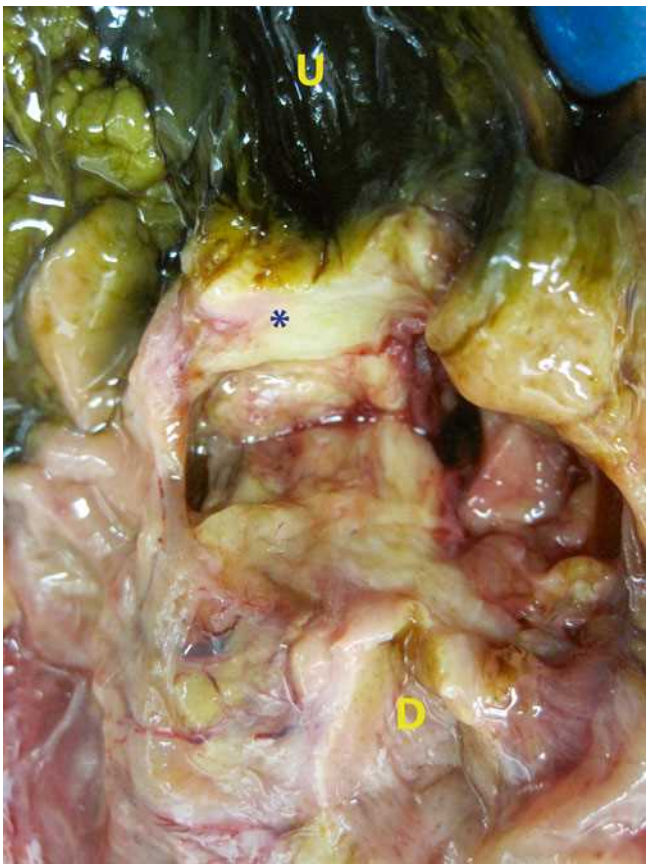


**Fig. 5.106** The common bile duct is seen in a close-up view. On section, a firm, cream-colored tumor (*) is present. On one side, there is intense bile discoloration within the upper (*U*) dilated duct, with the duct carcinoma blocking further flow of bile. The downstream (*D*) duct is smaller in transverse section and less bile-stained

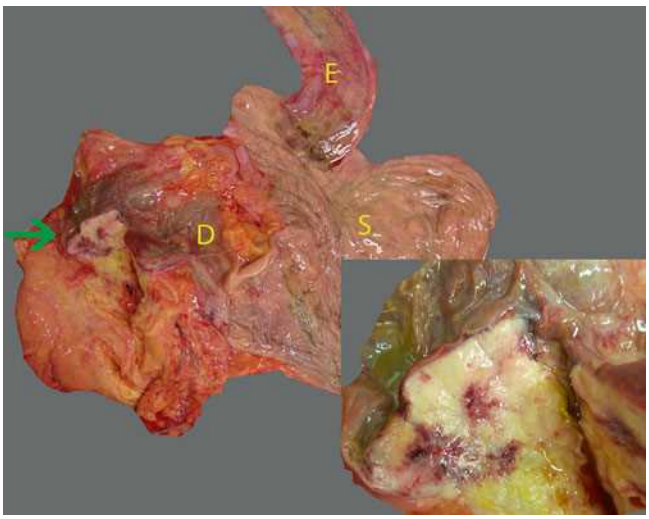


**Fig. 5.107** A tumor at the end of the bile duct, seen in a patient suspected of having carcinoma of the bile duct, but with histology more in keeping with carcinoma of the pancreas. Such cases can be the source of disseminated neoplasia; one needs to dissect the tissue carefully to confirm that the pancreas is the source of the neoplasia. Here, a firm, cream-colored tumor (*arrow*) is present adjacent to the proximal duodenum (*D*). At high magnification (*inset*), the tumor is seen lifting the duodenal mucosa upwards and extending into the adjacent soft tissues. The stomach (*S*) and esophagus (*E*) are unremarkable

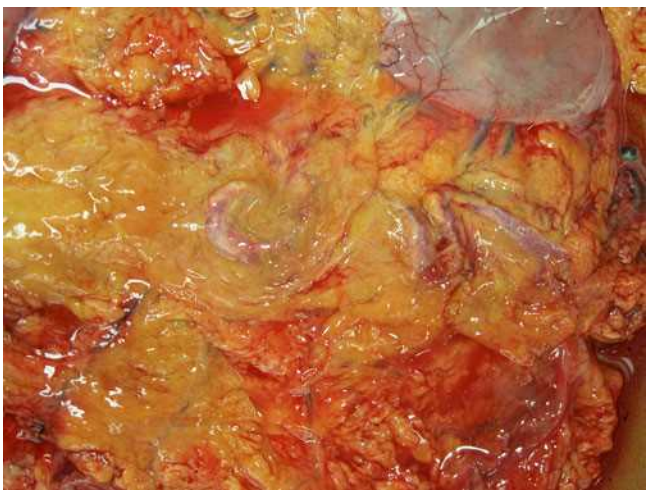

**Fig. 5.108** Calcification of the splenic artery is common. It is rarely associated with pathology, but is a marker for generalized arterial disease

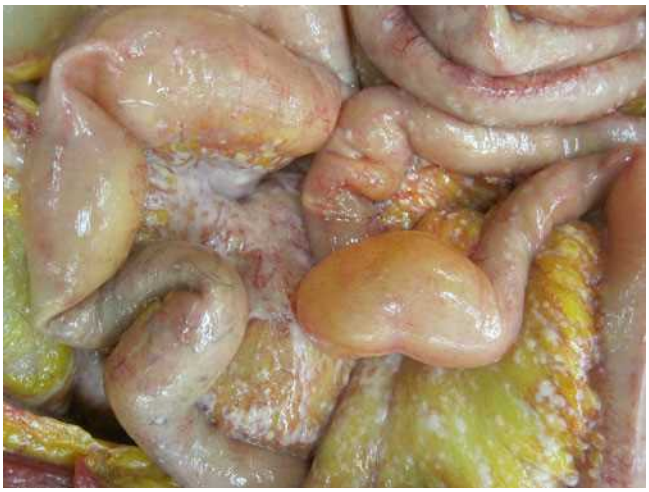

**Fig. 5.109** Pronounced serosal surface contamination by disseminated tumor is seen in this case. It is not possible to identify such tumors simply on morphology, although peritoneal carcinoma should be suspected in women. Mesothelioma may be more commonly suspected in men with an appropriate occupational history

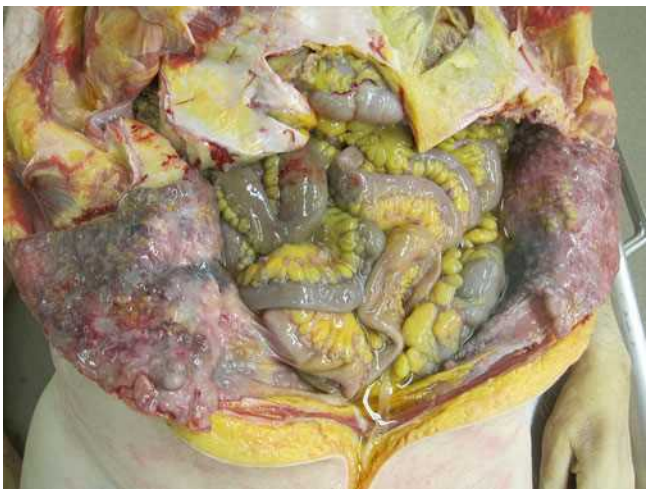

**Fig. 5.110** A case of advanced, diffuse peritoneal mesothelioma is seen with widespread infiltration of the abdominal wall and associated fluid collectiony

## Suggested Reading


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# Genitourinary and Breast Tissues, Including Pregnancy

6

S. Kim Suvarna

## Introduction

The chapter deals with a variety of tissues with some differences between the sexes and some things in common. The common elements include the kidneys, ureters, and bladder. These items are covered first, with gender-specific tissues and pregnancy covered later.

Genitourinary pathology is relatively commonplace in autopsy practice. Common themes include sepsis of the urinary tract, which can be acute or subacute/chronic. Chronic damage (from many causes) can have significant effects on the kidneys in particular, with secondary systemic effects. Among the common lesions are scarring processes related to cortical damage and glomerular loss, as can be been in hypertension, diabetes, glomerulonephritis, and other disorders. Obstructive realities also may be pertinent to the cause of death, as renal outflow obstruction is a ready cause of renal decompensation and failure. These tissues are also involved by a variety of tumours, both primary and secondary. Metastatic disease is common in the kidney, but primary kidney cancers can be widely disseminated and may be surprise findings at autopsy examination.

Pathology of the female genital tract principally reflects tumours, although sepsis is often encountered in examinations. The female genital tissues to be covered include vulval and vaginal tissues. The uterine corpus and cervix will be considered with the tubes and ovaries.

The breast tissues also are discussed in this chapter, reflecting their role in pregnancy and reproduction. The breast tissues are very rarely a significant factor in autopsies in terms of cause of death, aside from breast tumours with disseminated disease.

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Specialist examination protocols and techniques are required for maternal deaths, which inevitably demand a wide-ranging investigative protocol.

The male external genitalia (penis, scrotum/testes) and prostate are considered in turn. Aside from incidental findings of sexually transmitted disease, the male genital tract pathology at autopsy largely revolves around tumours, which can be locally aggressive or disseminated. Their impact will depend upon the sites involved.

## Kidney and Ureter

The kidneys exist as a paired group of glands with an average weight of 100–150 g. They are found in the retroperitoneal, paravertebral tissues. Looking at the Rokitansky block dissection, they are often examined early in the autopsy protocol and may be removed intact with the ureter, bladder, and associated pelvic tissues, or they may be taken separately. Traditionally, if they are taken separately, the left side ureter is left longer than the right to allow identification of sidedness.

The initial dissection and kidney tissues (Figs. 6.1, 6.2, 6.3, 6.4, 6.5, 6.6, 6.7, 6.8, 6.9, 6.10, 6.11, 6.12, 6.13, 6.14, 6.15, 6.16, 6.17, 6.18, 6.19, 6.20, 6.21, 6.22, 6.23, 6.24, and 6.25) can be involved by a variety of processes, which principally fall into three groups. First, degenerative and scarring processes are common in kidney disease when seen in the adult autopsy. The prime pathology is that of renal scarring, characterised by a roughening of the cortical tissues and thinning of the cortical parenchyma. Whilst there is some value in measuring the cortical thickness, the cortex will vary according to the body-frame of the individual. In general, a crude gauge is that the cortical thickness should be the same as that of the medulla (on average), and thinning of the cortex in relation to the medullary tissues is a marker for fibrosing processes and cortical tissue loss. Common pathologies underlying this process include hypertension, glomerulopathies (particularly immune complex glomerulonephritis), diabetes, and various tubular injuries (infections, drugs).



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These processes often are not the prime cause of death but are contributory factors. Indeed, this pattern of renal disease is often associated with hypertension, which may be the driving force behind the cause of death.

The second group of disorders to characteristically affect the kidney is infection. Acute infections, whether blood-derived or (more commonly) ascending (*i.e.*, along the ureter and pelvis) can rapidly debilitate an individual and potentially cause death. Such infections are particularly a problem for the elderly, and urinary samples should be considered if one suspects an unusual septic process. It should be remember that tuberculosis also can affect the kidneys, so some caution should be used when examining these tissues. Acute-on-chronic sepsis or previous sepsis can produce significant renal scarring and loss of function, which may be a driver for renal impairment (chronic kidney disease).

The third group is renal tumours, which are not uncommon in autopsy practice. Primary renal cancers are mainly renal cell carcinoma, which is often an incidental or unexpected finding. Other tumours include transitional cell carcinomas of the renal pelvis, but it is metastatic cancer that is often seen in cases of disseminated carcinomatosis.

Obstructions of the upper urinary tract do occur, with both ureteric and pelvic dilatation. The source of such obstruction should be sought when the features of hydronephrosis are seen at initial examination. In such cases, it is important not to simply remove the kidney without due dissection down the ureteric pathway towards the bladder. Obstructions can be mural, in terms of the junction of the pelvis and the ureter, or they can occur at the ureter/bladder interface. Stones and tumours also can provide obstructive pathologies. Sepsis often occurs in association with such conditions.

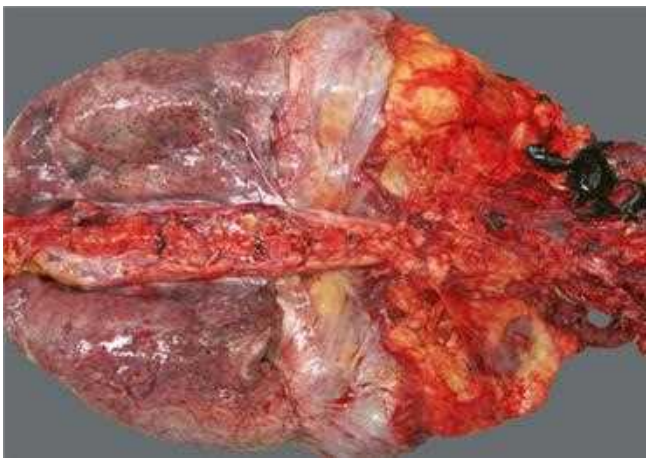

**Fig. 6.1** The posterior aspect of the eviscerated tissue provides the start point for examination of the genitourinary system, focusing on the kidneys. The initial examination is complicated because the kidneys, the ureters, and other tissues are often partially encased within fatty parenchyma, which must be sequentially and carefully incised to examine the tissues

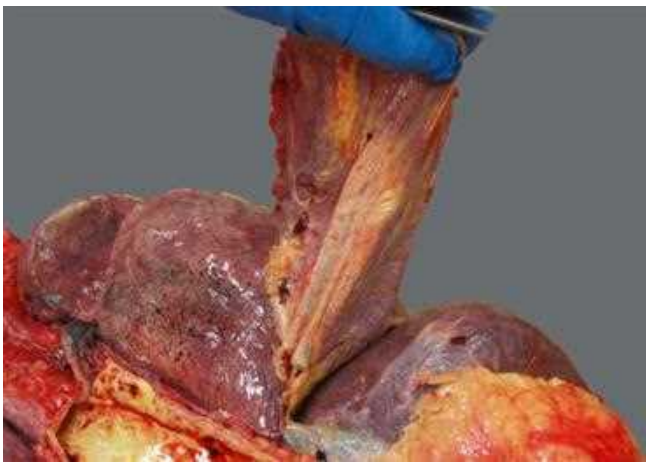

**Fig. 6.2** The diaphragm tissues are removed, folding this muscular parenchyma up and away from the liver and upper abdominal tissues on both sides

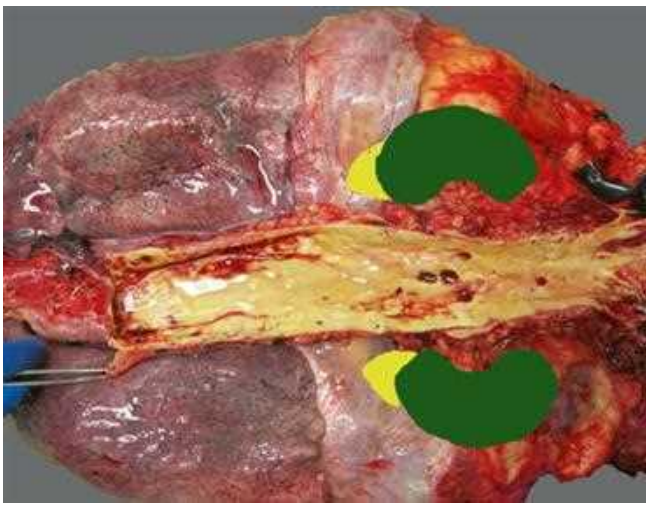

**Fig. 6.3** When one is faced with a fatty retroperitoneum, it pays to think about the likely position of the kidneys. The likely position of the kidneys (*green*) is marked on the overlay map, with the adrenals also marked (*yellow*)

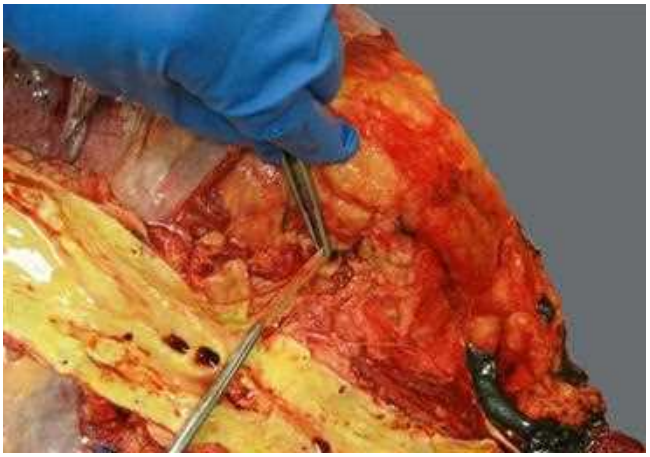

**Fig. 6.4** To assist with identifying the kidneys, one can trace the renal artery on both sides, as it will point towards the kidney. Here the aortic tissue has been opened and any residual blood content has been removed. The renal arteries should be explored on both sides to check for stenosis as part of the general autopsy. If one frees and folds the aorta down towards the pelvic tissues, then the same can be performed for the renal veins

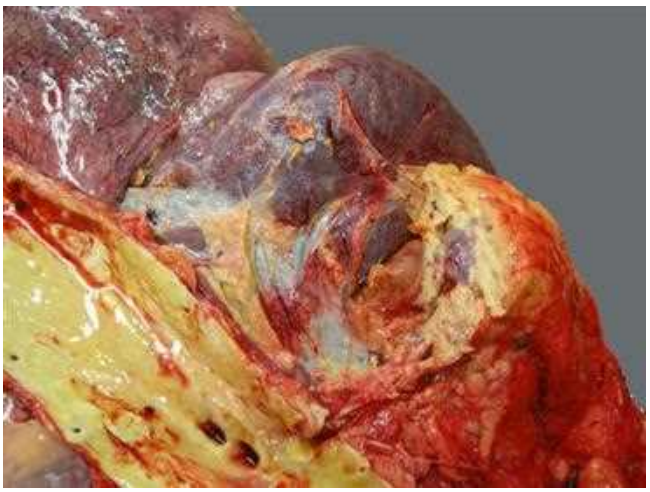

**Fig. 6.5** This view of the upper retroperitoneal tissues has had the fatty tissue and adrenal parenchyma removed. The *top part* of the kidney is now open for inspection, but the remainder of the kidney is still hidden from view, encased within the perinephric fat and fascia

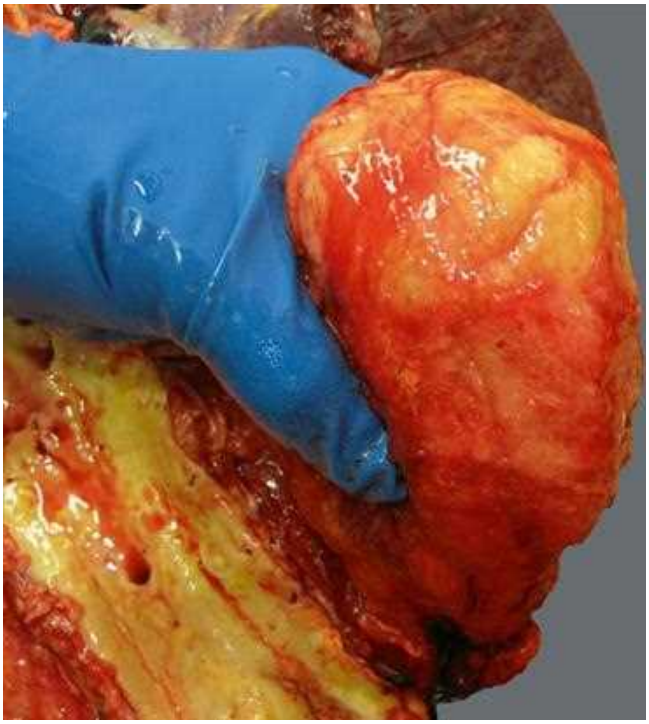

**Fig. 6.6** Grasping the soft tissue and fat around the kidney, one holds the outer (antihilar) aspect upwards, perpendicular to the cutting board/surface

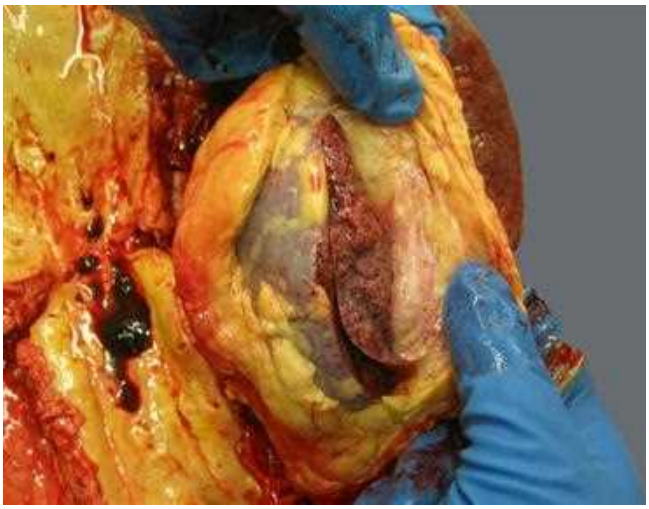

**Fig. 6.7** A knife (PM40 type) has been used to incise a few millimeters into the kidney from the antihilar aspect. The kidney fat and capsule are now pulled from the surface of the kidney by grasping the capsule with toothed forceps. The capsule and fatty tissue can be pulled away from the kidney parenchyma in a process that will facilitate examination of the kidney and also its removal. Here, the surface of the kidney is noted to be granular and pitted, in keeping with chronic kidney disease and hypertension. It does not matter at this stage whether the cut has gone deeper into the kidney, as this cut will be extended more deeply later

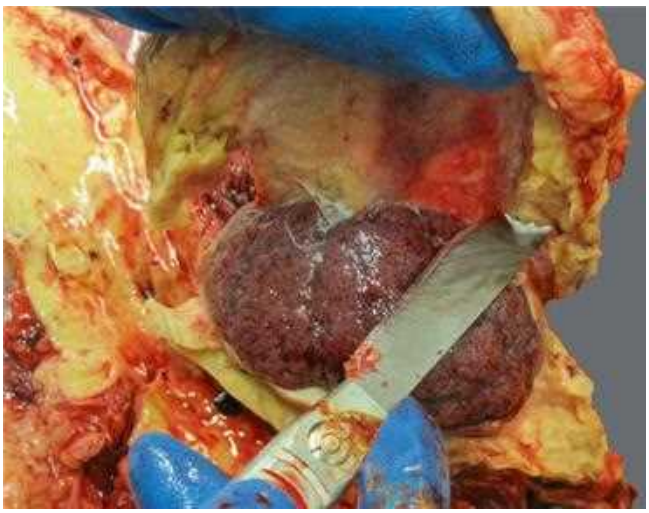

**Fig. 6.8** The kidney is now laid flat. Holding the fatty tissue (peripheral to the kidney) up at 90° away from the interface of the kidney surface and hilar blood vessels, ureter, and soft tissue, one may now resect the plate of perinephric fat and the capsule, being confident that no kidney damage will occur and simplifying the examination of the tissues and subsequent dissection

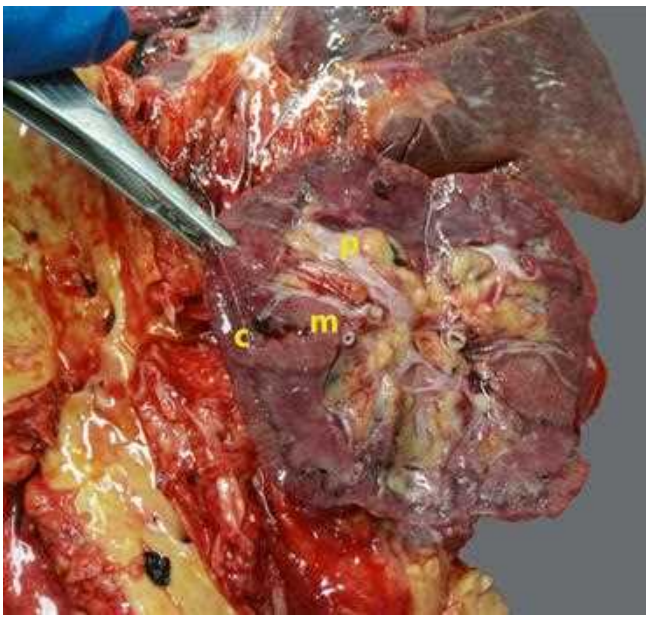


**Fig. 6.10** The cortex (*c*), medulla (*m*) and renal pelvis (*p*) are now displayed. Note that the tissues are still attached to the vasculature and ureter. The cortex and medulla are well demarcated from one another

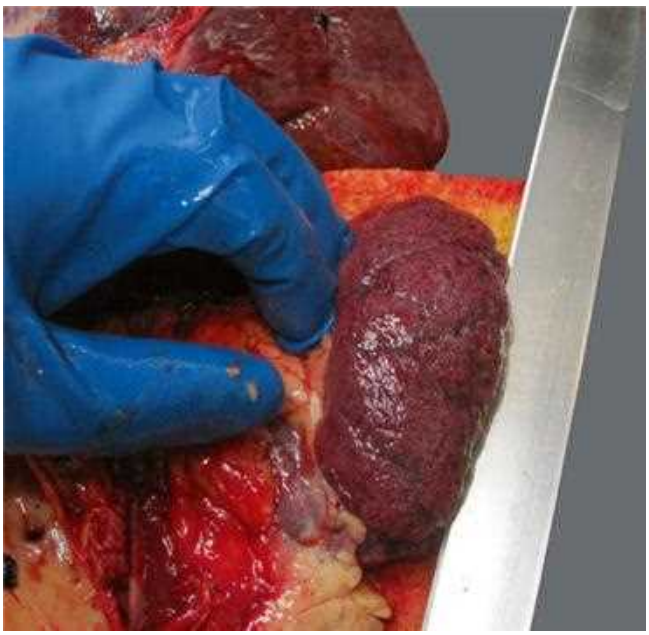

**Fig. 6.9** Once it is dissected free of the fat and fascia/capsule, the kidney can now been seen around all the cortical external surface. The granularity and scarring are noted. The next step is to lay the kidney on a sponge or other support and extend the initial cut made into the outer cortical plane. A long knife slices towards (but not through) the hilum, thereby opening into the pelvis. The cut surface of the renal medulla parenchyma and the cortex will now be easily seen

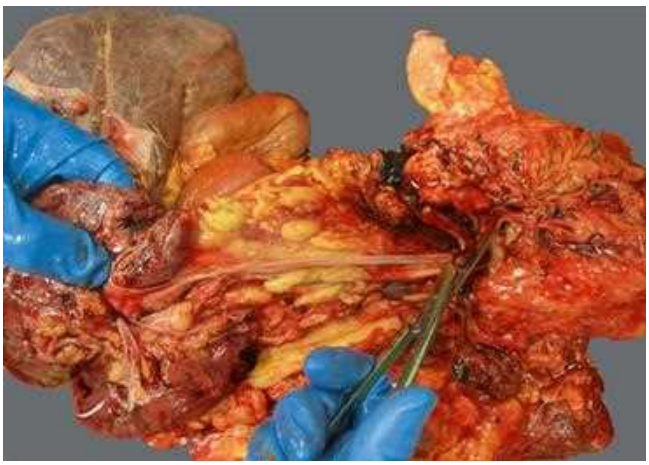

**Fig. 6.11** By passing one blade of the scissors into the pelvis, one can dissect steadily down along the ureter towards the bladder. This dissection should not be rushed, to avoid tearing the ureter. This procedure keeps the ureter, kidney, and vascular tissues still in one piece

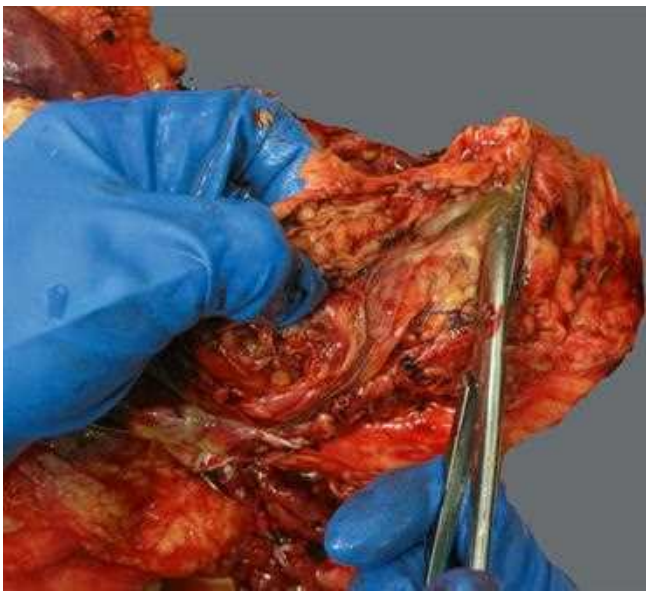

**Fig. 6.12** There is a small gain to be made by rotating the pelvic tissues one quarter turn towards the contralateral side, which keeps the ureter and bladder alignment in a straight line. Any cut through the ureter/bladder interface will thus be made easier. The blade of the scissors can now be passed into the bladder, with the ureter orifice being available for study

**Fig. 6.13** The normal kidney has a smooth cortical surface

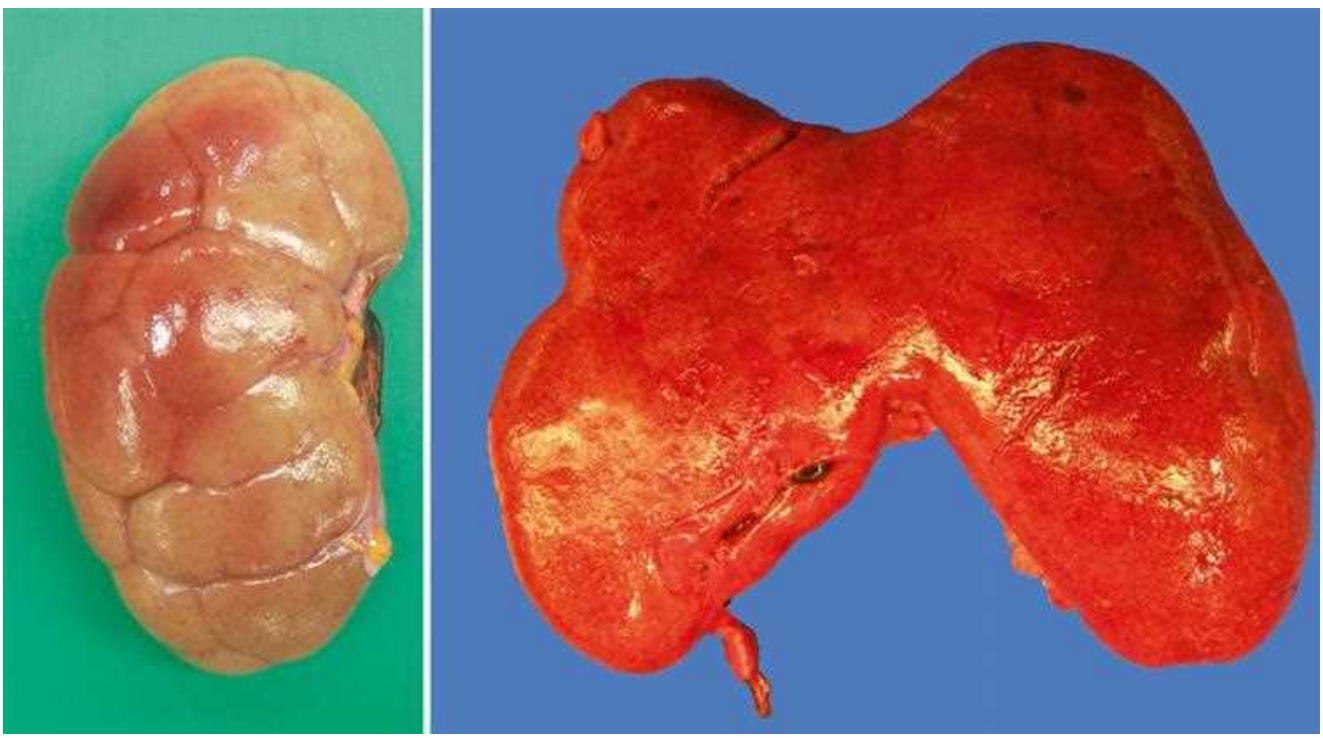

**Fig. 6.14** *Left side*, On occasion, some lobulation is evident, akin to that of fetal development. This is of no consequence, but the cortical surface should be smooth and shiny, as in the normal kidney. *Right side*, A congenital variation with normal renal function is the 'horseshoe' kidney, where a central/midline fusion of renal elements occurs and a single kidney is formed in utero

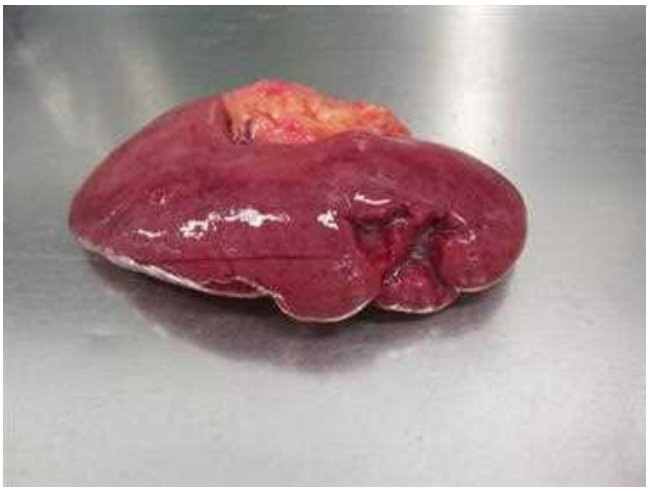

**Fig. 6.15** This kidney, dissected free from the vasculature and ureter, has a partly normal surface, but there is a focus of depressed scarring and distortion, in keeping with previous pyelonephritis and ascending infection. Note the thin cut evident on the outer cortical plane, which is in keeping with the dissection described above

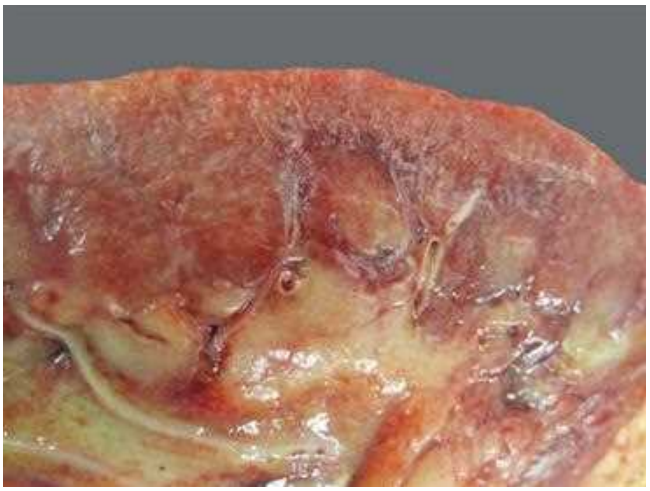

**Fig. 6.17** The renal parenchyma shows these medullary tissues to be slightly thicker than the cortex. A moderate amount of fat is present in the peripelvic compartment. The renal pelvis is seen in part, with some congestion of the surface tissues. The corticomedullary junction in this case is somewhat blurred, mapping to a period of hypoxic injury in this patient, who was treated on the intensive care unit

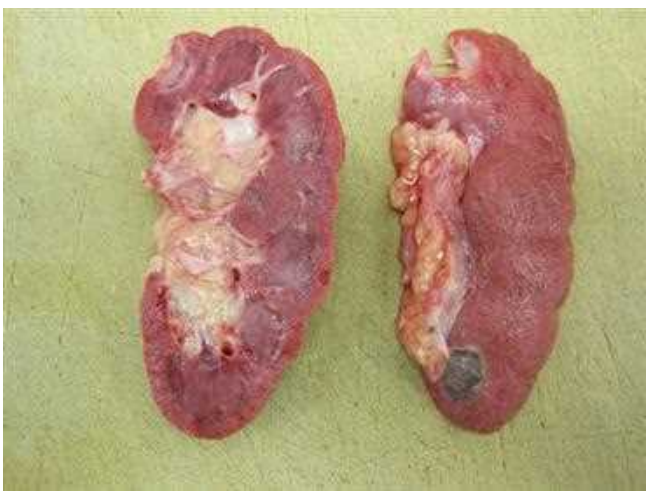

**Fig. 6.16** Many kidneys seen in autopsy show granular surfaces and some reduction in the cortex thickness. As a general rule, the cortex and medullary parenchyma should be roughly equal in terms of thickness. Diminished cortical thickness and granularity may indicate a background of hypertension, although diabetes and glomerulopathies may also be pertinent. An incidental cyst is noted

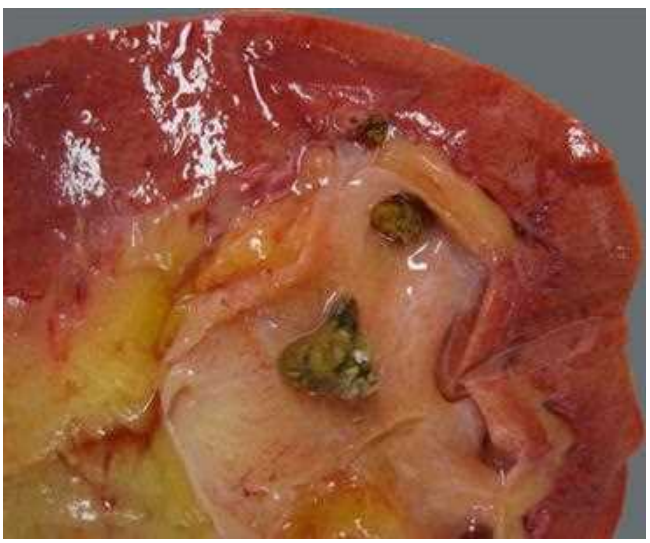

**Fig. 6.18** Renal stones are often found as incidental findings at autopsy. This somewhat fragmentary stone was present in the pelvis and calyx, and broke during dissection. The stones can be associated with sepsis, obstruction, inflammation, and ulceration, as well as neoplasia, but in many cases they cause no significant abnormality and are surprise findings at autopsy

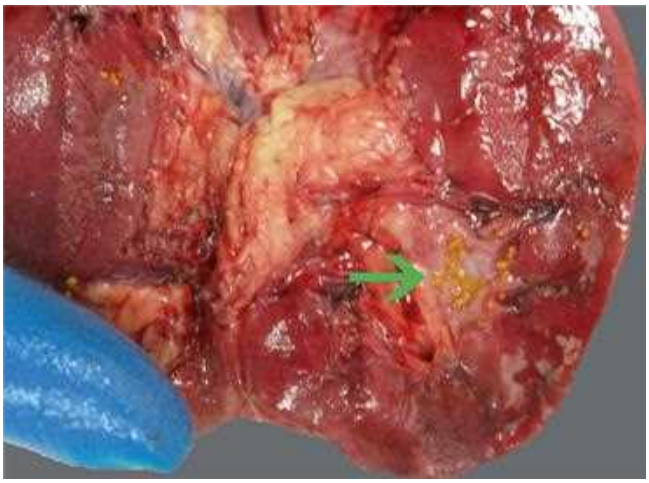

**Fig. 6.19** Small kidney stones can be seen in the renal pelvic tissues (*small*, *yellow*, gravel-like tissues, *arrow*). These stones are more prone to pass into the ureter and cause obstruction. When they are found, careful inspection of the ureters is recommended

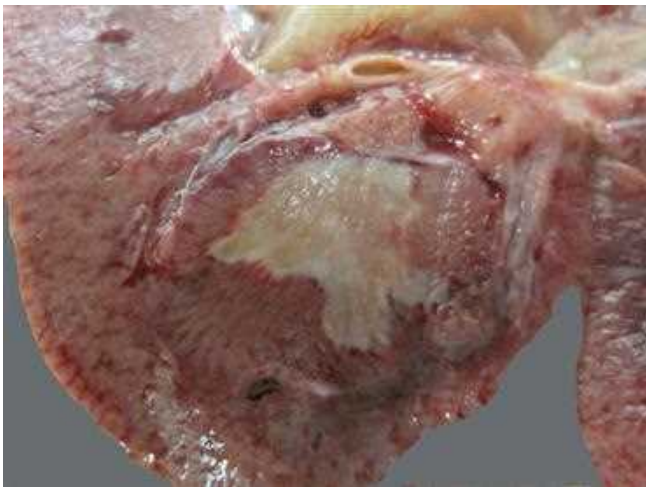

**Fig. 6.21** Renal papillary necrosis is commonly seen in diabetics. The necrotic tissue often has a slight hyperaemic boundary with the normal parenchyma. One should always consider local sepsis as a compounding factor, particularly in diabetics

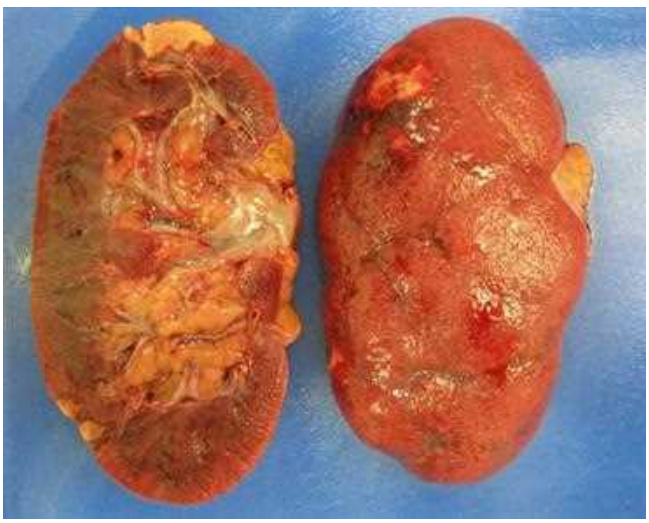

**Fig. 6.20** Systemic embolic disease from the cardiac valves can embolise into the kidney. This will produce peripheral, cortical wedge-shaped infarcts in the kidney parenchyma. Several are seen in this opened kidney, with lesions extending deeper into the renal parenchyma. Histology is recommended to exclude tumour or septic emboli

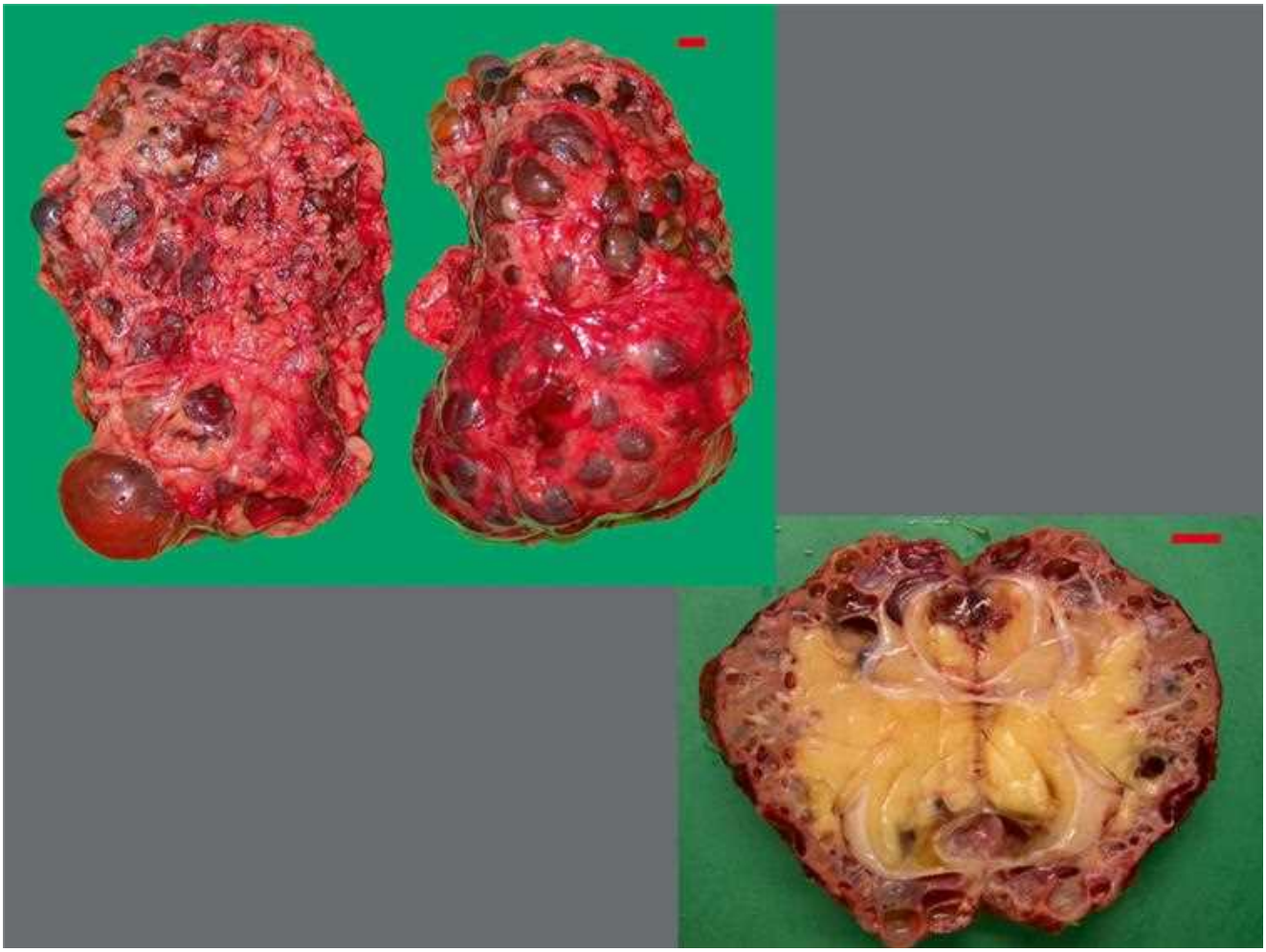

**Fig. 6.22** Cystic change is common in adult kidneys seen at autopsy, usually as a single, simple cyst (such as the one in Fig. 6.16) with no sinister potential. On the *upper left* is a case of adult polycystic kidney disease (APCKD), a nonneoplastic degenerative process that is sometimes responsible for kidneys weighing 1.5 kg. The cysts may enlarge as one progresses into adult life, with a risk of haemorrhage and/or sepsis, but the main problem is hypertensive disease and its consequences, as well as the realities of renal failure. By contrast, acquired polycystic kidney disease (*lower right*) often follows renal failure and dialysis. It is associated with a risk of renal cell carcinoma (Bar marker, 10 mm)

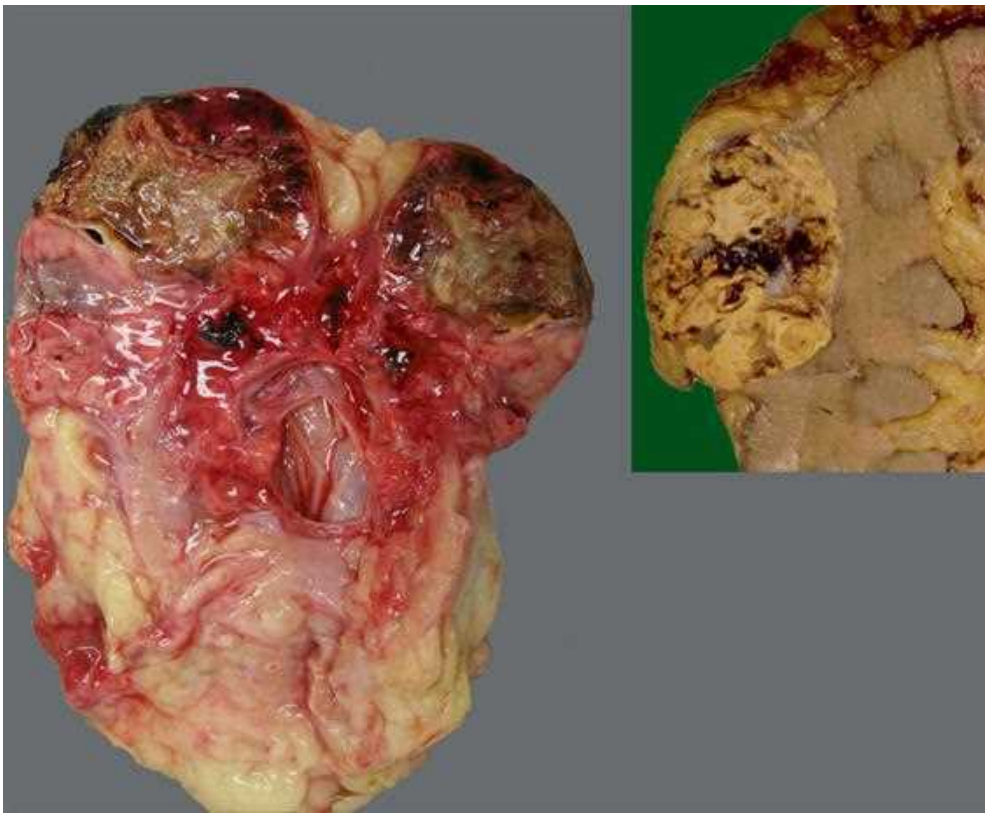

**Fig. 6.23** Primary kidney cancer is not uncommon at autopsy. The commonest type, renal cell carcinoma, may present as an incidental mass or as advanced and disseminated disease. The classic format (in the fixed sample, *upper right*) shows a *yellow* cut surface. Often at autopsy (*left side*), the tumours appear haemorrhagic and partly necrotic

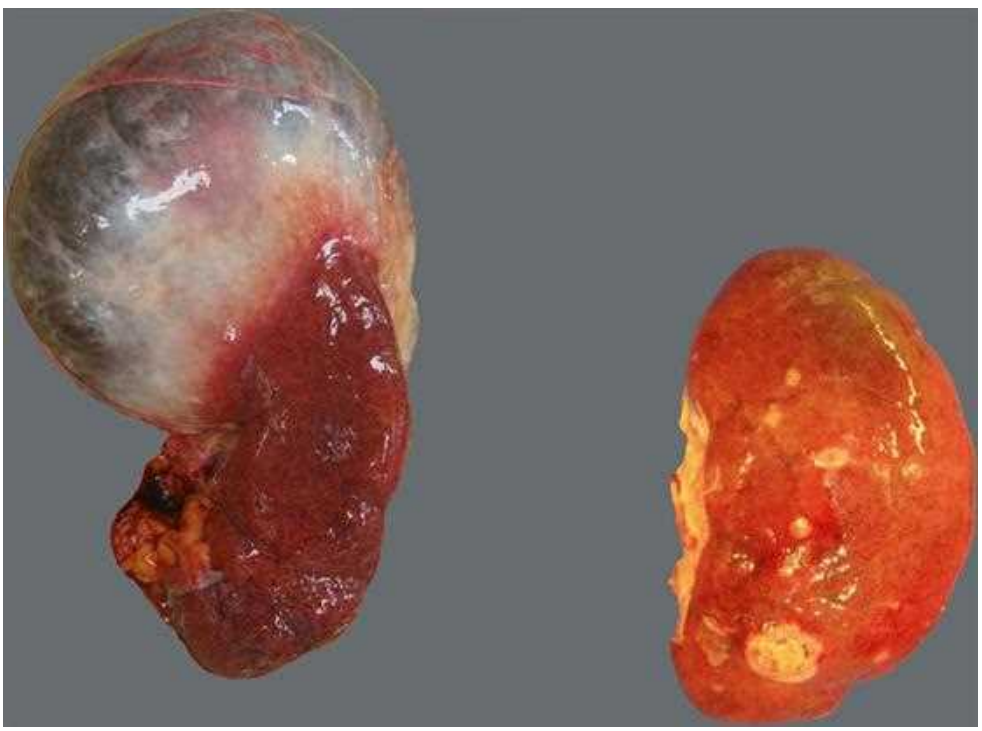

**Fig. 6.24** Another variant of renal cell carcinoma is seen in the form of cystic change (*left side*). The turbid and haemorrhagic content and minor yellow/brown lining was the clue that this was a carcinoma, which was confirmed by histology. By contrast, on the *right side* is a kidney with multifocal metastatic tumours from a primary lung cancer

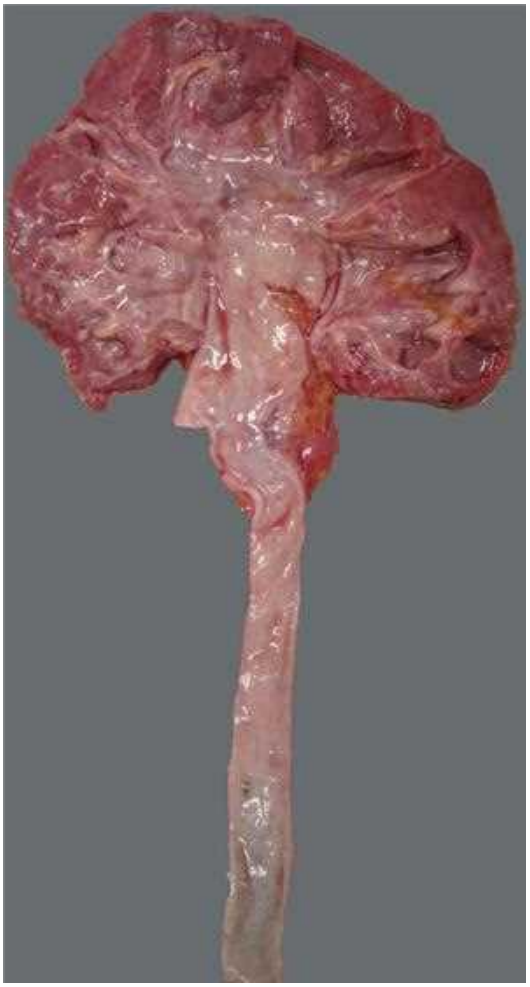

**Fig. 6.25** Ureteric obstruction is seen with dilated ureter and a dilated pelvi-calyceal system. The source of the obstruction should be considered, with extension of the dissection into the lower ureteric parenchyma and down to the bladder

## Bladder

The bladder (Figs. 6.26, 6.27, 6.28, 6.29, 6.30, 6.31, 6.32, 6.33, 6.34, 6.35, 6.36, and 6.37) is of particular interest in autopsy practice, mainly as a source of urine for toxicology testing (see Chap. 10). It should always be inspected internally to assess for inflammation and infections as well as for neoplasia. Urinary sepsis, in the form of cystitis, is not uncommon among adults, particularly those with catheters.

One should remember that post-mortem shedding of the urothelial surface occurs quickly, so some turbidity of urinary content does not automatically imply sepsis. Sampling of urine for cytological assay and to look for infective agents may be relevant, but bacterial overgrowth in the post-mortem setting does create difficulty.

Bladder neoplasms are principally transitional cell carcinoma in nature and may be associated with features of urinary stasis and/or calculus disease. In such cases, careful consideration of the pelvic soft tissues should be undertaken, looking for involved lymph nodes. Metastatic tumour from the bladder often also involves bony and lung tissues.

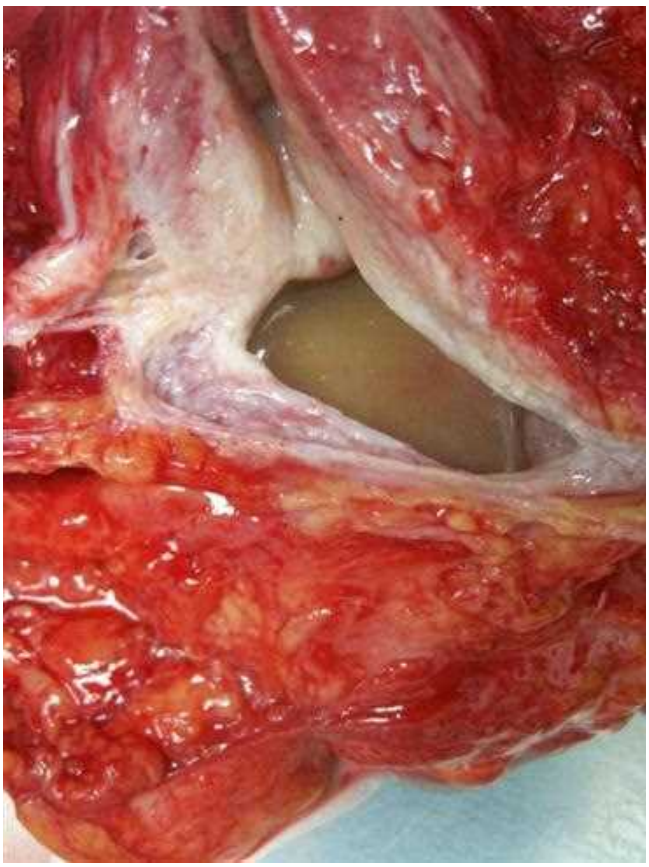

**Fig. 6.26** As one examines the pelvic tissues, the bladder is often opened from the roof. The urine may appear turbid at autopsy, but that turbidity is likely to reflect sloughing tissues from the urothelial surface mixing with the clear urine

**Fig. 6.27** Urine from the bladder often contains sloughed epithelial cells, as is evident in this sample spread onto a stainless steel surface. This material, whilst opaque in the bladder, should not be regarded as urinary sepsis without other supportive factors. It should also be remembered that culture of urine at post-mortem is often a less than ideal exercise, as post-mortem colonization is common

**Fig. 6.28** Urine is generally clear at autopsy, particularly in the recently deceased. Some urine ideally should be retained in suspicious deaths or in those with likely drug abuse. The amount of sample required may be quite large (when considering steroids); one should check with the toxicology department regarding how large a urine sample is required for unusual drugs

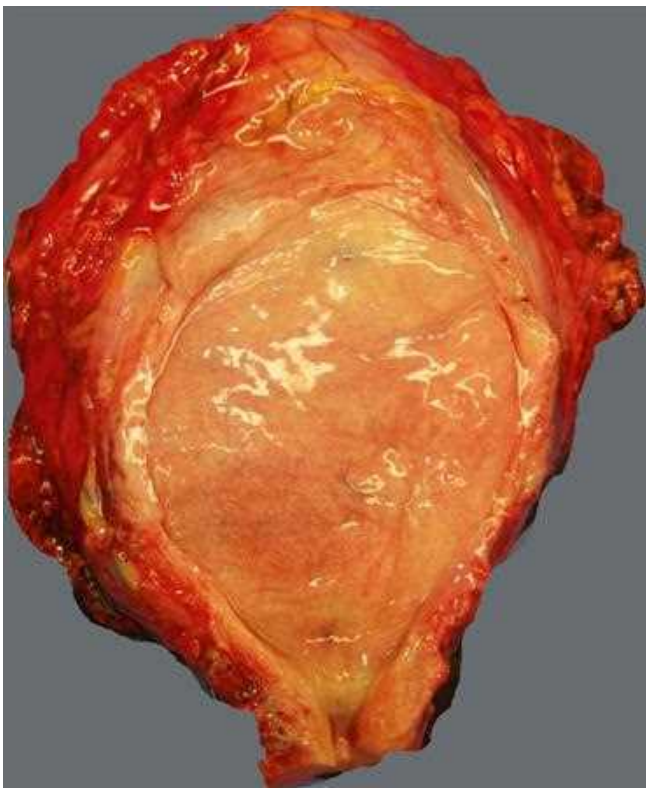

**Fig. 6.29** The normal bladder has a smooth, beige surface with only very mild vascular marking. The bladder wall should be soft and pliable

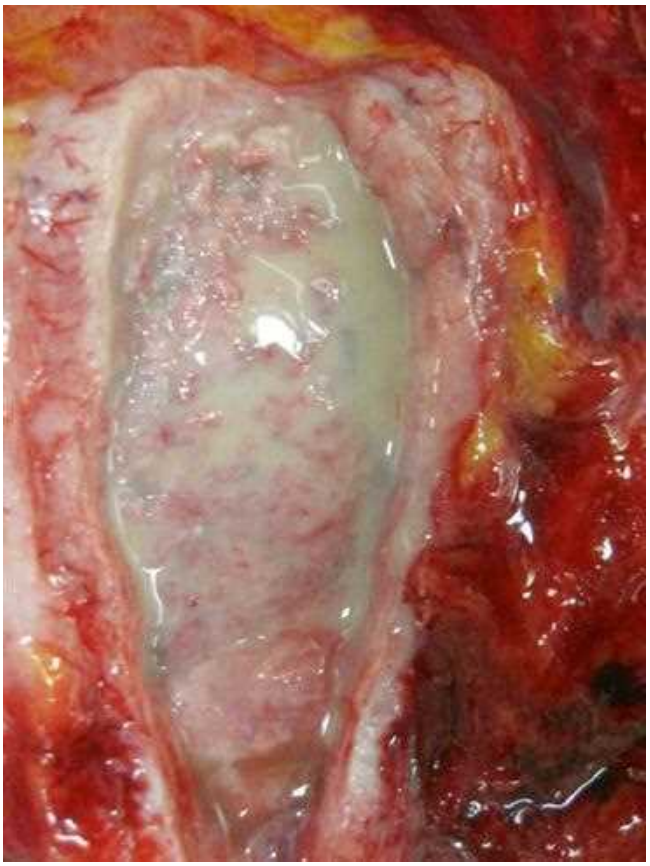

**Fig. 6.30** This bladder shows a somewhat congested and discolored surface. The *light green* and opaque material on the surface suggests active infection, but one must be aware of post-mortem overgrowth as a potentially complicating factor when considering microbiology sampling

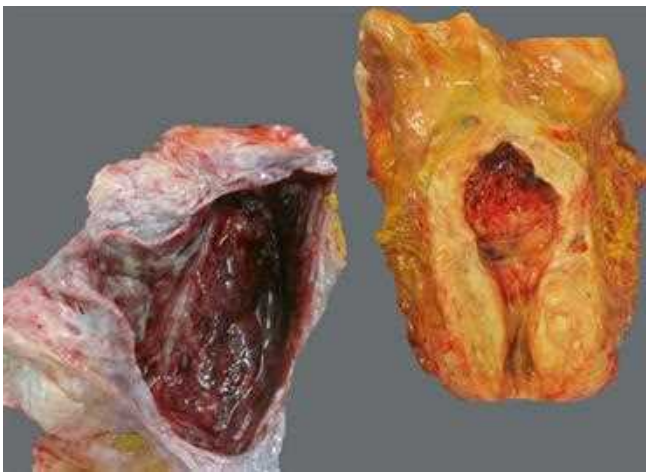

**Fig. 6.31** Two views of inflamed bladders are seen. On the *left* is frank bladder sepsis, with an inflamed surface lining and some grey-green purulent material. Variable ulceration of the bladder lining may be present, and the bladder itself appears intensely haemorrhagic and congested. On the *right*, a patient with a chronic catheter showed evident cystitis affecting the entirety of the bladder surface, with associated mural thickening and fibrosis. In such circumstances, one may also need to take samples of the bladder lining to exclude dysplasia and neoplasia


**Fig. 6.32** Urinary stones vary in shape and size. They can be small and gravel-like (as seen in Fig. 6.19), but others can be quite large, with rounded or irregular surface profiles

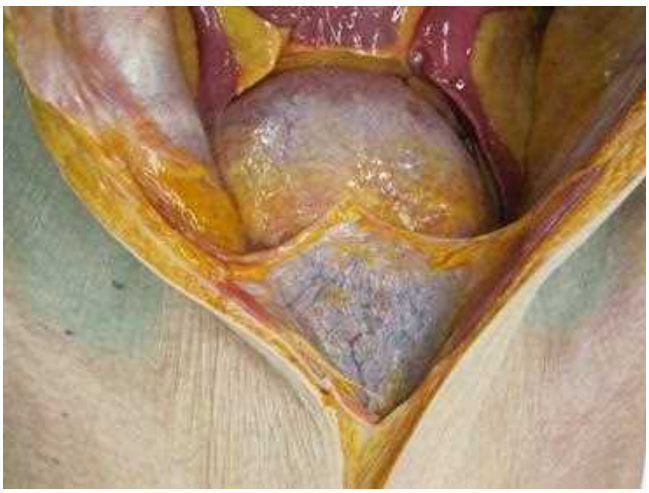

**Fig. 6.33** A distended bladder seen when opening the abdomen may reflect significant obstruction of urine outflow, particularly in males. It may be prudent to deflate the bladder, measuring the total urine volume, before proceeding to further tissue evisceration. As a rough guide, the weight of the urine is equivalent to the volume. Due diligence should be exercised to consider the cause of the urinary obstruction by means of diligent dissection and histology

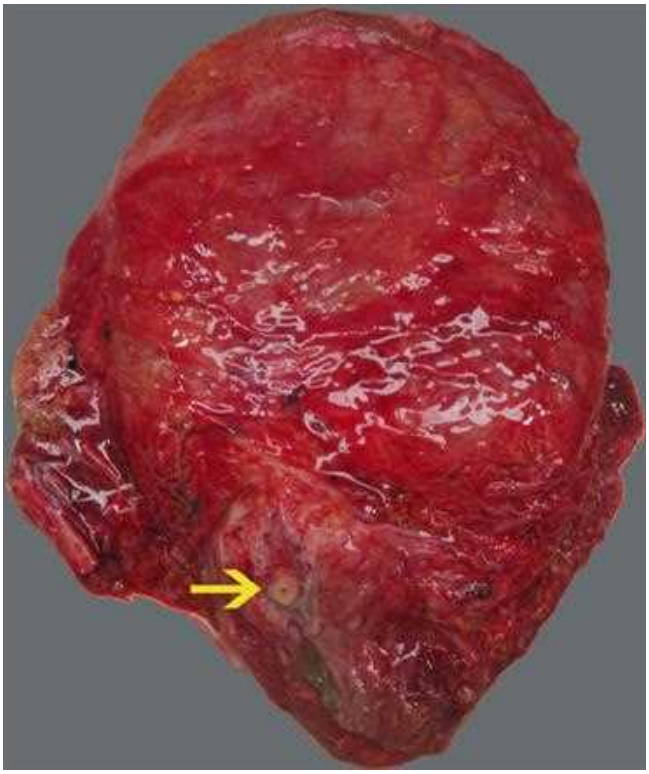

**Fig. 6.34** A full bladder can be resected intact, although dissection of the tissue is more complicated if it is full of urine. The *arrow* indicates the urethra at the prostate outflow

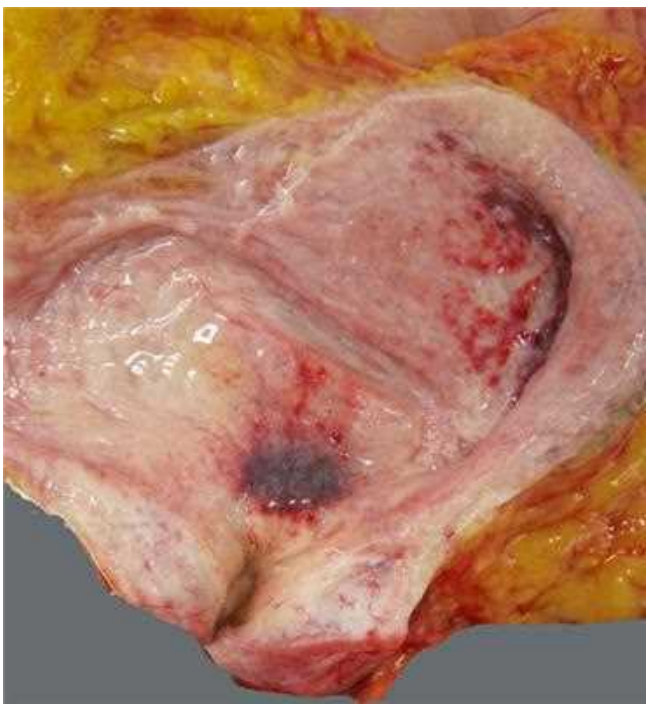

**Fig. 6.35** The bladder in men is normally resected together with the prostate. Here, the prostatic outflow tract and urethra are seen in association with a focally inflamed bladder, which has a thickened wall. This case of catheterisation clearly had some degree of associated bladder irritation

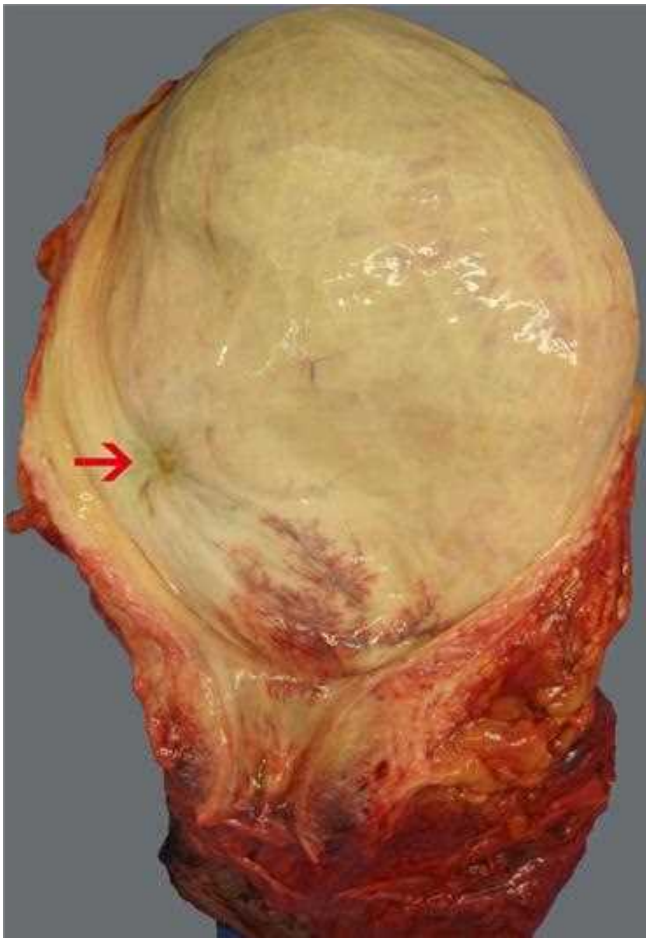

**Fig. 6.36** This case of treated bladder cancer shows an area of telangiectasia towards the base of the bladder/trigone. Towards the lower right-hand side (*arrow*) is an irregular yellow/grey nubbin of tissue, representing a treated bladder carcinoma. This patient had been treated with external beam radiation for bladder cancer

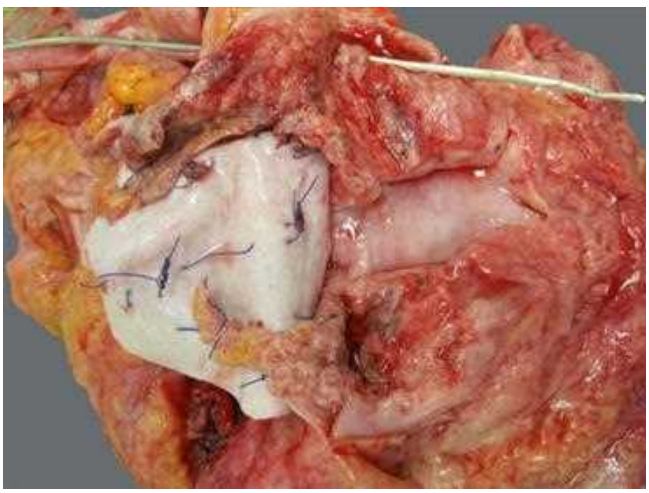

**Fig. 6.37** Bladder tissue can be augmented by small-intestine and other patches. In examining a case shortly after such augmentation procedures, one should pay attention to local sepsis, dehiscence, and local infective pelvic complications

## Prostate

The prostate gland at autopsy usually is considered from two main perspectives. First, an assessment of size and nodularity is often relevant in terms of benign nodular hyperplasia, which is the normal reality for males with increasing age. This 'benign' process is linked with urinary outflow obstruction from the bladder, with a potential risk of backpressure onto the kidneys, as well as sepsis. It may also provide a setting for the formation of stones. The maximal cross-section of the prostate is often measured in terms of diameter; but, alternatively, at the end of the autopsy, the prostate can be dissected free and actually weighed.

The prostate is a common site of malignancy, almost entirely prostatic adenocarcinoma. Whilst these tumors may be large, it is the disseminated disease that is most likely to be relevant to the cause of death.

The seminal vesicles rarely have significant pathology in autopsy examination and are rarely sought specifically. Likewise, the urethra and its passage into the penis is not usually a source of major pathology. Unless there is antemortem evidence of significant disease at this site, detailed dissection of this site is rarely needed.

## Male Genital Tissues

It is rare for the spermatic cord to be involved in significant autopsy pathology, although various degrees of lipomatous/fatty hypertrophy are often seen in the elderly. The main pathology involving the cord often relates to local hernia with compression of the cord elements and subsequent atrophy of testicular tissue.

The testis, by contrast, may be involved by tumours, with the commonest being germ cell neoplasms. Metastatic neoplasia (mostly from the prostate) is possible, but it is rarely sought or identified.

The remaining external genital male tissues comprise the penis and scrotum. The external genitalia (Figs. 6.38, 6.39, 6.40, 6.41, 6.42, 6.43, 6.44, 6.45, 6.46, 6.47, 6.48, 6.49, and 6.50) are commonly ignored during autopsy practice, but some inspection is required, even in the elderly. These structures are mostly uninvolved by pathology at autopsy, apart from occasional incidental sexually transmitted disease (*e.g.*, viral warts, condylomata, ulcers). Nevertheless, there may be variable degrees of sepsis in relation to genital studs, rings, or implants.

**Fig. 6.38** The normal male genitalia are seen with the pubic hair, penis, and scrotum (containing two testes)

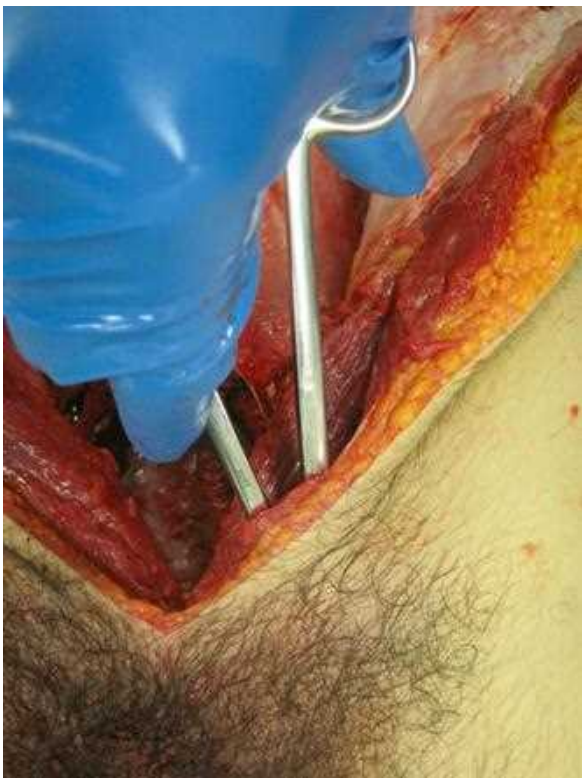

**Fig. 6.39** Rather than directly dissecting through the scrotum, examination of the testes requires a dissection plane to be opened at the pelvic rim, running into the superficial soft tissues outside the pelvis and down towards the scrotum

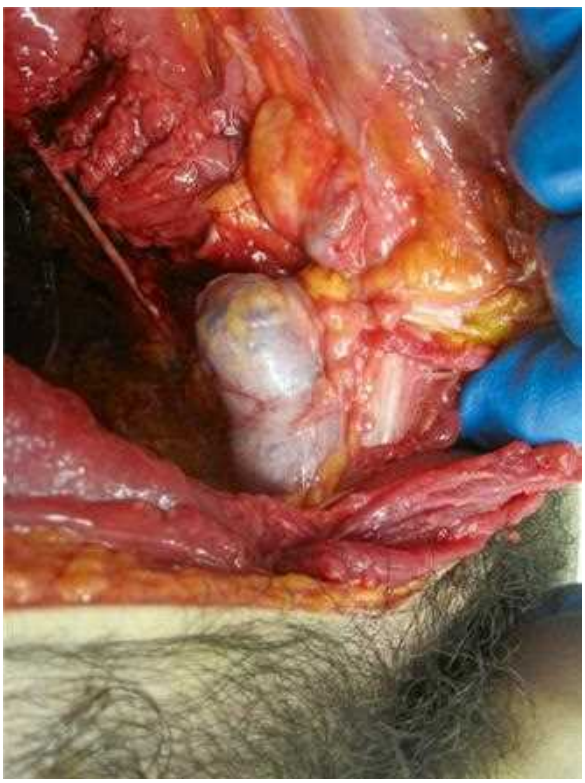

**Fig. 6.40** Having bluntly dissected down alongside the cord, the testis can be mobilised. By pushing the testis upwards (from outside of the scrotum), it is possible to push the testis through the plane of dissection made and to remove it at the pelvic brim

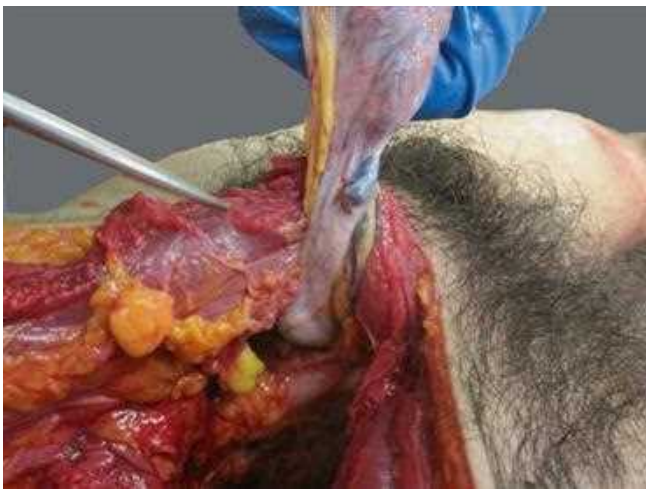

**Fig. 6.41** The testes and cord are pulled free and upwards at 90°. The testis is seen with the cord and local soft tissues adjacent

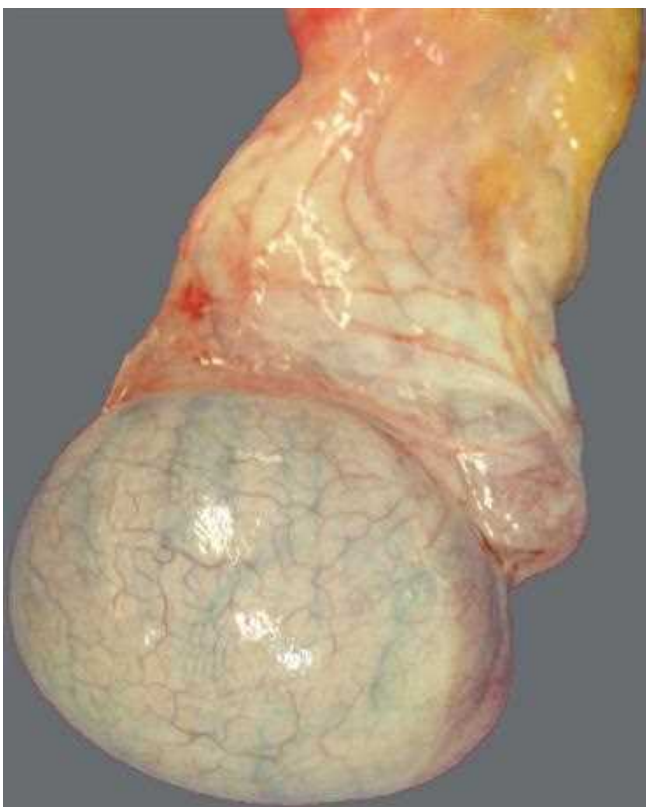

**Fig. 6.42** The testis has a smooth and shiny external surface. The normal testis should be soft, without nodularity or fibrosis

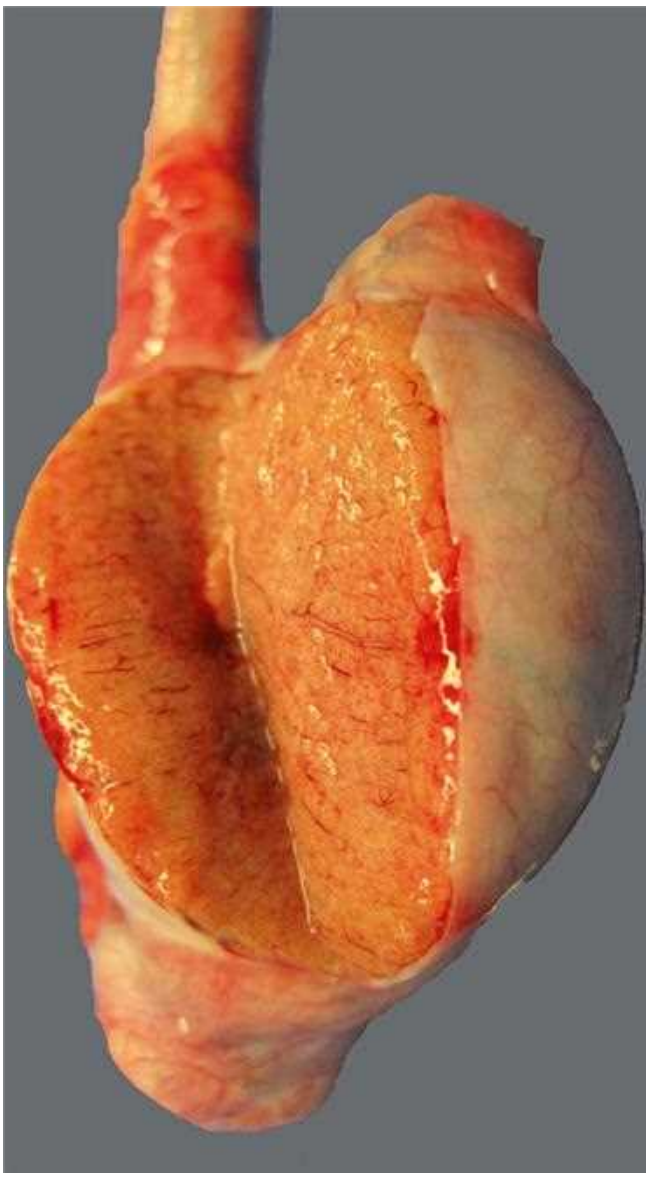

**Fig. 6.43** The testis can be sliced open, revealing a congested parenchyma, which often has a yellow/orange colour

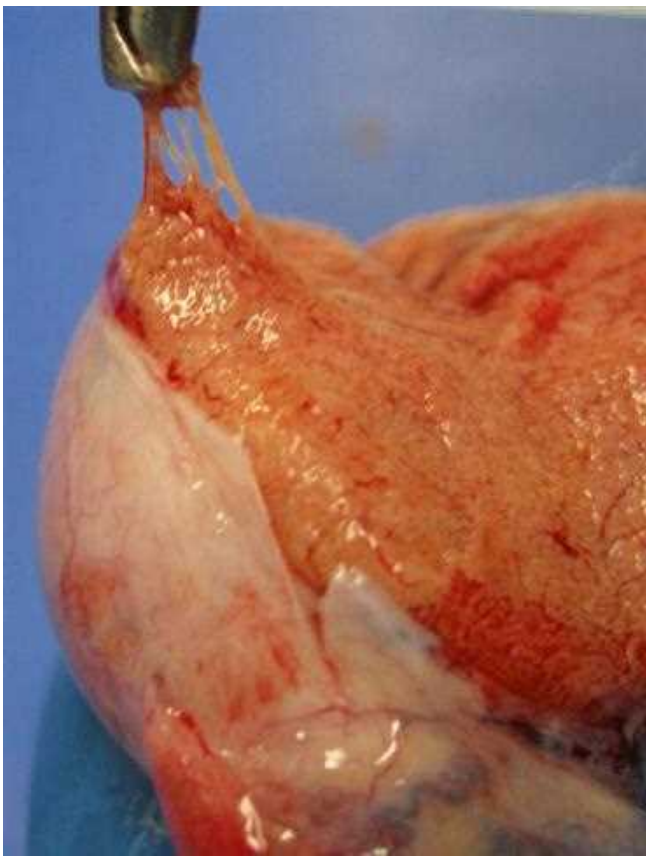

**Fig. 6.44** The individual tubules can be grasped with toothed forceps and pulled upwards to demonstrate the highly folding quality of the testicular tissues

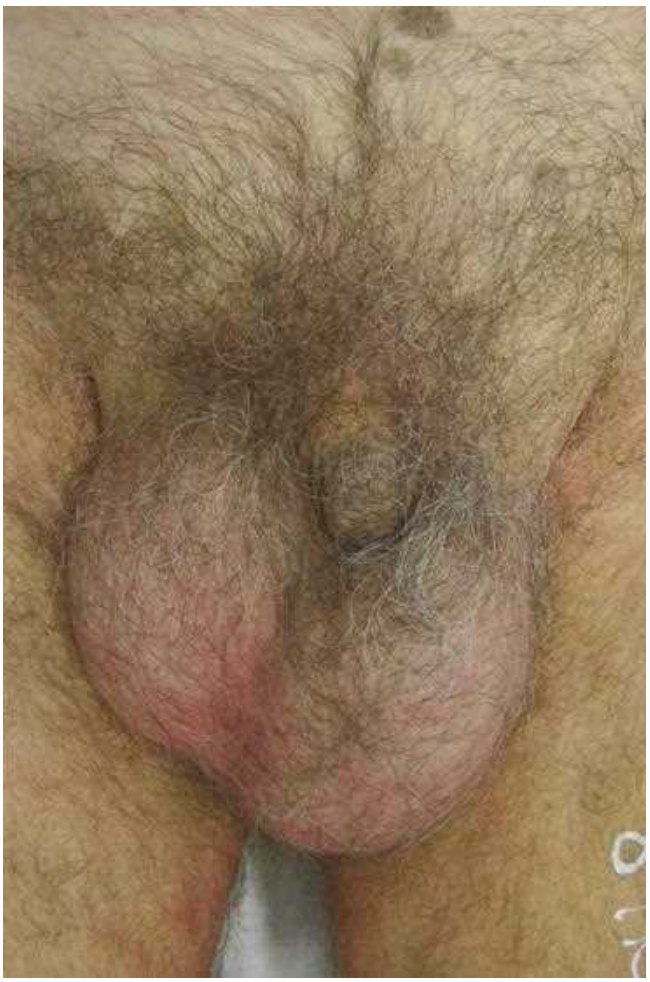

**Fig. 6.45** Variation in size of the two sides of the scrotum may point towards testicular tumours or, more commonly, fluid collections around the testes

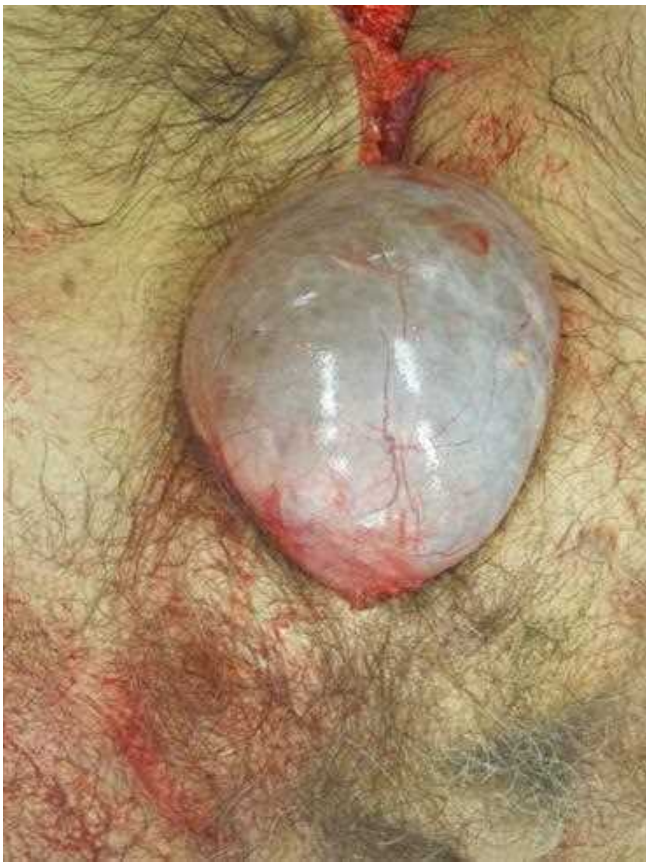

**Fig. 6.46** When extracting the testis, the sac may be removed at the same time if there is a significant fluid collection

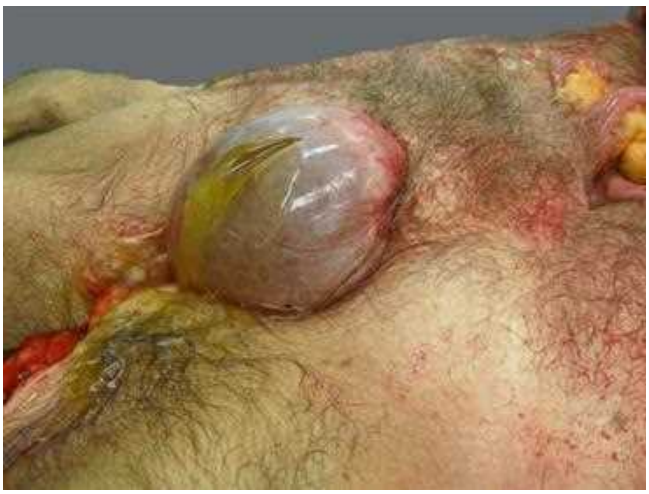

**Fig. 6.47** Looking from the side, the sac is then incised to check the nature and volume of the fluid. In this case, clear *yellow* fluid is allowed to flow from the cut surface

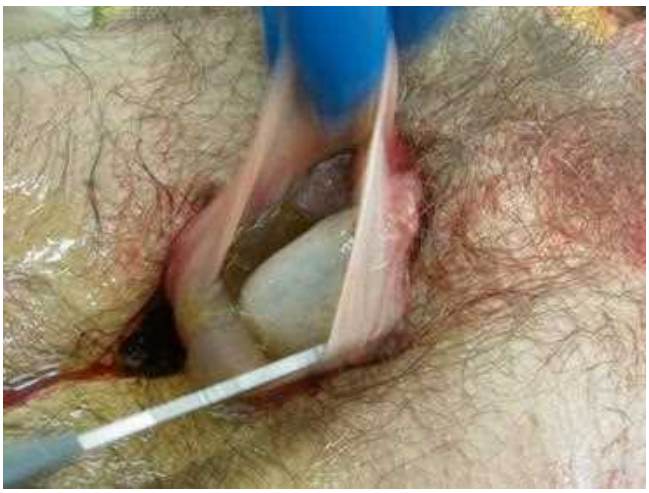

**Fig. 6.48** Inside the hydrocele sac is a testis with normal shape and architecture

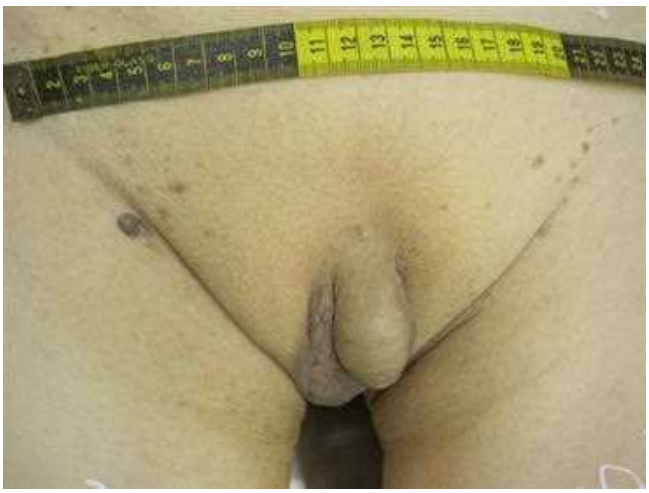

**Fig. 6.50** Atrophy of the penis and loss of secondary sexual hair pattern is characteristic of patients who have been treated with orchidectomy for prostate carcinoma. Such external findings should prompt consideration during the autopsy of disseminated prostate cancer

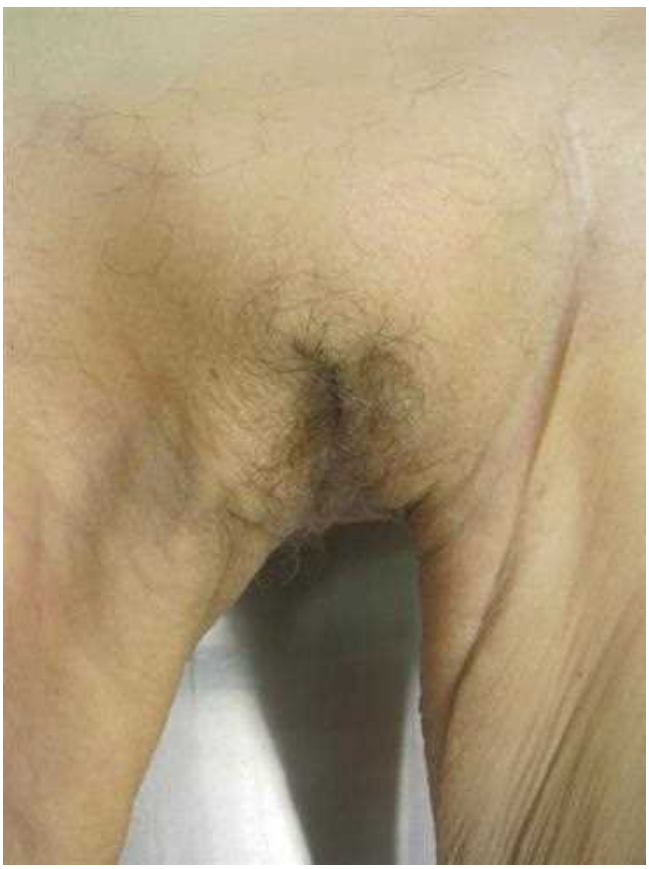

**Fig. 6.49** Malignancy involving the penis (commonly squamous carcinoma) may require resection of the external genitalia in the male. This procedure had been carried out on this man with a pseudo-female genital architecture

## Female Genital Tissues

The vulva and vagina (Figs. 6.51 and 6.52) are intermittently involved in disease ante-mortem, but minimal attention is often focused upon this area at autopsy. Nevertheless, these sites may have common infections (*e.g.*, *Candida*), but exclusion of sexually transmitted diseases should be considered, particularly in the young. As with males, the fashion for genital jewellery (rings, studs, etc.) means that standard skin commensal sepsis may be significant in the groin area. Identification of genital sepsis (standard infections, sexually transmitted infections) is important, as the infection may be easily overlooked and yet the disease may have generalized, systemic effects.

The uterus (Figs. 6.53, 6.54, 6.55, 6.56, 6.57, 6.58, 6.59, 6.60, 6.61, 6.62, 6.63, and 6.64) is best considered as the uterine corpus and cervix (*i.e.*, as one combined piece of tissue). In general terms, in the non-pregnant state, the uterus is rarely of significance, showing variable endometrial features in relation to the standard monthly hormone cycle. It is not uncommon, however, to find a degree of mucus inspissation within the endometrial cavity, and occasionally a contraceptive device. These devices may be associated with sepsis. The uterine body itself has perhaps the most common genital tract pathology—fibroids. Rarely, other uterine neoplasia may be found. Likewise, the cervix can be involved by carcinoma, and in such cases tumour spread to the pelvic tissues must be considered.

The tubes are rarely involved by pathology at autopsy, although cysts are intermittently found. Ties and clips from sterilization procedures should be documented as part of the routine examination.

The ovaries (Figs. 6.65, 6.66, 6.67, 6.68, 6.69, and 6.70) will vary in terms of architecture depending on the age of the individual. The presence of follicle cysts of various stages, corpora lutea, and various scars may be relevant, but these are generally accepted as normal. In cases of maternal death, of course, the presence of such elements is an important component of the description.

The most common pathology seen in the ovaries at autopsy is that of cystic tumours, ranging from small to very large. These are principally mucinous or serous adenomatous processes, although carcinomas can present with cystic change. Rarely, germ cell tumours can be seen, the commonest being dermoid tumours.

**Fig. 6.51** The normal female genitalia are seen with vulval and pelvic tissues. The general autopsy will not have issues of sexual activity or assault as part of the routine screen, but one should always be aware of this possibility

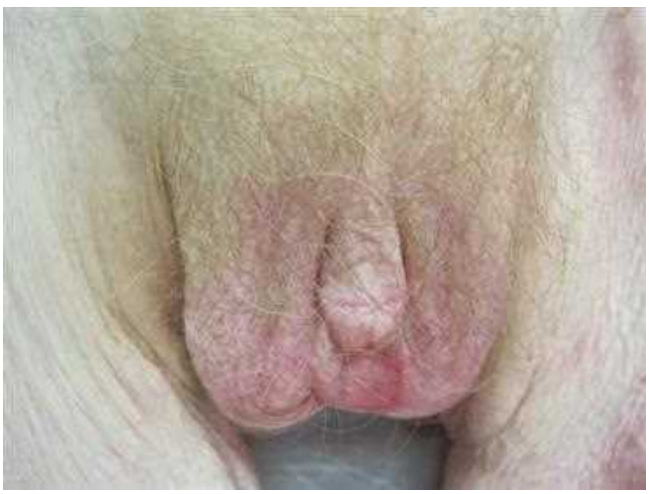

**Fig. 6.52** Pronounced oedema of the female genital tract can produce a distorted vulval appearance and should not be mistaken for neoplasia

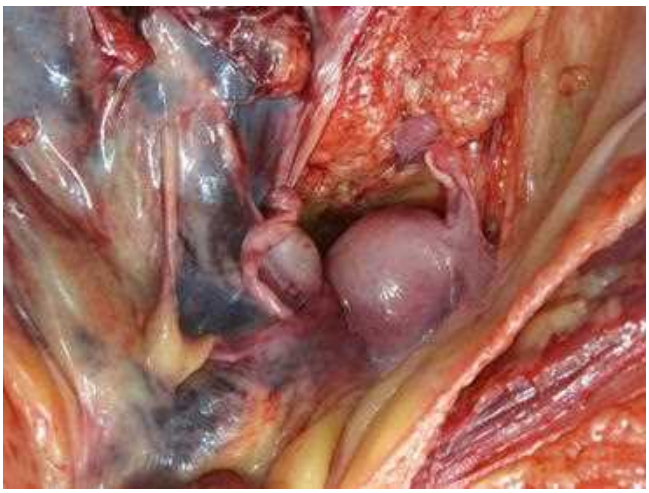

**Fig. 6.53** During evisceration, looking downward from the pubic brim, one can see the uterus, tubes and ovaries during examination of the pelvic content. The internal female genital tract tissues are removed as part of evisceration at the same time as the resection of the rectal and bladder tissues

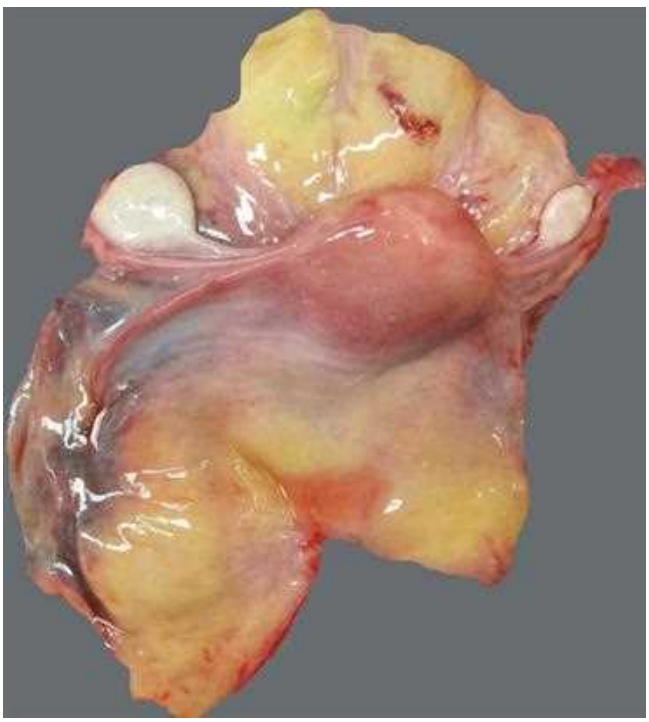

**Fig. 6.54** The uterus, both tubes, and both ovaries are seen with local soft tissues, prior to dissection. This is a postmenopausal case

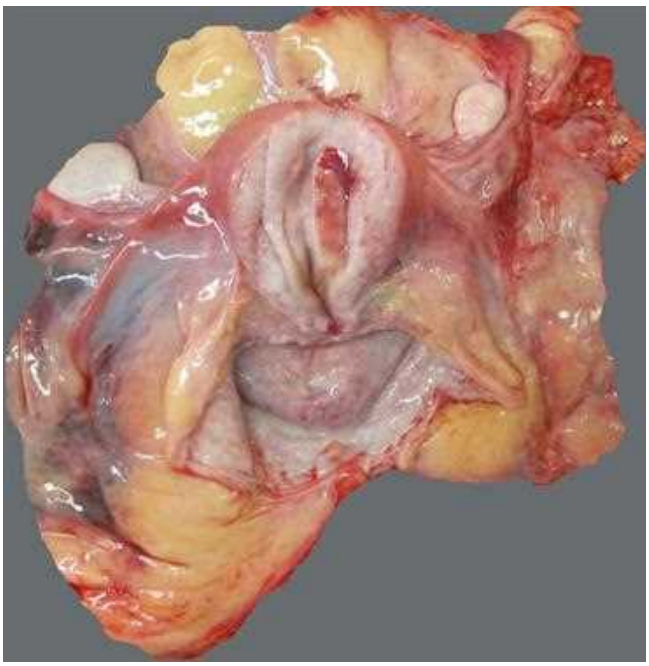

**Fig. 6.55** The same female genital tract tissues can be explored in different fashions. Here, the uterus and cervix have been opened anteriorly in association with the cut passing across the roof of the vagina anteriorly. The ovaries and tubes are seen in a normal position

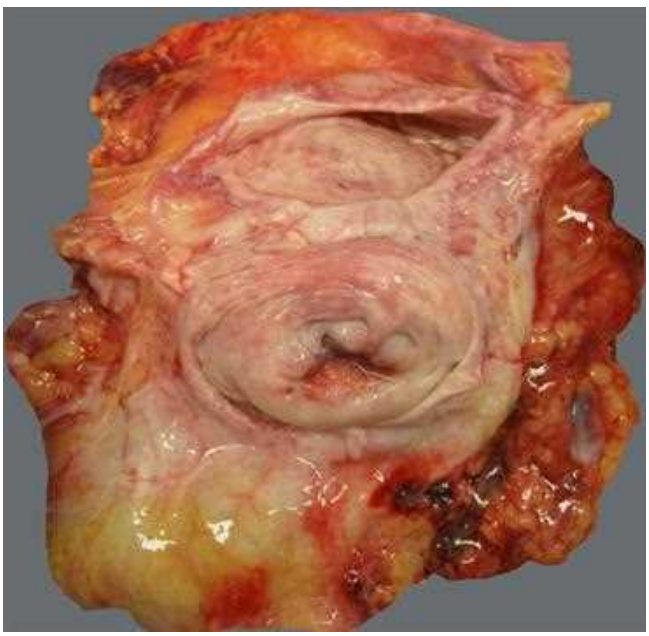

**Fig. 6.56** The bladder has been opened (in front of the uterus), and the cervix is seen from below, looking towards the fundus. The os is ovoid/transverse, in keeping with prior gestation (*i.e.*, multips os)

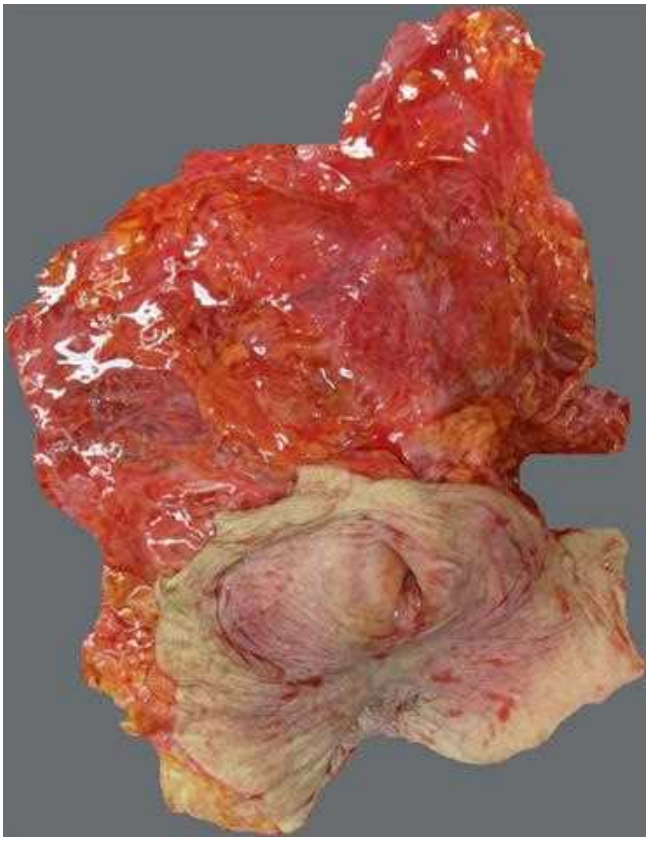

**Fig. 6.57** One may wish to examine the entirety of the perineum, vulva, vagina, internal genital organs, and anorectal tissues in one piece. These are removed in the style of an 'apple core' around the inner pelvic walls; then the incision runs around the perineum. This encloses all the tissues under consideration. If sexual assault is a significant consideration, a wider pelvic-perineal resection margin may be needed, with a more detailed forensic examination. Here one can see the vulva/vaginal tissues and fatty tissue around the internal genital block

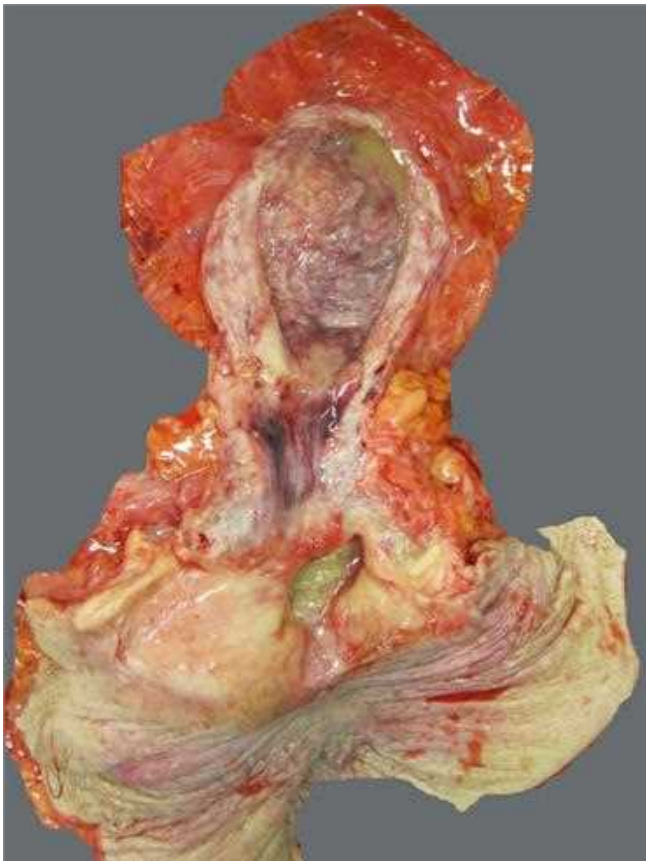

**Fig. 6.58** The bladder has been opened longitudinally through the urethra. Some inflammatory changes are noted

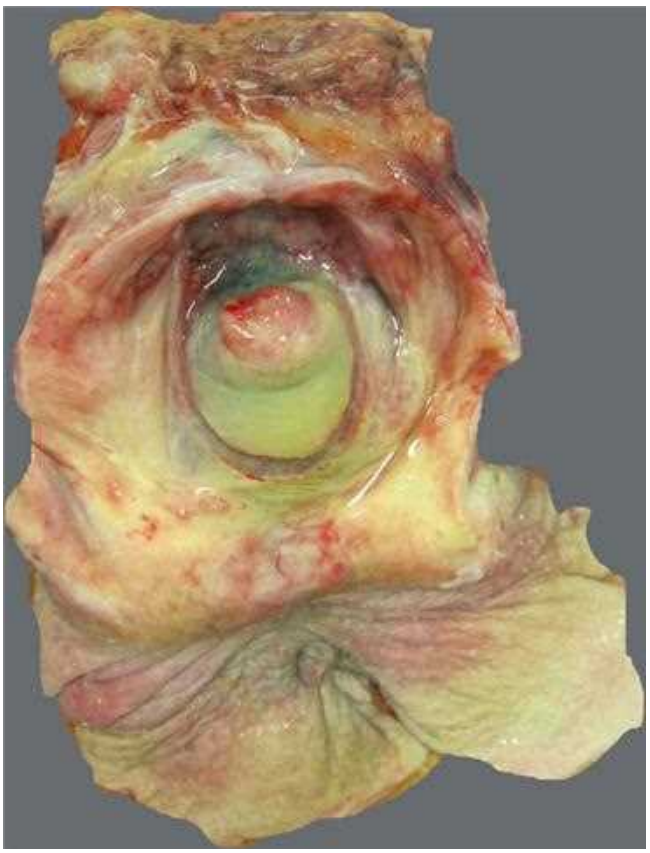

**Fig. 6.59** The vagina is visualized longitudinally; the cervix is seen at the end. Further swabs for microbiology and DNA analysis may be taken

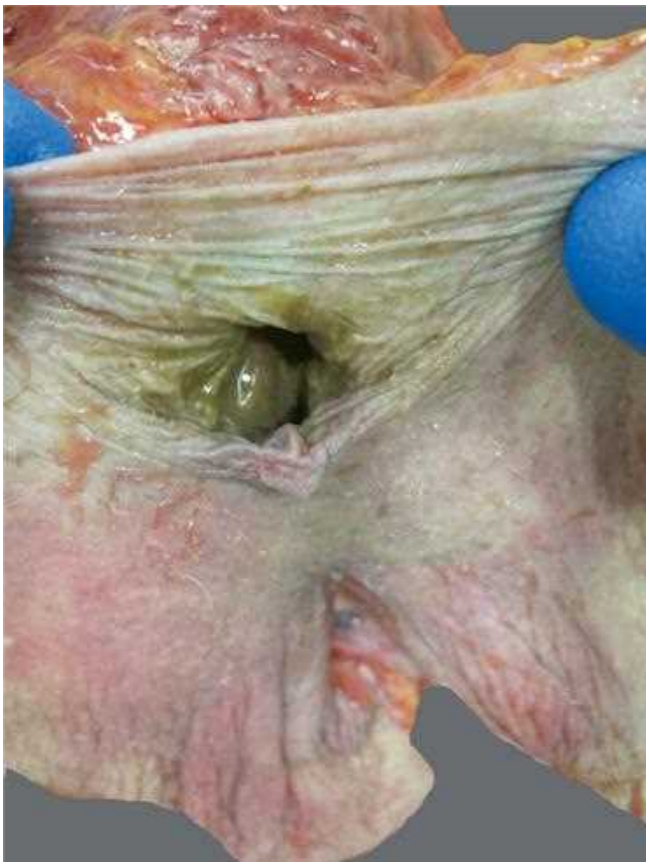

**Fig. 6.60** Turning the tissues around so the rectal tissues are uppermost allows the external features of the anus and rectum to be inspected, with the anal orifice. Any fecal matter can be sampled and swabs taken

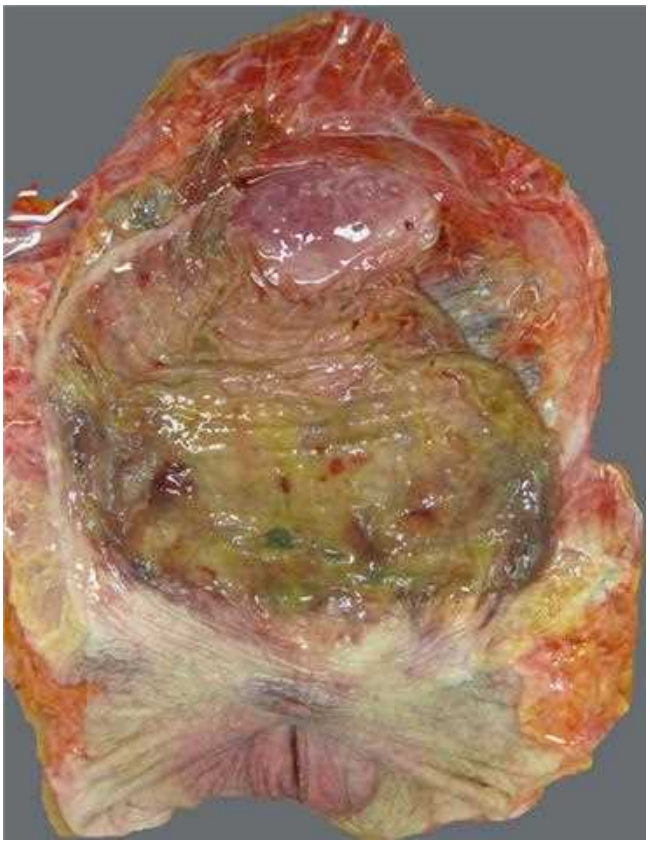

**Fig. 6.61** Keeping the rectal tissues uppermost, the rectum is opened along its length in the posterior midline, allowing a full review of the mucosa and anorectal interface

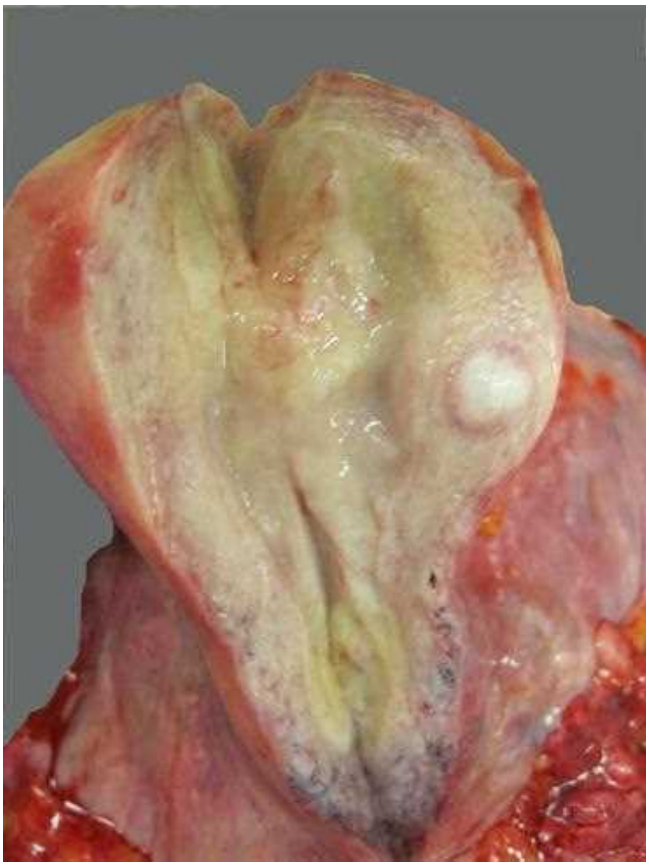

**Fig. 6.63** This uterus has been opened longitudinally. A largely normal myometrium and endometrium are revealed, but a rounded leiomyoma/fibroid is present in the midmural position focally

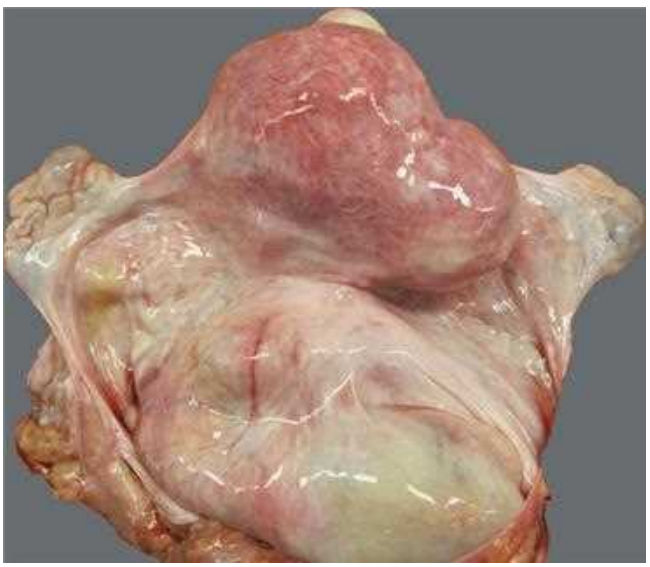

**Fig. 6.62** This uterus, with tubes and ovaries, is nodular and distorted, in keeping with fibroid change, which is a common finding at autopsy. It usually has no significance unless the fibroids are large enough to cause pelvic obstruction

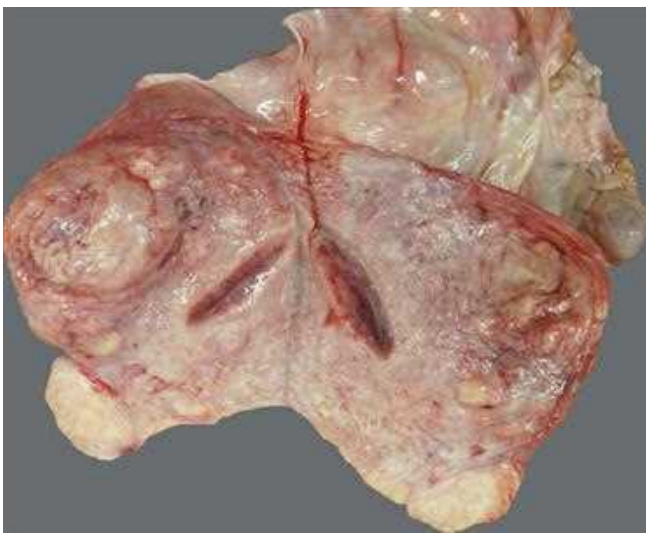

**Fig. 6.64** This uterus and the endometrial cavity are significantly distorted by multiple fibroids (leiomyomas) of varying sizes, making the section down through the cervix difficult

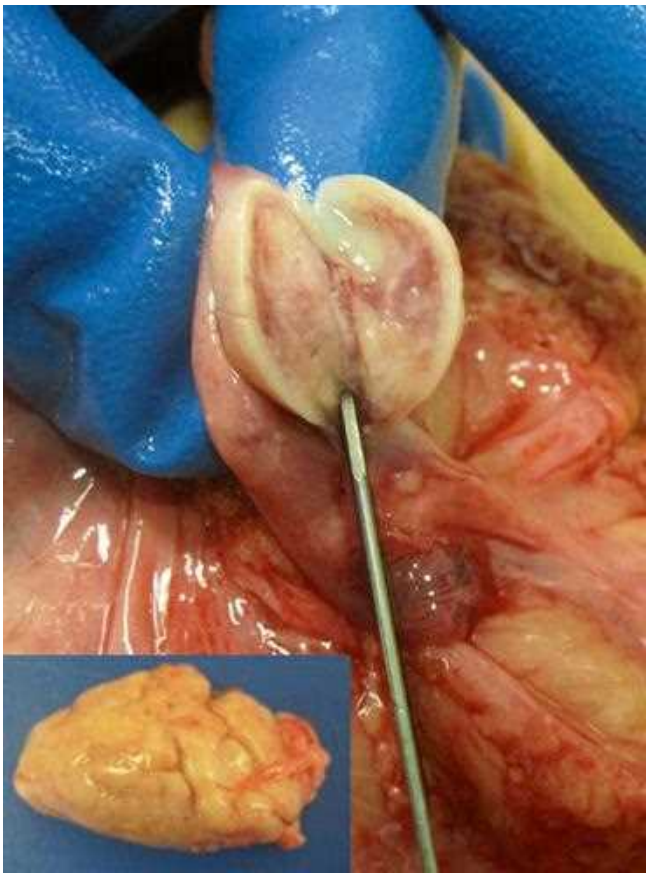

**Fig. 6.65** Ovaries are best examined while still attached to the genital tissue block by slicing them along the long axis. A slightly folded external surface (*inset*) is often seen in ovaries

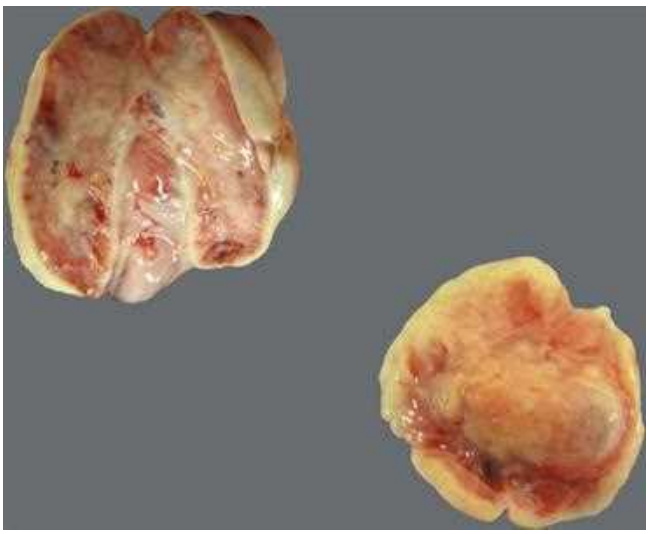

**Fig. 6.66** Two ovaries are seen with small follicles and a bland, pink-grey stroma

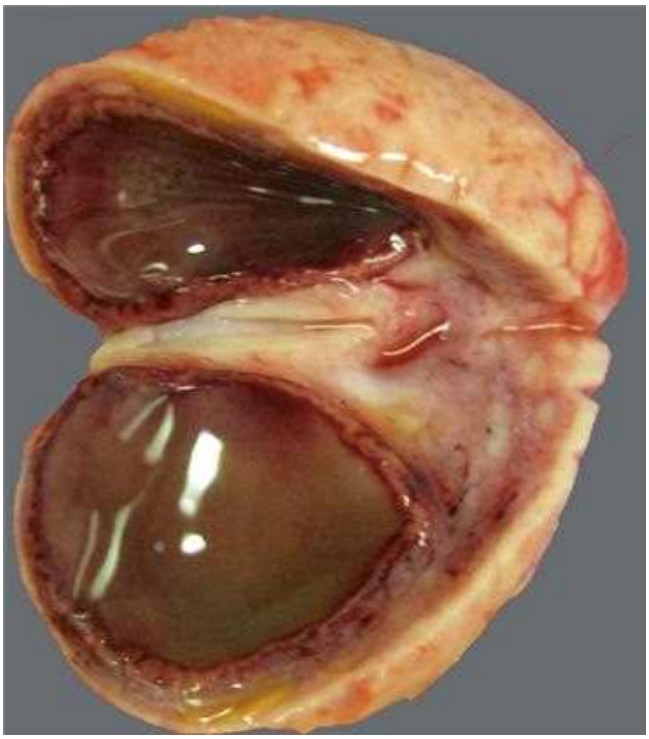

**Fig. 6.67** The ovary shows a corpus luteum cyst as part of early pregnancy

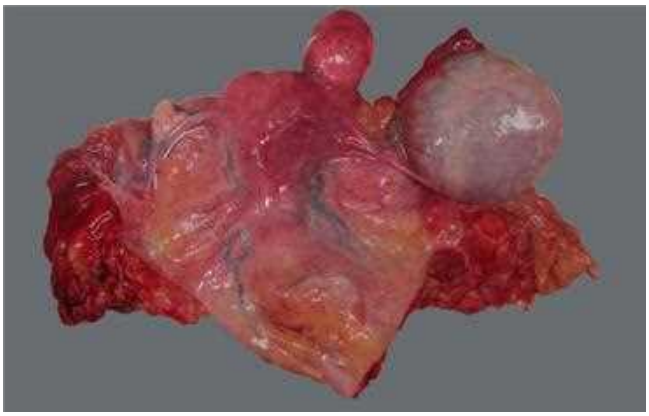

**Fig. 6.68** This ovarian cystadenoma was an incidental finding during evisceration and had no bearing on the cause of death. The tube is stretched around the cyst

**Fig. 6.69** Features of malignancy in an ovarian cyst are suggested by the presence of solid and papillary lesions. This ovarian carcinoma was found incidentally, and disseminated disease was not identified (Marker, 10 mm)

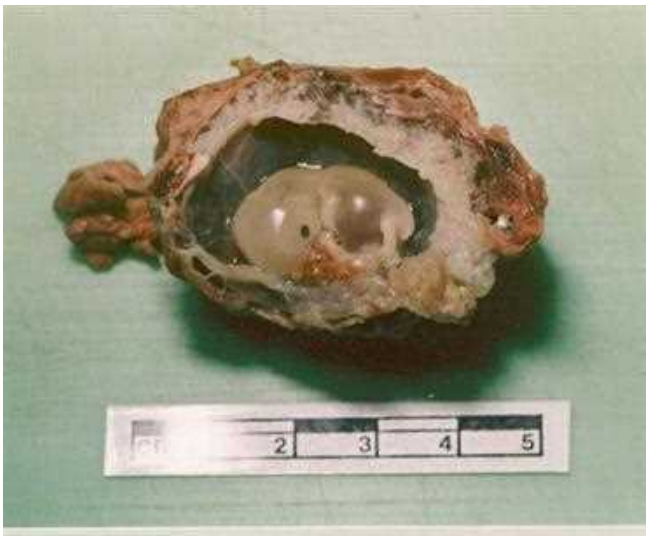


**Fig. 6.70** Other cystic lesions adjacent to the ovary may include cysts of the tubes. These are mostly benign and of no consequence, but pronounced bleeding and death may follow ruptured tubal ectopic gestation. This fixed surgical sample shows an ectopic early-stage pregnancy, without rupture of the tube

## Pregnancy

The maternal death autopsy (Figs. 6.71, 6.72, 6.73, 6.74, 6.75, 6.76, 6.77, 6.78, 6.79, and 6.80) is a specialist autopsy analysis, which must cover ante-mortem matters in detail, alongside aspects of the pregnancy and potentially the delivery. Often, the concept of the maternal death autopsy may be invoked for up to a year following the birth of a child.

Particular emphasis upon the genital tract tissues will be required in maternal autopsy, with close inspection of the entire female genital tract. Identification and sampling of any septic focus (using uterine swabs, blood cultures, spleen swabs, etc.) is a priority, and it is important to widely sample these tissues.

Likewise, histological assessment of the genital tract is important. If delivery has recently taken place, an attempt should be made to secure the placenta and membranes. If the gestation is still in situ, then gross examination of the fetus is often all that is required. This examination confirms normality in most cases.

It is recommended that a digital photograph of the genital tract tissues is undertaken for record purposes, but an image of the child may be helpful to the parents at a later date. At least one image should be taken using a background other than an autopsy station, such as an image akin to a sleeping child in blankets.

A fetal autopsy is rarely needed unless there is some specific issue with regard to development. It should be remembered that, on occasion, a small amount of fetal or placental tissue should be retained for paternity tests. The fetal autopsy full analysis is beyond the scope of this text, and the reader is advised to consult specialist texts and/or to take advice from local paediatric pathologists.

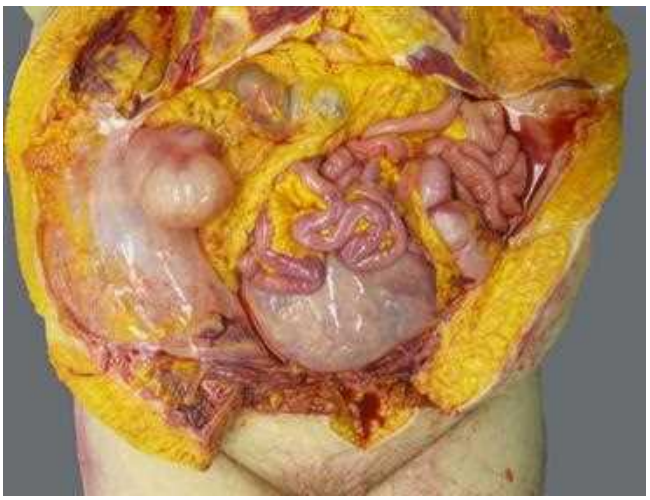

**Fig. 6.71** Autopsy cases involving pregnancy require special consideration and examination. If the fetus is still in situ, it is important to examine the genital tissues with particular care in order to exclude structural problems and sepsis pathology associated with the fetus. The uterus may appear enlarged, with the size reflecting the stage of gestation. One starts with the general overview as the abdomen is opened

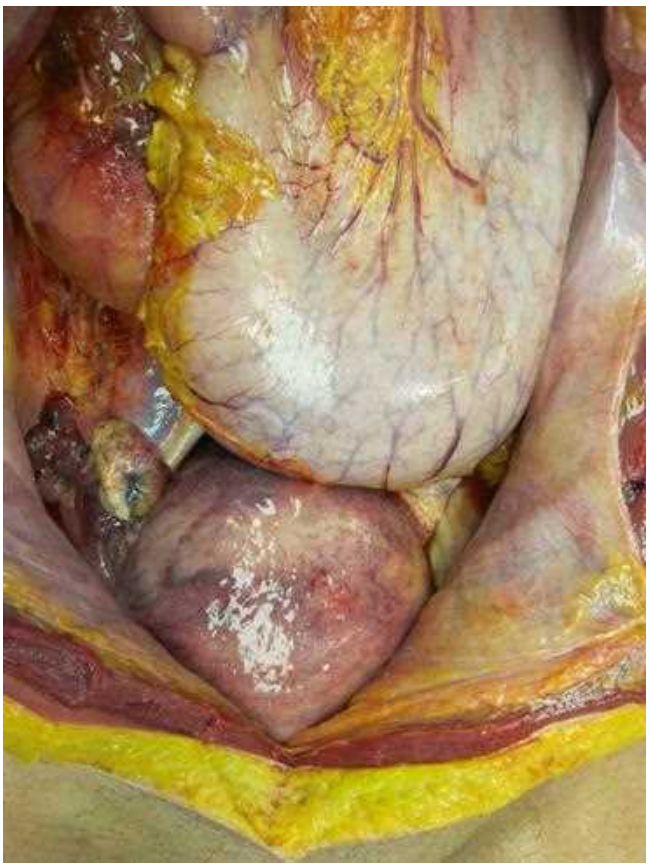

**Fig. 6.72** The gravid uterus should be considered beforehand in terms of the anatomical position, the other abdominal viscera, and the local vasculature

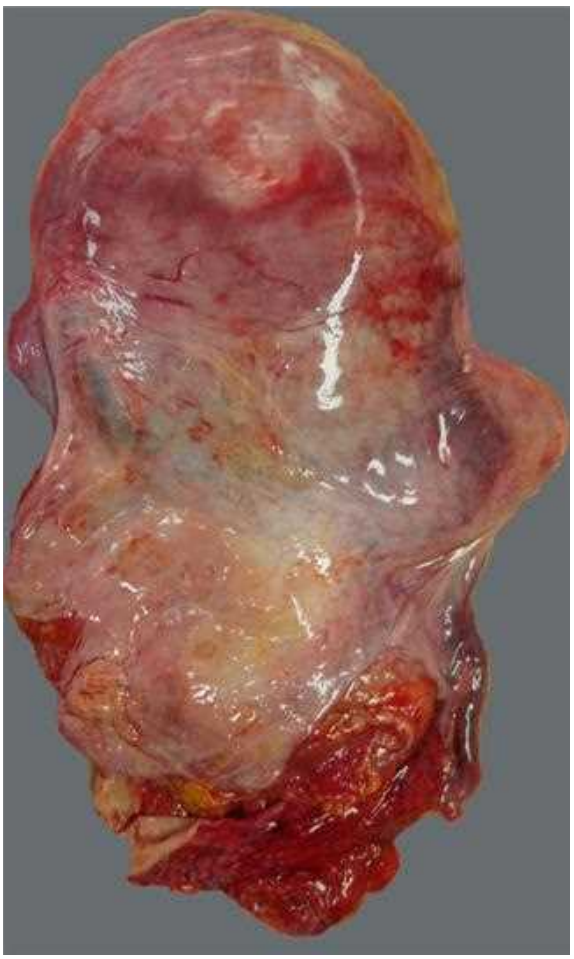

**Fig. 6.73** Pregnancy-associated uterine enlargement needs to be carefully considered in terms of uterine content as well as possible sepsis or significant bleeding (Neither was present in this case)

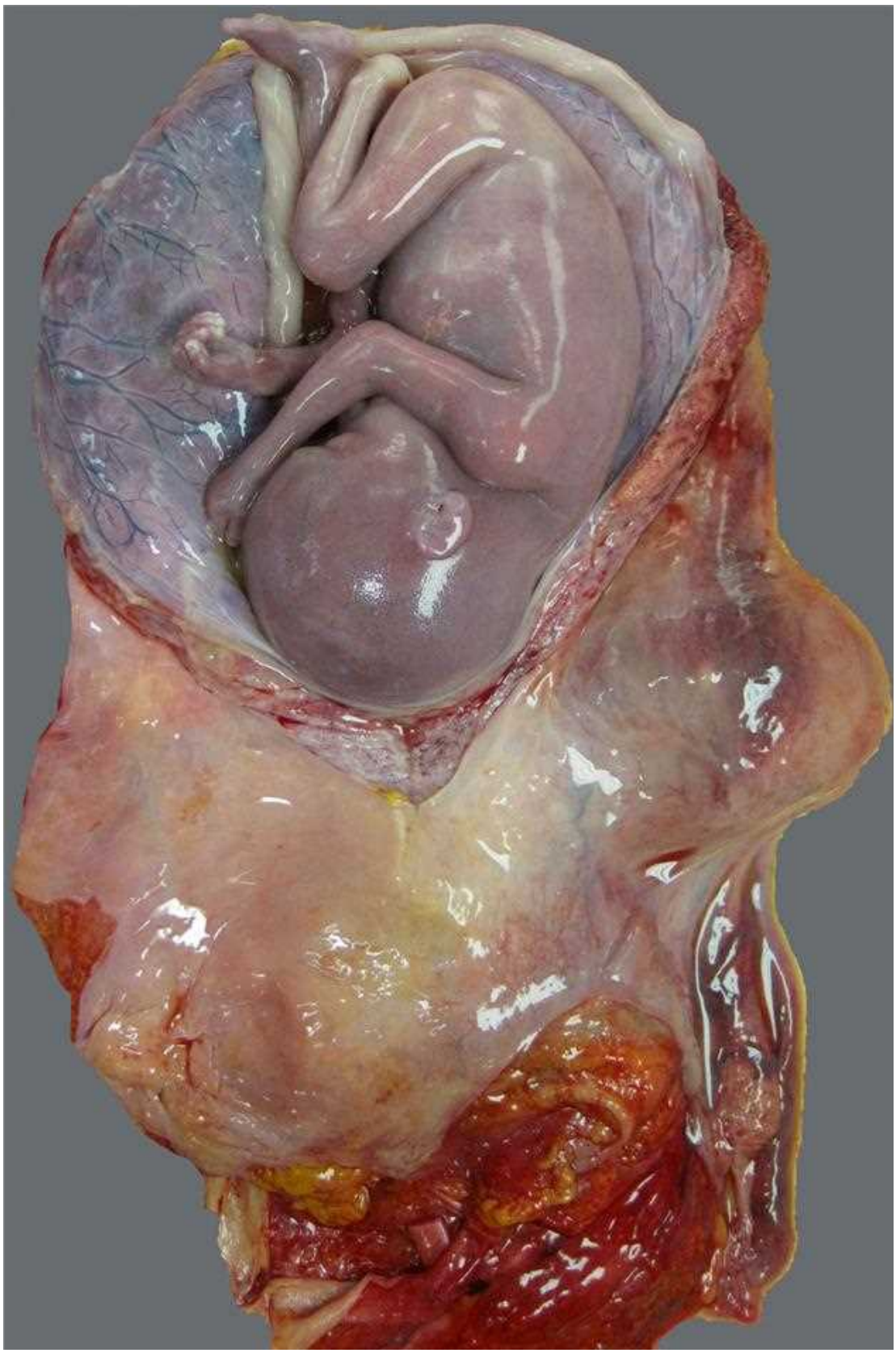

**Fig. 6.74** Opening the uterus—done carefully to avoid damage to the placenta and fetus—will allow direct inspection of the uterine cavity, the membranes, and the placental tissues. In most cases, the fetus has no abnormality and is not examined beyond recording standard measurements (foot length, crown rump length, crown heel length, head circumference, and weight parameters). Examination to determine sex and appropriate development is required. Exclusion of congenital abnormalities is recommended. The cord should be measured in terms of length and sectioned to confirm the presence of three normal vessels. The placental surface and membrane should be clear and translucent. The presence of plaque or discoloured membranes suggests sepsis. Swabs should be taken when the gestational sac is open, to exclude sepsis

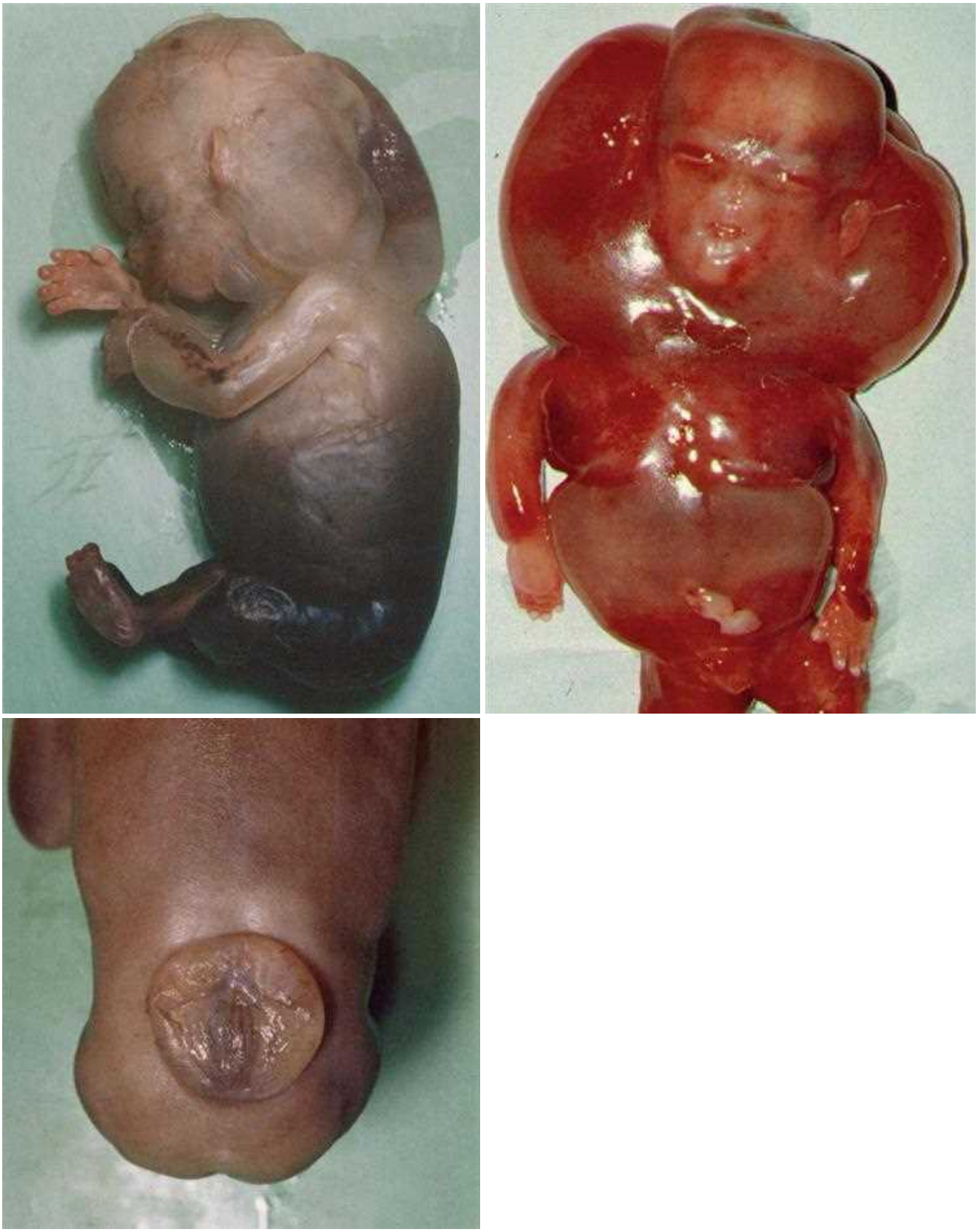

**Fig. 6.75** A fetus may have various pathology. Neural canal defects (*lower left*), nuchal oedema (*upper left*), or generalized hydrops (*upper right*) should be noted. Fetal tissue review by a paediatric pathologist may be desirable, but this will need consideration on a case-by-case basis

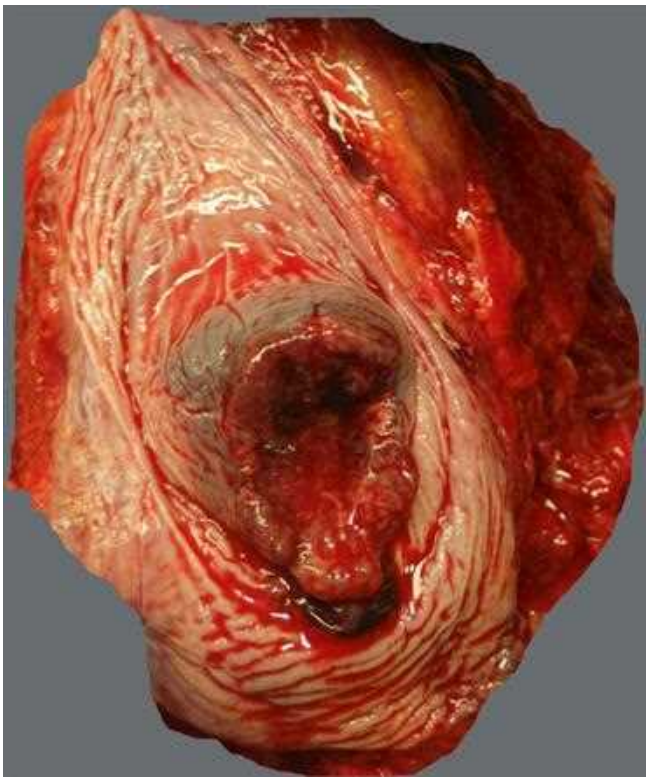

**Fig. 6.76** If vaginal delivery has already occurred, the uterus will be empty. Examination of the cervix may show an open and somewhat patulous cervix with variable degrees of congestion and haemorrhage, consistent with the delivery process

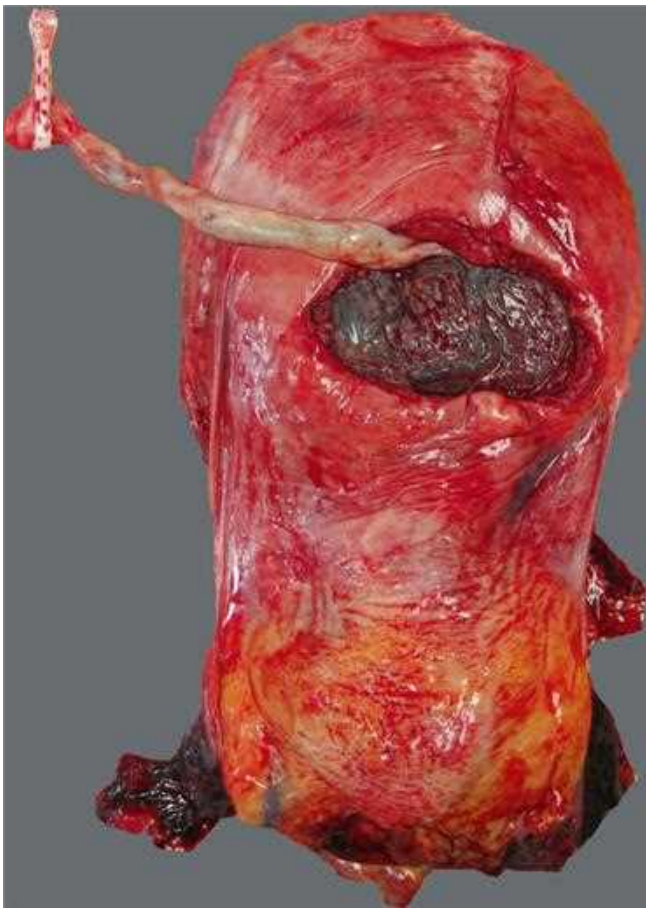

**Fig. 6.77** Deaths occurring in late-stage pregnancy often have had emergency caesarean section performed. In these situations, the uterus will be empty of the fetus but may still contain the umbilical cord and placental parenchyma. The same approach applies: the cord is measured in terms of length and the presence of three normal vessels is confirmed. The placenta may be examined in terms of gross anatomy, and there may be a need for histology

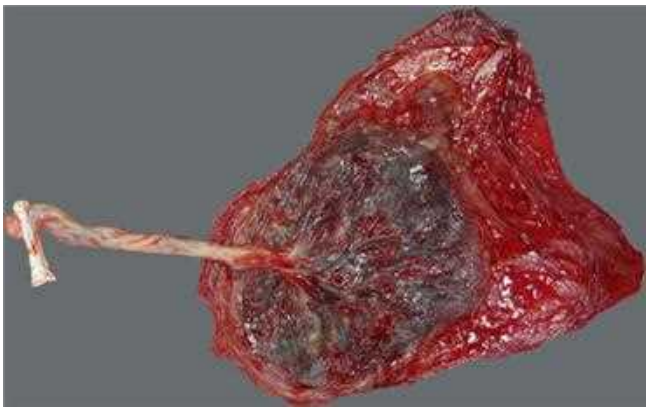

**Fig. 6.78** The placenta can be removed from the uterus, usually without any difficulty. It should be examined in terms of size and weight, and the placental tissue should be sliced to exclude infarction or frank sepsis. The membranes must be examined and the cord details checked

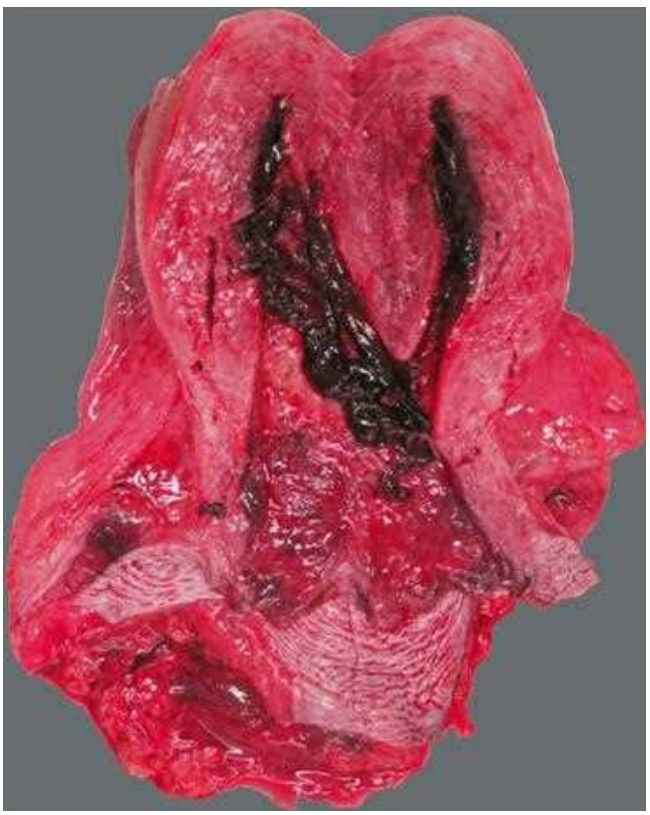

**Fig. 6.79** This sample, with pronounced endometrial surface haemorrhage, represents an emergency caesarean section taken shortly after the death of the mother. The degree of haemorrhage is entirely in keeping with the emergency procedure, but samples for microbiology and histology may still be of value

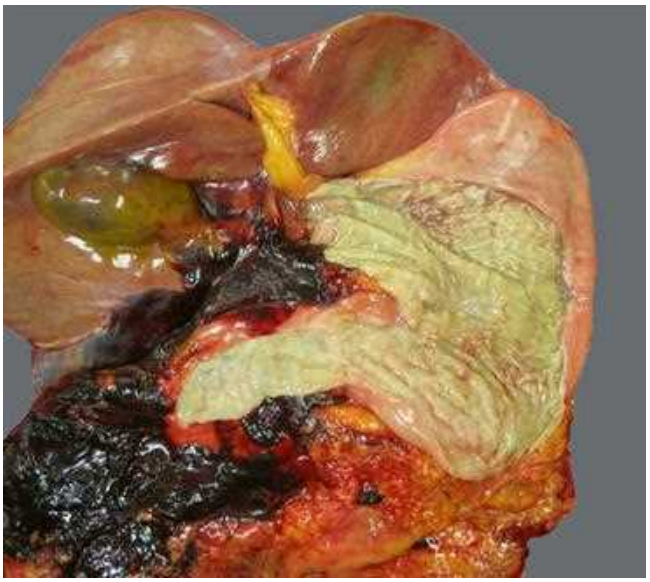

**Fig. 6.80** Seen here is a rare cause of sudden death in mid or late pregnancy: isolated arteritis/aneurysm rupture with catastrophic abdominal bleeding. This case involved a small branch of the celiac artery; the upper abdominal tissues are shown. The aetiology is unclear, but a diligent search for the bleeding point is advocated

## Breast Tissue

The breast tissues (Figs. 6.81, 6.82, 6.83, 6.84, 6.85, 6.86, 6.87, 6.88, 6.89, 6.90, and 6.91) are rarely an important matter at autopsy. Initially, they are covered as part of the external examination. In most cases, simple palpation of the breast tissue will suffice, to exclude pathology. Breast implants are not uncommon, and care should be taken to avoid damaging them during evisceration. Rarely, breast implants can be associated with sepsis, particularly in the early postoperative period. Natural-tissue breast infections are rare, but tumours should always be sought initially by internal and external palpation. Slicing the breast from the internal aspect (*i.e.*, after the chest has had the skin and soft tissues dissected free from the rib cage) will allow detailed review of the breast parenchyma. Ideally, one should not cut into the skin overlying the breast, for cosmetic and reconstructive reasons. Benign tumours such as fibroadenomas are occasionally found, and malignancy is a not uncommon reality in the autopsy room. Malignant tumours of the breast may be pertinent to the cause of death, although the disseminated disease is usually the pathology of significance.

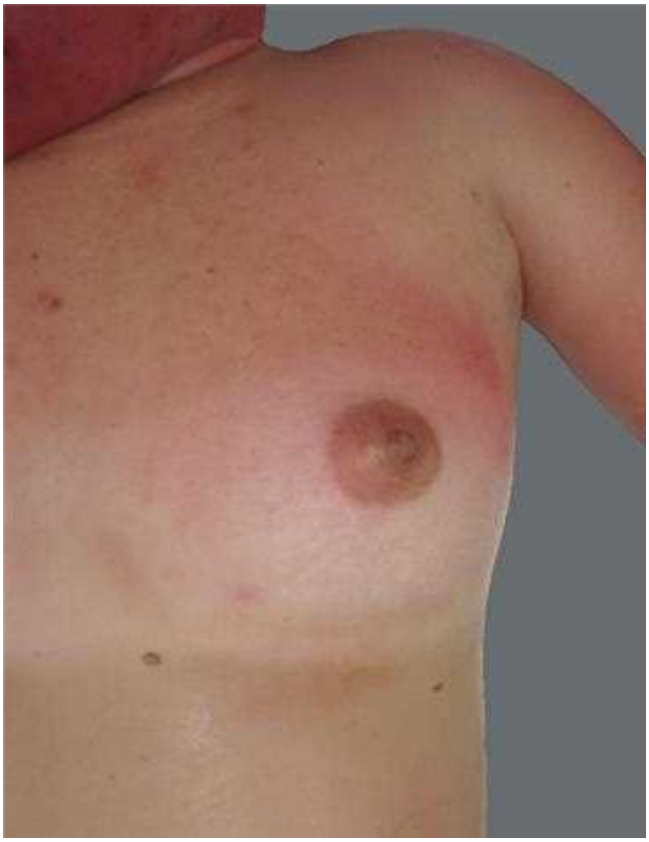

**Fig. 6.81** Normal breast tissue is usually confirmed by visual inspection prior to the start of the invasive autopsy. Palpation of the axillae (to exclude nodal enlargement) and of the breast parenchyma can be undertaken

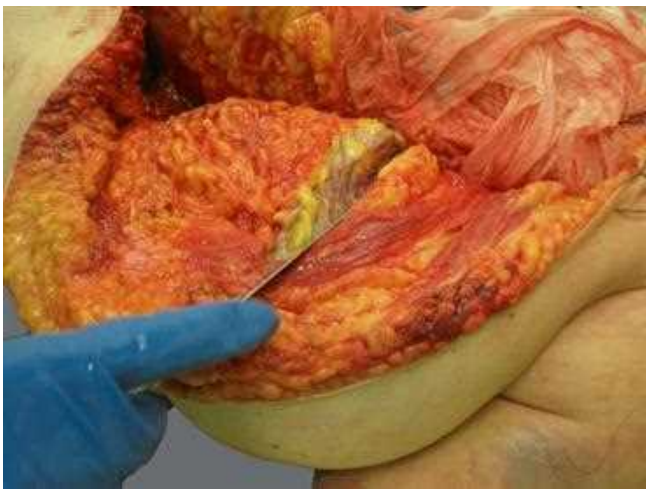

**Fig. 6.82** If there is concern about the breast tissue, serially slicing the parenchyma for the internal aspect (without damaging the external breast skin) can be undertaken to search for focal lesions. Shown is some wadding around the sharp cut ends of the rib cage

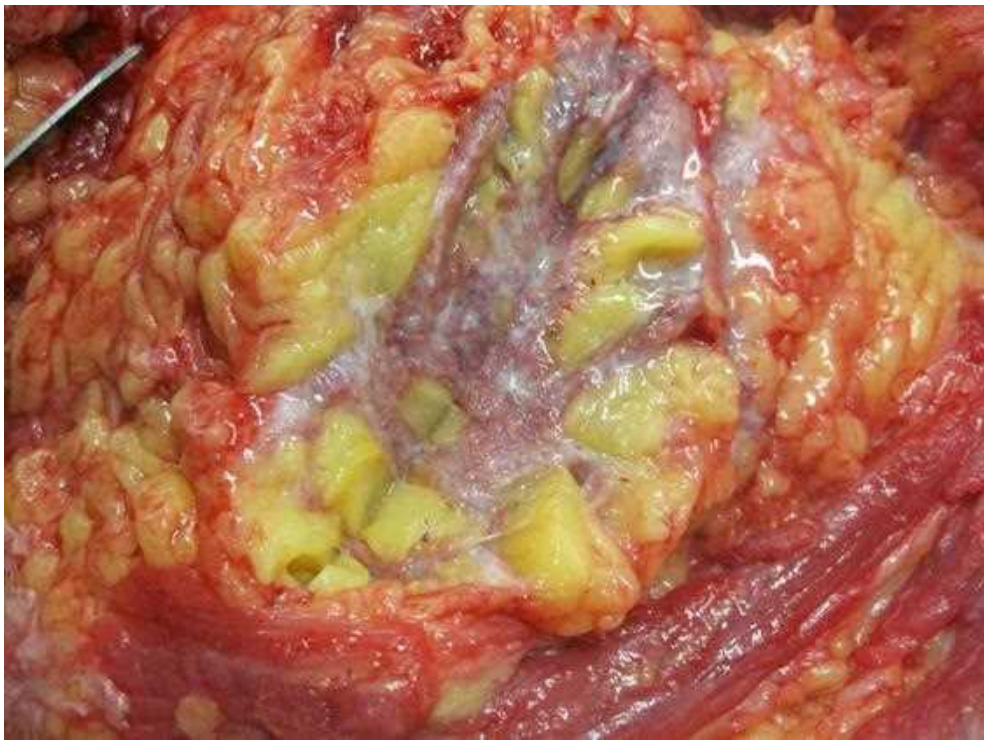

**Fig. 6.83** Breast tissue can change significantly during pregnancy, with the acinar parenchyma being particularly prominent. Significant secretory development occurs towards the end of pregnancy

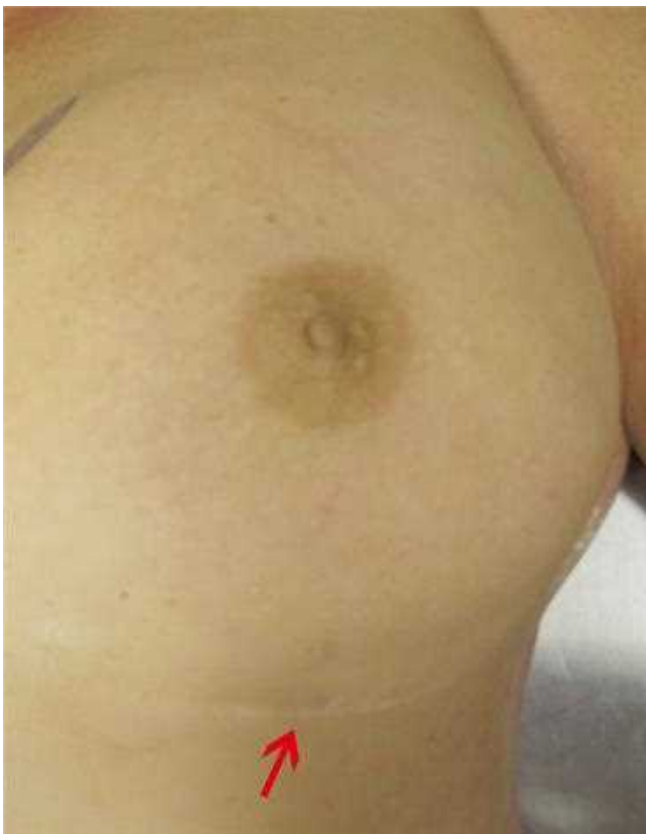

**Fig. 6.84** Breast implants are increasingly seen in the autopsy room. This apparently normal breast tissue is seen to have scarring under the breast, in keeping with prosthetic breast enlargement. Characteristic sites of scarring include centrally below the breast and high in the axilla. The scar (*arrow*) often gives the first indication of an implant

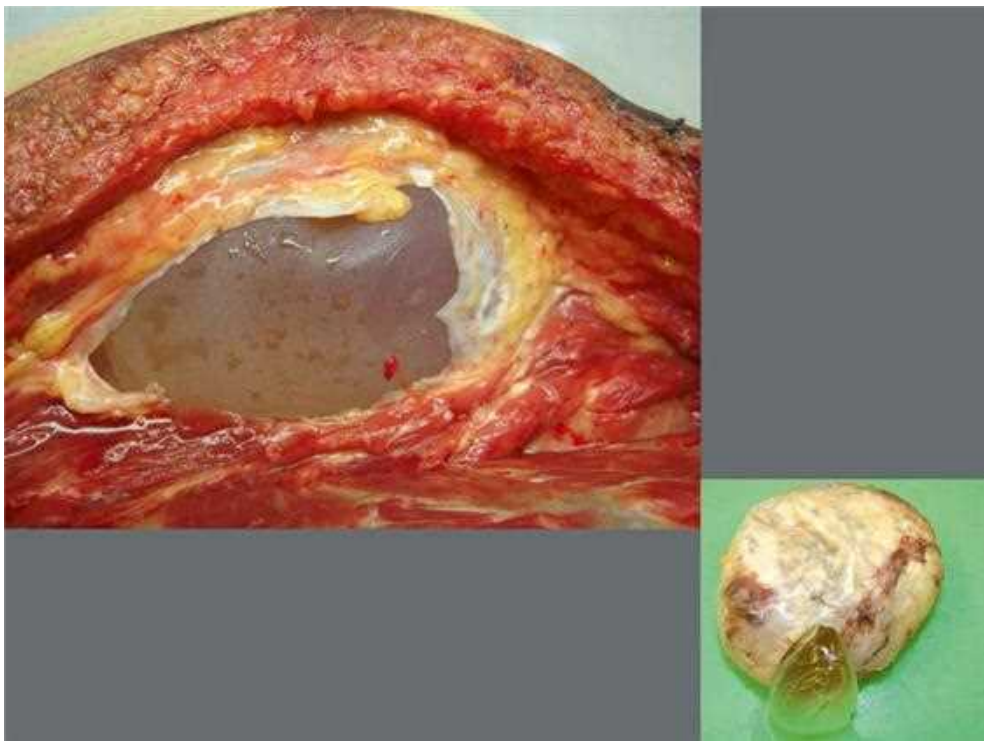

**Fig. 6.85** Opening the body as part of evisceration may demonstrate the prosthesis (*upper left*). It is important not to damage the prosthesis during evisceration, so that body reconstruction can proceed without significant change to the anatomical boundaries seen ante-mortem. Modern implants have less associated fibrosis than those used previously (*lower right*), and are no longer filled with silicone materials

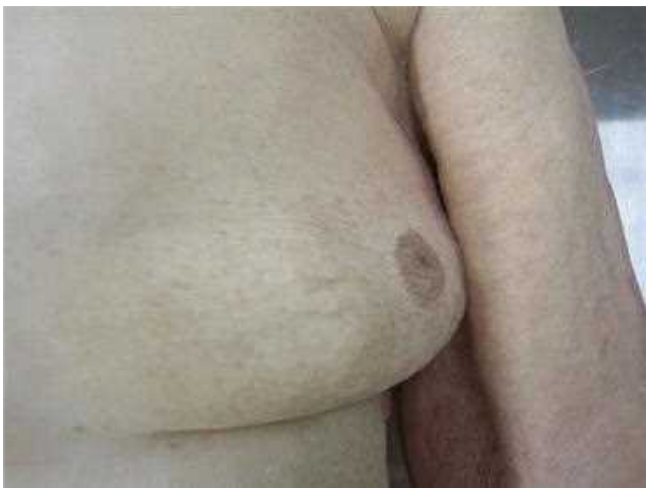

**Fig. 6.86** The presence of breast cancer often can be seen on naked-eye examination as an ill-defined lump (here, medial to the in-drawn nipple)

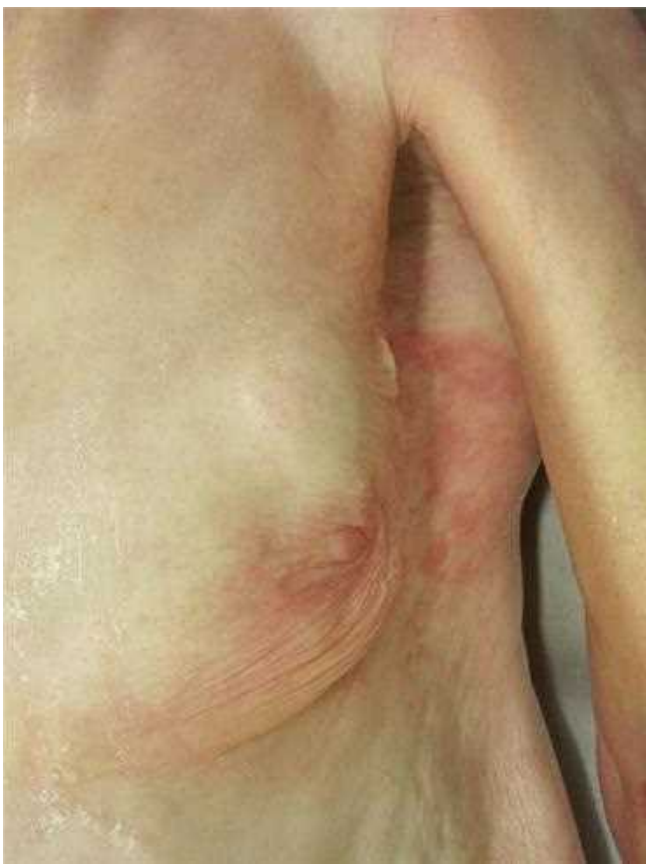

**Fig. 6.87** Another case of breast cancer is seen from the external aspect. There is a tumour above the nipple

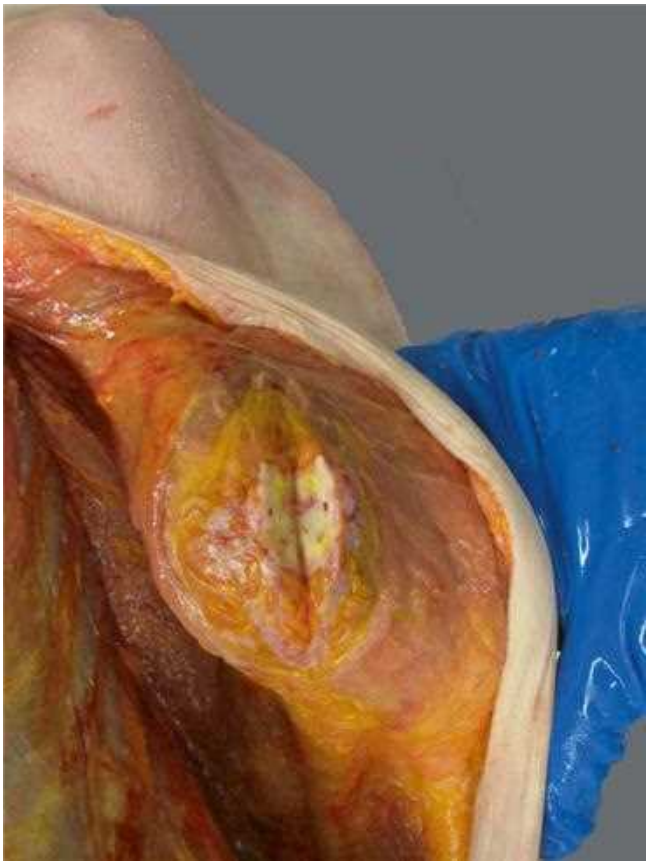

**Fig. 6.88** Primary breast cancer is seen with a fatty and scirrhous cut surface, sliced from the inner aspect

**Fig. 6.89** The breast cancer here has been treated with radiotherapy. Note the enhanced sclerosis and the zone of old haemorrhage and necrosis centrally

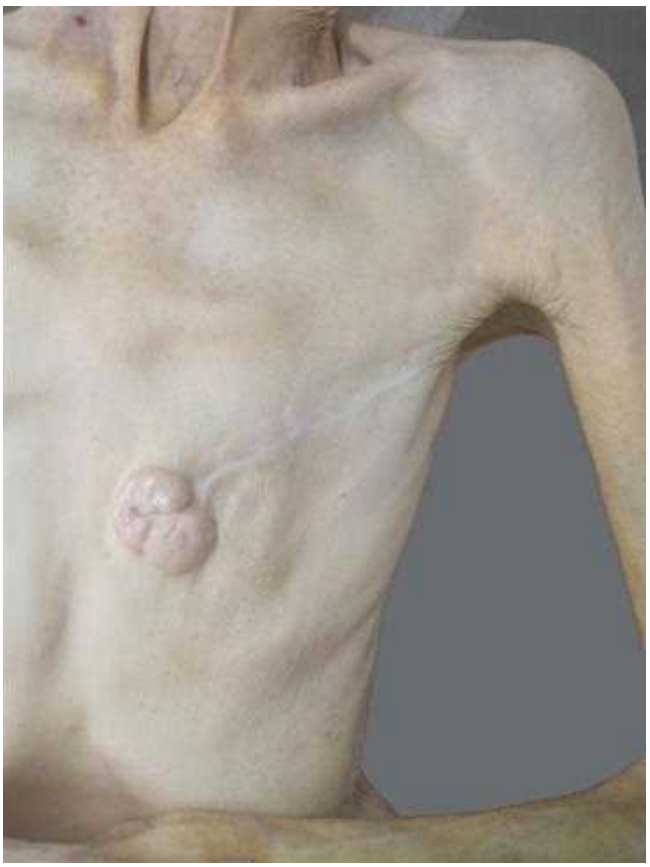

**Fig. 6.90** This previous mastectomy site is noted to have recurrent tumour at the medial end, which may suggest disseminated disease elsewhere in the body. Due diligence to consider metastatic sites is recommended

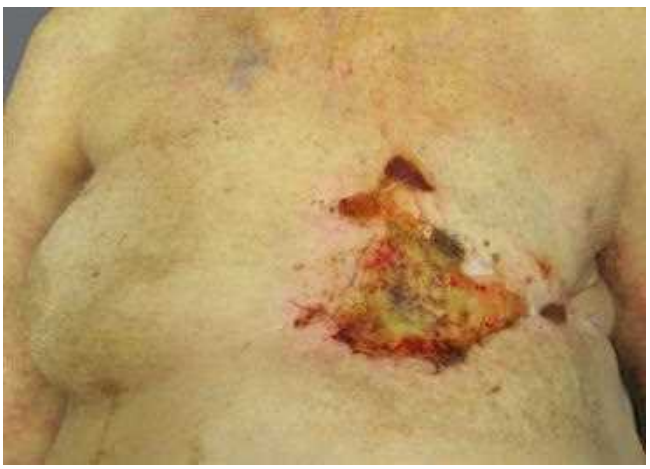

**Fig. 6.91** Recurrent breast cancer can be widespread on the chest and destructive. This previous mastectomy site showed extensive skin infiltration by tumour that ran into the local musculature, deeper parenchyma, and indeed the bone. Widespread metastases were also present

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# Lymphoreticular Tissues: Lymph Node, Spleen and Thymus, Bone and Marrow

7

S. Kim Suvarna

## Introduction

The tissues of the lymphoreticular system are often examined almost incidentally as one performs an autopsy. These tissues are rarely the primary focus of the disease, unless considering the bony tissues and fractures. Yet this widespread group of tissues is responsible for many aspects of haematological and immune function, so it is not uncommon to see minor pathology of relevance during the post-mortem examination.

The lymph nodes commonly vary in size and shape when one considers the various types of autopsy cases. Localised or generalised lymphadenopathy may point towards systemic infections, autoimmune conditions, or neoplasia. Some histology sampling of such nodes is recommended, but one also should not forget to consider reserving some tissue for microbiology testing. Correlation against clinical data is always important.

The spleen, found adjacent to the stomach and left kidney, is easy to identify. In most autopsies it will be of normal shape and configuration, but cystic change, nodularity, or gross enlargement may point to either local or systemic disorders. Softening of the tissues may point towards sepsis.

The thymus atrophies, in relative terms, as one ages. Whilst relatively easy to identify in infancy and early childhood, the thymus is more difficult to appreciate as the amount of mediastinal fat increases. Indeed, most autopsy practitioners say they cannot find the thymus in the elderly! It is often relatively easy to define at the superior boundary of the pericardial sac, however, even in the elderly. The thymus is rarely of significance unless a tumour is present.

Unless there are fractures, the bone and marrow are rarely pursued during the autopsy examination. The presence of osteoporosis is common with increasing age and may be a significant factor in terms of fractures and local complications. Histology may be relevant, but bone invariably needs fixation and decalcification.

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## Lymph Nodes and Related Tissues

The lymph nodes are often found to be variably enlarged at autopsy. In most cases, this reflects terminal septic conditions, principally in the chest. Reactive nodal hyperplasia and infections are a common reality of autopsy practice. For this reason, some lymph node tissue sampling (histology and microbiology) may be of assistance when faced with diffuse lymphadenopathy.

In the elderly, it is not uncommon to find low-grade, unsuspected chronic lymphocytic leukaemia or lymphocytic lymphoma. Metastatic carcinoma (sometimes unsuspected) requires both macroscopic assessment of abnormal tissue in the lymph nodes and histological confirmation. The primary site should be sought.

Rarely, atypical infections, autoimmune disease, and other infiltrates may present as nodal enlargement—highlighting the need for histology and for microbiology to exclude sepsis.

## Spleen

The spleen (normally 100–150 g) generally shows little relevance to the autopsy; that is, it is usually of normal weight, shape, and form. It may be enlarged, however, from background processes (*e.g.*, autoimmune conditions or cirrhosis). In these circumstances, the cut surface parenchyma usually is still unremarkable.

In terminal septicemia or bacteremia, the splenic tissues often appear soft and semifluid on section. If sepsis is suspected, the outer surface of the spleen should be sterilized by the application of alcohol and/or formalin, given that a hot metal to sear the surface is generally not a commonplace tool in the modern autopsy room.



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The splenic tissues also can be involved by tumours. These commonly comprise lymphomas although metastatic carcinomas and angiomatous processes are also well recognised.

Splenic trauma is of particular importance. Any capsular tear or deficit should be recorded in terms of position and size, with an assessment of any blood volume lost locally.

Gross enlargement should raise the suspicion of atypical infections such as malaria or leishmaniasis.

## Thymus

The thymus is normally found at the superior/anterior aspect of the pericardial sac, immediately behind the sternal tissues. Its size can be quite variable, but generally it is about 40 mm in cross-section. The weight varies depending on the amount of fatty tissue and the diligence with which one dissects normal fatty tissue away from the thymic parenchyma.

It is fair to state that the thymus is rarely involved in pathology of importance at autopsy. Mild prominence of fatty thymic tissues in the adult is often nonspecific macroscopically. Nevertheless, basic examination of the thymic tissues should occur at the time of opening the pericardial sac, in order to exclude localised neoplasia, cystic change, or indeed hyperplastic phenomena. If these are present, then histological interpretation is advised.

Thymic pathology in rare cases tends to occur in autoimmune disorders and also can occur in generalised inflammatory conditions. In such cases, histology is recommended for assessment. Likewise, thymic tissues can be enlarged by infiltration of the parenchyma by lymphoma. Rare tumours include carcinoid, germ cell, and thymic epithelial neoplasia. All possible neoplastic processes require histological assessment.

## Bone and Marrow

The bone tissues are rarely the focus of an autopsy, but if so, it is likely that the commonest task is to consider primary or metastatic neoplasia. Metastatic neoplasia is more common, but primary tumorifactive leukemic and lymphomatous neoplasms also may present with pathological fractures.

Another prime area of pathology involving bone is the issue of trauma in the form of fractures with local haemorrhage (sometimes significant). In many cases, these may involve osteoporosis, although one should always be aware of possible metabolic bone disease. Ante-mortem radiology is often of benefit in assessing cases, and one may argue that it provides better data than a standard autopsy. Post-mortem radiology (see Chap. 13) is also making a significant impact in the arena of fractures. If skeletal evaluation is needed, then the sites, degree, number, and types of fracture should be recorded as a minimum.

Osteoporotic bones break easily, and all autopsies should assess for osteoporosis. The direct pressure of one's thumb into the cut surface of an osteoporotic individual's vertebral body will often indent the bone. This yields a simple assessment of bone volume, since osteoporotic marrow is readily deformed. The alternative method is to use isolated ribs during initial dissection (slicing either side of rib tissues), with a subsequent attempt to break them by simple, direct finger pressure. In this test, osteoporotic bones break easily and yet can be re-aligned before body reconstruction.

Formal assessment of osteoporosis will require histology, in which decalcified tissues can be assessed in terms of bone volume (*i.e.*, the volume of ossified tissues over the total bone volume). In cases of metabolic bone disease, specialist techniques for assessment of bone mineralization may require consultation with specialist bone pathology services before one undertakes the autopsy.

### Cervical Vertebral Tissues and Cord

The approach to the spinal cord is particularly complex when dealing with cervical bone injuries. The close relationship of the skull and the complex bone and ligaments at the base of the skull means that the assessment of cervical spinal pathology is technically difficult. In addition, the standard incisions provide limited access to this site.

One approach to this issue is to remove en bloc the central base of the skull and the cervical bony structures. Thereafter, after resection of the vertebral tissues, the back of the spinal canal is removed, allowing access to the spinal cord. Lastly, the vertebral body block is cut (with an oscillating saw) in a sagittal plane, allowing inspection of the bony tissues. This method allows significant inspection of the cervical bone and neurological tissues. The alternative is to rely upon radiological images from ante-mortem studies.

### Complications Adjacent to Fractures

The degree of blood loss in association with any fracture should be estimated, as it can be significant and pertinent to the cause of death. Such blood loss may occur from the bone fracture site itself. Multiple fractures will necessarily incur a greater degree of blood loss than small bone fractures. Local trauma to large arteries and veins may also complicate even small fractures. Any preexisting anticoagulation will further aggravate matters.

The autopsy must consider aspects of medical and surgical interventions with bone fractures, particularly in the form of metallic and other prostheses.

Finally, one must remember that bony tissues can be involved by sepsis, mostly following surgery and involving prostheses.

Figures 7.1, 7.2, 7.3, 7.4, 7.5, 7.6, 7.7, 7.8, 7.9, 7.10, 7.11, 7.12, 7.13, 7.14, 7.15, 7.16, 7.17, 7.18, 7.19, 7.20, 7.21, 7.22, 7.23, 7.24, 7.25, 7.26, 7.27, 7.28, 7.29, 7.30, 7.31, 7.32, 7.33, 7.34, 7.35, 7.36, 7.37, 7.38, 7.39, 7.40, 7.41, 7.42, 7.43, 7.44, 7.45, 7.46, 7.47, 7.48, 7.49, 7.50, 7.51, 7.52, 7.53, 7.54, 7.55, 7.56, and 7.57 illustrate the approach in the autopsy to the lymph nodes, spleen, thymus, and bones, and illustrate many of the pathological processes that are commonly found in these tissues.

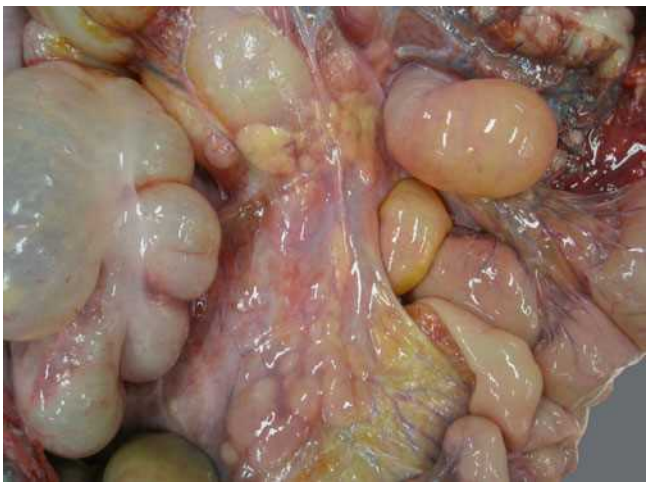

**Fig. 7.1** If there is relative paucity of fat, then lymphadenopathy is relatively easy to identify. The lymph nodes in this view of the mesentery appear to be of variable size and clustered within the mesentery support tissues. Palpation of the mesentery should take place during resection of the bowel tissues at evisceration, to avoid missing adenopathy in individuals with adipose-rich mesentery

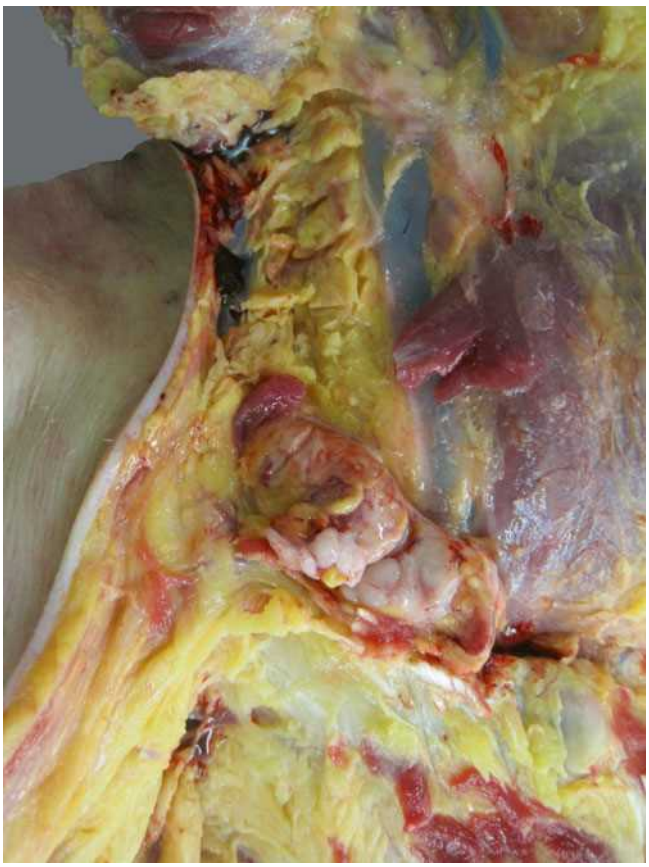

**Fig. 7.3** Lymphadenopathy in the neck requires histology. It can reflect not only sepsis (*e.g.*, mycobacterial infections) but also thoracic, cranial, or distal metastatic neoplasia and primary lymphomas

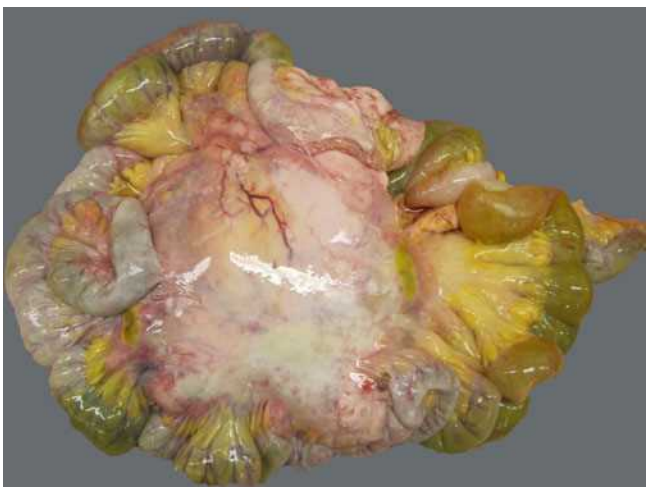

**Fig. 7.2** Lymphadenopathy can be seen within the abdomen with enlarged nodes, occasionally merging into one large tumour mass, in the mesentery. Such cases often reflect lymphomatous neoplasia

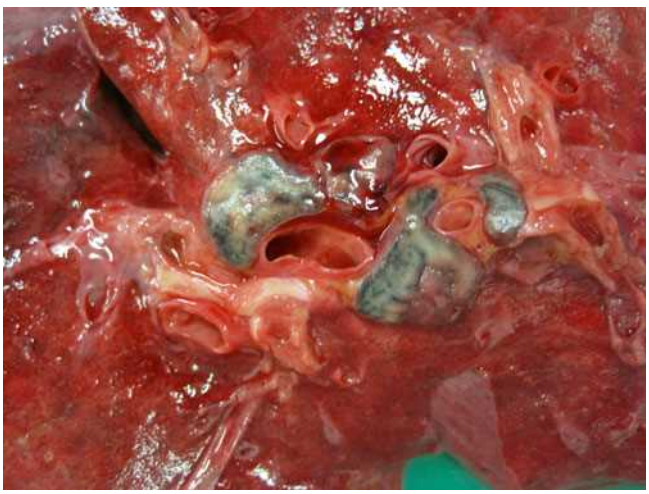

**Fig. 7.4** The lymph nodes within the chest are often pigmented owing to inhaled particulates. Consequently, lymph nodes are often *grey* and *black* in colour. Variegation of the nodes, as seen in this image, does not automatically imply pathology, but any large nodes should be sampled for histology

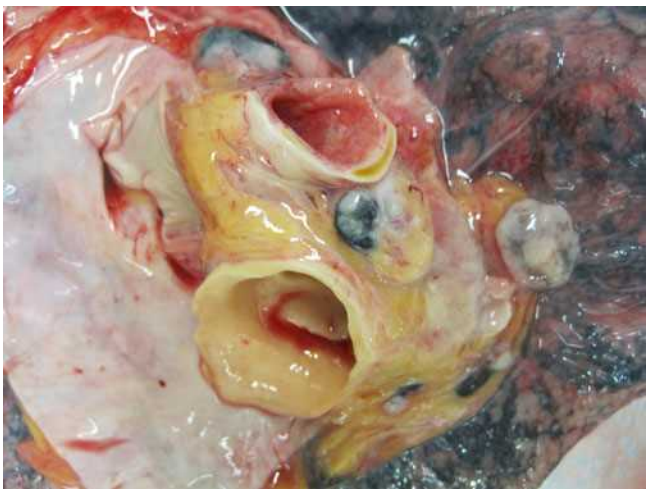

**Fig. 7.5** Lymphadenopathy within the neck may represent either thoracic disease or head and neck cancers. Some enlarged and partly necrotic nodes containing tumour are seen at the base of the neck structures immediately adjacent to the internal jugular vein and sternomastoid muscle

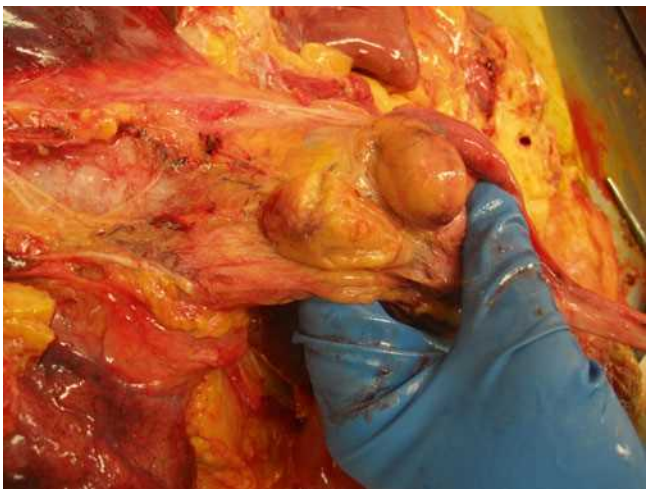

**Fig. 7.6** Even single enlarged nodes in the body merit histology to consider the possibility of neoplasia and/or inflammatory conditions

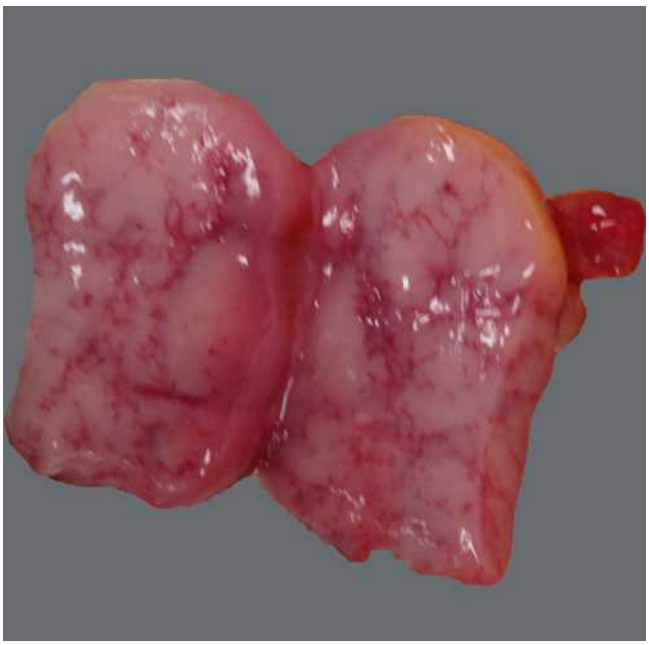

**Fig. 7.7** The cut section of an enlarged node gives a clue as to the pathology. Carcinomas are often fibrous or sclerotic, whereas soft, fleshy nodes (such as this) often point towards lymphomatous disease. Rapid assessment can be undertaken by frozen section or imprint cytology, even at autopsy

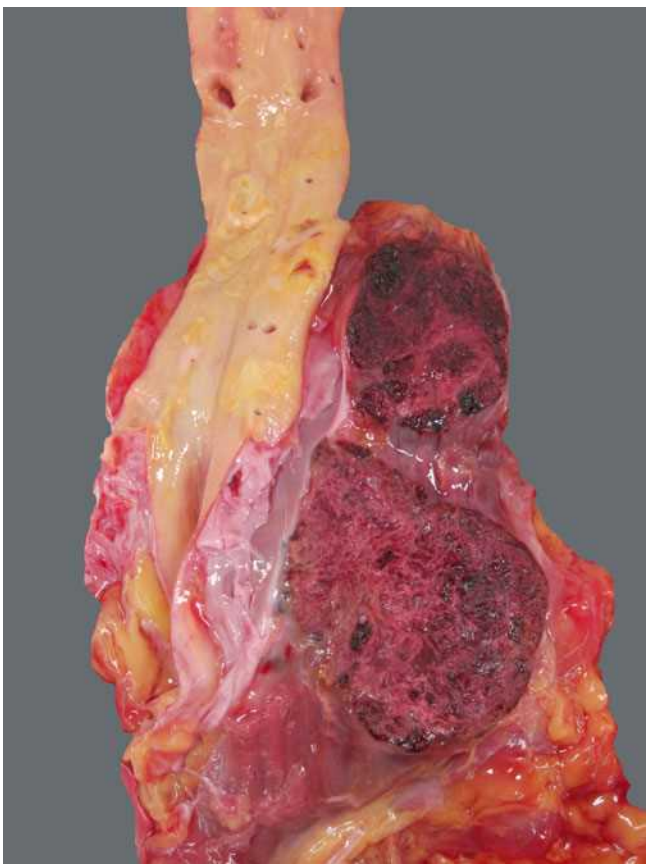

**Fig. 7.8** Mediastinal nodal involvement often can occur with metastases. This case of choriocarcinoma was noted to widely infiltrate the soft tissues around the aorta and to compress local lung and cardiac tissues. Note the haemorrhagic and fleshy quality, akin to placental tissue

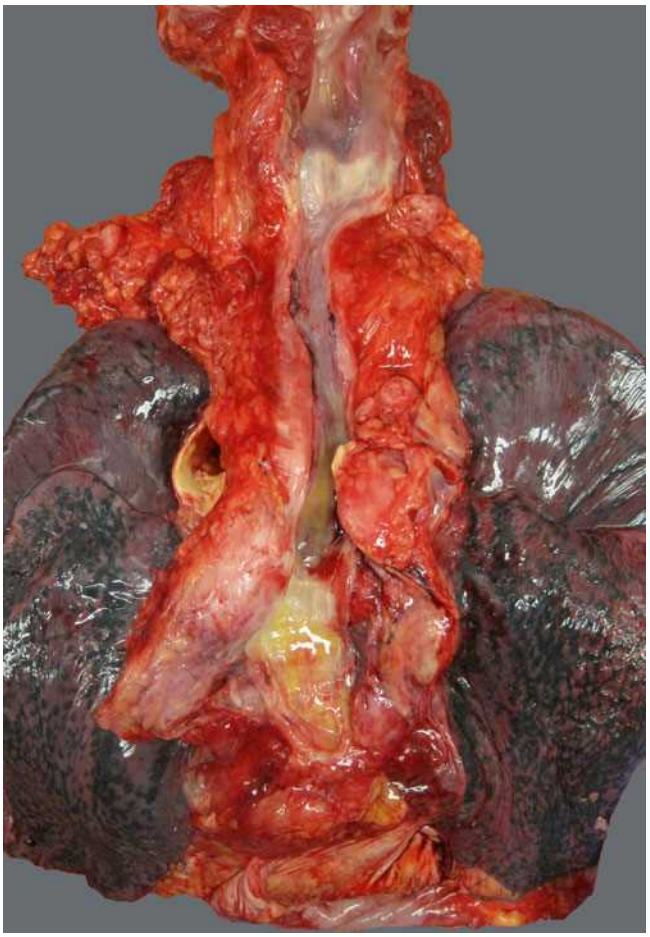

**Fig. 7.9** Multiple-site thoracic lymphadenopathy may be a feature of inflammatory conditions (*e.g.*, sarcoid or mycobacterial infection), apart from lymphomas. The final interpretation requires histology and microbiology

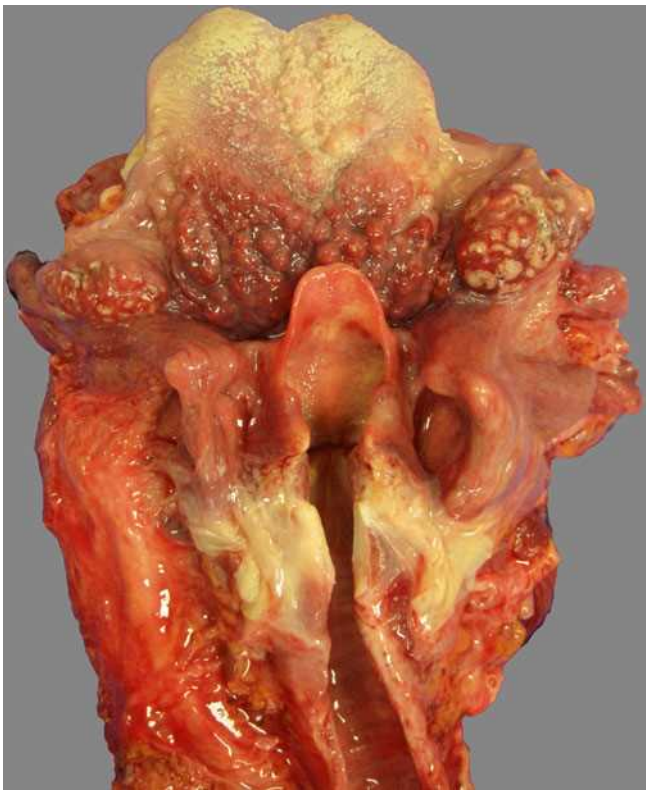

**Fig. 7.10** Tonsillitis is a rare pathology in the autopsy room. Most cases reflect sepsis, but lymphomas must be considered

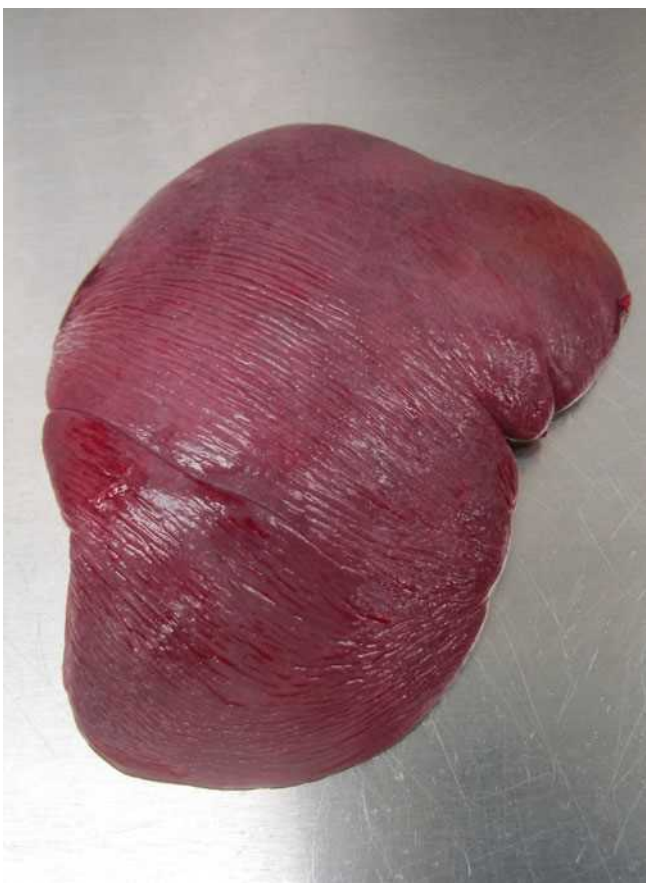

**Fig. 7.11** The normal spleen (100–150 g) shows a characteristic slightly wrinkled capsular surface and abuts the tail of the pancreas. It lies adjacent to the stomach and under the rib cage

**Fig. 7.12** The cut surface of the spleen shows a characteristic mottled architecture with different-coloured elements. These are described as white pulp and red pulp, with the white elements representing periarterial lymphoid sheaths and connective tissues, which are active in immune states. The *red* pulp tissues are sinusoids in which effete circulating blood cells are removed along with other extraneous matter. The *low right inset* shows a high magnification of the spleen

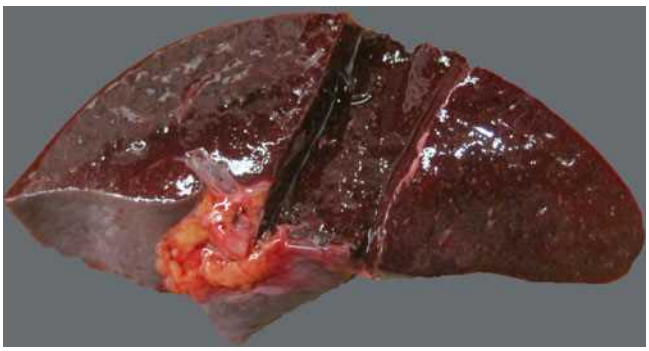

**Fig. 7.13** When considering tissues to be sampled for genetic studies, the high density of lymphoid elements within the spleen favours the spleen as a common source of DNA. It is recommended that a 1- to 2-cm portion of tissue be removed from the spleen (as indicated in this postsampling image) and placed in a sterile container. The DNA can be extracted immediately, or the sample may be frozen for subsequent DNA analysis

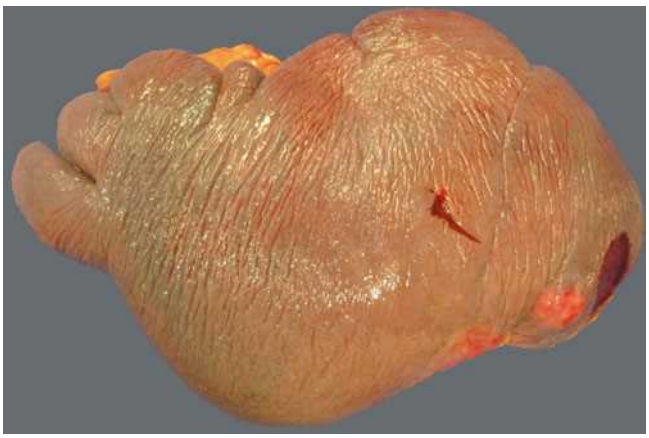

**Fig. 7.14** Small tears of the spleen capsule often occur during evisceration and handling. These artefacts should not be construed as significant unless local bleeding is apparent, in which case they reflect ante-mortem problems

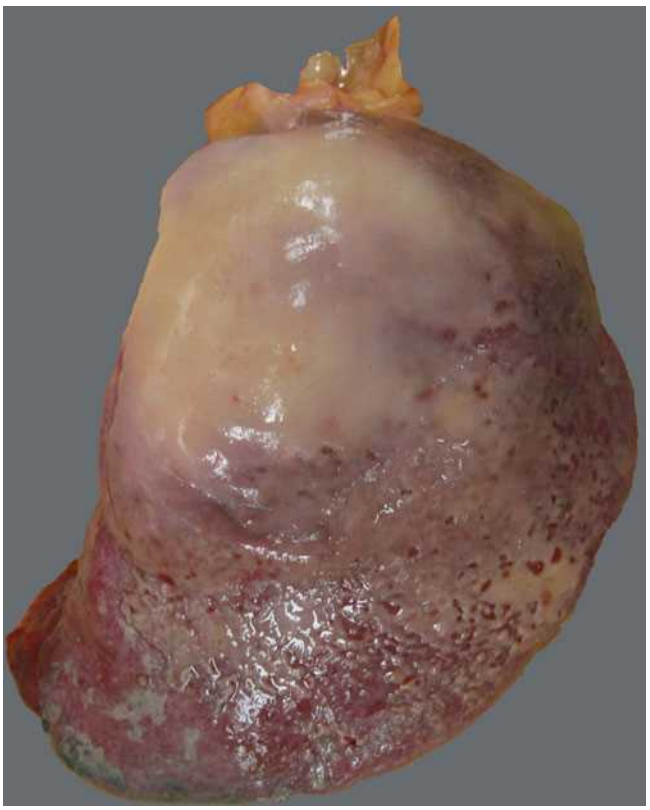

**Fig. 7.15** A common feature of splenic tissues is a surface fibrinous exudate (generally described as 'sugar-icing') that is organized focally or entirely around the spleen tissues. It is of no pathological consequence

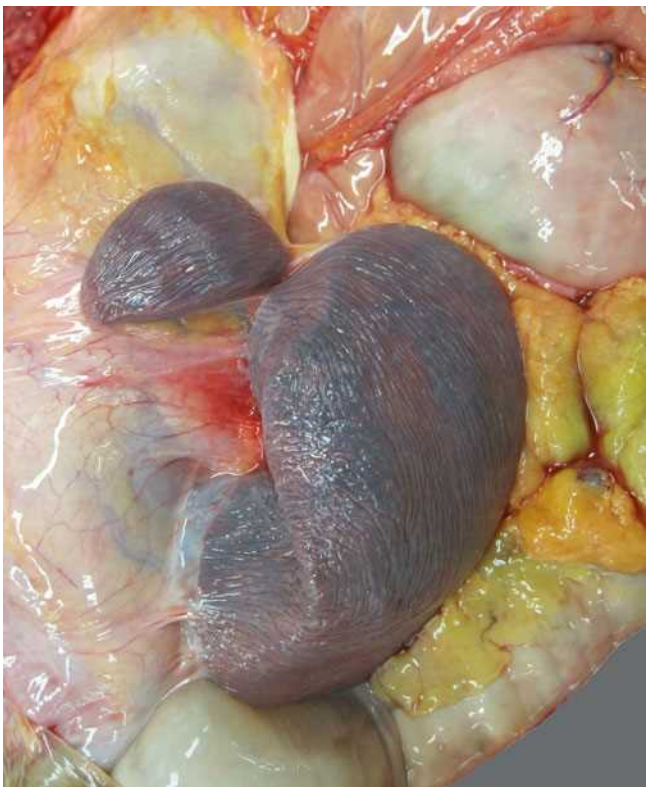

**Fig. 7.16** The spleen often has a small focus of similar spleen tissue adjacent. The splenunculus can vary in size and has exactly the same morphology and function as normal spleen tissues

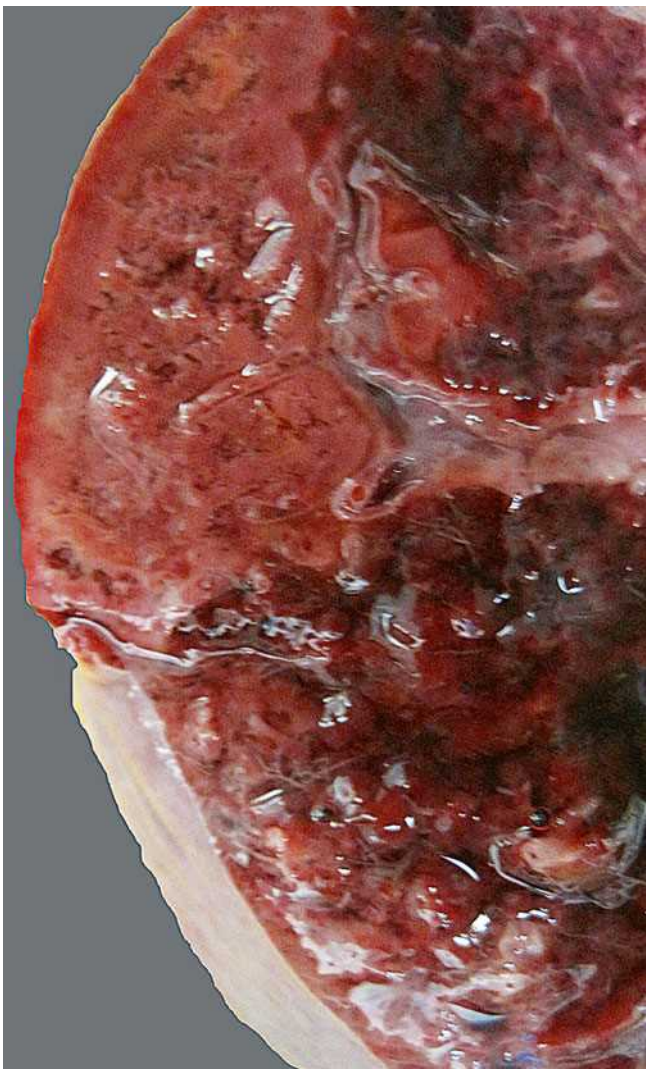

**Fig. 7.17** This close-up of the edge of a spleen shows a wedge-shaped splenic infarct, representing an embolus from a case of infective endocarditis

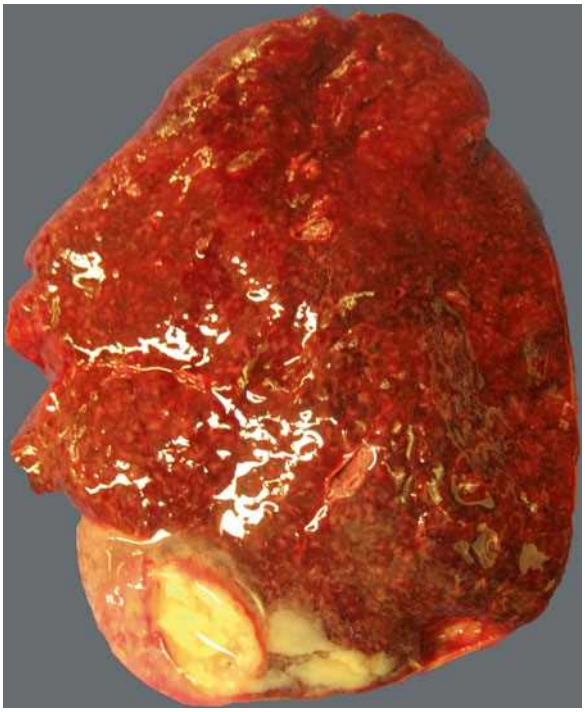

**Fig. 7.18** Splenic tissues can be involved by neoplasia. The commonest are tumours of the haemopoietic system (leukaemia or lymphoma), although metastatic lung cancer (as in this case) also can be found in splenic tissues

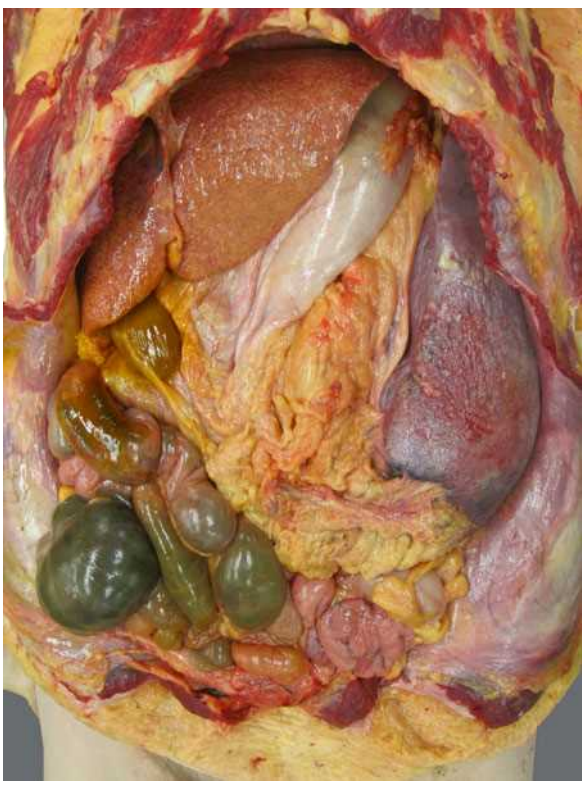

**Fig. 7.19** Gross splenic enlargement may be an unsuspected finding at autopsy. In this case, the splenic enlargement was due to chronic lymphocytic leukaemia. One should always be aware of the possibility of other malignant infiltrates, and one should also consider leishmaniasis in those coming from abroad

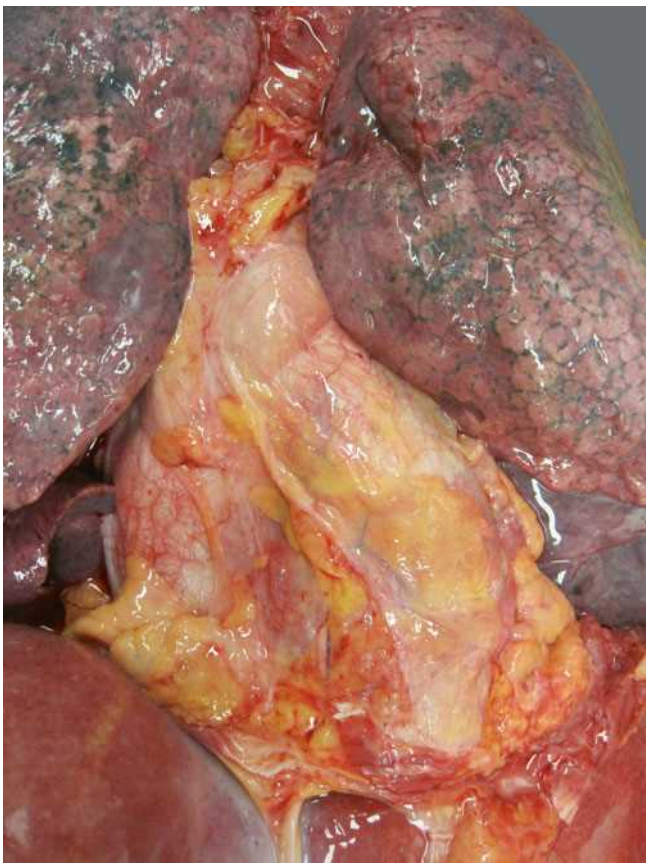

**Fig. 7.20** The position of the normal thymus may be seen central and high within the mediastinum of this 20-year-old man. There is a clear 'bulge' where the tissue lies

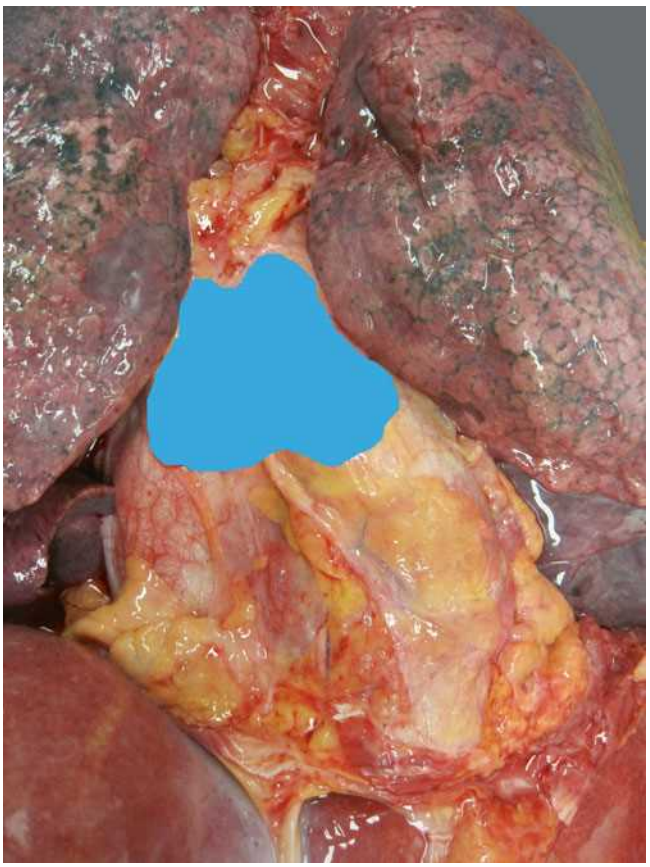

**Fig. 7.21** The same view as Fig. 7.20 is shown with the position and shape of the thymus marked in *blue*

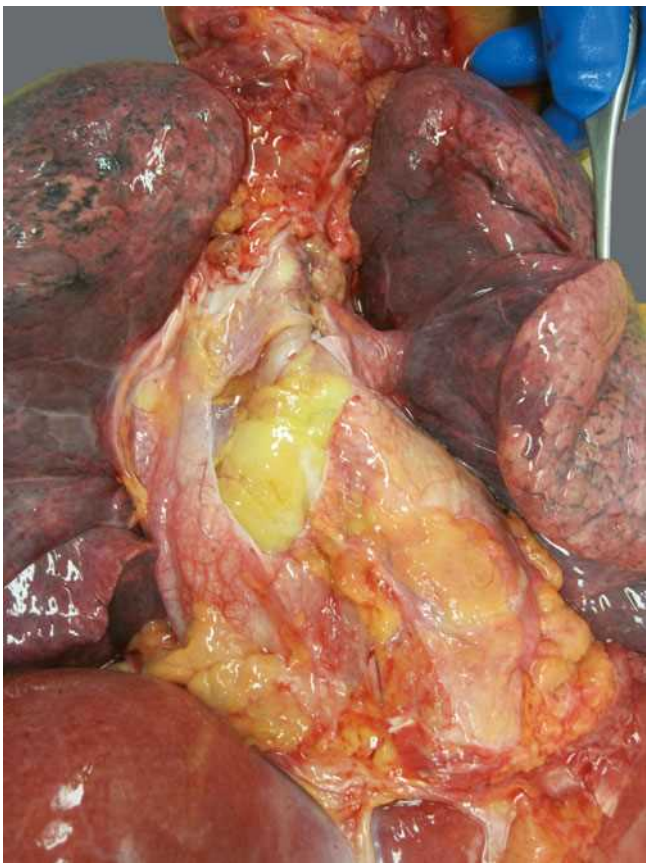

**Fig. 7.22** The upper mediastinal tissue is shown with the thymus now resected

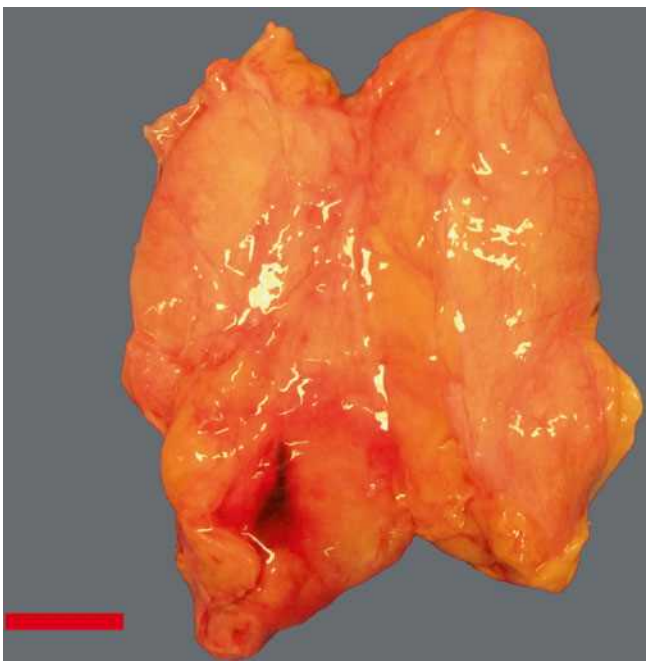

**Fig. 7.23** The resected thymus is seen from the anterior aspect

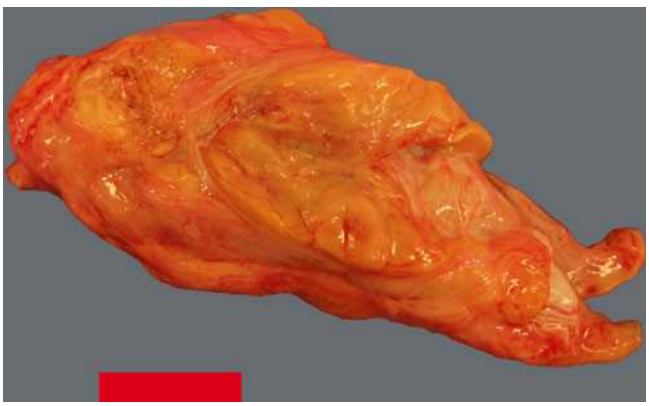

**Fig. 7.24** The thymic cut surface is displayed

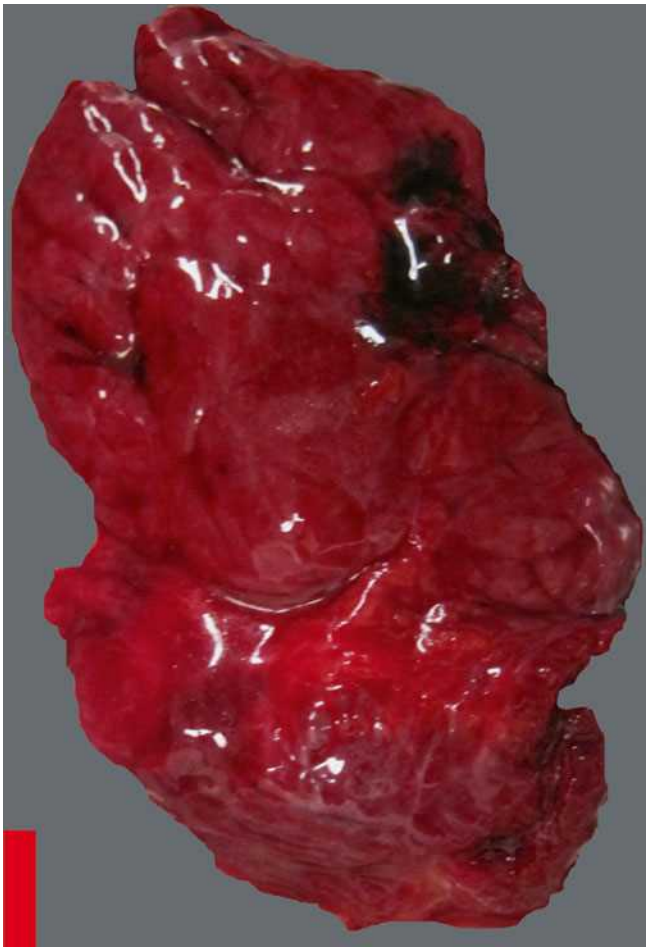

**Fig. 7.25** The thymus of a teenager often appears more congested and has less fat than that of an elderly person. The appearance does not automatically imply disease, but softening of the tissue may point towards inflammatory or septic processes

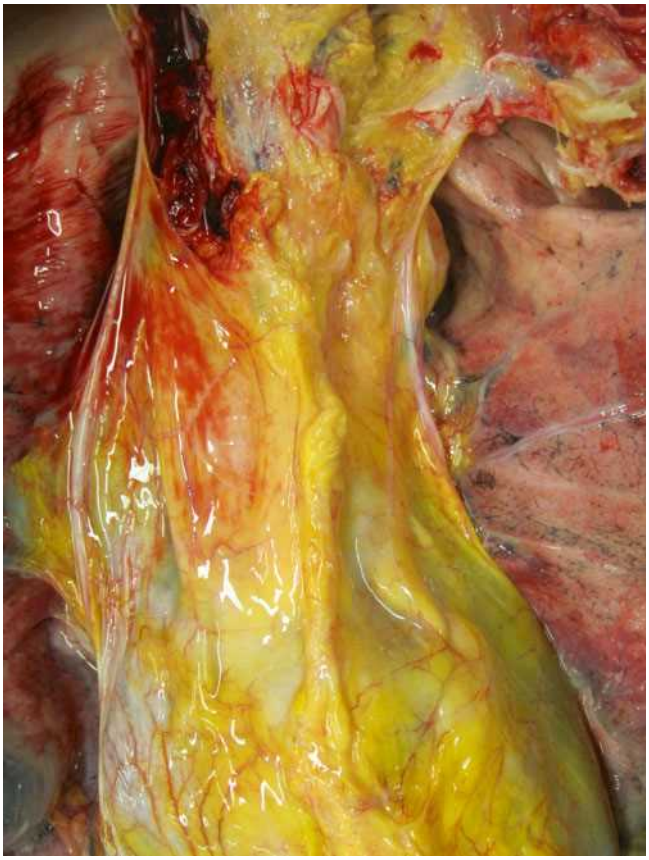

**Fig. 7.26** The atrophic thymus in the elderly is more difficult to identify within the mediastinal soft tissues. This image shows fatty tissue over the front of the heart and upper mediastinum without a clearly defined thymus being evident. Nevertheless, if one sections in the central part of the tissue, atrophic elements of thymic tissue will be identified. There is some haemorrhage towards the upper right side of the mediastinum in this case, in keeping with placement of an intravenous central line

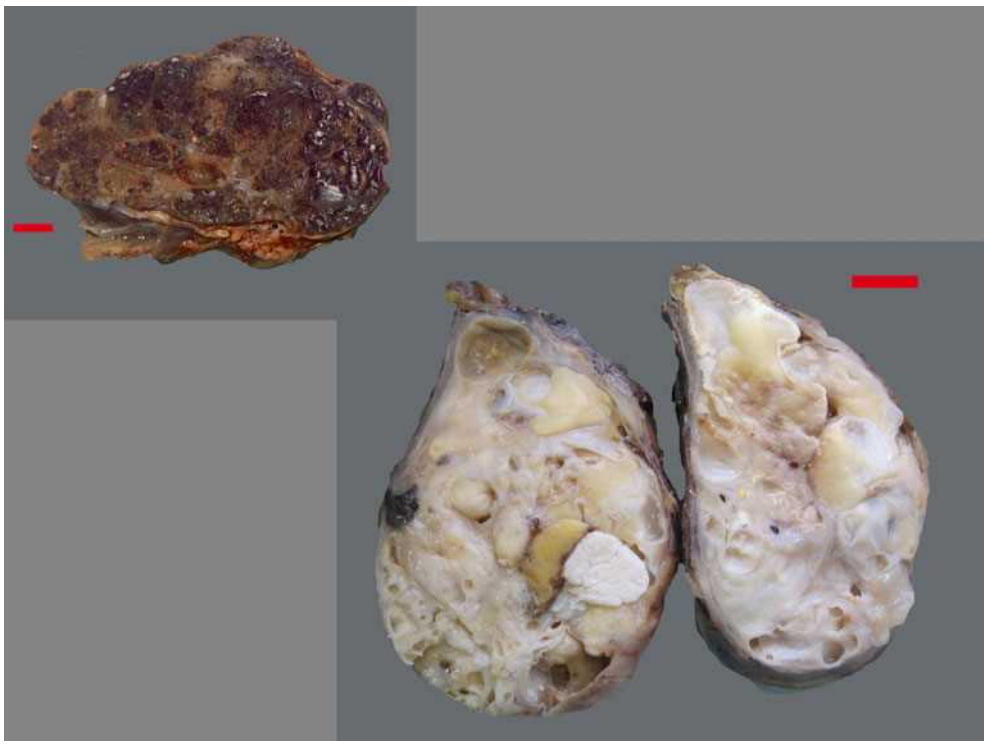

**Fig. 7.27** *Upper left*, This multicystic and focally haemorrhagic mass within the upper mediastinum comprised retrosternal thyroid tissue of benign format. Increasing heterotopic thyroid tissue enlargement can cause pressure on local structures, much as thymic and other neoplasia. *Lower right*, Another cystic thymic mass is seen in this surgical sample. This case had cysts with different epithelia, muscle, and cartilage. The features were those of a mature teratoma with no malignant features

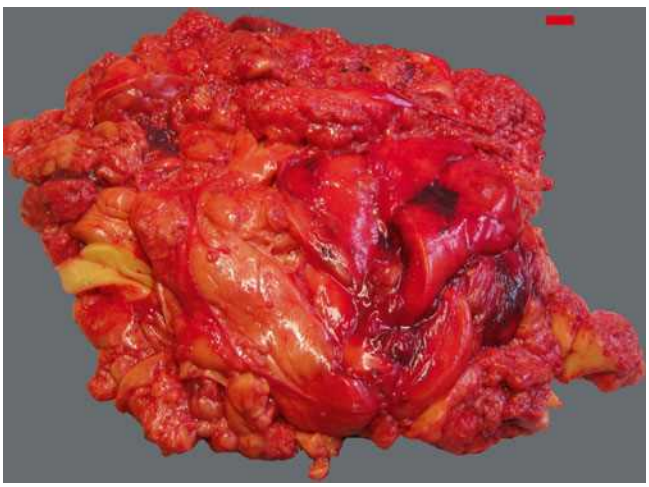

**Fig. 7.28** This large, fatty mass was surgically resected from the anterior mediastinum of an elderly individual. Histologically it was a liposarcoma. Soft-tissue neoplasia in the mediastinum is rare, but it is important to understand that not all anterior mediastinal tumours are thymomas or lymphomas

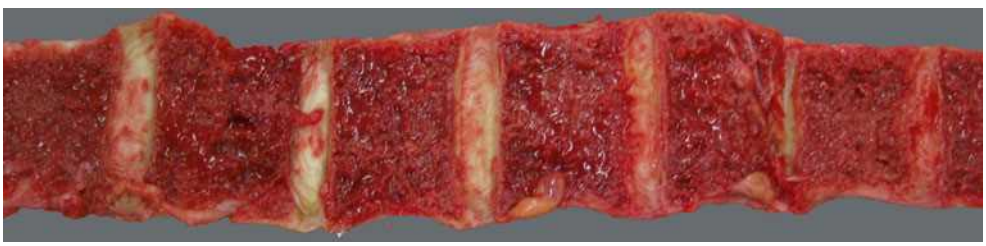

**Fig. 7.29** In an autopsy case, the normal vertebral bone must be seen both as the external tissue architecture (see Chap. 3) and the internal structure, which is more important. Here the normal bone cortex is seen with red medullary tissues. There is no fracture or tumour

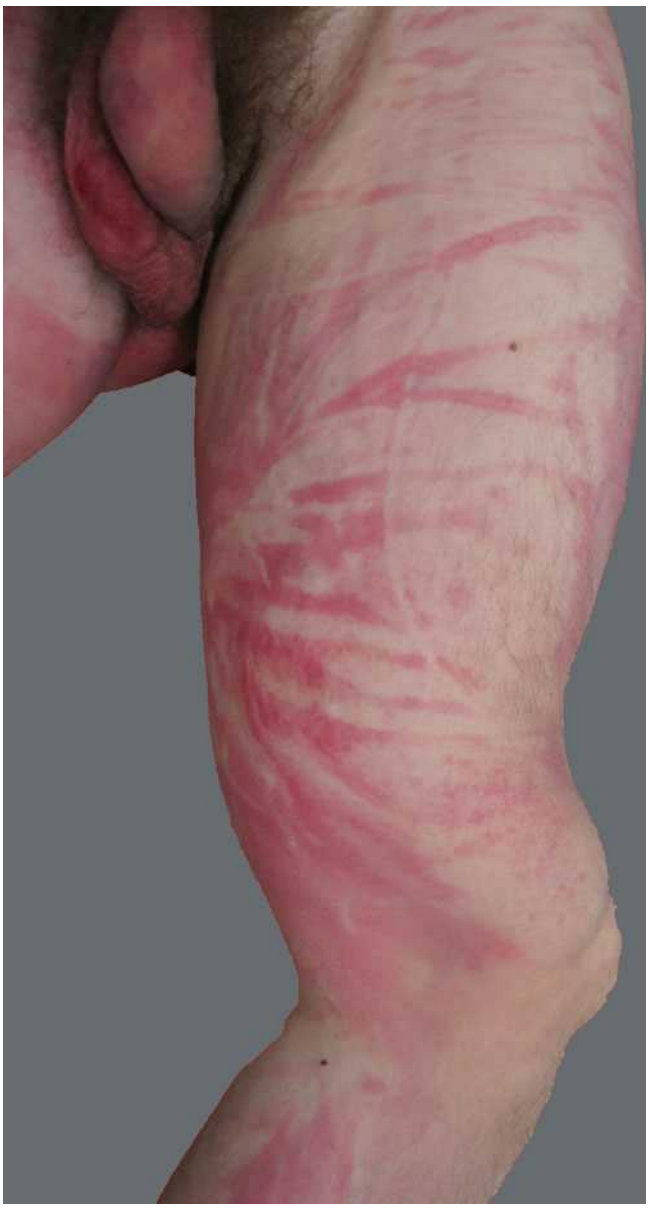

**Fig. 7.30** It is not necessary to incise joints or limbs if bony fractures are clearly evident. The gross distortion of this left thigh points towards a severe acute fracture—in this case related to a road traffic collision. Ante-mortem radiology, if available, may augment understanding of any bone fractures, and post-mortem CT scans (see Chap. 13) are also valuable

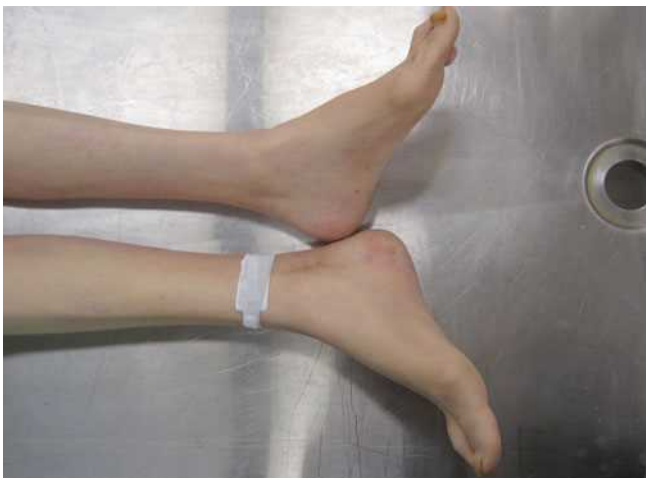

**Fig. 7.31** Fractures often can be easily interpreted externally. In this case, the external rotation and shortening of the left leg points toward hip fracture

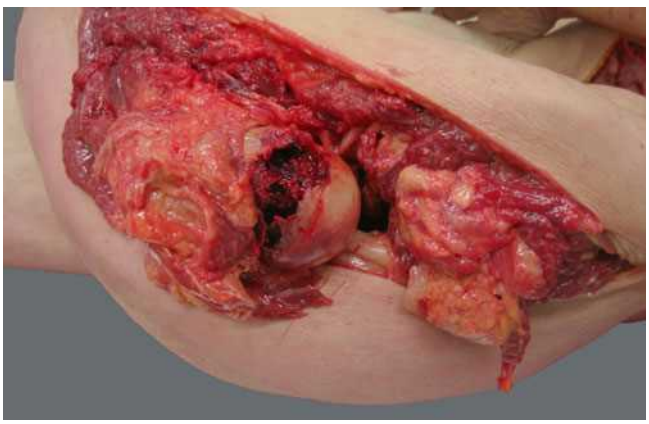

**Fig. 7.32** Demonstration of hip fractures, particularly those involving the neck of the femur, can be complicated. A wide incision has been made over the left hip lateral aspect, with the thigh folded toward the right side. Dissection down onto the articular tissues should allow the hip joint to be 'popped out'. Any fracture should be easily discernible, and samples may be taken if required. Photography is all that is needed for documentation in most cases

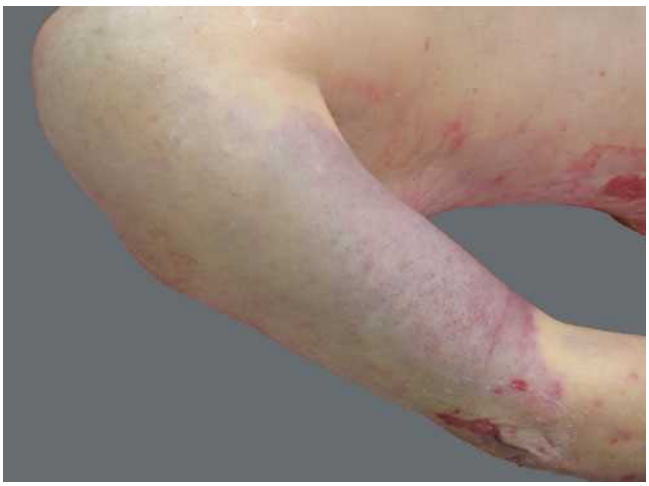

**Fig. 7.33** Some bone fractures are suggested by the presence of significant haemorrhage around the site of fracture. In this case, a subcondylar fracture of the humerus was present and had been noted on ante-mortem radiology. The pronounced haemorrhage in the soft tissues of the upper arm points towards significant bone trauma. In such cases, it could be argued that dissection of the fracture site does not advance case analysis

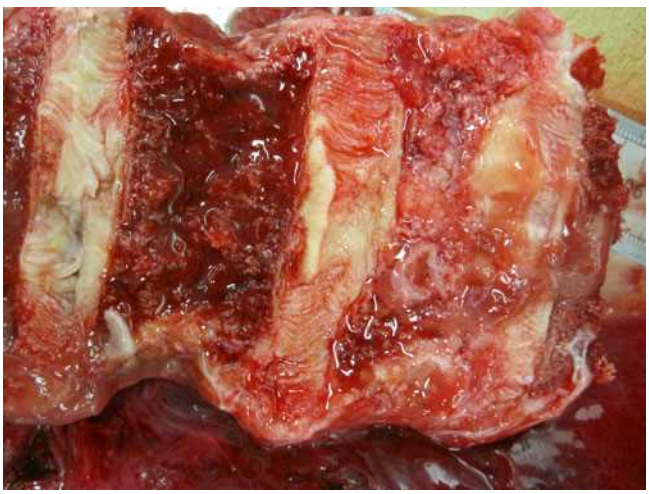

**Fig. 7.34** Examination of the cut surface of the bony tissues from the spine may show variable areas of distal distortion and bony collapse, often due to osteoporosis. The beige/grey tissue present around the site of fracture and collapse of one vertebral body—chronic features of osteoporotic fractures—may also point to the possibility of metastatic tumour at this site. Histology will be required to confirm the nature of the fracture

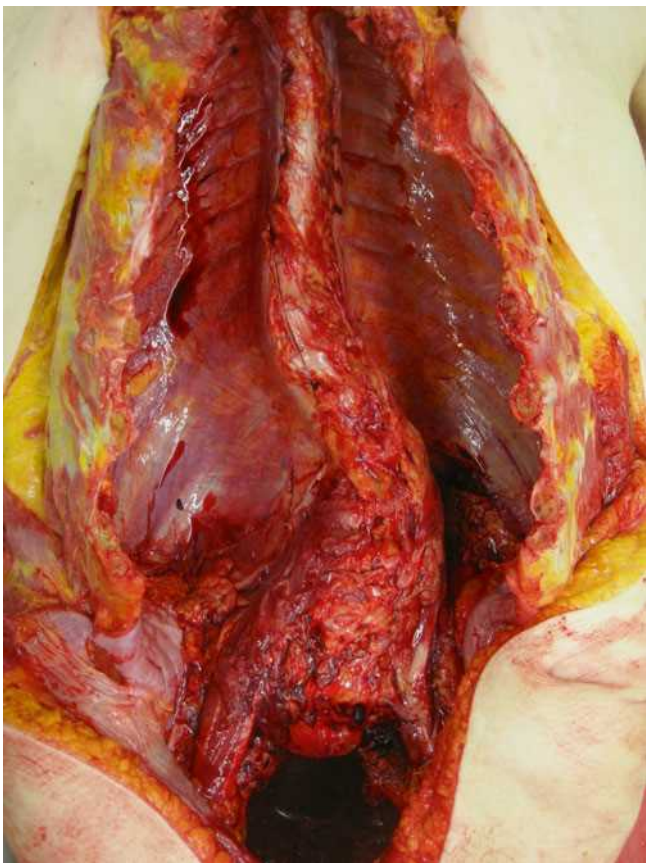

**Fig. 7.35** Severe kyphoscoliosis should always be recorded. It has significant bearing upon thoracic function and may be pertinent to the cause of death

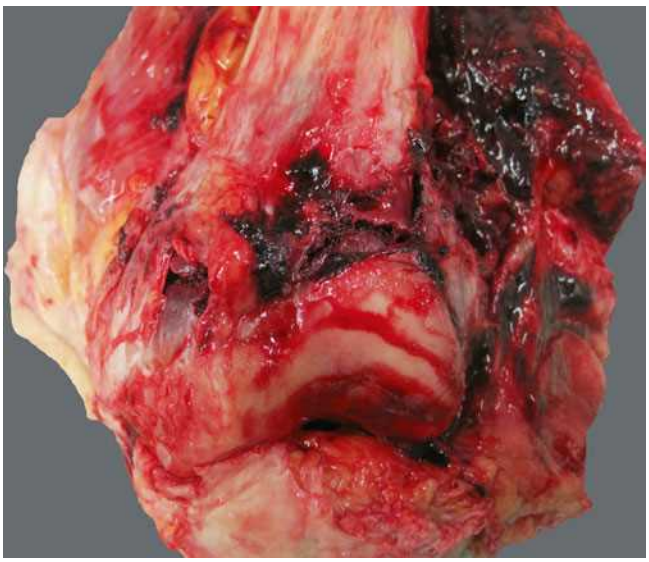

**Fig. 7.36** The bony tissues of long bones are commonly involved by fractures, particularly in the elderly with a background of osteoporosis and falls. This fracture ran across the distal femur. It is notable that considerable haemorrhage was incurred around the fracture site, adding to the debility and pain experienced ante-mortem

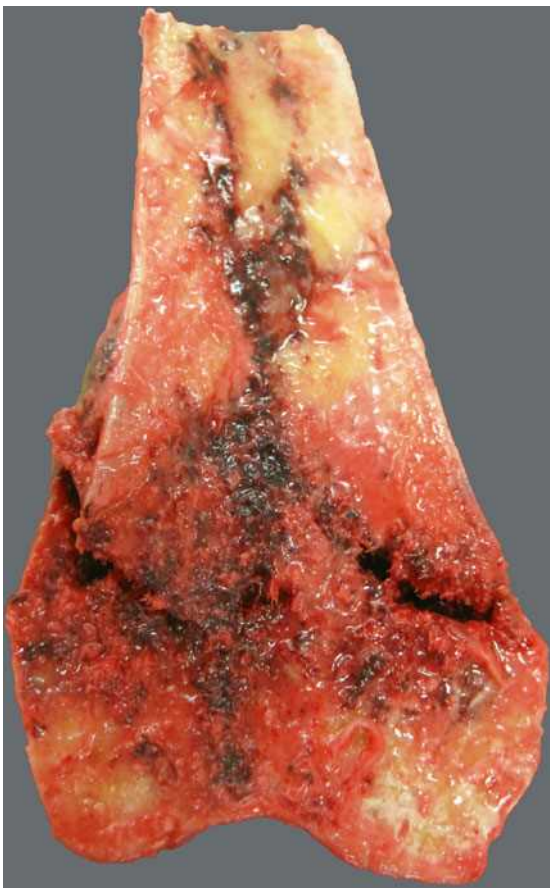

**Fig. 7.37** The same fracture is now seen, having being partially excised with a cut running in a coronal plane along the long axis of the bone. This view clearly demonstrates the fracture site and local bony tissues. Performing this task allows targeted sampling, fixation, decalcification, and then appropriate block sampling

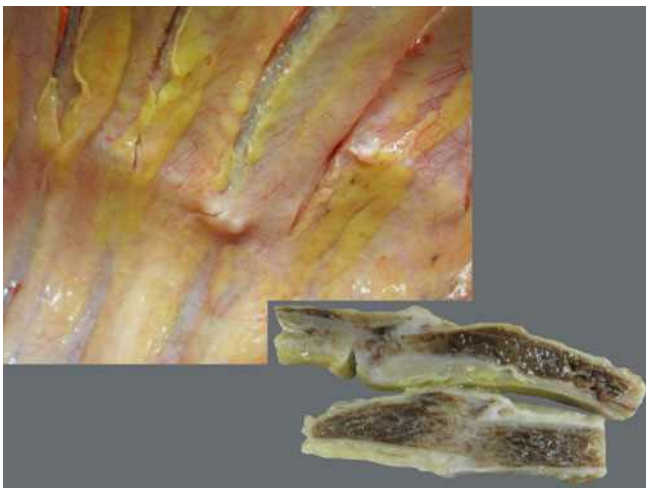

**Fig. 7.38** Fractures of the ribs are common. In this case, there is a multi-step deformity of the inner aspect of the chest wall, with focal reparative cartilaginous tissue being seen clearly on the pleural aspect. This patient had suffered rib fractures some 6 weeks before death, with poor alignment of the broken ends of the bone. The *lower right* image shows the bone tissues from the rib fractures on either side of the fracture site. In this case, fixation and then decalcification has allowed longitudinal slicing through the bone tissues to demonstrate the poorly aligned fusion of the bone tissues and some pseudoarthrosis

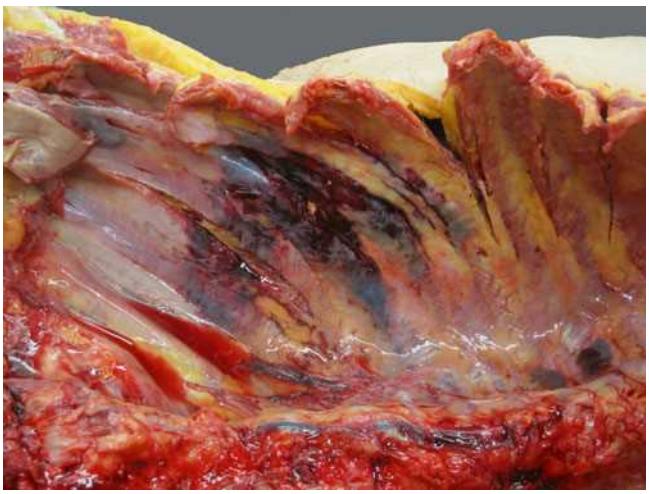

**Fig. 7.39** Significant injuries to the chest often incur rib fractures with local haemorrhage. In such cases, one of the most important techniques is slicing between the ribs to allow the fracture sites to be explored and to define the amount of blood lost

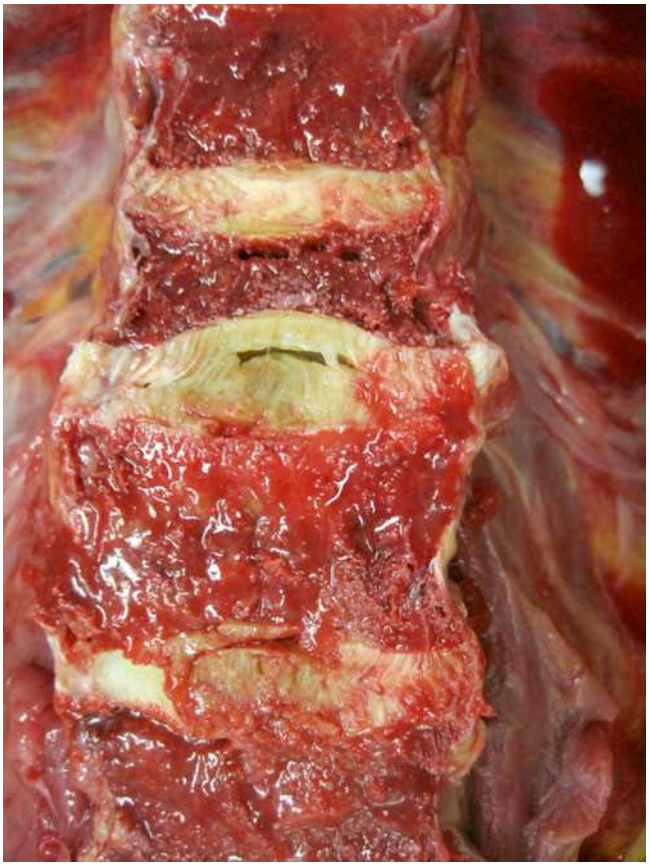

**Fig. 7.40** Osteoporosis is characteristically associated with multiple fractures and collapse of the vertebral column bones. The different sizes of the vertebral bodies seen in longitudinal sections assists in confirming this interpretation. Palpation of the cut surface of relatively normal vertebral bodies should allow assessment of osteoporosis. In these cases, the thumb can be easily pressed into the bone tissue surface, a maneuver that is not possible in the bones of the young, where a normal bone framework exists

**Fig. 7.41** This longitudinal section through the vertebral bodies shows multiple foci of tumour (disseminated lung cancer) involving the vertebral bony tissues

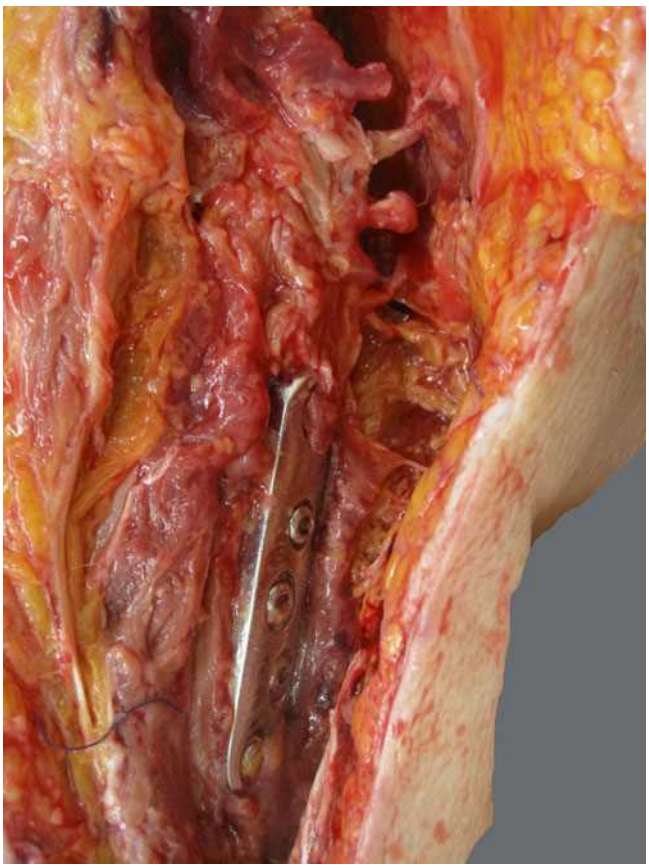

**Fig. 7.42** Bony tissues with fractures often are repaired with metal devices being left in situ. These are rarely of consequence later, but there is the potential for sepsis, so some examination of the tissues may be required. In this case, repair of the femur with a screw and plate was undertaken with good effect. There is no sepsis in the local soft tissues

**Fig. 7.43** Confirmation of the identity of a deceased individual, especially if significantly decomposed, may be assisted by checking the registry numbers of prosthetic devices such as hip implants

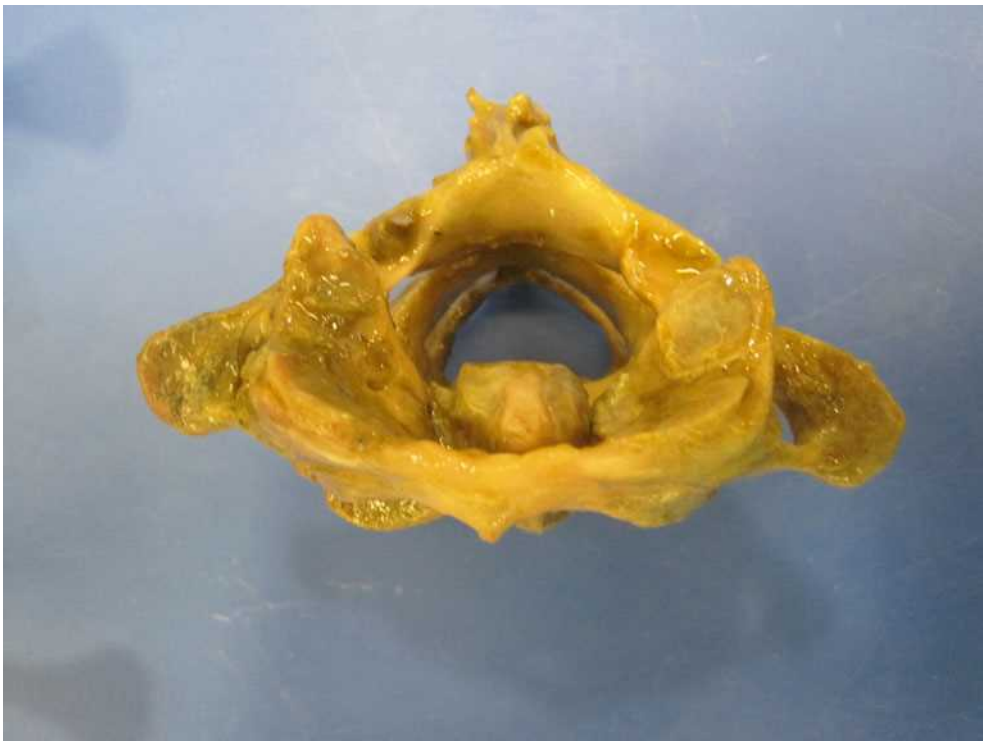

**Fig. 7.44** This is a superior view of the C1, C2, and C3 vertebral bodies from the cranial aspect downwards. The decomposed body in this case allowed full appreciation of the close interaction of C1, C2, and C3 with preservation of the odontoid peg and local bony tissues. The C1, C2, and C3 bony tissues are seen adjacent, with the odontoid peg of C2 projecting immediately behind the anterior ring of the C1 vertebral tissue

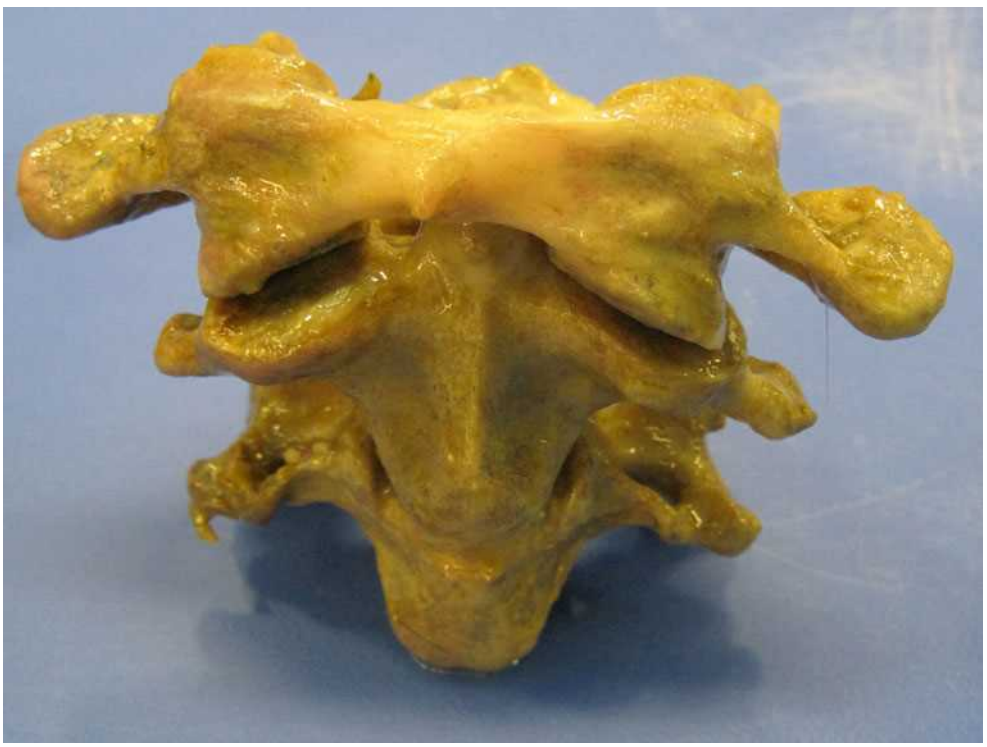

**Fig. 7.45** The same tissue as in Fig. 7.44 is seen from the anterior perspective, highlighting the normal anatomy. This view allows certain pathologies such as spine trauma to be excluded, whether it is achieved by decomposition or by excarnation/defleshing (Chap. 12)

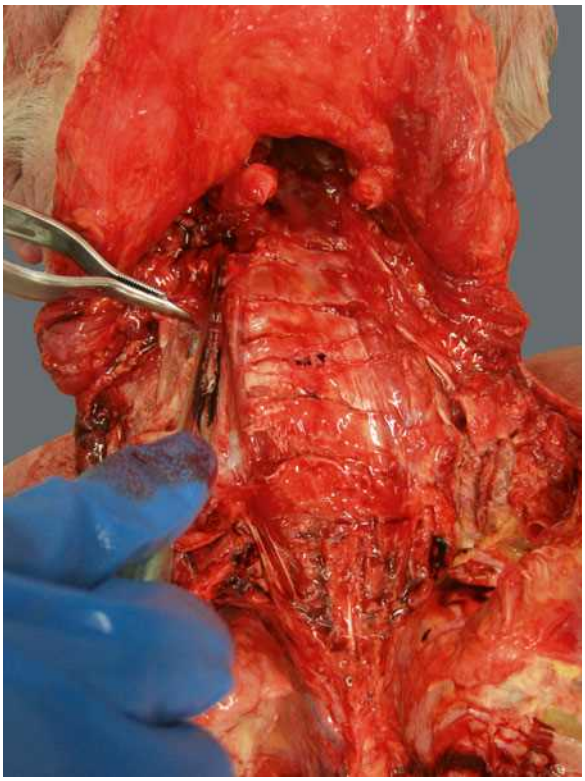

**Fig. 7.46** Assessing the cervical spine in neck injuries is complicated. Good access to the neck is required, with cuts being made into the soft tissues on either side of the cervical bony tissues

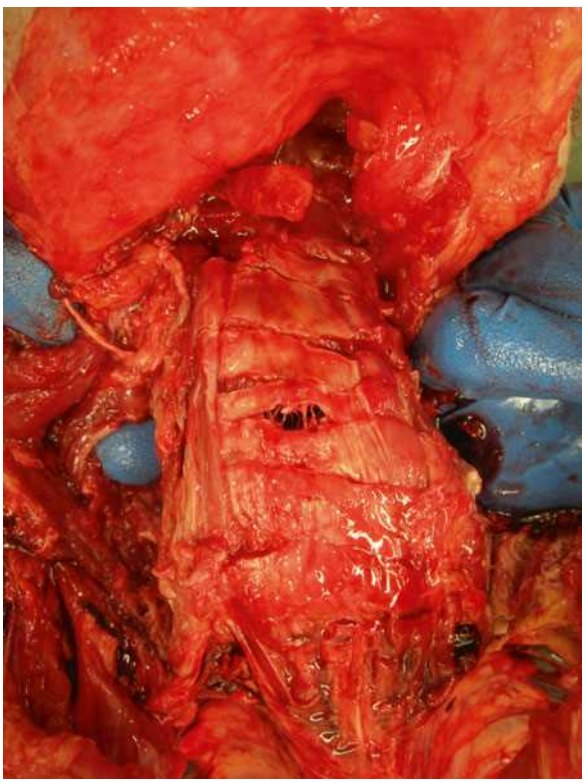

**Fig. 7.47** The tissues are undermined on both sides so that the hand can be passed from one side to the other, confirming that the vertebral bony tissue block is now free of its local soft tissue attachments. At this point, the fracture dislocation site is seen centrally. This pathology was incurred during a fall

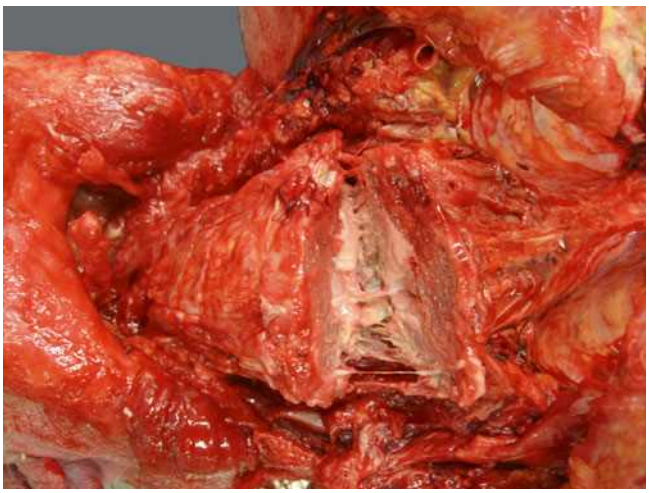

**Fig. 7.48** The lower cervical bony tissues are now cut horizontally using a saw, at least 20 mm below the spinal cord area of significance

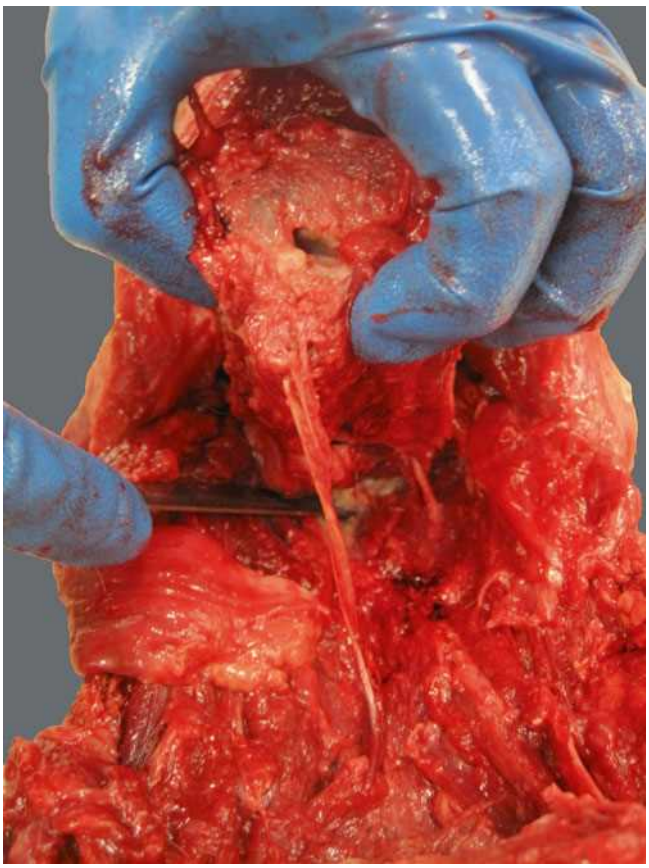

**Fig. 7.49** The base of the spinal vertebral block may now be lifted gently upwards and away from the back of the soft tissues of the neck and skin. The spinal canal can be seen in relation to the vertebral bodies. Soft tissue dissection continues upwards towards the base of the skull

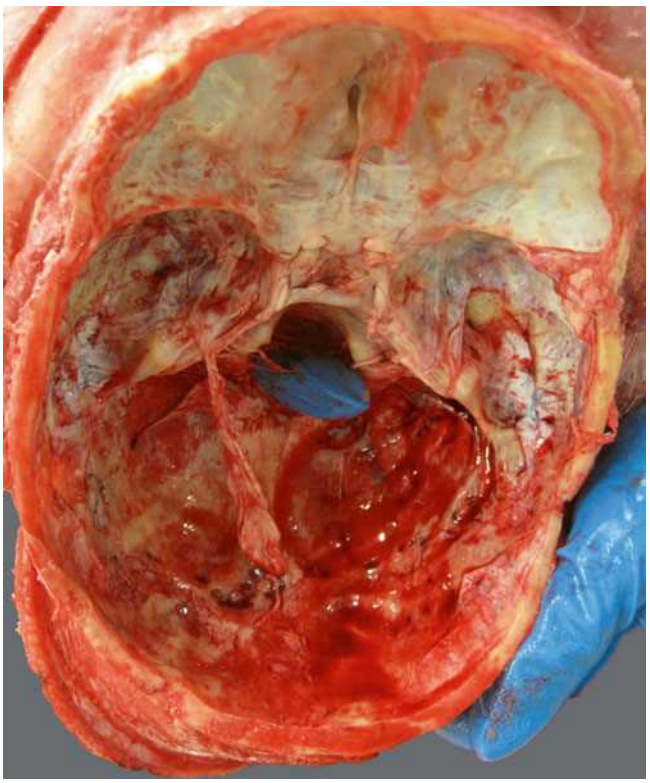

**Fig. 7.50** A set of four cuts are made into the base of the skull around the front of the foramen magnum. There is no way to illustrate this dissection process, as the saw blocks the view, but the resultant hole is shown here. A finger has been passed into the defect to highlight the size of the hole required

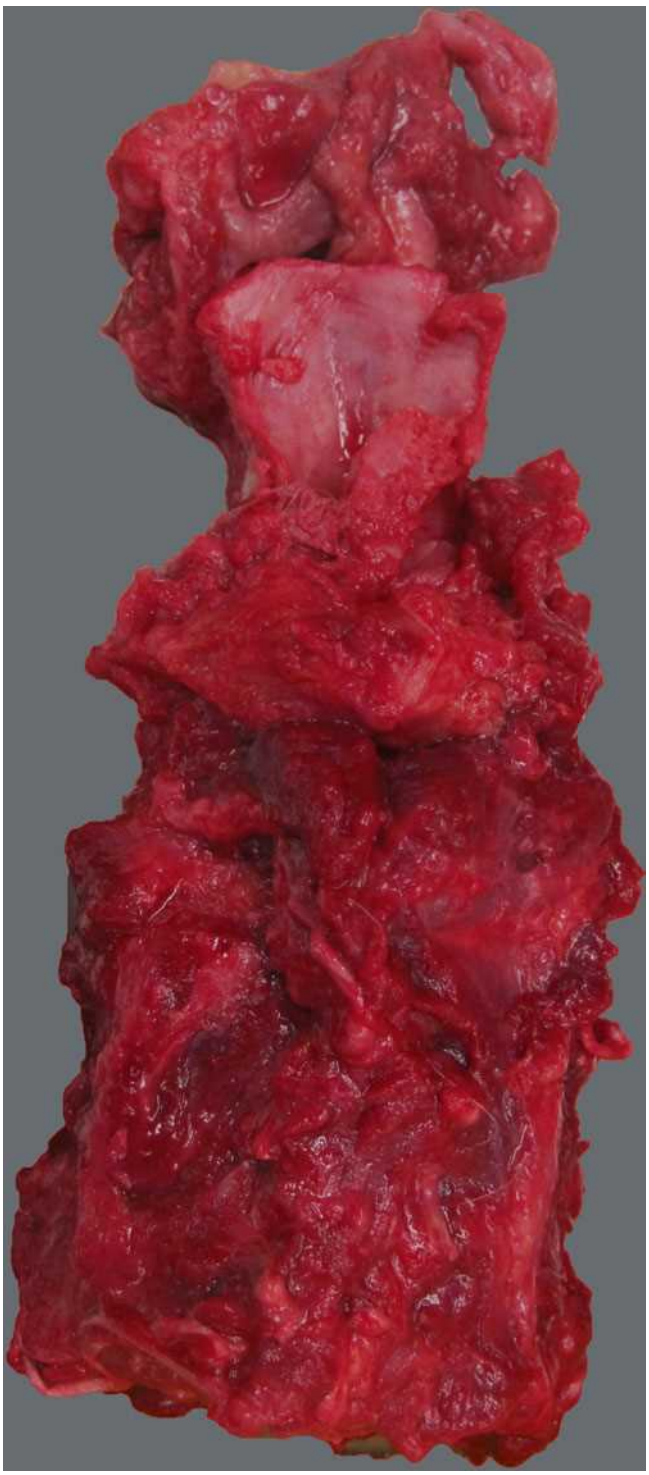

**Fig. 7.51** The tissue block, incorporating soft tissues, basal skull, and vertebral bodies, is seen from the posterior aspect in this view

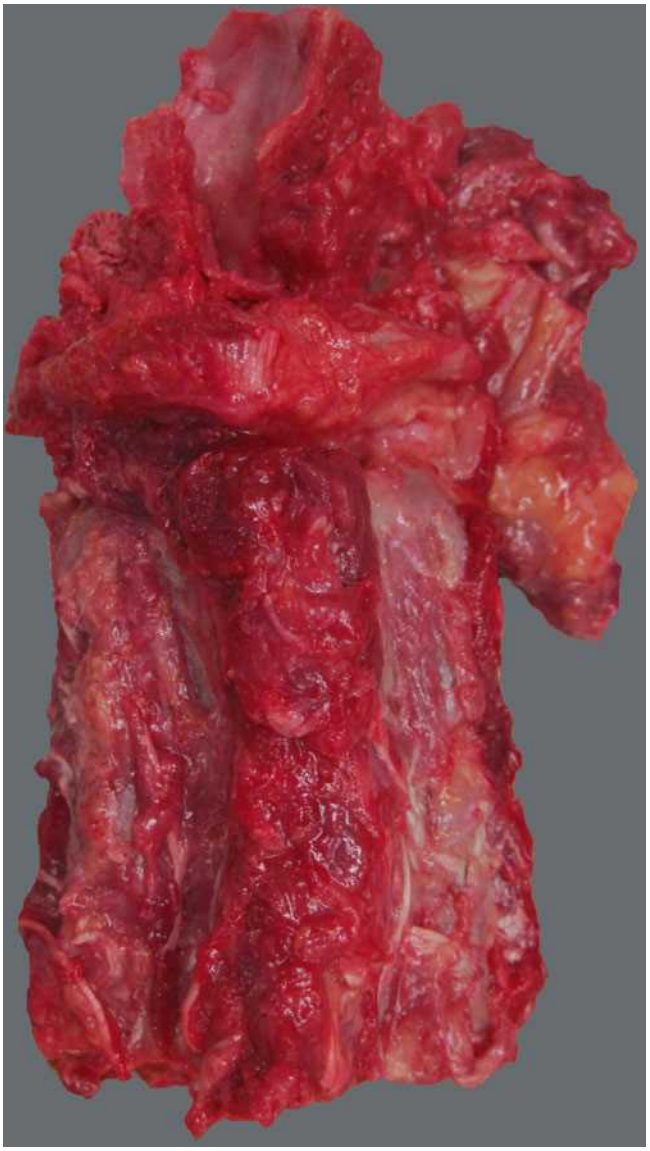

**Fig. 7.52** Further dissection of the soft tissues adjacent to the skull base and the sides of the vertebral bodies continues, with this tissue now being removed. The tissue at the top end is the base of the skull

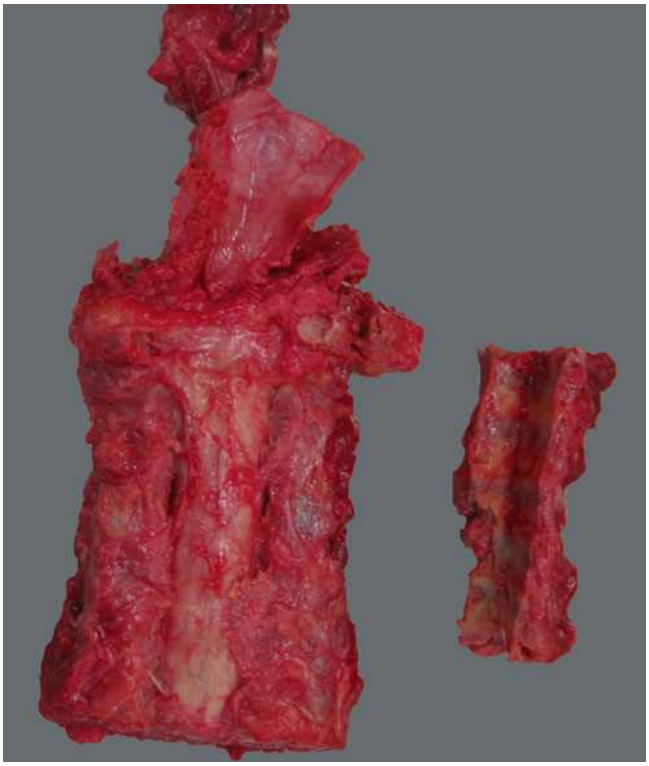

**Fig. 7.53** The back of the spinal canal is exposed by cutting through the bony structures at the back. The spinal cord is seen in situ and the posterior aspect of the spinal canal has been turned around so that one can inspect this aspect of the spinal canal. Any areas of tumour, haemorrhage, or sepsis can be identified readily in this view

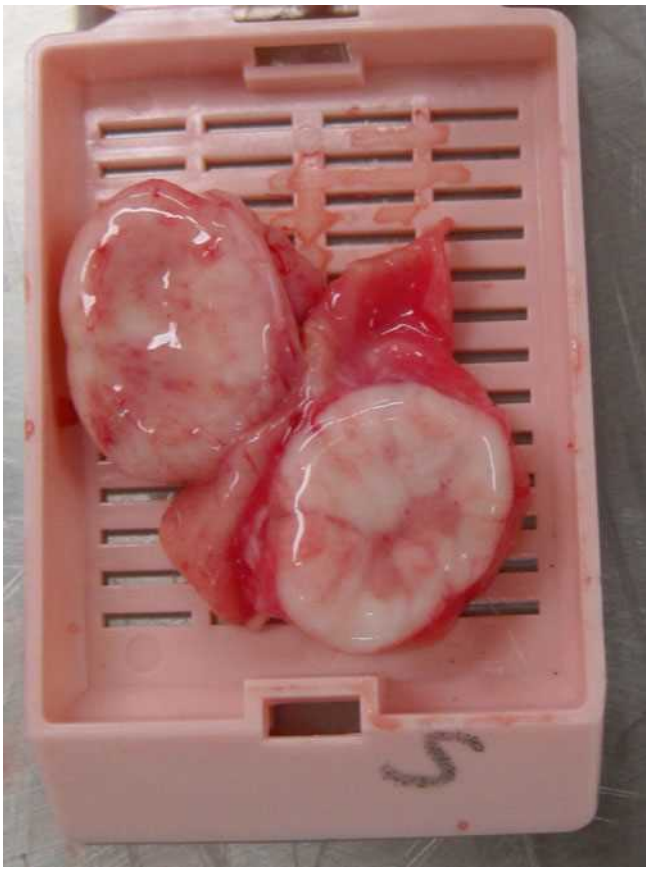

**Fig. 7.54** The spinal cord is removed carefully and without significant traction. Ideally, this segment of spinal cord is left to fix and then sectioned for histology, but if histologically sampling is required immediately, then transverse sections of the spinal cord can be taken, using a fresh scalpel blade to ensure a sharp cut and minimal fresh damage to the soft tissues of the cord. Do not use an old blade!

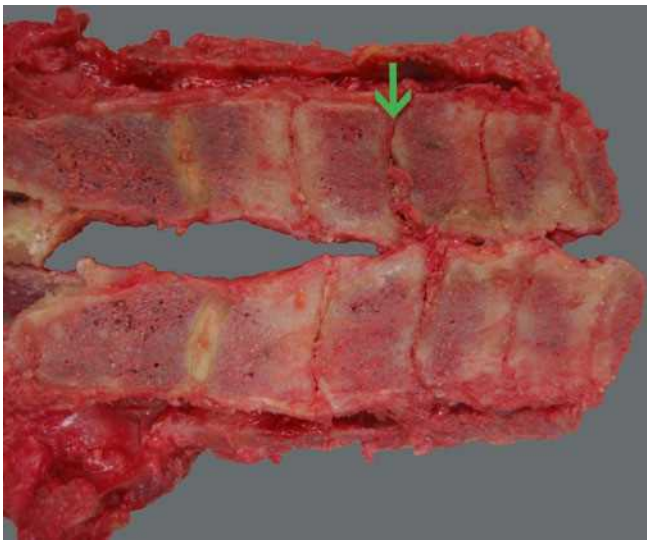

**Fig. 7.55** The bony tissues have now been cut in a sagittal fashion, exposing the alignment of the cervical spine. It is noted that there is some separation at the disc interface (*arrow*) that was responsible for cervical injury. These samples can be reserved for fixation, decalcification, and then histological study if required, although photography alone is usually enough for any medicolegal purposes

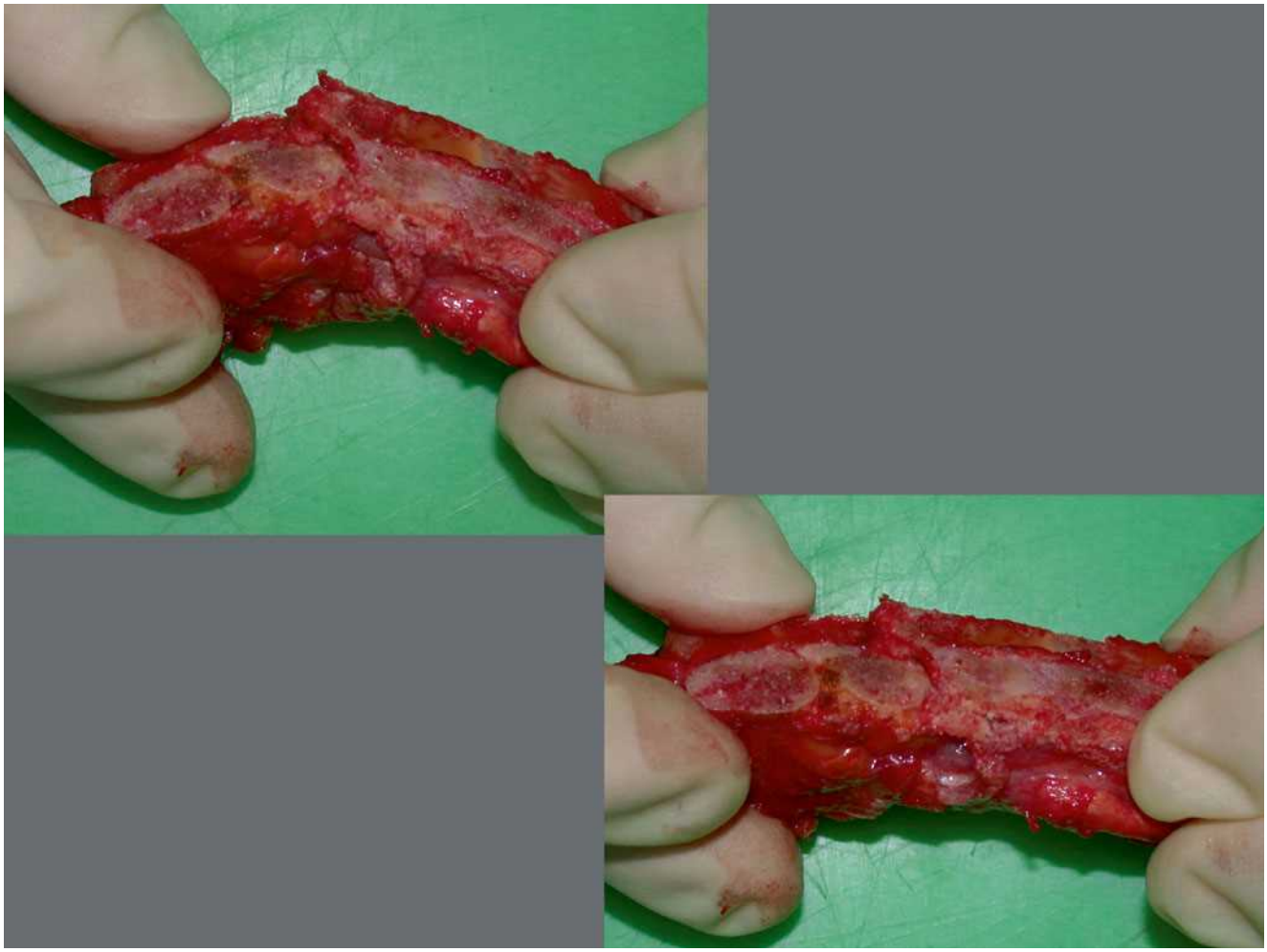

**Fig. 7.56** These two photographs show another case. Here the cervical spine flexion and extension reveals clear separation of the vertebral bodies from each other during this maneuver. This patient had a high-level cervical cord injury associated with a fracture dislocation. Dissection of the vertebral tissues and examination of the different components allowed a full understanding of the injuries sustained

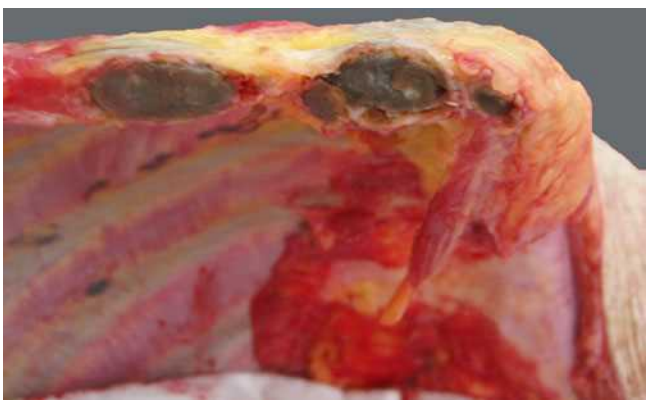

**Fig. 7.57** Examination of the bone and cartilage tissues occasionally shows unusual phenomena. This is a case of ochronosis (alkaptonuria) with brown/black discolouration of the sternal and other cartilage surfaces

## Suggested Reading

Burton JL, Rutty G. The hospital autopsy. 3rd ed. London: Hodder Arnold; 2010.

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# Endocrine Glands: Thyroid, Parathyroid, Adrenal, and Pituitary

8

S. Kim Suvarna

## Introduction

The general autopsy often gives scant or cursory attention to the endocrine tissues, but the possible impact of endocrine tissues on the individual's health—and death—should always be considered. To fully appreciate the pathology of these tissues, a good knowledge of normal physiology and the case history is vital. Furthermore, the knowledge of therapeutic treatments is often vital in case consideration.

This chapter covers the thyroid, parathyroid, adrenal, and pituitary glands. These endocrine tissues exist as separate glands. Of course, it is also recognised that many other tissues can secrete hormones and other circulating factors into the blood stream, particularly the pancreas (covered in Chap. 5). The pancreas is notably linked to diabetes and thereby to cardiovascular disease, sepsis, and metabolic issues that are covered elsewhere in this book.

For the four glands under consideration, in most cases review of the clinical data before the autopsy and subsequent macroscopic glandular tissue assessment will suffice as the examination needed at autopsy. Thus, general dissection and slicing of the tissues in situ may well be enough to confirm normality. Nevertheless, if there is any concern that the endocrine tissues are of pathological quality, then the glands should be dissected free of the local structures, carefully checked in terms of architecture, weighed, and sliced. Photography is often of value, and some tissue may be submitted for histology.

The endocrine glands should be fully sampled in cases of unusual deaths, especially all maternal or sudden, unexpected deaths.

In all the following images, the red bar marker, where present, is 10 mm.

## Thyroid

The thyroid gland (Figs. 8.1, 8.2, 8.3, 8.4, 8.5, 8.6, 8.7, 8.8, 8.9, 8.10, 8.11, 8.12, 8.13, 8.14, 8.15, 8.16, 8.17, 8.18, 8.19, 8.20, 8.21, 8.22, and 8.23) is not an uncommon site of pathology. In many cases, clues of thyroid disease are present externally in the form of macroscopic scarring and/or goiter.

The thyroid gland is situated immediately in front of the thyroid cartilage and upper trachea. It is a bi-lobed structure, with a weight of 20–40 g in health. Its size will reflect the individual, with some variation occurring during growth, health, pregnancy, and systemic disease. It is surrounded by a thin capsule, often with the four parathyroid glands adjacent. Heterotopic thyroid tissue also can be found within the low neck or even in the mediastinum.

The thyroid and parathyroid glands are best approached from a posterior perspective, rather than dissecting through the layers of the anterior strap muscles. By grasping the neck structures in the cup of the hand, some grasping-style tension can be applied on both sides of the neck tissue, around the trachea. This first allows a broad appreciation of where the thyroid will be, with the likely positions of the parathyroids.

The thyroid gland, once dissected free, is slightly irregular in terms of external morphology. A small amount of nodularity is probably within the range of normality. The cut section of the gland shows a meaty, red-brown architecture of an even quality.

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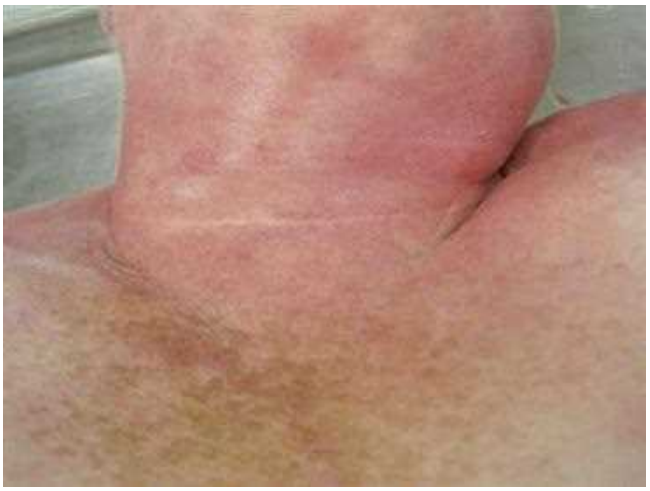

**Fig. 8.1** External inspection of the neck of all cases should look for features of previous thyroid surgery—in this case, a well-healed, transverse low cervical scar

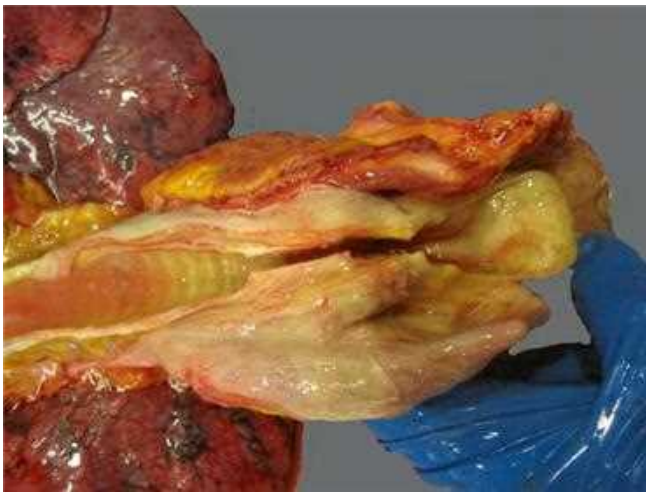

**Fig. 8.3** The upper neck structures are seen from behind. The thyroid and parathyroid tissues are not immediately apparent, but rather are eased into view by careful dissection of the fascial planes, as discussed below

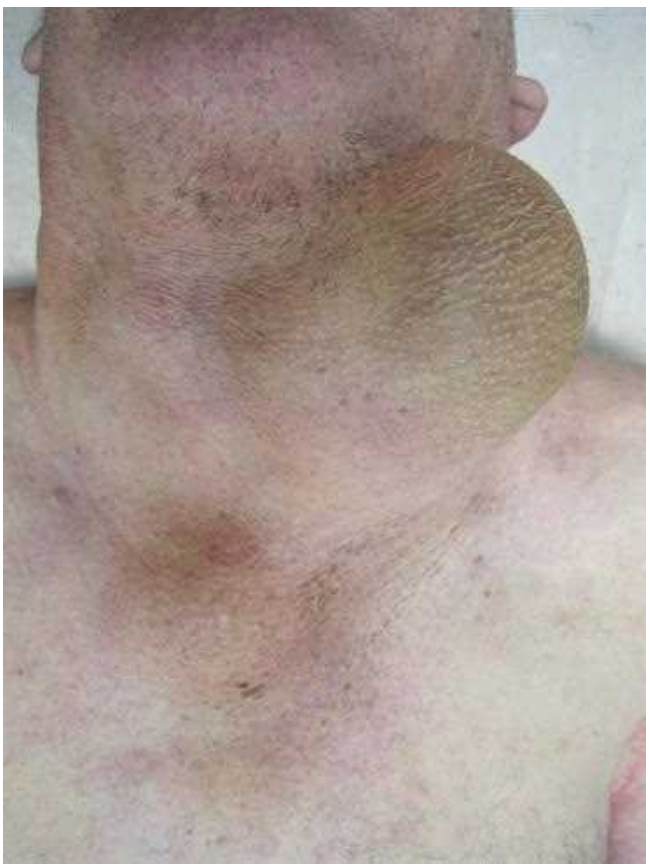

**Fig. 8.2** The macroscopic features of thyroid enlargement may be obvious. The cervical tissues appear distorted by a rounded, firm mass, which appears most pronounced on the *left side*, though some palpable fullness and enlargement of the contralateral thyroid tissues was also evident

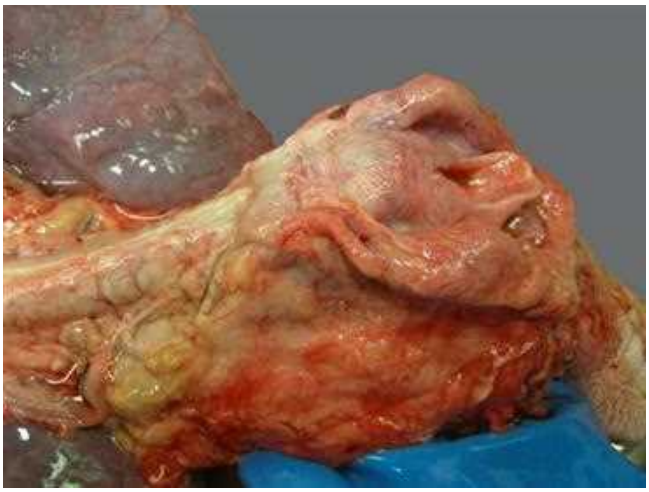

**Fig. 8.4** The posterior view of the neck structures is viewed from the right side oblique perspective, with tension applied to the neck parenchyma, allowing the broad position of the thyroid and parathyroid glands to be defined

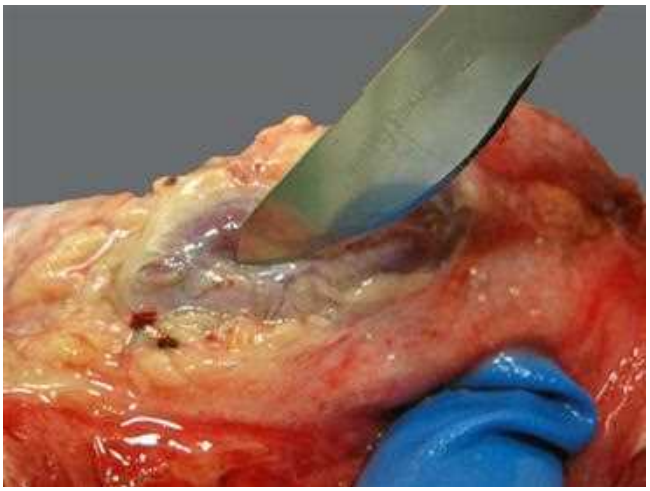

**Fig. 8.5** The scalpel blade is run gently along the thyroid capsule to allow separation of the tissues at this fascial plane

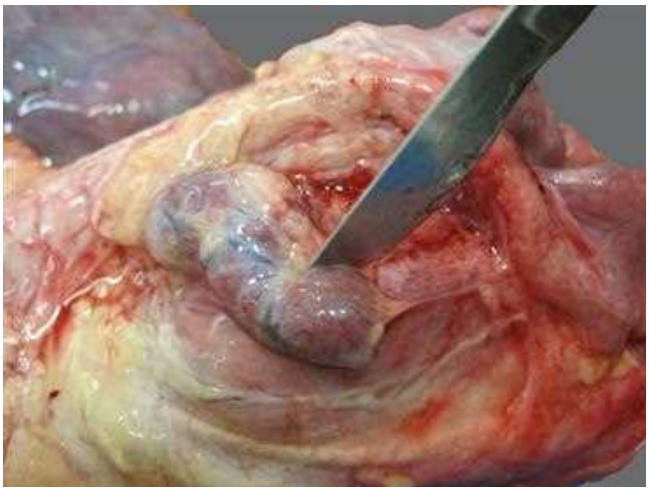

**Fig. 8.7** The thyroid gland can be now be gently prised away from the central neck structures, and the gentle incision of fascia tissues continues across the posterior aspect of the gland. The thyroid is dissected free from the local trachea by running the scalpel blade gently between the thyroid capsule and airway tissues. One should be careful not to incise into the thyroid gland

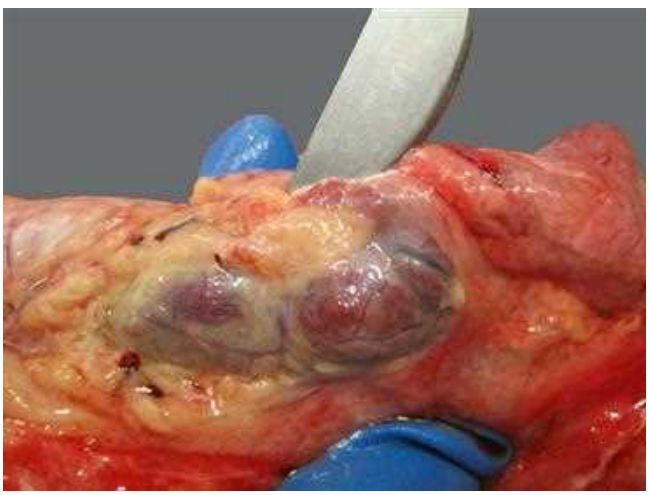

**Fig. 8.6** The tension and gentle dissection allow the thyroid gland to emerge into view. The scalpel blade has been 'stroked' gently across the lateral/posterior surface of the thyroid, allowing the gland, with slight nodularity, to be seen with local vasculature

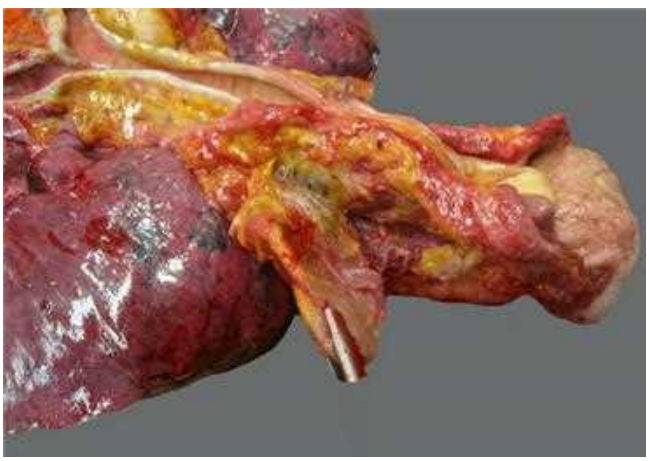

**Fig. 8.8** The thyroid parenchyma has been dissected to reveal the smooth external lobar architecture. It is being pulled forwards by forceps to aid dissection

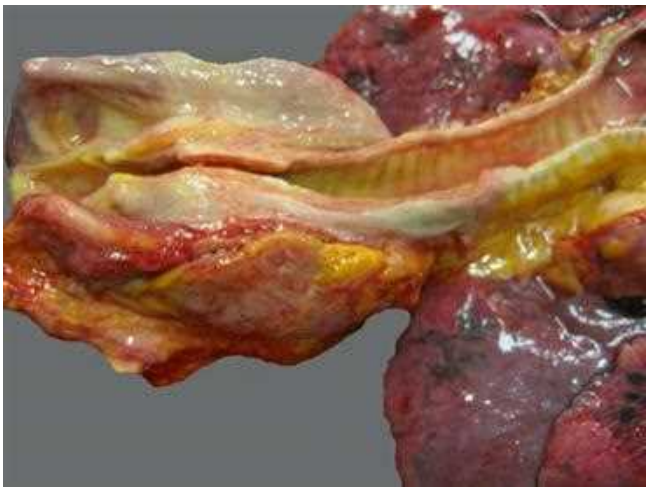

**Fig. 8.9** The same process is completed on the other side, with one clearly visualizing the gland

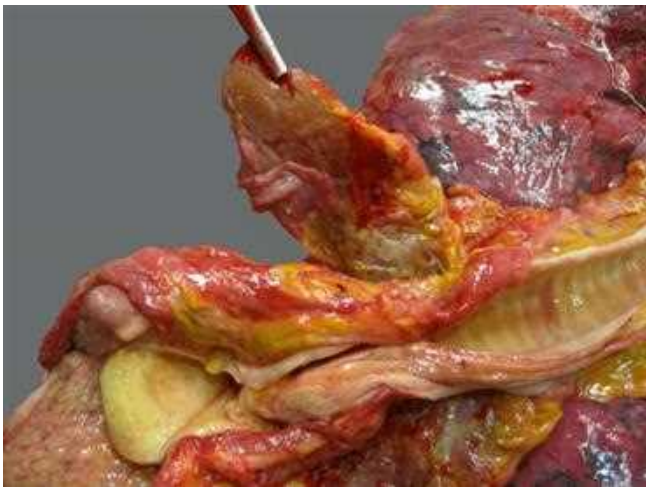

**Fig. 8.11** The right side of the thyroid is now free

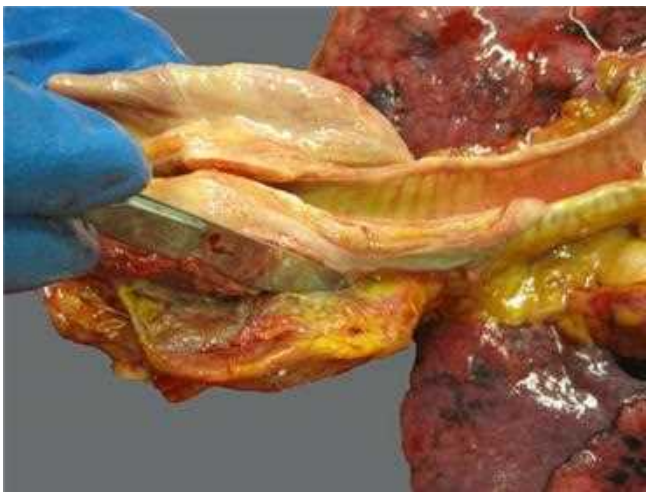

**Fig. 8.10** The posterior aspect of the thyroid is exposed by a very gentle stroke of the scalpel; not much pressure should be applied to the blade

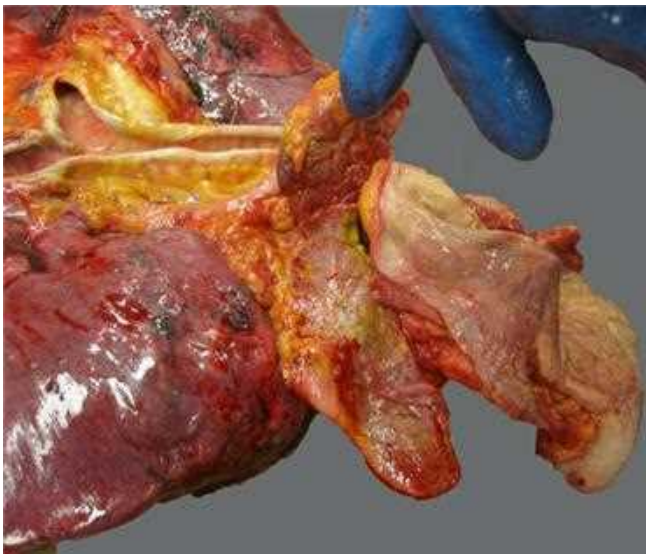

**Fig. 8.12** The left side of the thyroid is mobilized, and the dissection plane is extended onto the tracheal external surface. The left thyroid lobe may now be 'passed' to the right side, in front of the trachea. This allows the gland to be resected intact, and with minimal local soft tissues

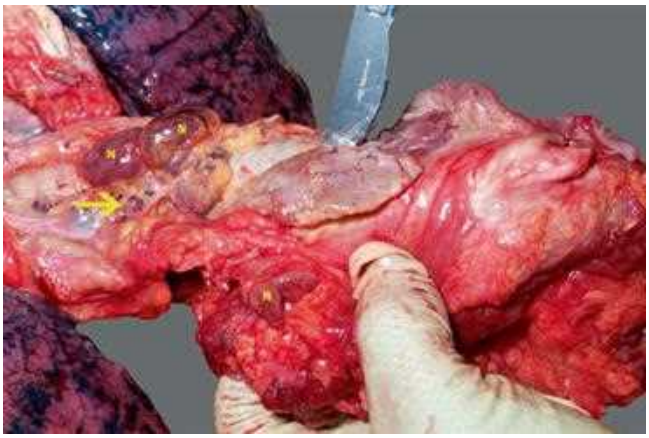

**Fig. 8.13** Note the tension applied (by the hand grip) to the neck structures, to allow the thyroid to be gently mobilized. Below the thyroid and towards the lung interface are several rounded, mid-brown lymph nodes (*N*). These nodes should not be mistaken for parathyroid elements, although one possible parathyroid is noted (*arrow*)

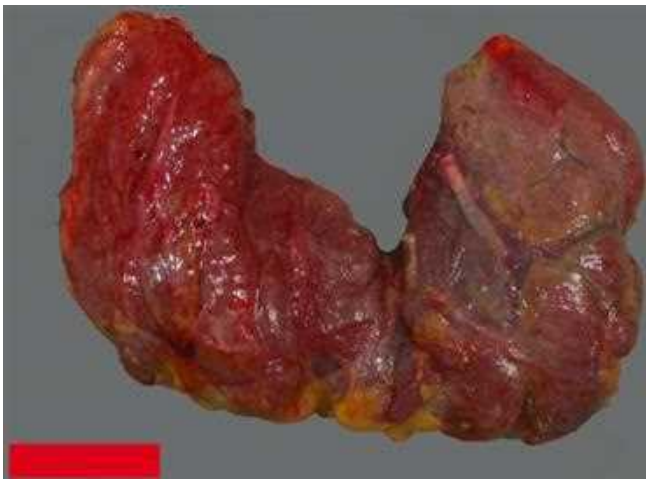

**Fig. 8.15** An enlarged thyroid is seen, with a very meaty architecture and some nodularity. This colloid goiter was clearly evident in the neck but was producing no direct pathological problem

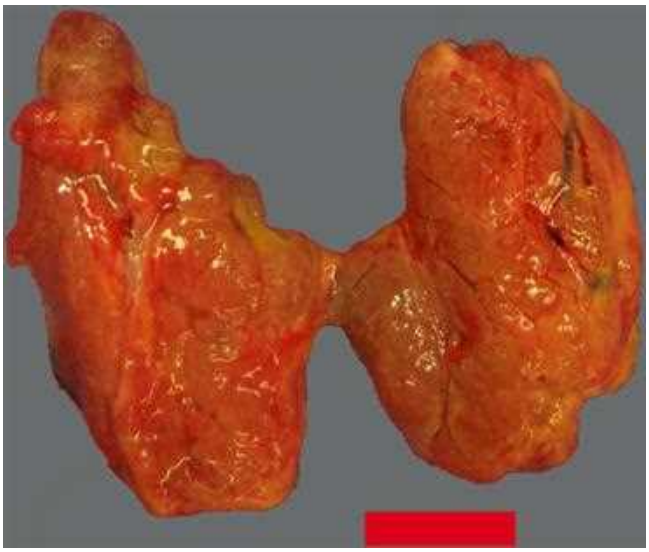

**Fig. 8.14** The normal thyroid has a 'bow tie' architecture, with two large lobes and a small central isthmus

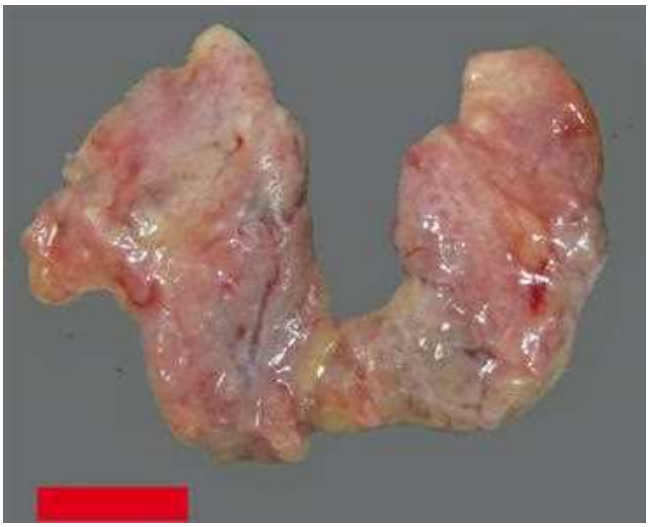

**Fig. 8.16** The thyroid gland in many elderly individuals is subject to autoimmune destruction, with tissue atrophy and fibrosis. In this case of Hashimoto's/autoimmune thyroiditis, the gland is shrunken and fibrotic

**Fig. 8.17** This thyroid tissue has a brown, colloid nodule seen towards the lower end. The thyroid gland in this case has simply been incised along the long axis of the lobe to demonstrate the lesion, rather than undergoing complete resection and display

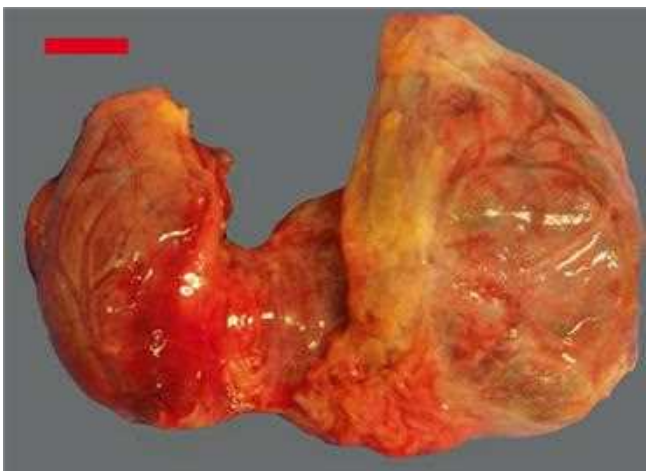

**Fig. 8.18** Disproportion between the lobes of the thyroid is quite common and may produce localised goiter externally, as well as clearly a defined overgrowth at autopsy. Such goiter may be associated with underproduction or overproduction of thyroid hormones, or indeed a euthyroid status

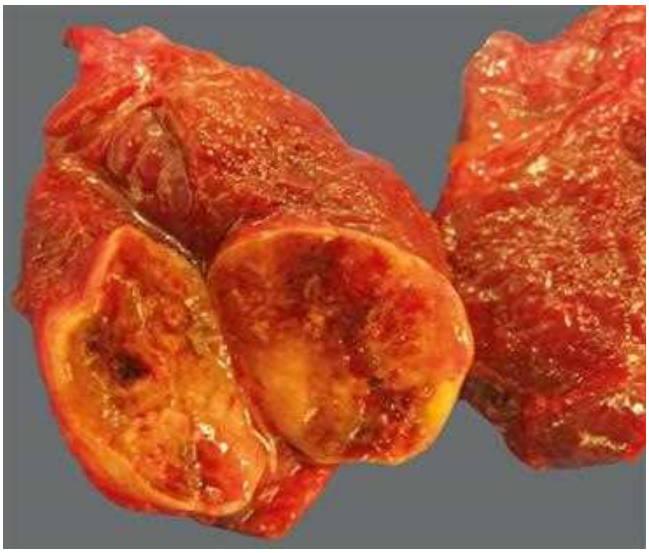

**Fig. 8.19** In many thyroid glands, some of the nodules have a cystic quality. This case histologically was one of cystic papillary carcinoma, but most cystic nodules are benign/colloid in type. These nodules should not be overinterpreted as malignant, although encapsulated thyroid malignancy should always be considered

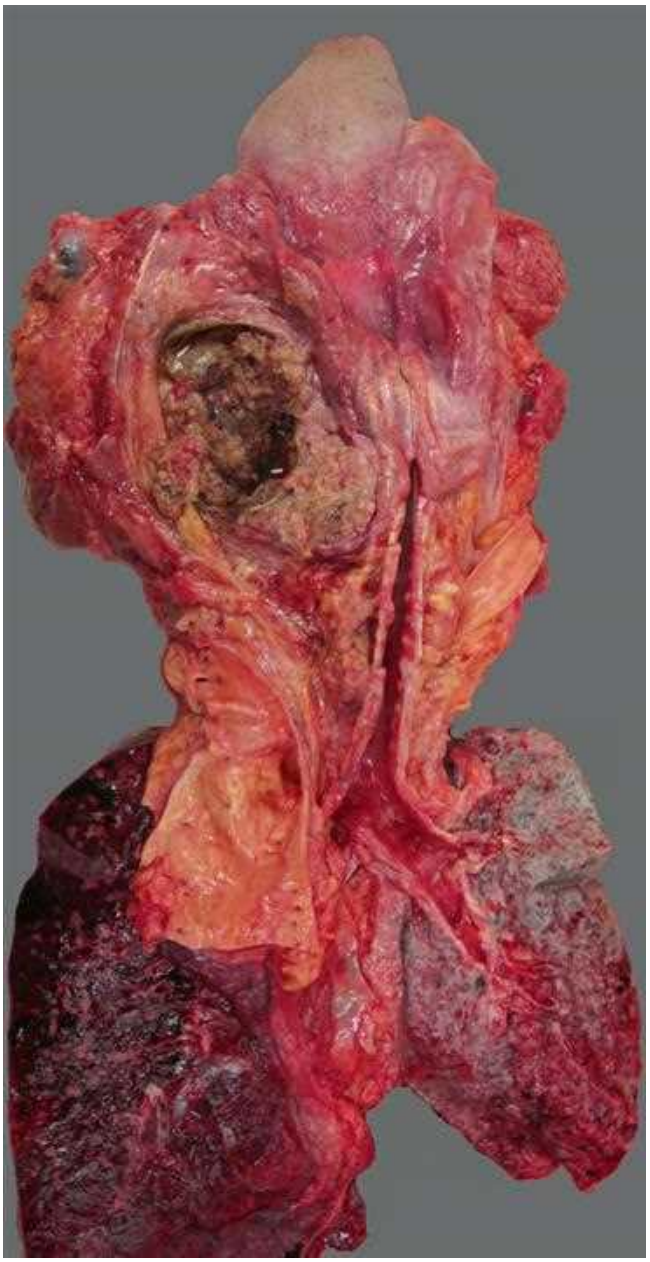

**Fig. 8.20** This dissection shows the tongue superiorly, neck structures centrally, and lungs towards the base. There is distortion of the tracheal parenchyma by a partly necrotic thyroid tumor, particularly in the middle and upper cervical region

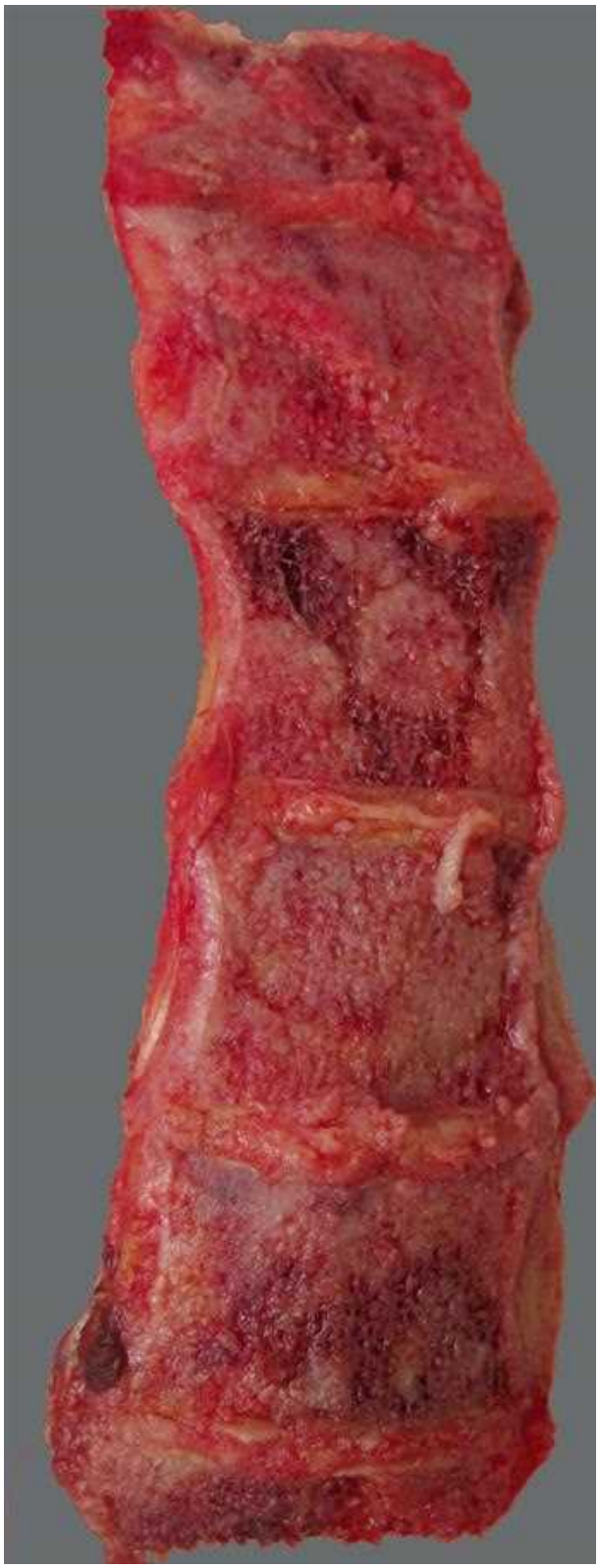

**Fig. 8.21** Metastatic follicular carcinoma is seen in the vertebral tissue block

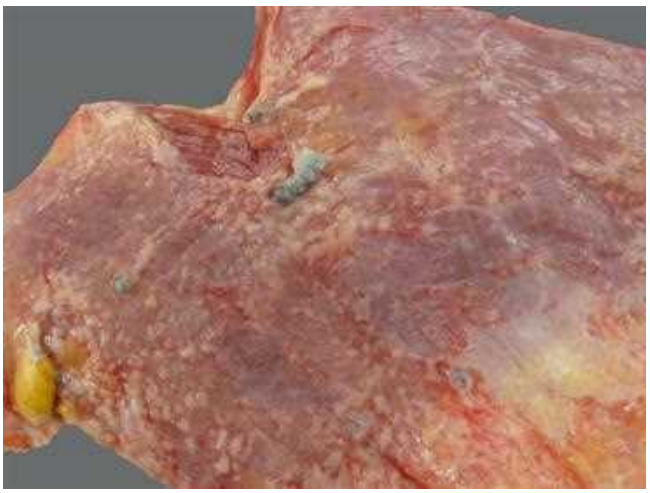

**Fig. 8.22** Metastatic thyroid cancer can be seen in many sites, some being unusual. Here, follicular carcinoma deposits are seen on the superior aspect of the diaphragm. Immunohistochemistry can be applied to autopsy tissues and is of use in identifying thyroglobulin-positive neoplasia

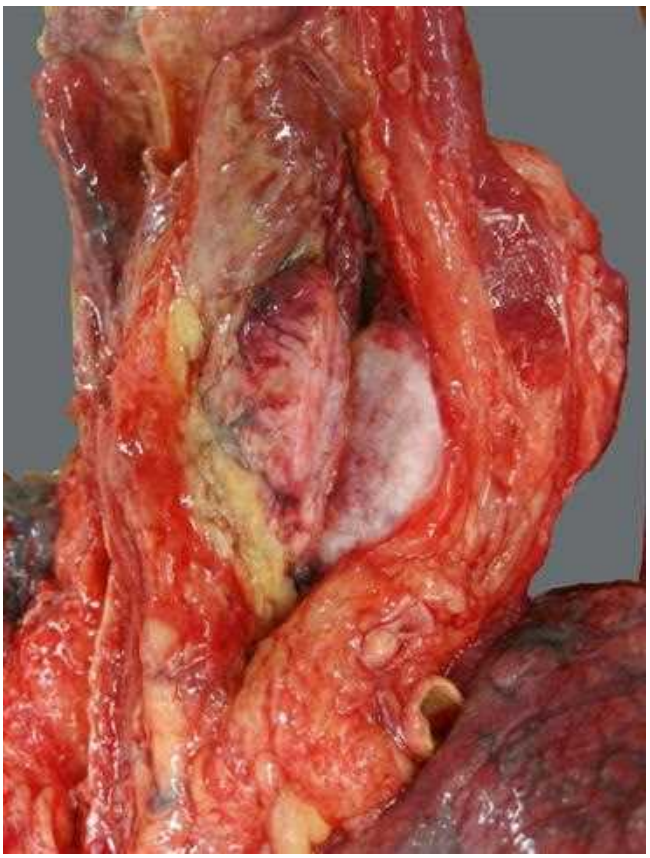

**Fig. 8.23** The thyroid can be the site of metastasis, often in late-stage, disseminated cancers. It can also be a primary site for lymphoma. This view shows lung cancer that has spread to the thyroid lobe, with widespread loss of the normal parenchyma

## Parathyroids

The parathyroid glands (Figs. 8.24, 8.25, 8.26, 8.27, 8.28, 8.29, 8.30, 8.31, and 8.32) are often best examined and appreciated during thyroid dissection. There are usually four macroscopically identifiable glands, with an average weight in surgical biopsies of 20–40 mg. In almost all autopsy cases, the parathyroid glands are unremarkable, being small. They are often easily missed. Many pathologists do not search for these small tissue elements, arguing that unless the glands are obviously enlarged, they will be normal.

Most parathyroid pathology seen at autopsy will be found in cases of renal disease, with secondary hyperparathyroidism. This is manifest as generalized gland enlargement and hyperplasia, affecting all four glands to a roughly similar extent. In these cases, the glands are very easy to spot, potentially being up to 20 mm in diameter.

Contrastingly, it is rare to find complete absence of the parathyroid tissues, except as part of a syndrome (*e.g.*, di George). In most cases, therefore, the absence of parathyroid gland enlargement (together with a lack of significant antemortem history) may allow one to judge that the glands are within normal limits.

The parathyroids are best identified by means of careful dissection of the thyroid. This section demonstrates a posterior approach, grasping the anterior neck tissues firmly and applying tension towards the front of the cervical block. This technique allows the scalpel blade to gently scrape along the edge of the posterior thyroid. As one applies gentle sweeping movements with the scalpel blade, the parathyroids will begin to 'pop' into view and often are clearly evident without any detailed dissection.

Firm incision into the soft tissues and thyroid is not recommended, as this will distort the thyroid parenchyma/capsule, and the loss of blood and fluid often obscures the dissection plane.

If the glands cannot be identified, then the soft tissues adjacent to the thyroid (including fat, lymph nodes, and possible parathyroid tissue) are resected en bloc in order to guarantee capture of the parathyroids. It should also be remembered that the upper thymic tissue can contain some embryologically derived parathyroid parenchyma. If it is important to understand parathyroid histology, then these sites may need to be resected for histology examination.

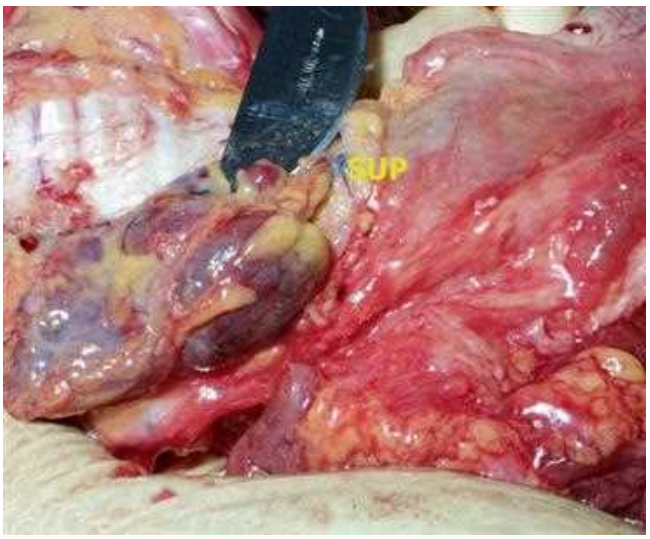


**Fig. 8.24** As one dissects the posterior aspect of the thyroid gland, one should look closely for parathyroid elements. This close-up view shows that the thyroid parenchyma has been largely exposed and is being defined with tension applied to the neck structures. The right superior parathyroid gland (*SUP* here under the point of the scalpel) stands out from the thyroid capsule

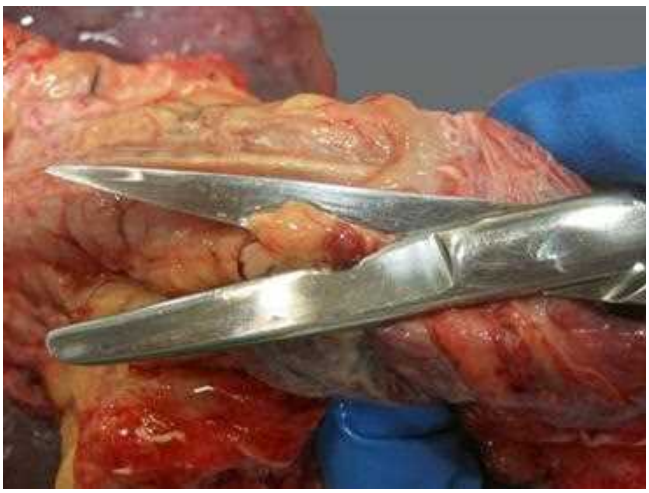

**Fig. 8.26** In this case, scissors are used to lift and gently resect the parathyroid gland tissues. This is a particularly useful technique, since the parathyroid glands stand out when the tissues of the neck are placed under tension

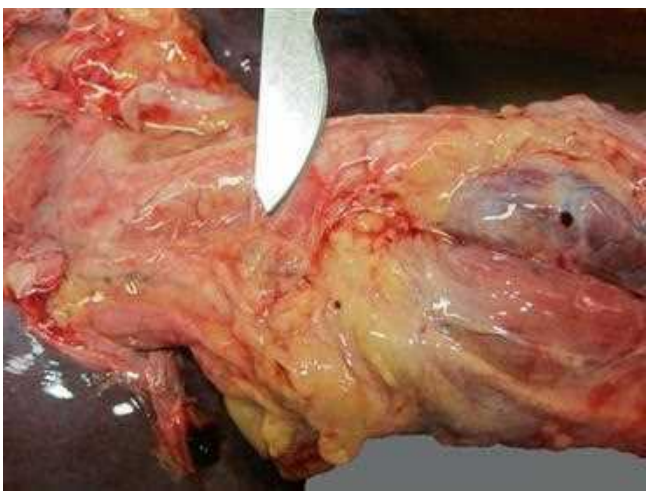

**Fig. 8.25** Often the parathyroids are not easily identified, lying within fatty cervical soft tissue. If one is unsure as to the position of the glands, then it is advisable to fully resect the fatty tissues around the likely position. These tissues can then be fixed 'en masse' and later examined by histology

**Fig. 8.27** Parathyroid glands tend to float when placed into formalin, contrasting with lymph nodes, which mostly sink. This crude technique is useful to confirm that one has captured all four glands!


**Fig. 8.28** This view of the four parathyroid glands (viewed from behind) serves to show the right superior (*RS*), right inferior (*RI*), left superior (*LS*), and left inferior (*LI*) glands. A local lymph node (*LN*) is included to emphasise how easy it is to mistake nodes and parathyroid tissues

**Fig. 8.29** Close-up of the parathyroid gland shows a fine vascular network around the gland tissue, which is closely attached to local fatty soft tissue and the back of the thyroid gland

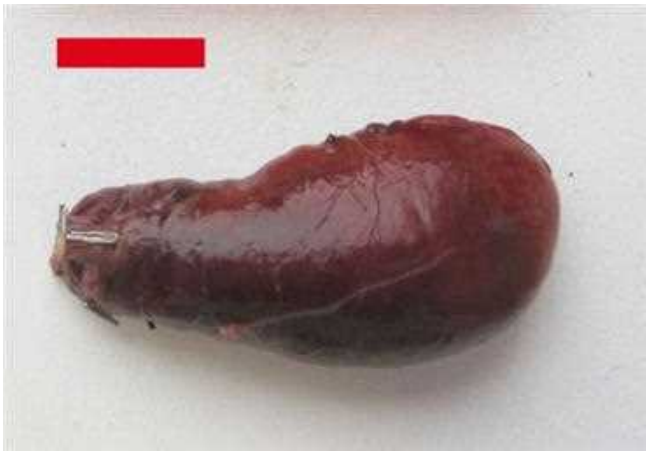

**Fig. 8.30** A parathyroid adenoma (surgical case), showing considerable glandular enlargement

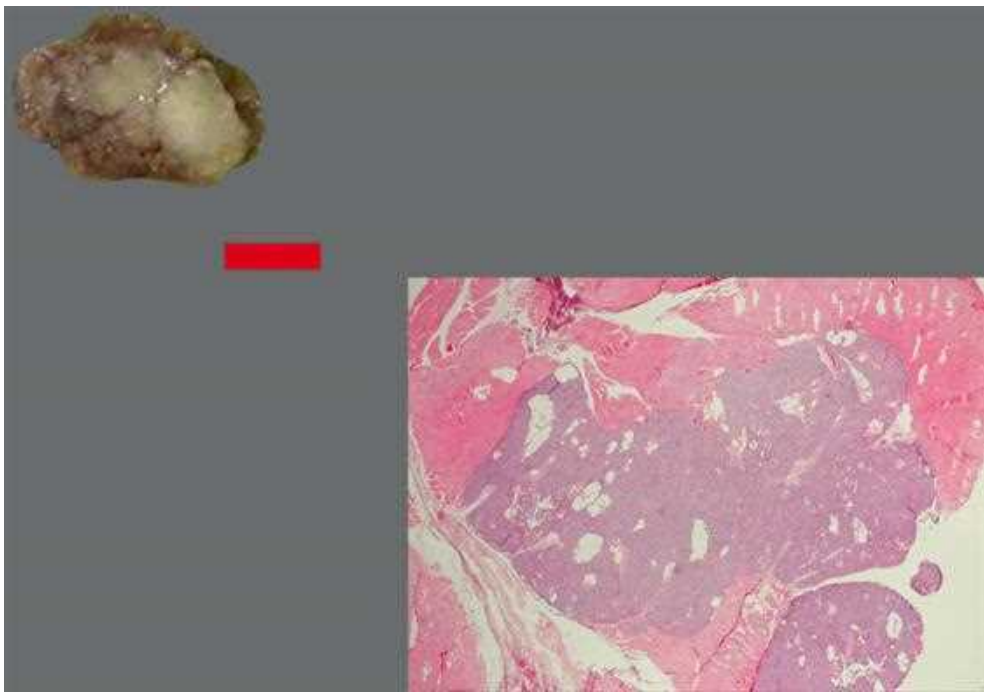

**Fig. 8.31** On occasion, parathyroid resection would have been undertaken previously, usually as a consequence of chronic kidney disease. A small amount of residual parathyroid tissue is often implanted into the forearm, and this too may become hyperplastic. This surgically resected sample shows somewhat grey tissue with surrounding brown muscle (*top left*), with the histology (*bottom right*)

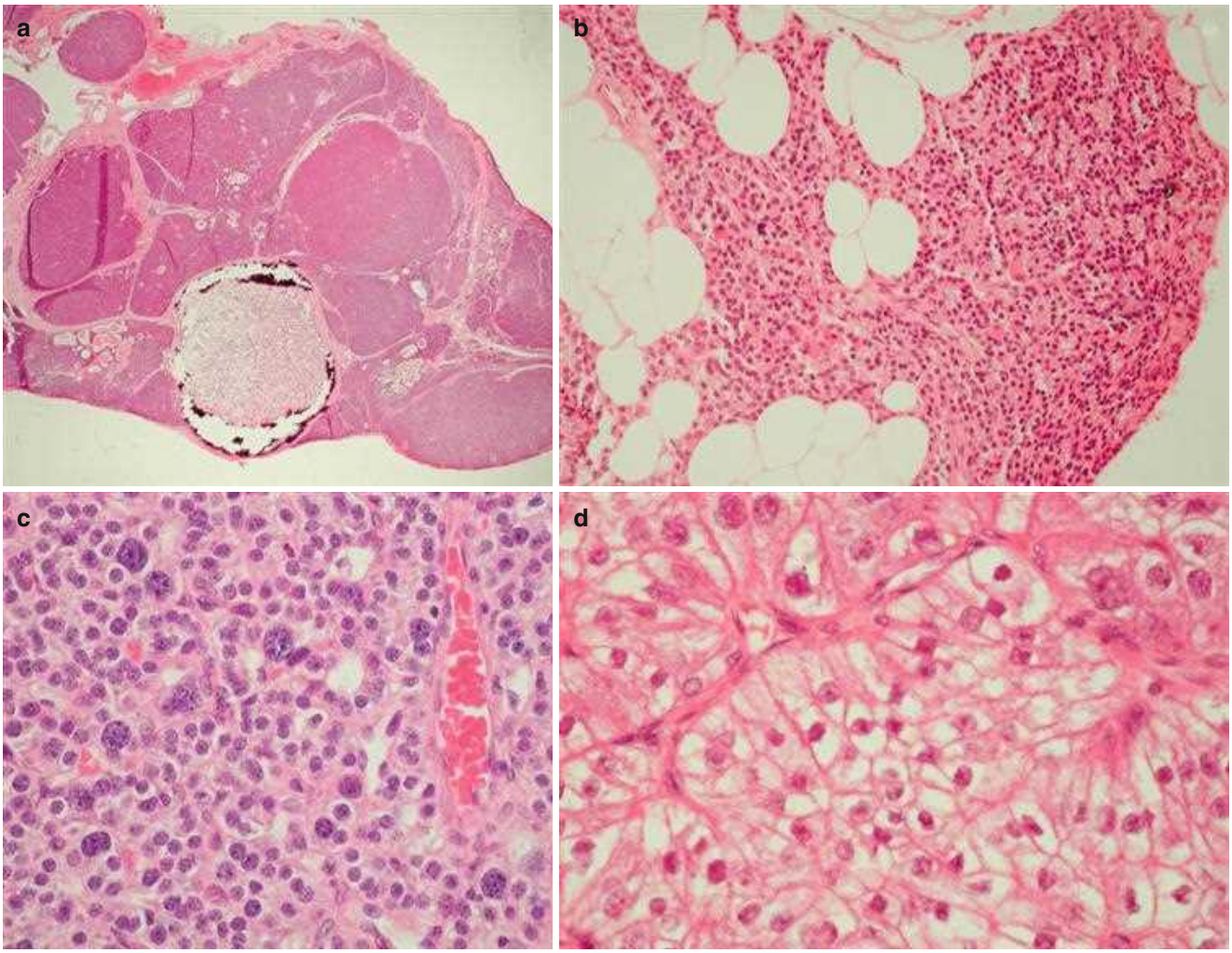


**Fig. 8.32** Many aspects of parathyroid tissues are really appreciated only under the microscope. (**a**) Parathyroid hyperplasia can have a variable nodularity with some calcification. (**b**) Normal parathyroid tissues have chief and oxyphil cells mixed with fat. (**c**) Nuclear pleomorphism may be found in hyperplastic glands and does not signify malignancy. (**d**) Parathyroid carcinoma may appear relatively bland but often has pronounced clear cytoplasm

## Adrenals

The adrenal tissues (Figs. 8.33, 8.34, 8.35, 8.36, 8.37, 8.38, 8.39, 8.40, 8.41, 8.42, 8.43, 8.44, 8.45, 8.46, 8.47, and 8.48) have two significantly different endocrine functions. The outer cortex, appreciated histologically as three discrete layers, is the site of production of a variety of steroids. These have effects in terms of mineral metabolism, general health, and sexual function. The inner medullary tissues are concerned principally with the production of adrenalin and noradrenalin. The weight of the adrenals varies according to the body frame of the individual but is generally 5–10 g per gland.

The adrenal gland tissues are sited close to the superior pole/capsule of the kidney. The glands are often deep within a significant amount of retroperitoneal fat, hindering identification. Blind dissection or slicing into the fat at a likely gland site should allow one to inspect the tissues directly. This process is obviously easier if the glands are not deep in retroperitoneal fatty parenchyma. Whether the tissues are found by direct vision or by serial slices, however, the direct visualization of the parenchyma during the course of a routine autopsy is often all that is necessary to confirm normality.

This basic examination technique allows confirmation of the normal arrangement of cortical and medullary tissues and exclusion of nodularity, tumors, haemorrhage, or necrosis, but histology beneficial in a review of metabolic dysfunction, particularly if implicated in the cause of death (*eg*, Cushing's disease, hypoadrenalism).

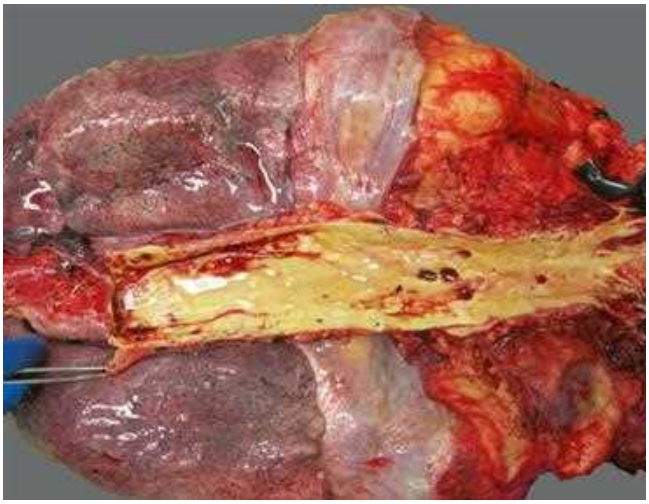

**Fig. 8.33** The adrenal glands require a staged dissection for identification in most cases. The eviscerated retroperitoneal block (see Chap. 3) is seen from the back. The aorta has been opened, in this initial dissection view, with the lungs being seen above the diaphragms (towards the left). The fatty retroperitoneal tissues are seen below the diaphragm. It is possible to palpate the kidney tissues and thereby derive a rough position for the adrenal glands, which lie above and medial to the kidney upper pole. Removal of the diaphragms will often assist identification of the likely adrenal sites, but in those with minimal fat, the adrenals are often clearly visible on direct inspection

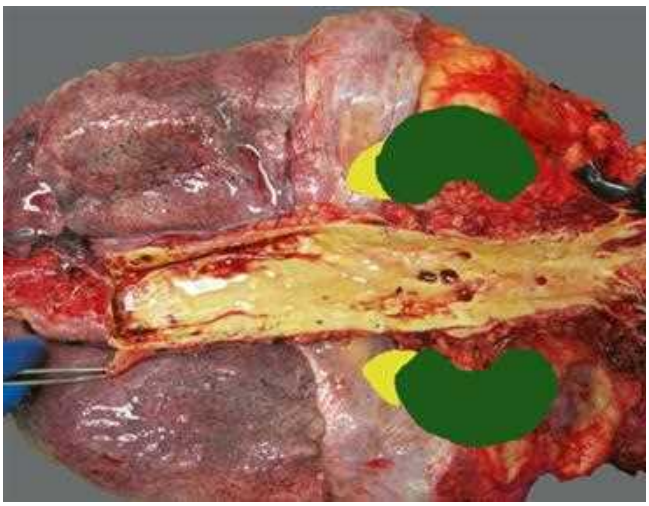

**Fig. 8.34** The same thoracic/peritoneal block of tissue has overlaid two *green kidney* shapes to represent the positions of the renal parenchyma. The approximate position of the adrenals (*yellow*) is seen on the superior aspect of both kidneys. Awareness of this localization can assist the search for these glands

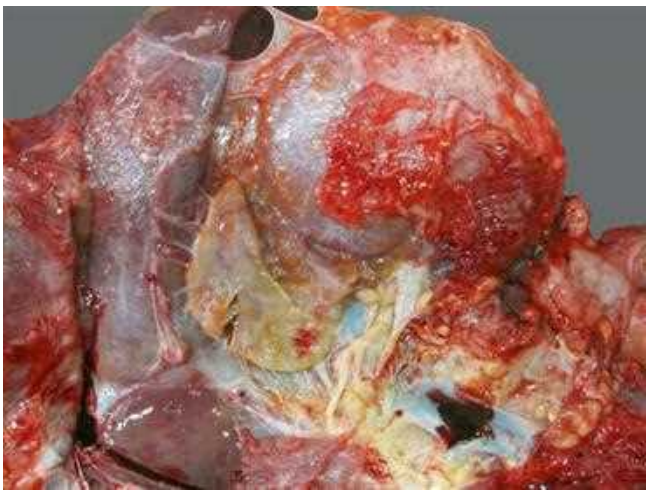

**Fig. 8.35** The right-side posterior retroperitoneum is seen from the back. The adrenal is positioned above and slightly medial to the kidney tissues. The relative paucity of fat in this case allows one to fully appreciate the shape and architecture of the adrenal in relation to the liver and kidney

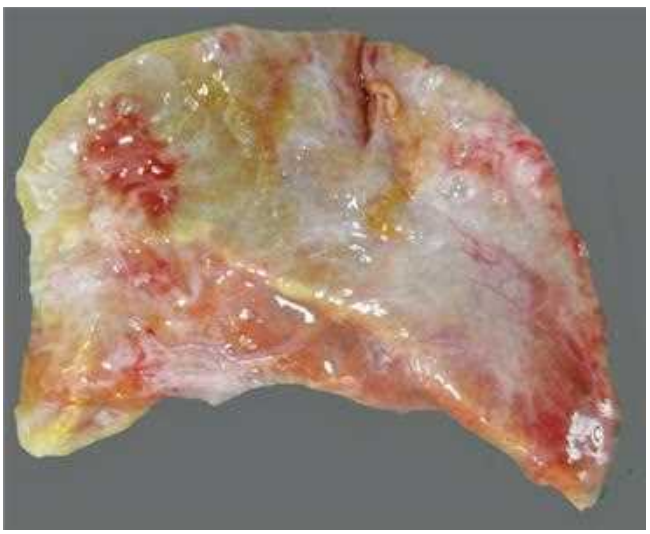

**Fig. 8.36** The adrenal has now been largely dissected free of the surrounding fat. There is a fine, fibrous capsule around the cortex

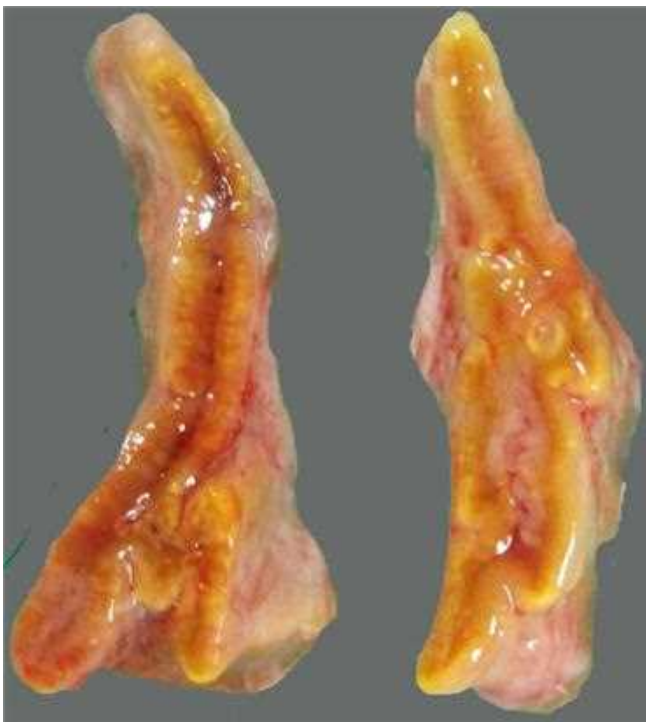

**Fig. 8.37** The cut surface of the normal adrenal shows a yellow/orange cortex, reflecting the steroid production of the three zones or layers of the cortex. The medullary tissue is often a tan/brown color, although it may be difficult to define on occasion. It is noted that the adrenal has a somewhat lumpy, irregular quality on the external and internal cut surface, without defined nodularity or other focal pathology. In some areas, the adrenal tissue is folded, potentially giving a false impression of cortical hyperplasia

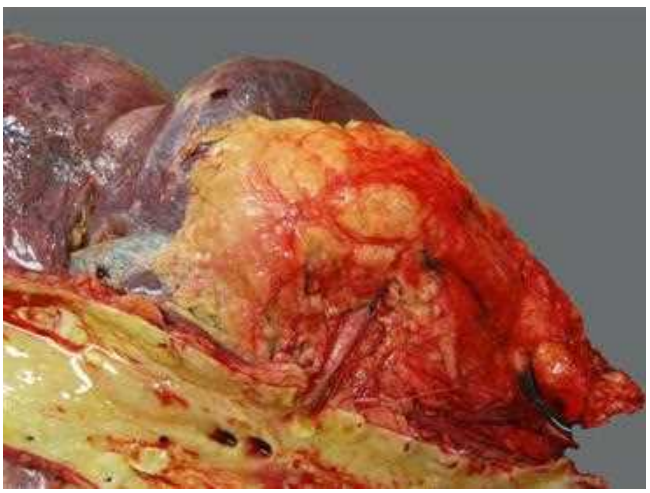

**Fig. 8.38** The aortic tissues have been opened, and the proximal renal artery is likewise open. The vague outline of the kidney can be identified, but the adrenal gland is not immediately apparent. One therefore has a choice as to whether to dissect directly into the fat in order to identify the gland, and then trace/dissect around the adrenal, or to resect the entire fatty tissue block that lies superior to the kidney and then serially section this tissue

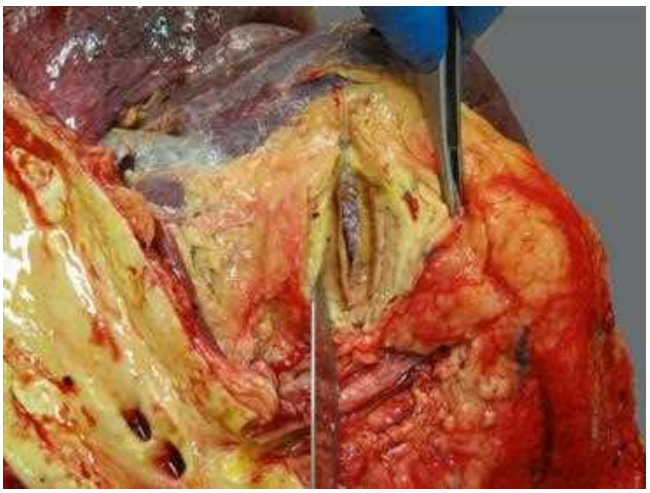

**Fig. 8.39** When pronounced fat is obscuring the adrenal position, one may choose to identify the adrenal by making a single cut into the fat and then dissecting around the adrenal once it is found. Alternatively, one can proceed along the course of the adrenal artery to identify the gland tissue, but this is a more time-consuming technique

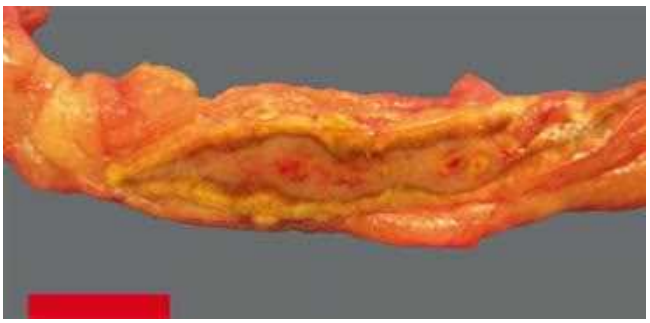

**Fig. 8.40** This adrenal, with some peripheral fat, shows a relatively thin cortex in relation to the medullary parenchyma. Although one might interpret this as medullary hyperplasia, it is in fact cortical atrophy in an individual who was systemically stressed by several episodes of surgery over the 2 weeks prior to death. In this case, the depletion and thinning of the cortical tissues is a stress response. One can see similar changes in individuals given long-term exogenous corticosteroids, with secondary ACTH suppression and thereby cortical atrophy

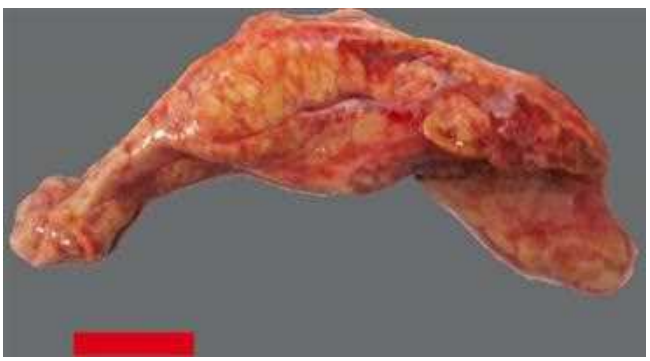

**Fig. 8.41** This adrenal shows nodular hyperplasia of the cortex with variable thickness of the cortical tissues. The hyperplasia is not uniform along the tissue length

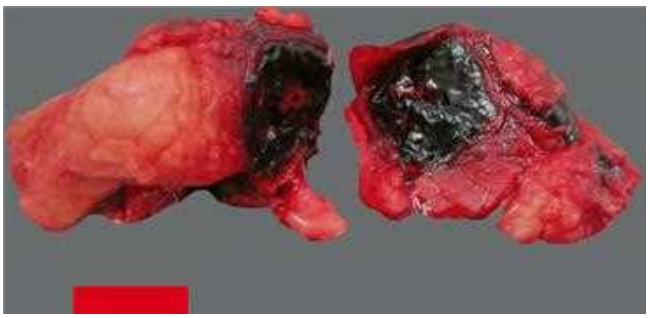

**Fig. 8.42** This adrenal has been opened to reveal marked parenchymal haemorrhage, principally in the medullary parenchyma, in a case of disseminated sepsis. Such changes are common in cases of significant bacteraemia (*e.g.*, meningococcal meningitis or group A streptococcal septicaemia)

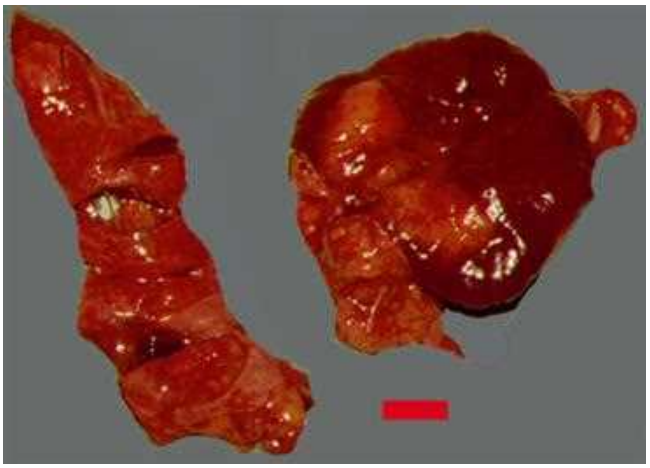

**Fig. 8.43** An adrenal myelolipoma is seen in a case with associated sickle cell disease (HbSS). The adrenal on the left side is normal. The right-side adrenal had a soft, haemorrhagic tumour. Histological examination confirmed the presence of all three types of hematological elements in this case, although there was pronounced hyperplasia of the erythron, in keeping with the sickle cell anaemia

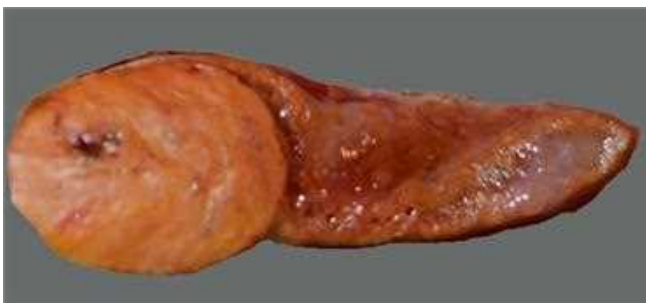

**Fig. 8.44** A rounded yellow adrenal adenoma is seen on the left-hand aspect of this adrenal gland. Such tumours may be found incidentally at autopsy. They may be functional (actively secreting hormones) or without endocrine secretion or effects

**Fig. 8.45** The retroperitoneal tissues of this case have been partially dissected and exposed in a case of adrenal cortical carcinoma. The tumour had been treated by both radiotherapy and chemotherapy. Tissue necrosis has occurred because the tumour outstripped its own blood supply and because of the oncological treatments, resulting in a large zone of haemorrhage and necrotic tissue. It is not easy to find viable tumour elements in necrotic tumors, and widespread sampling is recommended for histology

**Fig. 8.47** This fixed adrenal tissue has been widely invaded by metastatic lung carcinoma, with expansion of the tumour into adjacent soft tissues. Residual adrenal tissue can be seen focally, but the carcinoma has widely infiltrated and disrupted the gland

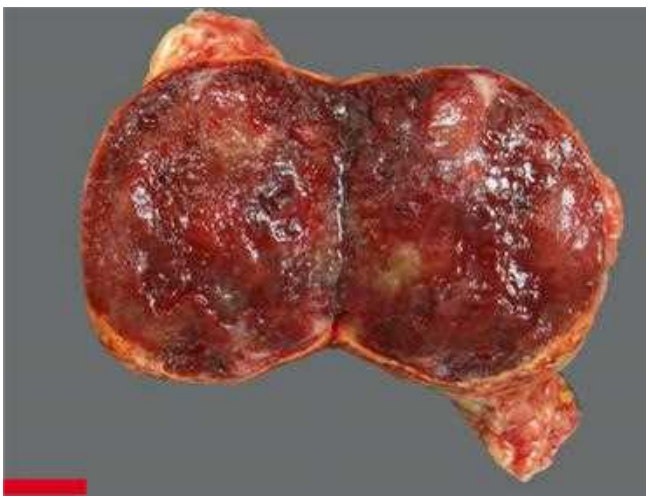

**Fig. 8.46** An adrenal paraganglioma is present, with a thin rind of normal adrenal tissue stretched around the mid brown adrenal tumour. Such tumours may be functional, with significant cardiovascular consequences, or they may be hormonally silent

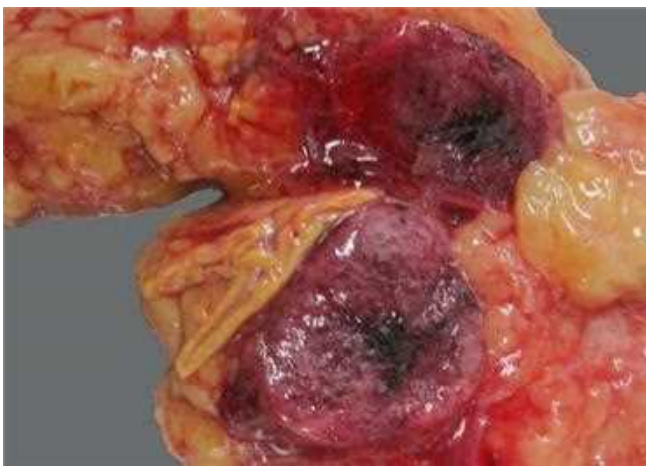

**Fig. 8.48** This case of alleged adrenal tumor actually reflected lymphadenopathy adjacent to the adrenal rather than tumour within the parenchyma. Metastatic tumours to the para-aortic site can be mistaken for adrenal neoplasia (either primary or secondary), so histological sampling is often important

## Pituitary

The pituitary gland (Figs. 8.49, 8.50, 8.51, 8.52, 8.53, 8.54, 8.55, 8.56, 8.57, and 8.58) sits in the sella turcica at the base of the skull adjacent to the optic chiasm. See Chap. 9 for information on the cranial/head dissection. Pituitary dissection is rarely an issue, given that the gland is removed whole after knocking the posterior clinoid processes backwards with the skull key. The pituitary is then 'scooped' out by running the scalpel blade around the sella boundary and gently extracting the gland with toothed forceps.

At autopsy, the gland is about 10 mm in diameter and generally weighs about 1 g. It comprises two main parts, termed *anterior* and *posterior*. There may be a small, macroscopically nondiscernible, embryological remnant (pars intermedia) between the two lobes.

Histologically, the anterior lobe contains polygonal endocrine cells with varying histology color (chromophobe, acidophil, basophil) in sections stained with hematoxylin and eosin. The cells have a rich vascular supply locally, and secrete hormones directly into the blood stream. The hormones secreted include follicle-stimulating hormone, prolactin, luteinizing hormone, thyroid-stimulating hormone, growth hormone, and adrenocorticotrophic hormone.

The posterior lobe may secrete oxytocin and antidiuretic hormone. It is a direct extension of the brain parenchyma. The hormones enter the blood stream from the ends of the nerves that extend from the local brain tissues.

The bulk of pituitary pathology at autopsy reflects adenomatous neoplasia and the effects of therapy. Other tumours and inflammatory conditions rarely may have a detrimental effect on pituitary status, however, so histology is often valuable.

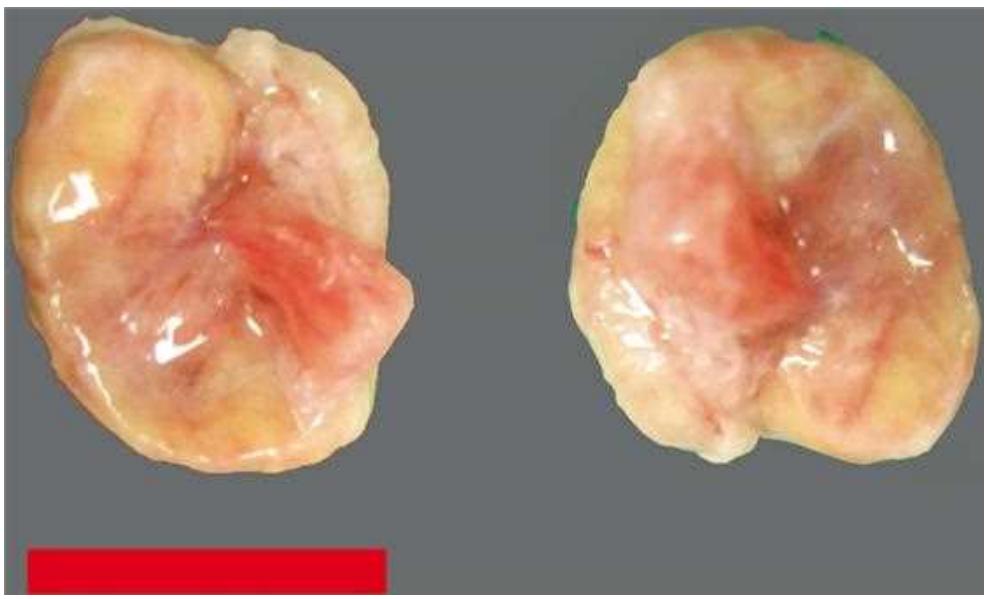

**Fig. 8.49** This normal pituitary is seen from the superior (*left*) and inferior (*right*) aspects. The stalk is present, entering the pituitary from the superior aspect

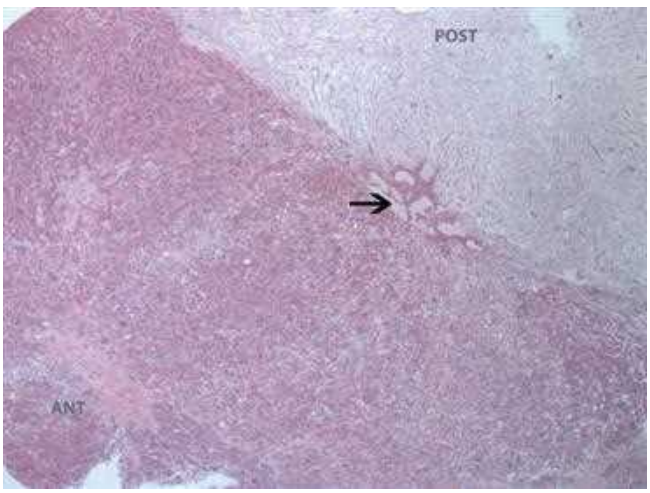


**Fig. 8.50** Histology of the pituitary demonstrates the anterior lobe (*ANT*) with closely packed polygonal cells and the neuronal elements of the posterior lobe (*POST*). The two parts are clearly separate entities, with the partly cystic pars intermedia (*arrow*)

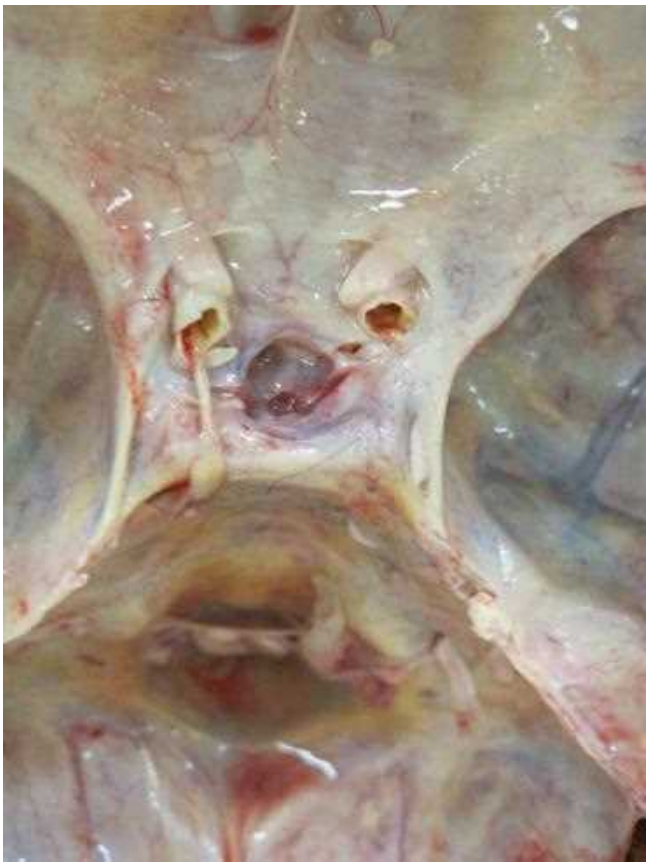

**Fig. 8.51** An empty sella can be found in autopsy studies in cases with no ante-mortem irregularity of hormone production, visual fields, or symptoms. There is often a thin rind of gland parenchyma around the base of the sella, which is still producing adequate levels of hormones

**Fig. 8.52** This pituitary site has been subject to previous surgery with dissolution of the adenomatous parenchyma. A hole at the base (transsphenoidal approach) indicates the point of entry of the surgical procedure

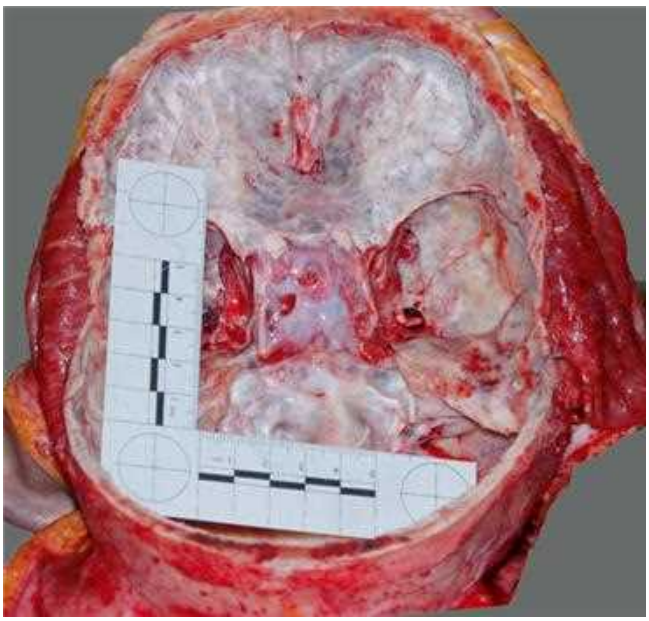

**Fig. 8.53** A pituitary adenoma is seen in situ, expanding and distorting the local sella tissues and bulging upwards focally in a diffuse fashion

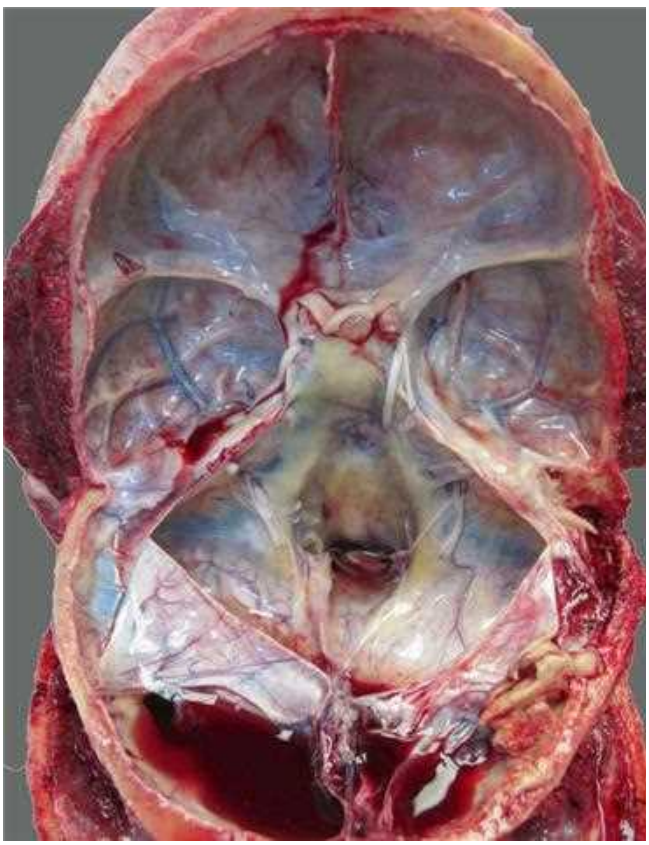

**Fig. 8.54** This pituitary adenomatous process, seen as cream-colored tissue, is seen expanding the sella and bulging upwards to interact with the optic chiasm

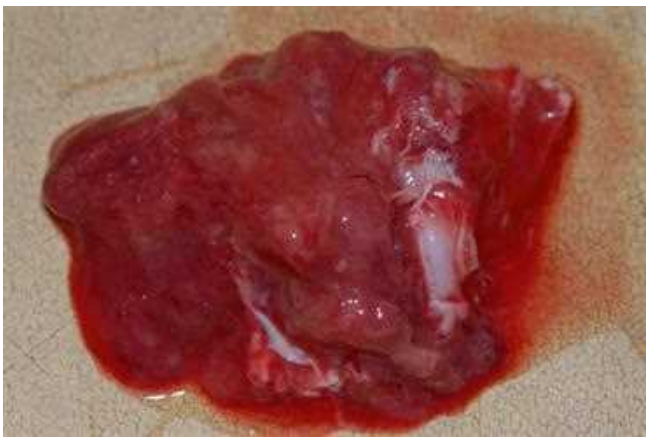

**Fig. 8.55** A close-up view of a pituitary adenoma, which had a very soft and focally liquefied quality, indicating partial necrosis. This individual had been treated for his adenomatous tumour, but clearly much of the lesion was viable. This tumour weighed 8 g and measured up to 3 cm, both expanding the sella and protruding upwards towards the optic chiasm

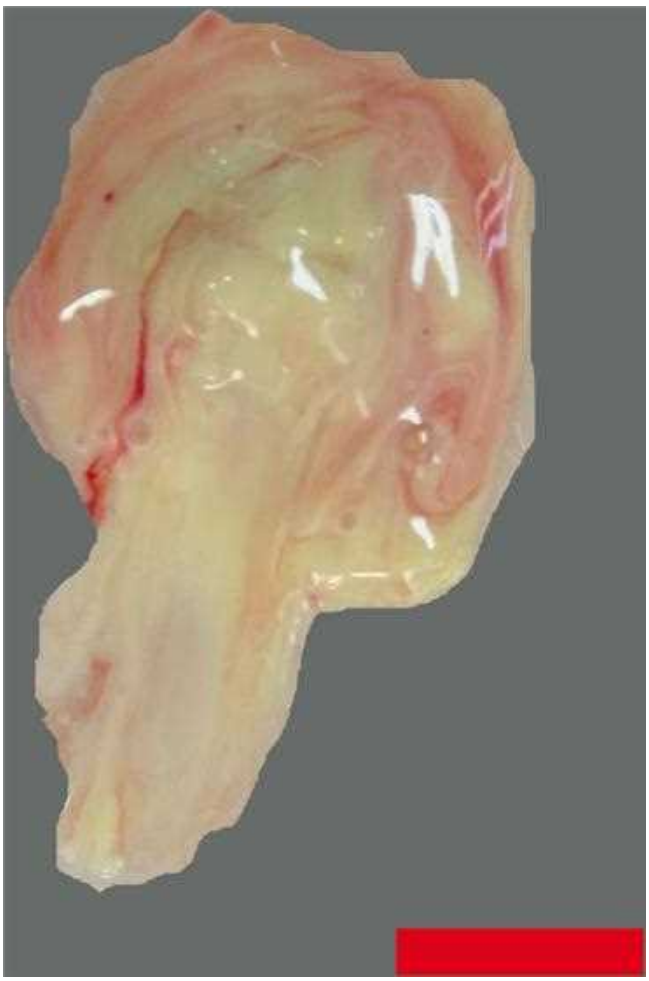

**Fig. 8.56** The liquefied pituitary fossa contents are seen in a case of adenomatous disease. The ante-mortem oncological treatment has caused significant degeneration of the pituitary neoplasm. Histology is often unrewarding, but it can be undertaken in order to assess the therapeutic effectiveness (*i.e.*, the tumor cell kill fraction)

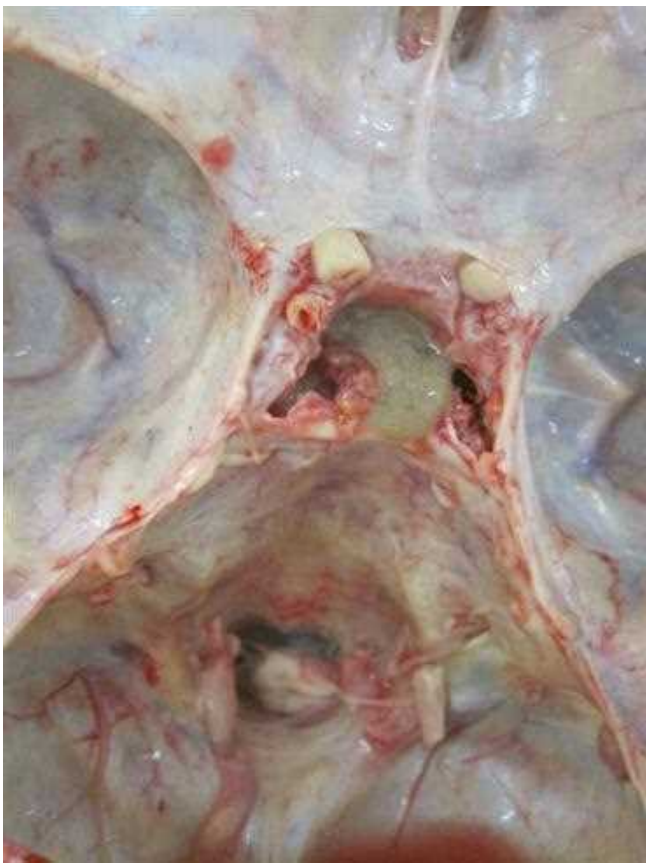

**Fig. 8.57** The skull base shows the sella turcica to contain partially liquefied grey/yellow tissue, in a case of treated pituitary adenoma. These cases are difficult to define macroscopically. Histology may be the only way to truly appreciate any residual tissue elements

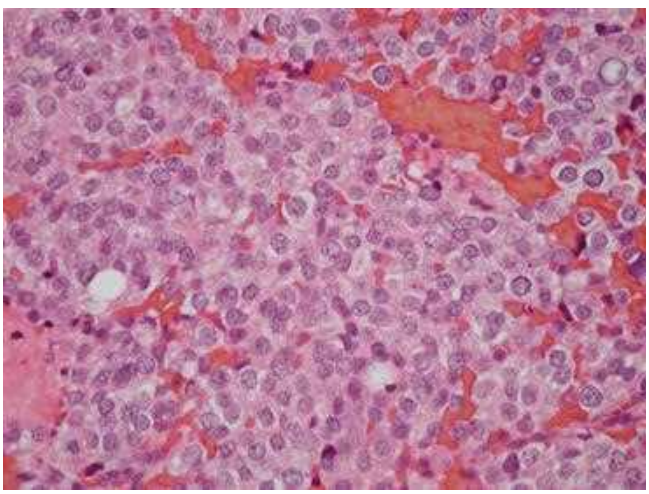

**Fig. 8.58** Histological examination of a pituitary adenoma (*viable*) shows closely packed polygonal cells of a classically neuro-endocrine quality, set within a vascular stroma

## Suggested Reading

Cooke RA, Stewart B. Endocrine system. In: Colour atlas of anatomical pathology. 3rd ed. Edinburgh: Churchill Livingstone; 2004. p. 183–204.

Curran RC, Jones EL. Endocrine organs. In: Curran RC, Jones EL, editors. Gross pathology: a colour atlas. Aylesbury: HM + M Publishers, London, United Kingdom 1974. p. 70–5.

Finkbeiner WE, Ursell PC, Davis RL. Autopsy pathology: a manual and atlas. Philadelphia: Churchill Livingstone; 2004. p. 257–61.

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Maitra A. Endocrine system. In: Kumar V, Abbas AK, Aster JC, editors. Robbins basic pathology. 9th ed. Philadelphia: Elsevier Saunders; 2013. p. 715–64.

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Strachen MWJ, Walker BR. Endocrine system. In: Boon NA, Colledge NR, Walker BR, Hunter JAA, editors. Davidson's principles and practice of medicine. 20th ed. Philadelphia: Churchill Livingstone; 2006. p. 739–804.

# The Central Nervous System, with Eye and Ear

9

Julian L. Burton and S. Kim Suvarna

## Introduction

Examination of the brain is an emotional issue for some relatives, as it involves opening the head. Some pathologists and medicolegal authorities prefer to examine the brain only when examination of the organs within the torso has not yielded a definitive cause of death. It may be argued that this approach is seen as more acceptable (i.e., less disfiguring) by the relatives of the deceased. This view may have some merit if ante-mortem CT imaging of the head is available and there is no likelihood of new intracranial disease.

Nevertheless, it may be argued that the brain should be examined in every autopsy, in line with best-practise guidelines [1, 2]. An argument against progressing to intracranial examination because of time constraints (cited by some pathologists) cannot be used. The autopsy examination must be sufficient to allow the pathologist to determine the cause of death reliably [2].

Intracranial pathology may be linked with systemic effects that result in death or that contribute to the cause of death. For example, raised intracranial pressure due to an intracranial haemorrhage may result in systemic hypertension that precipitates rupture of an abdominal aortic aneurysm. Similarly, systemic disease and its treatment may have an effect on the central nervous system with impact upon the cause of death. Examples include cerebral infarction following myocardial infarction, metastatic malignancy, and fat embolism following femoral fracture. Examination of the brain should not add significantly to the time taken to perform the post-mortem examination or reconstruct the body in a well-run mortuary.

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The pathologist should be familiar with the techniques used to extract the brain, though in practise this procedure is normally performed by the anatomical pathology technician.

Examination of the pituitary gland, middle ears, eyes, and spinal cord are not required in every post-mortem examination, but the techniques used to examine these structures are included in this chapter and the pathologist engaged in autopsy practise should be familiar with their use.

This chapter initially details the standard dissection process for the brain and then shows the various pathologies linked to this tissue. The final part of the chapter focuses on the less commonly assessed areas: the eye, ear, and spinal cord.

## The Brain

### Extraction of the Brain

The brain may be extracted/eviscerated before or after evisceration of the organs within the torso. When there are concerns that death may be the result of pressure to the neck, the brain should be removed prior to opening the neck. This procedure helps to decompress the vessels within the neck, reducing the risk of generating artefactual haemorrhages (Prinsloo-Gordon haemorrhages [3]) within the neck during neck dissection.

### Fixing the Brain for Neuropathological Examination

In cases where microscopic pathology of the brain is suspected (for example, dementia or demyelinating disease), the brain may be retained and fixed to allow subsequent transport and detailed examination by a neuropathologist. The brain is fixed in formalin, which should be changed after 3 days, and then every week thereafter for 3 weeks. A minimum of 3 weeks of fixation is needed before the brain is transported to a neuropathologist.



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## Dissection of the Brain

Examination of the fresh brain is a relatively crude form of examination, allowing initially inspection of the external gross morphology, the meninges, the cranial nerves, and the gyral and sulcal surfaces (Figs. 9.1, 9.2, 9.3, 9.4, 9.5, 9.6, 9.7, 9.8, 9.9, 9.10, 9.11, 9.12, 9.13, 9.14, 9.15, 9.16, 9.17, 9.18, 9.19, 9.20, 9.21, 9.22, 9.23, 9.24, 9.25, 9.26, and 9.27). It should also be sufficient, after sectioning, to allow review of the internal parenchyma.

**Fig. 9.1** The hair is parted and the scalp is incised over the occiput from ear to ear

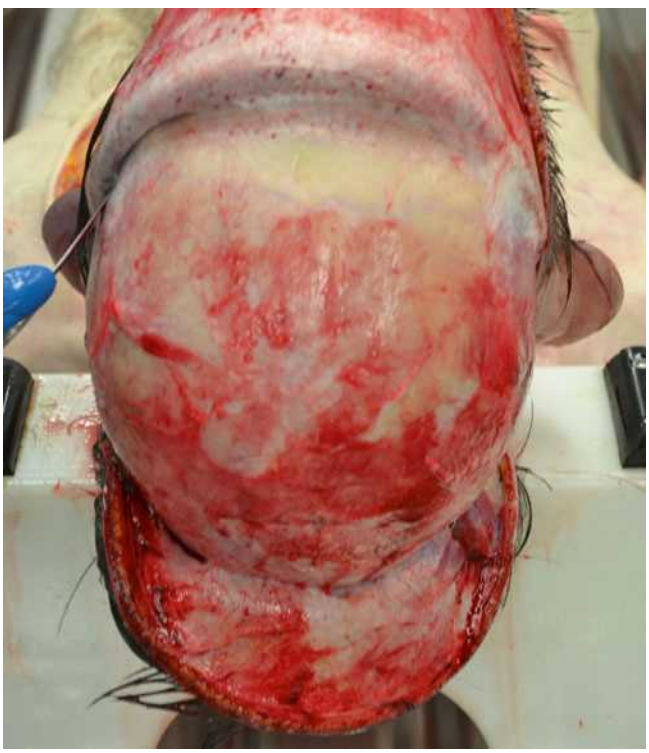

**Fig. 9.2** The scalp is reflected

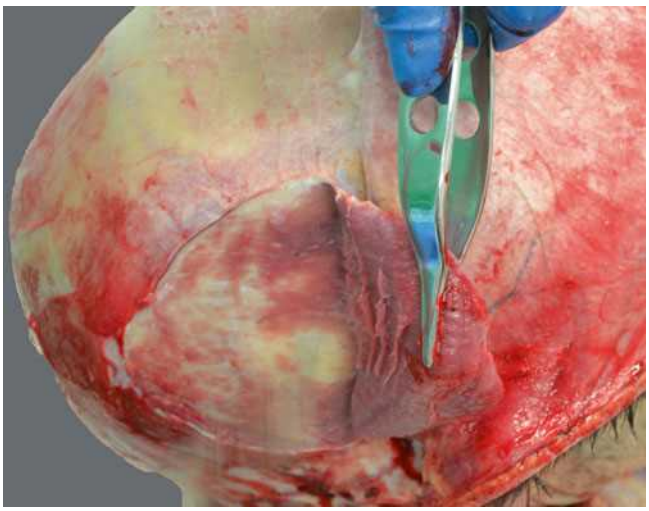

**Fig. 9.3** The temporalis muscles are incised and reflected

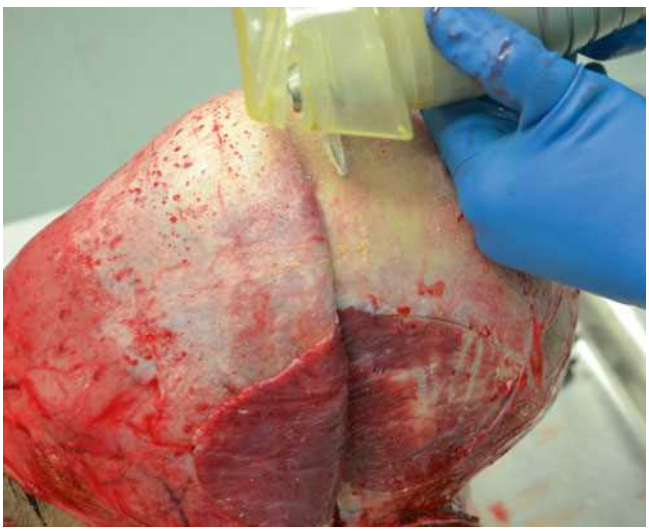

**Fig. 9.4** Incising the skull is normally performed with a motorised saw with extractor unit

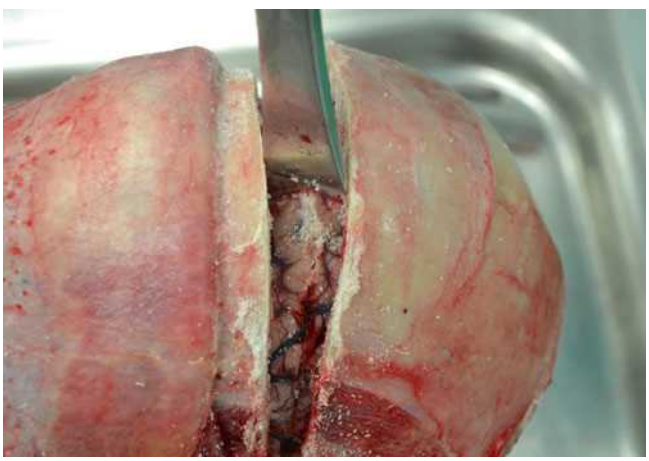

**Fig. 9.5** The calvaria are separated from the skull base using a skull key

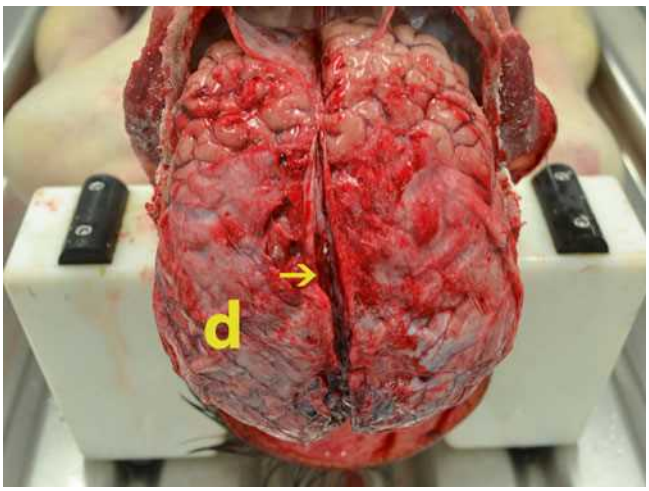


**Fig. 9.6** The brain and dura are exposed by lifting off the skull cap. The dura (*d*) may be seen on the brain surface, and the superior sagittal sinus (*arrow*) has been opened

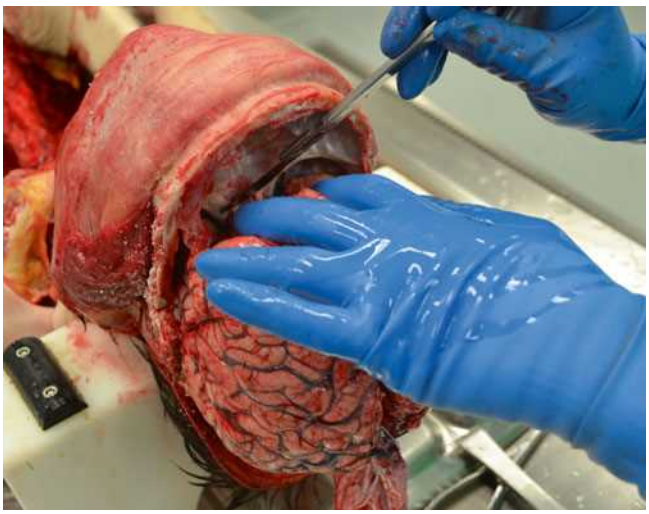

**Fig. 9.7** Working from front to back under the brain, the cranial nerves and spinal cord are transected

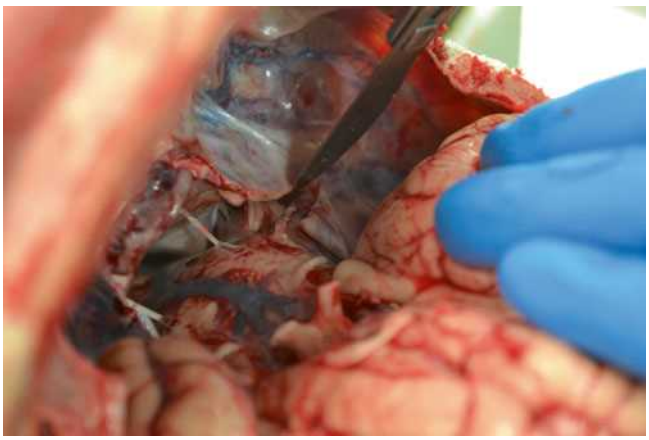

**Fig. 9.8** The falx cerebri and tentorium cerebelli are incised

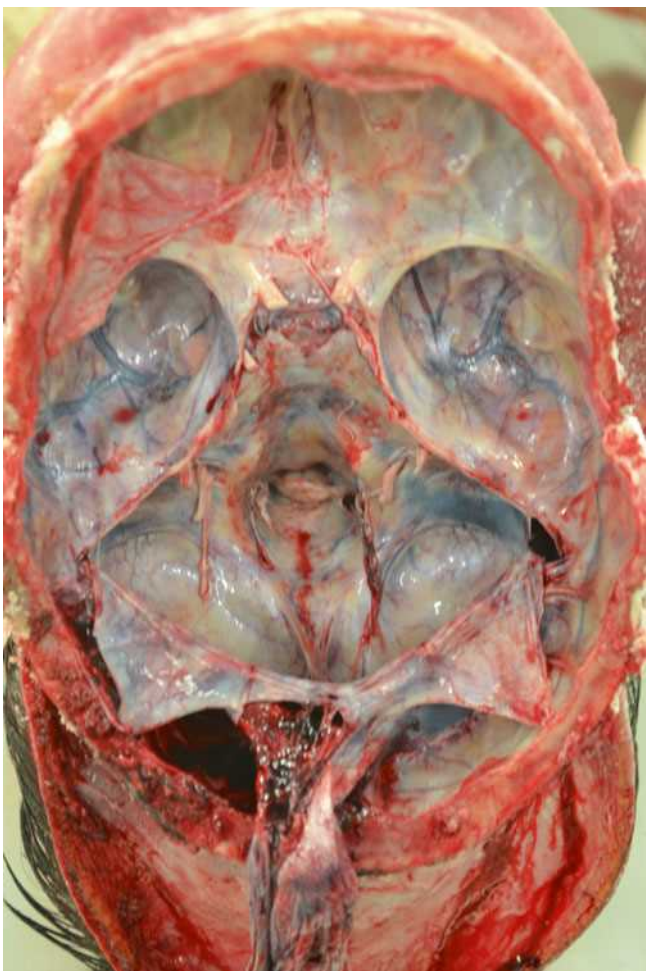

**Fig. 9.9** The brain is lifted free from the head, exposing the lower cranial cavity boundary

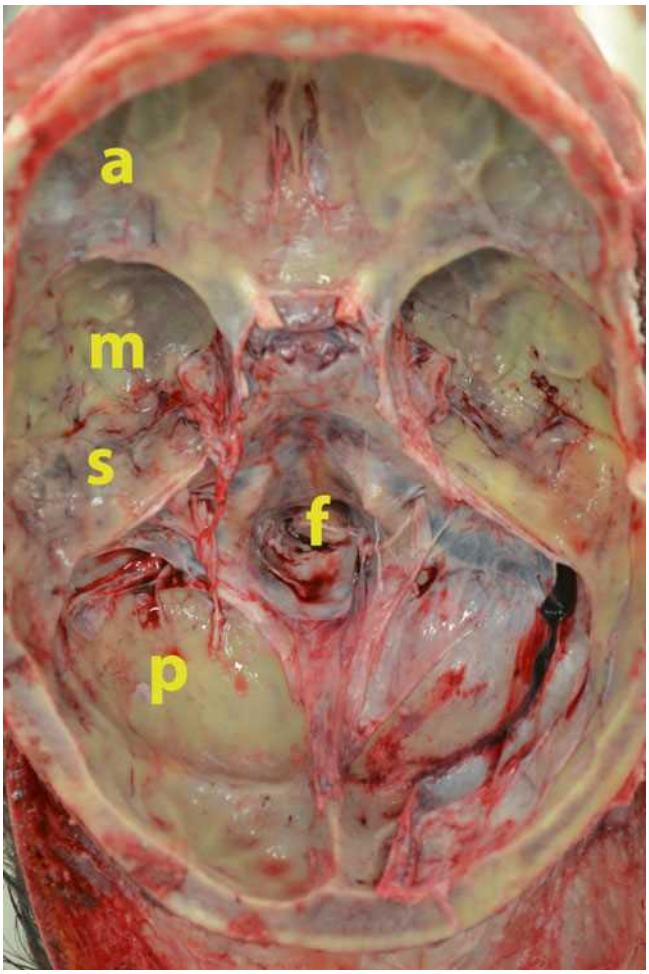


**Fig. 9.10** The dura is stripped, exposing the skull base. One may identify the anterior fossa (*a*), middle fossa (*m*), posterior fossa (*p*), transected cord in the foramen magnum (*f*), and sphenoid bone tissue (*s*)

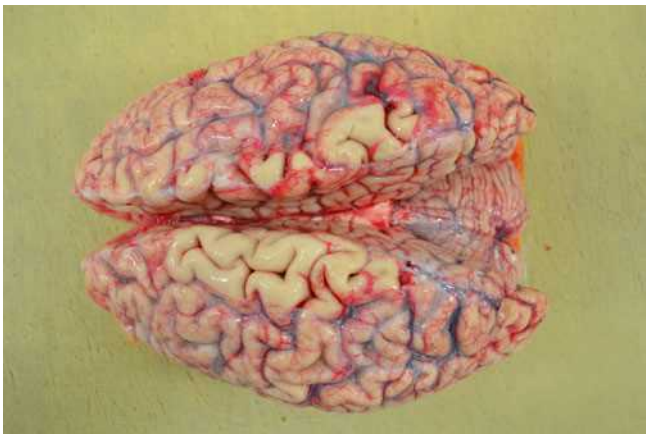

**Fig. 9.12** The brain is inspected: superior surface

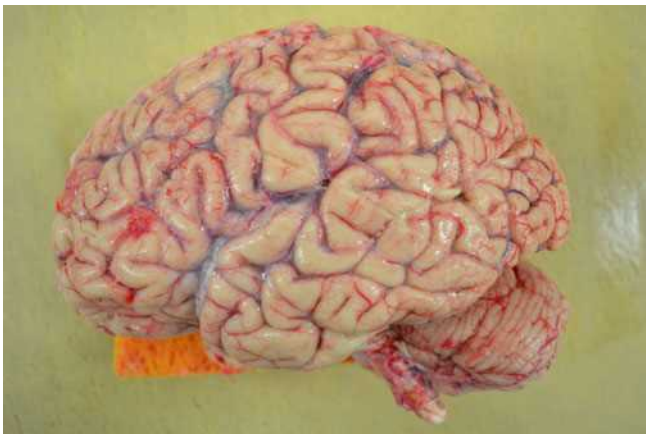

**Fig. 9.13** The brain is inspected: lateral view, using a sponge to balance the brain on edge

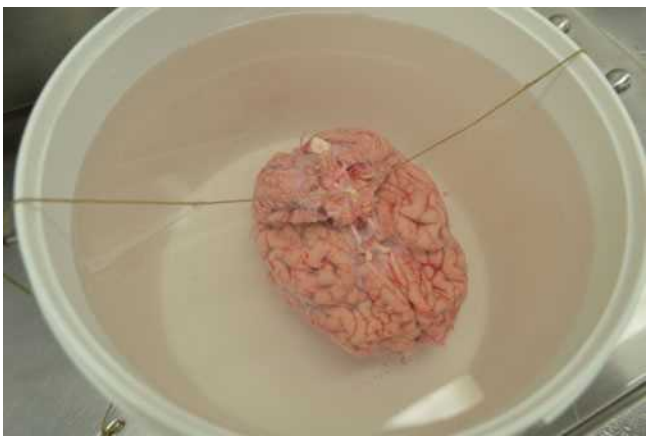

**Fig. 9.11** The brain is suspended in a bucket of 10 % neutral buffered formalin by a string that is passed beneath the basilar artery. The ends of the string are tied to the bucket handles, thus suspending the brain in the formalin

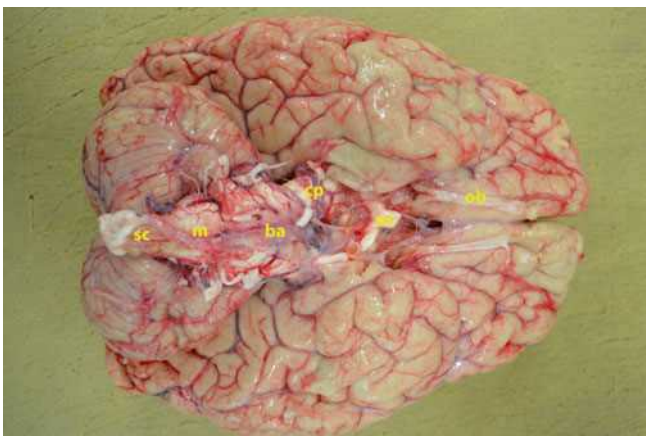


**Fig. 9.14** The brain is inspected: basal view showing olfactory bulbs (*ob*), optic chiasm (*oc*), cerebral peduncle (*cp*), basilar artery (*ba*), medulla (*m*), and spinal cord (*sc*)

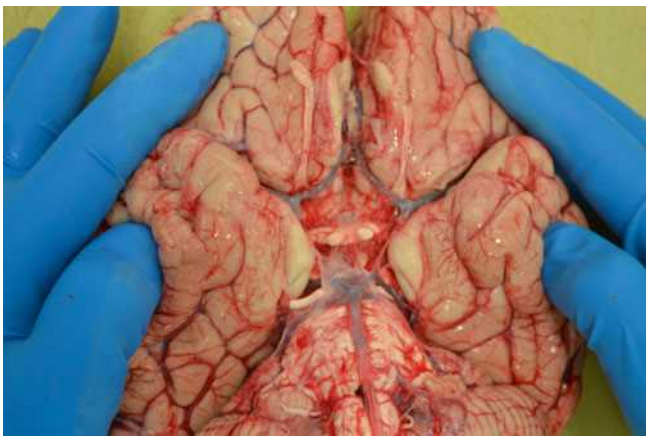

**Fig. 9.15** The circle of Willis is inspected and may be dissected free for demonstration purposes

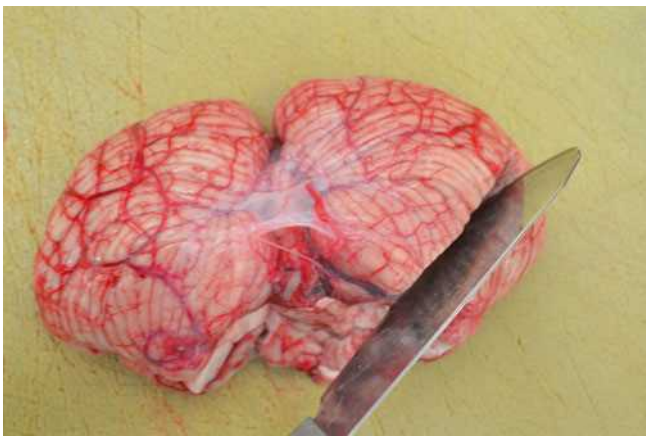

**Fig. 9.18** The cerebellum is radially sliced (Alternatively, the cerebellum can be bisected in a horizontal fashion)

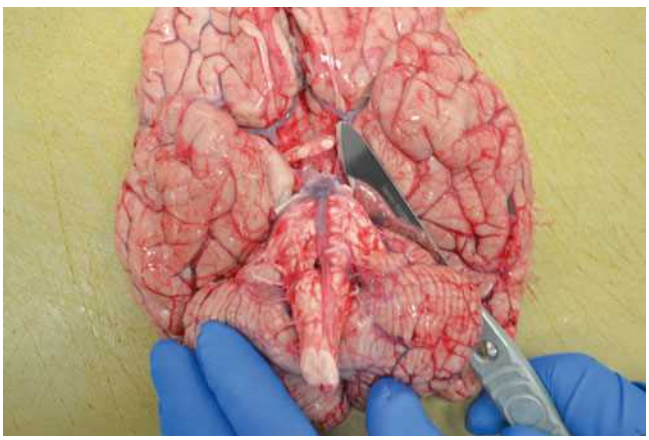

**Fig. 9.16** The cerebellum and brainstem are removed

**Fig. 9.19** The medulla and pons are sliced at 5-mm intervals

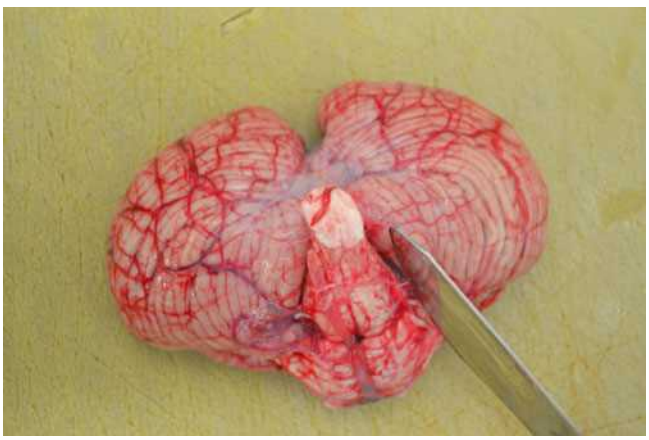

**Fig. 9.17** The cerebellar peduncles are divided

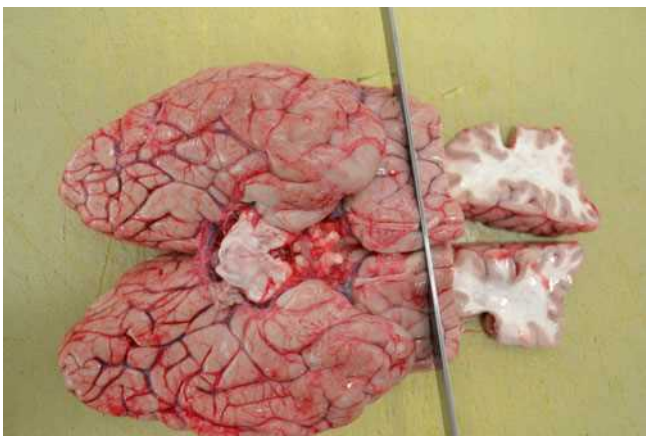

**Fig. 9.20** The brain is coronally sliced at 1 to 2 cm intervals

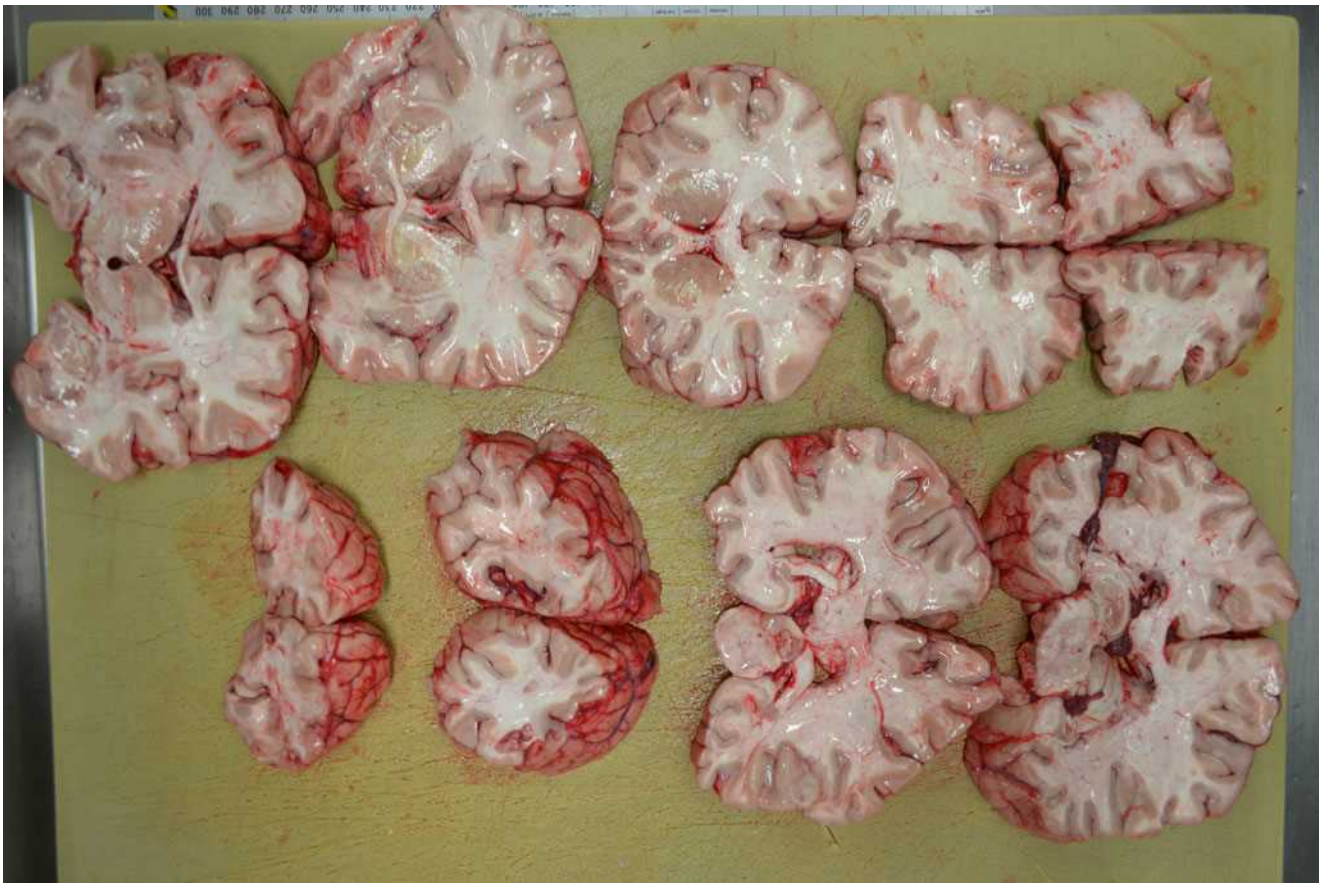

**Fig. 9.21** The brain slices can be laid out for demonstration

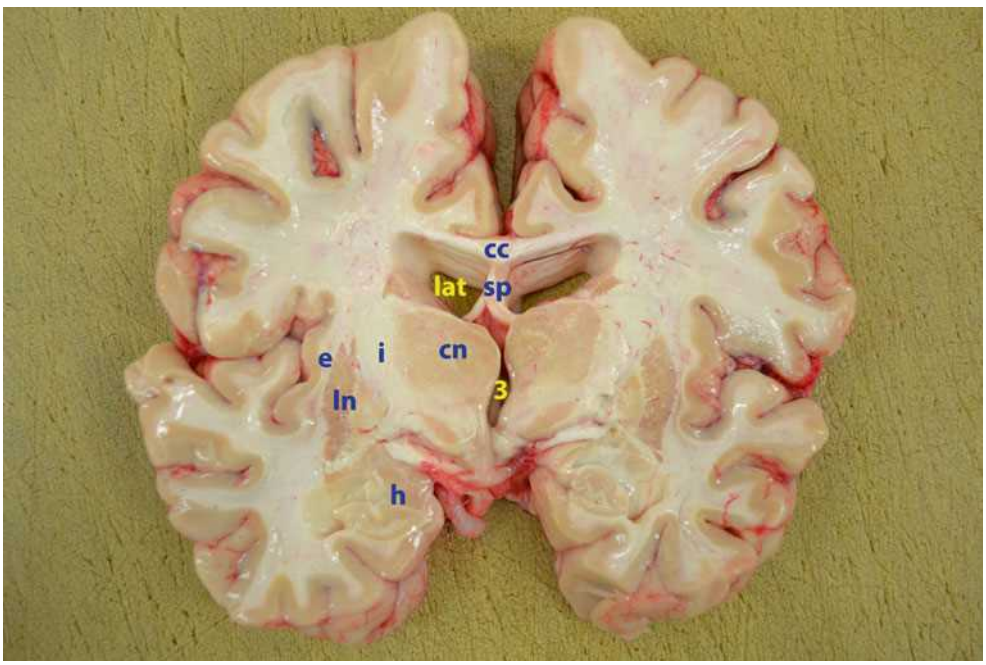


**Fig. 9.22** Neuroanatomy visible in a coronal section at the level of the mammillary bodies: Corpus callosum (*cc*), septum pellucidum (*sp*), lateral ventricle (*lat*), caudate nucleus (*cn*), third ventricle (*3*), hippocampus (*h*), lentiform nucleus (*ln*), external capsule (*e*), and internal capsule (*i*)

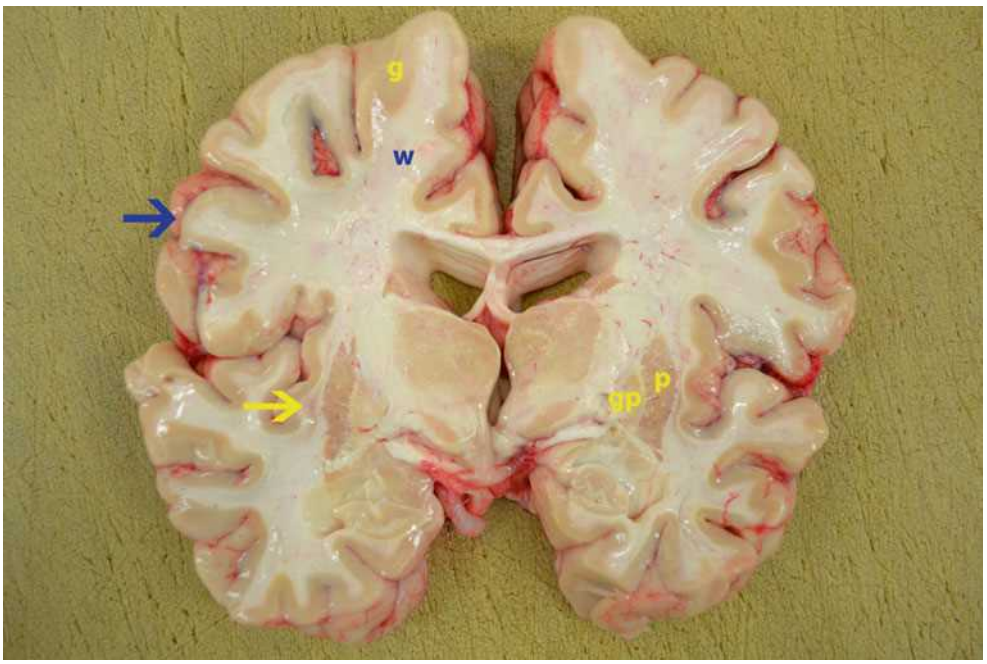


**Fig. 9.23** Neuroanatomy visible in a coronal section at the level of the mammillary bodies: Putamen (*p*), globus pallidus (*gp*), white matter (*w*), grey matter (*g*), surface arachnoid/pia meninges (*blue arrow*), claustrum (*yellow arrow*)

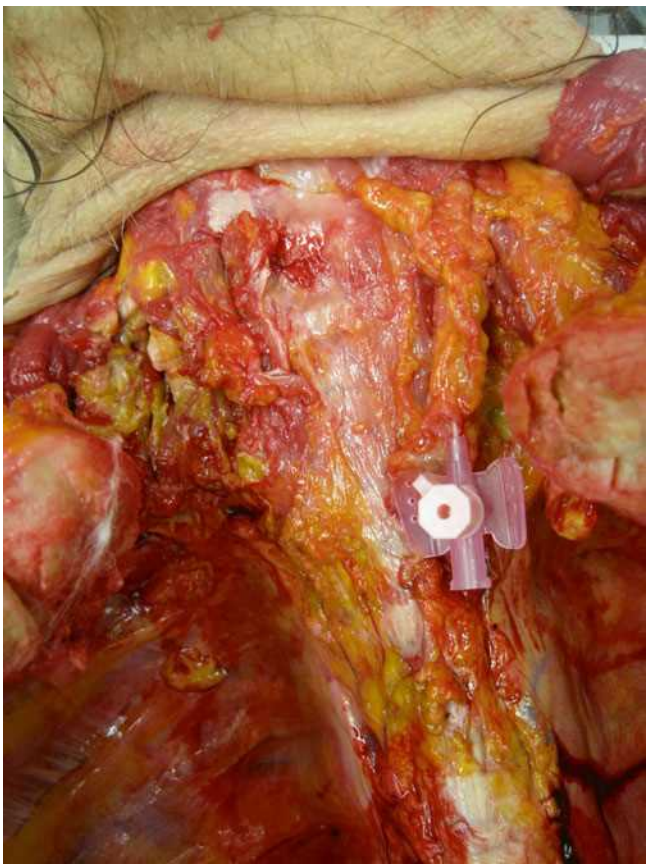

**Fig. 9.24** Testing the basilar artery system can involve dissecting out the artery on both sides of the neck. Alternatively, one can cannulate and perfuse the artery at the start of the subclavian artery

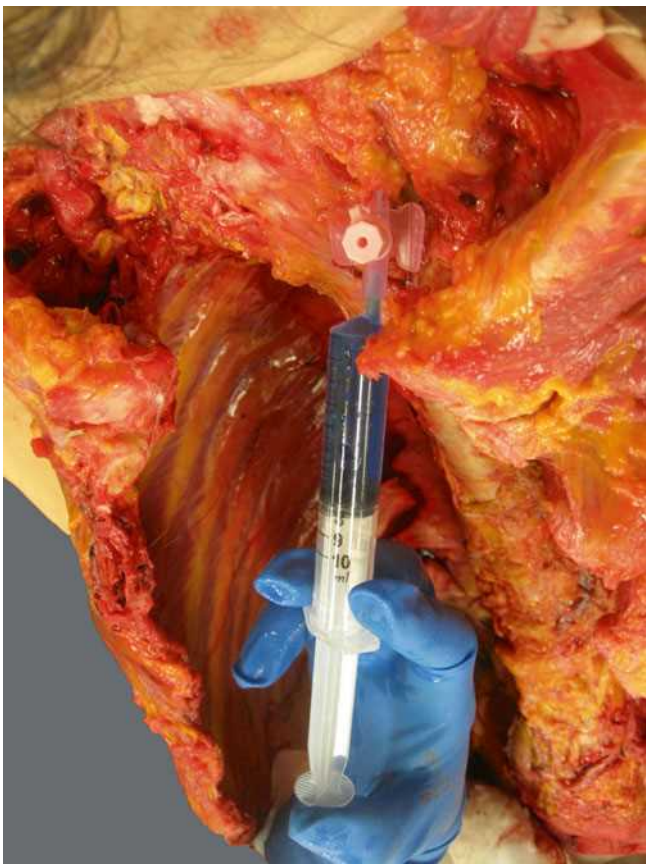

**Fig. 9.25** The artery is instilled gently with water containing blue dye

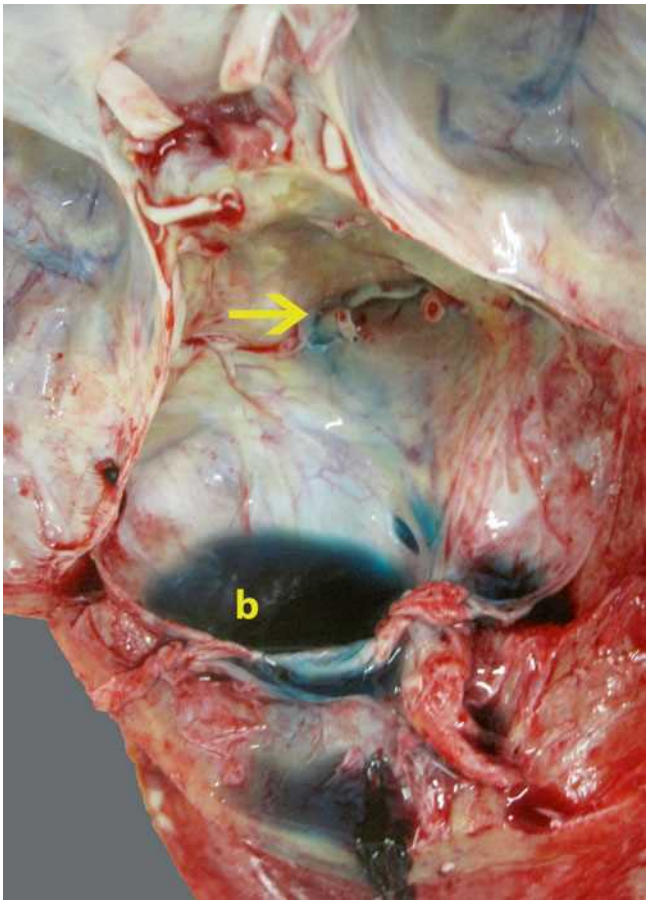


**Fig. 9.26** This artery was patent; blue dye is seen emerging from the cut end of the vertebral artery (*arrow*), pooling as blue water (*b*) in the posterior fossa

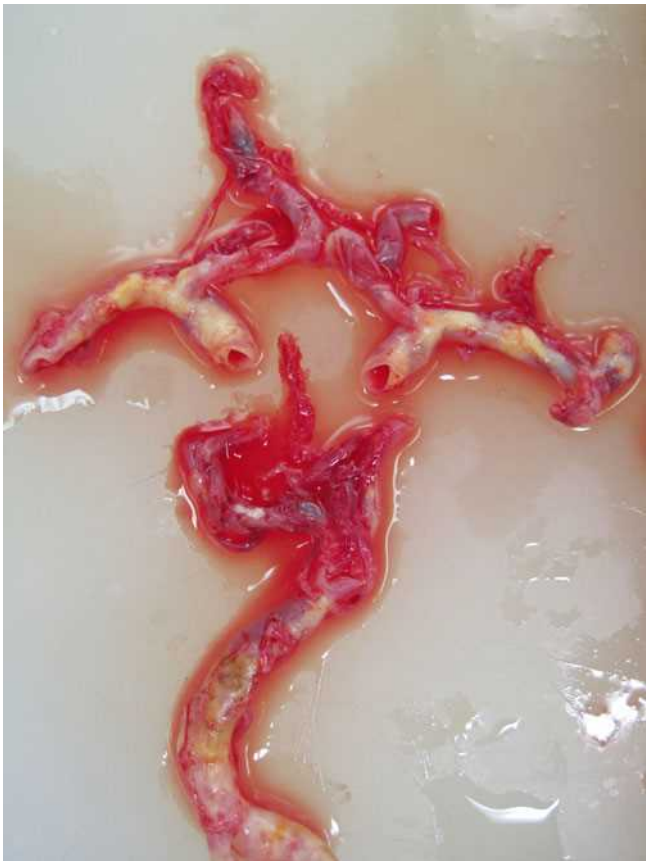

**Fig. 9.27** The circle of Willis can be dissected from the base of the brain, but atheromatous disease can make the tissues both rigid and fragile, often making dissection difficult. Atheromatous disease is strongly associated with cerebrovascular ischaemia and cerebral infarction

## Common Pathologies

Figures 9.28, 9.29, 9.30, 9.31, 9.32, 9.33, 9.34, 9.35, 9.36, 9.37, 9.38, 9.39, 9.40, 9.41, 9.42, 9.43, 9.44, 9.45, 9.46, 9.47, 9.48, 9.49, 9.50, 9.51, 9.52, 9.53, 9.54, 9.55, 9.56, 9.57, 9.58, 9.59, 9.60, 9.61, 9.62, 9.63, 9.64, 9.65, 9.66, 9.67, 9.68, and 9.69 illustrate a variety of pathologies that may be identified. Trauma to the head may result in skull fractures or bleeding between the skull and dura (extradural haematoma) or between the dura and arachnoid layers (subdural haematoma). Berry aneurysms of the circle of Willis are common, and rupture of such an aneurysm results in subarachnoid haemorrhage. Subarachnoid haemorrhage also may complicate a cerebral haemorrhage. Occlusion of the cerebral vasculature results in cerebral infarction. Benign tumours of the meninges (meningiomas) are common, and are usually asymptomatic, incidental autopsy findings. Tumours from many sites, particularly the lungs, may metastasise to the brain.

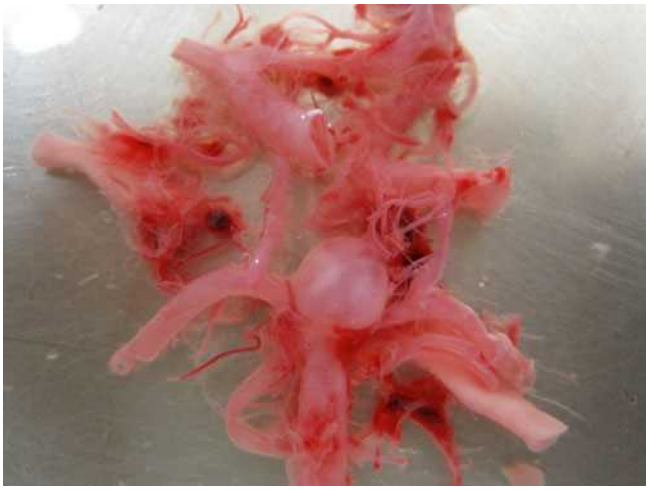

**Fig. 9.28** The circle of Willis is a common site for aneurysmal change. This circle of Willis has been dissected and then floated in water to show the relative positions of the arteries, as well as a saccular aneurysm close to the origin of the posterior cerebellar artery

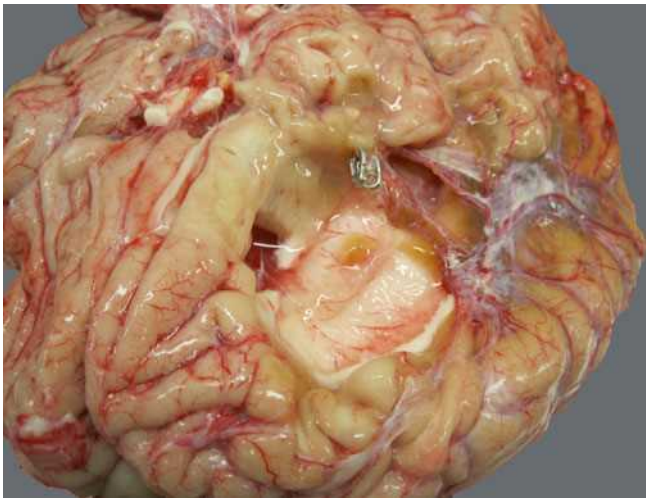

**Fig. 9.30** Cerebral infarction (haemorrhagic or ischaemic) often undergoes liquefactive degeneration. The relationship to an aneurysmal vessel had been recognised earlier in this patient. A surgical clip had been placed, controlling the flow of fluid through the diseased vessel and tributaries, but it had had no beneficial effect on the already infarcted brain tissues

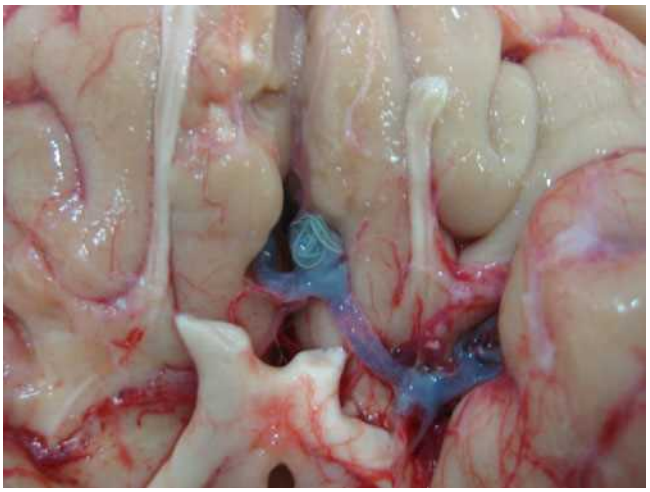

**Fig. 9.29** This aneurysm has been treated by placing a metal coil within the dilated, aneurysmal artery. This process aims to induce local thrombosis and diminish the risk of any subsequent haemorrhage from the weakened artery site

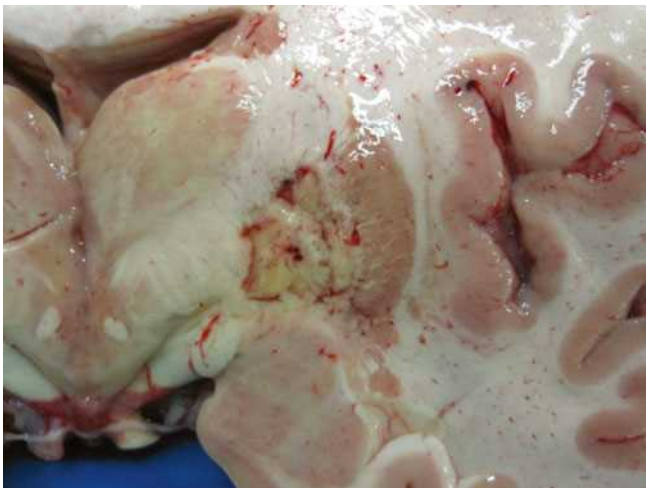

**Fig. 9.31** Hypoxic brain damage can occur in relation to hypotension (in this case a cardiac arrest). Yellow discolouration of the deep nuclei is present, with some swelling of the tissues. Liquefactive degeneration has not yet occurred

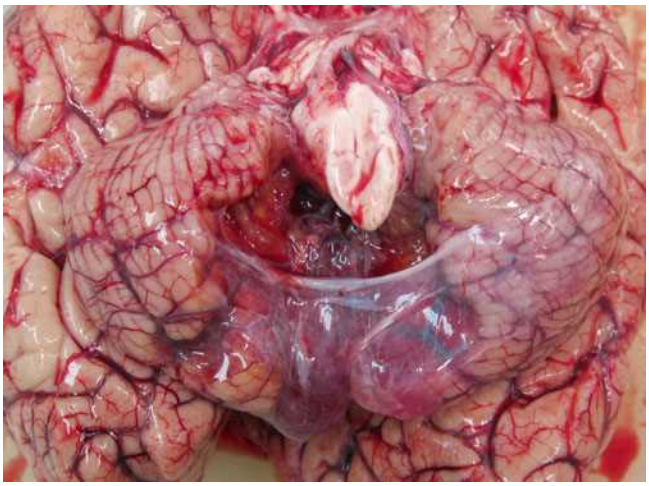

**Fig. 9.32** Old cerebellar infarction is often seen as a cystic area, in this case involving the central inferior cerebellar aspect

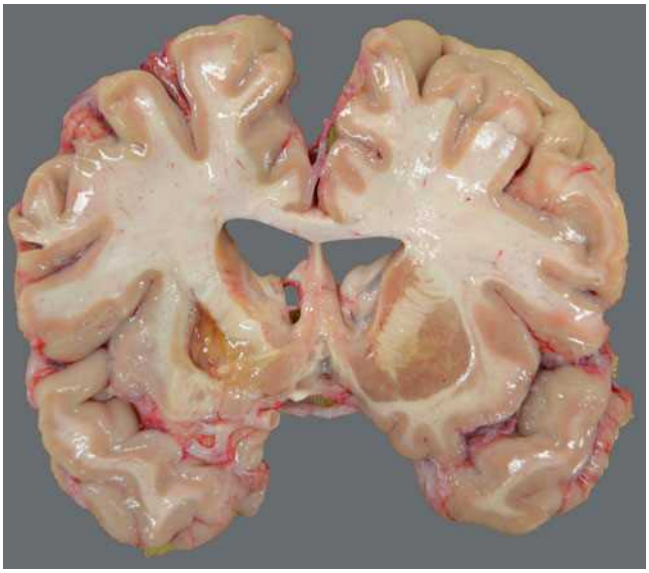

**Fig. 9.34** A lacunar infarction in the cerebral deep nuclei is noted, with cystic degeneration. The yellow discolouration is due to focal haemosiderin deposition. This suggests that the lesion may have been haemorrhagic in origin

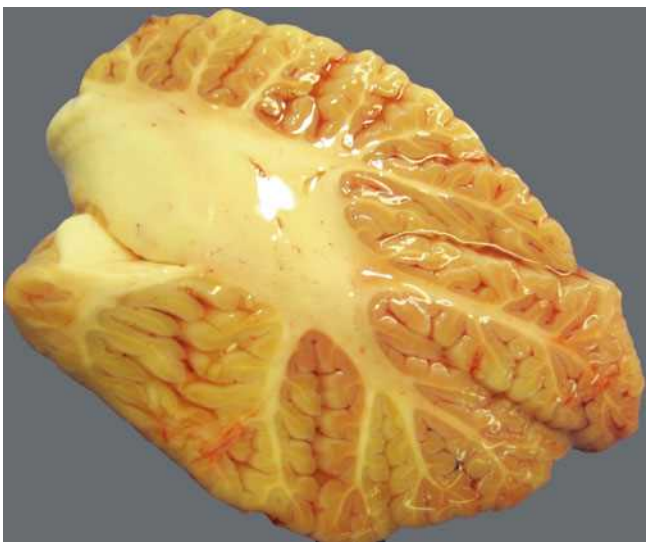

**Fig. 9.33** Hypoxic brain injury can cause localised swelling due to ischaemic damage. Very early changes are seen as swollen brain parenchyma, which is evident in this view of the cerebellum, where the inferior and posterior pole of the tissues appears more prominent, firm, and closely opposed than that of the superior surface

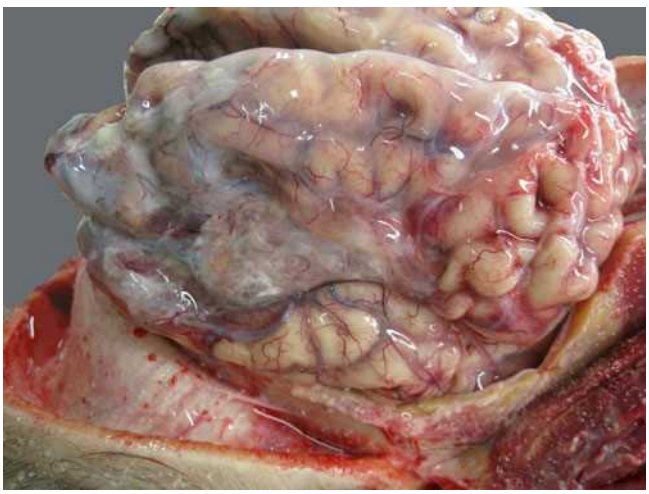

**Fig. 9.35** Significant brain injury can occur at birth. This adult had experienced significant hypoxic injury in the immediate perinatal period, resulting in gross tissue loss from the right cerebral hemisphere, and to a lesser degree the left. Significant neurological deficit was present throughout life, involving higher intellect functions as well as motor and sensory skills

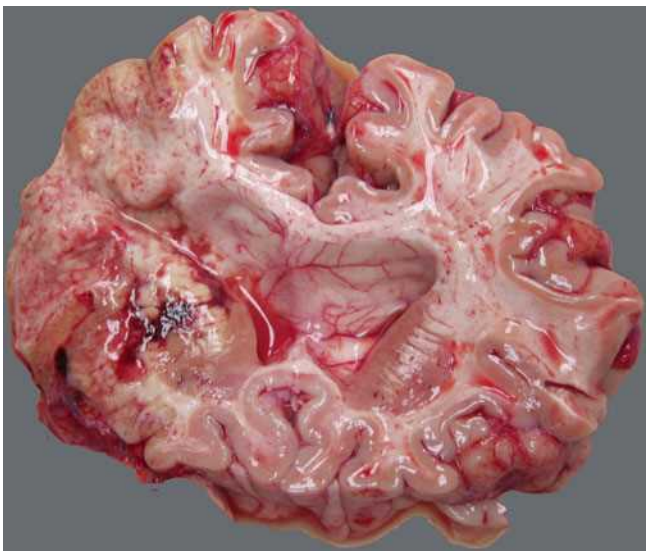

**Fig. 9.36** Brain injury in relation to localised trauma (road traffic collision) can show focal infarction and localised haemorrhage, often after several weeks. These findings mimic cerebrovascular disease, but clearly the history in this case is paramount. Of particular importance is establishing whether the cerebral lesion was the cause of the road collision or a consequence

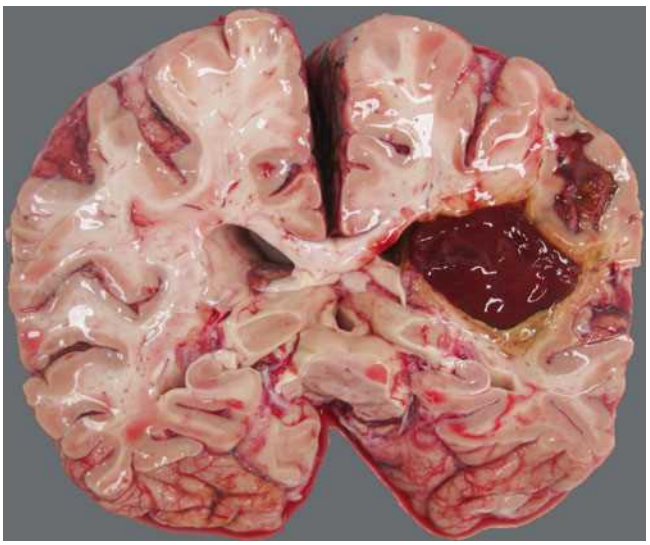

**Fig. 9.37** The parietal tissues show yellow discolouration and tissue loss in a case of cerebral infarction with secondary haemorrhage

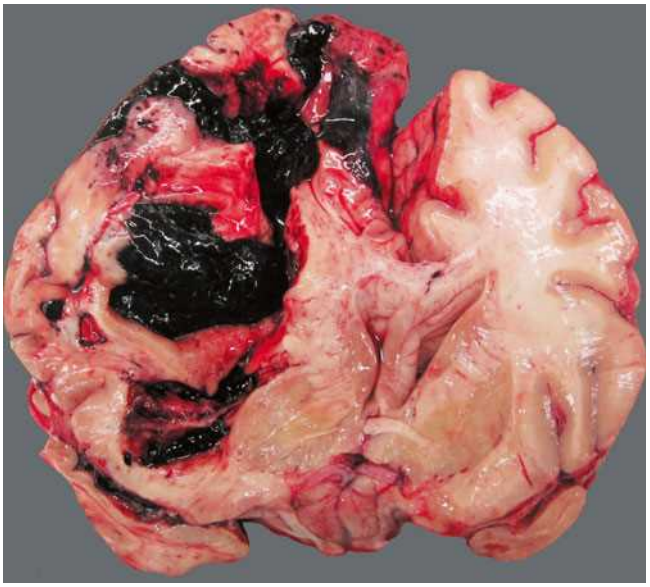

**Fig. 9.38** Fresh cerebral haemorrhage can be often be associated with massive brain disruption, causing death promptly

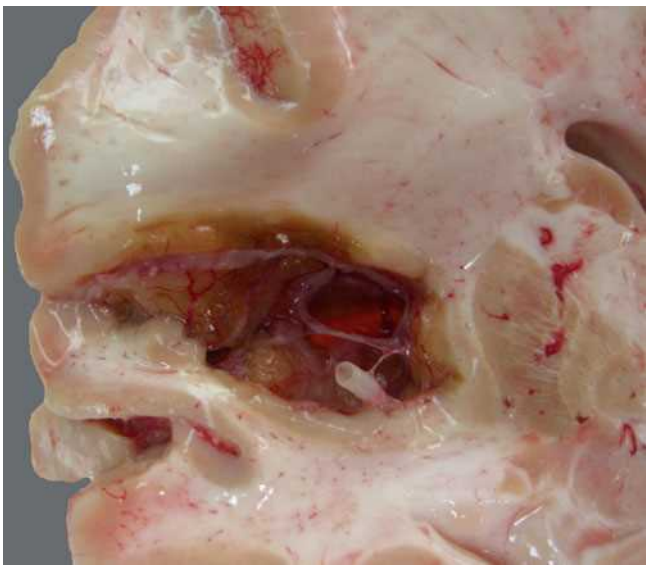

**Fig. 9.39** An old cystic infarct is seen in the brain parenchyma. Such cysts, with yellow discolouration, are often termed *apoplectic cysts* and reflect infarction with prior bleeding

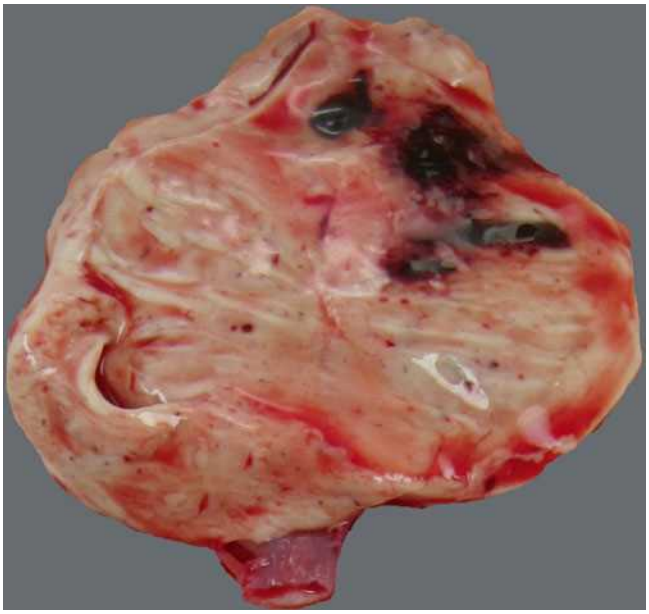

**Fig. 9.40** Haemorrhages in the brainstem, midbrain, and pons can occur in situations of raised intracranial pressure. These 'Duret' haemorrhages are a secondary consequence of the raised intracranial pressure and shearing of the brainstem tissues and blood vessels rather than primary pathology

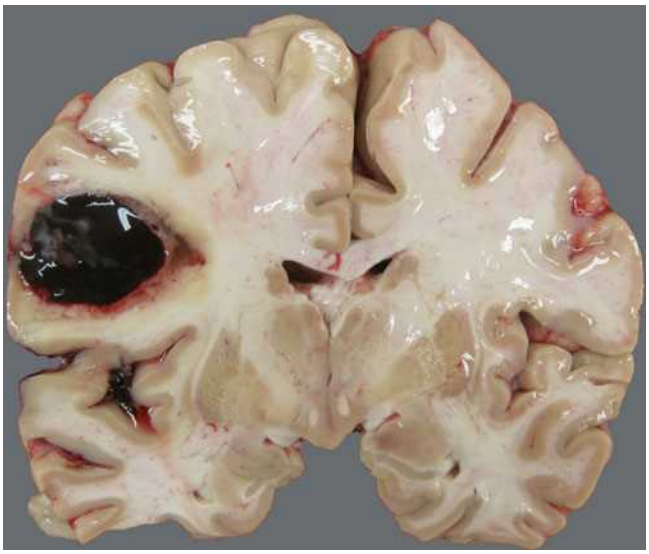

**Fig. 9.41** Examination of brain tissues following haemorrhage may be fraught with difficulty in that the brain parenchyma is soft and distorted. Some would say that radiological investigation of such events is superior to transverse slicing of the fresh brain. If significant study of the brain parenchyma is required, then the tissues ideally must be fixed for about 6 weeks and then sliced. Despite all these limitations, however, slicing the brain in a fresh state and taking photographs can provide significant information for clinical teams. This image shows a large, spontaneous cerebral bleed with some midline (mild left to right) shift

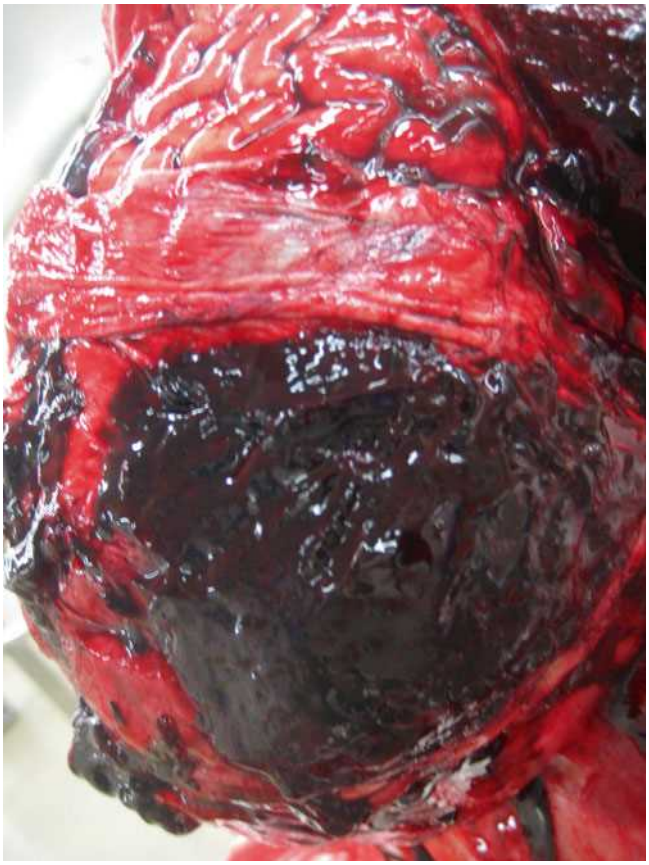

**Fig. 9.42** An extradural haemorrhage is seen, with blood that has accumulated above the dura and below the skull vault. This haemorrhage commonly follows significant head trauma tearing the middle meningeal artery

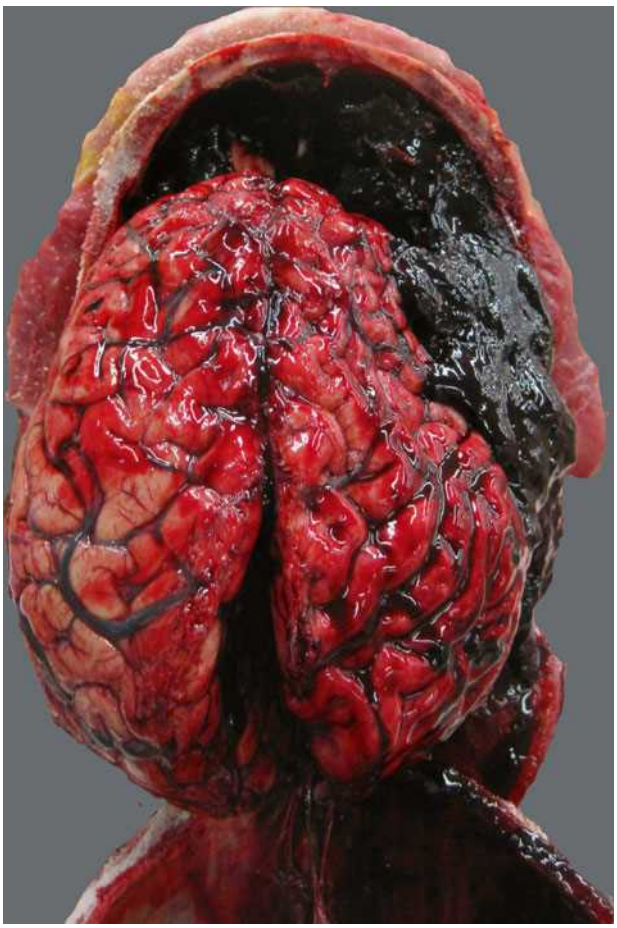

**Fig. 9.43** Subdural haemorrhage can be associated with significant bleeding, with compression of the local brain

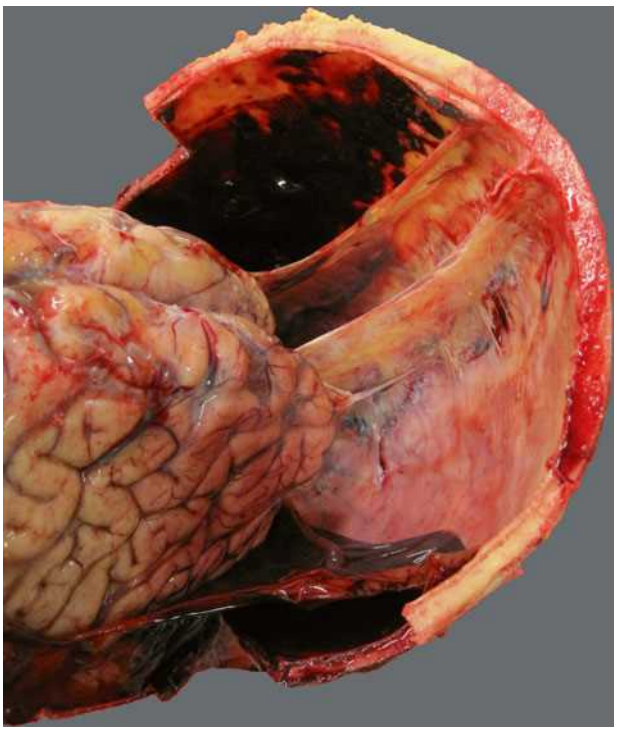

**Fig. 9.44** Subdural bleeding is seen under the right side dura as one removes the skull cap. One should try to estimate the volume of blood that has accumulated

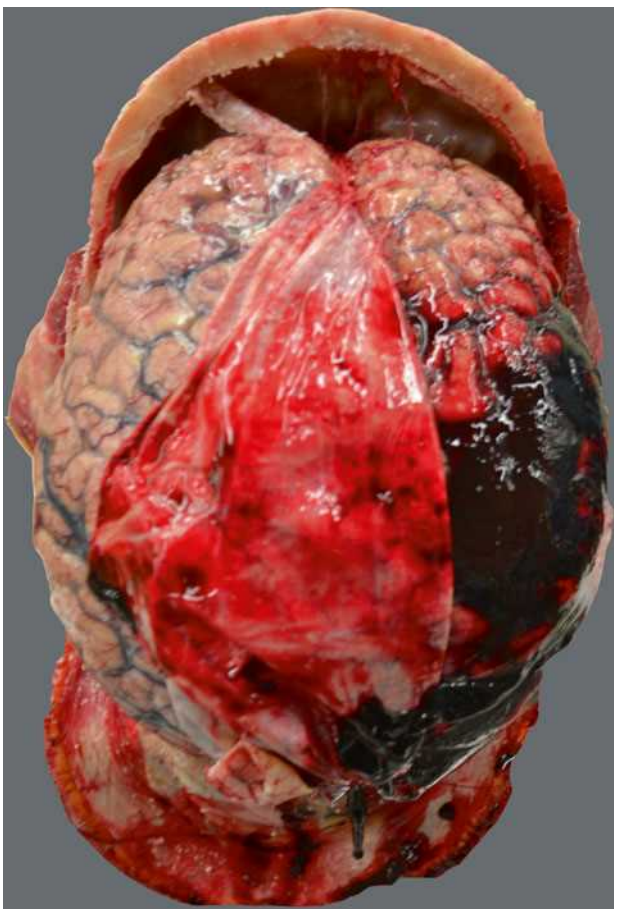

**Fig. 9.45** Subdural haemorrhage is seen with the dura reflected

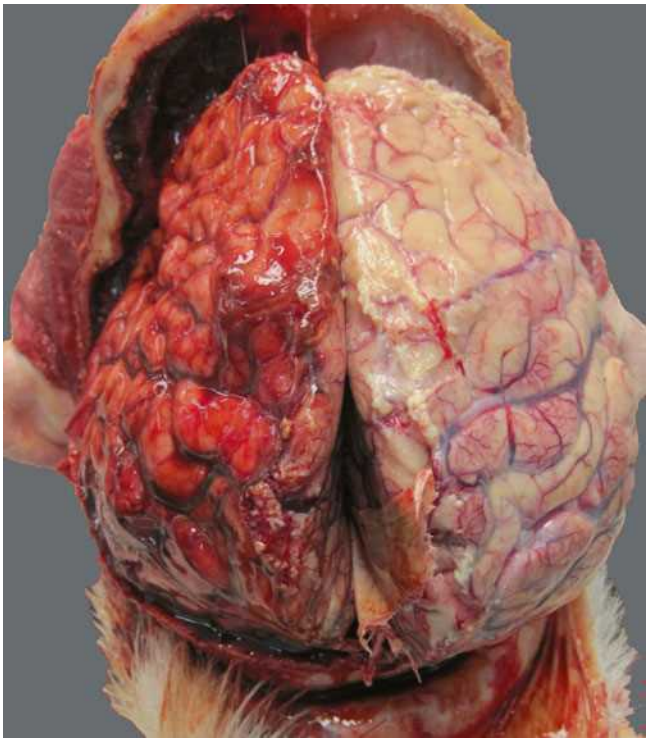

**Fig. 9.46** Old subdural haemorrhage is often associated with some flattening of the left cerebral hemisphere and yellow discoloration of the meninges

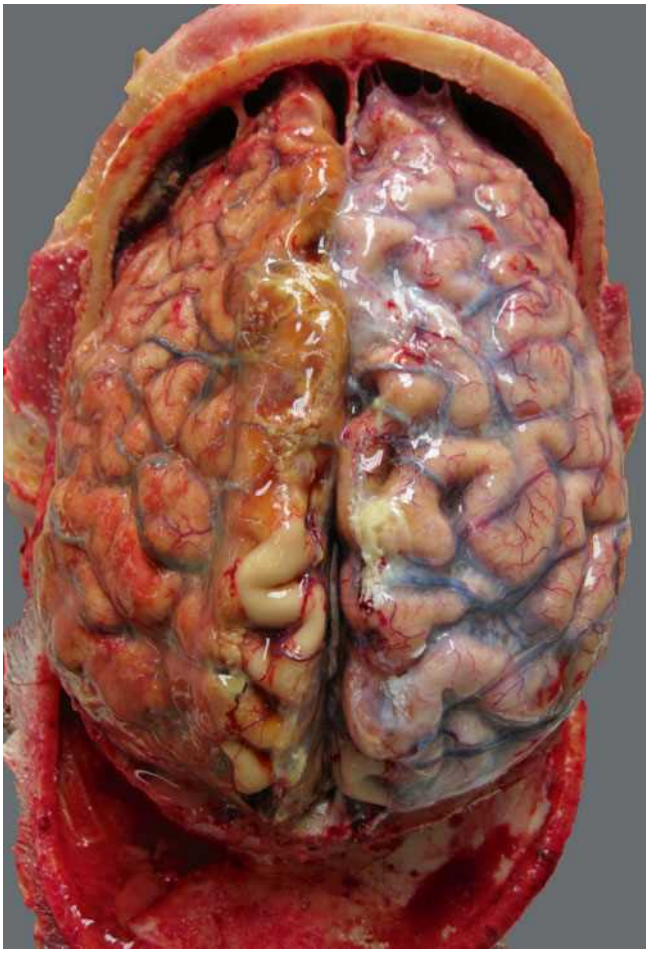

**Fig. 9.47** Chronic subdural haemorrhage is seen with blood-stained discolouration of the outer brain tissues and a hint of yellow discolouration

**Fig. 9.48** Significant bleeding around the brainstem has been identified during brain tissue removal. Such changes can occur in relation to fractures of the skull base or subarachnoid haemorrhages due to aneurysms or vertebral artery transection

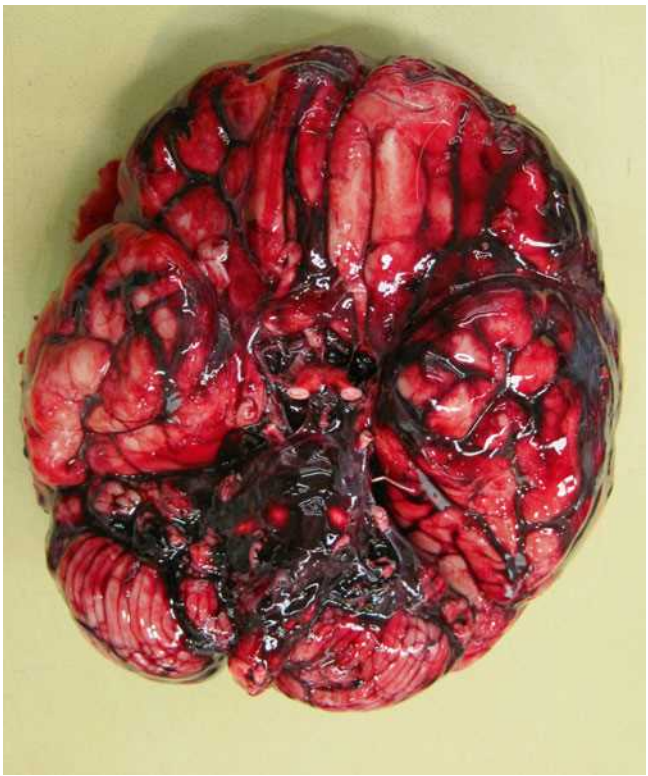

**Fig. 9.49** Significant subarachnoid haemorrhage is seen in a case of circle of Willis aneurysm. The heavy contamination of blood around the undersurface of the brain makes identification of the bleeding point difficult

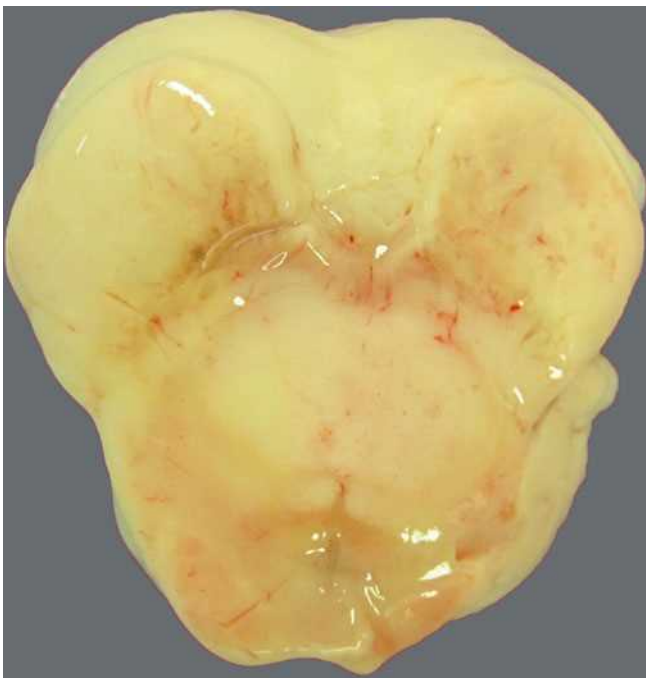

**Fig. 9.50** A section through the midbrain shows significant loss of pigmentation at the site of the substantia nigra in a case of idiopathic Parkinson's disease. Identification of this process in relation to falls, accidents, and natural disease (chest infections) is of value, as there is a strong link between the neurological impairment and secondary pathologies such as subdural haemorrhage

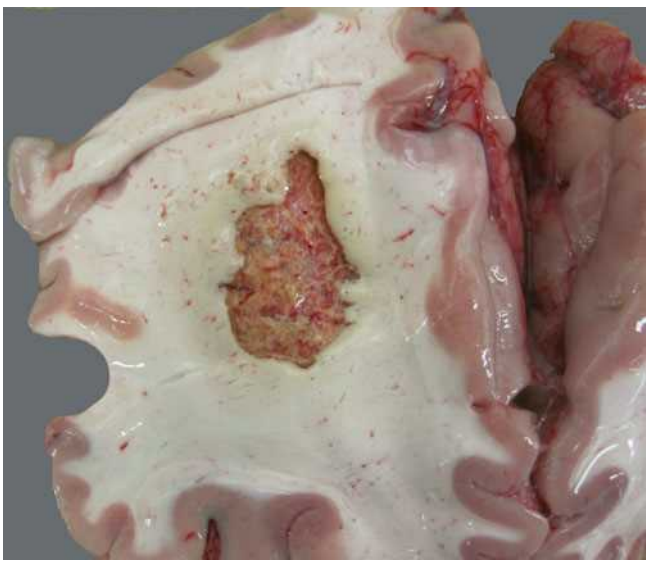

**Fig. 9.51** A central nervous system abscess is seen in a case of an intravenous drug user. Surprisingly, there were no ante-mortem symptoms, and the individual died as a result of an overdose

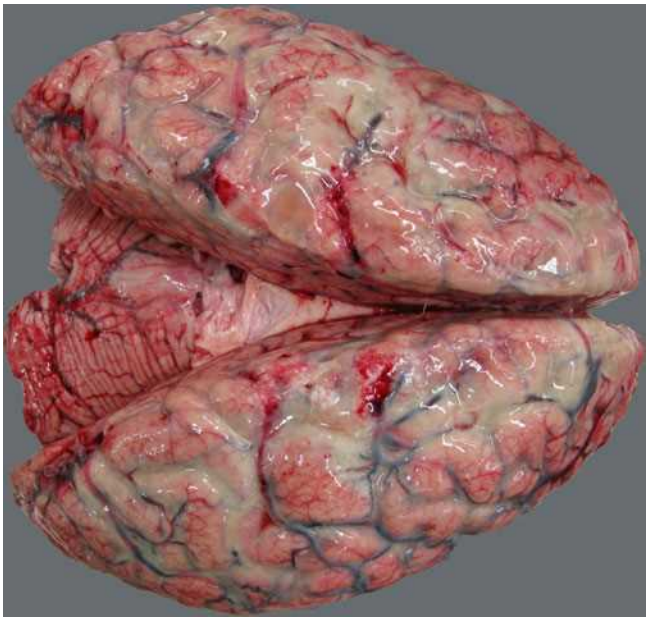

**Fig. 9.52** Purulent meningitis is seen in a case of a young adult student. This is a relatively rare, but well recognised, cause of sudden death. Material should always be sent to microbiology for typing of the organism, and notification of local authorities is often required. Contact tracing may be relevant

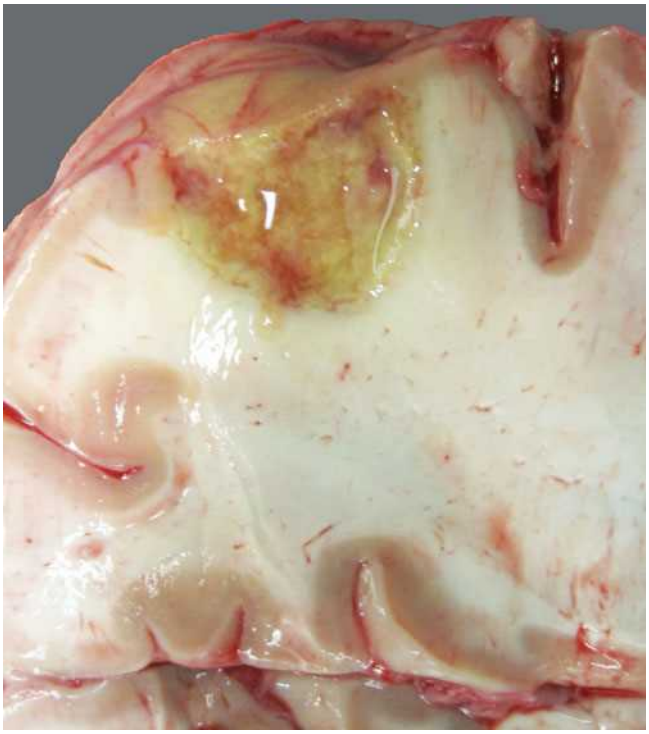

**Fig. 9.54** Metastatic neoplasia involving the brain often appears as relatively well-defined lesions. This case shows a largely necrotic, cystic focus of metastatic lung cancer involving the outer grey cortical ribbon

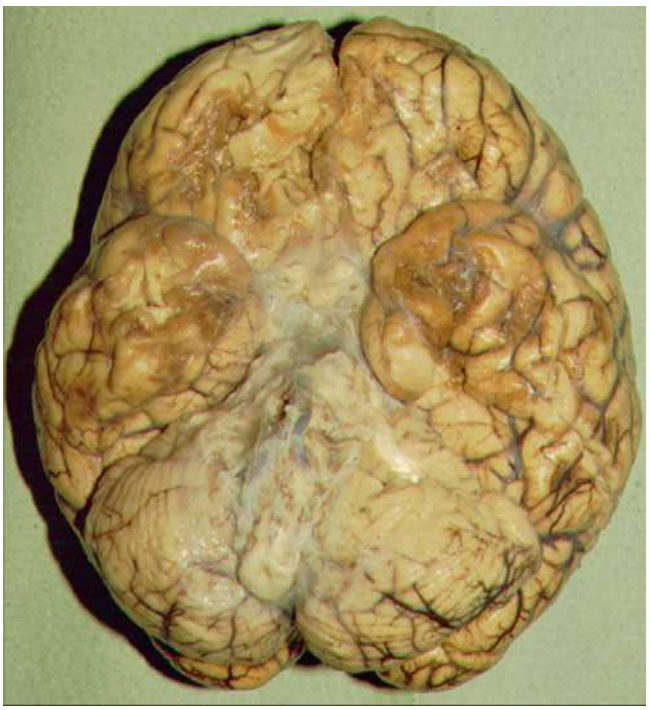

**Fig. 9.53** *Mycobacterium avium-intracellulare* meningitis in an individual with HIV infection

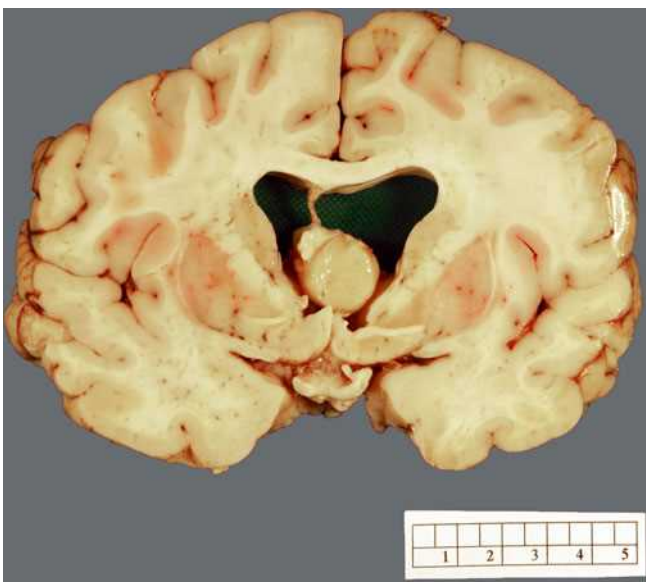


**Fig. 9.55** A colloid cyst of the third ventricle. This is a cause of sudden death

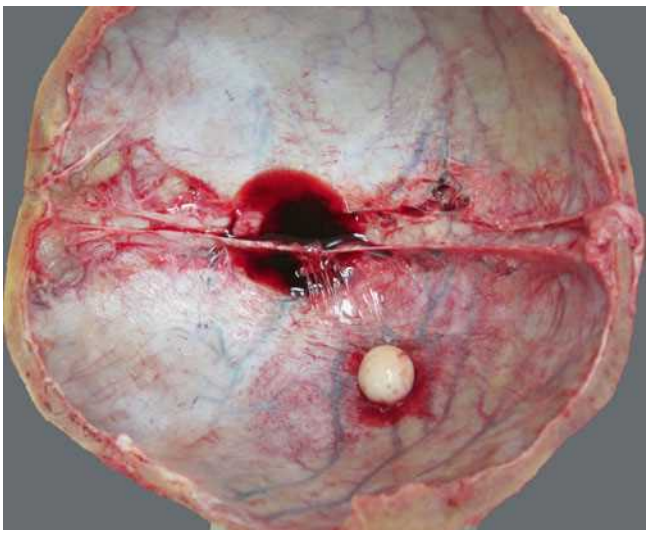

**Fig. 9.56** This meningioma was an incidental finding at autopsy

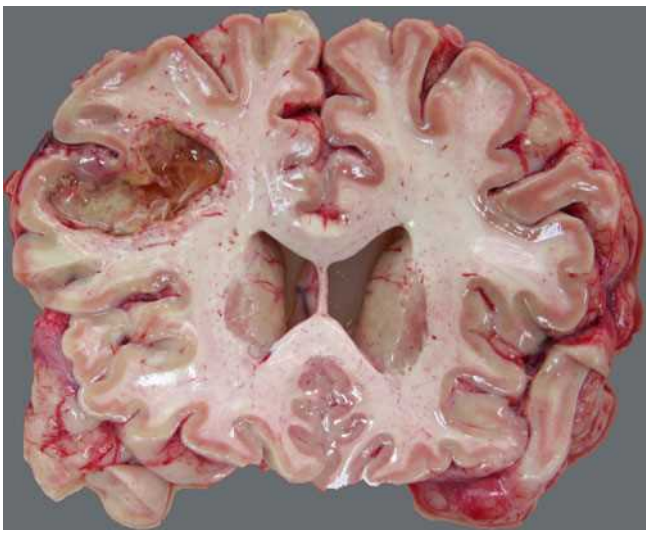

**Fig. 9.58** Gliomatous disease is often identified before death and treated. The pronounced cystic degeneration and necrosis points to significant tumour killing by means of external beam radiotherapy

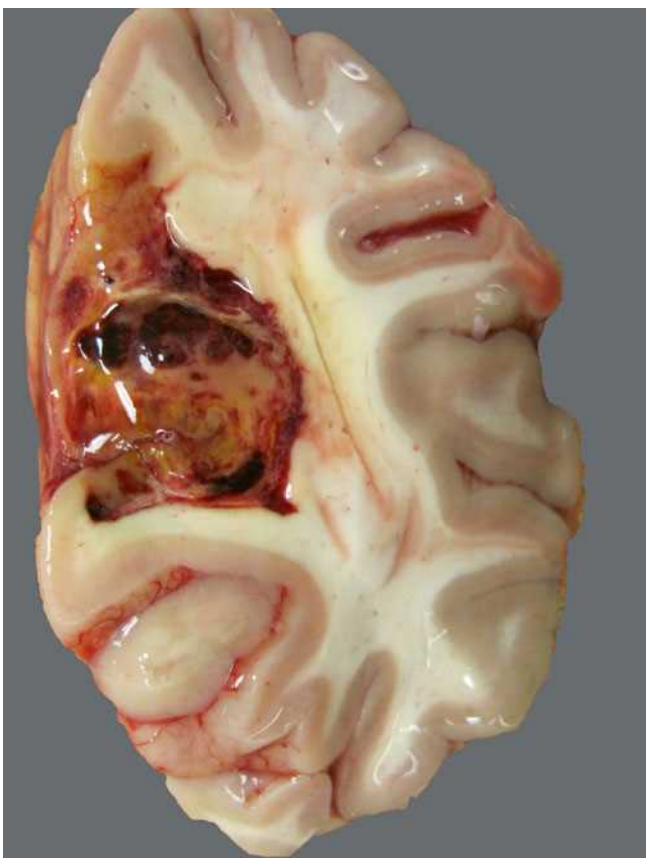

**Fig. 9.57** Primary brain neoplasia is an uncommon finding at autopsy, although it does explain sudden death on occasion. This case shows a high-grade glioma with significant brain destruction and necrosis

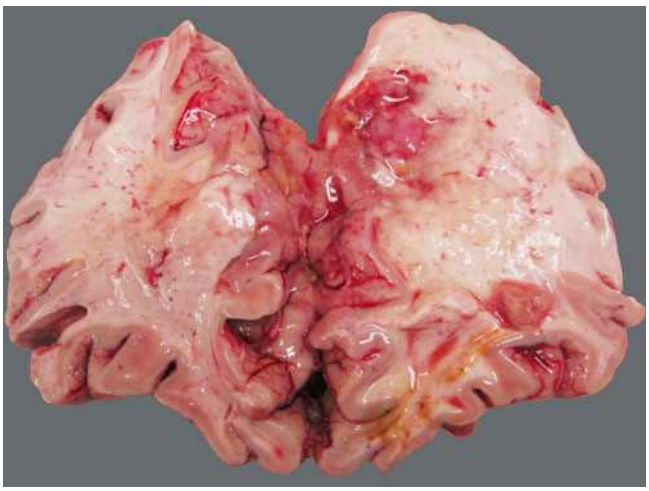

**Fig. 9.59** The brain tissues are widely softened, distorted, and friable in this case of advanced, high-grade oligodendroglioma

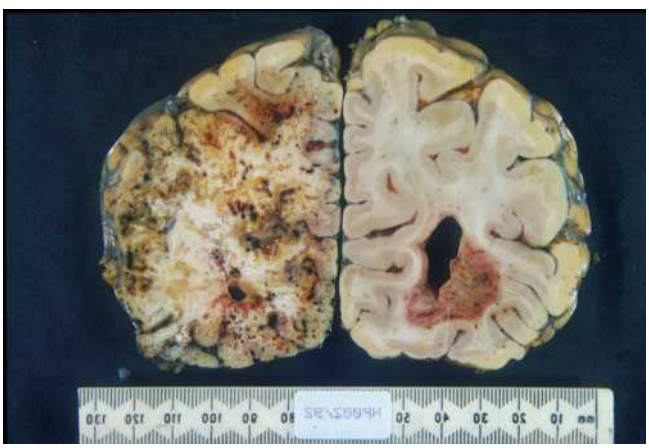

**Fig. 9.60** Widespread cerebral involvement by glioblastoma multiforme is seen with a significantly necrotic and haemorrhagic tumour affecting both cerebral hemispheres (*Courtesy of* Professor Paul G. Ince, Sheffield, UK)

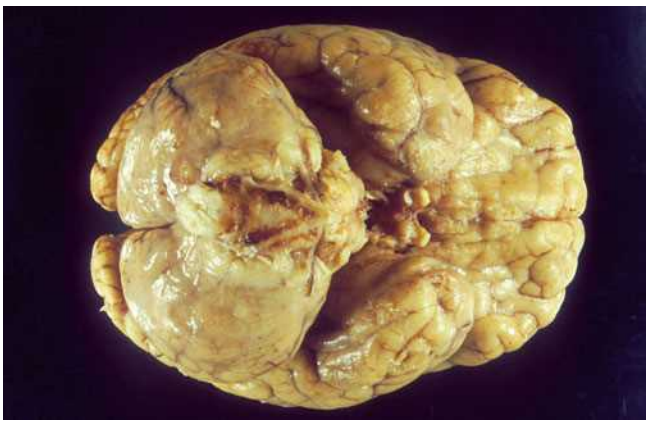

**Fig. 9.61** Diffuse meningeal spread of tumour is relatively rare, but it is a recognised cause of a dementing-type process and thereby the death of the individual (*Courtesy of* Professor Paul G. Ince, Sheffield, UK)

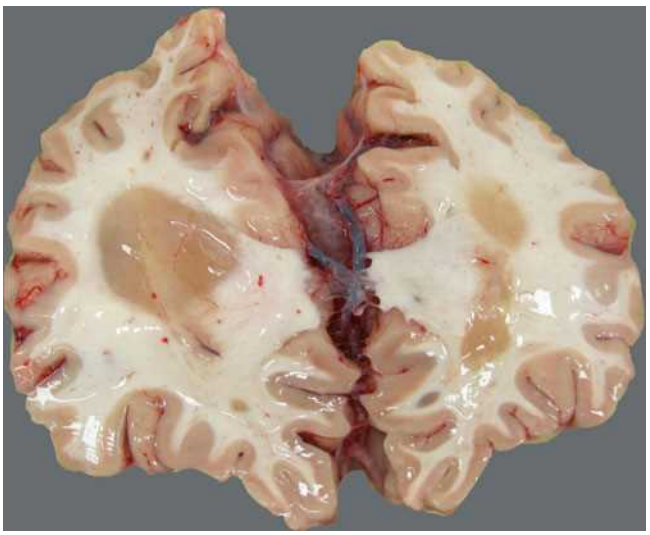

**Fig. 9.62** Multiple sclerosis is normally recognised before death. Several plaques are evident in the deep white matter, mapping to the ante-mortem symptoms experienced

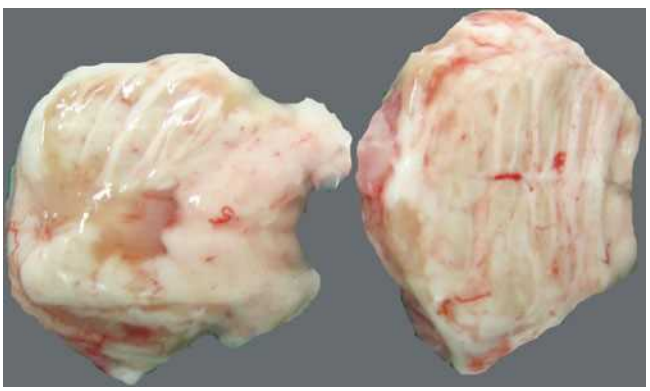

**Fig. 9.63** Multiple sclerosis can affect the brainstem, cerebellum, and spinal cord, as well as cerebral tissues

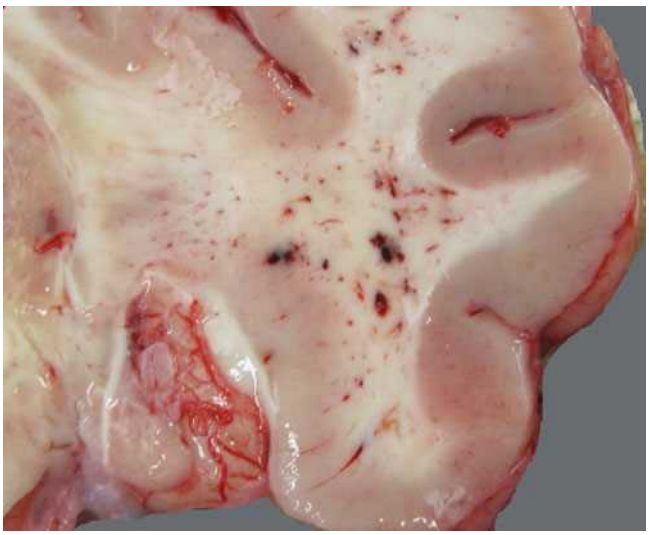

**Fig. 9.64** Cerebral trauma can produce significant vascular injury, usually reflecting shearing effects. Bleeding occurs diffusely across the brain tissues and is often rapidly fatal

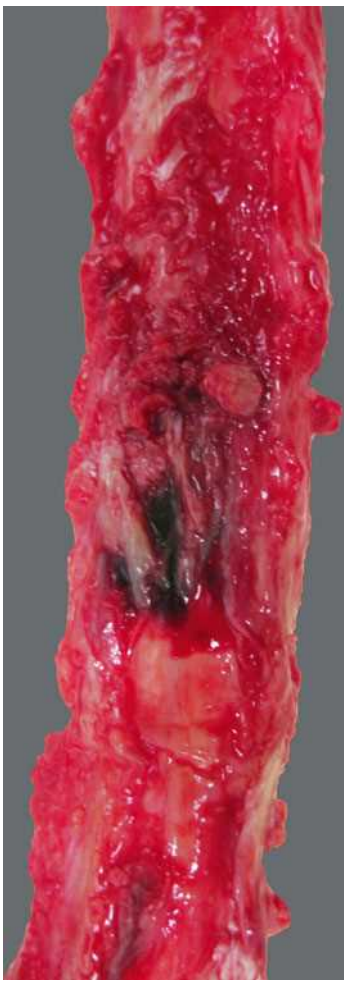

**Fig. 9.65** Cerebrovascular disease can affect the spinal cord and central brain tissues, with predictable consequences. Many of these situations involve vascular anomalies, as in the case illustrated

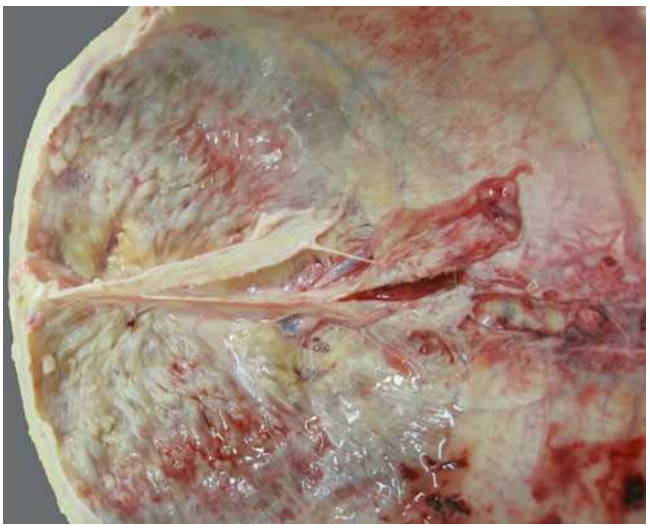

**Fig. 9.66** Hyperostosis frontalis interna is a benign condition of the cranial vault involving overgrowth of the bony tissues

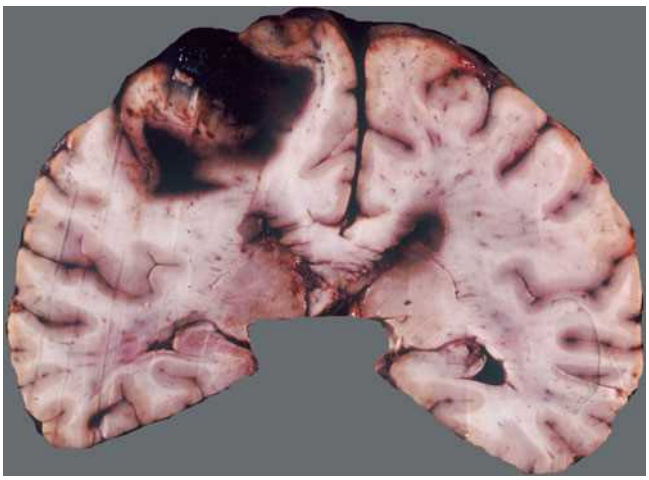

**Fig. 9.68** This view of a brain shows significant cerebral microvascular haemorrhage as well as a large cerebral bleed, in a case of eclampsia (*Courtesy of* Professor Paul G. Ince, Sheffield, UK)

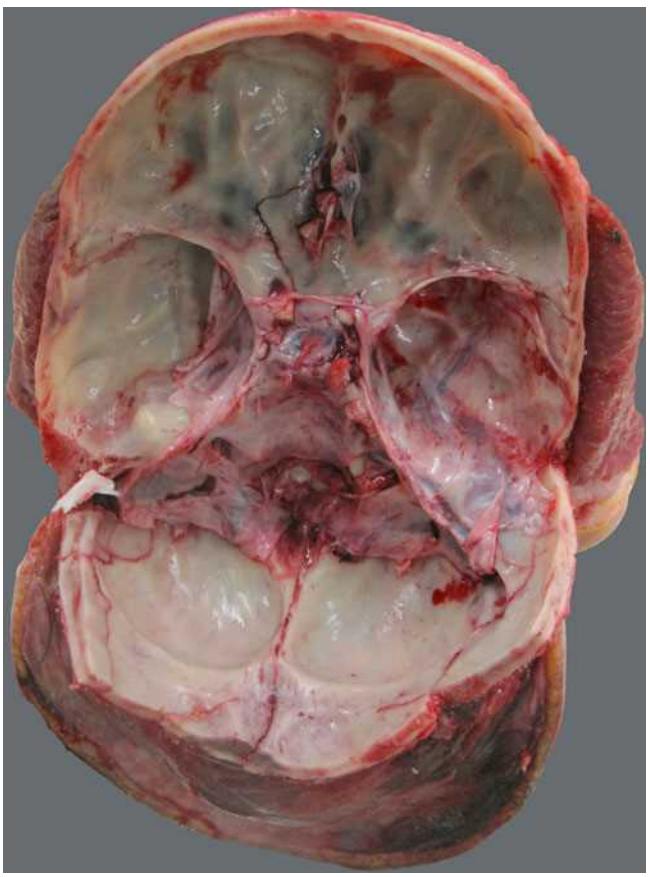

**Fig. 9.67** This complex skull fracture involved multiple bones of the skull and the skull base. It is often easiest to appreciate skull fractures by flexing and extending the skull tissues to demonstrate separation planes. A picture often is most valuable in these cases, rather than having to laboriously document the length and angle of every single fracture line

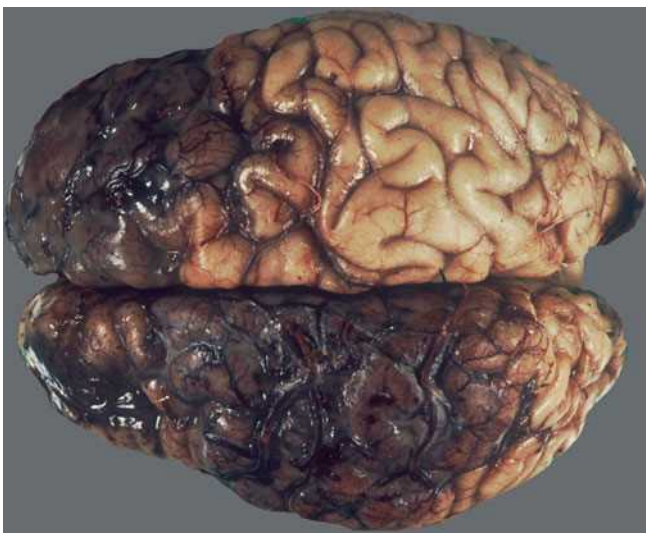

**Fig. 9.69** Thrombosis of the sagittal sinus produces congestion and venous infarction of local draining brain tissues. The degree of thrombotic change is clearly evident in the draining radicals that run on the surface of the cerebral hemispheres (*Courtesy of* Professor Paul G. Ince, Sheffield, UK)

## Commonly Encountered Surgical Interventions

Neurosurgical interventions may be encountered at autopsy (Figs. 9.70, 9.71, 9.72, 9.73, and 9.74). Burr holes may be drilled into the skull to relieve raised intracranial pressure; these are typically located over the parietal bones and are 10–12 mm in diameter. When wider access to the brain is needed, a craniotomy is used. Intracranial pressure monitors are commonly used to monitor patients who have undergone neurosurgery (see Chap. 2).

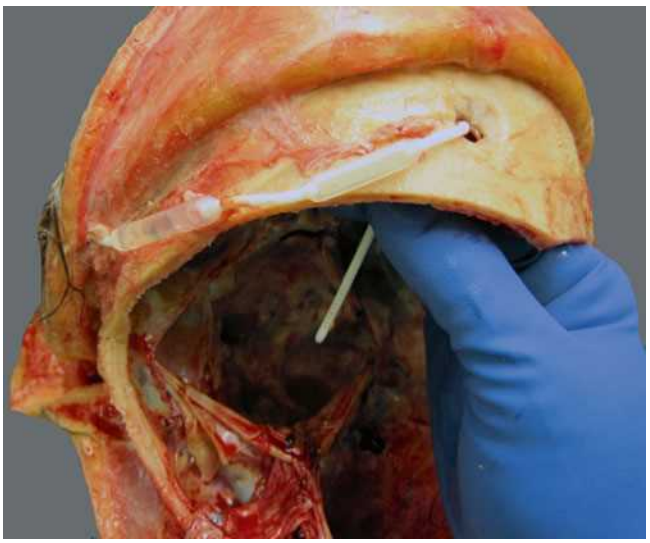

**Fig. 9.70** The entry point of a ventriculoperitoneal shunt is illustrated. The catheter running into the cerebral ventricles has been left intact so that the depth of penetration can be demonstrated

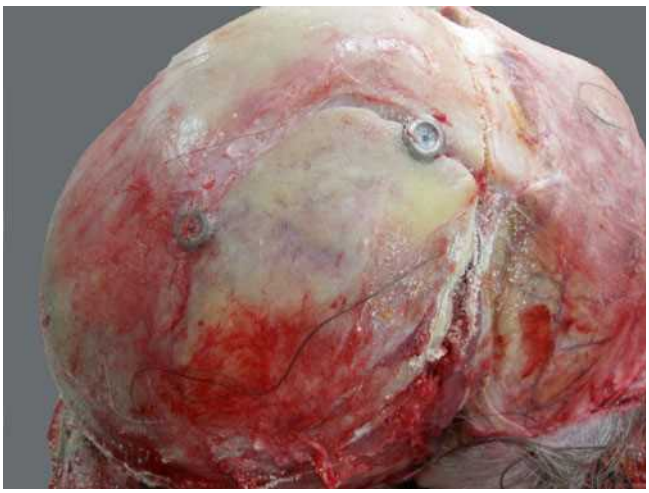

**Fig. 9.72** Previous cranial surgery is seen, with a bone flap and several anchoring screws. The space between the edges of the bony tissues has been filled by fibrous tissue

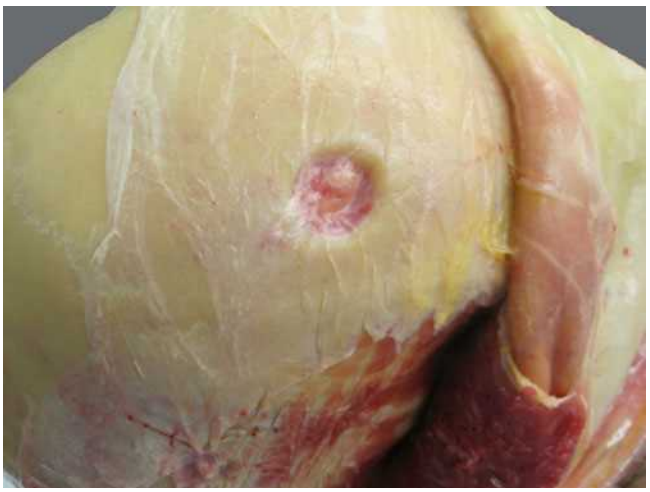

**Fig. 9.71** A burr hole is present from previous drainage of a subdural collection

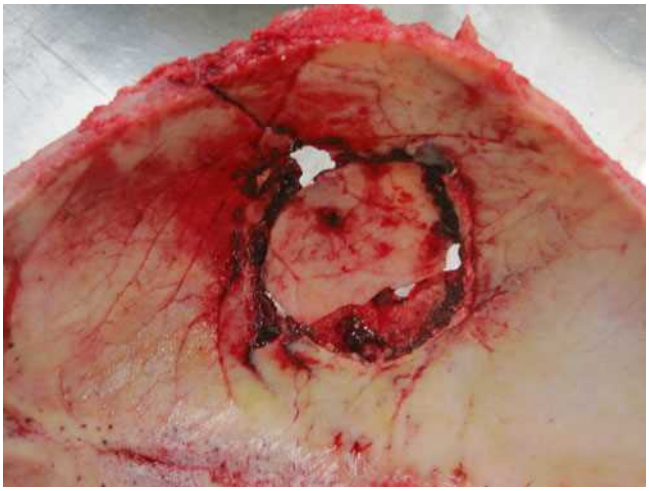

**Fig. 9.73** This repair of an acute fracture of the skull involved fixing it in position with screws. The inner aspect has no metal projecting into the cranial cavity

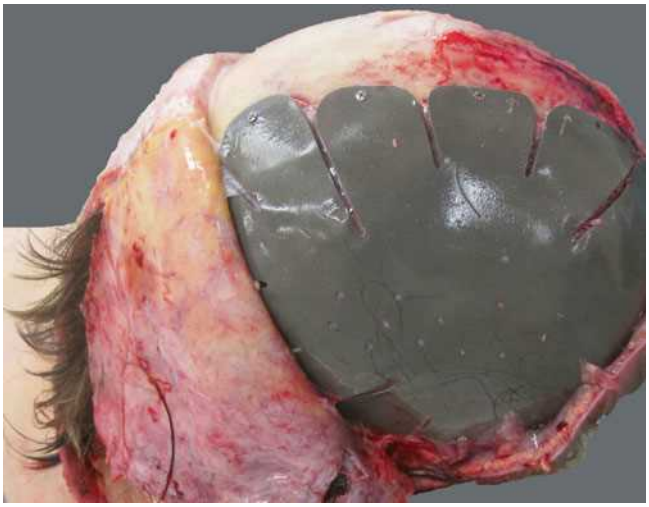

**Fig. 9.74** Where cerebral tissues have had a significant pathological insult—in this case, cerebral trauma and skull fracture with secondary infection. Treatment may require the removal of quite large areas of skull bony tissue. The integrity of the cranial vault is re-established by using a metal plate. The individual metal leaflets are folded directly onto the cranial vault and screwed into place

## The Eye

On rare occasions, it may be necessary to remove one eye or both eyes for detailed examination by an ophthalmic pathologist. For example, examination may be necessary if there is concern regarding possible ocular or orbital tumours or an acute disease that may have affected visual acuity, contributing to death.

To achieve an adequate cosmetic result on reconstruction, great care must be taken not to damage the eyelids or eyelashes when removing the eyes. Following the removal of the eye, the orbit must be reconstructed. The back of the orbit is packed with wet cotton wool, and dry cotton wool is then placed in front. An eye shield is placed over the top of the cotton wool, and the eyelid is then closed. Methyl methacrylate glue may be used to hold the eyelid in place, but care must be taken not to allow the glue to come into contact with the external surface of the eyelids.

## Removing the Eye: Anterior Approach

The anterior approach (Figs. 9.75, 9.76, 9.77, 9.78, and 9.79) is appropriate when examination of orbital structures other than the eye is not required.

## Removing the Eye: Posterior Approach

The posterior approach is preferred when there are concerns that the deceased may have had an ocular tumour. This approach allows the globe to be removed in continuity with the other orbital contents, allowing staging of such tumours. The conjunctiva is first incised, as for the anterior approach.

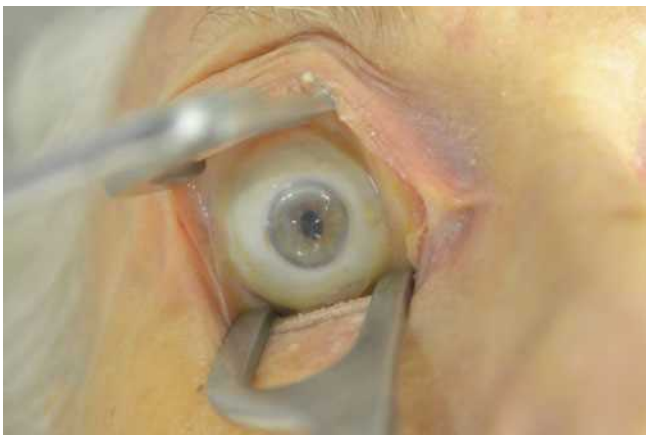

**Fig. 9.75** The eye is removed in stages. One begins by retracting the eyelids

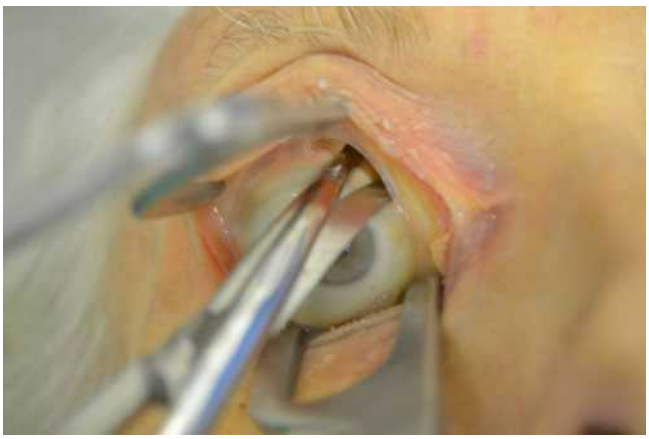

**Fig. 9.76** The conjunctiva is incised and the muscles are cut, followed by the optic nerve

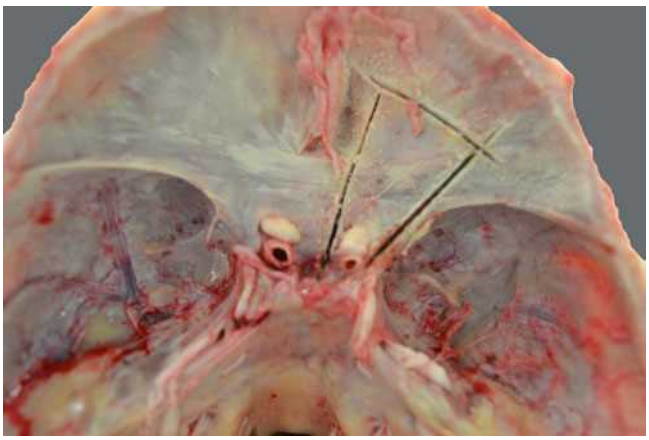

**Fig. 9.77** Using the oscillating saw, three cuts are made in the floor of the anterior cranial fossa

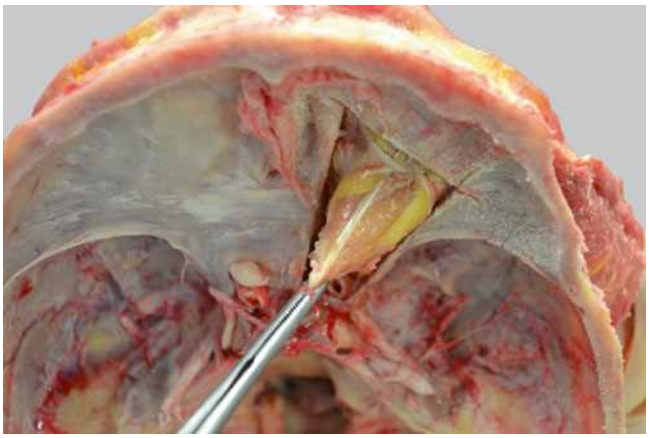

**Fig. 9.78** The bone segment is removed

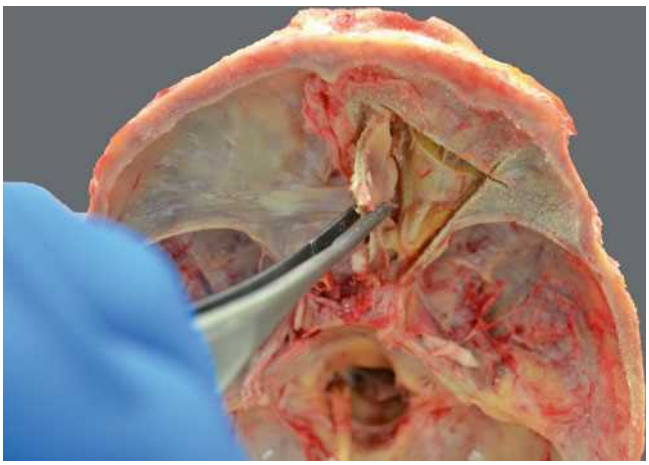

**Fig. 9.79** The eye connections can be removed from the superior approach, using curved scissors

## The Middle Ear

The middle ear should be examined in cases of sepsis of unknown source, and in some cases of meningitis, to look for a source of infection. The middle ear cavities can be swabbed for microbiological examination (see Chap. 10).

The middle ears are exposed once the brain has been eviscerated and the dura stripped from the base of the skull (Figs. 9.80, 9.81, 9.82, and 9.83). No specific reconstruction is required beyond that used to reconstruct the skull following evisceration of the brain.

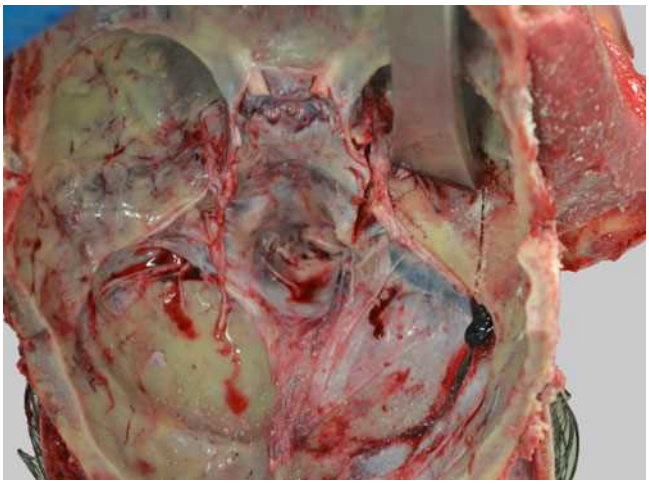

**Fig. 9.80** The ear is approached by cutting into the sphenoid. Two parallel cuts are made. The bone segment is levered out with the skull key or osteotome

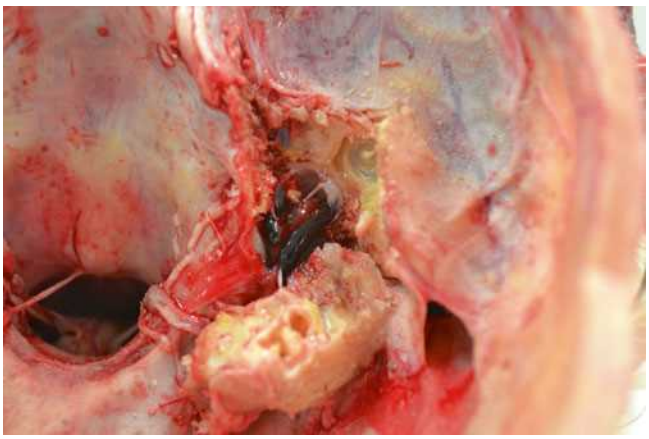

**Fig. 9.81** The inner ear is exposed

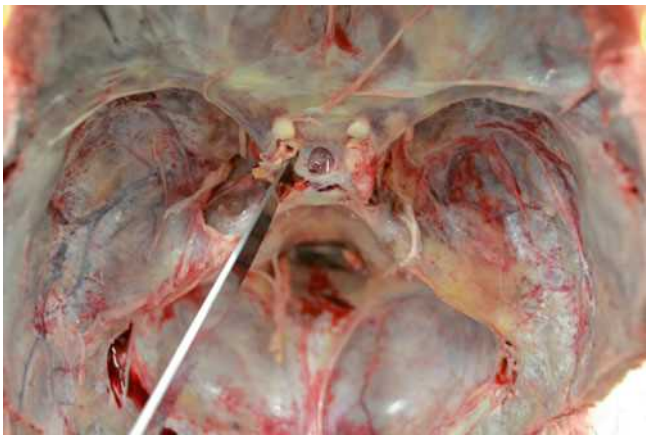

**Fig. 9.82** Cuts are made around the sella turcica

## The Pituitary

The pituitary gland (also discussed in Chap. 8) should be examined in cases of suspected tumour, and in cases of systemic inflammatory response syndrome (SIRS), when a septic focus cannot be identified.

The pituitary gland is removed (Fig. 9.84) once the brain has been eviscerated and the dura has been stripped from the base of the skull.

**Fig. 9.83** The posterior parts of the clinoid processes are cut backwards by the skull key

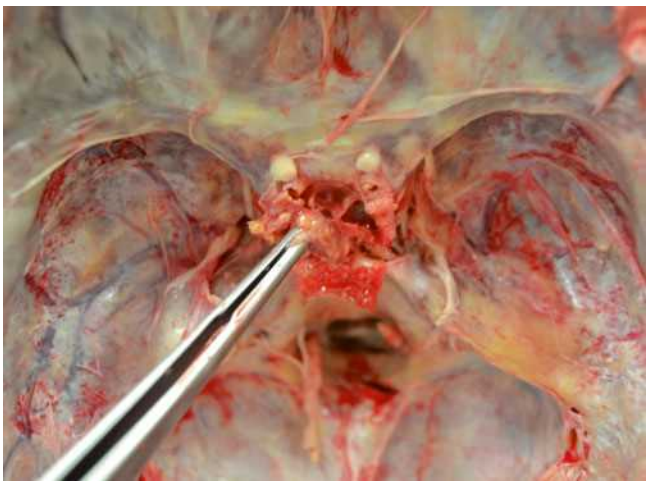

**Fig. 9.84** The pituitary is gently dissected and lifted out

## The Spinal Cord

The spinal cord may require examination in cases where trauma, tumour, inflammatory diseases, or degenerative diseases are suspected. Care must be taken when handling the cord to keep it as straight as possible. Angulation of the cord may produce artefacts that make histopathological interpretation difficult.

On rare occasions, it may be necessary to remove the cord in continuity with the brain. This is best done by using the anterior approach for removal of the cord, as the body is then in the correct position for removal of the brain.

### Exposing and Removing the Spinal Cord: Anterior Approach

Most often, the spinal cord is removed using an anterior approach (Figs. 9.85, 9.86, 9.87, 9.88, 9.89, and 9.90) once the organs of the neck, thorax, and abdomen have been eviscerated. This approach allows the spinal cord to be removed in continuity with its associated ganglia.

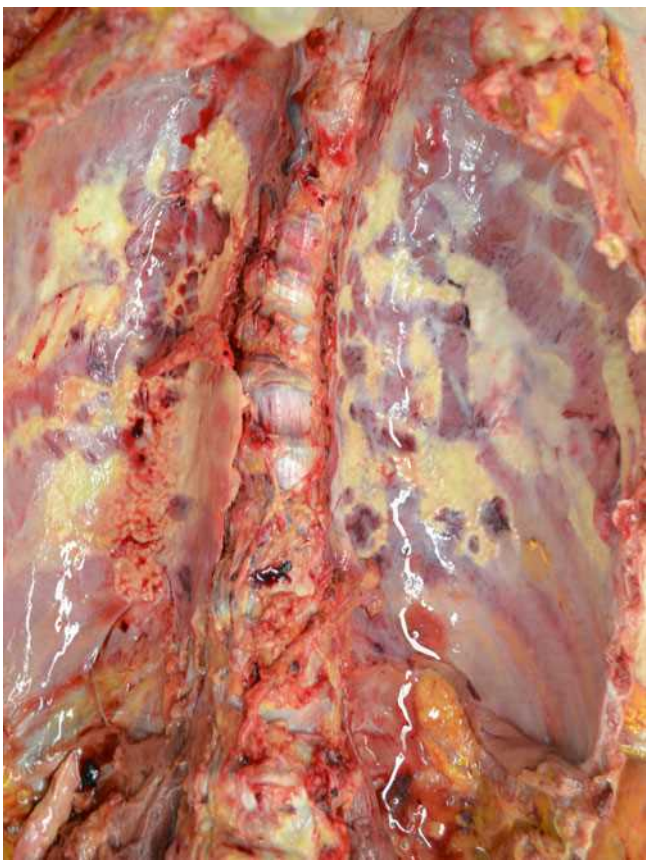

**Fig. 9.85** The examination and removal of the spinal cord is a complex task. Here, one starts by having the thoracic and lumbar vertebral bone exposed. The muscle and other soft tissues are removed on either side

## Exposing and Removing the Spinal Cord: Posterior Approach

When the spinal cord is to be examined without its associated ganglia, it may be removed via a posterior approach. This approach is easier than the anterior approach, as the angle of the saw cuts is uniform in all regions of the spine, but it requires the body to be placed into a prone position and it generates a further skin incision. (see also Chap. 7, cervical vertebrae dissection Figs. 7.46–7.56).

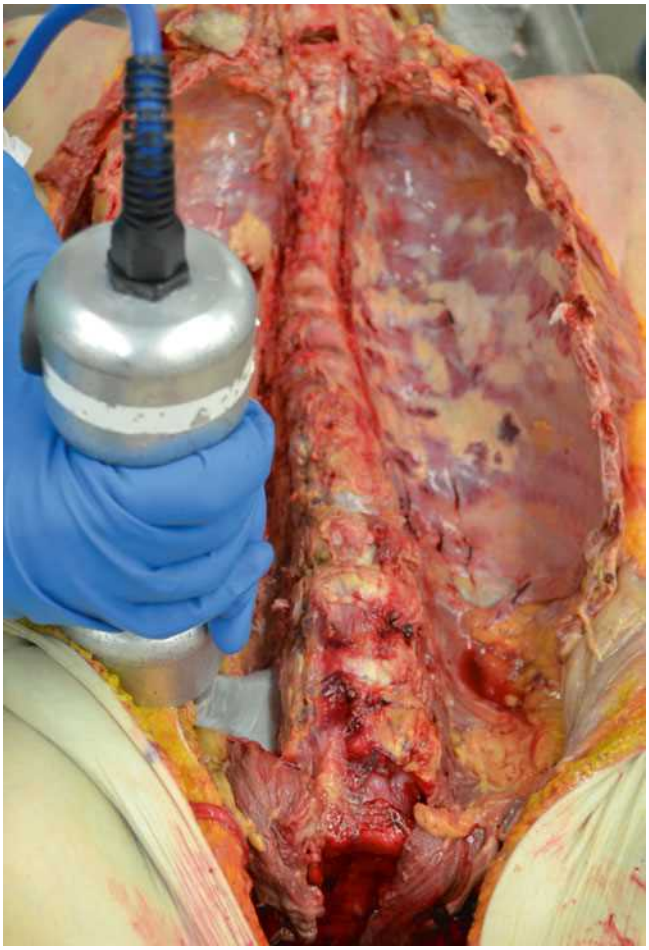

**Fig. 9.86** The lateral bone boundaries of the spinal canal are cut with the oscillating saw. Each vertebral body must be cut. The lower end (L5/S1 disc) is transected by a long knife

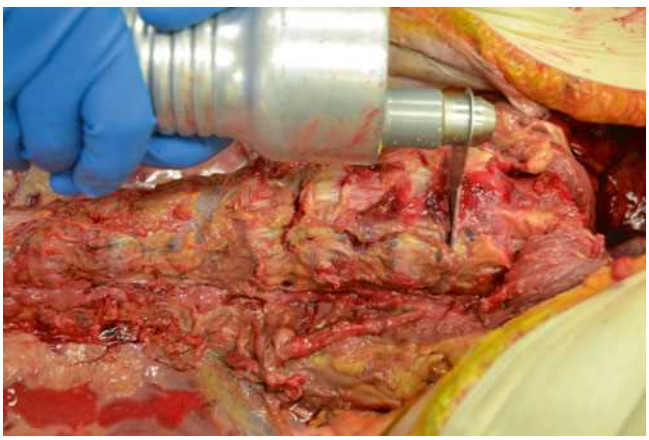

**Fig. 9.87** Alternatively, one can use the saw to cut across the bone transversely. This cut, or that made at the disc, will be below the distal end of the cord

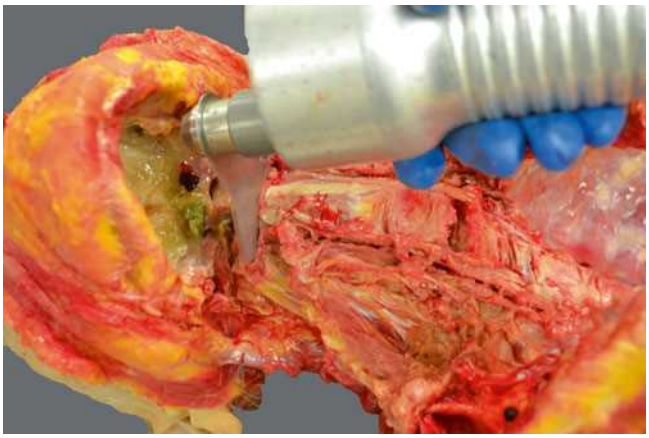

**Fig. 9.88** The upper end of the cord is accessed by a high cervical vertebral bone cut

**Fig. 9.89** The vertebral bodies are levered upwards, gently exposing the spinal cord in the spinal canal

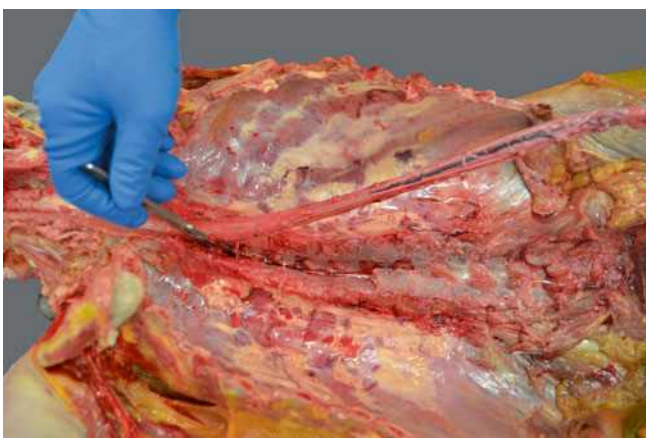

**Fig. 9.90** The side branches of the spinal nerves are sequentially cut, allowing the cord to be removed gently

## Reconstructing the Head

It is not practicable to attempt to replace the brain in the cranial cavity because of its soft consistency and the risk of later leakage. When the examination of the brain and skull is complete, the brain is returned to the body with the organs from the torso.

The head is normally reconstructed by an experienced anatomical pathology technician, who will ensure good cosmesis, but the pathologist should be familiar with the process. The cranial cavity is first packed with cotton wool, and the calvaria are replaced. The calvaria can be secured in place using methyl methacrylate glue. The temporalis muscles are returned to their anatomical positions, and the scalp is then replaced over the calvaria. The scalp incision is sutured closed.

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## Suggested Reading

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# Standard and Special Tests

# 10

Julian L. Burton

## Introduction

In many autopsies, the cause of death can be determined by macroscopic examination alone, or with macroscopic and histopathological examination of the major organs. Frequently, however, further investigations are needed to determine or refine the cause of death.

This chapter illustrates the methods used to collect samples for toxicology, microbiology, virology, immunology, and histology. It should be noted that the technique shown to collect blood for toxicological analysis is also largely appropriate for the collection of blood for virology and/or immunological investigation, although an aseptic technique is more appropriate for microbiology samples. The initial set of images deals with toxicology samples, with microbiology samples following.

Consideration should be given to the potential collection of these samples prior to the start of every autopsy. Once the organs have been eviscerated, the opportunity to collect these samples is frequently limited, or even lost.

When samples are submitted for analysis, it is imperative that they be placed into the appropriate containers and correctly labelled with patient identifier information (ideally name, date of birth/death, and unique numerical coding). The samples should be accompanied by adequate clinical and case history details.

Toxicology samples should be accompanied by details that include the circumstances surrounding the death, the nature of any prescribed medication or suspected illicit drugs, and the nature and site of collection of the samples.

Samples submitted for bacteriological or virological examination should likewise be accompanied by details of the clinical history, the deceased's address (for contact tracing purposes), any antimicrobial therapies given in life, and the post-mortem interval.

Autopsy microbiology poses its own special challenges of interpretation. It should be remembered that organisms detected may be due to post-mortem colonisation of the body (usually originating from the intestines), and that a test may be negative because there is no infection (true negative) or because the infecting organism has died in the interval between death and autopsy. Sampling error also may yield false negative results.

All samples obtained should be submitted to the appropriate laboratory as soon as possible. If immediate transportation is not possible, they should be stored at 4 °C.

## Toxicology

The collection of samples for toxicology should be considered in every autopsy. Samples are best collected before or during full evisceration, or potentially as the autopsy starts. Where possible, both plain and preserved samples should be collected and placed into the appropriate specimen containers. Tissue samples should be placed dry into a sterile container that has never contained formalin or another product.

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S.K. Suvarna (ed.), *Atlas of Adult Autopsy: A Guide to Modern Practice*, DOI 10.1007/978-3-319-27022-7_10

## Vitreous Humour

Vitreous humour may be collected for both clinical chemistry and toxicological analysis by direct aspiration of the eye contents using a syringe and needle (Figs. 10.1, 10.2, 10.3, 10.4, and 10.5). An unpreserved sample is required for sodium ion, urea, and creatinine quantification. A preserved sample is required for glucose, alcohol, cocaine, and 6-monoacetylmorphine quantification. The structure and shape of the eye should be reconstituted by instilling water afterwards.

**Fig. 10.1** The eyelids are retracted

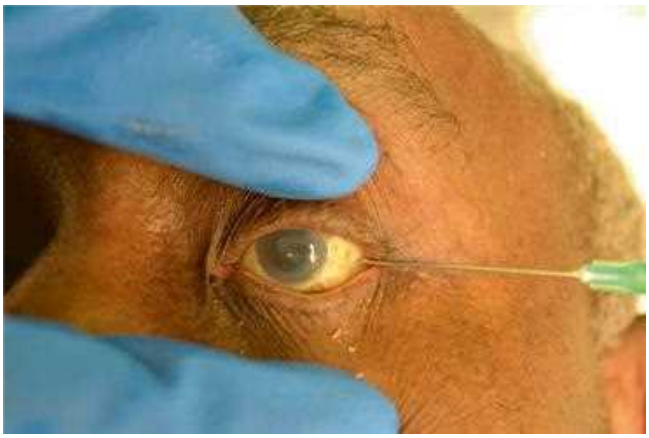

**Fig. 10.2** Using a 19G needle attached to a 10-mL syringe, puncture the sclera as distally and laterally as possible. The needle should be angled inwards at 30–45°

**Fig. 10.3** Aspirate 2 to 3 mL of vitreous humour

**Fig. 10.4** Remove the syringe from the needle, leaving the needle in situ

**Fig. 10.5** Reinflate the globe using 2 to 3 mL of water, and then remove the needle

## Blood

Blood for toxicological analysis (ideally 10–20 mL) should be obtained either by needle puncture of the femoral vein prior to evisceration (Figs. 10.6, 10.7, and 10.8) or from the common iliac vein on opening the body, prior to evisceration of the internal organs. It is not common practice to sample blood from the abdominal or thoracic tissues, to prevent issues with post-mortem redistribution of drugs or toxins. On rare occasions, when little peripheral blood is available, the venous sinuses in the cranial cavity may be accessed directly with syringe aspiration of their contents.

**Fig. 10.6** Blood for toxicology can be aspirated from the femoral vein, which lies medial to the midpoint of the inguinal ligament

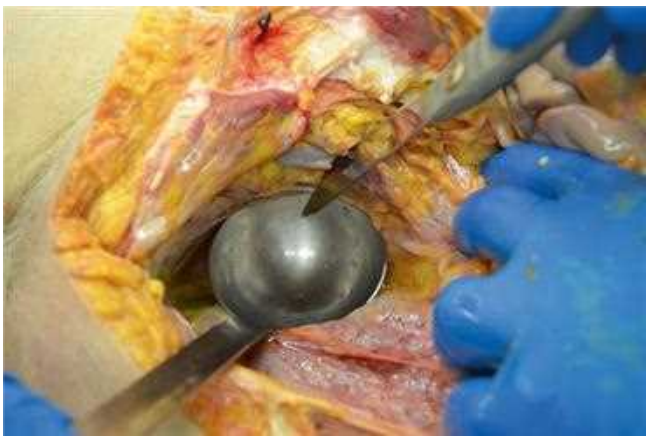

**Fig. 10.7** On opening the abdomen, use blunt dissection to free the pelvic organs from the pelvic side walls. Place a specimen container or clean ladle into the pelvis, below the common iliac vein

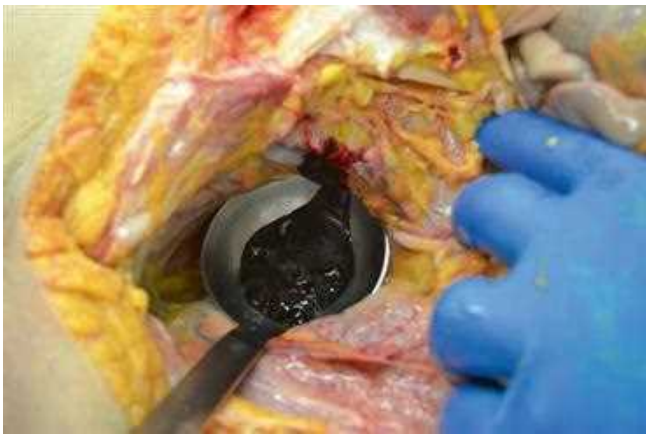

**Fig. 10.8** Incise the iliac vein using a knife. Blood usually flows freely into the specimen container or ladle. If necessary, gently massage the inner thigh, from knee to groin, to express blood

## Urine

Urine is most easily obtained immediately after the peritoneal cavity has been opened, using a needle and syringe (Figs. 10.9 and 10.10). If the volume of urine within the bladder is small, a sample may be obtained by incising the dome of the bladder to visualise the sample to be collected.

**Fig. 10.9** Urine for toxicology is best obtained using a needle and syringe, on opening the abdomen. Take care not to contaminate the sample with blood. Insert the needle through the dome of the bladder

**Fig. 10.10** Aspirate 10 mL of urine and place it into the appropriate specimen containers

## Gastric Contents

It is useful to collect a sample of gastric contents if there is suspicion that the deceased may have ingested a toxin or drug overdose. The entire contents should be extracted (Figs. 10.11, 10.12, and 10.13). The total volume of the stomach contents should be noted and a representative sample (generally 20 mL) should be submitted for analysis. The total volume of the stomach contents should be recorded. Close attention to the contents is advised, looking for solid or partly broken-down tablet residues. If there is concern that death may be due to anaphylaxis, the stomach contents should be photographed.

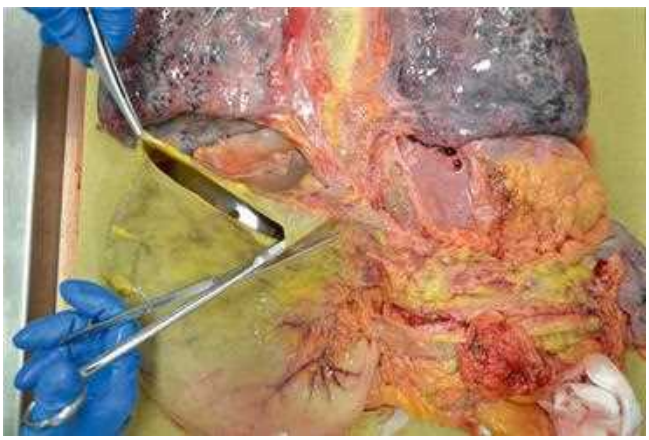

**Fig. 10.11** A 10-cm incision is made into the gastric fundus using scissors, taking care not to contaminate the gastric contents with blood

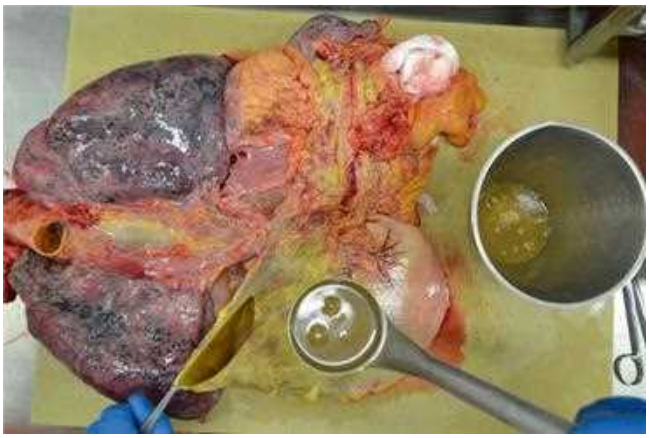

**Fig. 10.12** The gastric contents are removed into a clean, dry measuring jug using a clean, dry ladle

**Fig. 10.13** Record the volume of the gastric contents, noting the presence or absence of any tablet residues. Reserve a sample for toxicology, but the entire volume may not be required

## Bile

When it is not possible to obtain samples of blood or urine, a sample of bile may be collected and submitted for toxicological analysis (Figs. 10.14 and 10.15). The range of drugs that can be tested may be more limited than with blood.

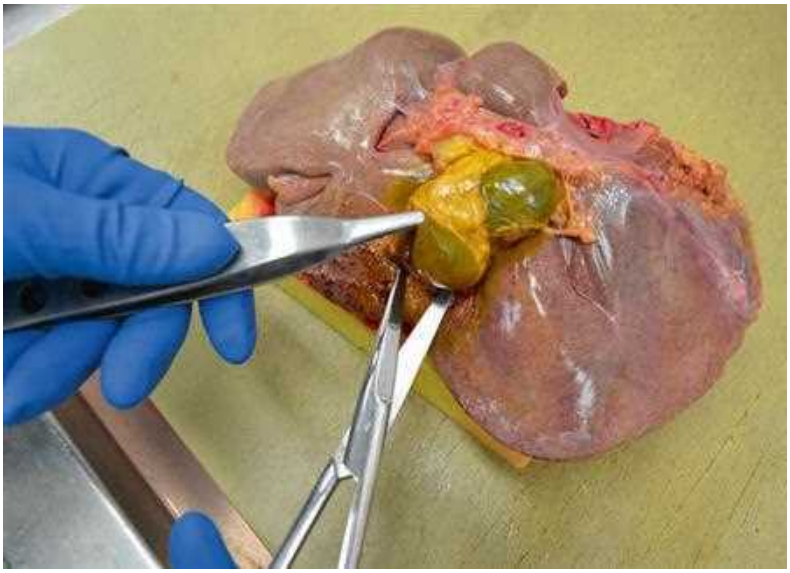

**Fig. 10.14** Carefully dissect the intact gallbladder free of the gallbladder bed. Alternatively, take the sample by syringe aspiration of the gallbladder at the start of the examination, after opening the abdomen

**Fig. 10.15** Invert the gallbladder over an appropriate specimen container and squeeze gently, taking care not to contaminate the sample with blood

## Liver

If no fluid samples can be obtained from the body, a sample (generally 2–3 $cm^3$) of the right lobe of the liver can be submitted for toxicological analysis (Figs. 10.16 and 10.17). A sample of psoas muscle is also acceptable, either in place of liver or as an additional specimen.

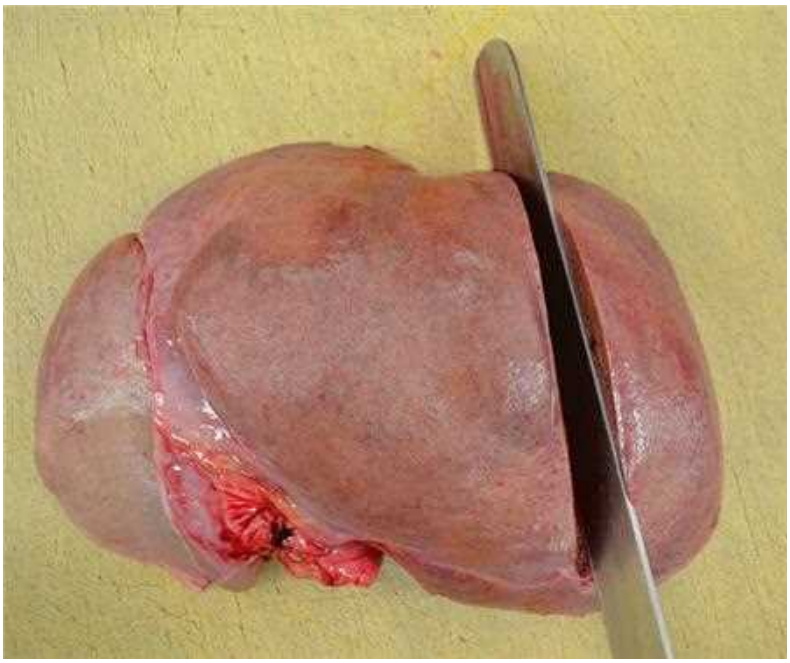

**Fig. 10.16** Having dissected the liver free of its attachments, make a parasagittal slice through the middle of the right lobe, using a clean, dry knife

**Fig. 10.17** Use a clean, dry scalpel to excise a portion of liver from deep within the right lobe of the liver

## Microbiology and Virology

In cases where infection is suspected, samples should be collected for microbiology. When collecting transdermal samples, the skin should be cleaned with alcohol, povidone-iodine solution, or chlorhexidine and allowed to dry. If these are not available, then formalin-soaked tissue may be used.

Sterile instruments must be used, and needles should not be changed.

Specimens must be placed into empty, sterile universal containers that have never contained formalin or other products. When tissue is collected, it may be placed into a sterile container containing sterile 0.9 % saline to prevent it from drying out. Samples should be stored at 4 °C if they cannot be immediately transported to the laboratory. Modern virological investigations examine for the presence of viral DNA/RNA rather than viral culture, and viral transport medium is generally no longer required.

## Blood

Blood for microbiological culture should be collected prior to opening the body. It is best collected from a site remote from the intestines. The jugular or subclavian veins are often ideal sites (Figs. 10.18 and 10.19).

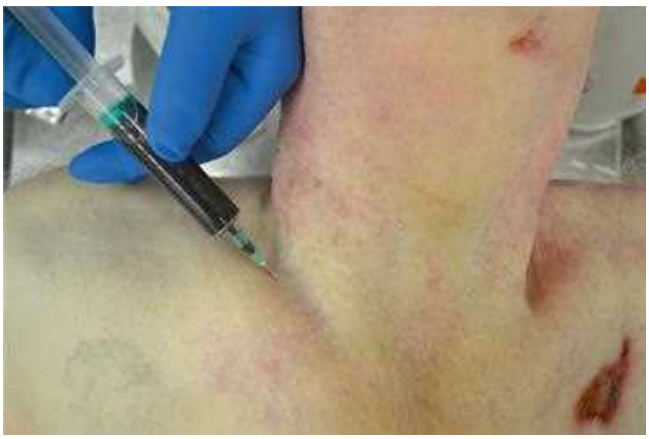

**Fig. 10.18** Taking blood for microbiology starts with cleaning the skin of the anterior neck using alcohol, chlorhexidine, or povidone-iodine solution, and allowing it to dry. Using a green needle attached to a 10 or 20 mL syringe, insert the needle above the junction of the inner and middle thirds of the clavicle. Angle the needle at 80° from the vertical, and aim towards the contralateral sternoclavicular joint. Gradually advance the needle while gently aspirating on the syringe. Aspirate 10 to 20 mL of blood

**Fig. 10.19** Withdraw the needle, and inject directly into blood culture bottles. *Do not change the needle*

## Urine

Urine for microbiological examination is best obtained from the bladder prior to opening the body (Figs. 10.20, 10.21, 10.22, and 10.23). If this is not possible, the method described for the collection of urine for toxicology can be used. If there is no urine within the bladder, the bladder mucosa can be swabbed. It is emphasised that urine present within catheter bags is not appropriate for microbiology.

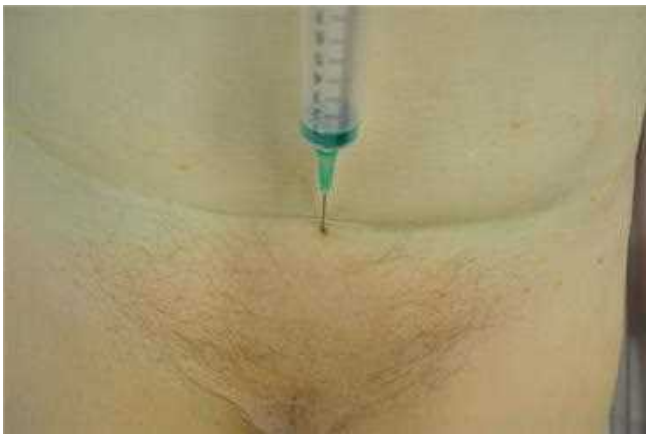

**Fig. 10.20** Microbiology assessment of urine may be approached using a green needle attached to a 10 mL syringe. Insert the needle above the symphysis pubis

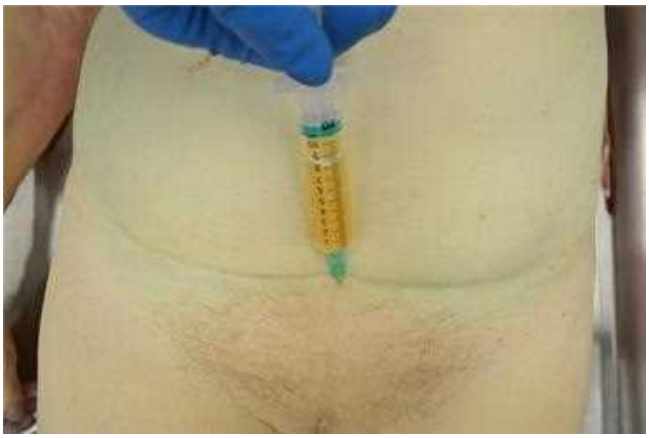

**Fig. 10.22** Gradually advance the needle, while gently aspirating on the syringe. Aspirate 10 mL of urine

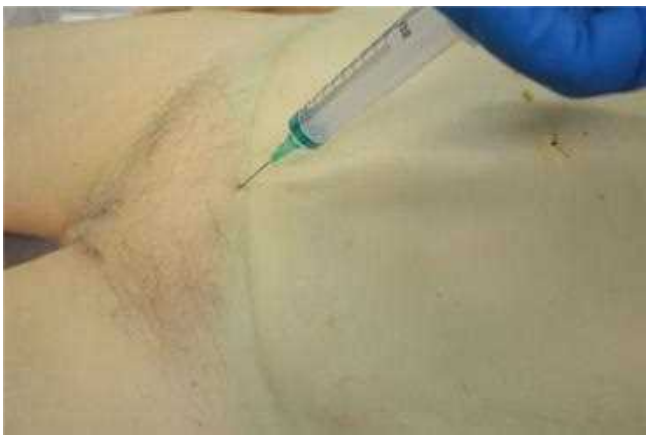

**Fig. 10.21** Angle the needle inferiorly, at an angle of 45°

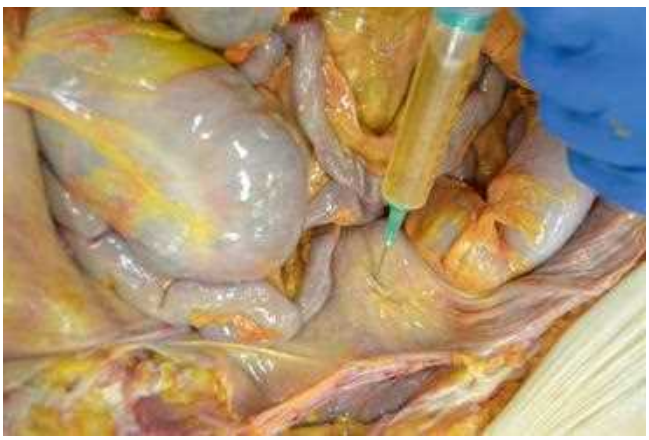

**Fig. 10.23** Alternatively, use a needle and syringe to aspirate urine via the dome of the bladder immediately on opening the peritoneal cavity

## Lung

Lung tissue should be collected for standard microbiology (bacterial) studies and/or virology when either pneumonia or viral pneumonitis are suspected from the history (Figs. 10.24 and 10.25). One should not overlook possible fungal infections. A sample for histology should also be reserved for cross comparison.

**Fig. 10.24** One may sample lung for microbiology. This sampling is accomplished by opening the chest cavity as normal, taking care not to handle the lungs directly

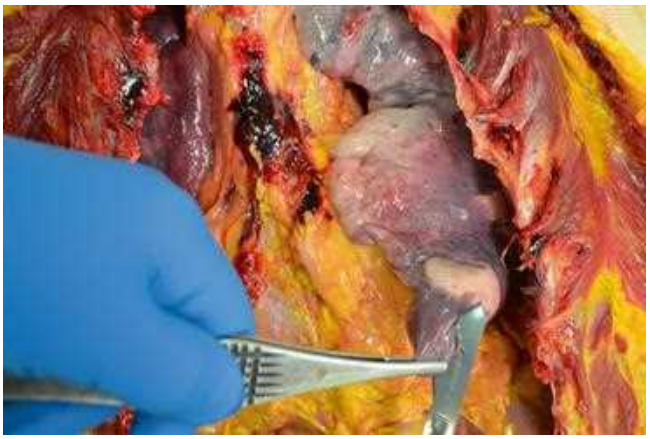

**Fig. 10.25** Using a sterile scalpel blade and forceps, remove a cube of lung (1–2 $cm^3$) from the lower lobe or area of interest

## Cerebrospinal Fluid

When meningitis or encephalitis is suspected, samples of cerebrospinal fluid (CSF) should be collected for microbiology and virology. (Swabs and samples of brain tissue also can be collected, and a pharyngeal swab for microbiology should also be collected.) Samples can be easily obtained from the cisterna magna by direct aspiration (Figs. 10.26, 10.27, 10.28, and 10.29).

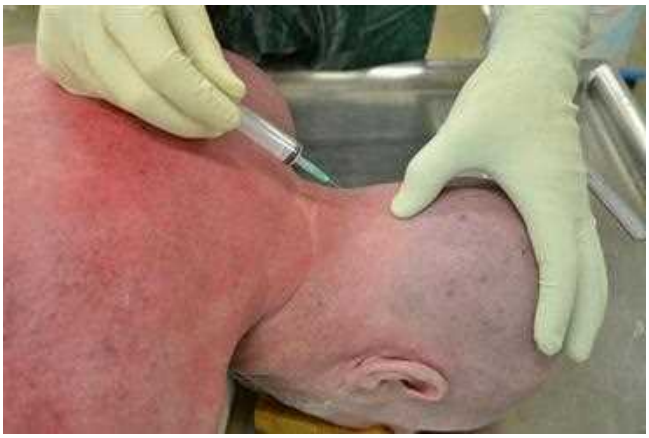

**Fig. 10.26** Cerebrospinal fluid (CSF) sampling involves access via the back of the neck. Lay the body prone, with a block under the chest to flex the neck. Palpate the atlanto-occipital membrane in the midline. This will be the site of needle insertion

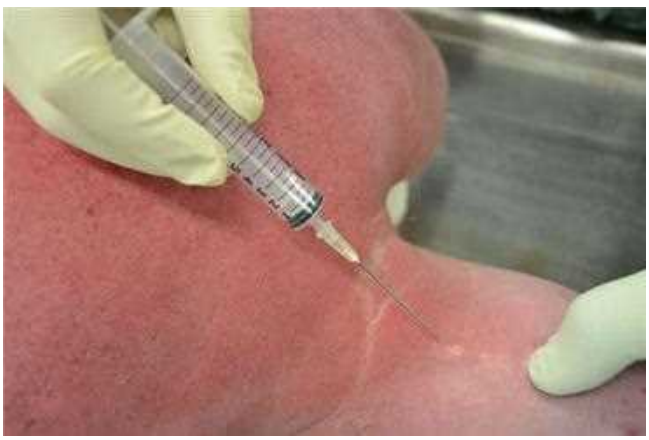

**Fig. 10.27** Use a wide-bore needle attached to a 5 or 10 mL syringe

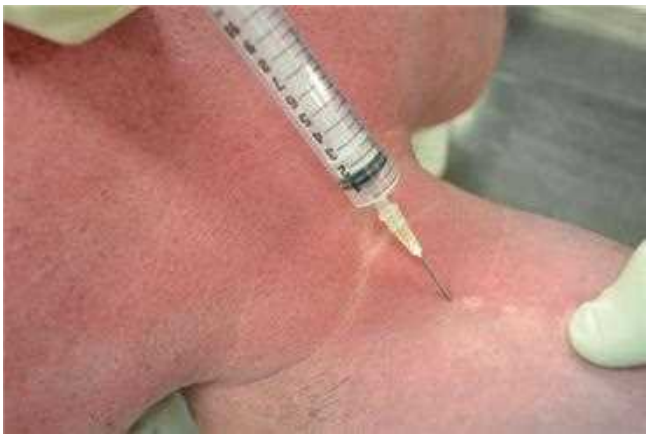

**Fig. 10.28** Slowly advance the needle, aiming towards the bridge of the nose

**Fig. 10.29** At a depth of approximately 20 mm, you will feel the needle "give" and will be able to aspirate between 2 to 10 mL of CSF

## Ascites

Patients with cirrhosis of the liver may develop spontaneous bacterial peritonitis. Collecting a sample of ascites for microbiological examination helps to confirm the diagnosis and is best done before the body is opened (Fig. 10.30).

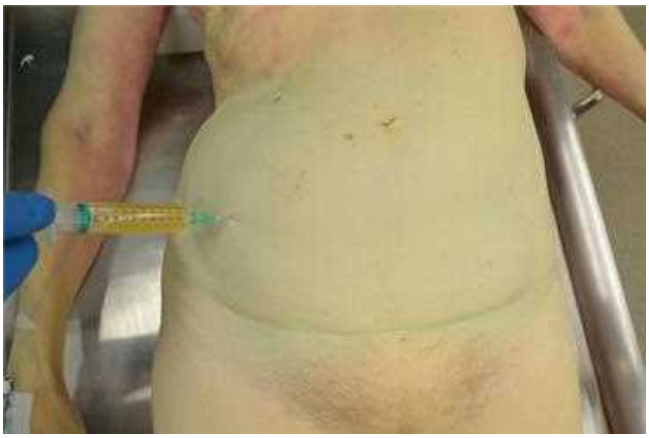

**Fig. 10.30** To sample ascites, use a green needle attached to a syringe. Insert the needle in the mid-clavicular line, aiming for the spine, and aspirate 10 mL of ascites

## Spleen

If blood cultures are desirable but unobtainable, a sample of spleen can be collected for microbiological culture as soon as the peritoneal cavity has been opened (Figs. 10.31 and 10.32).

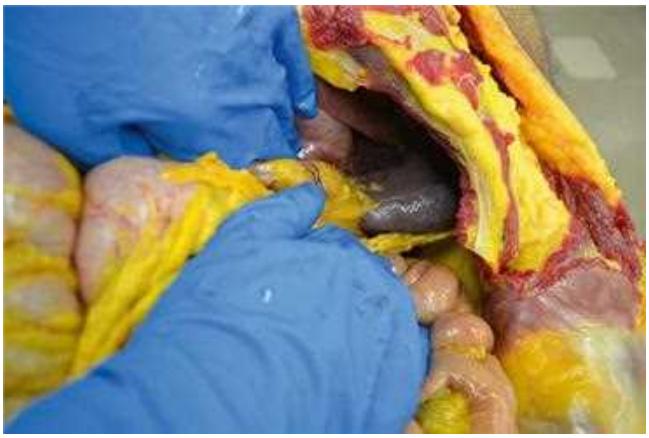

**Fig. 10.31** To collect a sample of spleen for microbiological culture, have an assistant retract the intestines towards the right iliac fossa, taking care not to touch the spleen

**Fig. 10.32** Using a sterile scalpel blade and forceps, remove a 1 to 2 $cm^3$ cube of spleen

## Bile

When biliary sepsis is suspected, a sample of bile may be collected for culture. This is best done on opening the peritoneal cavity, prior to evisceration of the internal organs (Fig. 10.33).

**Fig. 10.33** To collect a sample of bile for microbiological culture, use a white needle attached to a 10 mL syringe to puncture the gallbladder fundus and aspirate up to 10 mL of bile

## Faeces

If gastroenteritis (including viral causes) or pseudomembranous colitis is suspected, sample of faeces can be collected during evisceration or organ dissection for stool culture (Fig. 10.34).

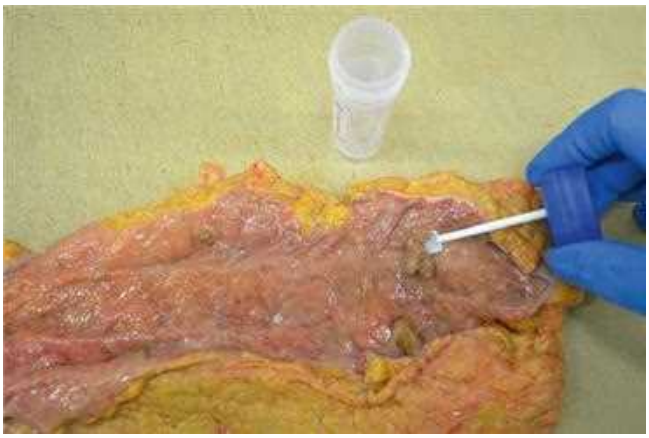

**Fig. 10.34** A sample of faeces can also be collected by opening the sigmoid colon after evisceration

## DNA Sampling

Any tissue with nucleated cell content will have DNA available for extraction and testing, but the best source will be tissue with a high cell nuclear content, generally taken to be spleen. A sample of spleen is generally advocated for DNA testing purposes. The sample is taken in a similar manner to that employed for microbiology. The sample may have the DNA extracted soon after sampling for storage, or the spleen sample may be frozen for later DNA extraction and assessment (*see* Chap. 7).

## Histopathology Sampling

If the cause of death is not evident from macroscopic examination, or if there is concern about diseases that can be accurately identified only using microscopic examination, samples of tissues should be collected for histology.

Samples of the major organs and other tissues of interest are collected using a knife and are placed directly into appropriately labelled cassettes. The tissue samples should be approximately 2 mm thick. Soft tissues may need more than 24 h of fixation. Heavily calcified tissues require fixation and decalcification prior to histology sampling. Samples of similar density, or those that require similar staining techniques (for example, liver and kidney) may be placed into the same cassette for efficiency.

## Suggested Reading

Burton JL, Rutty G. The hospital autopsy. 3rd ed. London: Hodder Arnold; 2010.

Finkbeiner WE, Ursell PC, Davis RL, editors. Autopsy pathology: a manual and atlas. Philadelphia: Churchill Livingstone; 2004.

Saukko P, Knight B. Knight's forensic pathology. 4th ed. Boca Raton: CRC Press; 2016.

# Devices: Foreign Items Encountered During the Autopsy

11

S. Kim Suvarna

## Introduction

Modern medicine has developed a variety of lines, tubes, joints, and electronic instruments to be implanted during life. These devices (for want of a better word) are many in type and are varied in design. They have symptomatic, reconstructive, and therapeutic functionality.

Among the devices often seen at autopsy are cardiac valves, pacemakers, and joint replacements. Many bodies arrive in the mortuary without full notes. However, and one may be perplexed to find an unexpected or unusual item (for which the function is not immediately apparent) during the examination. Whilst not all-encompassing, this chapter aims to demonstrate many of the range of items that may be found, with some basic data about their functionality.

## External Devices

Modern medicine provides a range of different devices that are used to assist monitoring and treatment of patients in the surgical and medical setting. Many, such as the pulse oximeter and ECG electrodes, are temporary and are removed before the patient is transferred to the mortuary after death. Nevertheless, general advice to those looking after the deceased is that any device or item that could have an effect upon the body is generally left in situ following death. If the body is not subject to autopsy examination, then the devices may be removed before funeral preparations.

For those cases requiring autopsy, the presence of any device that may have a bearing upon the cause of death requires the device to be considered as part of the post-mortem examination process. Examples include a variety of lines that access the internal aspects of the body, including intravenous, intra-arterial, intraosseous, subcutaneous, intrathecal, percutaneous, and intra-abdominal lines (Figs. 11.1, 11.2, 11.3, 11.4, 11.5, 11.6, 11.7, 11.8, 11.9, 11.10, 11.11, 11.12, 11.13, 11.14, 11.15, 11.16, 11.17, and 11.18). It must be understood that these all provide a portal through the skin barrier directly into the body and may be the source of sepsis. On occasion, the lines may also interact adversely with underlying structures, causing pathology in their own right, such as significant haemorrhage from arterial puncture in a patient on anticoagulant medication. Internal lines must be traced to the point of interaction. Thus, intravenous and intra-arterial lines may need to be cut flush with the skin and pushed slightly inwards to allow the autopsy incision to then consider whether the lines have been placed correctly.

Some knowledge of the types of lines is required, if one is to understand the pathophysiology of the patient prior to death. In this regard, one should be familiar with long lines that monitor central pressures and that provide portals for multiple drug access (e.g., Swan-Ganz catheter). Haemodialysis and intra-arterial lines should be identified and understood in terms of their basic function. Haemodialysis lines, for instance, remove blood for filtration/dialysis and then return it to the circulation.

Surgical operation closure is often an easily identified external feature, particularly if the surgery was performed shortly before death. Many surgery incisions are closed in layers with different types of suture, which can be prone to infection. Skin closure is usually made via surgical suture, or on occasion metal staples. These external closures are often removed after the skin has begun to heal, with deeper tissue sutures being left in situ. On occasion, closure is not possible and the wound is left open, either to allow the

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pathology to subside or to permit secondary closure by scarring. In this regard, the absence of a device for closure is potentially a pathology in its own right or an indicator of other issues.

Drainage lines and tubes are generally placed into cavities and are often connected to bags and other drainage devices. The amount and type of fluid in the drain collection point should be considered, because significant fluid collections, particularly those with blood, may be relevant to the cause of death. It is important not to just pull such tubes out at the start of the autopsy. Any tubes and lines that penetrate into cavities below the skin surface, such as chest or surgical drains, also need to be checked in terms of their pathway.

The presence of drug patches should be noted, with the name of the drug and the dose being recorded. It follows that the provision of therapy will often point to the terminal realities of the deceased.

Personal devices attached to the body are common. Such devices include hearing aids and false eyes. Some may indicate significant underlying disease (e.g., surgery for ocular neoplasia). Other items include piercings (commonly in the ear, but perhaps almost anywhere). The site, type, and number of piercings should be noted. Although they are not usually matters to be considered at autopsy, skin-related sepsis may be pertinent. Unusual devices that have been placed in position by the deceased may also be relevant to the cause of death.

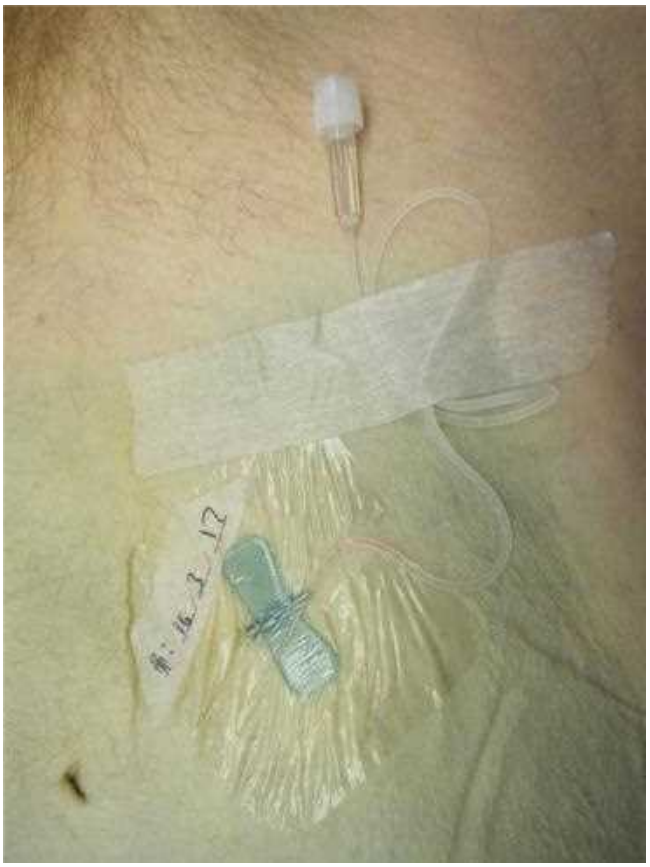

**Fig. 11.1** A butterfly needle is present on the abdomen. This simple subcutaneous drug delivery device is useful for instilling small volumes of drugs, particularly in the palliative care setting. A date is written on the dressing, indicating when it was positioned

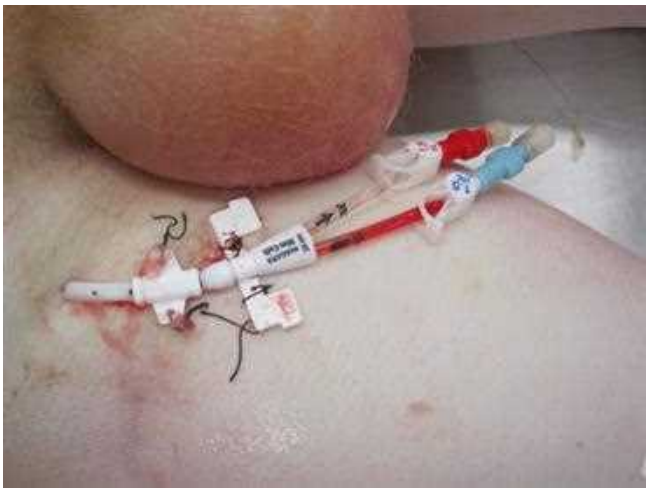

**Fig. 11.2** Various external-sited vascular lines can be seen around the torso. This groin red and blue coded line with twin arms is characteristic of a haemodialysis line, mapping to the connections for a dialysis unit. Pronounced scrotal oedema is noted adjacent to it

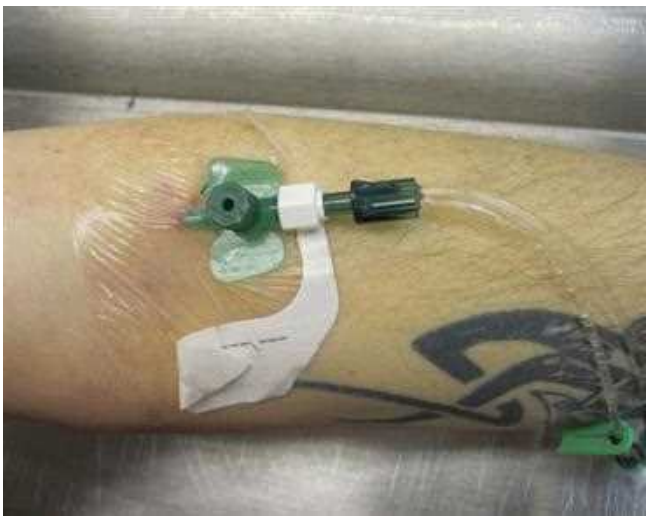

**Fig. 11.3** Many types of intravenous line exist. This one is covered with a clear dressing to assist early identification of skin inflammation or sepsis

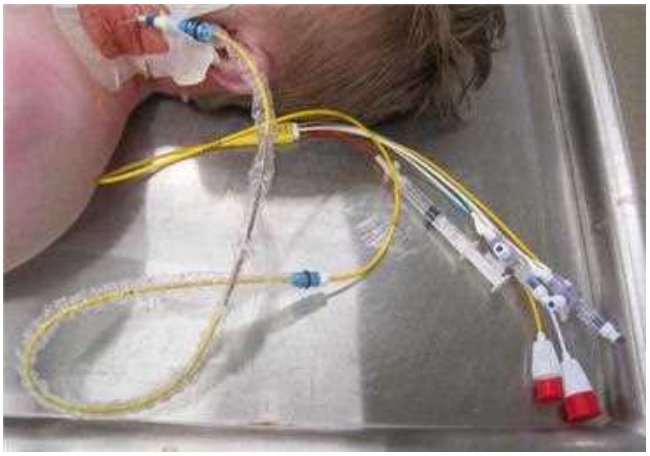

**Fig. 11.4** A multiple-channel catheter is present on the left side of the neck. This device allows monitoring of central pressures as well as the instillation of a range of different drugs or fluids

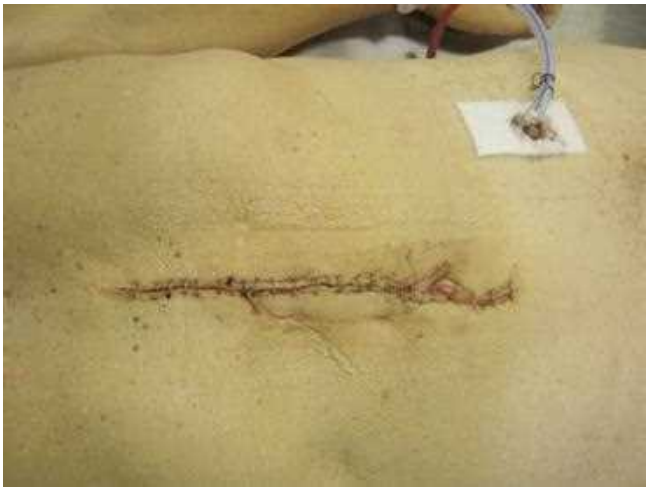

**Fig. 11.6** A stapled surgery closure is seen with a local peritoneal drain tube. Knowledge of the surgical procedure is advised before starting the examination, in order to secure the best analysis of the case

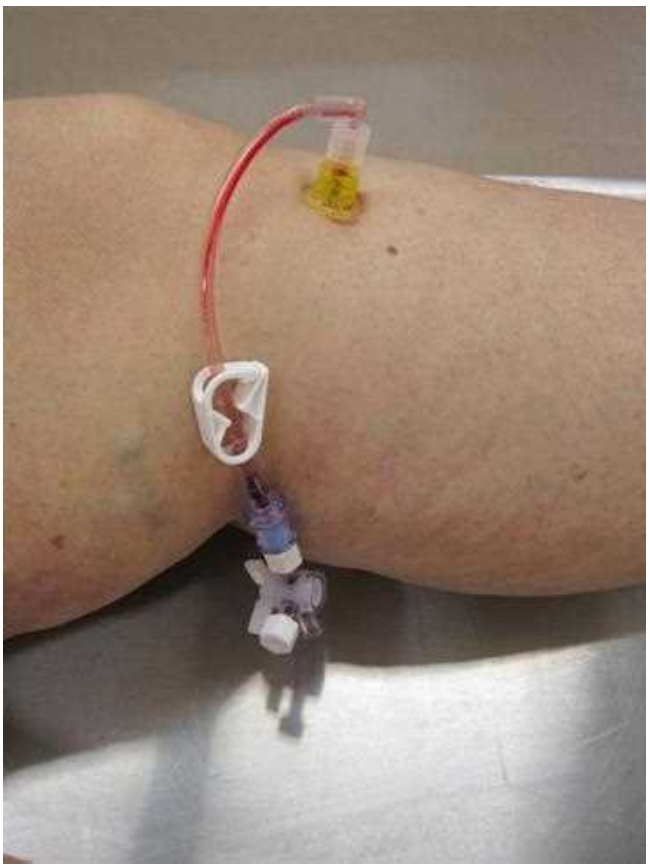

**Fig. 11.5** An intraosseous vascular line to secure rapid access to the circulatory system is seen in a patient with 'vascular shut-down'. This type of line is increasingly used in emergency settings

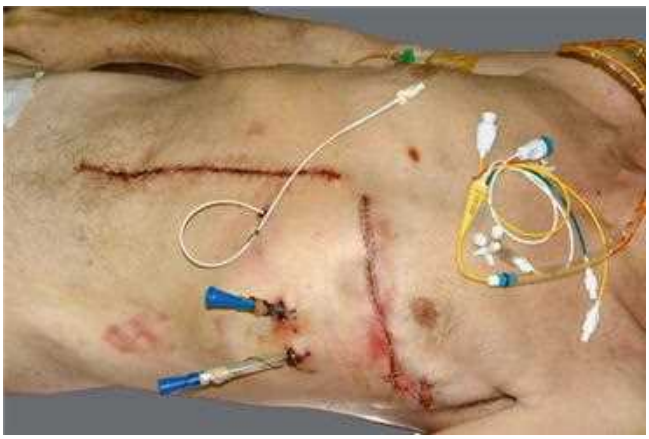

**Fig. 11.7** Investigating the death of an individual with complicated medical problems and recent surgery often is seen externally as "a complicated area". Two drainage tubes are present immediately below the left costal boundary, which were positioned within the left pleural space. A pericardial drain site is present, entering under the xiphisternum. A multiple-channel catheter was present on the right side of the neck, the end of which has been curled around onto the front of the chest. Freshly sutured and stapled wounds are present in the midline of the abdomen and the left lateral chest, and a freshly sutured chest drain point is seen in the lateral midline of the chest (*left*)

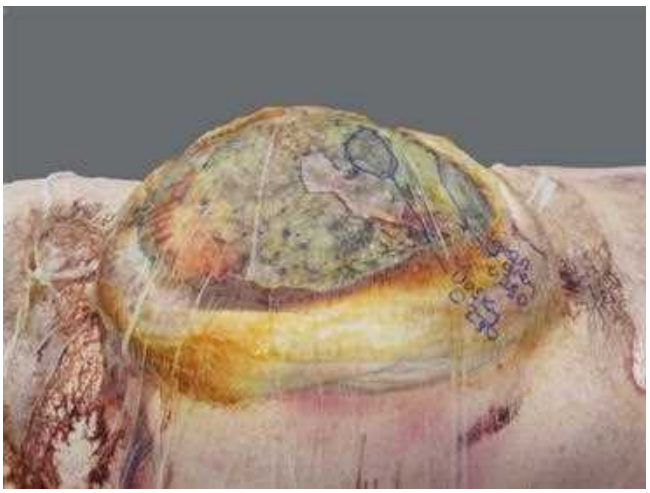

**Fig. 11.8** This abdomen has been left open because the abdominal content is grossly distended and any attempt to close would cause tissue necrosis (akin to compartment syndrome). Several gauze packs are also present within the abdomen. Critically compromised tissues may recover with this approach, with later abdominal closure. Note the plastic dressing and comments written onto the surface. This may assist monitoring of sepsis or peri-ischaemic phenomena

**Fig. 11.9** A chest drain is seen in situ. It is secured by suture and covered in a clear dressing

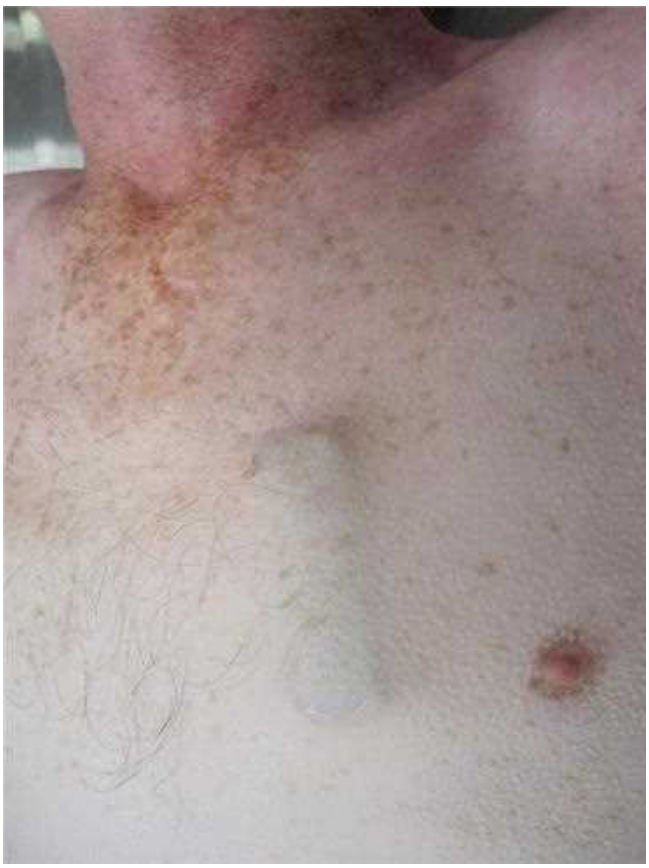

**Fig. 11.10** A Reveal® electronic sensor unit (Medtronic) is seen below the skin surface. This is an internal cardiac monitor that can have data remotely downloaded about the cardiac cycle

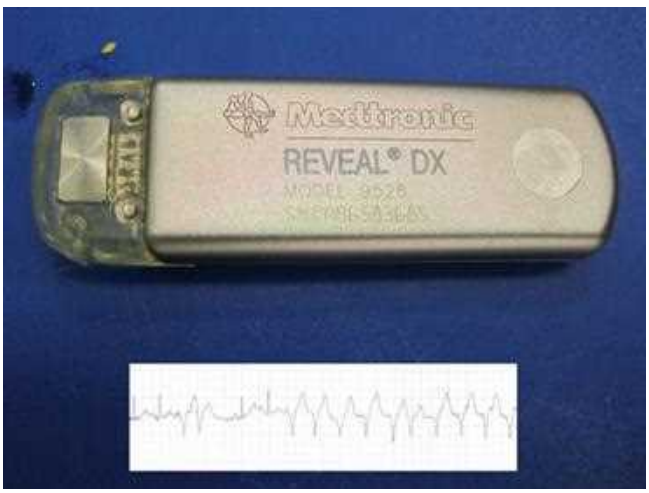


**Fig. 11.11** The Reveal® ECG monitoring device, once removed, comprises a single sealed box capable of assessing cardiac rhythm and later having that data downloaded. In the recording shown, the device has captured a ventricular tachycardia arrest

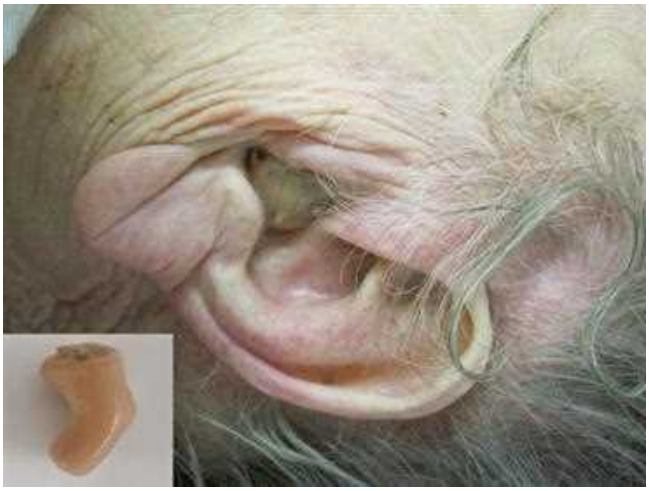

**Fig. 11.12** A hearing aid, seen in situ and outside the ear canal (*inset*)

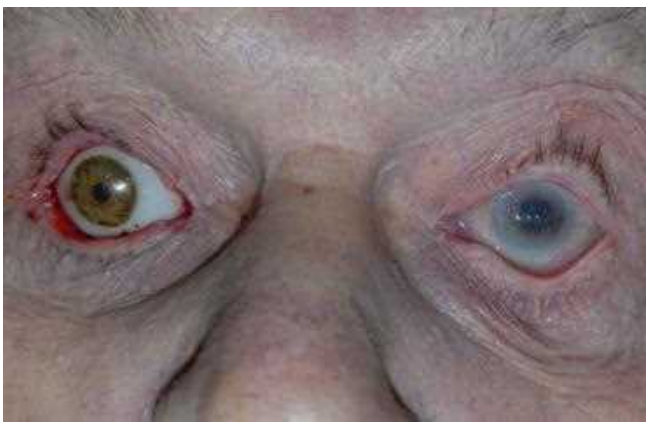

**Fig. 11.13** A glass eye (*right side*) is an unusual finding in autopsy. Often it is marked by appearing normal, compared with a suffused post-mortem eye (*left side*)

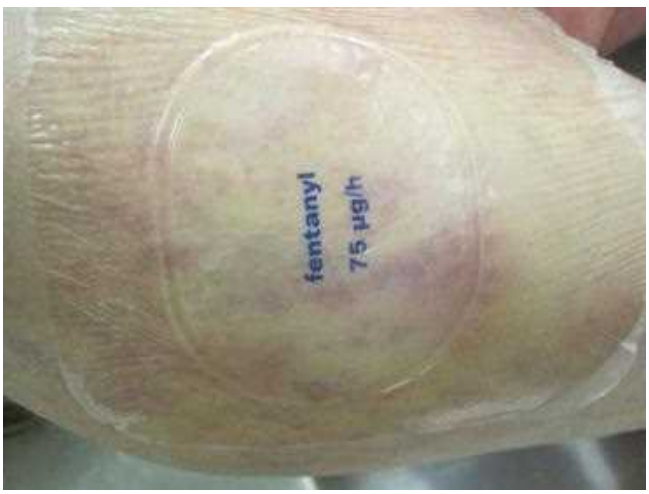


**Fig. 11.14** A drug-delivery patch, on which the drug, the dose, and the delivery rate are clearly marked

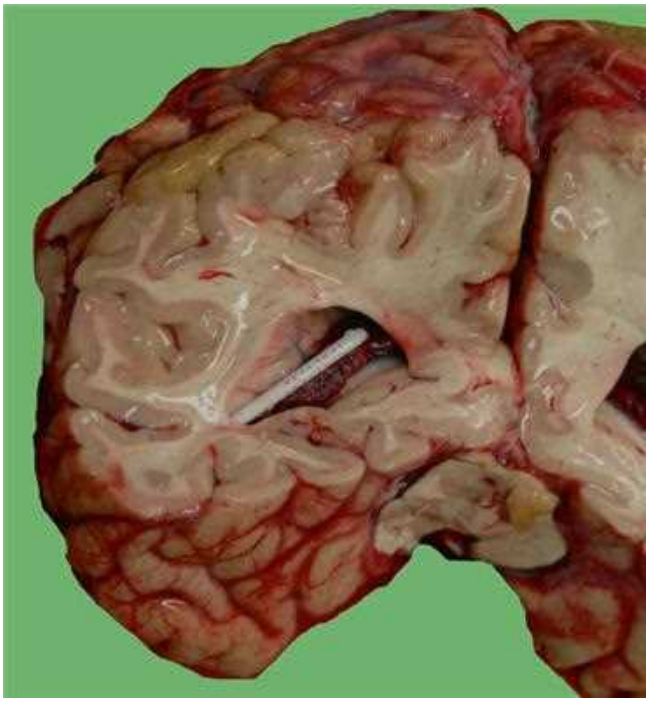

**Fig. 11.15** The tip of a hydrocephalus cerebrospinal fluid (CSF) drainage tube is present in the lateral ventricle. This tube ran into the scalp soft tissues, and then downwards alongside the body to end in the peritoneal cavity

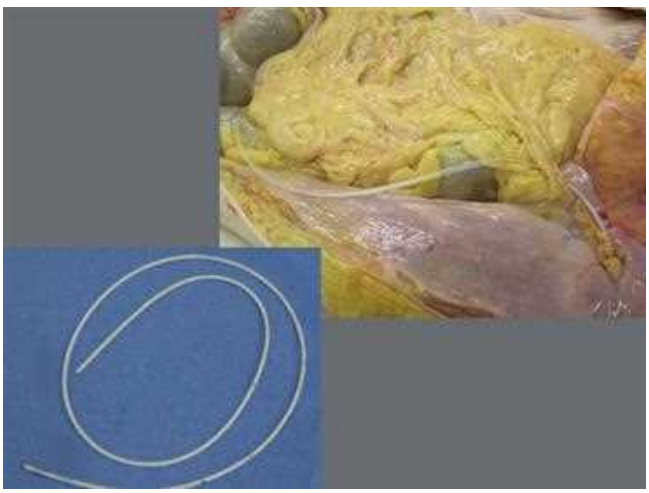

**Fig. 11.16** This CSF drain tube is seen entering the abdomen. The drain fluid is discharged into the peritoneal cavity. The *inset* shows the coiled tube after extraction, with a total length of 85 cm in this case

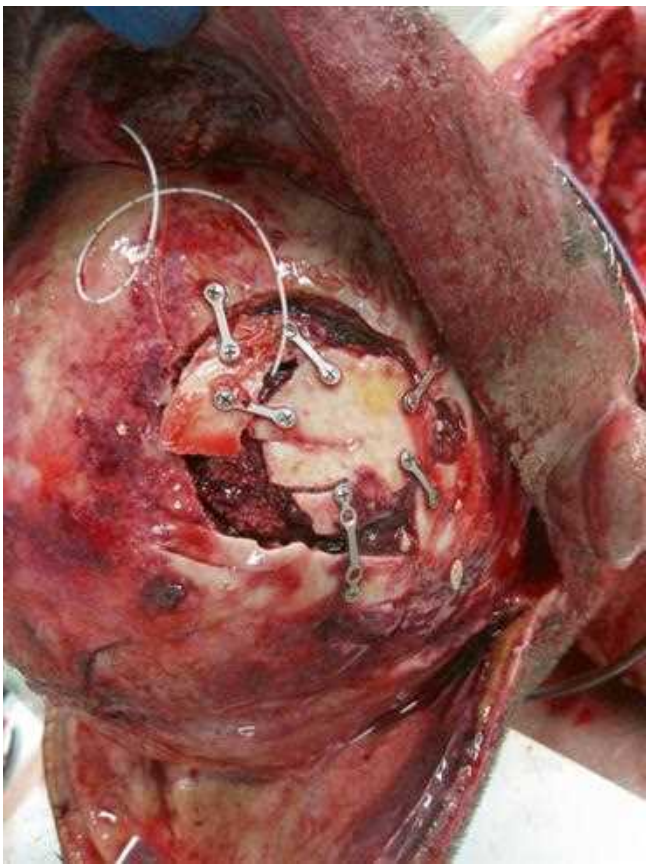

**Fig. 11.17** A complex and depressed skull fracture has been closed with original skull bone, anchored by metal struts. A central nervous system pressure transducer is entering the intracranial tissues through part of the defect left after surgery

**Fig. 11.18** Some drugs are introduced into the intrathecal space by means of a drug-eluting reservoir sitting immediately below the skin, with the drug delivery point running from the unit towards the site desired. The injection-site hub unit and part of the tube are seen

## Electronic Devices

A variety of electronic devices are available (Figs. 11.19, 11.20, 11.21, 11.22, 11.23, and 11.24). Pacemakers are covered principally in Chap. 4. Dissection of the entire unit should be considered before the autopsy starts if there is a suspicion of pacemaker malfunction.

Other pacemaker-like devices occasionally are found within the body. These include stimulator devices for the treatment of Parkinson's disease and vagal stimulators in cases of persistent epilepsy.

There are also devices with muscle stimulatory function, such as those assisting diaphragm function. These are usually activated by external electrical generators. Aural cranial nerve stimulators and other similar items act on nerve tissues around the spine. Often review of the clinical record and interaction with the relatives or caregivers will assist in the identification and understanding of these devices.

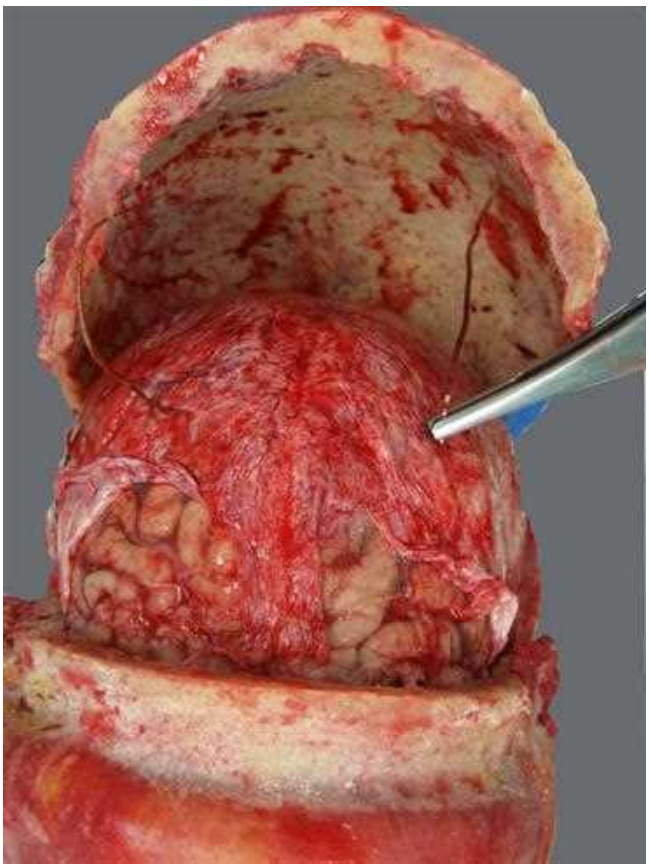

**Fig. 11.19** Two thin wires are present running through the skull and into the brain tissues. This patient had significant Parkinson's disease and had direct electrical stimulation of relevant nerve nuclei and pathways to control symptoms

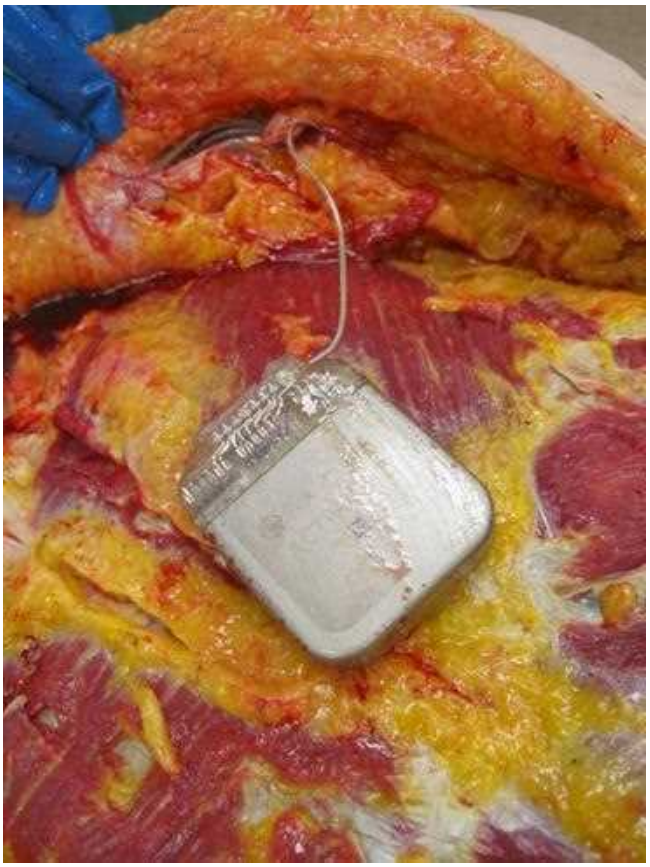

**Fig. 11.20** The stimulator unit for the Parkinson's patient was situated in a subcutaneous pocket, akin to a pacemaker, but the electrical lead ran towards the brain tissue rather than into the mediastinal/vascular compartment

**Fig. 11.21** A vagal nerve stimulator can be used in cases of epilepsy. This device, with the wires progressing towards the base of the skull, should not be confused with a pacemaker


**Fig. 11.22** A baclofen pump/drug-delivery device is seen with a drug instillation portal towards the front and a central metallic body. This device is occasionally seen in spinal injury patients who have significant pelvic floor muscle/sphincter problems


**Fig. 11.23** An aural nerve stimulator is present in the soft tissues around the neck. This is an unusual device to find within the body. It often requires review of the clinical notes or information from medical practitioners to confirm its nature

**Fig. 11.24** These electrical transducers with wires running to the low chest served to stimulate the diaphragms. An external electrical stimulator device produced electrical activity in these receivers

## Structural Cardiovascular Devices

Intracardiac devices are complex and multiple (Figs. 11.25, 11.26, 11.27, 11.28, 11.29, 11.30, 11.31, 11.32, 11.33, 11.34, 11.35, 11.36, 11.37, 11.38, 11.39, 11.40, and 11.41). Cardiac valves, as discussed and illustrated in Chap. 4, are now frequently encountered in the autopsy room. Less commonly, one may find devices related to septal defects. These congenital and acquired holes through septal tissues often can be closed by means of devices delivered by intravenous access. Such devices placed across the septal defect will permit thrombosis around the metal device, closure of the defect, and ultimately re-endothelialisation. These devices have a potential risk of infective endocarditis, but it is rare.

In the immediate postoperative setting, coronary bypass surgery and valve placement need to be considered from the point of view of clips and sutures. If significant haemorrhage occurred at the time of surgery, then pledget sutures (fabric-buttressed sutures) can be placed to assist surgical closure, and procoagulant matrix (e.g., Surgicel®) can be employed.

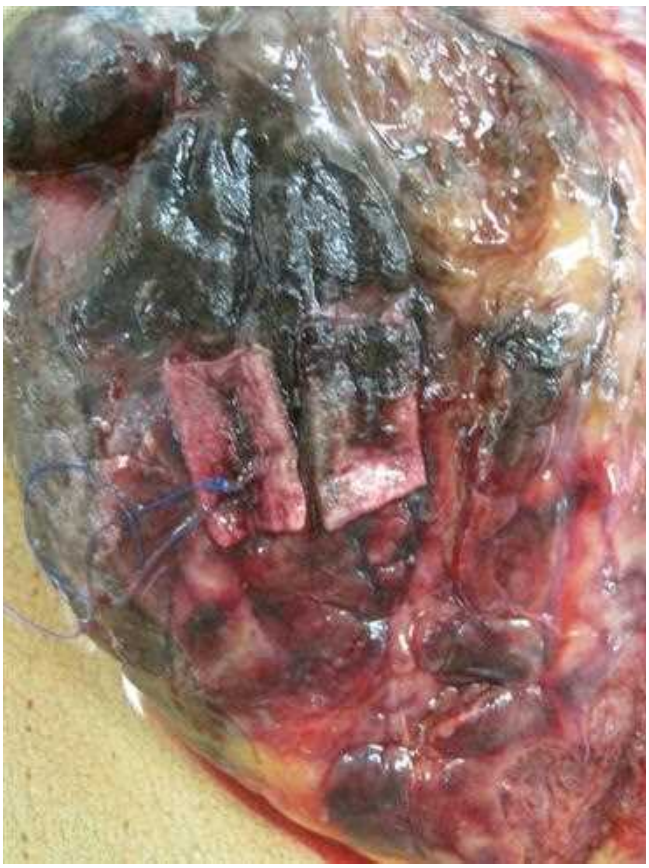

**Fig. 11.25** Pledgets (cloth-buttressed sutures) are used to try to seal bleeding points on friable cardiac tissues

Vascular stents are increasingly common, both within the coronary system and in the arterial and venous circuits. Issues to consider at autopsy are the presence and degree of internal thrombosis, any issues with regard to infection, and the effectiveness of the device. Many such devices are placed internally by means of an endovascular approach, although open surgery is occasionally employed. Sepsis is an important issue with stents, and any infection at the skin surface can potentially spread to the internal vascular device, with significant consequences.

Some intravascular devices (inferior vena cava filters) are designed to capture embolic fragments of clot. If these are present, then commentary with regard to the amount of clot and effectiveness of the device should be made.

Another vascular device, often used in the intensive care setting, is the intra-aortic balloon pump. If present, its position should be confirmed, although it will invariably be deflated at the time of autopsy examination.

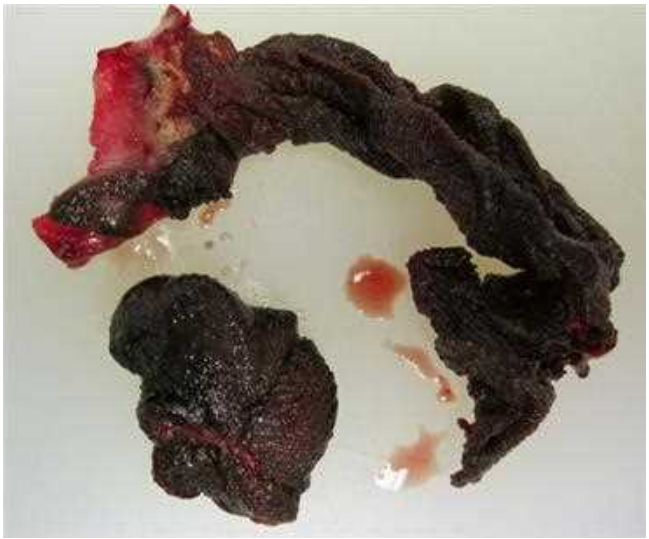

**Fig. 11.26** Surgicel® matrix is occasionally used in cardiac surgery to enhance coagulation around the heart. It is invariably heavily blood-stained and has a rather granular quality


**Fig. 11.28** A range of cardiac pacemakers: The modern variety have a smooth external case, with the manufacturer's data and serial numbers. Leads emerge from one point on the device and pass onwards to the heart. At autopsy, most cases have the generator unit (*box*) cut free from the wires to allow easy extraction. If the device may have not worked adequately, the entire unit with wires and electrodes needs to be retrieved intact and sent for specialized analysis

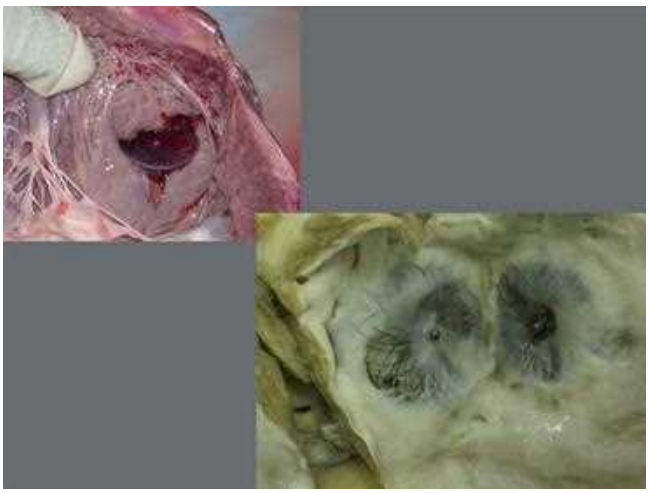

**Fig. 11.27** The left upper side shows a septal closure device that was deployed into an area of infarction-related ventricular septal defect. The device could not be deployed adequately and the individual died. The metal device is seen in an atypical position, preventing deployment. The right lower side shows two septal closure devices that have served their purpose in closing the defects. With time, local fibrosis and re-endothelialisation occur; no sepsis is seen

**Fig. 11.29** Prosthetic cardiac valves are broadly of two main types: tissue and metal disc varieties. The types currently favoured are natural (tissue) trileaflet valves and twin metal leaflet devices

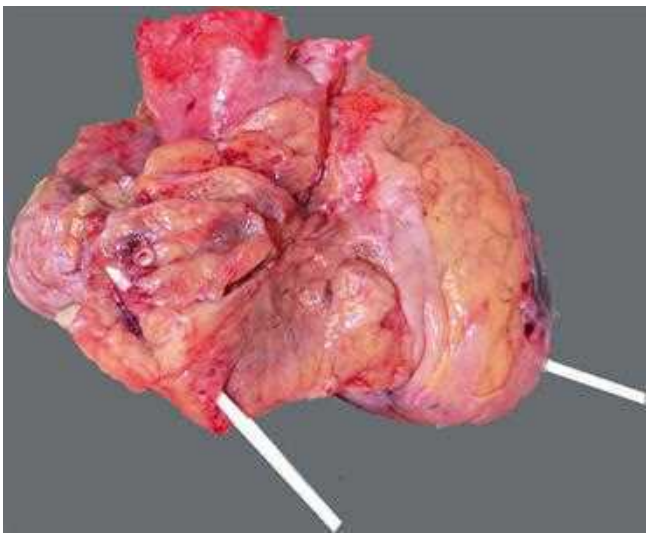

**Fig. 11.30** This heart demonstrates two attempts to drain a pericardial fluid collection. Both lines penetrated the cardiac tissues and caused death. The role of photography is clearly evident, as it demonstrates the cause of death and can be used as evidence in court if needed

**Fig. 11.31** An open operative trouser graft repair, using Dacron, has been successful on one side of the tissues, but the luminal aspect of the other limb shows abundant grey thrombotic material, indicating complete occlusion of the graft

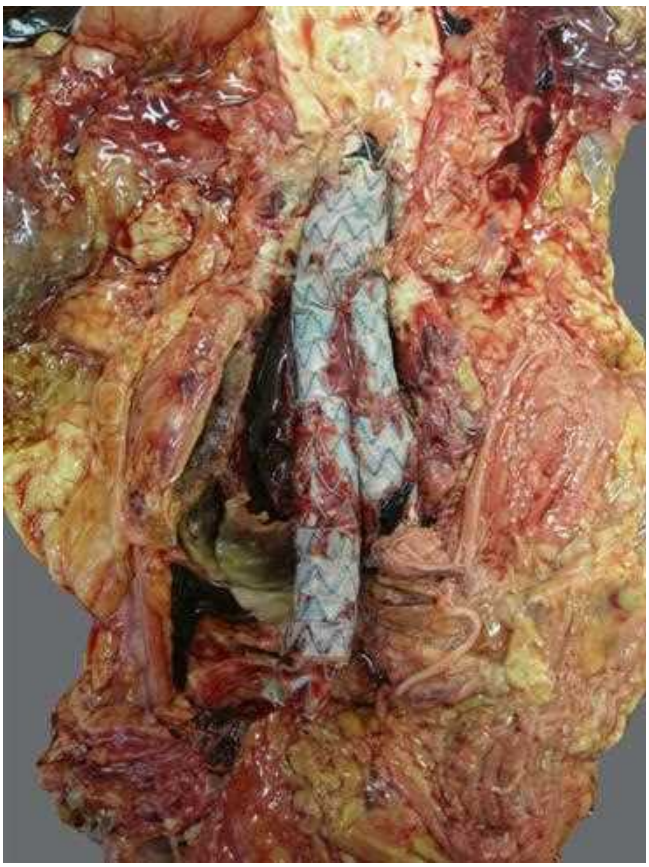

**Fig. 11.32** This endoluminal trouser graft was used with good effect in an area of aneurysm and atheromatous disease of the lower aorta. The local aneurysmal changes and thrombus are seen compartmentalized away from the lumen of the low aortic graft

**Fig. 11.33** The same graft as shown in Fig. 11.32 has been freed from the local tissues, to allow confirmation of lumen patency and absence of sepsis

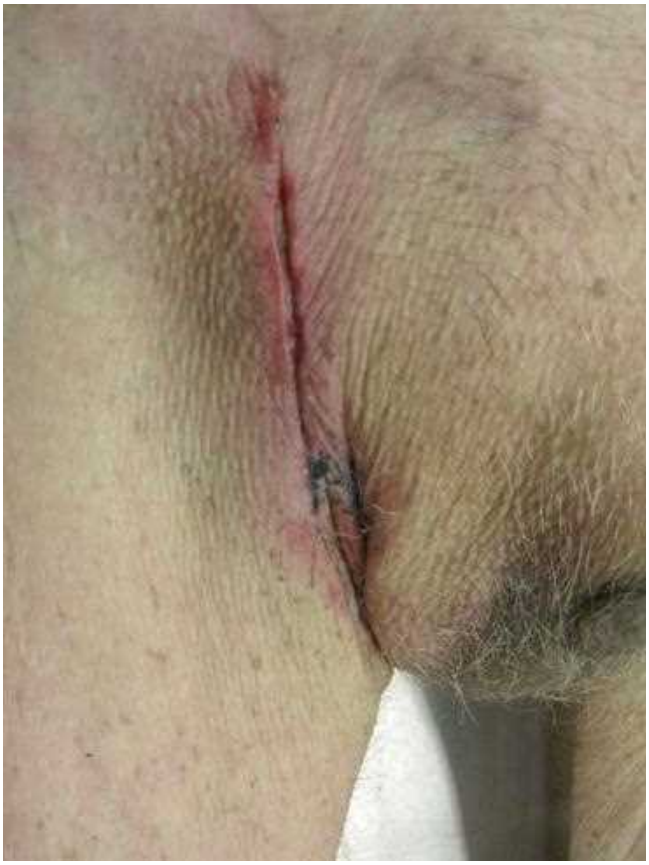

**Fig. 11.34** Grafted tissues may become infected via sepsis extending from the skin surface. Close inspection of any surgery site is recommended, with swabs or tissue sampling if infection is suspected

**Fig. 11.35** Another material used for vascular conduits is polytetrafluoroethylene (PTFE), which appears as a featureless, white-grey plastic. Histologically it has a characteristic quality. It can cause local fibrosis, calcification, and occasionally local squamous epithelial metaplasia. It can be used to assist the creation of an arteriovenous fistula for haemodialysis

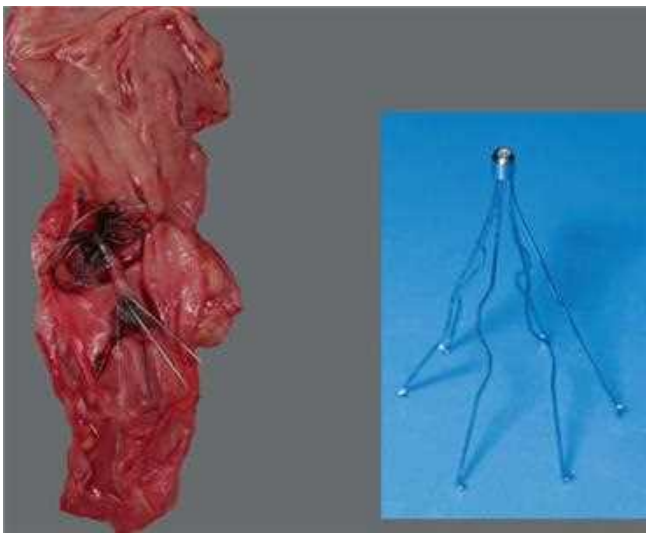

**Fig. 11.36** The venous system is occasionally used as a site for placement of endovascular cages to prevent venous embolization from leg to lung. The inferior vena cava (IVC) is a common site for a cage filter, which is aimed at preventing such thromboembolic events, particularly in patients with issues with anticoagulation control. Here the device is seen with a small amount of thrombus captured. An explanted filter is seen on the right side

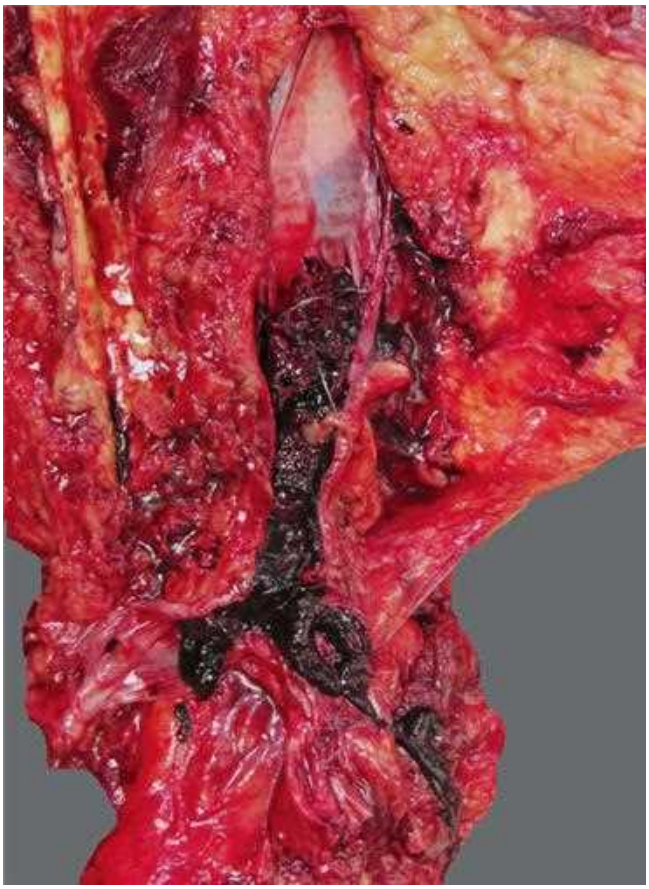

**Fig. 11.37** This case demonstrates the value of the IVC filter, having captured many embolic fragments of thrombus

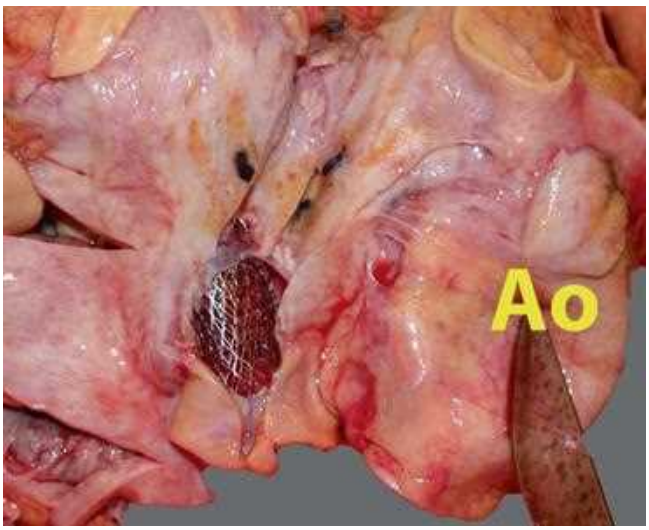


**Fig. 11.38** A superior vena cava (SVC) stent is often used to hold open the SVC when there is significant compressive tumour locally. This view shows the dense, sclerotic thoracic tumour around the SVC, adjacent to the aorta (*Ao*)

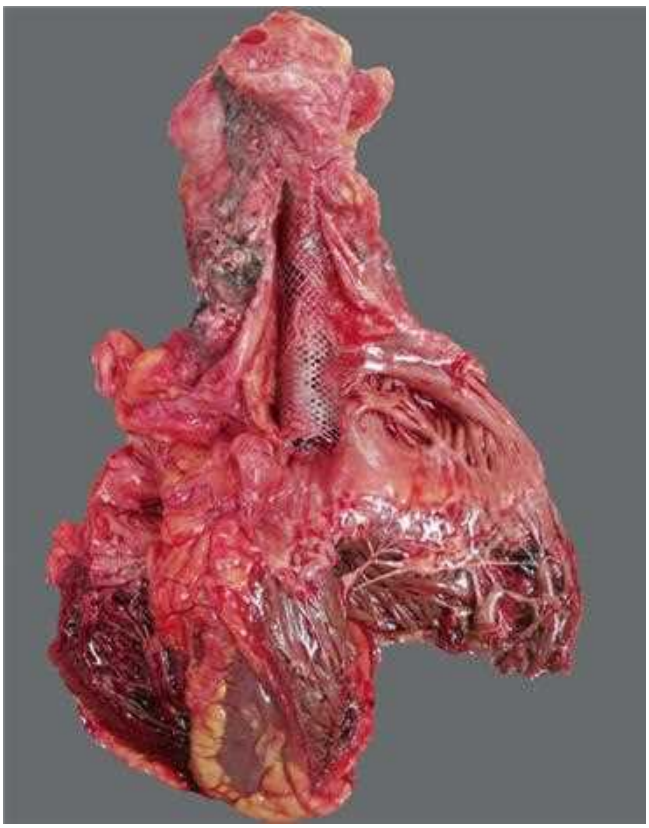

**Fig. 11.39** Another case of SVC stenting is seen with the metallic cage protecting the lumen of the SVC as it runs towards the right atrium

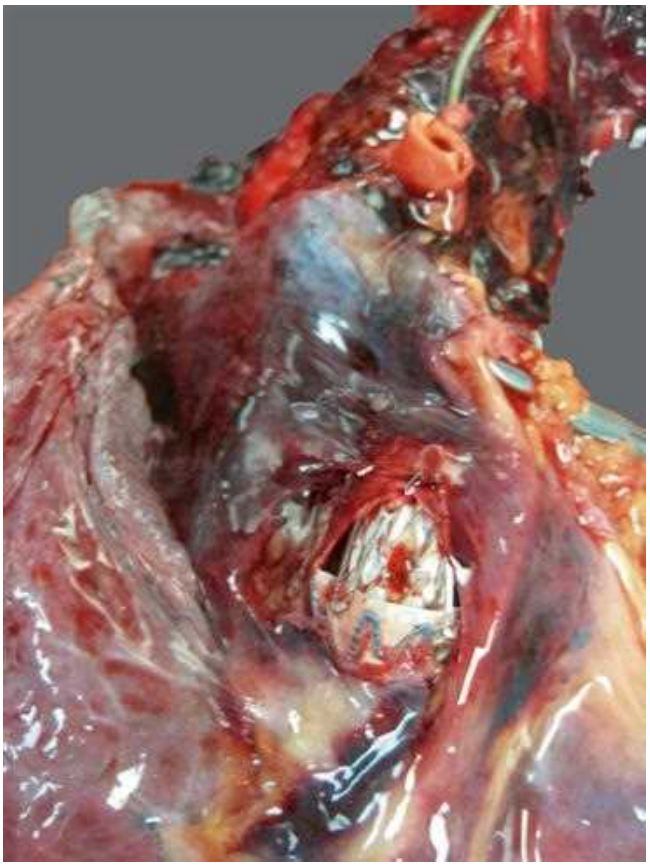

**Fig. 11.40** Long-standing pacemaker leads can become adherent to the SVC. An attempt to remove these leads may very rarely result in SVC tearing, causing massive right-side haemothorax. An emergency endovascular approach, attempting to seal the hole, was undertaken without success. The endovascular device is seen directly at the site of the large tear

**Fig. 11.41** An aortic balloon pump is often used to help maintain systemic blood pressure to vital structures in cases of acute cardiac failure. It normally lies within the aorta and will invariably be in the collapsed state at autopsy

## Gastrointestinal Devices

Gastrointestinal devices are principally of two main types, stents and feeding tubes (Figs. 11.42, 11.43, 11.44, 11.45, and 11.46). Stents are applied across inflammatory or neoplastic strictures. The most common sites include the oesophagus and colon, although the biliary system and other parts of the bowel may be treated similarly. Feeding tubes (see Chap. 5) usually are interacting with upper gastrointestinal tissues. A common example is a percutaneous endoscopic gastrostomy (PEG) tube. On occasion, some unusual devices may be encountered, such as an external flatus tube.

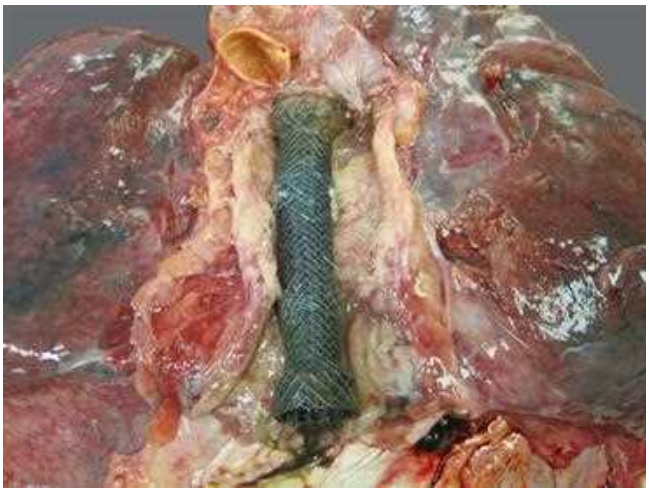

**Fig. 11.42** The oesophagus is commonly stented in cases of malignant narrowing or inflammatory strictures. Several interlocking metal stent devices have been inserted to allow oesophageal lumen patency. Whilst tumour can grow into the lumen itself, it is often not a significant problem, as further endoscopic treatments can be used, and it is also degraded by food materials passed along the oesophageal lumen. Such stents can cause oesophageal rupture (and thereby mediastinitis) at the time of placement. It is important to look for infections and involvement of local structures when considering large stents

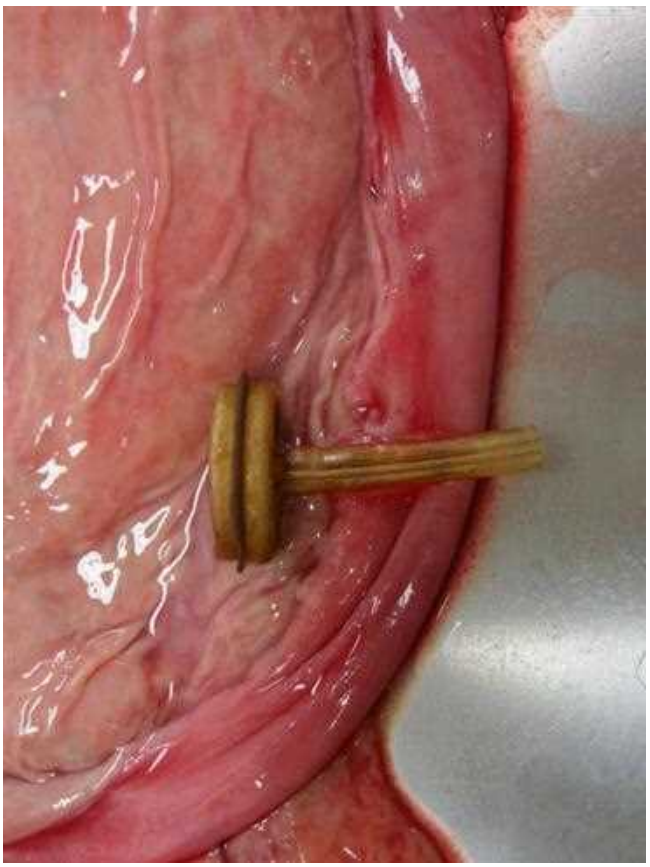

**Fig. 11.43** Percutaneous endoscopic gastrostomy (PEG) tube feeding is a common procedure in patients unable to take fluids and foods by oral means. Whether placed using endoscopic support or directly through the abdominal wall, the feeding tube is passed directly into the stomach. A similar process can be used for the proximal small bowel. The cushioned, soft, disclike end prevents the unit from being pulled backwards out of the bowel site and allows direct instillation of fluids, foods, and drugs at an appropriate rate into the bowel tissues. An autopsy with a PEG tube in situ requires consideration of the tube's entry point to the abdominal tissues as well as the tissues around the point where the tube interacts with the bowel

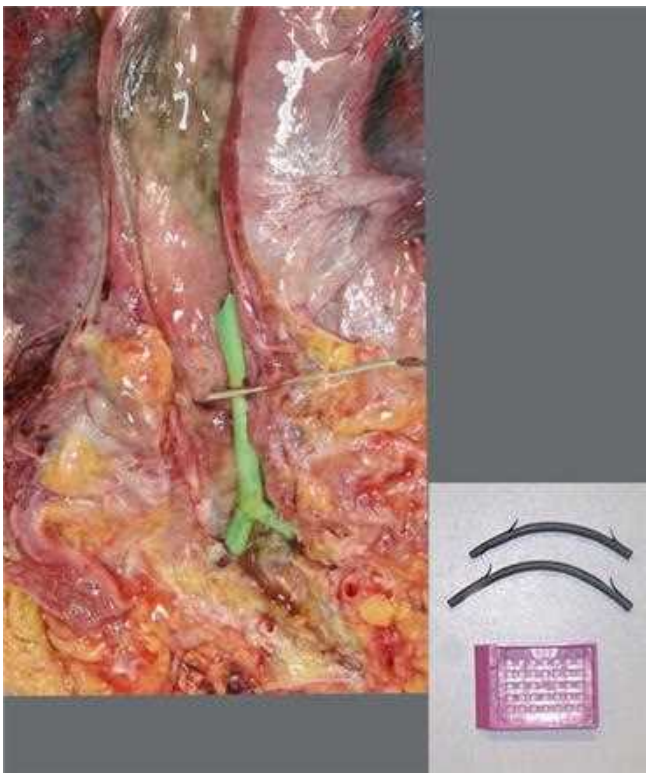

**Fig. 11.44** Plastic-type tubes are often employed in allowing materials to flow along bowel or related structures. On the left, a green T tube allows esophageal content to flow towards the anastomosed small bowel (following an esophagogastrectomy). Suture line dehiscence is noted, with a probe being passed through the defect. On the right, two plastic stents from a case of biliary obstruction due to cholangiocarcinoma are seen. Note the flanges to prevent stent slippage. These stents are normally placed by endoscopic routes


**Fig. 11.45** Although stomas can be placed directly into the upper bowel tissues for feeding purposes, one occasionally can find a flatus stoma (*f*) sited in the colon to prevent excessive gas production and thereby a risk of colon volvulus. A PEG-like flatus decompression tube was present in this loaded colon

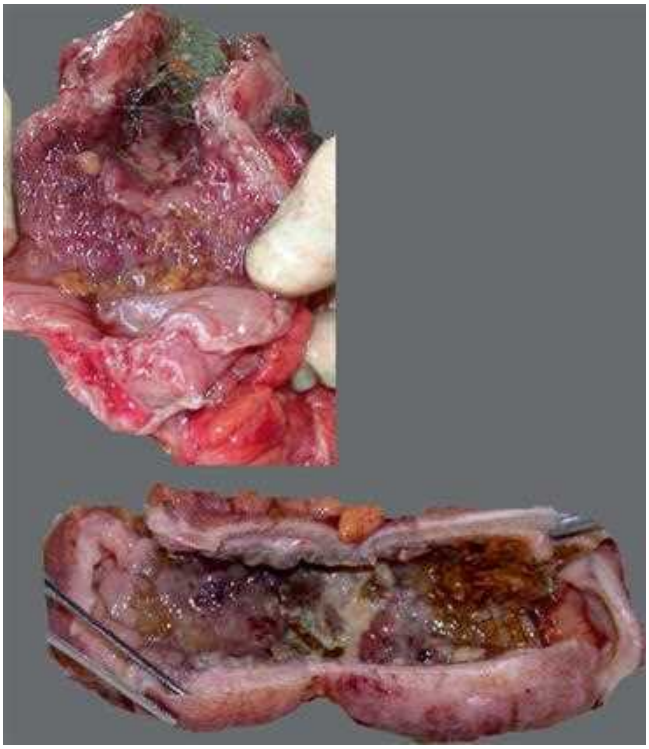

**Fig. 11.46** Strictures at the site of inoperable rectal cancers often may be treated by means of stents. These metal cages will hold open the lumen of the bowel tissues and prevent intestinal obstruction. They do not treat the tumour itself and do not become incorporated. These devices are for palliation only. The stent may fail because of tumour ingrowth into the lumen, and necrotic or infected material around the tumour may involve the metal stent frame itself and thereby local tissues. Two views of such devices are seen with the stents in situ

## Devices in the Genitourinary System

The urinary catheter is a common finding at autopsy. Some degree of bladder reddening is a consequence of inflating the tip of the urinary catheter, causing bladder surface irritation. Exclusion of sepsis around the catheter is important, but it is difficult to confirm because the urothelium is often shed early, mimicking urinary septic change (see Chap. 6). If frank pus is present, however, then a swab of the material may be pertinent. Other urinary devices found at autopsy include urostomies to the skin, pigtail catheters running between the kidney and bladder, and occasionally intraprostate metal devices (Figs. 11.47, 11.48, 11.49, and 11.50).

Devices may also be encountered in the genital tract (Figs. 11.51, 11.52, 11.53, and 11.54). Many are commonly used in the female genital tract, including ring pessaries or other pessaries, intrauterine contraceptive devices, and uterine (Fallopian) tube clips. In the male, penile implants are rarely encountered.

Hazardous materials applied as a bead or needle to deliver radiation to the local tissues may be found in some tumours. This therapy often focuses on breast or prostate tumours, but it is possible to encounter radioactive devices anywhere. Care with handling these items is mandatory, and disposal of radioactive materials will always be governed by strict regulations.

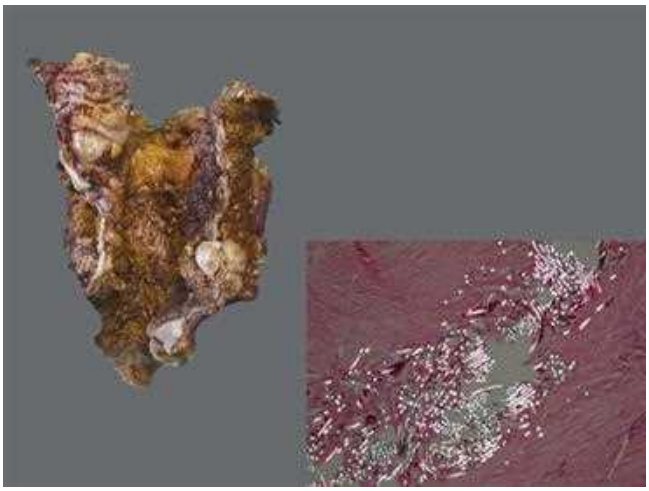

**Fig. 11.47** Laparoscopic hernia surgery with synthetic mesh devices is increasingly common. This left-side image shows a case involving chronic sepsis and extensive fibrosis into the local tissues. Samples for microbiology should be sent. The histology (*inset*) shows the mesh highlighted (in a nonseptic case) with polarized light

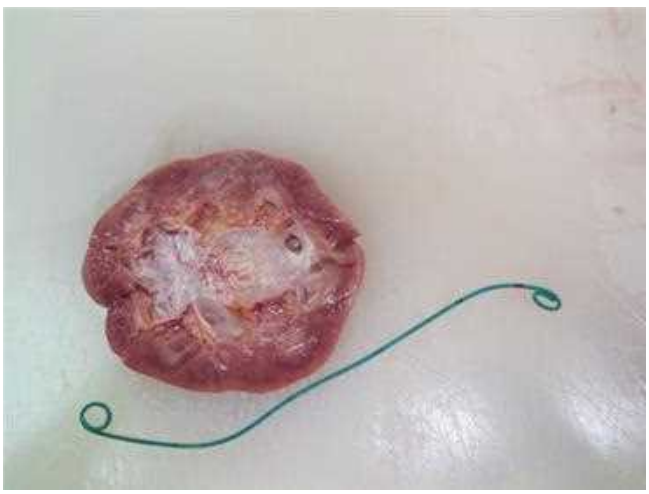

**Fig. 11.48** A pigtail catheter is used to drain urine from an obstructed kidney. One end of the pigtail will be present in the renal pelvis, with the other end in the bladder. *Pigtail* refers to the coiled nature of the distal end of the drainage tube

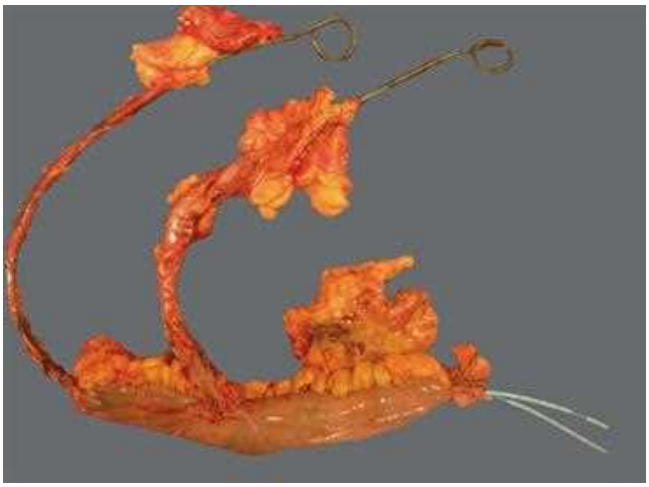

**Fig. 11.49** A dissected urinary ileal conduit has been assisted in terms of drainage by inserting two tubes that ran into the renal pelves. Such tubes normally accompany ileal conduit surgery, as a method of initially assisting the kidney urine outflow and thereby recovery

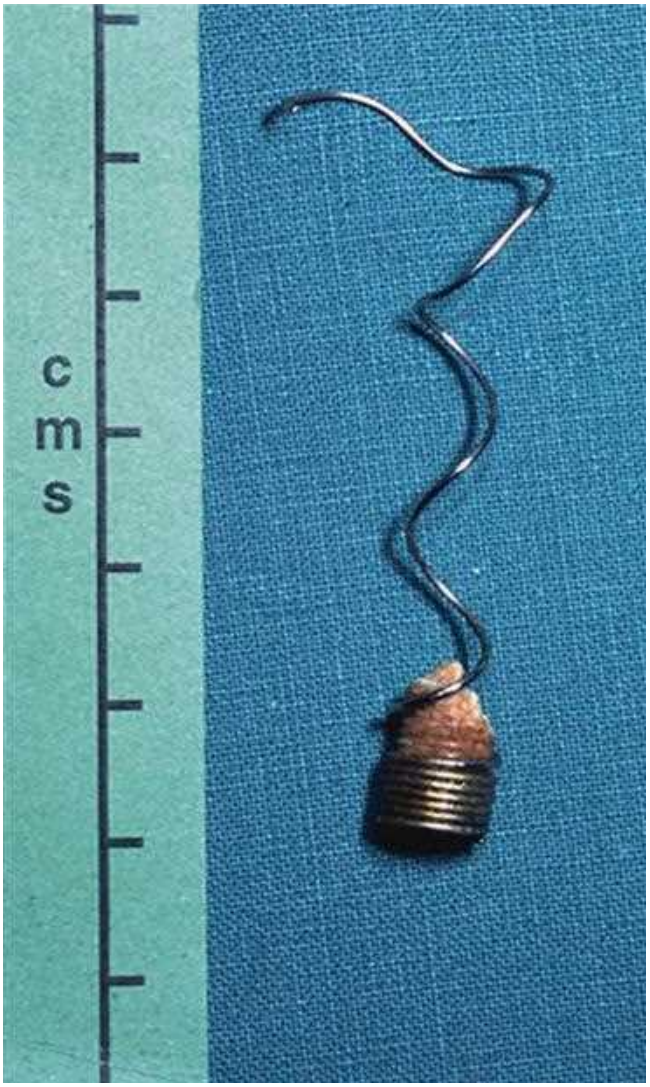


**Fig. 11.50** Some urinary stents are placed at sites of strictures in the prostate and bladder outlet. These can become encrusted with bladder stone material and thereby obstruct the urine outflow. This stent has been partially pulled apart to reveal the calculus material

**Fig. 11.51** This set of rings was placed around the genital region in an individual found deceased in bed, surrounded by various erotic literature. The cause of death reflected ischaemic heart disease, rather than a specific complication of the rings

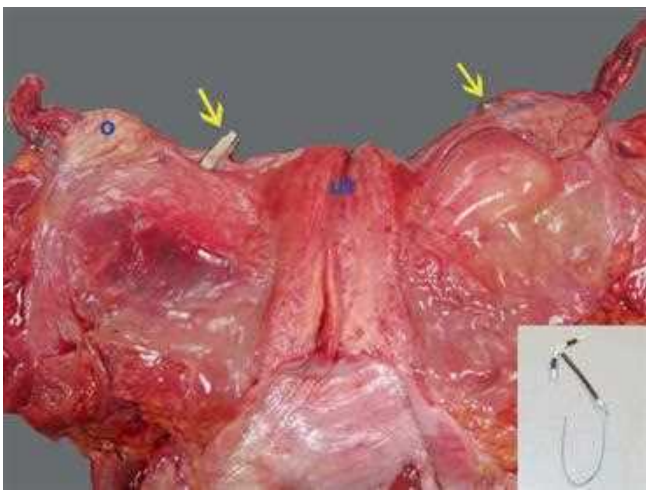

**Fig. 11.52** This view of the uterine tissue (*ut*) shows sterilization clips (*arrow*) on the tubes leading to the ovary (*o*). The inset shows an intrauterine contraceptive device (*IUCD*)

**Fig. 11.53** A vaginal pessary. Not all are ring-shaped

**Fig. 11.54** A large skull fracture and bone defect can be treated with a metal plate to protect the brain. This metal plate is bent to fit the patient's outer cranial vault and fixed in position by small screws at the edge. Here it has been dissected free (See also Fig. 9.72)

## Supports for Soft Tissues and Bones

Many devices are used to support structures, such as mesh for abdominal hernias, bone prostheses, and metal plates for craniotomy wounds (Figs. 11.55, 11.56, and 11.57). In all autopsies where such items are present, their position must be recorded and sepsis must be considered. In most cases, if there is no overt pathology, then the device does not need to be removed, although one should be able to extract metal devices and bony tissues or soft tissue and support devices en bloc if there is an issue with regard to the alignment of the repair or consideration of neoplasia or sepsis. Clearly, if the device has failed, then photography and radiology may be of assistance. Review by the manufacturer also may be helpful.

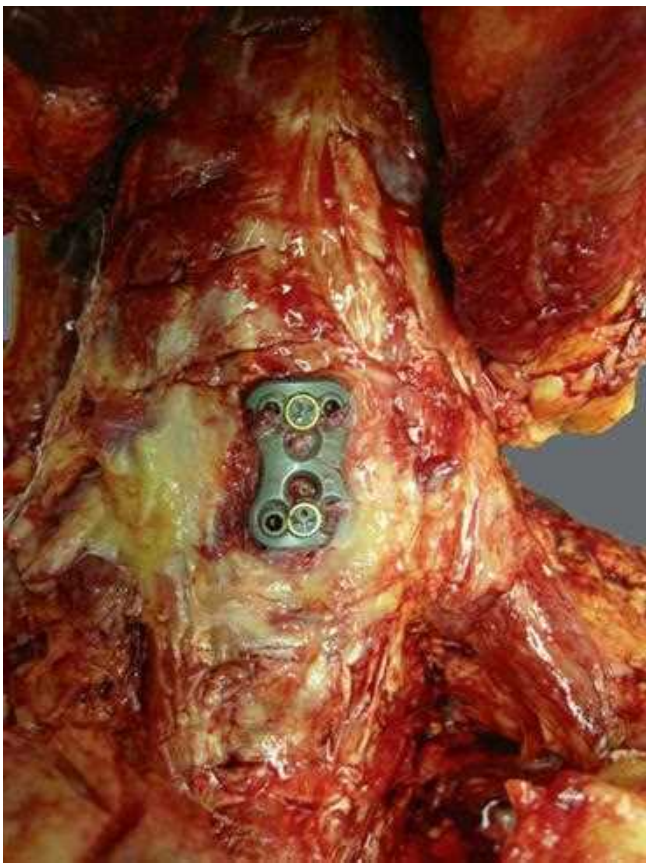

**Fig. 11.55** Features of spinal stabilization may be evident when the body is open and the cervical, thoracic, and abdominal tissues have been removed. This metal device had been positioned many years beforehand, to stabilize a fracture/dislocation of the spine. In cases of recently completed surgery, it is important to check for local sepsis

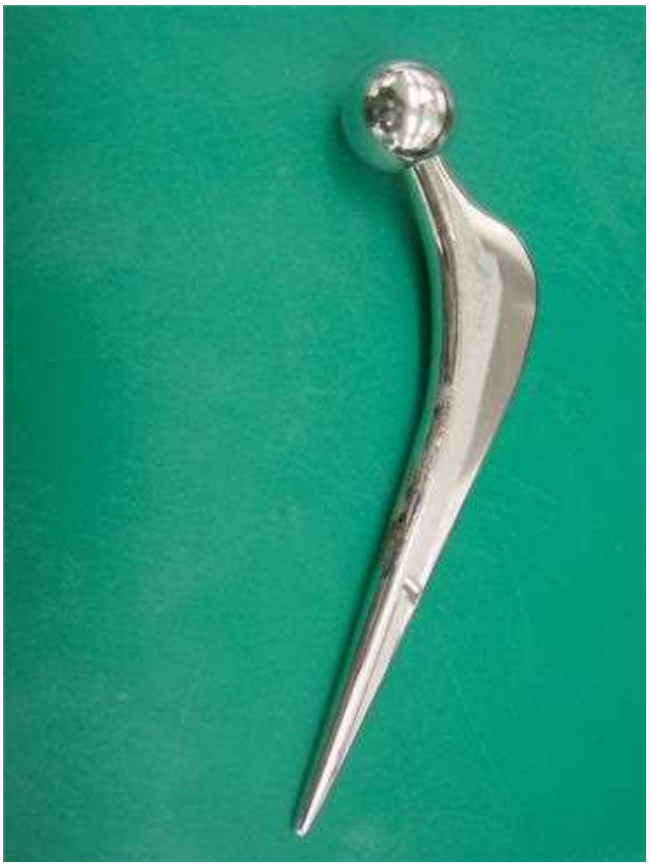

**Fig. 11.56** A variety of metal joint replacements exist, as exemplified by this hip prosthesis. This type of device is commonly found in autopsy cases, following acute fractures or elective replacement surgery. If the bone alignment is good and there is no ante-mortem issue, then complete explantation is not needed, but if there is any issue of sepsis or mechanical failure, then prosthesis examination is advised

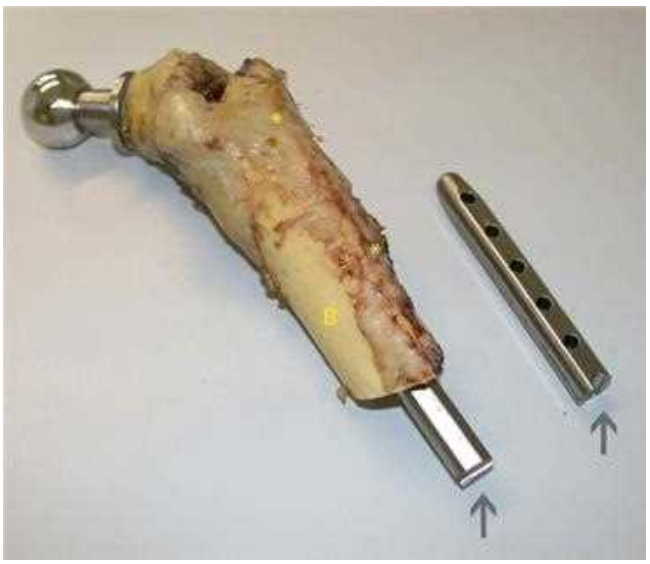

**Fig. 11.57** This complicated hip fracture needed a metal prosthesis and reinforcement with allograft bone and cement (*asterisk*). Whilst this procedure was a success with regard to allowing incorporation into native bone (*B*), the metal device failed, with fracture of the metal stem adjacent to a drilled hole (*arrow*)

## Respiratory System Devices

In the respiratory system, endotracheal tubes are regularly seen. It is important to verify that the tube has been positioned in the larynx and trachea rather than misplaced into the oesophagus (see Fig. 4.138). On occasion, laryngeal masks may be found as an alternative and simpler method of ventilating the unconscious patient (Fig. 11.58 and 11.59). Unusual devices that may be found, particularly in the context of previous treatment for laryngeal cancer, include valves within the esophagus/trachea to assist phonation (Figs. 11.60 and 11.61).

**Fig. 11.58** Antibiotic beads are an uncommon therapy that was previously used. These drug-loaded beads elute antimicrobial therapy directly into a cavity or other site that may have poor vascularity

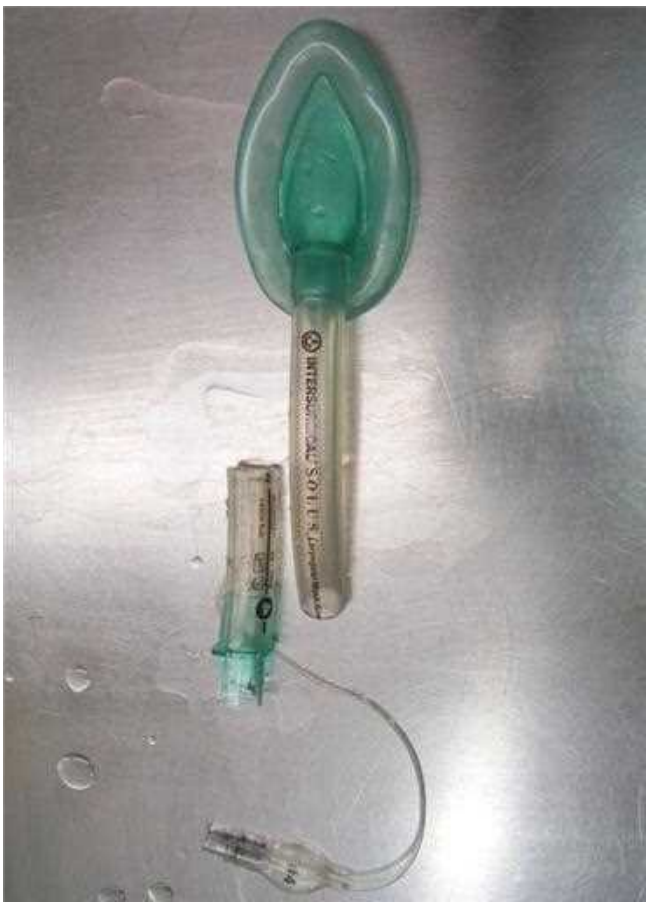

**Fig. 11.59** A laryngeal mask has been used in this individual, rather than an endotracheal tube. To facilitate its extraction and analysis in situ, the outer end of the tube has been cut, allowing the airway end to be left in situ for inspection at the time when the neck tissues are mobilized

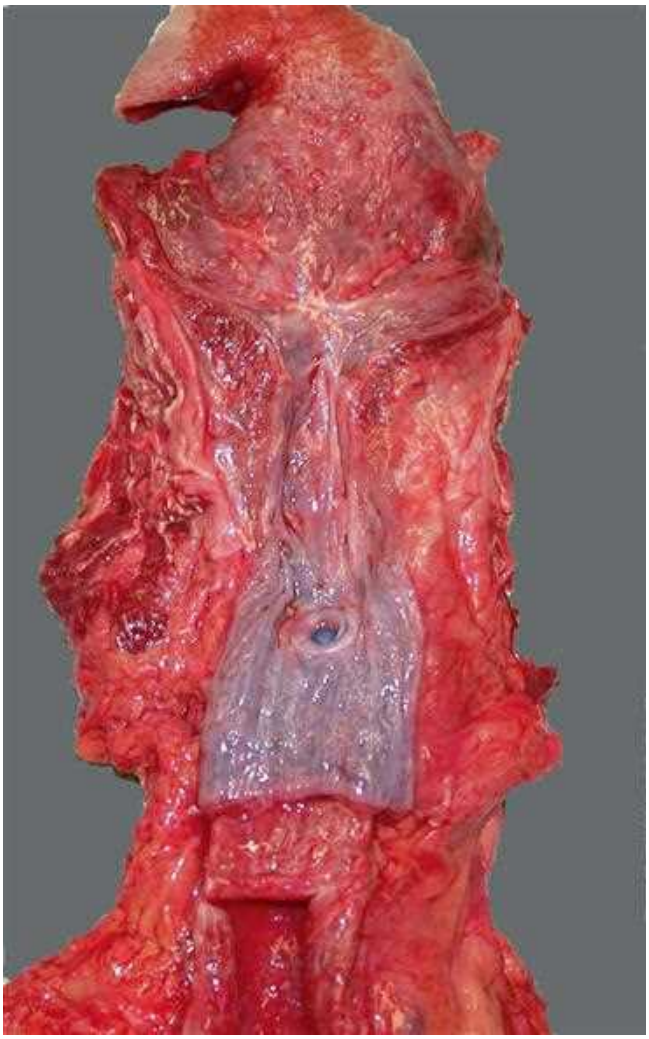

**Fig. 11.60** A valve is present at the upper end of the esophagus, linked to the trachea, to allow some phonation in those who have had laryngeal cancer treatments and surgery

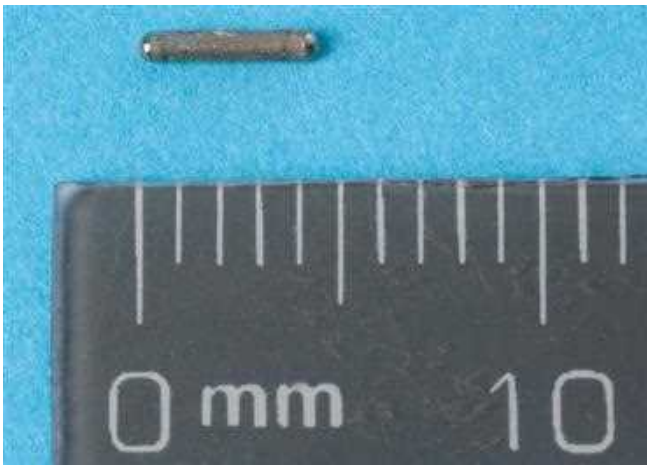


**Fig. 11.61** Radioactive beads (and other implants) are a potential hazard for those performing autopsies. Although they often appear innocuous, one should have a high index of suspicion if such implants are present in an area of tumour, even if they were not declared beforehand (*Reprinted with permission from* Start RD, Tindale W, Singleton M, Conway M, Richardson C. Radioactive prostatic implants: a potential autopsy hazard. *Histopathology*. 2007;51:246–8)

## Suggested Reading

Burton J, Saunders S, Hamilton S. Atlas of adult autopsy pathology. Boca Raton: CRC Press; 2015.

Finkbeiner WE, Ursell PC, Davis RL. Autopsy pathology. A manual and atlas. Philadelphia: Churchill Livingstone; 2004.

Singleton M, Start RD, Tindale W, Richardson C, Conway M. The radioactive autopsy: safe working practices. Histopathology. 2007;51:289–304.

# The Forensic Autopsy

12

Philip D. Lumb

## Introduction

Not all cases coming to autopsy examination reflect deaths that are natural. Some have medicolegal considerations reflecting matters of nursing care, medical negligence, violent deaths, industrial accidents, suicide, homicide, and so on. The investigating police officers may require urgent examination of a deceased individual to determine if a crime has been committed, so that they can manage their own resources. For those undertaking any autopsy examination, whether anatomical technician or pathologist, a degree of dutiful suspicion should always exist.

This careful scrutiny must continue throughout the entire examination, and one should always be prepared to consider stopping the case if any suspicious features are found. To this end, those performing the autopsy must be able to distinguish pre-mortem versus post-mortem injuries to the body. One should also be able to discern mimics of injuries and appreciate how injuries may have been inflicted.

Various types of injury need specific consideration. These include deaths at home and the workplace, including fire deaths and electrocution. Deaths on the highway, whether in a vehicle or as a pedestrian, need a thorough assessment of the pattern of injuries, particularly if others are to be held liable for the death. Finally, there is a group of post-mortem changes that may be suspicious but are actually consequences of local and natural phenomena.

In simple terms, the forensic autopsy covers the same ground as a standard autopsy, but it goes into greater detail in terms of analyzing the pathology, recording the injuries (if present), retaining samples (e.g., evidence and other tests), and liaising with the police and other relevant parties. To this end, performing the autopsy with other personnel (police, scene of crime officer, photographer, etc.) is required.

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## The Forensic Autopsy Specialist Dissections

The examination often must go beyond the routine incisions, extending the dissection around various parts of the body.

### The Neck

Detailed examination of the neck should take place in a "dry" bloodless field in order prevent artefacts such as the Prinsloo-Gordon lesion. Decompression of the neck should be made by initially removing the brain, thereby assisting by draining the head of some blood. A wide "V" incision should then be extended from the region of the upper chest, along the lateral aspect of the neck, and behind both ears. The angle of the 'V" is just below the top of the sternum centrally. The skin should then be reflected over the chin. Each of the strap muscles are sequentially dissected, layer by layer. Each muscle can also be individually incised to examine for subtle internal bruises. For completeness, the posterior triangles of the neck also should be exposed, and the fold of skin over the occiput also can be dissected inferiorly to reveal the posterior musculature at the back of the neck. Once the musculature has been inspected, the other neck structures can be readily exposed and carefully dissected away from the spinal column. The laryngeal inlet and any obstruction process are readily visualized at this point, using this technique.

### The Face

Standard forensic autopsies usually involve dissection of the subcutaneous tissues of the face. The "V" neck incision fully exposes the neck structures and is then extended behind both external auditory meatuses and over the mental process of the chin. The buccal surfaces are exposed and the incision can be extended to the orbits, which also allows access to the orbital contents. This dissection allows



S.K. Suvarna (ed.), *Atlas of Adult Autopsy: A Guide to Modern Practice*, DOI 10.1007/978-3-319-27022-7_12

assessment of deep facial bruising and fracturing. The lips also can be examined in detail. Reconstruction is relatively straightforward; in the hands of a skilled technician, the individual's appearance will not be altered, even for identification purposes.

### Posterior Skin Flay, Including Upper Limbs

Dissection of the back can reveal very extensive injuries that are not visible on the skin surface but may cause or contribute to death. Such injuries include large hematomas causing exsanguination, or pulping of the subcutaneous fat, producing fat embolism. A cruciate incision should be made and extended down the back. Extension can also be made down the arms to reveal deep bruising, such as that which may be caused by restraint or gripping. The dorsum of the neck can also be exposed to assess the posterior ligamentous attachments, spinous processes, and laminae of the cervical vertebrae.

## The Lips

Following deep dissection of the face, the inner aspects of the lips are exposed. Some incisions can be made to assess for deep lip bruising. Deep lip bruising may be encountered in blunt force assaults, although subtle bruising seen in smothering cases may also be exposed by using this technique.

### The Orbit

Following dissection of the face, it is then possible to extend the dissection to expose the tissues and bony walls of the orbit. This is particularly useful when investigating the cause of periorbital hematomas. These "blowout" fractures of the floor of the orbit suggest direct force to the ocular globe, such as from a punch. Indeed, it is important to remember that, rarely, oculocardiac reflexes can be the cause of death. The lateral orbital margin is exposed and blunt dissection is made posteriorly into soft tissues of the orbit using snub-nosed scissors. Freeing this tissue should expose the optic nerve, which is then cut with scissors. Gentle retraction on the globe and further blunt dissection posteriorly and medially free the globe from the orbit. Note that the globe remains attached to the inner aspect of the eyelids by the conjunctivae, allowing simple reconstruction of the face afterwards.

### The Vertebral Arteries

The extracranial and intracranial portions of the vertebral arteries may become damaged with blows causing rotation of the head upon the spinal column, leading to basal subarachnoid haemorrhage (see also below). Careful dissection of the extracranial vertebral arteries is mandatory in such cases, as dissection and stretch-related changes may be identified. The vertebral artery canal is deroofed with small bone clippers (tailor-made equipment is also available), and the arteries are carefully removed. It is recognized that accessing the upper extracranial vertebral artery (between C1 and the dural insertion) is difficult and is best done by exposing the area with the deceased lying face downwards.

### Anterior Skin Flay

Subcutaneous dissection of the front of the chest and abdomen can reveal subtle bruising that may not be visible externally.

### Forearm and Hand Flay

The incision lines should be extended from the base of the proximal phalanx of the little finger, ulnar aspect, along the ulnar aspect of the forearm towards the elbow. A second incision can then be extended from the metacarpophalangeal joint of the thumb and then down the radial aspect of the forearm. A third transverse incision then should join the outer forearm incisions, and the skin can be flayed back from the forearm over the metacarpophalangeal joints of the knuckles, facilitating the assessment of subtle deep bruising and hidden needle puncture marks. The knuckles can be assessed for offensive injuries (e.g., punching). Reconstruction preserves much of the skin of the hand and is thus more satisfactory for relatives of the deceased than the more traditional hand incisions along the length of each finger.

### Anogenital Block

A marker should be placed at the anal verge in the 12 o'clock position, so that the location of injuries can later be identified after dissection. Internally, the bladder, uterus, and rectum should be freed from the pelvic wall by blunt dissection. An ellipse of skin is then made around the pudenda to include the anus and vulva. The subcutaneous tissues are then carefully dissected and deepened into the pelvic outlet, to free up the entire block. Following removal, the anus, rectum, vulva, and vagina are easy to access for detailed inspection and photographic documentation of injuries.

### Excarnation/Defleshing

Decomposition of the corpse can hamper careful inspection of the skeleton for features that may be relevant to the iden-

tity of the deceased or the cause of death (such as subtle bony incisions from stab wounds). After general tissue examination and preservation of samples for additional tests (e.g., toxicology, DNA), bone cleansing may be used. This specialist technique is simple and effective. Different methods are available, but they generally involve the incubation of partially defleshed tissue in a bath of proteolytic enzymes.

## Sharp Force Trauma

Sharp force injuries can be generally categorized into incised wounds and stab wounds. Puncture wounds often are also included in this group and can be defined as shallow penetration of the tip of an implement. Sharp force injuries can be differentiated from lacerations by examination of the wound edges, which are cleanly divided and without tissue bridges. Hairs crossing over the wound also may be divided neatly. Unlike with lacerations, abrasions at the edge of the wound are relatively uncommon, unless severe force has been used (for example, the hilt of a weapon had been driven into the skin).

### The Stab Wound

A stab wound can be defined as a sharp force injury that has a wound tract that is deeper than the width of the injury at the skin surface. Stab wounds are usually caused by bladed weapons such as knives, which are thrust into the tissues.

The following information should be gathered about each stab wound: location, in terms of height from the heel and distance from the midline on the torso (or other fixed point on the body); length; shape of the ends of the wound, direction of the track; tissues penetrated; and depth of the track. These features can help identify the weapon used.

Stab wounds that are orientated across Langer's lines (tension lines in the skin and subcuticular tissues) can gape open and become shortened, misrepresenting the width of the blade. In this situation, gentle tension should be applied to reform the wound. Sharp or pointed wound ends indicate that it is likely that there has been contact with the forged cutting edge of the blade, whereas a squared or blunt end indicates contact with the unforged spine of the blade.

### Stabbing Injuries: Force

The force used to cause a stab wound should be assessed. Many pathologists use a scale of mild, moderate, and severe force. Mild force usually implies slow movement of the weapon; deeply penetrating wounds caused by mild force are usually seen only when the individual is restrained and unable to fend off the weapon as it is relatively slowly inserted into the tissues. By contrast, severe force has been used when thick, bony buttresses such as the sternum, the full thickness of a rib, or the calvarium are penetrated. Moderate force lies somewhere in between—with the blade moving quickly enough to overcome the evasive actions of the deceased but not necessarily having the force to penetrate through thick bone. When assessing the force used to describe a deeply penetrating stab wound without bony injury (or with minor bony injury), the term "at least moderate force" is used in the autopsy report. Whilst moderate force may have been used to cause such deep injuries, severe force cannot be excluded; it just so happens that the stab wound has not struck bone that might serve as a marker of force. Hilt marks (or more rarely, fingernail marks) alongside the stab wound suggest that the weapon has been driven in deeply with severe force.

### Defence Injuries

The discovery of incised wounds or stab wounds to the hands or ulnar aspects of the upper limbs raises the possibility of sharp force defense injuries and that the individual has been attacked by an assailant. During the course of an assault with a sharp weapon, the hands and forearms are often instinctively raised to protect the torso and head from injury. The bladed weapon may even have been grasped in the hand and, upon removal, incised injuries can be seen across the palm or palmar aspects of the fingers, not uncommonly in the interphalangeal skin creases. The weapon also may be drawn through the web of skin between the index finger and thumb, causing deep incision. Similarly, as the individual fends off a sharp force attack with the upper limbs, the back of the hands and often the ulnar aspect of the forearm are presented to the oncoming weapon, causing incised wounds to be inflicted.

### Self-Inflicted Fatal Stab and Slice Wounds

It is critical to consider whether sharp force injuries might have been be self-inflicted, as opposed to being inflicted by an assailant. One should be mindful of the circumstances surrounding the death, but assumptions must not be made on the history alone. Fatal self-inflicted stab wounds are not uncommon, being more frequently encountered in males. The individual is more likely to lift their clothing to inflict the fatal injury, and the weapon may remain in the deceased's body or may be close by. Occasionally, however, the weapon may be tidied away! Some stab wounds can permit a period of purposeful activity, and the individual may even be capable of quite vigorous activity until overwhelmed by blood loss or other tissue damage.

Self-inflicted injuries must be in a location accessible to the deceased's own hand; fatal stab wounds or incised wounds are most frequently encountered in the torso or neck. The fatal injury is often accompanied by tentative or hesitation marks—shallow stab wounds or puncture wounds caused by the tip of a knife, presumably testing out the pain to be expected prior to the fatal blow. Self-inflicted stab wounds usually do not penetrate through thick, bony buttresses such as the sternum, and penetration of such bones should elevate the suspicion of the forensic pathologist. Incised wounds to the upper limb can injure the main vessels (e.g., radial or ulnar arteries), leading to severe and fatal haemorrhage.

A word of caution is necessary at this point regarding post-mortem incised and stab wounds. Although the accompanying injuries may suggest that the wound is self-inflicted, it would never be entirely possible for the forensic pathologist, when considering the pathological findings alone, to entirely exclude third-party intervention with regard to the fatal injury. Indeed, old self-harm scars may not necessarily indicate that a single stab wound to the chest is self-inflicted.

## Blunt Force Trauma and Friction Injury

Blunt force trauma is caused by blunt, non-sharp, broad impacts to the skin tissue. Two characteristic injuries occur: the bruise and the laceration.

The bruise appears as an area of discolouration upon the skin, sometimes accompanied by tissue swelling. The bruise is caused by bleeding from crushed and ruptured vessels in the tissues. Not all bruises are visible from external inspection of the body, and deep body dissections are required to reveal them (see above). The age of bruises can be broadly gauged by colour and their histological appearances. Haemorrhage from basal skull fractures and other deep injuries can emerge upon the skin surface to mimic localized injuries (for example, a "black eye" caused by a basal skull fracture).

Lacerations are skin splits caused by blunt force. They can be rather irregular, with an associated abrasion rim. Invariably, a degree of bruising accompanies lacerations. Friction injuries occur when a rough surface rubs across the skin surface, leading to the development of abrasion. Abrasion typically involves only the epidermis, although bleeding may ensue from the injury if the papillary dermis (or deeper) becomes involved.

### Patterned Bruise

Patterned bruising may be critical in identifying a weapon or offending footwear. The pathologist is required to carefully document and measure the pattern and ensure that it is appropriately recorded photographically, with scale. Patterns may be modified or lost due to the interposition of clothing or the healing process.

### Tramline Bruises

Tramline bruises are characteristic of impacts from cylindrical objects, such as bats and poles. The tramline comprises two outer linear bruises, separated by an area of sparing. As the cylindrical object impacts, the area of most skin deforming occurs at the edges of the impacting object—tearing blood vessels and leading to the development of bruising. Centrally, the skin is depressed inwards rather than deformed, with less vascular injury. Broader weapons cause broader tramlines, although it is important to be cautious about estimating the width of a potential weapon from the width of the tramline bruise alone.

### Laceration

Lacerations occur in areas where the skin is relatively tightly applied to the underlying soft tissues, such as in the scalp. The blunt force applied is sufficient to split the skin and underlying tissues. These lacerations may be difficult to differentiate from incised wounds, but careful inspection of the injury should be able to differentiate between them. To assist in cases with possible laceration or incision to the scalp, the overlying head hair may also be cut (as part of the initial external examination). Linear lacerations may have thin rims of abrasion along the wound margin. In addition, within the depths of the wound there are tissue bridges, rather than the neat division encountered with incised wounds. Bruising and swelling are often more marked with blunt force trauma than with a sharp force wound.

### Bite Marks

Curved bruises and earlobe injuries (particularly if part of the ear is missing) should always raise the consideration that an injury has been caused by teeth (human or other). A forensic odontologist should be consulted for specialist advice and interpretation. Dissection of a bite mark may alter its shape and size, so ideally the odontologist should be asked for advice prior to dissection. Swabbing of the marks for possible DNA is also required, potentially assisting future identification of the perpetrator.

### Grip Marks

Ovoid bruises on the inner aspects of the upper limbs or thighs suggest tight gripping. Such injuries on the upper

arms are often caused during the resuscitation procedure, usually by attempts to move the collapsed individual into a favorable position for chest compressions. Bruising on the thighs—particularly the inner aspects—may indicate antemortem sexual activity.

### Abrasion Injuries

Many of these injuries will be familiar to all, such as common knee abrasions or grazes caused by collapse or falls to the ground. These injuries are often natural, but recording their presence and details is part of the examination.

### Abrasion Scrolls

The direction that the friction force was applied to the skin can be determined by the observation of abrasion scrolls. Scrolls of skin (and sometimes large skin flaps) are raised, with the base of the scroll (or flap) showing the direction of travel.

## Deaths on the Highway

Deaths on the highway should be considered from the perspective of what the person was doing when death occurred. Realities to be considered differ for driver versus pedestrian deaths, cyclist versus motor vehicle, and passenger/driver injuries. Persons injured as pedestrians need a different approach.

For the sake of simplicity, this section assumes the vehicle is a car, although one must accept that different injuries will follow collisions involving motorcycles, lorries, coaches, and other vehicles. In this regard, close interaction with traffic police, forensic collision reconstruction experts, and civil highway engineers may be needed.

### Pedestrian Injuries: Primary, Secondary, and Tertiary Impacts

Impacts with pedestrians can be classified into primary, secondary, and tertiary impacts. The primary impact is the point of first contact of the oncoming vehicle with the pedestrian and should be identified, if possible.

Primary impacts caused by the vehicle bumper on the lower limbs often appear as horizontal bruises, and the distance from the heel should be measured. This information can assist the forensic collision reconstruction experts in their assessment of the case; low bumper marks could indicate braking of the oncoming vehicle. Radiographs or CT scans of fractured lower limbs, assessing the shape of the injury, can also identify the direction in which the collision occurred. Horizontal or wedge-shaped fractures to the tibia are typical of primary impacts, with the base of the wedge to the point being in the same direction as the travel of the vehicle.

Secondary collisions often involve the head striking an upper part of the vehicle, such as the window screen or vehicle frame. The secondary injury is often fatal. Tertiary impacts are caused by contact with the roadway and include large abrasions or fractures. Long bone fractures from these impacts are often spiral in nature, rather than transverse.

### Pedestrian Injuries: Patterned Injuries

Patterned injuries discovered upon the body of a "hit and run" victim can be critical in the identification or exclusion of a suspect vehicle. Grills and other patterned parts of the vehicle may leave imprinted bruises. Careful, scaled photography can assist the investigating officers in comparing the injury to a suspect vehicle. If possible, the injuries can also be traced upon paper, to allow rapid comparison of a suspect vehicle to the injury in the field.

### Vehicle Occupants

Critical to many investigations is identification of the driver of the vehicle at the time of the fatal collision. Often, this is straightforward, with the driver remaining strapped in the driver's seat, but the collision may alter the position of deceased individuals in the vehicle or may project individuals out of the vehicle.

The pathologist can help the investigating officers determine who was driving the vehicle at the time of a fatal collision. Transmitted forces through the foot pedals can leave impressions on footwear, which can be recovered at autopsy for the officers. Examination of the chest may identify specific features of the steering wheel, if it has been in contact with the deceased. One caveat should be considered, however: mechanical resuscitators used by some emergency services can leave rounded or curved bruises upon the chest, which may be mistaken for steering wheel injuries.

The position of a seat belt mark also can assist in determining who was sitting where in the vehicle. Classic seat belt marks or abrasions are uncommonly encountered, as clothing often protects the skin. More commonly encountered are broad but linear abrasions at the base of the neck, caused as the upper part of the seat belt tightens against exposed skin. Horizontal marks across the abdomen, caused by the lap belt, may also indicate the use of a seat belt. These abdominal injuries sometimes are not evident externally, but

subcutaneous dissection can reveal a band of linear laceration in the lower abdominal soft tissues.

### Road Traffic Collision with Natural Disease

For both pedestrian versus vehicle collisions and fatal vehicle collisions, natural disease is often relevant. For example, pedestrians may collapse in front of a vehicle as a result of epilepsy or syncope from coronary artery atheroma. Alternatively, one may find an acute natural pathology affecting the driver of a vehicle. All such disease data must be evaluated alongside the forensic injuries.

Consideration of the senses can be difficult to assess as a pathologist. The best information with regard to an individual's vision or hearing comes from ante-mortem clinical data or statements of witnesses (such as family). It may be appropriate under some circumstances to remove the eyes for formal ophthalmological assessment. Recovery of items such as contact lenses may also help investigators enquire into why a collision occurred.

## Specific Neck Considerations

Although the neck is opened during routine examination, one must be aware of possible pathology that will be missed or lost without attention to specific considerations. Given the airway vulnerability in the neck, special attention must be paid to cases with features of asphyxia, strangulation, hanging, or choking.

### Signs of Asphyxia (Petechiae and Congestion)

Petechiae are small, pin-prick sized haemorrhages visible upon the skin and on the inner aspects of the eyelids, which appear like a fine rash. They are commonly encountered in cases of death by asphyxia. The precise mechanism by which they develop is not known, although they are likely related to vascular overpressure, which may involve aspects of hypoxia on small blood vessels and continued arterial supply of blood to the head during asphyxia, whilst venous return may be impaired. Petechiae are not exclusive to asphyxial deaths. They may be encountered in other circumstances, including natural deaths. Hypostasis may also mimic the development of petechiae in the face, particularly if the deceased assumed a "face down" posture.

Congestion, causing a purple discolouration to the head, often accompanies other asphyxial signs such as petechiae, nose bleeds, and facial oedema. Indeed, congestion is responsible for artefactual haemorrhages in the neck, such as the Prinsloo-Gordon lesion, and decompression of this congestion is required when assessing deaths caused by pressure to the neck.

### Pressure to the Neck: Internal Injuries

Ligatures or hand grips (manual strangulation) are capable of causing fractures to the hyoid bone and larynx. Younger individuals may be less susceptible to these fractures, as these joints and cartilages are often flexible and not ossified. Bruising often accompanies these injuries. Occasionally, the cricoid cartilage may also be fractured. To assist, histological assessment should be made. A lack of an inflammatory infiltrate indicates death soon after the injuries were inflicted (i.e., death was caused by the pressure to the neck), whereas a well-established inflammatory infiltrate may indicate a period of survival, and death by another mechanism.

### Hanging

Hanging is death caused by exerting pressure upon the neck, usually using the weight of the body through a ligature tethered to a suspension point. This is quite unlike judicial hanging, which causes death by cervical spinal column disruption following precipitation of the individual from a height with a ligature about the neck. Self-suspension with a ligature usually causes unconsciousness very rapidly, with death ensuing within a few minutes. Respiratory movements, whilst suspended by the neck, along with decorticate/decerebrate posturing during the hanging process, can occasionally cause additional injuries over the bony prominences of the face. One must be aware of these minor injuries, as they do not necessarily indicate third-party participation or additional suspicious circumstances. For death to occur, full suspension of the body is not necessary; partial suspension with the feet or knees touching the floor can exert enough pressure to bring about death.

Several critical features should be assessed at autopsy. Firstly, the ligature mark must be compared to the ligature that is believed to have been used to cause death. Any mismatch in the patterns must be highlighted to the investigating officers. Secondly, the point of suspension should be sought; it is often characterized by the ligature marks on either side of the neck rising towards a single point. Horizontal or encircling ligature marks are more suspicious of ligature strangulation. Thirdly, other suspicious marks to the neck (or elsewhere) need to be assessed to exclude other modes of pressure to the neck or additional injuries used to subdue a victim.

### Manual Strangulation

Manual strangulation can be defined as pressure to the neck exerted by a third party by use of the hand or hands. Manual strangulation is often characterized by multifocal areas of irregular abrasion and ovoid bruising to the neck—injuries that occur during a dynamic struggle between two individu-

als. Curved abrasions may also be identified, caused by the fingernails either of the perpetrator or of the victim, attempting to remove the gripping hands. Objects such as clothing or necklaces may be pushed into the skin, simulating the application of a ligature. Examination of the internal neck structures often identifies widespread, multifocal bruising to the strap muscles, mirroring the external findings. The larynx, cricoid, and/or hyoid may be fractured, but there may be no injury in younger individuals because the cartilages are flexible and mainly cartilaginous.

## Ligature Strangulation

Ligature strangulation can be defined as pressure to the neck exerted by a third party by use of a ligature. Ligature strangulation is usually characterized by the presence of a ligature mark upon the neck. Such ligature marks may not necessarily be particularly well defined. Some marks are characterized by linear bruises only, and broad ligatures may not leave any ligature mark. Ligature marks caused during ligature strangulation do not have suspension points such as those encountered in hanging, and the marks tend to be horizontal as they cross the sides of the neck. Signs of a struggle may also be present, with fingernail marks about the ligature mark. Documentation of the injuries must capture any patterning within the ligature mark, and it is often of value to measure the circumference of the neck. These records may assist in determining the nature of the ligature.

## Choking

Exposure of the neck by a wide "V" incision (see above) permits careful access to the oropharynx, preventing items from becoming dislodged, which may occur with single neck low incisions that require removal of the tongue from low in the neck. Choking should be suspected particularly in cases where an individual has died suddenly whilst eating a meal, particularly if they have any disorder that may alter the ability to swallow, such as degenerative neurological conditions.

## Traumatic Asphyxia

When the chest becomes compressed and it is no longer possible to ventilate the lungs owing to failure of the chest to expand and contract, the situation is best termed traumatic asphyxia. Traumatic asphyxia can be encountered in a number of situations, including motor vehicle accidents, industrial accidents, and crowd crushes or stampedes. Although the exact mechanisms are debated in the medical literature, the back pressure in the venous system and continued cardiac output may account for the intense congestion of the head, neck, and upper limbs; intense petechiae of the face; and scleral haemorrhage. Examination of the chest can identify accompanying external injuries and rib fractures, although little injury may be present in the young because of relative chest flexibility.

## Postural/Positional Asphyxia

These terms describe asphyxial deaths caused when the body adopts such a posture or is placed in such a position that ventilatory movements become compromised. If the individual cannot escape from the situation, then fatal postural or positional asphyxia may take place. Flexion of the lower limbs and abdomen can displace the abdomen viscera upwards, making downward movement of the chest difficult. This can be further compromised by flexion of the upper chest and neck. Support of the body weight by extension of the upper limbs away from the chest wall, with elevation above the shoulders, can also prevent normal expansion of the chest, leading to positional asphyxia.

Intoxication with alcohol or drugs is a risk factor for postural or positional asphyxia, as the individual may be rendered incapable of escape. Natural disease, particularly if affecting muscle power (such as motor neuron disease, muscular dystrophy, or multiple sclerosis) also can increase the possibility of accidental postural asphyxia, although practically any condition than can cause a person to fall into unconsciousness might lead to death by this mechanism. Diagnosis of fatal postural/positional asphyxia cannot be made without assessing the scene. Post-mortem examination reveals typical features of asphyxia, with congestion and petechiae often extending onto the chest. Hypostatic pallor can also assist in the assessment of the position of the deceased.

## Electrocution

Sudden death in the workplace (or elsewhere, for that matter) without obvious explanation should raise the suspicious of possible electrocution, as the autopsy findings can be minimal. The mechanism of death is usually disturbance of cardiac rhythm, although extensive burns may also sometimes be a feature.

Electrocution burns can be subtle and easily overlooked. They can appear as small blisters or focal areas of carbonization and occasionally can appear similar to abrasions. Histological assessment of electrical burns reveals characteristic features: the epidermis is focally denuded, and nuclear streaming is identified at the edge of the burn. Deep to the

burn site, the dermal collagen is often hypereosinophilic. Embedded metallic particles may also be identified.

## Fire Deaths

Fires may kill from the effects of direct thermal injury, but fatalities more commonly result from smoke inhalation. Fires often occur in accidental settings, although fire investigations (which may take considerable time) may later indicate arson. Fires may also be set in order to hide evidence of a homicide. The autopsy needs to establish whether the individual died prior to the fire or during the fire, and if the person died during the fire, the autopsy must attempt to discover why the individual did not escape from the heat and/or smoke.

### Vital Signs

The presence of soot below the level of the vocal cords indicates active inhalation of smoke. It indicates that the individual was alive whilst the fire was producing smoke. Individuals who are deceased prior to commencement of the production of smoke have airways free of soot, although soot may percolate into the mouth, onto the tongue and pharynx.

External examination may reveal the typical cherry pink discolouration of the skin caused by carboxyhaemoglobin (the combination of carbon monoxide with haemoglobin, which displaces oxygen). The internal tissues and blood also show this similar discolouration.

Carboxyhaemoglobin saturations of 50 % and above may prove fatal in healthy adults. In individuals with other conditions, such as ischaemic heart disease, lower carboxyhaemoglobin saturations may lead to death.

For those surviving a blaze for a period of time, when medical assistance is rendered, fatalities will necessarily have modulation of their injuries. In the case, case note/record assessment will be important, as well as consideration of the physiological effects of the treated injuries (e.g., sepsis on a large zone of burn injury, or respiratory distress syndrome from exposure to smoke or fumes).

### Artefacts of Fire: Heat Haematoma

Some caution is needed in looking at bodies retrieved from fires. Artefacts of heat may mimic intracranial haemorrhage, skin lacerations, or bony fractures. One artefact of concern is the heat hematoma. These are usually associated with carbonization and loss of the scalp over the head. The heated blood from the venous sinuses of the dura leaks into the extradural space, where it coagulates and occasionally may form a substantial hematoma. These are not to be confused with true extradural hematomas, which are invariably caused by skull fractures.

### Artefacts of Fire: Heat Lacerations

Heated skin, particularly when carbonized, can shrivel and split, thereby mimicking injury. Under most circumstances, these can be distinguished from true injuries by a lack of haemorrhage into the underlying subcutaneous fatty tissues.

## Post-mortem Phenomena on the Boundary of Natural and Non-natural Death

A complicated group of post-mortem changes confuse many pathologists. One needs to be aware of these issues, first to identify when a non-natural death has occurred and second, to avoid mistakenly raising an alarm over a case.

### Post-mortem Injuries

Post-mortem injuries may be sustained when the body is moved to the mortuary from the scene of the death. Many are unintentional. These injuries may be inflicted by the pathologist or technician during the autopsy. Resuscitation also may be a cause of peri-mortem injury.

Rarely, an assailant may inflict additional injury on a body as part of the assault/mutilation after death has occurred. In general terms, post-mortem injuries show no vital reaction, no bleeding, or no swelling. Decomposition increases the risk of accidental skin tears, but medical conditions (e.g., steroid medication causing thin skin) also may increase the risk of injury.

### Hypostasis

Hypostasis (or post-mortem lividity) is the discolouration of the skin caused by gravitational pooling of blood after death. Relatives who view the body after death can sometimes confuse hypostasis with true injury—a question sometimes raised at inquest.

After several hours, hypostasis becomes fixed or partially fixed within the tissues. Thus, it can be used to determine whether a body has been moved from one position to another after death. Hypostasis also can be identified within the internal organs and must be distinguished from naturally occurring disease processes. In the heart, posterior hypostasis can even be mistaken for myocardial infarction.

### Skin Slippage and Depigmentation

Skin slippage and depigmentation is a particular problem often encountered in bodies recovered from water, when the identity is not known. Of course it is useful in identifying an individual to know the person's ethnic origin and skin colour, but maceration and slippage of the skin due to decomposition can lead to loss of the pigmented layer, exposing the underlying dermis and making identification of the deceased more difficult.

### Post-mortem Animal Predation

Individuals may die in a sealed environment with their pets. The pets, when starving, will resort to feeding on the corpse. The post-mortem injuries can be widespread and may mimic true injury. Rodents, other vermin, insects, and crustaceans also can inflict a variety of post-mortem injuries if they have access to the body.

## Hypothermia

Hypothermia is typically associated with exposure to very low ambient temperature, although it can be encountered in environments where the temperature may be as high as 12 °C. Disease processes or intoxicants that interfere with homeothermic mechanisms (e.g., hypothyroidism or acute alcohol intoxication) can increase the risk of developing hypothermia.

The diagnosis of hypothermia requires careful consideration of the circumstances of death. Of course, the individual needs to be in a cold environment. Although not fully understood, unusual behaviours can develop in the hypothermic individual. Paradoxically, the affected individuals before death may remove items of clothing, and the individual may be discovered seminaked or naked. This situation can be of concern to investigating officers, particularly with females, as it raises the possibility of sexual assault.

Confusion often also ensues and a scene may show signs of great disturbance or chaos, again mimicking the scene of an assault. The hypothermic individual also may be found in an enclosed space (sometimes referred to as the "hide and die" syndrome or "terminal burrowing". Presumably this behaviour is an attempt to escape from the cold.

Hypothermia may produce reddish discolouration over the main joints and sometimes over the nose and cheeks. When exposure to cold has been prolonged, the extremities occasionally may show blistering or tissue necrosis (frost nip and frost bite).

Multiple gastric erosions (Wishnewski's ulcers) are also characteristic of hypothermia deaths. Microscopically, the ulcers usually involve only the mucosa. A heavy polymorph infiltrate is also characteristic of these ulcers. Pancreatitis sometimes is also encountered.

## Drowning

The post-mortem features indicating that death was caused by drowning are often minimal. Froth (caused by the mixture of water, mucus, and surfactant) may be present in the airways. In addition, silt or other material may also become deeply inhaled or swallowed. Occasionally, large quantities of extruded foam may be seen at the nose and nostrils—sometimes referred to as a "foam cone" or "plume de champignons".

The lungs typically are hyperinflated and feel crepitant. Other features, such as middle ear haemorrhage and maceration of the limbs, are nonspecific and only indicate immersion in water, rather than specifically death by drowning.

Microscopic inspection of the lungs may show emphysema aquosum. This irregular expansion of the alveolar spaces is not specific for drowning. Specialist tests, such as assessment of bone marrow, liver, and splenic diatoms can assist in the diagnosis of drowning. The pathologist should also consider why an individual has drowned. Why were they incapable of saving themselves? The answer to these questions may lie in an assessment of the circumstances (such as fast-flowing rivers and strong currents), although it also is paramount to assess natural disease and toxicology in such cases.

## Firearms

Firearms injuries can generally be categorized into those caused by rifled weapons and those caused by shotguns firing pellet cartridges. Rifled weapons fire bullets, and the rifling of the spiral-grooved gun barrel causes the bullet to spin and remain in stable fight. Shotguns usually fire a bolus of small projectiles (shot), housed in a cartridge. Both the pellets, cartridge, propellant, and expelled gases are capable of causing injuries from shotgun discharges.

Standard assessment of firearm injuries includes description of the number of missiles fired, direction of the shot, internal injury, and range of fire. Prior to post-mortem assessment, full body radiographs should be undertaken to identify the number of retained missiles. A ballistics expert should also be consulted prior to the external examination, so that the correct samples can be retrieved for later forensic assessment in the laboratory.

If the gunshot wound is believed to have been self-inflicted, it is important to assess the hands for minor injury or soot staining, which may be caused when the weapon discharges. Swabbing for gunshot residue using specially

designed kits (to reduced artefactual contamination) may be important to perform on such cases. Measurements of the limbs should also be recorded, to ensure that the individual is capable of pulling the trigger of a weapon.

## Rifled Weapon: Entry Wounds

A typical entry wound comprises a central defect surrounded by a rim of abrasion, which is caused as the bullet passes through the epidermis. At the abrasion rim, oils from the missile may be wiped off, which may be of forensic significance. The shape of the entry hole and width of the surrounding abrasion rim should be carefully noted, as this can indicate the direction that the missile passed into the skin. A wider abrasion rim on one side of an oval gunshot entry, with under-shelving of the wound opposite, indicates that the strike was angled towards the skin towards the abraded end.

## Rifled Weapon: Exit Wounds

Exit wounds are often rather ragged and are sometimes accompanied by additional lacerations caused by bone being driven into the skin. A typical exit wound has no abrasion rim. If the skin is resting against a firm object when the missile exits, however, the edges of the wound can become crushed, leading to the formation of abrasion. This shored-up exit wound, because of the abrasion, can sometimes be confused with an entry wound.

## Bone Injury

When it is unclear from the external examination of the skin whether a gunshot wound is an entry or an exit wound, bone injury may be able to assist. Skull bone comprises an inner and outer table. A smaller hole is formed in the table that the missile first penetrates, and a larger hole is formed in the second table passed through. Thus, with an entry wound, the inner table has a larger defect than the outer table, and with an exit wound the reverse is true: the outer table has the larger defect. If multiple entry wounds are present in the skull, causing radiating skull fractures, it is possible to determine the order that each missile was fired using Puppe's rule—wherein a new fracture will terminate when it comes to a pre-existing fracture. This means that the pattern of interlocking fractures may be seen as a sequence of successive injuries, allowing the first and later events to be defined.

## Shotgun Entry Wound

Contact shotgun wounds can be devastating, and it may be difficult to identify the precise location of discharge. Soot staining can assist in determining the location of the weapon discharge, and careful reconstruction of the edges of lacerations can re-form the entry hole. Contact wounds may also show a distinct muzzle mark: as the gases from the discharge enter the skin, the skin is forced back against the metal of the muzzle to form a well-defined rim of abrasion. Double-barreled shotguns can leave two muzzle marks, one with a ragged central hole from the discharge.

Upon exiting the barrel of a shotgun, the pellet mass begins to spread out. With increasing range, the shape of the shotgun entry wounds changes. Scalloping of the wounds edges and separate entry holes caused by individual pellets are observed with increasing range. The presence of any of these features must raise the suspicion that the discharge was made by a third party, rather than being a self-inflicted wound.

# Blunt Force Head Injury

Blunt force head injury is a very common mode of death in forensic practice. Detailed external examination of the face and scalp can assist in determining the implement used to cause injury. Indeed, the head hair should be shaved off to reveal subtle injuries, so that they may be documented photographically. The under surface of the scalp and face should be inspected for deep bruising not otherwise visible externally. The calvarium should be inspected for fractures. Removal of the temporalis muscles and aponeurosis (which can be rubbed off with a blade or abrasive pad) should be undertaken to expose subtle hairline fractures. Similarly, it also may be necessary to strip muscle from the bones of the face to reveal fractures.

The calvarium should be opened carefully, so as not to damage the underlying dura mater. The cranial contents can then be inspected for extradural haemorrhage, before the dura is incised. Observation of the subdural space can then be made for bleeding. At this stage, the front lobes can be gently lifted in order to visualize the base of the brain, enabling assessment for basal subarachnoid haemorrhage. (If present, a different path to the investigation should be instigated; see Chapter 9.) Once satisfied, the brain can be removed in the standard way. The dura at the base of the brain can then be stripped to expose the base of the skull, permitting inspection of the basal skull bone.

## Extradural Haematoma

Extradural haemorrhage is accumulation of blood between the undersurface of the inner cranial bone and the external surface of the dura. Invariably it is associated with skull fracture, as the fracture lines damage underlying calvarial arteries. The middle meningeal arteries are particularly vulnerable, as they are covered by the relatively thin squamous part of the temporal bone. As with subdural haemorrhage, individuals with extradural haemorrhage may be characterized by a variable period of lucidity before later collapse.

## Subdural Hematoma

Subdural hematomas accumulate beneath the dura mater, above the surface of the brain. When force is transmitted through the cranial cavity, the brain tissue can deform to an extent. The bridging veins, which are tethered to the dura mater, stretch as the brain tissue deforms, and can then shear. The sheared vessels result in bleeding, causing subdural hematoma formation. In individuals with cerebral atrophy, the bridging veins become slightly stretched and are more vulnerable to shearing, so less force may be necessary to cause subdural bleeding. Subdural hematomas are almost invariably caused by trauma. Very rarely, ruptured cerebral aneurysms can force jets of haemorrhage through the brain tissue to erupt into the subdural space, mimicking injury. Similarly, other natural intracranial haemorrhages can enter the subdural space, but they are usually readily distinguished from true trauma.

## Cerebral Contusions, Coup and Contre-Coup

Cerebral contusions, bruises to the brain surface, are often accompanied by superficial laceration. Direct blows with a blunt weapon may cause contusion to the brain directly beneath the impact site—a "coup" lesion contusion. Conversely, falls onto a hard, unyielding surface, such as the pavement or tarmac, cause contusion on the opposite side of the brain skull. Typically, unprotected falls onto the back of the head cause abrasion about the occiput with skull fracturing radiating forwards towards the foramen magnum. Subdural haemorrhage is invariably present, but the characteristic finding is of contre-coup contusions over the poles of the frontal and temporal lobes and contusion in the region of the olfactory bulbs. Another common finding that accompanies contre-coup head injuries is fractures to the supraorbital plates. The precise mechanism by which the supraorbital fractures develop is poorly understood, although radiated force or changes in the intracranial pressure may be responsible. They are of forensic significance, and bleeding from these fractures may spread to the periorbital tissues, mimicking a "black eye" caused by a blow such as a punch.

## Traumatic Basal Subarachnoid Haemorrhage

When there is a forceful angular rotation of the skull upon the spinal column, the vertebral arteries may traumatically rupture, causing fatal basal traumatic subarachnoid haemorrhage. The rupture may be caused by direct shearing forces placed on the arteries at the junction, although a water hammer effect may also cause rupturing. Typically, deep dissection of the facial tissues reveals injury in the region on one of the angles of the jaw. Dissection of the extracranial vertebral arteries is detailed above, and the intracranial portion of the vertebral arteries should also be carefully examined. Removal of the cerebral hemispheres above the tentorium gives much easier access to the intracranial portions and basilar artery, facilitating easy removal. In addition, sampling of other arteries elsewhere in the body is valuable, to exclude a vasculopathy. Histological assessment of the entire vertebral arteries should be made to exclude a natural cause for the haemorrhage. Trauma is characterized histologically by areas of nuclear lysis, focal dissection, and fibrin plugs.

## Late Complications of Head Injury

Late complications of head injury can lead to death a considerable time after the injuries were originally sustained. Complications include posttraumatic epilepsy, bacterial meningitis, and bronchopneumonia from reduced mobility. In every case where epilepsy is believed to be the cause of death, the medical records should be carefully inspected—even traced back for years—to ensure that head injury was not the cause of the seizure activity.

Bacterial meningitis can complicate head injuries in which skull fractures traverse the airway, middle ears, or frontal sinuses. This complication of head injury usually occurs relatively early (days or weeks) following infliction of the head injury. Bronchopneumonia can be a terminal feature of head injury that can develop many months or years after the incident. In order to consider whether the pneumonia was caused by the head injury, a clinical review must be made to ensure that the head injury was the cause of immobility or gastric content aspiration, rather than another disease process acting independently.

## Skull Fractures

Broadly, skull fractures can be classed as linear (simple or complex) or depressed. Higher-energy impacts can cause severe basal skull fractures, such as the "hinge" fracture or the "ring" fracture. Linear injuries can be encountered from practically any form of blunt force trauma applied to the skull, but depressed skull fractures always raise the possibility that a blunt weapon has been used, and caution should be employed upon their discovery.

Skull fractures also may be hidden beneath the dura mater, so this should be carefully stripped away from the calvarium to expose the underling bone. Occasionally fractures may also develop away from the impact site, commonly within the thin supraorbital plates. The precise mechanism is not fully understood, but this phenomenon is likely the result of transmitted forces or rapid changes in intracranial pressure caused by the initial impact. The form and length of skull fractures should be documented. Where possible, it is also of value to document the thickness of the skull in the region of the fracture. Puppe's rule may be relevant in these cases.

Figures 12.1, 12.2, 12.3, 12.4, 12.5, 12.6, 12.7, 12.8, 12.9, 12.10, 12.11, 12.12, 12.13, 12.14, 12.15, 12.16, 12.17, 12.18, 12.19, 12.20, 12.21, 12.22, 12.23, 12.24, 12.25, 12.26, 12.27, 12.28, 12.29, 12.30, 12.31, 12.32, 12.33, 12.34, 12.35, 12.36, 12.37, 12.38, 12.39, 12.40, 12.41, 12.42, 12.43, 12.44, 12.45, 12.46, 12.47, 12.48, 12.49, 12.50, 12.51, 12.52, 12.53, 12.54, 12.55, 12.56, 12.57, 12.58, 12.59, 12.60, 12.61, 12.62, 12.63, 12.64, and 12.65 illustrate the points made in this chapter.

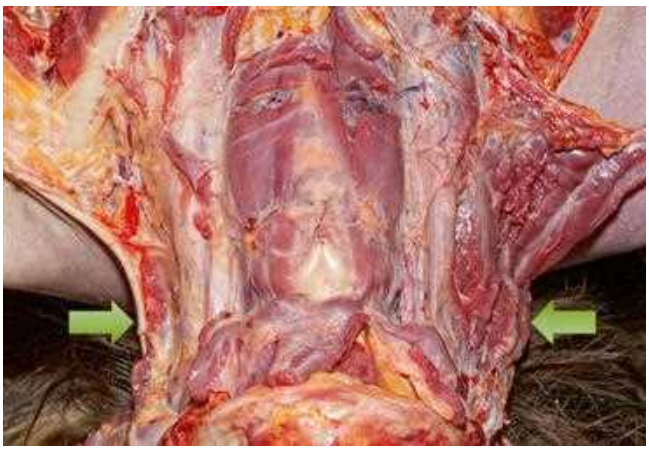

**Fig. 12.1** The neck is shown exposed. Each individual muscle can be stripped back and inspected both externally and internally. The scalenes (*arrows*) can also be dissected

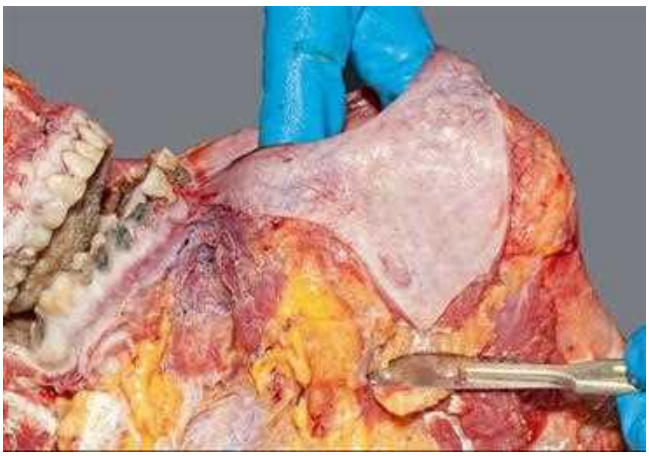

**Fig. 12.3** The lips are carefully incised to expose subtle deep bruising. This is a case of smothering

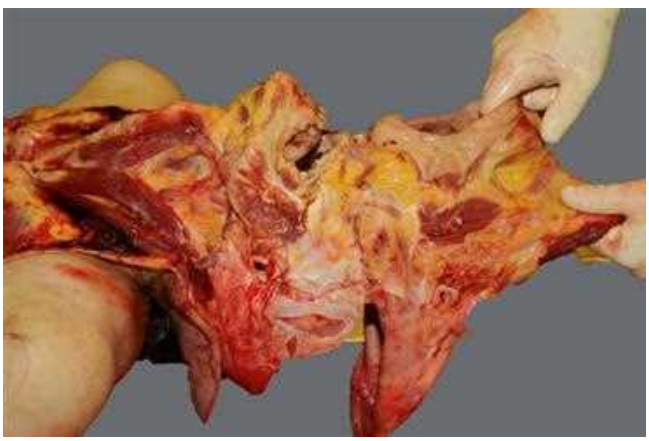

**Fig. 12.2** The exposed facial tissues can be readily inspected for deep bruising and fractures

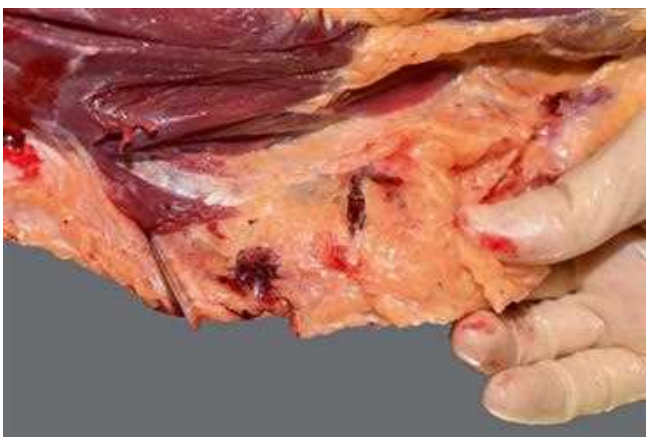

**Fig. 12.4** The superficial fatty and deep subcutaneous tissues are carefully examined for hidden bruises

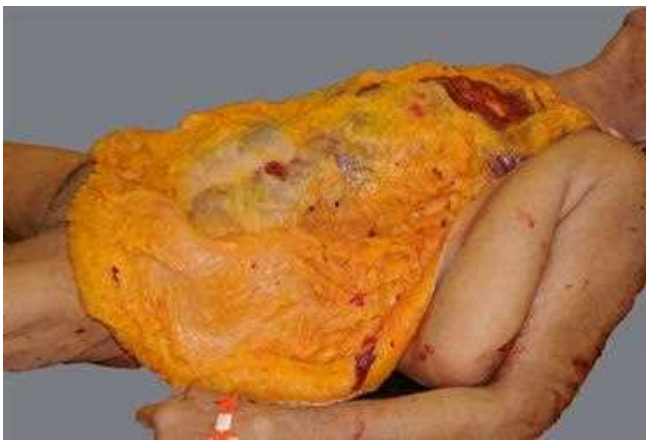

**Fig. 12.5** The abdominal subcutis has been extensively dissected to look for deep bruising

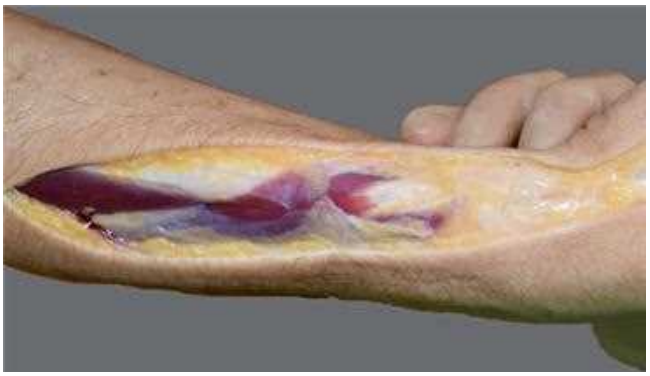

**Fig. 12.6** Subcutaneous forearm dissection. Stage 1

**Fig. 12.7** Subcutaneous forearm dissection. Stage 2

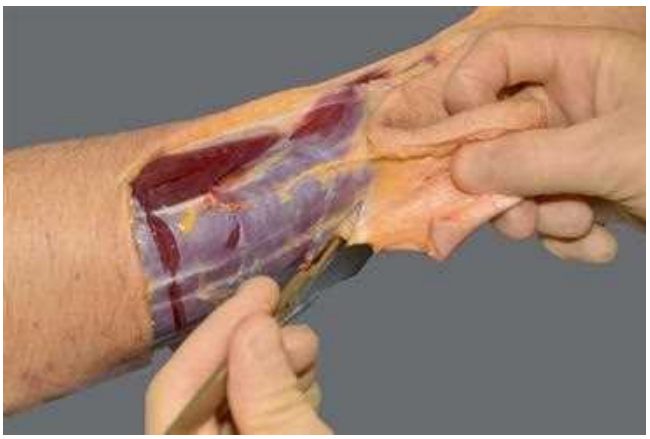

**Fig. 12.8** Subcutaneous forearm dissection. Stage 3

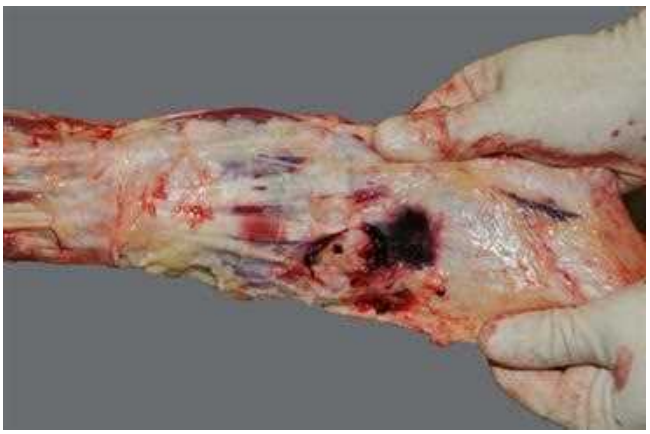

**Fig. 12.9** Subcutaneous forearm dissection. Stage 4. The superficial incisions form the flap, which is dissected distally

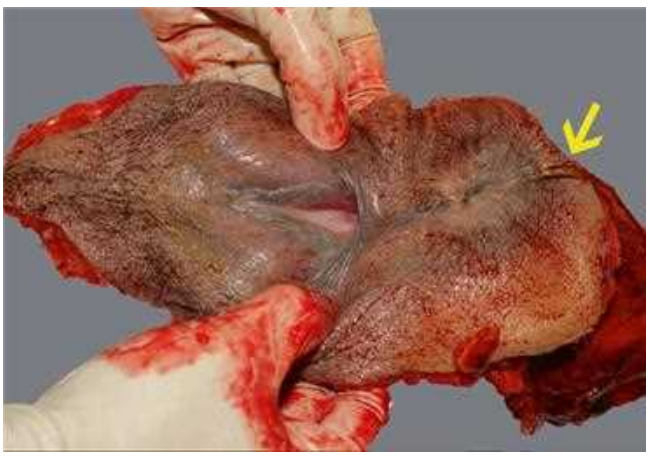

**Fig. 12.10** Ano-genital block shown fully excised, permitting ready assessment and access for imaging. A marker is placed (*arrow*)

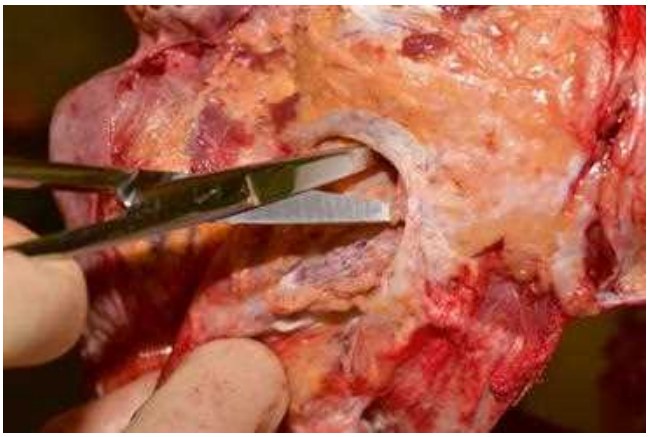

**Fig. 12.11** Orbital dissection (1). Blunt dissection of the lateral wall tissues of the orbit is made

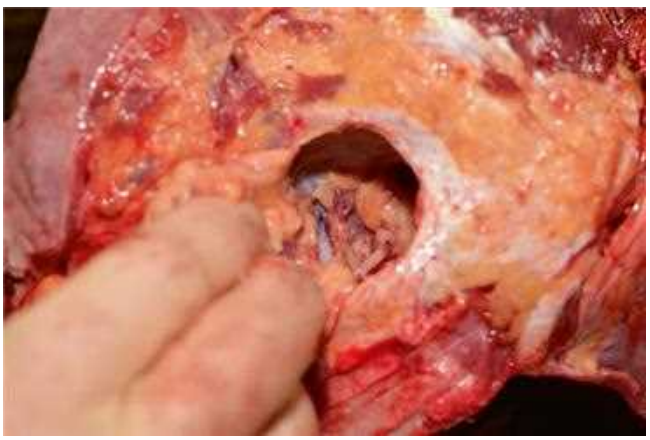

**Fig. 12.12** Orbital dissection (2). The eye is removed after the optic nerve is cut

**Fig. 12.13** Deroofing the vertebral canal with bone clippers will expose the vertebral canal and artery

**Fig. 12.14** Excarnation or defleshing. Partial defleshing is done using dissection initially

**Fig. 12.15** The partially defleshed bones are placed in the bath with biological washing powder at about 37 °C for a week

**Fig. 12.16** The tissue had loosened enough for simple removal with an abrasive pad

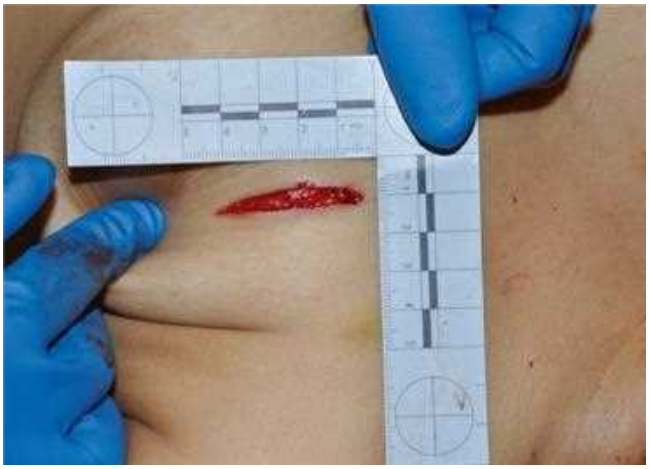

**Fig. 12.17** Stab wound. Note the slight tension applied to reflect the true length of the injury. The shape of this injury indicates the passage of a single-edged weapon

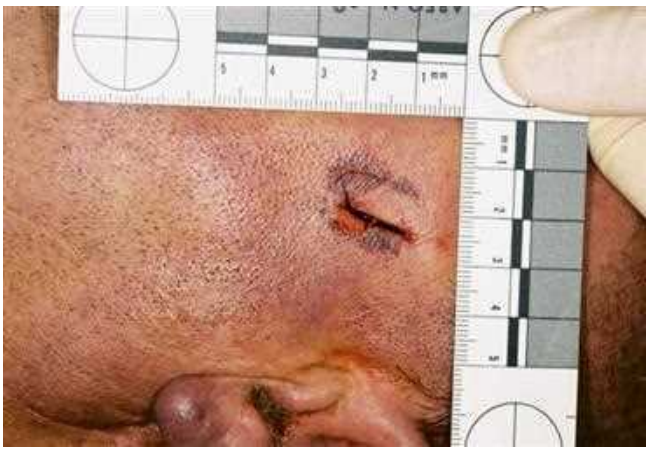

**Fig. 12.18** This stab wound penetrated the calvarium, indicating the use of severe force. The curved injury is the assailant's fingernail penetrating the skin, also implying that severe force was used

**Fig. 12.19** Injuries to the palm caused by grasping a knife—defence injuries

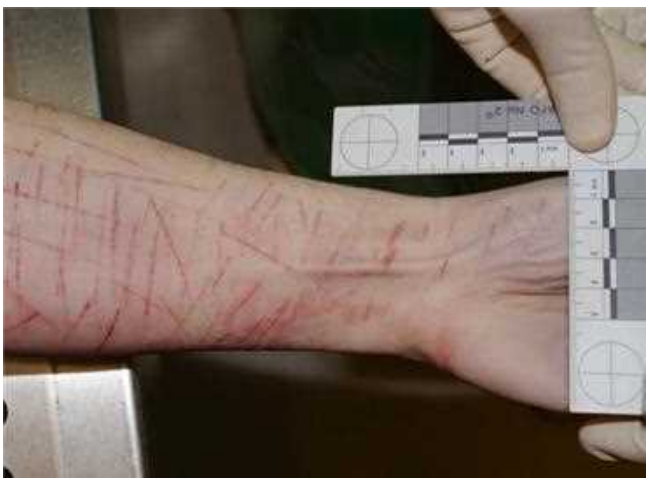

**Fig. 12.20** Parallel, superficial, and horizontal linear incised wounds, typical of self-inflicted wounds

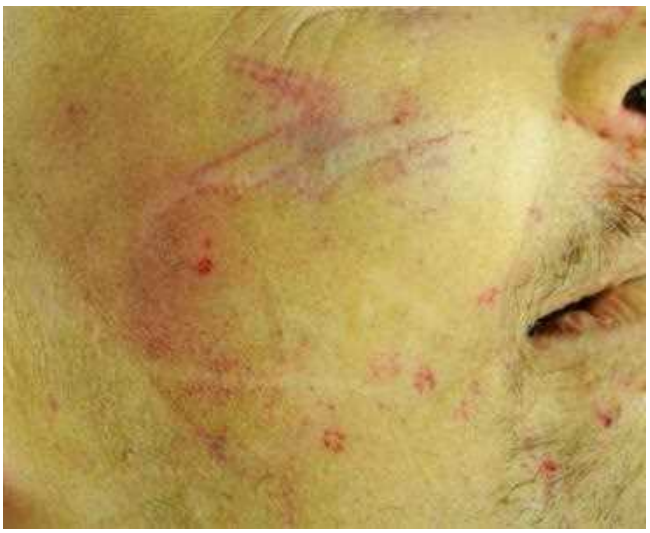

**Fig. 12.21** Footwear mark on the face, with red intradermal bruising

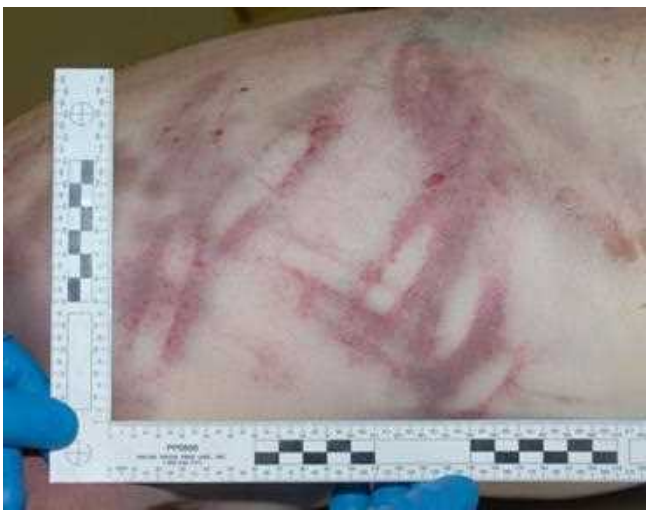

**Fig. 12.22** Tramline bruise caused by repeated strikes from a baseball bat

**Fig. 12.23** Linear laceration mimicking an incised wound. Tissue bridges are evident deep within the injury, indicating the use of blunt force

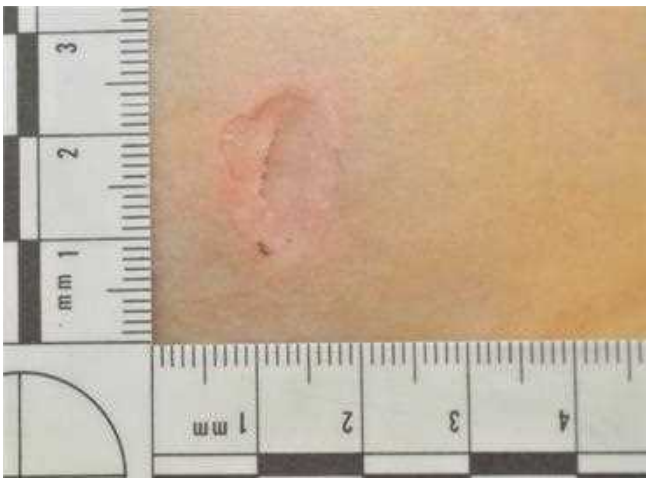

**Fig. 12.24** Depressed skull fracture underneath a curved laceration: a typical hammer blow

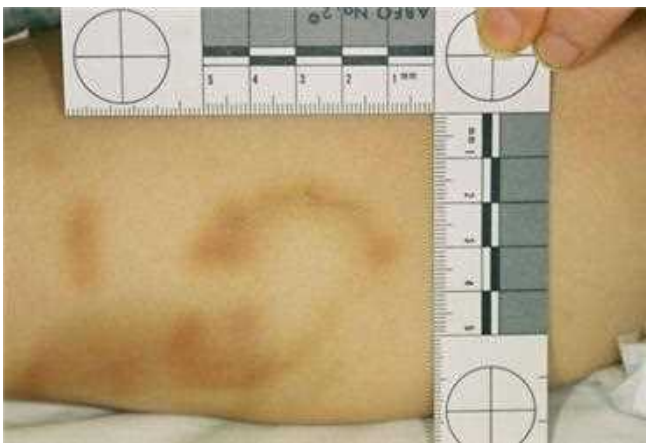

**Fig. 12.25** This is a curve of bruising. An odontologist confirmed that this was a bite mark caused by an adult

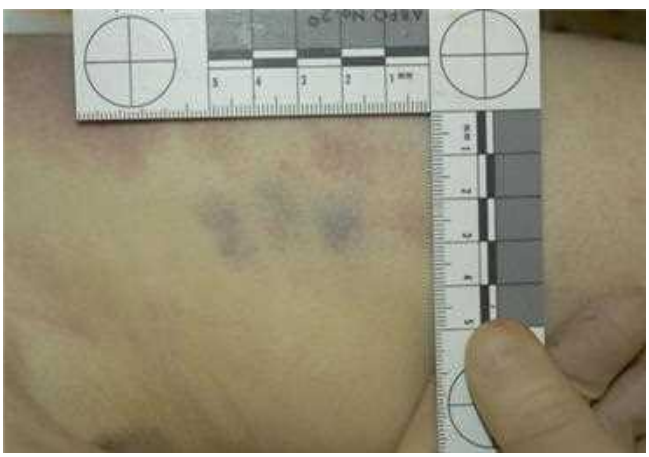

**Fig. 12.26** Grip mark on the inner thigh in a suspicious death

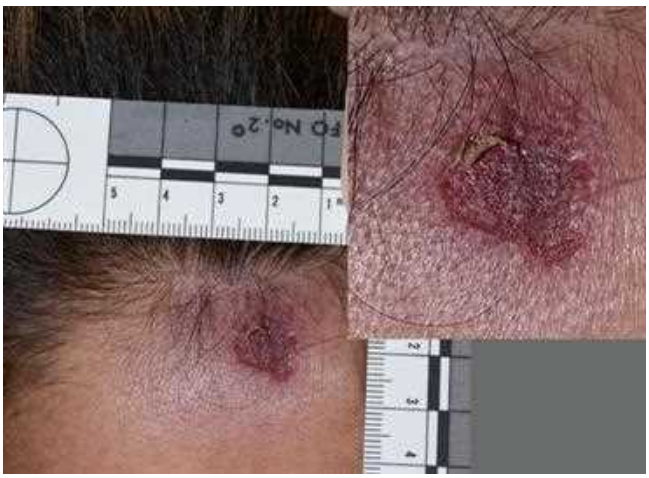

**Fig. 12.27** An abrasion scroll seen at the upper edge of the injury. The individual was dragged by the heels. High magnification (*inset*) shows the scroll towards the edge

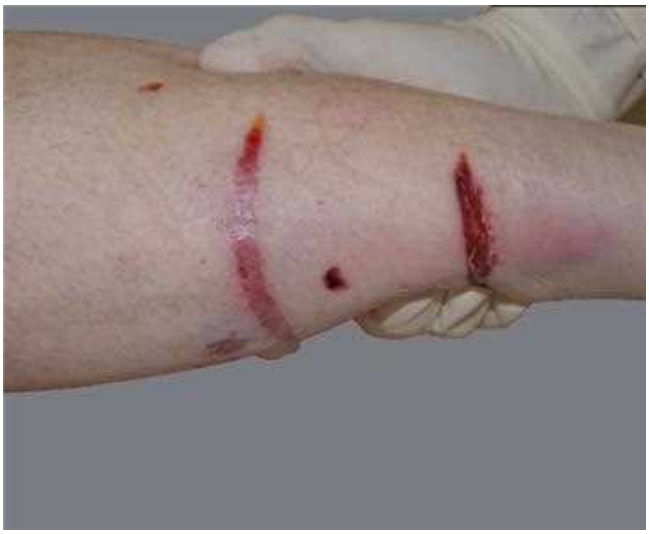

**Fig. 12.28** Primary impact of a vehicle striking a pedestrian: linear bumper strike injuries

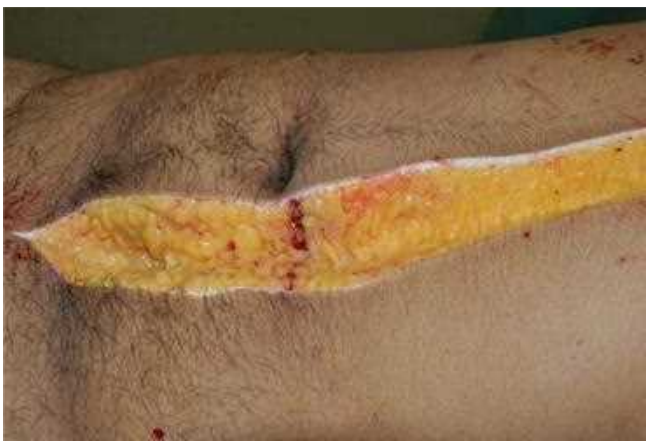

**Fig. 12.29** Horizontal panniculus injury caused by a seatbelt

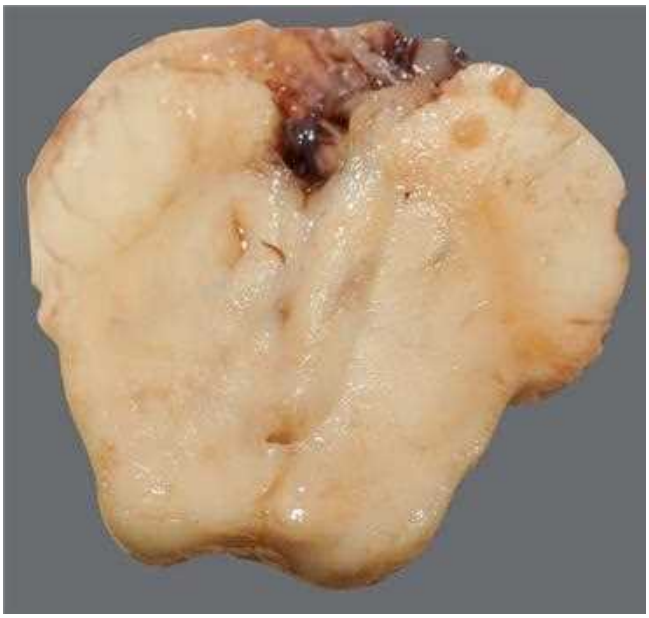

**Fig. 12.30** Pale substantia nigra in a case of Parkinson's disease. The deceased had a festinant gait, which caused this pedestrian to walk unexpectedly into the roadway

**Fig. 12.31** Removal of the contact lenses for expert assessment. This individual crashed his car shortly after visiting the optician

**Fig. 12.32** Conjunctival petechiae in a case of strangulation

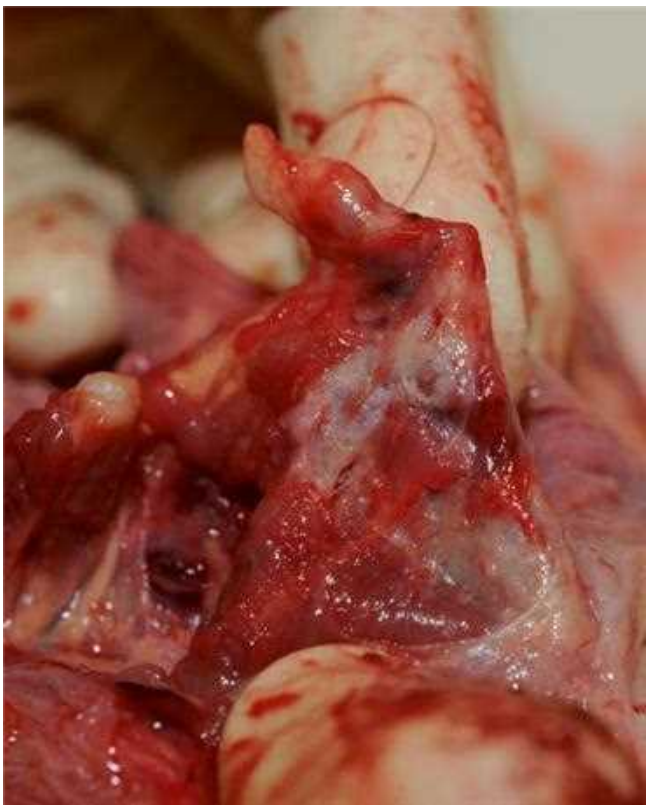

**Fig. 12.33** Fracture to the larynx (laryngeal horn) in a case of manual strangulation

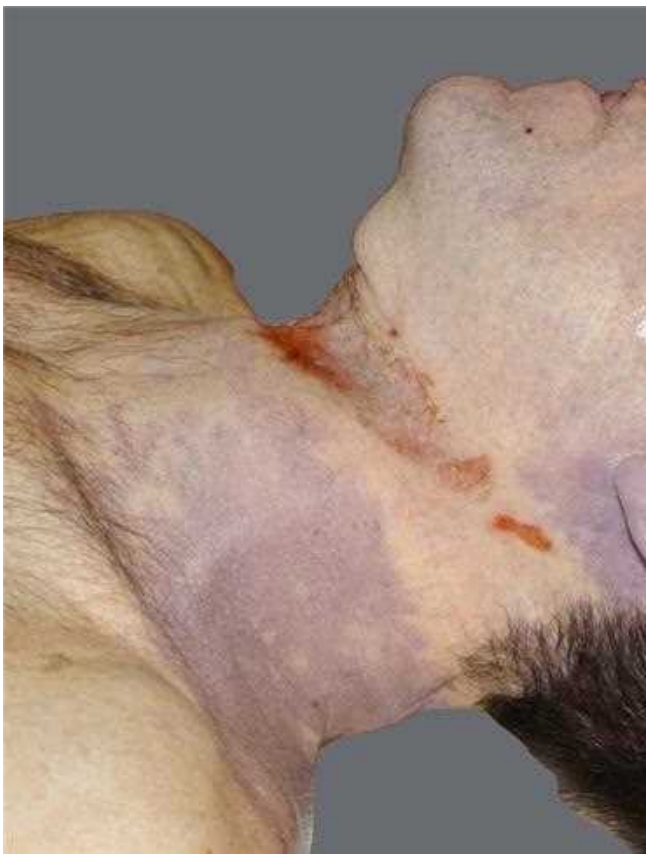

**Fig. 12.34** Typical ligature mark with suspension point in a hanging death

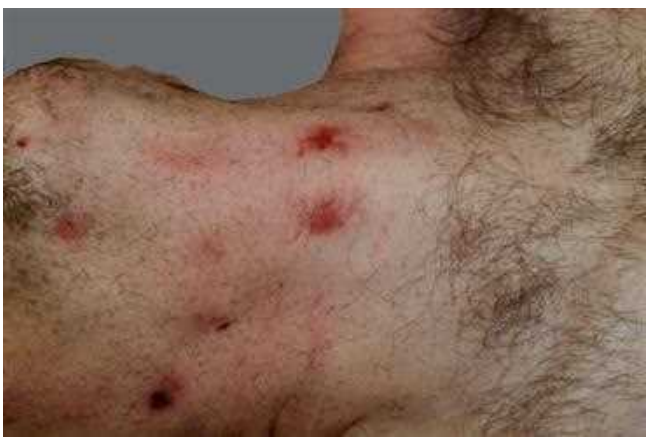

**Fig. 12.35** Manual strangulation: multiple foci of bruising and abrasion over the neck, with fingernail marks

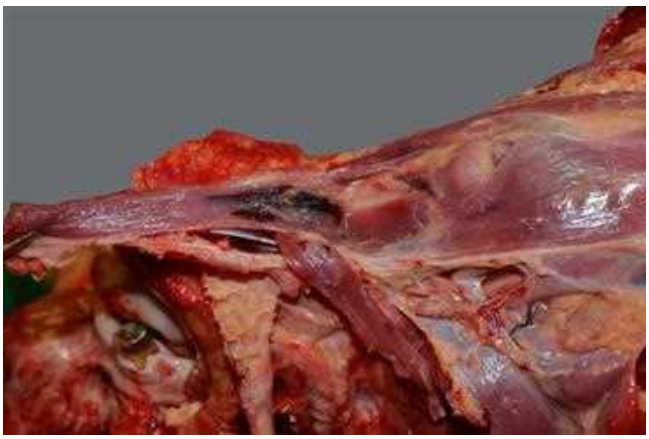

**Fig. 12.36** Manual strangulation: multiple foci of bruising in the deep soft tissues and muscle

**Fig. 12.39** Scleral haemorrhage, florid petechial haemorrhages, and intense facial congestion in a case of traumatic asphyxia

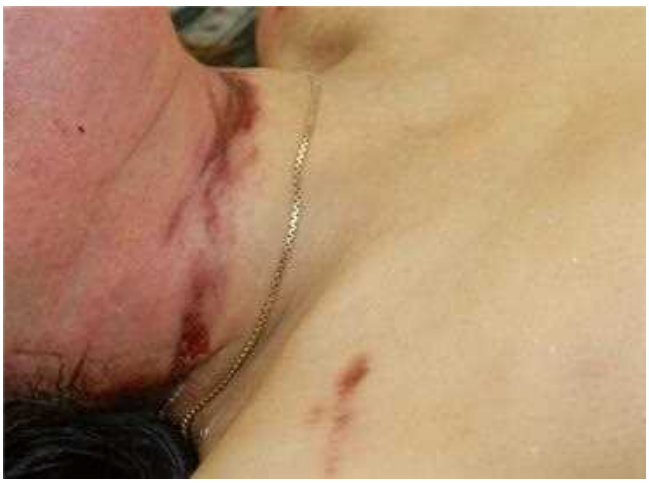

**Fig. 12.37** Ligature strangulation of the neck. Note the congestion rising above the ligature mark

**Fig. 12.40** Postural asphyxia. This heavily intoxicated man became trapped between the wall and a bed, resulting in airway occlusion and respiratory muscle inefficiency (*Note*: The bed has been pulled to the side to assist photography)

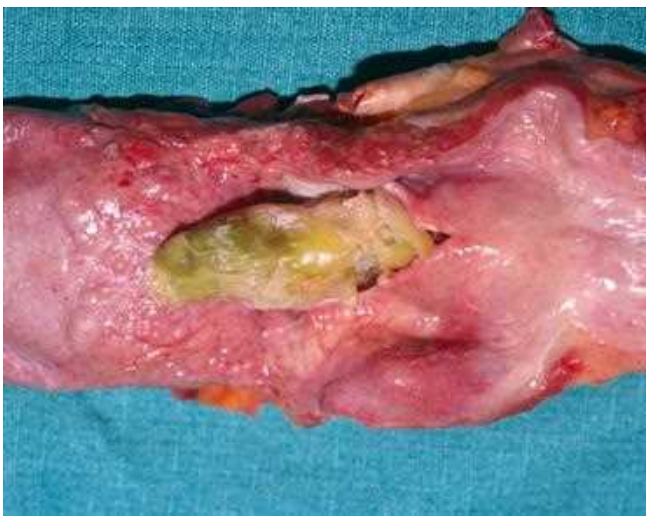

**Fig. 12.38** Choking in an elderly individual who had swallowing difficulties due to cerebrovascular accident. A bolus of food is lodged in the laryngeal inlet, blocking the airway

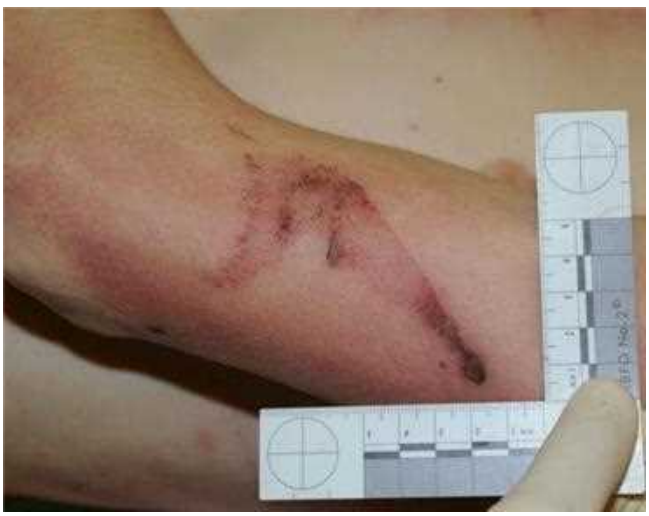

**Fig. 12.41** An electric burn on the skin mimicking an abrasion. Close inspection revealed carbonization

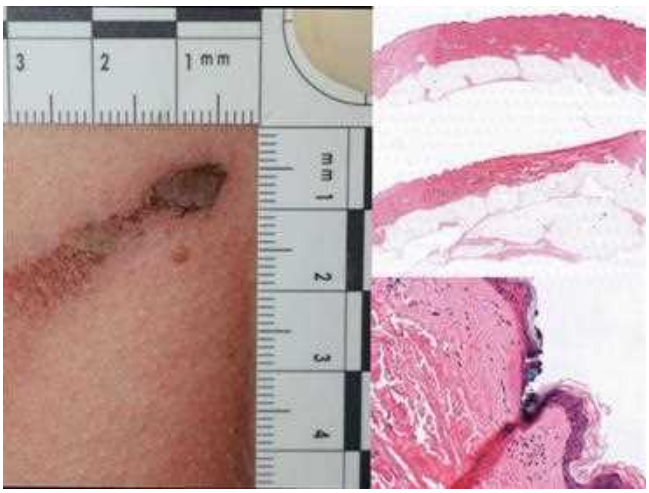

**Fig. 12.42** Electrical burn macroscopy with histology. Typically, the nuclei at the edges of the lesion show streaming artefact. The dermis is hypereosinophilic. Embedded copper from the electric cabling with which the deceased came into contact is seen in this example

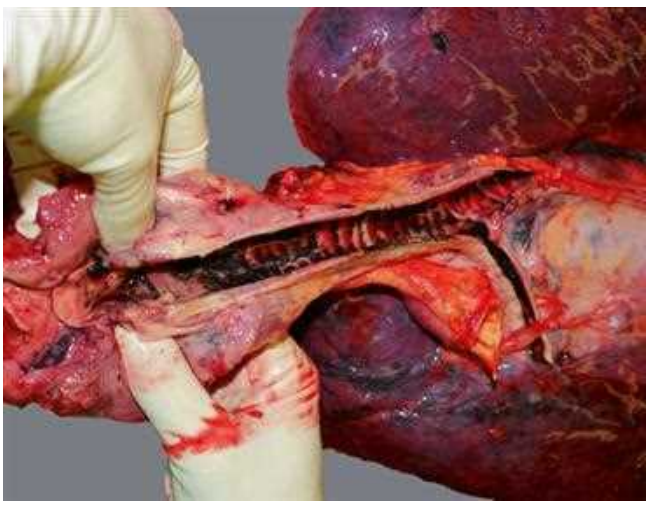

**Fig. 12.43** Soot below the level of the vocal cords, in the trachea, indicating active inhalation of smoke

**Fig. 12.44** Heat hematoma: the overlying skull and scalp are carbonized. The skull often shows superficial fracturing above (a patina effect)

**Fig. 12.45** Heat skin splits. These splits can be confused with true laceration, but the underlying fat shows no haemorrhage

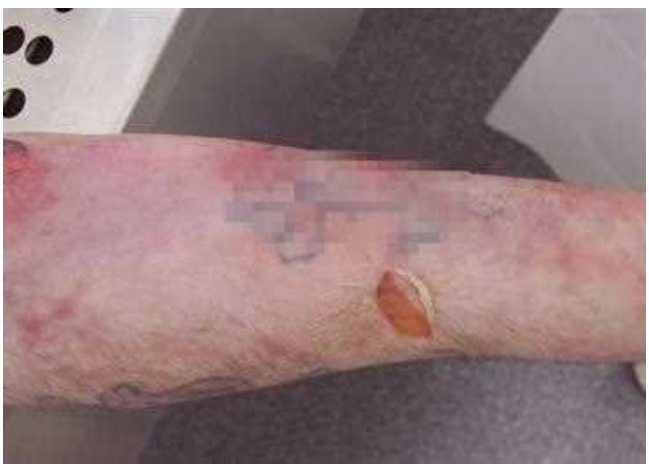

**Fig. 12.46** Post-mortem skin laceration without any associated bleeding or tissue haemorrhage

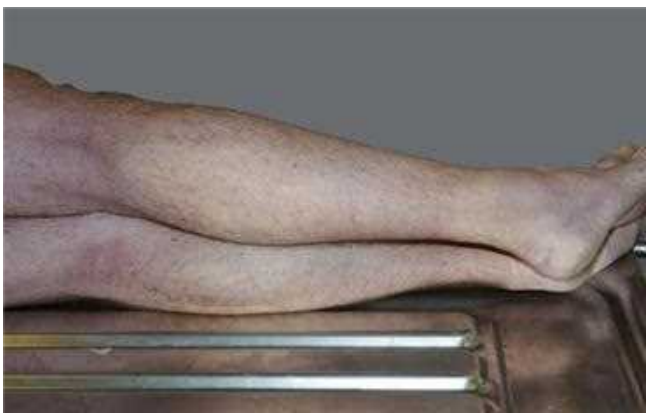

**Fig. 12.47** Hypostasis in the soles of the feet in a case of partial suspension hanging. Hypostasis has also moved to the front of the shin, sparing the calves, reflecting the deceased's position after death

**Fig. 12.48** Loss of the epidermis in a dark-skinned individual, exposing the underlying white dermis. When the loss is extensive, the ethnicity of the individual can be misinterpreted

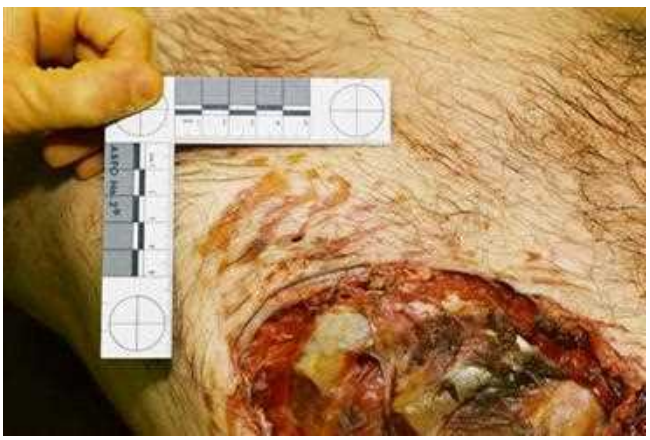

**Fig. 12.49** Post-mortem decapitation by a pet dog left for approximately 10 days, locked in a residence without food

**Fig. 12.50** Hypothermic death. The previous evening, this individual was intoxicated and never arrived home. Clothing is partially removed, and he has tried to enter a bush. The blood alcohol concentration was more than 280 mg/dL

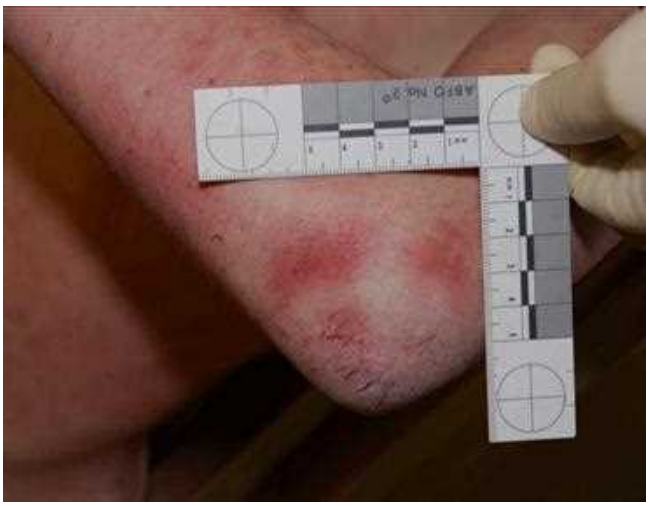

**Fig. 12.51** Reddish discolouration of the joint in an individual who died of hypothermia

**Fig. 12.52** Gastric ulcerations in a case of hypothermia

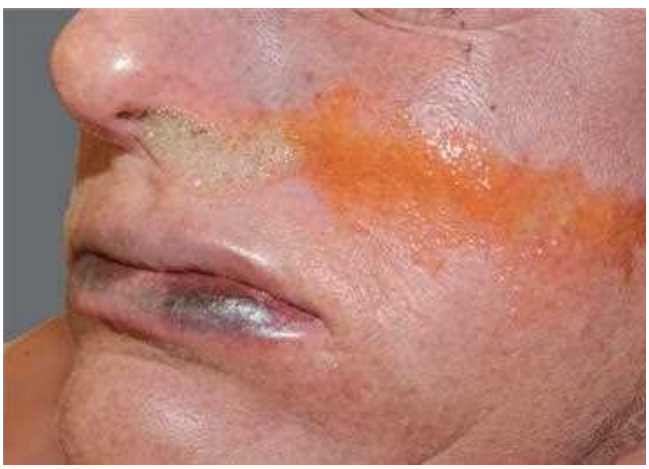

**Fig. 12.53** Foam exuding from the nostrils of a drowned individual

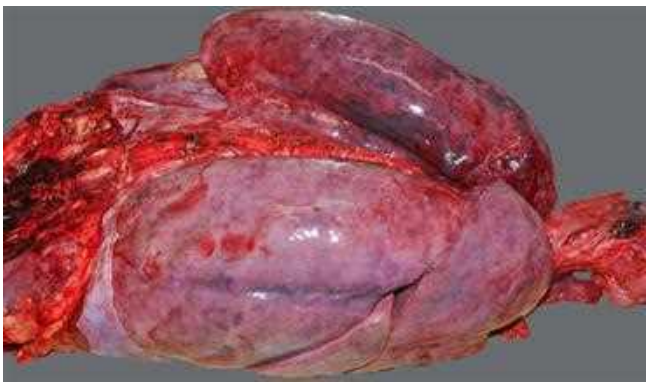

**Fig. 12.54** Hyperinflated lungs in a case of drowning

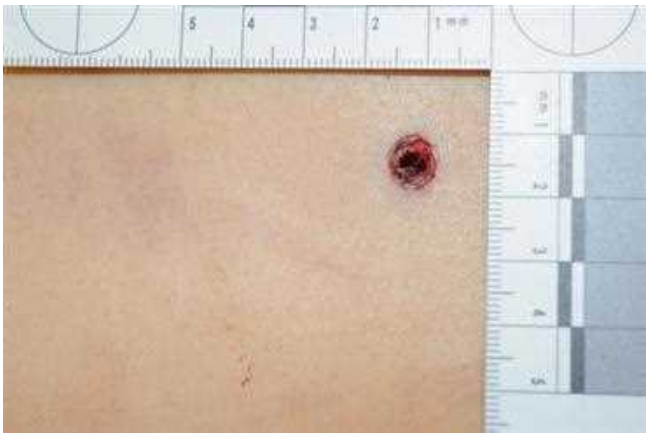

**Fig. 12.55** Gunshot entry wound with typical abrasion rim

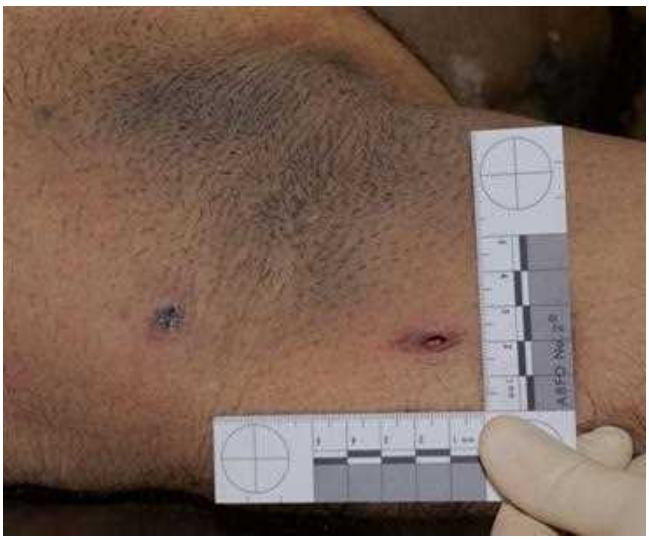

**Fig. 12.56** Shored up exit wound on inner arm, showing abrasion around the exit wound. This exit wound was shored up against a jacket that was being worn on the chest (A second entry wound is visible on the chest)

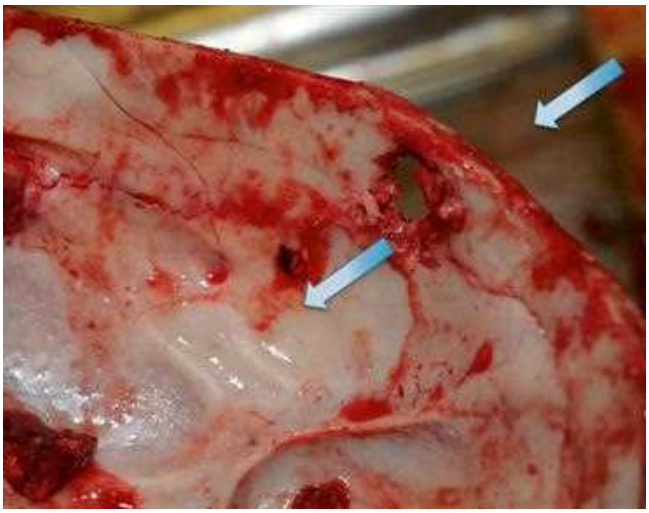

**Fig. 12.57** Gunshot wound through the skull. Collapse of the inner table confirmed the impression that the external injury was an entry wound

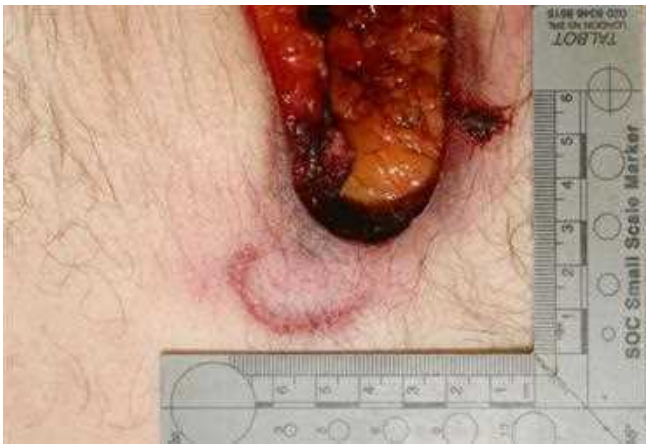


**Fig. 12.58** Contact gunshot wound from a double-barreled (side-by-side) shotgun. One barrel has discharged, and the expansion of the gases has compressed the skin against the nondischarged barrel to produce an abrasion ring

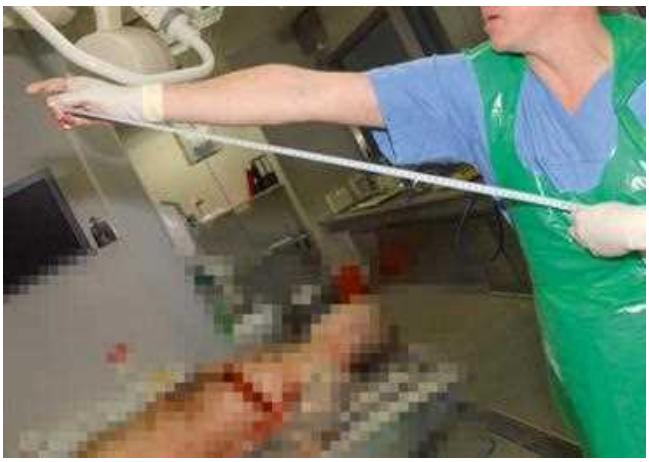

**Fig. 12.59** Coincidentally, the pathologist's upper limbs were the same length as the deceased's. Using a tape measure, it was possible to demonstrate that the deceased was capable of reaching the weapon trigger to discharge the shotgun into his chest

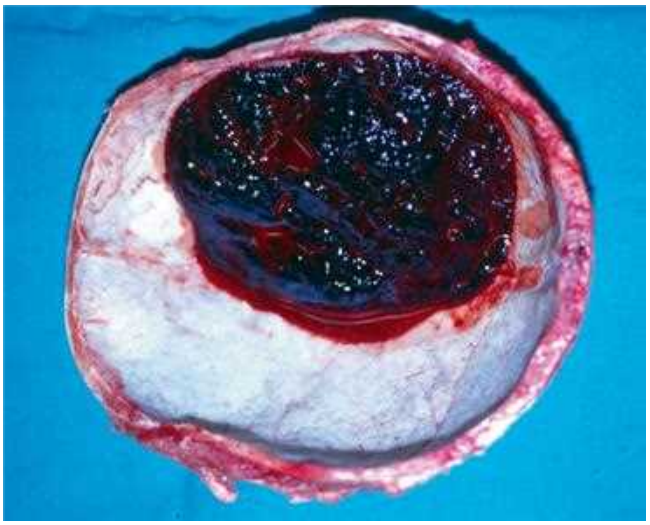

**Fig. 12.60** Extradural hematoma: skull fracture following an assault

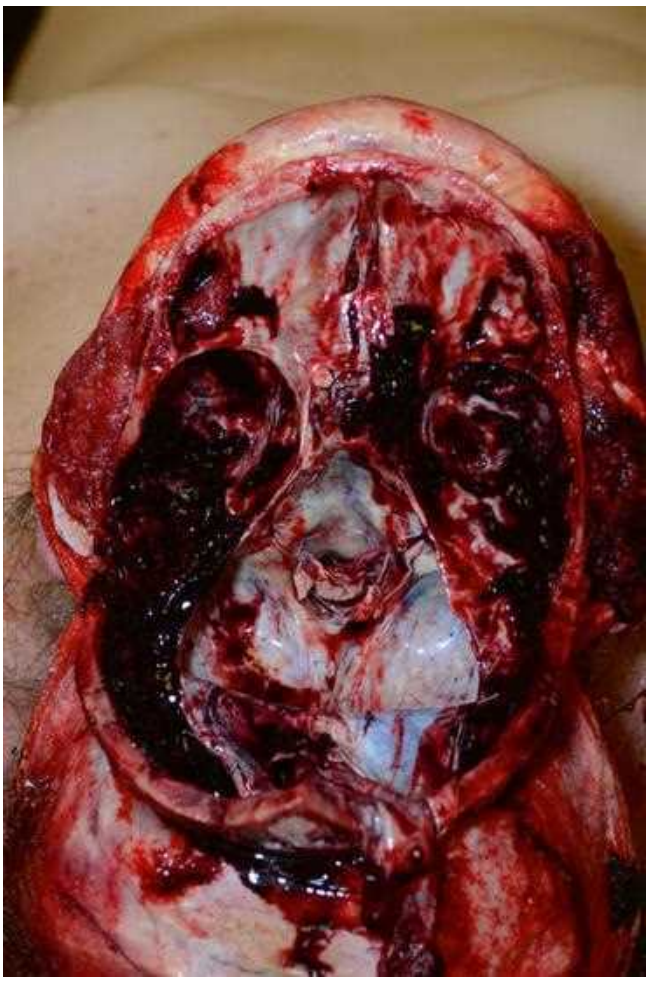

**Fig. 12.61** Subdural hematoma shown within the cranial fossae

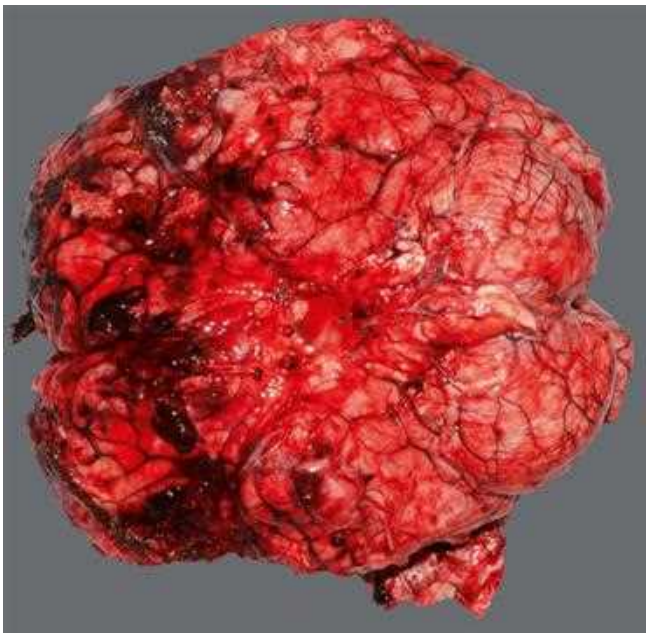

**Fig. 12.62** Contre-coup pattern of brain injury following a fall onto the back of the head

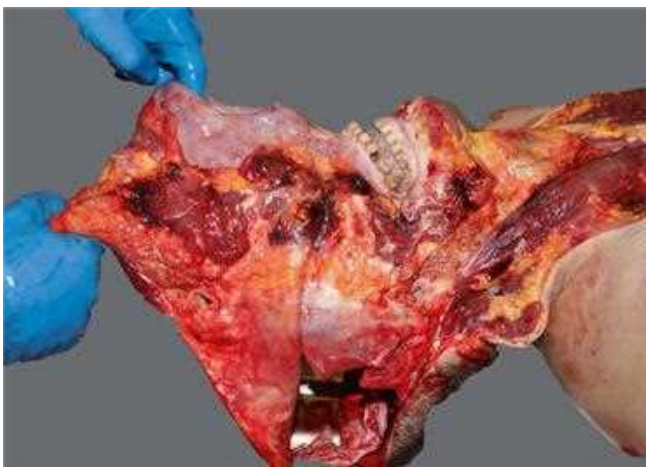

**Fig. 12.63** Traumatic basal subarachnoid haemorrhage. Deep dissection of the face revealed a bruise at the angle of the right side of the jaw from a single punch delivered in a brawl

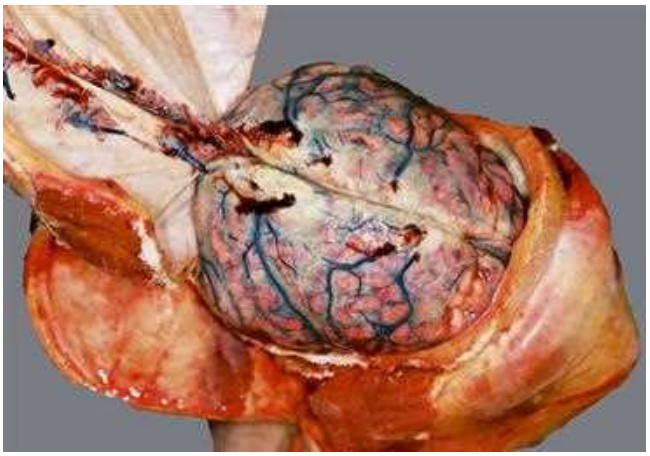

**Fig. 12.64** Post-traumatic meningitis. This individual died several days after sustaining a skull fracture following a push forwards onto the floor. The meningeal membranes are thickened and inflamed

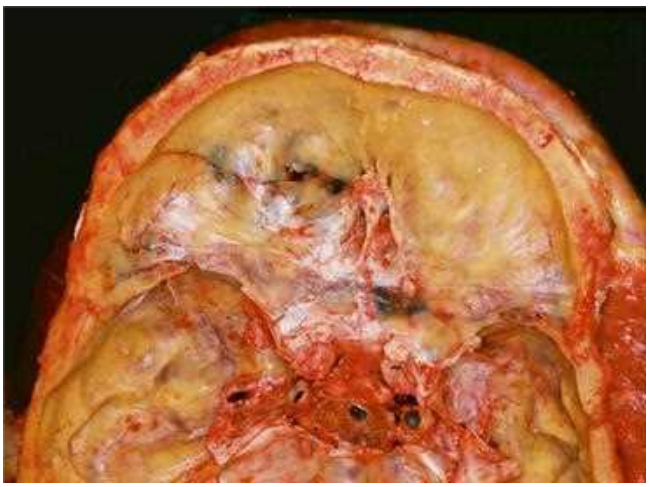

**Fig. 12.65** Post-traumatic meningitis (2). The fracture crossed the cribriform plate, presumably admitting bacteria (streptococcus) into the normally sterile environment of the skull cavity

## Suggested Reading

Burke MP. Forensic medical investigation of motor vehicle incidents. Boca Raton: CRC Press Taylor & Francis Group; 2007.

Di Maio DJ, DiMaio VJM. Forensic pathology. Boca Raton: CRC Press; 1993.

Mason JK, Purdue BN. The pathology of trauma. 3rd ed. London: Arnold Publishing; 2000.

Saukko P, Knight B. Knight's forensic pathology. 3rd ed. London: Hodder Arnold; 2004.

# The Radiological Autopsy

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## Introduction

Post-mortem imaging is increasingly being used to supplement dissection in the forensic investigation of traumatic deaths. Post-mortem computerised tomography (PMCT) is particularly effective for the identification of fractures, internal haemorrhage, and foreign bodies. Unlike dissection, it is nondestructive and produces a permanent record of the findings. The imaging findings can be demonstrated in court in order to facilitate understanding of how the deceased came to be injured.

Increasingly, post-mortem imaging is being used as an alternative, rather than merely a supplement, to traditional autopsy. Such autopsies are described as 'virtual autopsies' or digital autopsies, and are now the primary method of death investigation in some countries. In England and Wales in 2012, 46 % of all deaths were reported to a Coroner, and post-mortem examinations were ordered in 42 % of these cases [1]. In contrast, there is a low autopsy rate (2 %) in Japan, and post-mortem imaging is widely used both as an alternative to an invasive autopsy and as a screening tool to determine whether autopsy is necessary in the case of a death of unknown cause. Indeed, 89 % of large hospitals in Japan perform post-mortem imaging, with 20,000 post-mortem CT scans reported each year [2].

The emergence of the digital autopsy has also been driven by religious and cultural objections to invasive autopsy. The accuracy of early digital autopsy services was questionable because they lacked a strong evidence base and appropriate governance [3, 4]. Research in recent years, however, has defined the accuracy of the various imaging techniques, identified their strengths and weaknesses, and optimised imaging protocols [5]. The development of angiographic techniques has improved the diagnostic accuracy of imaging, particularly in the assessment of coronary heart disease [6–8].

Imaging can be combined with (minimally) invasive techniques. Of particular value, for example, are aspiration of fluids for toxicology in suspected drug-related deaths [9] and needle biopsy of tumours or solid organs in order to obtain a precise histological diagnosis [10, 11]. When an invasive autopsy is necessary, the imaging findings can be used to direct and/or limit dissection. A knowledge and understanding of the applications of post-mortem imaging is essential if it is to be used appropriately.

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## Decision Making During the Digital Autopsy

A typical digital autopsy protocol is illustrated in Fig. 13.1. It is a staged process, with the aim of limiting the invasive component of the autopsy. At each stage, the following set of questions must be answered:

### Is This Case Suitable for a Digital Autopsy Approach?

The majority of sudden adult deaths, both natural and unnatural, can be diagnosed with post-mortem CT when combined with angiographic techniques [12]. CT is more accurate than traditional autopsy in the identification of certain injuries, such as fractures and pneumothorax, which might be missed on dissection.

Relative contraindications to the digital autopsy are deaths suspected to be due to metabolic disorders or sepsis, or deaths in hospital following extensive investigation in life, including imaging. Nevertheless, even in such cases, post-mortem imaging might be used to answer specific questions and enable a limited invasive procedure to be performed.

### Which Imaging Techniques Are Required? Is Angiography Indicated?

In general, CT is more accurate than MRI in the diagnosis of sudden adult deaths [5]. The converse is true for the investigation of fetal and infant deaths. If initial noncontrast CT identifies a definitive cause of death, no further procedure is required. Conditions that can be diagnosed with certainty on CT alone include ruptured abdominal aortic aneurysm, haemopericardium due to ruptured myocardial infarct, or massive intracerebral haemorrhage.

In the absence of a definitive cause of death on noncontrast CT, the addition of coronary angiography will identify coronary artery occlusion and early myocardial infarcts. It can be argued that such angiography is more accurate than dissection in the assessment of coronary stenosis.

### Following Imaging, Is an Invasive Procedure Necessary to Determine the Cause of Death?

Approximately three quarters of sudden adult deaths can be diagnosed with CT and coronary angiography. In the remaining quarter of cases, other investigations or invasive procedures are required for one of the following reasons:

- The cause of death is unascertained on imaging. Examples include septic, metabolic, and drug-related deaths.
- An abnormality detected on imaging requires further investigation. Examples include pneumoperitoneum, suggesting a perforated viscus, or pulmonary lesions in which the differential diagnosis is between malignancy and infection.
- There is a discrepancy between the clinical history, the circumstances of the death, and the imaging findings. An example of such a case would be a history of severe abdominal pain or "coffee ground" vomit prior to sudden collapse, in which imaging demonstrates severe coronary heart disease but does not explain the abdominal symptoms.

### Can Invasive Autopsy be Limited?

It is rare for a full invasive autopsy to be required following digital autopsy. In general, opening the cranial cavity is unnecessary if no abnormality is detected on CT. Imaging is

**Fig. 13.1** A protocol for the use of post-mortem imaging to avoid traditional medicolegal autopsy, and to guide and limit dissection in those cases in which an invasive element to the examination is required

used to direct the invasive procedure. In the case of pneumoperitoneum, it is often necessary to explore only the abdominal cavity in order to identify the cause of perforation. In the case of a suspected pulmonary embolus, minimally invasive examination of the main pulmonary arteries might be the only procedure required to confirm the diagnosis.

## Are Additional Investigations Required, Such as Aspiration of Fluids for Toxicology?

Toxicology is indicated in suspected drug-related deaths and in many traumatic deaths. Needle biopsy of tumours detected on CT can be performed if a definitive histological diagnosis is required. These investigations can be performed with minimal intervention with the body.

## Issues Around Decomposition

Post-mortem changes such as free air and fluid collections and decomposition cause particular difficulties in radiological diagnosis. Post-mortem accumulation of gas in the abdominal cavity may be mistakenly attributed to a perforated viscus (Figs. 13.2, 13.3, and 13.4). In advanced decomposition, post-mortem imaging must be interpreted with particular care. This is illustrated in Fig. 13.5, in which post-mortem CT of a decomposed body suggested bronchial obstruction as the probable cause of death. Subsequent autopsy demonstrated that the soft tissue mass noted in the airways on CT was in fact maggots crawling down the airways from the mouth. In advanced decomposition, however, PMCT can be a valuable adjunct to autopsy, enabling the demonstration of skeletal injuries that might be obscured by the decomposing soft tissues.

**Fig. 13.2** Post-mortem decomposition. Axial CT image showing widespread gas, particularly in hepatic vessels, spleen, and pancreas

## Trauma

PMCT is sensitive for the detection of fractures and is superior to dissection in documenting skeletal injuries. In a large series of digital autopsies, PMCT detected evidence of trauma that was missed at standard autopsy, such as femoral neck fracture and pneumothorax [12]. In some cases, it is clear that PMCT is superior to dissection in identifying unnatural deaths. It is also the investigation of choice for the detection of metallic objects such as shot and pellets from gunshot wounds (Figs. 13.6 and 13.7). Furthermore, three-dimensional reconstruction images provide additional information about how an injury was sustained and are valuable in explaining pathology to the public for illustration in court. PMCT is also sensitive for the demonstration of internal collections of blood secondary to trauma. Finally, the use of angiographic techniques can demonstrate the precise source of haemorrhage, which frequently is not possible by dissection. Figures 13.8, 13.9, 13.10, 13.11, 13.12, 13.13, and 13.14 show other CT images involving trauma.

**Fig. 13.3** Post-mortem decomposition. Axial CT image showing pneumoperitoneum (*star*) and extensive soft tissue gas

**Fig. 13.4** Post-mortem decomposition. Axial CT image showing gas in cardiac chambers (*star*)

**Fig. 13.5** Maggots in a bronchus (*arrow*), which may result in a misdiagnosis of bronchial obstruction as the cause of death

**Fig. 13.6** Gunshot wound to the chest. Axial CT image showing entry wound (*arrow*) with haemopericardium (*arrowhead*), and haemothorax (*star*)

**Fig. 13.7** Gunshot wound to chest (same patient as 13.6). Axial CT image showing pellets in the left hemithorax (*arrow*)

**Fig. 13.8** Blunt injury to abdomen. Axial CT image showing deep laceration of liver (*arrow*) with intraperitoneal haemorrhage (*arrowheads*)

**Fig. 13.9** Femoral fracture. Coronal CT image showing an intertrochanteric fracture of the left femur (*arrow*)

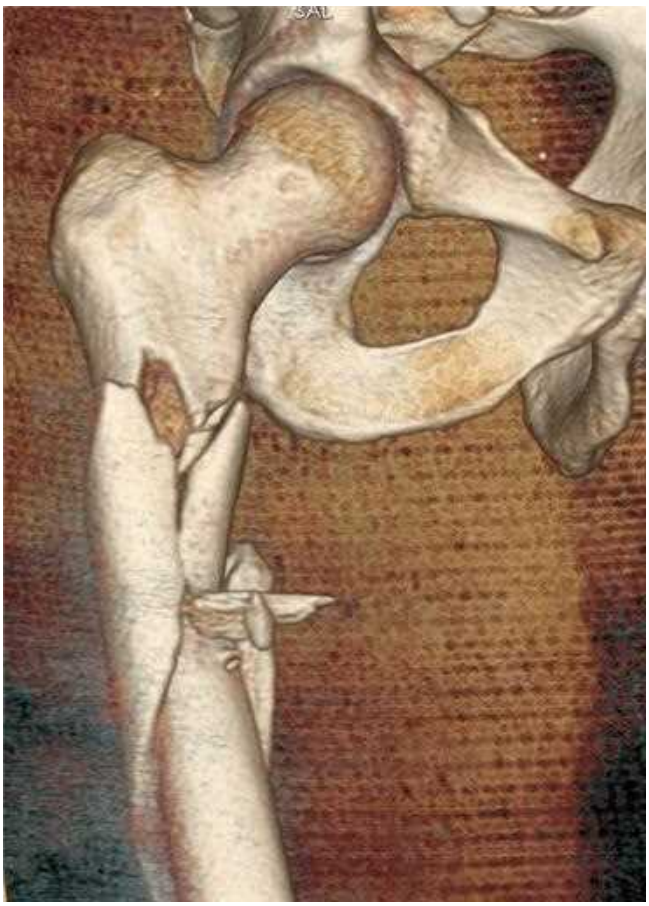

**Fig. 13.10** Femoral fracture. 3D volume-rendered image showing a comminuted fracture of the proximal right femoral shaft

**Fig. 13.11** Head injury. Axial CT image on bone windows showing multiple skull fractures and intracranial air (*arrows*)

**Fig. 13.12** Head injury (same patient as in Fig. 13.11). Axial CT image on soft tissue windows showing subarachnoid and intraventricular haemorrhage (*arrows*)

**Fig. 13.13** Sternal fracture. Sagittal reformatted CT image showing a fracture of the sternal body (*arrow*) secondary to attempted cardiopulmonary resuscitation

**Fig. 13.14** Rib fractures. Axial CT image showing bilateral anterior rib fractures (*arrows*) secondary to attempted cardiopulmonary resuscitation

## Head

PMCT is sensitive in the detection of intracranial haemorrhage, cerebral infarction, and intracranial tumours (Figs. 13.15, 13.16, 13.17, and 13.18). MRI provides superior detail of most cerebral abnormalities but is less sensitive in the diagnosis of subarachnoid haemorrhage. In a large validation series of post-mortem imaging, the only significant intracranial pathology missed on CT and MRI was meningitis.

**Fig. 13.15** Intracerebral haemorrhage. Axial CT image showing haemorrhage into the left cerebral hemisphere (*arrow*)

**Fig. 13.16** Subarachnoid haemorrhage. Axial CT image showing extensive subarachnoid blood (*arrows*)

**Fig. 13.17** Subdural haemorrhage. Axial CT image showing a right-sided subdural haematoma in the middle cranial fossa (*arrow*)

## Lungs

The lungs almost invariably appear abnormal on post-mortem imaging. A very common finding is the presence of ground-glass attenuation (a hazy increase in opacification with preservation of bronchial and vascular margins) in a dependent position, usually with a straight, horizontal anterior border, due to post-mortem hypostasis (Fig. 13.19). Perihilar consolidation and/or ground-glass opacification, frequently with interlobular septal thickening, usually indicates pulmonary oedema (Fig. 13.20). Whilst this might be secondary to left ventricular failure and fluid retention in life, it is also commonly present as a consequence of attempted cardiopulmonary resuscitation. Consolidation (a homogeneous increase in pulmonary parenchymal attenuation that obscures the margins of vessels and airway walls) is commonly due to pneumonia (Fig. 13.21). Figures 13.22, 13.23, and 13.24 are CT images showing other lung conditions.

**Fig. 13.18** Cerebral infarct. Axial CT image showing extensive cerebral infarction (more marked *on the left*) with predominant low density but with some small, high-density parenchymal haemorrhages (*arrow*)

**Fig. 13.19** Dependent ground-glass attenuation at the lung apices (*arrows*), secondary to post-mortem hypostasis

**Fig. 13.20** Pulmonary oedema. Axial CT image showing perihilar ground-glass attenuation and interlobular septal thickening secondary to pulmonary oedema

**Fig. 13.21** Consolidation. Axial CT image showing bilateral upper lobe consolidation and pleural effusions

**Fig. 13.22** Pleural mesothelioma. Axial CT image showing pleural-based tumour in the left hemithorax (*arrow*). Artefact from the arms obscures views of the posterior hemithoraces

**Fig. 13.23** Pulmonary tuberculosis. Axial CT image showing a cavitating mass (*arrow*) in the left upper lobe secondary to tuberculosis

**Fig. 13.24** Pulmonary fibrosis. Axial CT image showing honeycombing (*arrow*) with diffuse ground-glass attenuation and interlobular septal thickening

## Heart

Haemopericardium can be easily demonstrated on PMCT, but without using angiographic techniques, it is not always possible to distinguish the two commonest causes, ruptured myocardial infarction and aortic dissection (Figs. 13.25, 13.26, 13.27, and 13.28). Correlation against clinical data might assist in this regard.

The most frequent diagnostic errors made on the basis of imaging alone are in coronary heart disease and pulmonary embolism, the latter being missed in 100 % of cases in the largest validation study of the digital autopsy [5]. For this reason, the role of angiography is considered central to PMCT.

As coronary disease is the single commonest cause of death found at Coroner's autopsy, it is essential for coronary stenosis to be visualised if imaging is to be recommended as an alternative to autopsy. Coronary calcification is very commonly demonstrated on PMCT, but this finding does not necessarily equate to high-grade arterial stenosis or occlusion. Indeed, cases of patent calcified vessels are frequent, as are occluded noncalcified arteries. Visualisation of coronary artery lesions is not possible on post-mortem MRI, although myocardial abnormalities may be identified. In one series, post-mortem MRI, performed prior to autopsy, accurately identified old and recent myocardial infarcts [13]. In addition, imaging revealed myocardial abnormalities in cases with normal-appearing myocardium on macroscopic and histological examination. These abnormalities correlated with severe coronary stenosis, suggesting that MRI may be able to demonstrate early ischaemic lesions, which are not detected at macroscopic autopsy.

**Fig. 13.26** Left ventricular aneurysm. Axial CT image showing a calcified aneurysm (*arrow*) secondary to previous myocardial infarction

**Fig. 13.25** Haemopericardium. Coronal CT image showing haemopericardium (*arrows*) secondary to ruptured myocardial infarction

**Fig. 13.27** Coronary angiography. Oblique sagittal reformatted image showing the catheter balloon within the ascending aorta (*arrowhead*), calcification in the aortic valve cusps, and contrast within the left circumflex coronary artery (*arrow*)

## Abdomen

Post-mortem imaging is sensitive in the detection of tumours and other mass lesions. In this regard, as one is dealing almost entirely with soft tissues, MRI is generally superior to CT in identifying tumours in solid organs. For example, hepatic metastases frequently are not visible using CT alone, but both techniques easily detect intra-abdominal haemorrhage such as ruptured abdominal aortic aneurysm (Figs. 13.29, 13.30, 13.31, and 13.32). PMCT is sensitive for the diagnosis of pneumoperitoneum secondary to a perforated viscus, but it may be impossible to identify the precise site of perforation.

**Fig. 13.28** Coronary angiography showing a perfusion defect in the inferior wall of the left ventricle (*arrow*) with haemopericardium secondary to a ruptured myocardial infarct

**Fig. 13.29** Ruptured abdominal aortic aneurysm. Axial CT image showing a calcified aortic aneurysm (*arrow*) with retroperitoneal haemorrhage (*arrowhead*) secondary to rupture

**Fig. 13.30** Perforated duodenal ulcer. Axial CT image showing pneumoperitoneum (*arrow*) and free intraperitoneal fluid (*arrowhead*)

**Fig. 13.31** Strangulated inguinal hernia. Axial CT images showing a left inguinal hernia (*arrow*)

**Fig. 13.32** Strangulated inguinal hernia. Fluid in the pelvis secondary to peritonitis (*arrow*)

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# Toxicology

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## Introduction

Post-mortem toxicological analysis should be considered in all deaths in which there is a possibility of an overdose or drug toxicity. It should also be used to consider the cognitive function of an individual prior to death in scenarios such as road traffic collisions and suicides.

Toxicological analysis can be used to identify the presence of any substance that might be relevant to the death. Post-mortem biochemical analysis also may be of value in a proportion of sudden deaths, particularly those involving alcoholics, diabetics, or those with renal impairment, in whom the disease states may have reduced drug excretion and hence increased toxicity.

As with all such cases, when looking at any positive results, one should have some understanding of the background disease of the individual, and ideally should take advice from the toxicologist.

## Post-mortem Redistribution (Vd) and Half-Life (t½) and Pharmacokinetics

The level of any drug or toxin may be different after death, compared with levels encountered in life and in studies of healthy subjects. In life, drugs are absorbed into the bloodstream and tissues by active processes. The apparent volume into which drugs distribute is based primarily on their fat and water solubility. Drugs such as warfarin (primarily water-soluble) have a volume of distribution of approximately 1 L per kilogram, whereas many lipophilic compounds such as the antipsychotics (which have their primary effect in the brain, a tissue with high fat content) have volumes of distribution over 20 L per kilogram.

After death, the drugs will move out of the tissues in which they have been stored, to redistribute and re-equilibrate back into the blood. Therefore, drugs with a high volume of distribution (Vd), where one may use a pragmatic figure of 3 L per kilogram, tend to be significantly higher in post-mortem blood than may be expected from ante-mortem dosing studies.

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The other factor that contributes to post-mortem redistribution is the proximity to the bowel, liver, or other organs in which drugs may be concentrated. In life, active processes allow drugs to pass across the bowel wall and be absorbed into the blood. It is considered that any drugs that remain in the bowel after death are no longer actively retained in the bowel, but may undergo variable passive diffusion into surrounding tissues.

It is therefore essential that when interpreting post-mortem blood concentrations, the site of sampling and the volume of distribution are known. One should be mindful that in some cases it can be difficult to ascertain whether a high drug concentration is due to an overdose or to redistribution.

If a drug is injected, it immediately enters the bloodstream. The same is essentially true for inhaled drugs, as they pass easily into the pulmonary vasculature. Oral drugs will reach a peak concentration within approximately 30–45 min. Drugs are seen as foreign substances and so the body will metabolise them as rapidly as possible. This process requires a functioning liver and, when the drugs have been made water-soluble, excretion by functioning kidneys. The time taken for the removal of the drug is defined by its half-life (t½). The half-life is the time taken for the drug concentration to halve.

## Drug Concentrations

There is no standard nomenclature for reporting post-mortem drug concentrations. It is recommended that drugs are reported in mass units, and in an SI volume:

1000 nanograms (ng) = 1 microgram (μg) = 0.001 milligram (mg)
1000 milliltres (mL) = 1 liter (L)

There is a wide variation the drug concentrations required for clinical effects. For example, cannabis has an effect at about 2 ng/L, whereas valproate often requires a blood concentration of up to 100 mg/L (100,000,000 ng/L).

The other nuance is that in the UK, forensic blood alcohols are reported as mg/100 mL (mg/%), whereas clinical cases are reported as mg/L.

Metal concentrations (including lithium) are reported as millimoles per litre (mmol/L).

## Samples Required for Toxicology Analysis

Most bodies are recovered soon after death, and many different samples are possible. This situation may differ with a decomposed body or after embalming has taken place. See also Chap. 10 regarding samples used for special tests.

## Routine Cases

In routine cases, the following samples are recommended:

- Blood
  - Unpreserved (*i.e.*, plain) blood: ideally 20 mL or greater (drug screen and quantitation)
  - Fluoride oxalate preserved blood: ideally 10 mL or greater (ideal for ethanol, cocaine, or diazepam quantitation)
- Urine
  - Unpreserved (*i.e.*, plain) urine: ideally 20 mL or greater (general screen)
  - Fluoride oxalate preserved urine: ideally 10 mL or greater (ideal for ethanol analysis)
- Vitreous fluid
  - Unpreserved vitreous fluid: as much as is available for biochemical analysis
  - Fluoride oxalate preserved vitreous fluid: 1 mL for ethanol analysis
- Stomach content
  - Only worth sending if an overdose close to the time of death is suspected. Ideally all content should be sent. If an aliquot is sent, it must be as representative of the whole content as possible, and the total volume of stomach content must be recorded.
- Solid tissues
  - Liver (deep within the right lobe) and muscle (*e.g.*, psoas) may be sent. A 1-cm cube is often sufficient for screening purposes.

Rarely sampled sites are covered in Table 14.1.

**Table 14.1** Sample requirements for toxicology analysis

| Specimen | Common uses |
|---|---|
| Blood/plasma/serum | Preferred specimen (recent use with respect to death) |
| Bile (rarely collected) | |
| Bone/bone marrow (rarely collected) | |
| Fat (rarely collected) | Volatiles after increased postexposure period |
| Gastric contents | Orally administered drugs and poisons |
| Hair (not often collected for medicolegal cases) | All substances, especially basic/most metals |
| Muscle (preferably psoas) | Most drugs; useful in decomposition if no other fluids |
| Pleural effusion (rarely collected) | |
| Vitreous fluid | Ethanol, some biochemistry, glucose, electrolytes |
| Liver (deep in right lobe) | Most drugs; useful in decomposition if no other fluids |
| Lung | Volatiles/anaesthetic gases |
| Kidney (rarely collected) | Heavy metals |
| Faeces (rarely collected) | Porphyria |
| Brain (rarely collected) | Volatiles |

## Embalmed Bodies

Embalming chemicals consist of a variety of preservatives, sanitising and disinfectant agents, and additives used to temporarily prevent decomposition. Embalming fluid typically contains a mixture of formaldehyde, methanol, ethanol, and other solvents. The formaldehyde content generally ranges from 5 to 29 %, and the ethanol content may range from 9 to 56 %. There is no international standard.

It is self-evident that any embalming of the corpse may cause considerable changes of initial drug levels, due to alterations of the biological matrix, dilution of samples, and release or degradation of the drug or poison. If one can identify a source of uncontaminated fluid, then some testing may be possible, but liaison with the toxicologist is often beneficial. One area to consider is the cavernous sinus veins, if the head has not been perfused with embalming fluid or opened.

If one is sending any sample from an embalmed corpse for toxicology analysis, one also should always send an aliquot of the embalming fluid to compare with post-mortem fluid volatiles.

## Commonly Encountered Drugs and Toxins

The following section deals with the various drugs and toxins regularly encountered in toxicology analysis.

## Ethanol

Ethanol is an alcohol. It is consumed as a social drug, but along with other alcohols, it is also produced as part of the post-mortem process of the body breaking down. One unit of ethanol (8 g) of will lead to a peak blood alcohol of approximately 20 mg/100 mL of blood. Ethanol is excreted and/or metabolised at a rate of approximately 20 mg/100 mL per hour (range 10–35 mg/100 mL per hour).

Toxicology laboratories are often well versed in measuring ethanol levels and confounding realities, such as post-mortem production. Most laboratories should also measure ethyl glucuronide (a nonenzymatic metabolite of ethanol) as a confirmatory marker for ethanol consumption.

Post-mortem ethanol is highly variable and depends on the ambient circumstances of death [1]. Production is increased if there is infection or bacterial contamination of blood (and less commonly, urine). It is common to see concentrations of up to 20 mg/100 mL in most cases, and it is not unusual to see concentrations up to 100 mg/100 mL, and some concentrations up to 250 mg/100 mL have been reported. As vitreous fluid is found within a "contained" space, it is often the ideal sample for ethanol analysis.

**t½:** Ethanol shows zero order kinetics, falling by approximately 20 mg/100 mL per hour (range, 10–35 mg/100 mL)
**Vd:** Not applicable
**Samples:** Preserved blood, urine and vitreous

The clinical effects of ethanol vary with the blood concentration (Table 14.2).

**Table 14.2** Blood alcohol concentrations and general effects

| Concentration | General effects |
|---|---|
| 10–50 mg/100 mL | Minimal outward effects; feelings of relaxation and well-being, increased sociability |
| 50–100 mg/100 mL | Increased self-confidence and talkativeness, mild euphoria, reduced co-ordination and slightly slowed reactions (Legal limit for driving, 80 mg/100 mL in England and Wales; 50 mg/100 mL in Scotland) |
| 100–150 mg/100 mL | Impaired balance, clumsiness, reduced alertness, lowered social reserve, increased excitability |
| 150–200 mg/100 mL | Slurred speech, glazed eyes, flushed complexion, staggered gait, drowsiness, exaggerated emotional responses, impaired coordination, reduced inhibitions, dizziness, nausea, disorientation |
| 200–250 mg/100 mL | Marked or heavy drunkenness, confusion, grossly impaired coordination, vomiting, reduced awareness; often impaired short-term memory |
| 250–300 mg/100 mL | Extreme drunkenness, stupor, impaired consciousness, reduced reflexes, depressed respiration, incontinence |
| 300–400 mg/100 mL | Unconsciousness, absence of reflexes, coma; possible death |
| About 400 mg/100 mL and above | Possible death by respiratory depression or cardiac arrest |

This table is for guidance only and may not apply to any specific individual. The symptoms can overlap considerably and gradually increase in severity with increasing concentrations. The effects described apply to a social drinker but depend on the degree of habituation. A heavy drinker will show less effect, and a person unaccustomed to alcohol will have more pronounced symptoms. Therefore the effects of any given blood alcohol level cannot be determined simply by reference to a table. Tolerance should be considered, together with other information such as evidence from witnesses and the findings of any medical examination. It is not possible to determine a particular level of alcohol that may cause memory loss in a specific individual or render an individual unable to form an intent or to give informed consent, such as for sexual activity.

There is a legal limit for driving a motor vehicle: 80 mg/100 mL blood in England and Wales, and 50 mg/100 mL in Scotland. Different legal limits apply elsewhere in the world. It is often difficult to be definitive as to whether a post-mortem blood alcohol level represents a level above the legal limit.

## Ethylene Glycol

Ethylene glycol is a toxic alcohol used in engine antifreeze or as a radiator coolant. It is metabolised to four toxic metabolites: glycoaldehyde, glycolate, glycolic acid, and glyoxylate. It can be used as an adulterant of other alcohols or even deliberately as a poison.

**t½:** 3 h
**Vd:** 0.8
**Samples:** Preserved blood

Toxic effects are usually associated with serum ethylene glycol concentrations greater than 50 mg/100 mL (500 mg/L).

During the first few hours after ingestion of ethylene glycol, the individual may experience transient inebriation, euphoria, nausea, and vomiting. The clinical course of ethylene glycol poisoning has been described in three stages:

- Neurological stage (first 12 h): The accumulation of toxic ethylene glycol metabolites produces neurological symptoms including nystagmus, ataxia, myoclonic jerking, hypotonia, seizures, and coma.
- Cardiopulmonary stage (12–24 h): Tachycardia, hypertension, metabolic acidosis with a compensatory hyperventilation, and multiple organ failure may occur. It has been reported that death most commonly occurs during this second stage.
- Renal stage (24–72 h onwards): Renal failure with oliguria may result, even with ingestion of ethylene glycol at a nonfatal level. The ethylene glycol metabolites may cause kidney tissue destruction primarily from deposition of calcium oxalate crystals.

## Ketoacids and Ketoacidosis

Ketoacidosis should be suspected by the toxicologist when excess acetone is detected on analysis of volatiles (ethanol). This finding should prompt measurement of beta-hydroxybutyrate (BHB) in the blood and/or vitreous fluid. Acetone may also result from post-mortem autolysis, but BHB is unlikely to result from changes in the post-mortem period.

Ketonaemia is relevant in two main scenarios, chronic alcoholism and diabetes mellitus (Table 14.3). Excessive formation of ketone bodies (acetone and BHB) is also observed in conditions associated with decreased availability of carbohydrate (such as starvation or frequent vomiting) or decreased use of carbohydrates (such as diabetes mellitus and alkalosis).

**t½:** 3 h
**Vd:** 0.8
**Samples:** Preserved urine (volatile screen), preserved blood (volatile screen); preserved and unpreserved vitreous (for BHB and vitreous glucose); plain blood if assessing HbA1c

The concentration of BHB indicative of a ketoacidosis is not well defined. Some papers quote concentrations as low as 1 mmol/L as pathologically significant, but individuals may show no effects with concentrations up to 5 mmol/L. Hence levels from 1 to 5 mmol/L are a grey area.

**Table 14.3** Patterns of ketoacidosis

| Diabetic | Alcoholic |
|---|---|
| History of type 1 diabetes mellitus | No history of type 1 diabetes mellitus |
| Vitreous glucose raised/detectable | Undetectable vitreous glucose |
| Raised HbA1c | Normal HbA1c |
| No alcohol history | History of chronic alcohol misuse |

Ketosis also occurs with starvation, and beta-hydroxybutyrate (BHB) may also be a marker for a chronic stress response such as hypothermia.

## Opiates and Opioids

Opiates are a wide family of drugs, including morphine, heroin, codeine, pholcodine, and others. Opiates are chemically related to the morphine molecule and so act on the morphine opiate receptors. Opioids are not chemically related to morphine, but they act on some of the receptors that are reactive to opiates.

### Heroin (Diacetyl Morphine) and Morphine

Diacetyl morphine (diamorphine) may be used therapeutically for pain relief in a hospital or palliative care setting. It is identical to illicit heroin, but illicit heroin is likely to have contaminants and cutting agents added. Heroin is widely abused.

Heroin is metabolised almost instantaneously to 6-monoacetylmorphine (6-MAM), which is then further metabolised to morphine. Morphine is usually quantified in toxicology analyses. The presence of 6-MAM is consistent with the use of diamorphine/heroin within the previous 12–24 h. In the absence of 6-MAM, unless other contaminants such as noscapine or meconin are detected, it is not possible to analytically distinguish morphine administration from the use of diamorphine/heroin. 6-MAM is detected better in vitreous and urine than in blood; detection in blood is likely to represent rapid death after use of illicit heroin or diamorphine.

Morphine is likewise used as for pain relief, with similar realities to diamorphine. It is metabolised to morphine-3 and morphine-6 glucuronides in the liver. The morphine-3 glucuronide is pharmacologically inactive, whereas morphine-6 glucuronide is at least as potent as morphine with regard to sedation and respiratory depression.

Morphine in its nascent form is described as "free" morphine. The sum of free morphine, and morphine-3 and morphine-6 glucuronides is described as "total" morphine. The ratio of total to free morphine was found to range from 1:1 to 2:1 when death had occurred within 3 h of the administration of heroin in a naïve user. As the glucuronides are excreted by the kidney, renal impairment leads to accumulation of the morphine metabolites, including the active morphine-6 glucuronide.

**t½:** 2–6 h
**Vd:** 3–5 L/kg
**Samples:** Preserved or unpreserved blood for screening and quantitation; preserved or unpreserved urine for initial screening. 6-monoacetyl-morphine is well detected in vitreous humour

Fatal concentrations are highly dependent on tolerance and time from administration to death. In a naïve individual, concentrations as low as 100 ng/mL may be fatal. Deaths are more likely to occur at lower concentrations if other sedative drugs (*e.g.*, alcohol, benzodiazepines, methadone) are concurrently present. Tolerance may occur, but is lost with a few days of abstinence.

Morphine toxicity is associated with progressive depression of the central nervous system, which may lead to coma, marked reduction of respiratory rate, and respiratory arrest. Induction of vomiting increases the risk of aspiration pneumonia. Opiates may also cause a prolonged period of respiratory depression, which then leads to pulmonary oedema. Morphine may be metabolised during this period, perhaps accounting for what appears to be a low fatal opiate concentration.

### Codeine

**t½:** 1.9–3.9 h
**Vd:** 3.5 L/kg
**Samples:** Preserved or unpreserved blood for screening and quantitation; preserved or unpreserved urine for initial screening

Codeine has analgesic properties, but the narcotic effects are substantially mediated by morphine. In the absence of other contributing factors, there is strong evidence that free codeine concentrations as low as 400 ng/mL may be sufficient to cause death [2].

### Dihydrocodeine

**t½:** 3.5–4.5 h
**Vd:** 1–1.3 L/kg
**Samples:** Preserved or unpreserved blood for screening and quantitation; preserved or unpreserved urine for initial screening

In deaths attributed to dihydrocodeine alone the blood dihydrocodeine levels ranged between 900 and 19,900 ng/mL (median 3800 ng/mL). In polydrug intoxication that included dihydrocodeine, the blood dihydrocodeine levels ranged from 30 to 17,500 ng/mL (median 900 ng/mL) [3].

### Methadone

Methadone is a potent opioid narcotic analgesic. It is the most common substitution drug in the treatment of heroin addiction. Methadone is metabolised to its major inactive metabolite EDDP (2-ethylidene-1,5-dimethyl-3,3-diphenylpyrrolidine). Peak methadone levels occur 2–4 h after ingestion.

The interpretation of the methadone result requires knowledge of the history of drug use and therefore the individual's degree of tolerance.

**t½:** 10–35 h
**Vd:** 3–5 L/kg
**Samples:** Ideally plain urine and blood for screening; plain blood for quantitation

In fatal cases in which methadone is the only drug found in the blood, methadone concentrations vary greatly, with a mean of about 500 ng/mL but a range of 84–2700 ng/mL [4]. Concentrations of up to about 1200 ng/mL are observed in "well" methadone users, so the interpretation is dependent on details of the death.

Methadone, in common with other opioids, causes respiratory depression even at therapeutic doses. It can cause a prolonged period of unconsciousness that then leads to gradually worsening hypoventilation and respiratory failure. Methadone also causes arrhythmias independent of its effects on respiration and hypoxia.

## Fentanyl

Fentanyl is a potent synthetic narcotic analgesic usually administered in the form of a transdermal patch for the management of chronic pain, as a transmucosal lozenge, or intravenously for enhancement of anaesthesia. Fentanyl is thought to be 75–100 times more potent than morphine.

**t½:** 3.7 h
**Vd:** 3–8 L/kg
**Samples:** Ideally plain urine and blood for screening; plain blood for quantitation

Mean serum fentanyl concentrations have been reported to range from 0.3 to 2.5 ng/mL within 24 h after the therapeutic administration of transdermal patches with dosages of 12.5–100 μg/h, respectively. Chronic users develop a significant degree of tolerance. In naïve individuals, death may be attributed to therapeutic-range fentanyl levels, but it is not common for concentrations to be above 10 ng/mL [5].

Significant rises in blood fentanyl may occur if a patch is applied to damaged skin or if there is a rise in body temperature, such as occurs with infection. It is theoretically possible that fentanyl may continue to be released from a patch in the post-mortem period, so sampling should be distant from the patch application site.

As with other opioids, the primary side effects are reduction of consciousness and respiratory depression. There is also an increased risk of potentially fatal tachyarrhythmias.

## Tramadol

**t½:** 6 h
**Vd:** 3 L/kg
**Samples:** Ideally unpreserved urine to detect metabolite; ideally plain blood for quantitation

Tramadol is an analgesic with a complex mode of action. It has both opioid and nonopioid analgesic effects, as well as serotonin effects. Tramadol is metabolised to the active metabolite O-desmethyltramadol, which is thought to have a longer half-life.

The potentially lethal blood tramadol concentration has been reported to be greater than 2000 ng/mL. In some case reports in which tramadol was the only drug thought to have caused intentional or unintentional death, its concentration ranged between 6600 and 20,000 ng/mL. When tramadol was taken with other drugs, its concentration in fatal cases ranged between 30 and 22,600 ng/mL [6].

Side effects include respiratory depression, lethargy, seizure, coma, cardiac arrest, and death. Concomitant use of tramadol with tricyclic antidepressants such as amitriptyline increases the risk of seizures and cardiac arrhythmias.

## Benzodiazepines

Many benzodiazepines are available for therapeutic use, but many are also abused. Benzodiazepines are widely prescribed for the treatment of anxiety and insomnia. They also may be added to illicit heroin as a bulking agent. This chapter cannot cover all benzodiazepines, so the most common, diazepam, is used as a paradigm; all others in the class have similar realities and measurements. Special comment is given to chlordiazepoxide, which is used in the management of alcohol consumption issues.

## Diazepam

Diazepam, like many benzodiazepines, is metabolised to other active benzodiazepines. Diazepam is metabolised to nordiazepam, temazepam, and oxazepam. This metabolism may occur in vitro in blood samples but can be reduced by using fluoride oxalate preservative vials for samples.

**t½:** 20–40 h (nordiazepam, 40–100 h)
**Vd:** 06–2.4 L/kg
**Samples:** Unpreserved urine screen; preserved blood sample for quantitation

The acute toxicity of benzodiazepines is extremely low. Even large overdoses taken alone rarely cause death, although risks are increased in the presence of respiratory or cardiovascular disease. Post-mortem blood levels of diazepam above approximately 5000 ng/mL may be considered as causing death.

Oversedation is thought to be a dose-related extension of the sedative-hypnotic effects of benzodiazepines. Symptoms include drowsiness, poor concentration and vigilance, ataxia, dysarthria, motor incoordination, diplopia, muscle weakness, vertigo, and mental confusion. In view of the prolonged half-life, the effects may persist for more than 24 h. The elderly appear to be particularly vulnerable to amnesic/forgetfulness effects of benzodiazepines. Tolerance to the sedative effects of benzodiazepines has been known to develop over 1 or 2 weeks of their use.

### Chlordiazepoxide

Chlordiazepoxide is a benzodiazepine used for the treatment of anxiety and insomnia, and also as an adjunct in acute alcohol withdrawal. It is metabolised to nordiazepam, oxazepam, and desmethylmedazepam, which are also active benzodiazepines.

**t½:** 5–30 h
**Vd:** 0.3–0.6 L/kg
**Samples:** Unpreserved blood and urine for screen; preserved blood sample for quantitation

Post-mortem blood levels above approximately 5000 ng/mL were associated with ataxia, hyperreflexia, tachycardia, and seizures. Blood levels above 20,000 ng/mL are reported as fatal when present as the only drug detected.

### Cannabis (Tetrahydrocannabinol [THC])

Over 100 different cannabinoids are present in the cannabis plant. They are generally abused for their mind-altering properties, although a mild analgesic effect is recognised. Tetrahydrocannabinol (THC) is the main active ingredient of cannabis, and there are medicinal forms of cannabis that contain only THC. Metabolic degradation of THC produces 11-delta-9-THC-carboxylic acid (THC-COOH) as the main metabolite.

**t½:** 20–57 h (infrequent users); 3–13 days (frequent users)
**Vd:** 4–14 L/kg
**Samples:** Plain blood and urine for screening; plain blood for quantitation

A typical peak blood cannabis concentration of 100 ng/mL may be expected after use. THC is rapidly distributed from blood to tissues, and blood levels quickly drop by greater than 90 % within 2 h of administration. Blood THC levels greater than 2 ng/mL are suggestive of recent use. THC-COOH is inactive. This product and other cannabinoid metabolites can be detected in the blood for up to 5 days and in the urine for up to about 30 days.

If cannabis is smoked, the effects are seen within 2–3 min, peaking at 10–20 min and lasting for 90–120 min. At higher doses, the effects may last for up to 3–4 h. If taken orally, the onset of action is 30–60 min, with symptoms typically persisting for 3–8 h.

The symptoms associated with low to moderate doses include relaxation; well-being; euphoria; drowsiness; distortions of perception of time, body image, and distance; mild confusion; decreased concentration; decreased muscle strength and balance; impaired ability to perform complex motor tasks; and panic, or even mild paranoia. With high doses, hallucinations may occur. Cannabis at high doses can induce acute psychotic symptoms, particularly in susceptible individuals, but there is debate as to whether regular use of potent-variety cannabis causes chronic psychosis or schizophrenia. Cannabis can cause exercise-related tachyarrhythmias, but it is very rarely implicated as a cause of death.

Cannabis has a detrimental effect on complex tasks such as driving. In England and Wales, the legal limit for driving is a whole blood concentration of 2 ng/mL [7].

### Cocaine

Cocaine is a widely abused, naturally occurring stimulant with mood-altering (euphoric) actions. It also has anaesthetic properties and is utilised in local anaesthetics. Cocaine is metabolised to the inactive metabolites benzoylecgonine and methylecgonine, both of which are inactive. In persons co-consuming ethanol, cocaine is also metabolised to cocaethylene, which has toxicity similar to cocaine.

Hydrolysis of cocaine to its metabolites is thought to start immediately after exposure and to continue in the post-mortem period. Sampling into fluoride oxalate preservative tubes reduces the rate of hydrolysis.

**t½:** Cocaine 0.7–1.5 h; benzoylecgonine, 3.5–8 h; methylecgonine, 3.5–6 h
**Vd**: Cocaine 1.6–2.7 L/kg
**Samples:** Preserved blood (preferably fluoride-oxalate preserved) for screening and quantitation; ideally preserved urine for initial screening

Cocaine levels less than 50 ng/mL are generally thought not to produce measurable physiologic effects, let alone toxicity. Thus, in the absence of any confirmatory histopathologic change, cocaine levels less than 50 ng/mL should not be deemed to be the cause of death. If appropriate histopathologic changes are present, however, cocaine may be the cause of death even when the blood cocaine levels are zero. "Fatal" ranges are wide owing to differences in dose, route of administration, period of survival, and storage of the sample, but the association appears stronger when concentrations are greater than approximately 500 ng/mL [8].

Cardiovascular damage is relatively common in young cocaine users. Ischaemic events have been reported regularly (due to both intravascular thrombus formation and vasoconstriction). There is a strong relationship between cocaine use, heart contraction bands, and sudden arrhythmic death. Associations have also been made with myocarditis, cardiomyopathy, and valvular heart disease in those who abuse cocaine. Chronic use can lead to significant tolerance to the stimulant effects of cocaine.

## Amphetamine

Amphetamine is a stimulant that is pharmacologically related to the catecholamines, adrenaline and noradrenaline. It is used for its euphoric effect and ability to augment physical and mental work speeds. Tolerance develops to both the stimulant and euphoric effects of amphetamine. so there is considerable overlap between the nontoxic and toxic blood drug levels.

**t½:** 12 h
**Vd:** 3–4 L/kg
**Samples:** Plain blood and urine for screening; plain blood for quantitation

Amphetamine use may lead to reactions such as agitation, fever, and aggression, which can lead to very high body temperatures and elevated heart rate and blood pressure. These reactions may lead to rhabdomyolysis (muscle breakdown), intravascular coagulation, convulsions, haemorrhages in blood vessels, and renal failure. Cardiotoxicity associated with the use or abuse of amphetamine or its synthetic derivatives can manifest itself as acute myocardial infarction, cardiomyopathy, or arrhythmia. Amphetamine use may also be associated with poor cognition and decision making, putting one at risk of traumatic death.

Death from amphetamine toxicity often will not occur immediately, but rather after a few hours of agitation, fever, aggression, and hyperthermia, so post-mortem blood levels may not reflect peak concentrations. Arrhythmias can occur at a blood concentration of 20 ng/mL, but tolerant individuals may have minimal effects with a concentration of 600 ng/mL. Death from amphetamine toxicity is usually considered when post-mortem concentrations are above 500 ng/mL [9].

## MDMA (Ecstasy) and MDA

3,4-Methylenedioxy-methamphetamine (MDMA) is a synthetic psychostimulant that is used widely in the club scene. MDMA displays stimulant effects such as euphoria, well-being, happiness, stimulation, increased energy, extraversion, feeling close to others, increased empathy, increased sociability, enhanced mood, mild perceptual disturbances, and changed perception of colours and sounds. About 10 % of MDMA is metabolised to methylenedioxy-amphetamine (MDA), which is also active and possesses similar properties to MDMA.

**t½:** 6–7 h
**Vd:** 6 L/kg
**Samples:** Plain urine and blood for screen; plain blood for quantitation

After oral administration, peak blood levels of MDMA are reached within 1–3 h. A 75 mg dose of MDMA leads to a peak blood MDMA level of 130 ng/mL within 2 h, with MDA peaking at 7.8 ng/mL within 5 h.

MDMA also has a range of effects that can lead to acute toxic reactions, including hyperthermia (which may result in rhabdomyolysis, dehydration, and renal failure), raised blood pressure, raised heart rate, cardiac arrhythmias, increased coagulability state, hypertension (which may in turn lead to strokes), and the "serotonin syndrome" (increased muscle rigidity, hyperreflexia, seizures, and hyperthermia). These acute effects of MDMA are not dose-related. It has also been reported that chronic use of amphetamines, including MDMA, may result in cardiotoxicity in relatively young individuals without pre-existing cardiac disease. This cardiotoxicity may manifest itself as acute myocardial infarction (due to vascular spasm), cardiomyopathy, or cardiac arrhythmia.

## Carboxyhaemoglobin (COHb)

Carbon monoxide is normally respired, but low levels are produced with combustion of carbon fuels (fires, internal combustion engines). It readily complexes with haemoglobin (Hb) to produce avid binding and exclusion of oxygen binding. The bound gas has a characteristic pink colour, reflecting the carboxyhaemoglobin (COHb). In high concentrations, it may deprive the body of oxygen, causing end-organ hypoxic damage.

Carbon monoxide is the major toxicant in modern fires, as it has a greater affinity than oxygen for haemoglobin and thus results in tissue hypoxia. Deaths usually occur either from fires or from inhalation of exhaust fumes from petrol engines.

**t½:** 4 h on air; 60 min when administered 100 % oxygen
**Vd:** Not applicable
**Samples:** Plain or preserved blood

The COHb normal range is less than 10 % total haemoglobin. Toxicity occurs variably between 15 and 50 %, and death is expected in a healthy individual at greater than 50 %. Death may occur at a lower percentage of haemoglobin in those with pre-existing heart or lung disease.

Symptoms due to carbon monoxide exposure vary from person to person. Prolonged or high exposure may result in symptoms such as nausea, dizziness, headaches, vomiting, confusion, collapse, loss of consciousness, angina, and muscle weakness. Severe exposure can result in brain and heart damage or death. Medical problems such as heart and lung conditions, vascular disease, anaemic conditions, and cigarette smoking may increase susceptibility to carbon monoxide poisoning.

## Cyanide

Cyanide is a pure toxin that is not normally encountered in life. The burning of common substances such as rubber, plastic, and silk can create cyanide fumes. The main uses of cyanide are in the photographic, chemical research, synthetic plastic, metal processing, and electroplating industries. During cyanide toxicity, mitochondrial energy production is inhibited. The brain and heart are most vulnerable to the toxic effects of cyanide. Rarely, it is used in suicide and homicide cases.

The lethal range for cyanide is 1 mg/L. The reported symptoms of cyanide toxicity include nausea, headache, dizziness, disorientation, seizure, tachycardia, arrhythmia, and in severe cases, cardiopulmonary arrest. The blood cyanide level is well within the quoted lethal range.

*Note:* Suspected cases of cyanide poisoning should be approached with caution to avoid the breathing in of cyanide fumes by the autopsy team.

## Paracetamol

Paracetamol is a mild analgesic also available in many proprietary preparations that may contain mild opiate analgesics such as codeine (*eg*, co-codamol). Paracetamol is not toxic unless levels are above 500 mg/L, in which case a de novo metabolic acidosis may occur. Rather, the paracetamol metabolite can cause fatalities, generally as a result of liver necrosis and failure.

**t½:** 1–4 h
**Vd:** 0.8–1 L/kg
**Samples:** Plain blood for quantitation

The interpretation of blood paracetamol levels should take into account the time between ingestion and blood sampling. The peak blood paracetamol level is reached 1–3 h (mean, 1.4 h) after a single dose. The half-life of paracetamol in adults after a therapeutic dose is thought to be 1–4 h, but when liver damage is present, the half-life may be greater than 4 h. Liver necrosis (damage) is maximal 3–4 days after ingestion of a paracetamol overdose.

Nonspecific symptoms may include anorexia, nausea, vomiting, malaise, and features of hypoglycaemia, coagulopathy, and encephalopathy. There may be upper quadrant pain or tenderness, hepatomegaly, and oliguria. The symptoms usually worsen within the next 3–5 days, and central nervous system depression, shock, hypothermia, and metabolic acidosis may develop. It has been reported that repeated administration of as little as 20 mg/kg per day of paracetamol over a protracted period of time may be associated with liver failure.

## New Psychoactive Substances

Increasing numbers of new drugs are causing harm, have been linked to deaths, and are driving public and media concern. These drugs, known as new psychoactive substances (NPS) and often misleadingly referred to as "legal highs", pose a challenge to government, local authorities, healthcare services, and the criminal justice system.

These drugs are often sold (on the Internet or in "head shops") in foil packages with "Not for human consumption" clearly marked. They often contain stimulant compounds similar to cocaine, or cannabinoid-like compounds that have significantly greater potency than cannabis and often have stimulant characteristics.

Many of these compounds will not be detected by the first-line screens performed even in the leading toxicology laboratories. Specific analysis will be required, often prompted by the history. At present, there is no standardised sampling, so both blood and urine should be collected whenever possible.

**t½:** Unknown
**Vd:** Unknown
**Samples:** 10 mL plain blood

## Other Drugs and Toxins

Many other drugs and toxins exist, and the reader is referred to specialist texts for those not covered here, as this chapter has by necessity been brief. Any specific questions should be directed to your toxicology supplier. Use of the UK National Poisons Information Service is recommended.

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## Suggested Reading


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# Appendix

Two items merit inclusion at the end of this book.

The first item is a one-page document (used in the United Kingdom) to indicate the initial basic data after an autopsy has been performed. This details the name of the deceased along with basic information such as date of death and examination. There are choices regarding whether the case is concluded without histology or other tests or that require more work. The cause of death is given on this form, although on occasion "awaiting further tests" or "pending" is a reasonable response if the case has no clear/immediate solution. Clearly one should always indicate what samples have been taken. This can be generic as here or precise in terms of the volume/weight/number of samples. Of particular use is the box for comments. This allows the pathologist to record initial thoughts and finally sign off at the form's base.

This form is not meant to be proscriptive, or all encompassing. It is a guide for the reasonable end point of the case analysis.

The second item is a two-sided document. It is recommended that it be copied onto an A3 sheet, which is then folded in half. The first page is the generic data and external examination sheet, with the second page detailing the internal examination data, weights of the organs, and a summary of what samples were taken (if any). It is recommended to have both pages on the outer side of the folded A3 sheet (rather like the external cover of a book). This leaves two blank sides internally for additional notes, documents, correspondence, and so on. In effect, it is its own file binder. The boxes for data entry are largely self-explanatory, but this simple document serves to provide a quick written summary sheet for those working in the mortuary.



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## Notification to Coroner upon Completion of Postmortem Examination

Regs 14 &15 Coroners (Investigations) Regulations 2013

**CVS**

pericardium
heart ext
myocardium
chambers tvs diam R/L
valves / / /

LAD RCA CxA etc
aorta large artery
venae cavae

RV LV/S

**RS**

larynx ETT ok
trachea
bronchi pleural cavity R L
lungs parenchyma
arteries emboli

**GIT** mouth teeth tongue
esophagus
stomach
small intestine
appendix
colon
mesenteries peritoneum

liver ext section
gall bladder bile ducts
pancreas

B
H
RL
LL
Liv
RK
LK
S

**GU** kidney surfaces
parenchyma

pelves ureters bladder

prostate testes/ext genitalia

ut/cx FT ov
breasts

**Endocrine**

thyroid
parathyroids
adrenals
pituitary

**RES** thymus
spleen ext cut section
LN thorax abdo
bone marrow

**CNS** skull venous sinuses
dura leptomeninges
cranial n arteries
brain
cerebrum
cerebellum
midbrain pons medulla

Tissue retained Y / N | Histology blocks n= | Retained organs Y / N = which one/s
Toxicology Y / N | Microbiology Y / N | Additional data

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