Second Edition

COLOR ATLAS OF FORENSIC MEDICINE AND PATHOLOGY

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Edited by Charles Catanese

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This atlas is dedicated to my family, including my father, S. John Catanese, my deceased mother, Helen J. Amendola-Catanese, and my older siblings, John Catanese PhD, BMF, Anthony Catanese MD, and Gerald Catanese MD.

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Acknowledgments

I thank my father, S. John Catanese, my mother Helen Amendola-Catanese, and my older siblings, John Catanese PhD, Anthony Catanese MD, and Gerard Catanese MD.

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My sincerest thanks to all of you,

Charles A. Catanese

Editor

Charles A. Catanese, MD, completed his medical school education at SUNY Downstate and his residency training at State University Hospital–Kings County Hospital complex in Brooklyn, New York. He is board certified by the American Board of Pathology in anatomic pathology and forensic pathology. Following his forensic pathology fellowship training in New York City, Dr. Catanese was employed as a full-time medical examiner in the Brooklyn office of New York City for more than 10 years. He has performed in excess of 5000 autopsies, including more than 500 homicides, and has supervised many more. As educational coordinator of Brooklyn, he accumulated many academic images demonstrating a wide variety and spectrum of presentation for many forensic topics. The Brooklyn office is the busiest of the five boroughs and New York City has the largest medical examiner's system in the country. Dr. Catanese has also worked as a private consultant for many years and has provided locums coverage in the states of New Hampshire, New Jersey, Tennessee, Rhode Island, Vermont, and New York. He also worked through several disasters, including TWA Flight 800, AA Flight 587, and more than 9 months on the World Trade Center fatalities. He has been the chief medical examiner in the lower Hudson Valley of New York for more than 7 years.

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Sudden Natural Death in a Forensic Setting

1

CHARLES A. CATANESE AND AMY V. RAPKIEWICZ

Contents

Introduction

This chapter offers a brief overview of some common and some not-so-common natural deaths that typically may occur in a medical examiner system. Also demonstrated are examples that may alter the appearance of tissue, such as formaldehyde fixation and variation due to different types of photographic imagery. There are also examples of normal organs in both a fresh and a formaldehyde-fixed state that can be used by the reader to compare with diseased organs.

Deaths under this category are often unexpected, sometimes unwitnessed, and without documented medical history. There may be a suspicion of foul play. Families may say, "But Doctor, he was in fine health. I saw him an hour ago. It cannot be natural. Somebody must have harmed him," etc. Because of the sudden, unexpected nature of these deaths, it is best to do an autopsy to clarify exactly what happened. This decision to autopsy depends on many factors, including the decedent's age, medical history, family wishes, decedent's wishes (wills, etc.), religious beliefs, circumstances at the time of death, resources of a particular system, etc. As one becomes less certain of the cause of death, the level of suspicion will increase. At some point, the decision to autopsy becomes obvious and absolutely necessary. This decision is based on experience, knowledge, and sound judgment. Not infrequently, seemingly natural deaths can have unnatural or traumatic previous circumstances; therefore, when uncertain, an autopsy is best performed. In many medical examiner systems, the majority of deaths end up being certified as natural.

Sudden death may be defined in different ways. It may indicate a death that occurs within 24 hours of the onset of symptoms. It may also indicate a death that occurred within 1 hour or even within seconds. There are not many diseases that can cause death within minutes of the onset of symptoms.

In order to know what is natural, one must be able to differentiate it from other manners of death such as homicide, accident, suicide, undetermined, and possibly therapeutic complication (see Chapter 2). Natural deaths occur exclusively as a result of etiologically specific diseases such as cancer, chronic obstructive pulmonary disease, atherosclerosis, diabetes mellitus, lobar pneumonia, chronic environmental insults, etc. Natural death means the manner of death is exclusively 100% natural. Accidental deaths are caused by violent means, not by intentional or criminal acts of another. Homicides are defined as death at the hands of another, or that occurred during an illegal act. Suicidal deaths that occur as a result of self-murder where the decedent intended to kill themselves. Undetermined are deaths where a reasonable classification is not possible. If there is a 1% component of an unnatural event, which contributed to death, of another manner, it is no longer natural. If there are multiple components of different manners of death identified during a case investigation, the following rule will apply: a homicide overrides all, then an accident, then a therapeutic complication. For example, someone with end-stage metastatic liver cancer ingests 100 acetaminophen tablets to commit suicide. In the process of waiting to die, he decides to walk to a store. On the way, he trips in a pothole, falls in the street, strikes his head, and has an expanding subdural hemorrhage. While he is lying there groaning in pain, waiting for EMS, a stolen car fleeing the scene of a robbery runs him over, lacerates his heart in half, and he dies within seconds. The manner of death in this case would be homicide. The death occurred as a result of being run over by a car during an illegal act. A lacerated heart is universally fatal regardless of the other violent and natural processes. The death certificate should include only the trauma from the car. If the trauma from the car was not lethal by itself, one may add "other finding" to part two of the cause of death, but the manner would remain homicide.

Heart Disease

Heart disease leading to ventricular irritability and fatal cardiac arrhythmia is the most significant cause of death in this category. The most common arrhythmia leading to sudden cardiac death is ventricular fibrillation. Ventricular tachyarrhythmias are most commonly seen within 12 hours of a myocardial infarction. Critical coronary atherosclerosis and hypertension are by far the leading causes of these processes. Some diseases that contribute to atherosclerosis and arteriosclerosis formation include hyperlipidemia, high blood pressure, diabetes mellitus, obesity, cigarette smoking, stress, and sedentary lifestyle.

Having 75% or greater blockage in any of the epicardial vessels is considered critical stenosis and is consistent with being alive 1 second and having loss of consciousness leading to death the next. Hypertensive cardiovascular disease is usually essential in origin from an intrinsic abnormality of sodium metabolism. Other significant causes of hypertension include many types of kidney disease, including adult polycystic kidney disease and renal artery stenosis. Hypertension may be sporadic and missed on routine doctor appointments. High blood pressure is also associated with small-vessel coronary artery disease, as is diabetes mellitus, which is a reasonable cause of death by itself. Once people reach a pivotal point of myocardial irritability and go into ventricular fibrillation, they usually have approximately 15 seconds of consciousness left. Prior to losing consciousness, decedents may reach up to chest or neck and mention a fluttering sensation in the chest. They may have pressure, pain, or no expectation of what is to come. Ventricular irritability associated with coronary artery ischemia is due to lack of oxygen and nutrients reaching the conducting system of the heart. If the heart is not cardioverted back to a normal rhythm within 4–6 minutes, there is usually irreversible brain damage.

Another major cause of ventricular irritability leading to fatal arrhythmia is hypertension. Concentric left ventricular hypertrophy, usually defined at autopsy as having a left ventricular wall thickness greater than 1.5 cm for most average-sized adults, is a known risk factor for sudden cardiac death. Left ventricular thickness is best measured approximately 2 cm below the mitral valve annulus and excludes trabeculations and papillary muscles. As the disease process causing cardiac hypertrophy advances, heart failure may ensue with chamber dilatation. Although the overall heart size is enlarged, the left ventricle wall thickness may be less than 1.4 cm in cases of cardiac chamber dilatation with wall thinning due to failure. Although hypertensive cardiovascular disease is the major risk factor for the development of left ventricular hypertrophy, other risk factors include aortic stenosis, either congenital or acquired. The hearts of patients with hypertensive or arteriosclerotic cardiovascular disease typically show evidence of prior infarction and interstitial fibrosis. Both findings also predispose to abnormalities in the conduction system, predisposing to myocardial irritability and fatal arrhythmias.

Complications other than tachyarrhythmia and pump failure of myocardial infarctions can result in sudden cardiac death; the most common include the myocardial rupture syndromes, including ventricular wall and papillary wall rupture. Typically, these insults occur approximately 1 week following a myocardial infarction, the point at which there is removal of necrotic myocytes by macrophages. Hemopericardium with ensuing cardiac tamponade can occur following ventricular free wall rupture; this scenario is rapidly fatal in most cases, causing decreased venous return to the heart with jugular venous distention.

In young patients, hypertrophic cardiomyopathy is a common cause of sudden death. Fatal arrhythmia occurs during or following exercise. These patients can be asymptomatic prior to the sudden event or may have past episodes of palpitations or syncope. Typically, in the classical type, macroscopic heart evaluation shows cardiac hypertrophy with significant asymmetry of the subaortic septal region, which poses as an outflow obstruction. Microscopic sections from this region show variable degrees of myocyte disarray, fibrosis, myocyte hypertrophy, and small-vessel disease. The disease is due to an autosomal dominant mutation in the cardiac sarcomere apparatus, most commonly the myosin heavy chain, but many mutations have been described. As this disease process becomes more defined, it has been described with a spectrum of change ranging from asymmetry of the left ventricle, in classical presentation, to the rarely described variant with no cardiac hypertrophy at all.

Arrhythmogenic right ventricular cardiomyopathy can present with sudden unexpected death. At autopsy, the right ventricle is thinned, with microscopic evaluation showing significant transmural infiltration by fibrofatty tissue.

Myocarditis due to a variety of causes, including viral, bacterial, fungal, parasitic, autoimmune, and hypersensitivity, can present as sudden death. The degree of activity, myonecrosis, and the location of the inflammation (i.e., conduction system involvement) are important in determining the significance of the infiltrates. Notably, eosinophils are seen quite commonly in hypersensitivity myocarditis, which is a delayed type for hypersensitivity reaction and can be a clue to the underlying etiology, which begins around blood vessels and tracks into the parenchyma. The origin of this process is thought to be mostly autoimmune where the immune system produces an immune response against heart muscle components.

Some viruses have been identified in myocardial cultures, such as coxsackievirus. The presence of giant cells indicates chronicity of infection and should not lead to mistaken diagnosis of giant cell myocarditis. Giant cell myocarditis is associated with extensive necrosis and high mortality rate, and is of unknown etiology.

Dilated cardiomyopathy is common, and has many etiologies that include idiopathic arteriosclerotic disease, hypertensive cardiovascular disease, alcoholism, elevated catecholamines, myocarditis, postpartum, doxorubicin, endocrinopathies, and genetic diseases. The heart typically is enlarged with a globoid configuration. The microscopic analysis shows interstitial fibrosis.

Rare infiltrative cardiac diseases such as amyloidosis, hemochromatosis, primary or metastatic tumors, and sarcoidosis can result in sudden death. Microscopic evaluation in these cases is necessary, with particular attention to nodal tissues.

Staphylococcus aureus is the most common organism found in infective endocarditis (IE). *S. aureus* endocarditis is associated with the highest mortality and risk of embolism. Increasing age, periannular abscess, heart failure, and absence of surgical therapy were identified in multivariate analysis as independent poor prognostic factors for increased mortality in patients with *S. aureus* IE. Other risk factors for the development of IE include congenital or acquired anatomic valve abnormalities such as stenosis. Impaired cardiac conductivity and function with heart failure not infrequently develop in patients with multiple septic myocardial emboli and infarcts due to IE, particularly with paravalvular abscess formation. According to a recent study of a cohort of 606 cases of IE, 99 cases have embolization, of which 32 cases involve the central nervous system (CNS) with significantly higher mortality (65%) than those without CNS emboli.

Recently, genetic abnormalities have been found to underlie many of the intrinsic abnormalities of conducting systems, including Wolff Parkinson–White syndrome (WPW) and long Q-T syndrome. Sudden death in WPW is thought to occur as a result of an induction of ventricular tachycardia via an atrioventricular re-entry pathway. Long-QT syndrome can also present with sudden death. Investigations are ongoing around the association of sudden infant death syndrome with long-QT syndrome. Recent data suggest a genetic basis for the arrhythmogenic disease with the identification of the long-QT genes.

Sudden death related to cardiac valve pathology other than endocarditis is relatively uncommon, as valve replacement surgery has become a standard therapy. Patients with aortic stenosis, especially when acutely symptomatic, can experience sudden cardiac death. Most cases of aortic stenosis are caused by either rheumatic heart disease or valve calcification, which can occur on trileaflet or congenitally (uni)bicuspid valves. The mechanism for death in severe aortic stenosis (valve area <1 cm^2) appears to be through left ventricular hypertrophy and subsequent myocardial instability. In rare instances of severe aortic valve calcification, the deposits can erode the region and involve the conduction system. Mitral valve prolapse has long been associated with sudden cardiac death. The underlying etiology is not well understood, but seems to most frequently involve a severe valve deformity with a redundant, thickened, myxomatous mitral valve and ventricular arrhythmias such as ventricular fibrillation. On histologic sectioning, the mitral valve will show deposition of acid mucopolysaccharides.

Coronary artery anomalies are not uncommon but only certain anomalies result in ischemia, such as anomalous origin of a coronary artery from the opposite sinus (ACAOS), anomalous left coronary artery from the pulmonary artery (ALCAPA), ostial atresia/stenosis, and coronary artery fistulas. Left-sided ACAOS can result in acute takeoff angles with an increased risk of sudden death during or shortly after exercise. Besides the acute angle takeoff, there may be ridgelike defect at the coronary ostia, further decreasing blood flow in times of accelerated heart rates with increased oxygen demand. Myocardial tunneling is another anomalous coronary artery distribution that may be associated with increased arrhythmogenic potential. There is debate about the significance of this anomaly. Some still believe it may be significant when a large portion of the epicardial coronary artery dips deeply into the left ventricle wall for a considerable distance, during times of rapid muscle contraction.

Vascular Disease

Causes of sudden death associated with vascular disease include those that lead to occlusion, narrowing, or rupture of a blood vessel. Atherosclerotic aneurysms can rupture, leading to rapid loss of consciousness and death. These aneurysms can occur just about anywhere, but are by far most common in the abdominal aorta. Most abdominal aortic aneurysms occur below the renal artery. The risk of rupture increases with the size of the aneurysm, smoking history, and hypertension. The annual risk of rupture over 7 cm in size is 33%. Retroperitoneal rupture is typically associated with hematoma formation, whereas rupture into the abdominal cavity can be rapidly fatal, with hemoperitoneum and shock. Patients who have a ruptured aortic aneurysm and reach the hospital have a 50% mortality rate, with the overall mortality rate greater than 85%.

Aortic dissection is characterized by an intimal tear followed by a dissection of blood within the wall of the aorta, most commonly the tunica media. Rupture of this

dissecting aortic hematoma may lead to hemothoraces, hemopericardium, or fatal arrhythmia. Aortic dissection is a major cause of sudden death, mostly in patients over 50 years of age with the underlying risk factor being essential hypertension. However, pregnant women and patients with connective-tissue diseases such as Marfan syndrome also make up a significantly affected patient population. Aortic dissection can also occur following accidental or iatrogenic trauma to the aortic intima. In younger patients and those with connective-tissue disease, microscopy may reveal cystic medial degeneration of the aortic media.

Most spontaneous subarachnoid hemorrhages (SAH) (90%) are caused by ruptured intracranial saccular (berry) aneurysms. SAH occurs at a peak age of 55–60 years. Rupture of an intracranial aneurysm is believed to account for 0.4%–0.6% of all deaths. SAH is associated with a greater than 50% mortality rate. Some hospital-based studies suggest that approximately 10% of patients with aneurismal SAH die prior to reaching the hospital, 25% die within 24 hours of SAH onset, and about 45% die within 30 days. It is not unusual to perform forensic autopsies where death was almost instantaneous and outside of a hospital. The mechanism of death in such cases is cardiac arrhythmia, which is described in greater depth later. Most intracranial aneurysms (approximately 85%) are located in the anterior circulation, predominately on the circle of Willis. Risk factors for both SAH and intracranial aneurysms are similar and include hypertension, cigarette smoking, and alcohol consumption. Atherosclerosis is an independent risk factor for the development of intracranial aneurysms. The natural history of subarachnoid hemorrhage shows that rupture often occurs when they reach a size over 7 mm. Rupture of an aneurysm releases blood directly into the cerebrospinal fluid (CSF) under arterial pressure. The blood spreads quickly within the CSF, rapidly increasing intracranial pressure. A major symptom associated with SAH includes patients describing the worst headache of one's life. Increased intracranial pressure is associated with the Cushing's triad (hypertension, bradycardia, and abnormal respiration). SAH is associated with cerebral edema and subsequent herniation. Tonsillar and central transtentorial herniation is associated with compression of cardiovascular and respiratory centers in the medulla and, as such, is rapidly fatal. Other less common causes of SAH include angiomas and arteriovenous malformations. Ruptured berry aneurysms are the most common natural cause of SAH, whereas trauma is the most common overall cause. Ruptured berry aneurysms are a leading cause of sudden death in women during sexual activity, whereas for men, it is heart disease.

Cerebrovascular accidents (episodes), which include ischemic or intracerebral hemorrhage, can lead to sudden death. I recommend not using the term "accident" because there is nothing accidental about this process and its use often adds confusion in forensic proceedings. The term "stroke" or "event" as an alternative is less confusing to nonmedical personnel. Thromboembolic events can underlie ischemic cerebral events and are associated with heart disease, valvular pathology, or carotid artery disease. Hypertension is a major risk factor for intraparenchymal hemorrhage and may lead to increased intracranial pressure, herniation, and death.

The greatest percentage of thrombi resulting in pulmonary embolism is thought to originate in the deep veins of the lower extremities. Deep venous thrombosis can also occur in the pelvis or other locations. Fragments of blood clot may break off and embolize to the pulmonary arteries. An occlusion greater than 50%–75% of the large pulmonary vessels results in a rise of the pulmonary artery pressure greater than 40 mmHg. This rise of pulmonary arterial pressure is accompanied by an increase in right ventricular diastolic, right atrial, and systemic venous pressures, with a decrease in cardiac output resulting in sudden death. Patients who have multiple small pulmonary emboli or in situ thrombus formation over time may present with increasing shortness of breath and right-sided heart failure. Because the lungs have dual circulation, infarctions are less common unless there is significant underlying natural disease with decreased cardiac function.

Various types of vasculitis or blood vessel inflammation can cause wall thickening, thrombosis, dissection, and rupture. Mesenteric thrombosis may be associated with polyarteritis nodosum and other autoimmune conditions.

Other Causes of Sudden Death

Rare undiagnosed brain tumors may present with sudden death. The mechanism may be infiltration, or edema formation, into the key respiratory/cardiac centers of the brain, with possible herniation. Early- or late-stage malignancies may sometimes metastasize to the heart and interfere with the conducting system, causing a fatal arrhythmia. Other causes of sudden death in patients with malignancies include cardiovascular events such as acute myocardial infarction, therapeutic complications (i.e., anaphylaxis), and metabolic derangements. Rare causes of sudden death in patients with tumors or malignancies include erosion of large vessels or visci with fatal hemorrhage. A colloid cyst of the third ventricle may lead to sudden death and is usually associated with premortem postural headaches. In certain positions, the cyst will act like a ball valve and suddenly

block the flow of cerebral spinal fluid, resulting in acute obstructive hydrocephalus. One may be fine standing but develop symptoms when he or she lies down. This buildup of cerebral spinal fluid pressure can cause a fatal arrhythmia. Bacterial pneumonia with the combination of hypoxia and bacterial toxins and end products can cause sudden death.

Status asthmaticus and sudden asphyxic asthma are life-threatening forms of asthma. These cases are not unusual in a forensic setting. Status asthmaticus is defined as an acute attack of respiratory failure due to airway inflammation, edema, and mucous plugging. Sudden asphyxic asthma is due to bronchospasm rather than airway inflammation. Viral infections and other causes have been implicated as precipitants of these potentially fatal complications. Grossly in both cases, the lungs may appear so much hyperaerated that, at times, rib indentations will show. Thick mucous plugs may obstruct the upper airways. Sudden death in asthmatic patients is thought to be secondary to fatal arrhythmia, occurring as a consequence of global hypoxia and right-sided heart failure.

There is a condition known as sudden unexpected death in epilepsy (SUDEP). The mechanism is unclear, but this phenomenon occurs in up to 18% of patients with epilepsy, presumably in those with subtherapeutic levels of anticonvulsants. Autonomic dysfunction has been proposed as a mechanism. Other mechanisms for death in patients with epilepsy include accidental/traumatic incidents such as drowning and choking that occur during a seizure. Hypoxia as a result of respiratory compromise can result in ischemic cardiac events. This may be part of the final mechanism of death in epileptic patients experiencing status epilepticus. Another interesting point to remember is that there is often very rapid rigor mortis formation in deaths directly following static epilepticus due to substantial adenosine triphosphate (ATP) depletion associated with prolonged muscle contractions from prolonged convulsions. Usually there are few pathologic findings that explain the sudden death in epileptic patients. Autopsy findings may include bite marks to the tongue with hemorrhage or a voided urinary bladder. There may be no finding at all. These are nonspecific findings, and seizure activity may also occur prior to many other nonepilepsy-related deaths.

Fatal anaphylaxis can result from exposure to insect stings, foods, latex, drugs, chemicals, and exercise. This mast cell-mediated systemic reaction results in severe angioedema and bronchoconstriction of the upper respiratory tract along with hypotension, resulting in respiratory and circulatory collapse. Death caused by anaphylaxis is primarily due to airway obstruction when laryngeal edema fills the rich lymphatic supply of the epiglottic folds. Increased mast cell tryptase levels in the patient's serum can be detected that peak approximately 15–60 minutes after the onset of anaphylaxis and then decline with a half-life of about 2 hours.

The mortality for gastrointestinal bleeding (GI) in the case of ruptured esophageal varices, most commonly encountered in patients with portal hypertension, is high. Intra-aortic balloon pumps are lifesaving procedures but only if the patient presents in a timely fashion. Other causes of fatal upper GI include stomach and duodenal ulcers; in this scenario, the source is arterial as opposed to venous in esophageal varices. Fatal lower GI can be seen in patients with angiodysplasia, diverticulitis, and carcinoma; however, this scenario is less common than upper GI bleeding.

Mostly complications of morbid obesity are thought to underlie the association with sudden death. Hypertension, left ventricular hypertrophy, and cardiomegaly are all independent risk factors for sudden death. Postural asphyxia may occur as a result of obesity. Morbid obesity is a reasonable cause of death by itself due to stress on the heart. An individual who is three times the expected body weight has roughly three times the vasculature with three times the blood volume to pump. In times of other stress, this can have devastating consequences on the heart, with death by arrhythmia.

Waterhouse–Friderichsen syndrome was first described as occurring in patients with meningococcemia, and is characterized by severe bacteremia and bilateral adrenal hemorrhages. This combination results in overwhelming shock and, if untreated, sudden death can occur. Organisms other than *Neisseria meningitidis*, such as *Escherichia coli*, have been reported to produce this syndrome.

Multiorgan failure and death can be seen in sickle cell anemia patients with an acute crisis. Precipitants may include infection, dehydration, hypoxia, physical excretion, vaso-occlusion, or fat embolus following bone infarction. This acute hemolytic sickling crisis results in severe hypoxemia with end organ failure. Patients with sickle cell anemia have auto-infarcted spleens and are much more susceptible to encapsulated organisms such as pneumococcal bacteria. Even patients with sickle cell trait may develop crisis in times of great physical exertion with dehydration, such as basic training in the army or boot camp.

Natural disease processes may weaken the body, making fatal traumatic injury more likely. Osteoporosis from aging, Cushing syndrome, steroid use, and other natural disease processes will make bones more fragile and allow fractures to occur more easily.

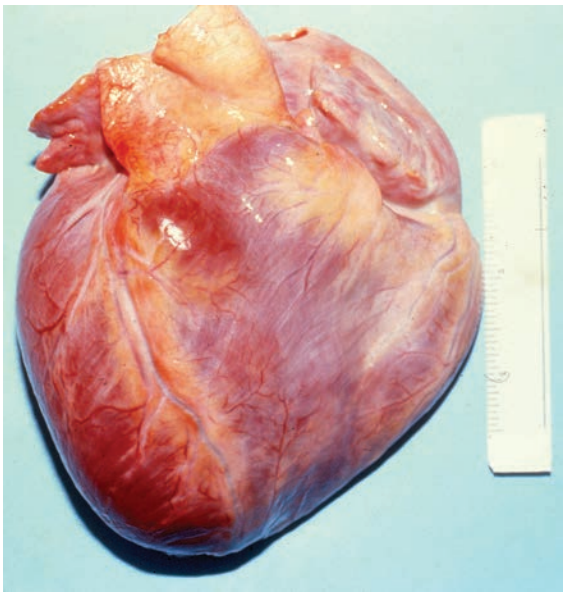


Figure 1.1 Normal heart from a recently deceased individual without formaldehyde fixation.

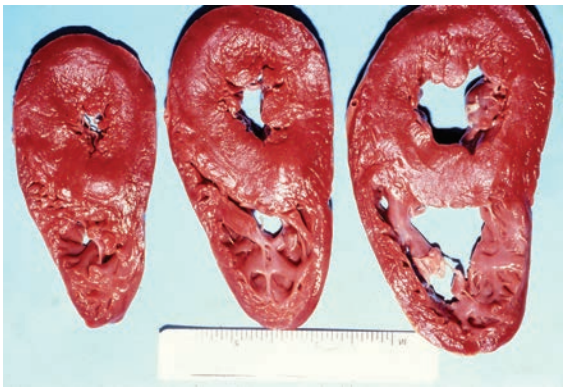


Figure 1.2 Sections of a normal heart from a recently deceased individual without formaldehyde fixation showing right and left ventricles.

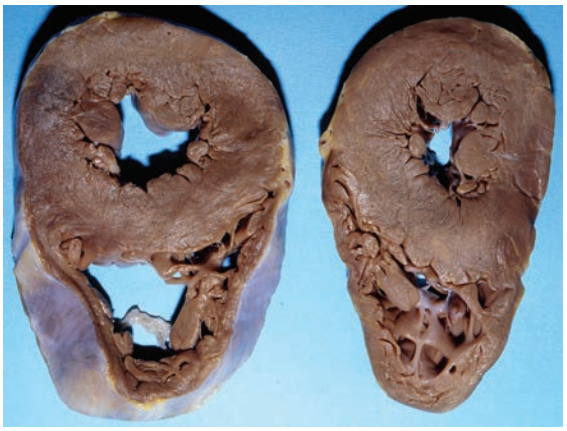
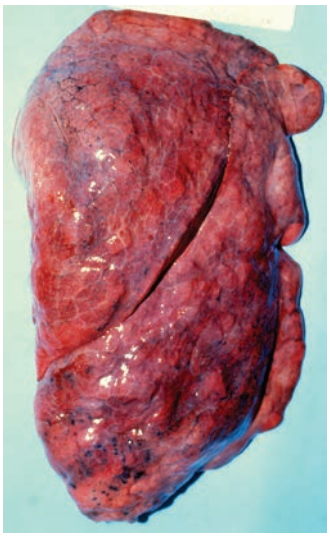
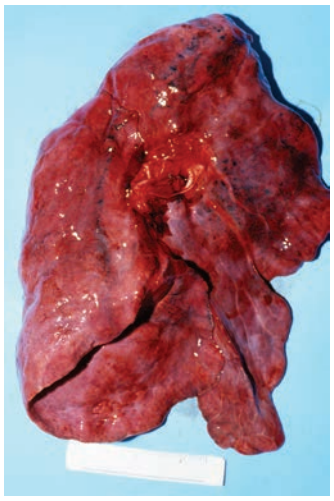


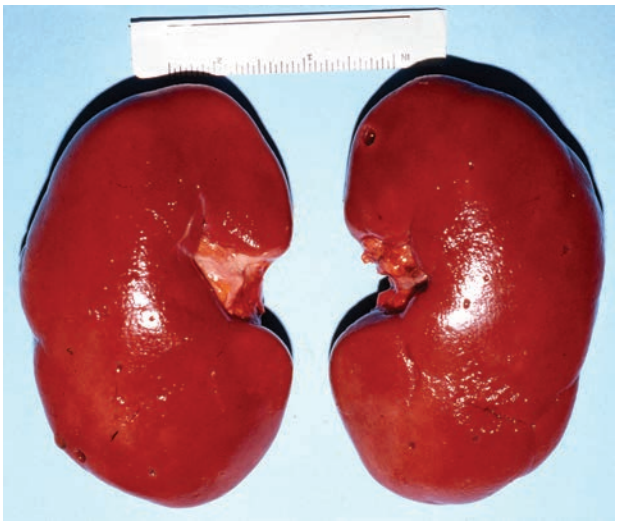
Figure 1.3 The same larger two sections, as depicted in Figure 1.2, after formaldehyde fixation, showing gray discoloration and slightly shrunken firm parenchyma.

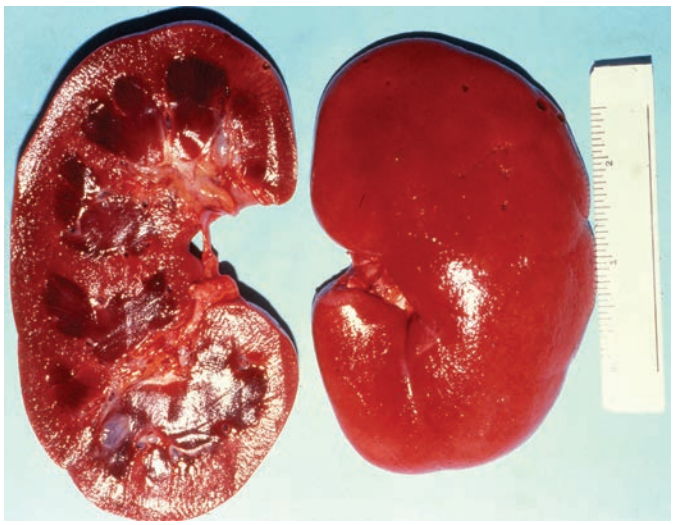




Figures 1.4 and 1.5 Normal left lung demonstrating two lobes from a recently deceased individual without formaldehyde fixation. Right lungs have three lobes.

Figure 1.6 The same lungs, as in Figures 1.4 and 1.5, after formaldehyde fixation, showing gray discoloration and slightly shrunken firm parenchyma.





Figures 1.7 and 1.8 Normal kidneys.

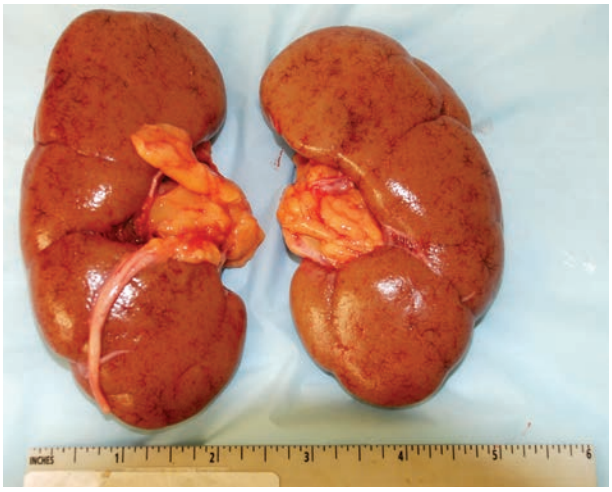


Figure 1.9 Nonfixed kidneys. Note the pale discoloration resulting from fatal blood loss prior to death due to a gunshot wound.

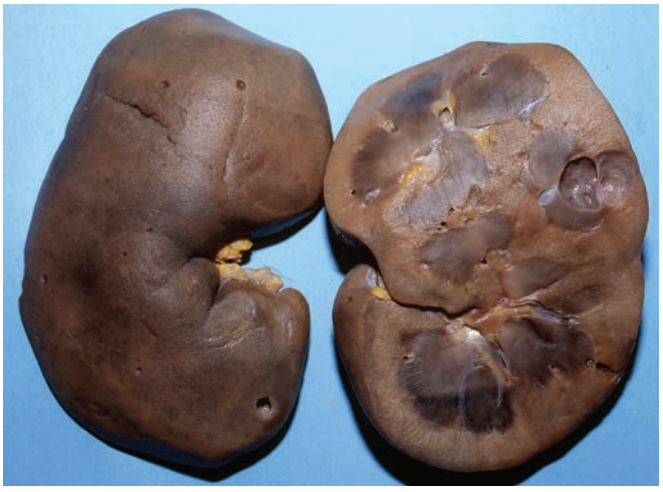
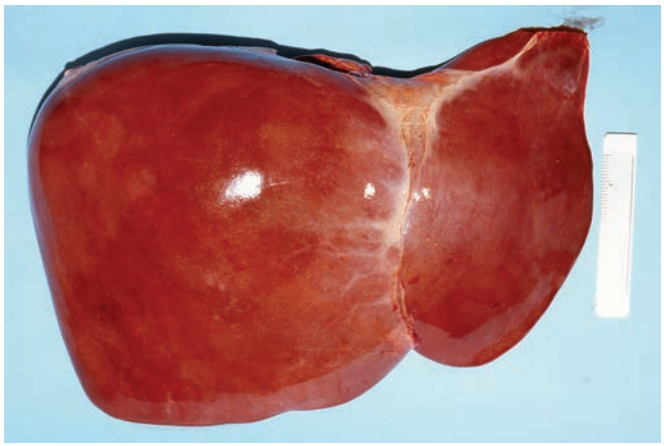
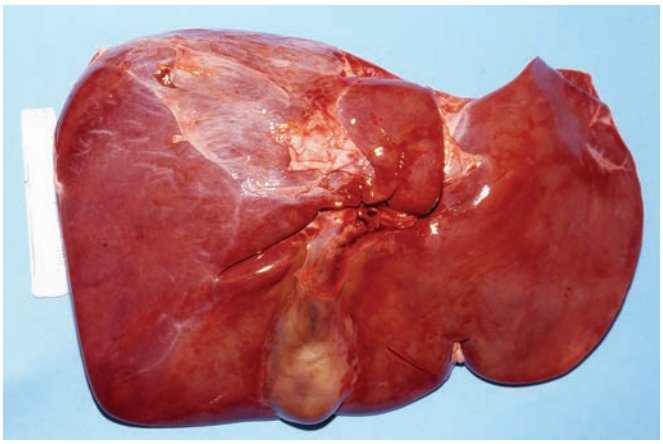


Figure 1.10 Normal bisected kidney fixed in formaldehyde.





Figures 1.11 and 1.12 Normal liver.

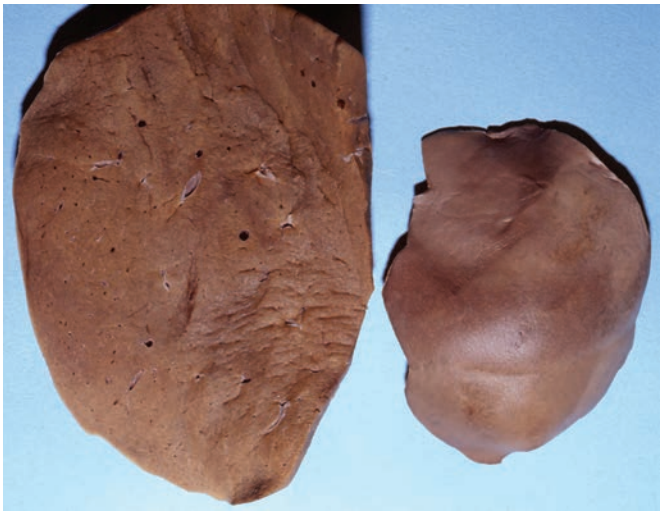


Figure 1.13 Normal liver fixed in formaldehyde.

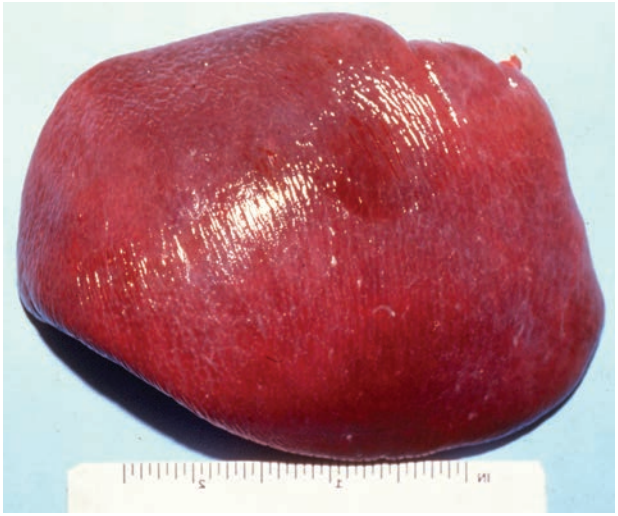


Figure 1.14 Normal spleen.

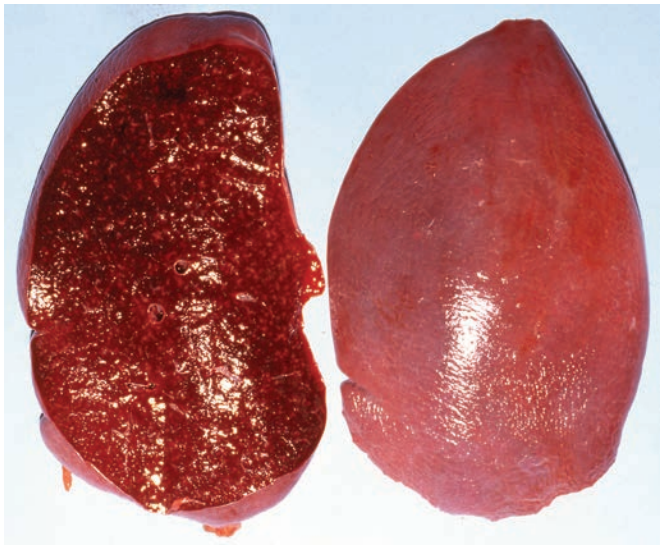
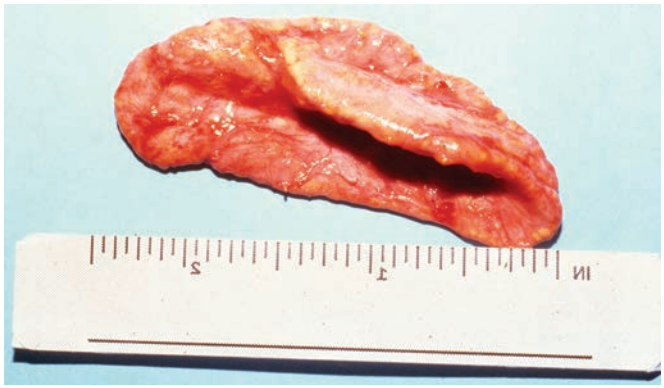
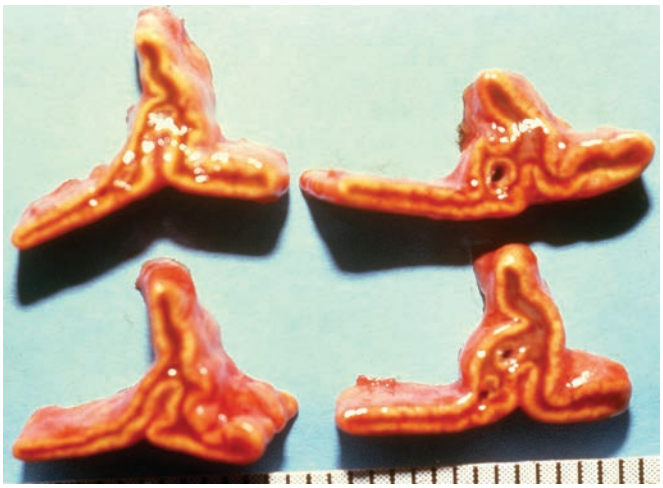


Figure 1.15 Normal spleen.

Figure 1.16 Normal spleen fixed in formaldehyde.





Figures 1.17 and 1.18 Normal adrenal gland intact and sectioned.

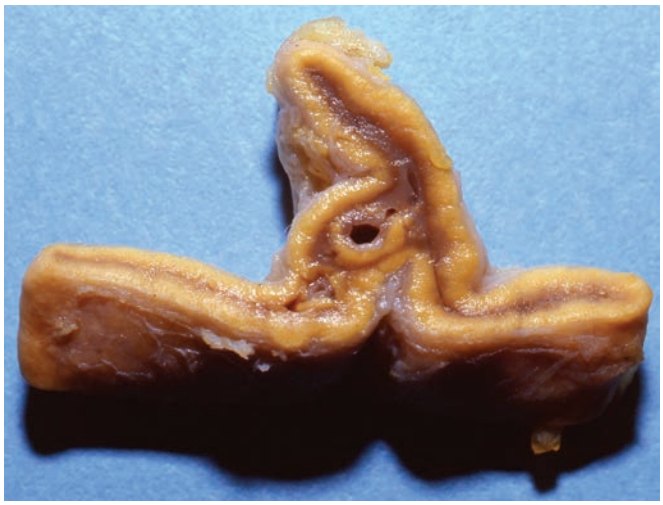


Figure 1.19 Normal adrenal gland section fixed in formaldehyde.

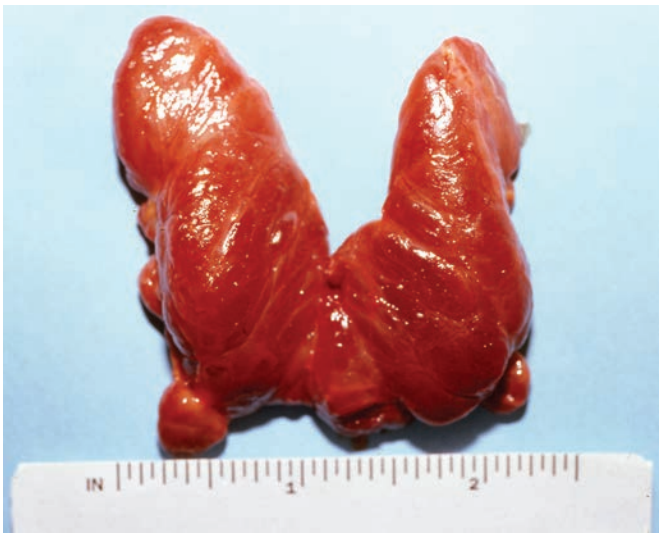


Figure 1.20 Normal thyroid gland.

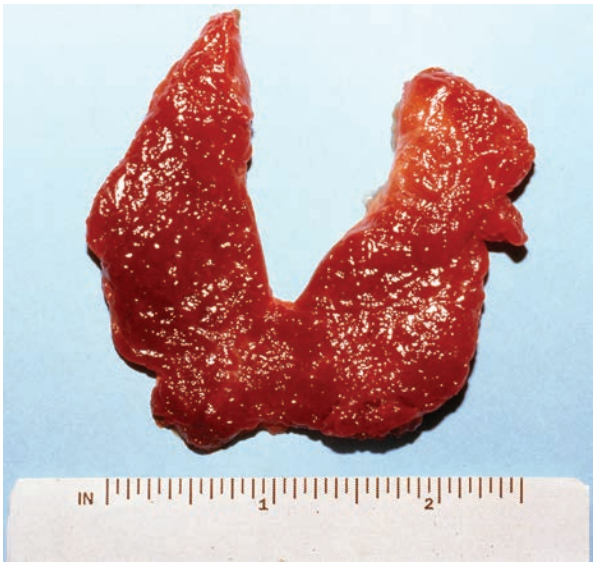


Figure 1.21 Normal thyroid gland.

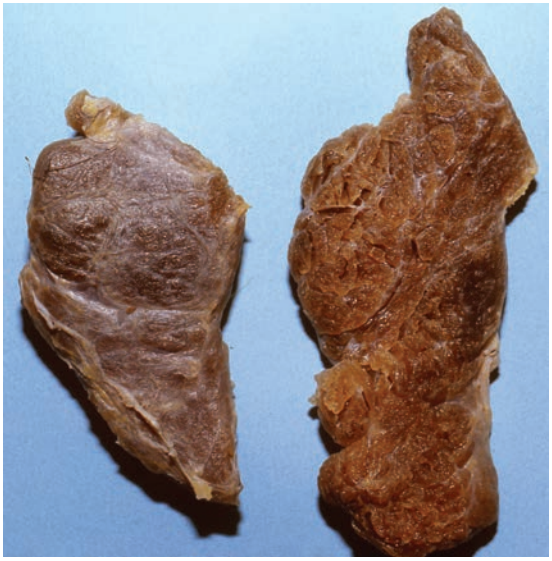
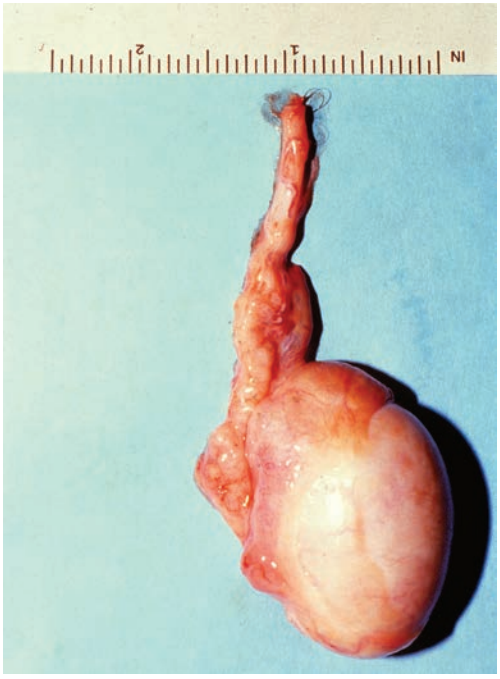
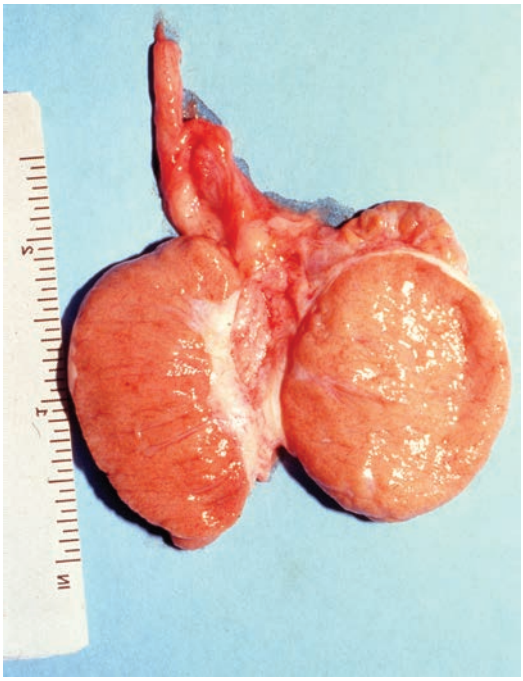


Figure 1.22 Normal bisected thyroid gland fixed in formaldehyde.





Figures 1.23 and 1.24 Normal testes.

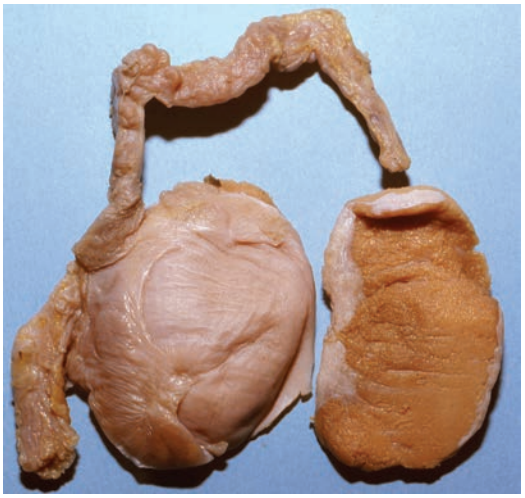


Figure 1.25 Normal testes fixed in formaldehyde.

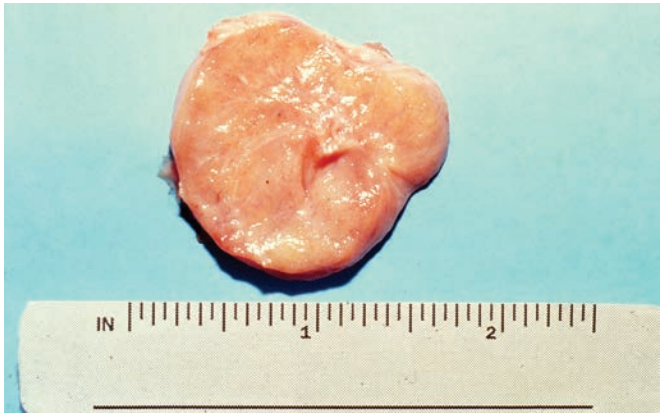


Figure 1.26 Normal prostate.

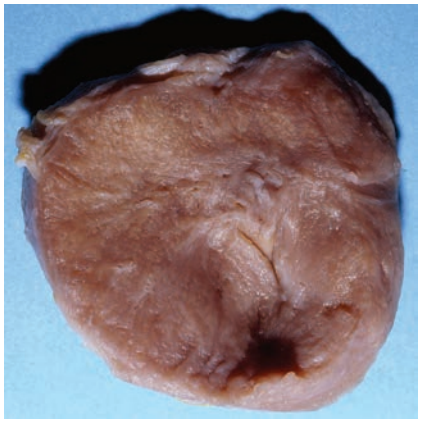


Figure 1.27 Normal prostate fixed in formaldehyde.

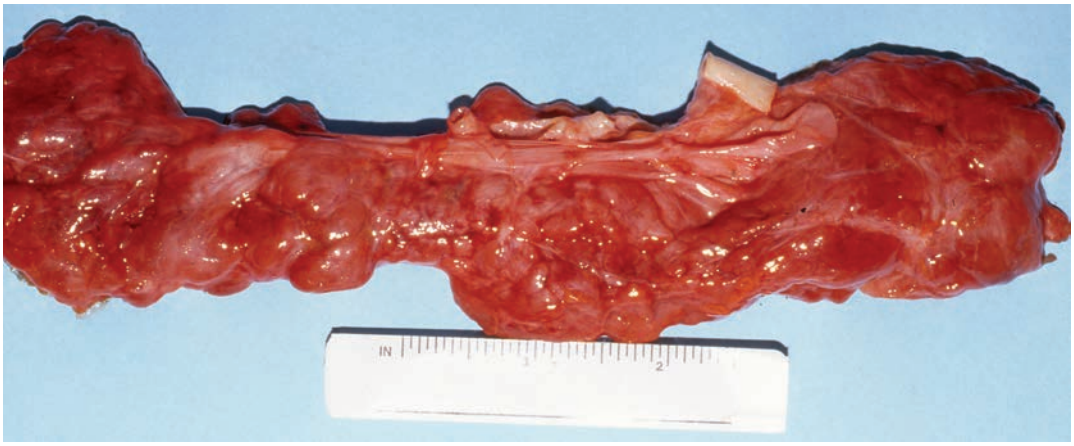


Figure 1.28 Normal pancreas.

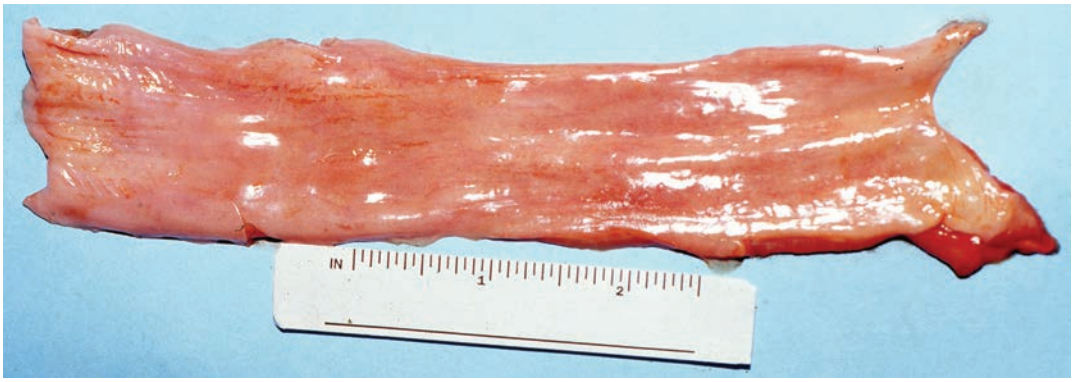


Figure 1.29 Normal esophagus.

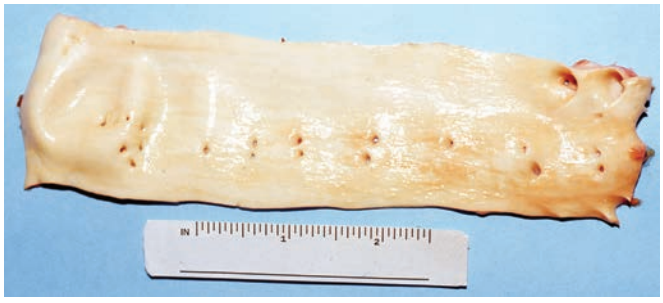


Figure 1.30 Aorta with slight atherosclerosis. Note the fatty streaks on the intimal surface from this individual in their early 20s.

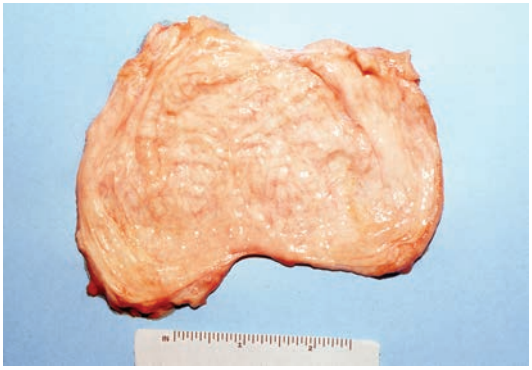


Figure 1.31 Normal bladder.

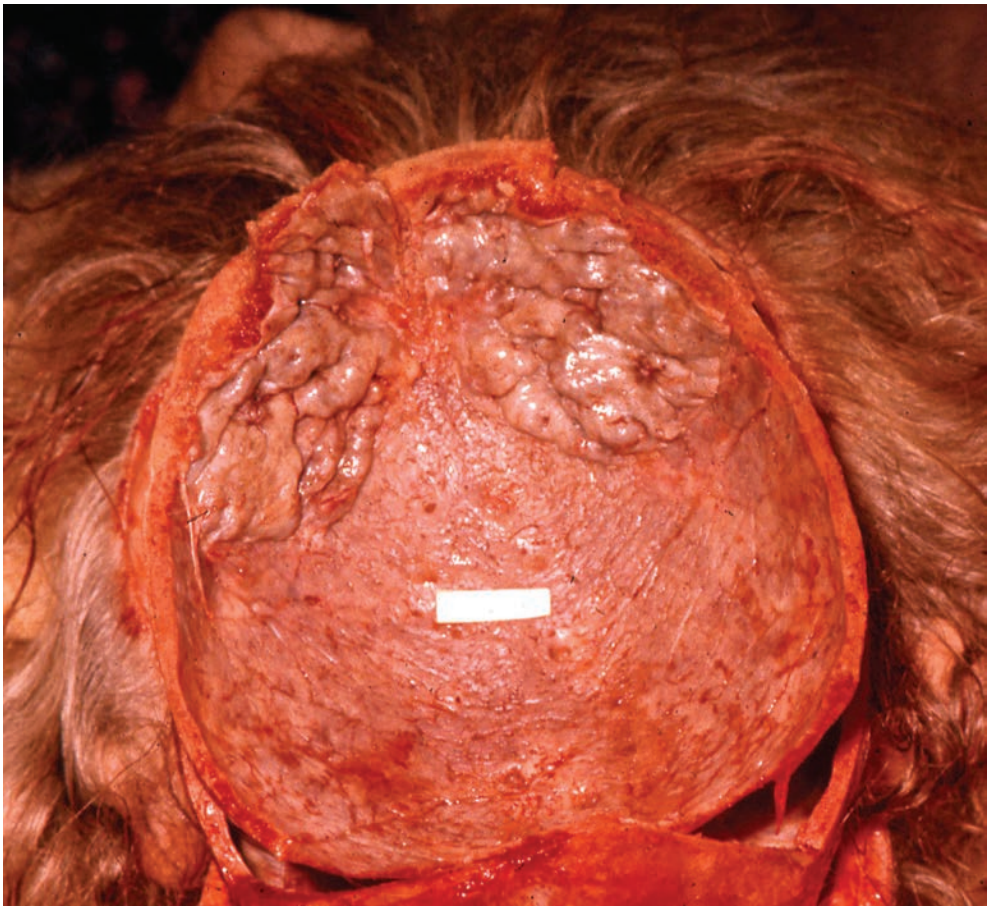


Figure 1.32 Hyperostosis frontalis is a benign process characterized by irregular thickening of the internal frontal bone that may help with identification.

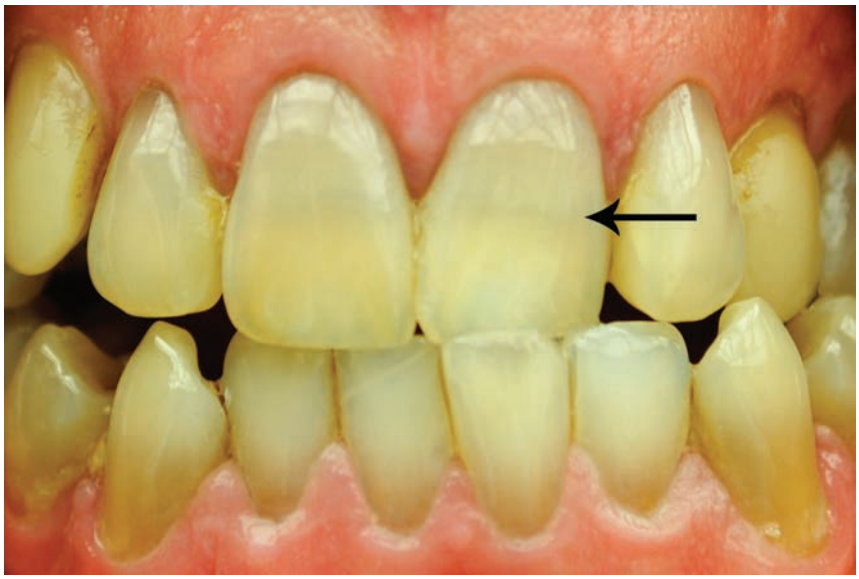
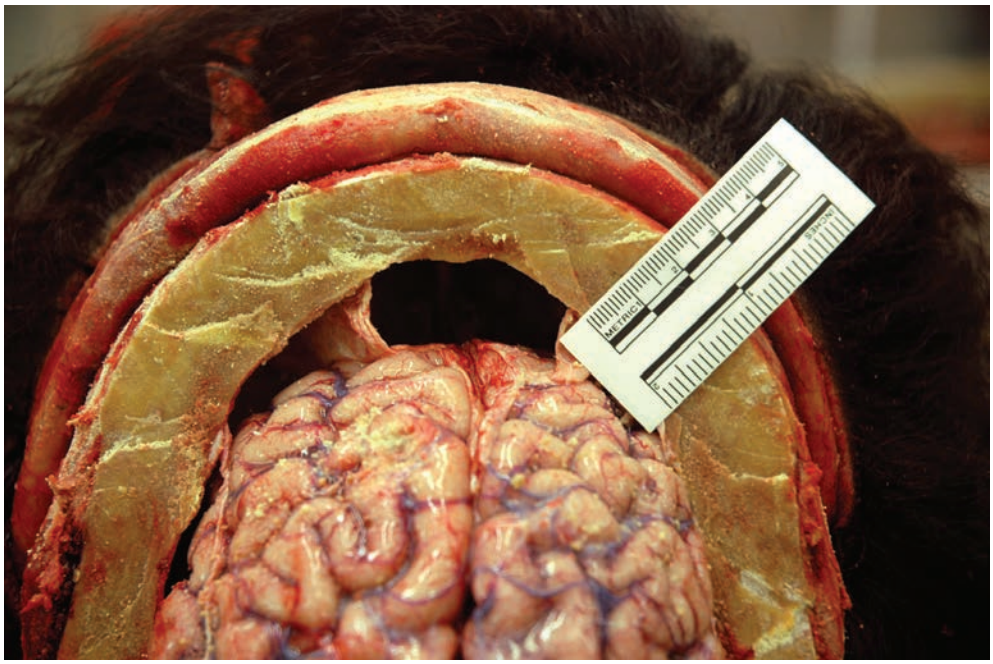
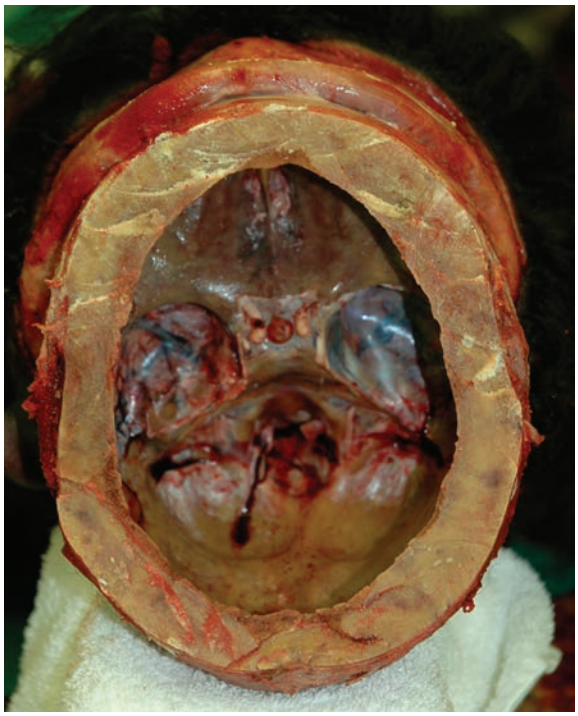
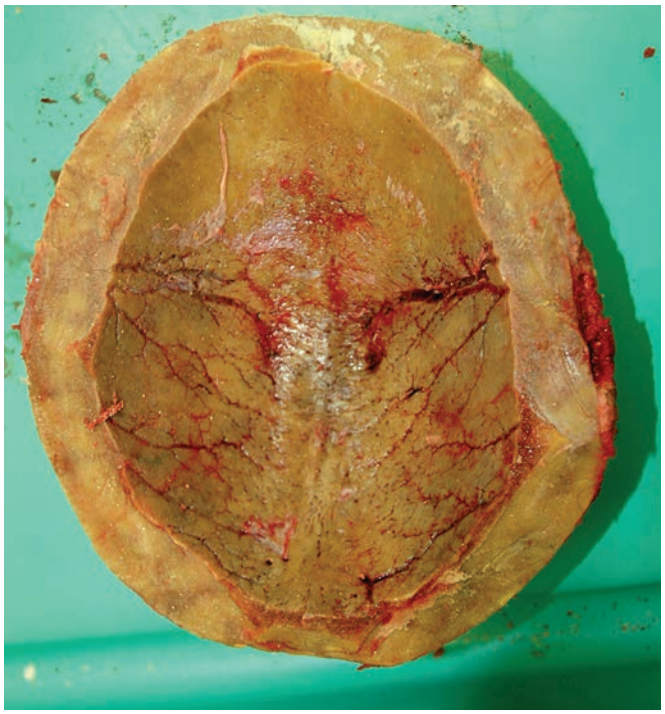


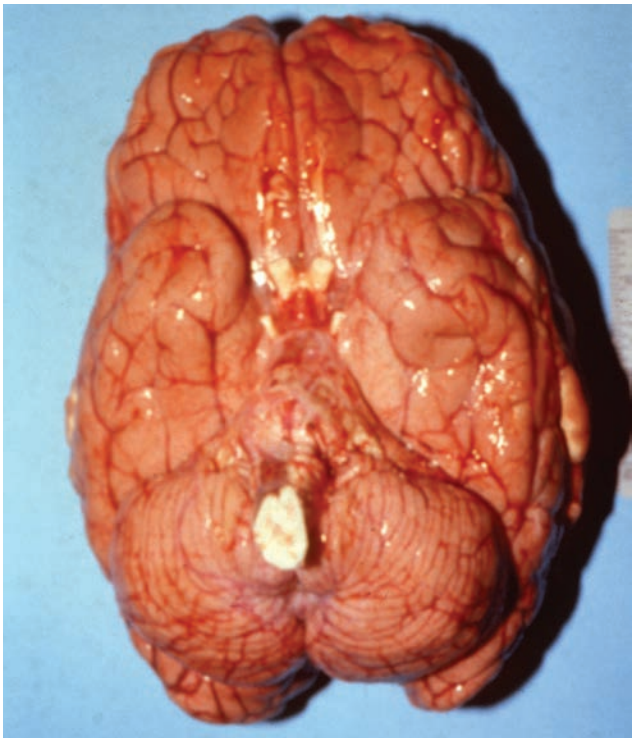
Figure 1.33 Tetracycline staining of the teeth due to exposure to this antibiotic as a child. This discoloration may be helpful as a feature for identification when other modalities are not possible. This discoloration, superior to the arrow, occurred during childhood when the teeth were still forming.

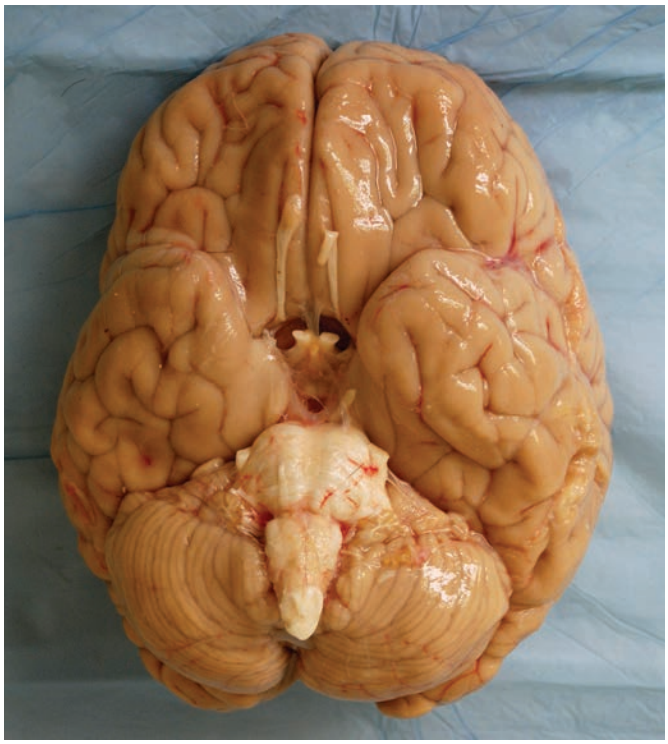






Figures 1.34–1.36 Paget disease is a disease involving the accelerated breakdown and formation of bone. Note the markedly thickened skull wall, which occurs gradually over time. See Figures 11.123–11.125 in the histology chapter.





Figures 1.37 and 1.38 The first picture demonstrates congestion of non-formaldehyde-fixed brain. Note the slight pink color. The second picture demonstrates non-formaldehyde-fixed brain in an individual who exsanguinated from a ruptured aortic aneurysm. Note the pale discoloration due to blood loss.

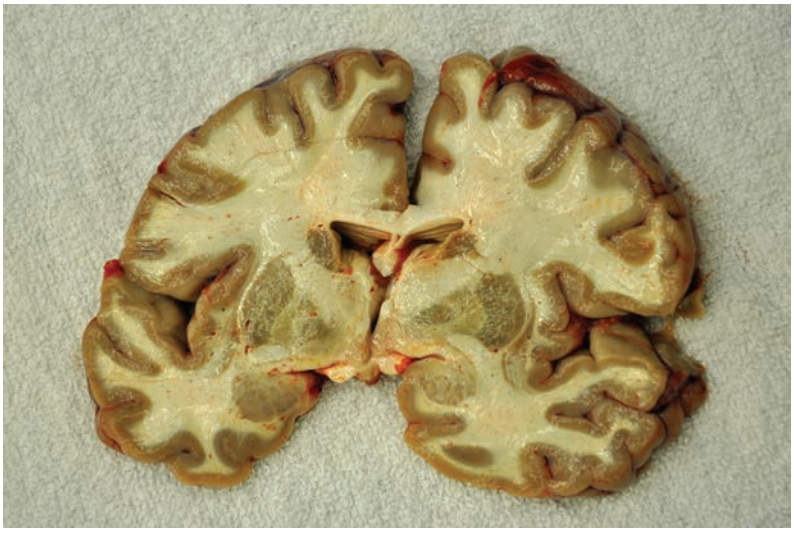


Figure 1.39 Note the green discoloration of the brain section from a person who died of hydrogen sulfide poisoning.

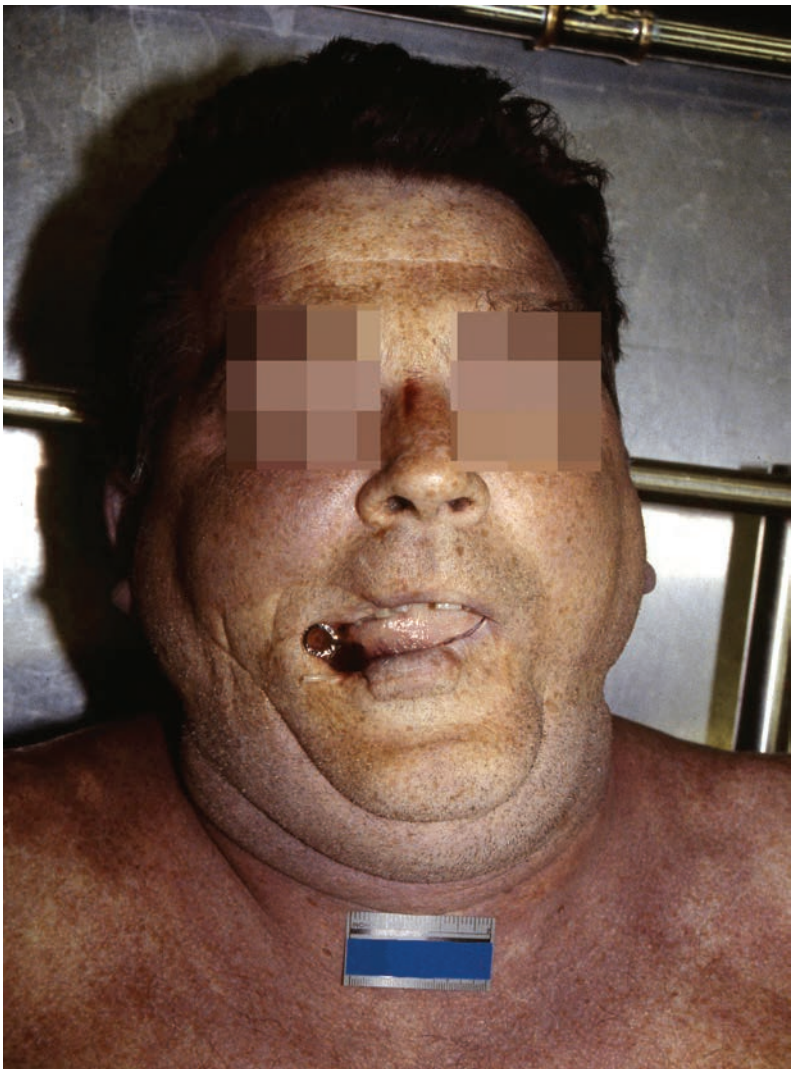


Figure 1.40 This picture depicts a decedent with an endotracheal tube in the right side of his mouth. It also demonstrates the "purple head sign," a common finding in victims of sudden death, particularly cardiac death. The explanation for this finding is not known in entirety but is attributed to uncontrolled terminal sympathetic nervous system discharges, which open free capillary sphincters and produce a gush of capillary blood.

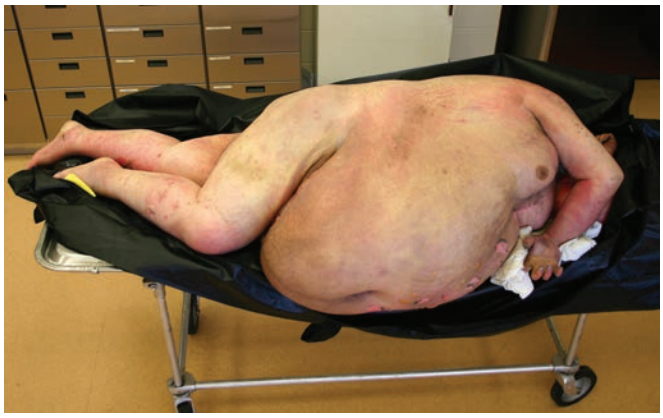


Figure 1.41 Morbid obesity. This is a legitimate cause of death and can stand alone on a death certificate.

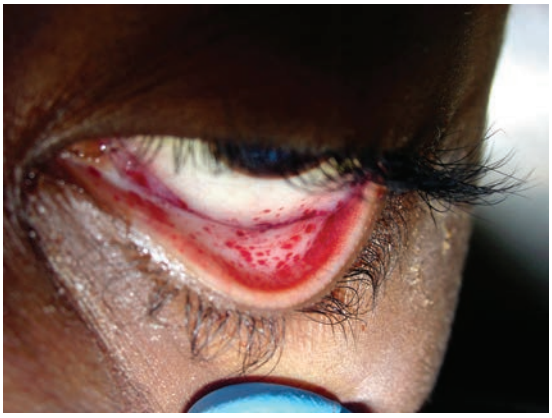
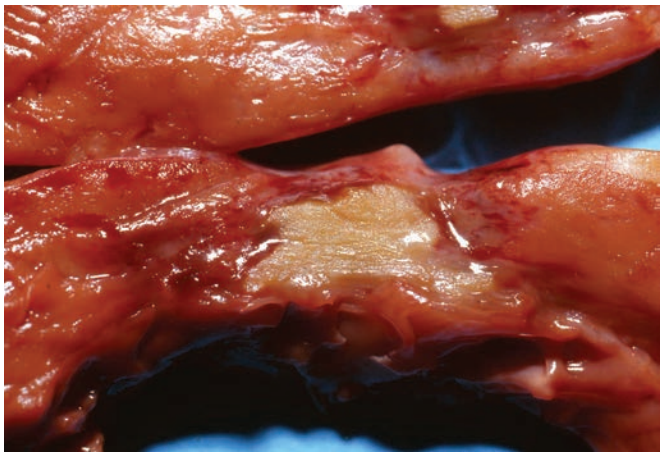
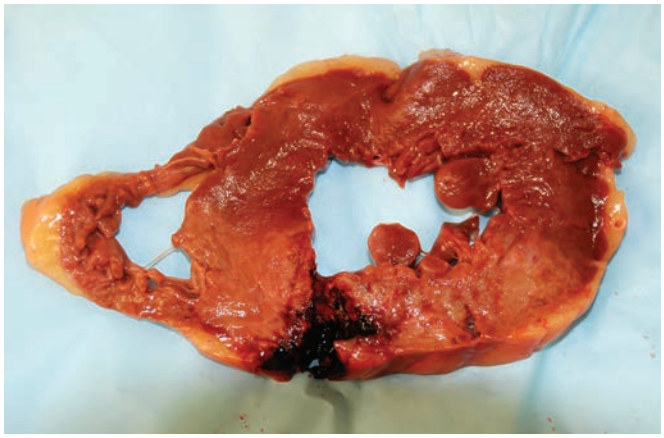
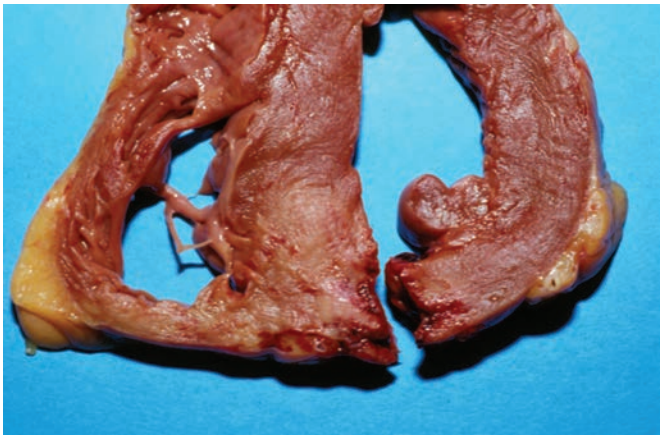


Figure 1.42 Petechiae associated with heart disease and resuscitation.







Figures 1.43–1.45 An acute myocardial infarction. Note the yellow discoloration due to necrosis. This infarction is approximately 3–6 days old. In Figure 1.45, there is a transmural acute myocardial infarction with rupture.

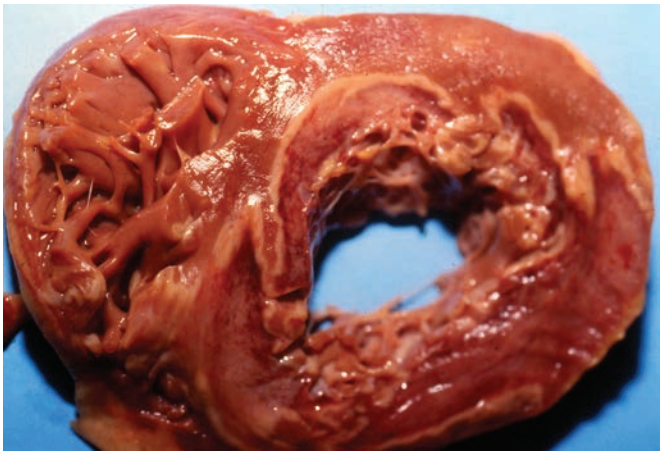


Figure 1.46 Acute myocardial infarction.

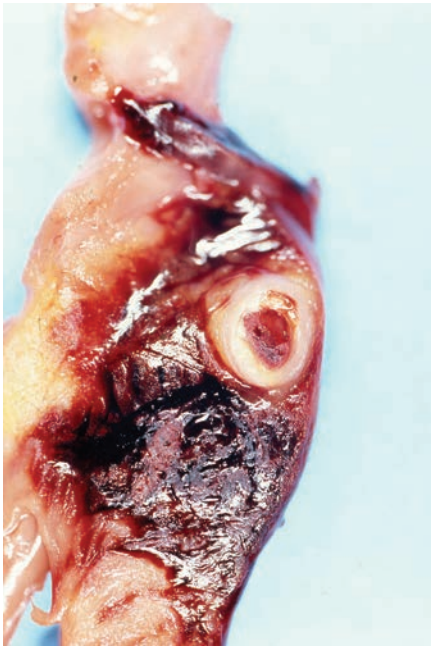
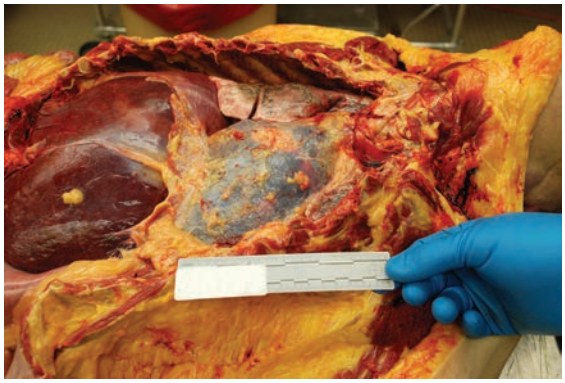
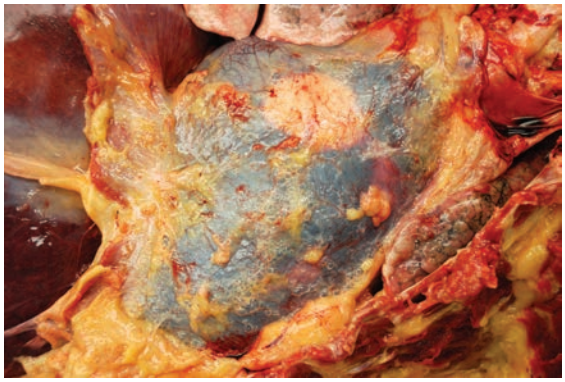
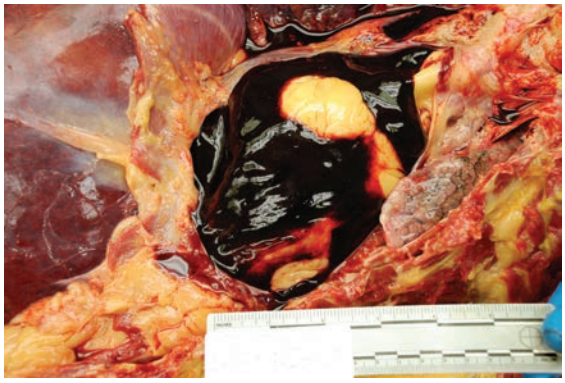
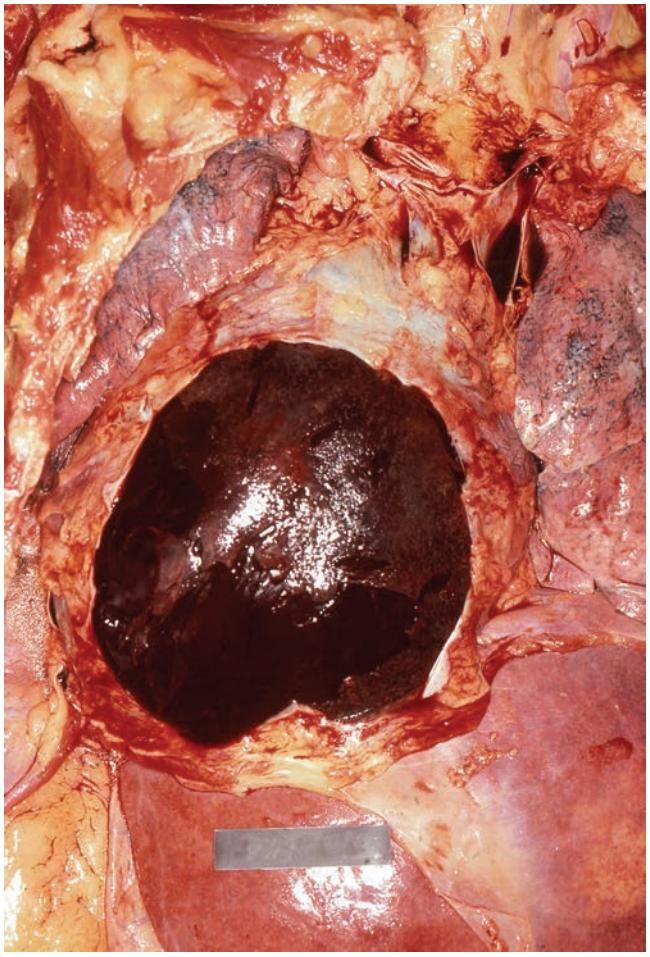


Figure 1.47 Epicardial vessel with complete occlusion by organizing thrombus. Note the adjacent epicardial hemorrhage.

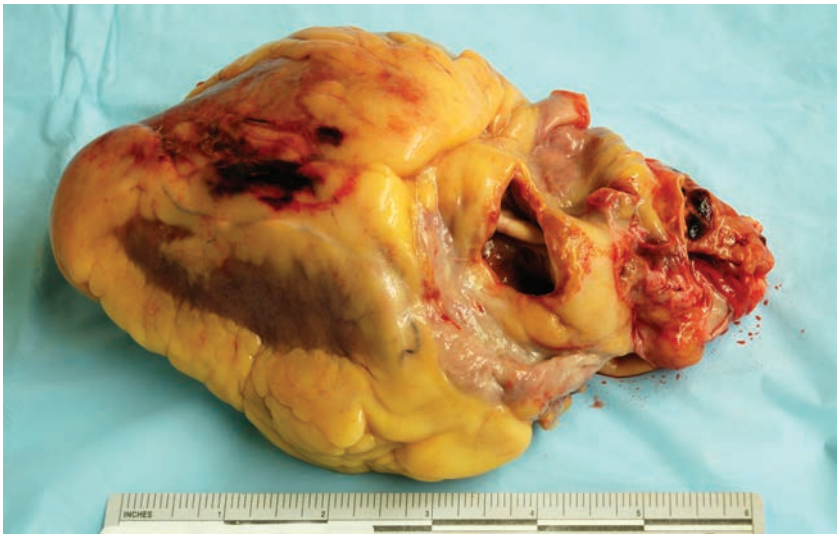


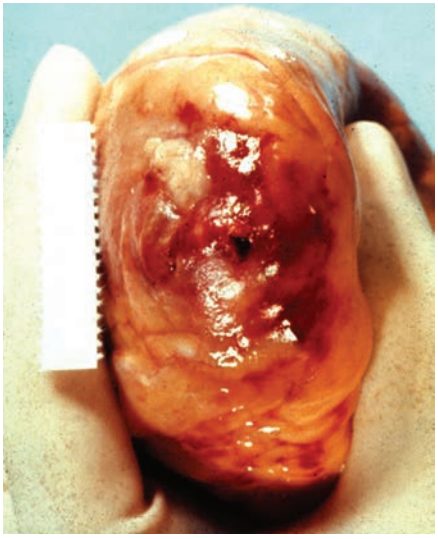




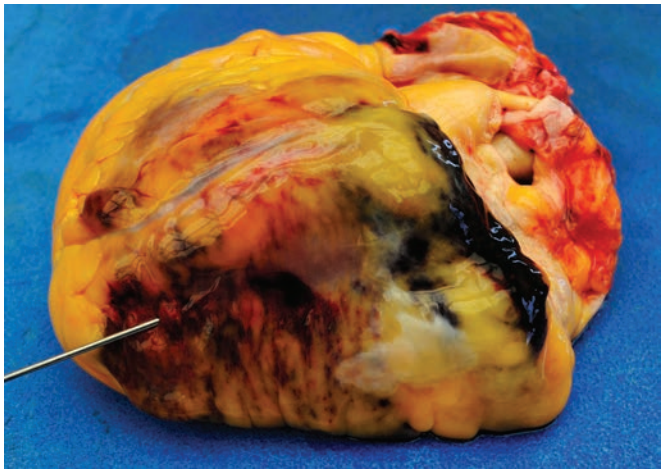


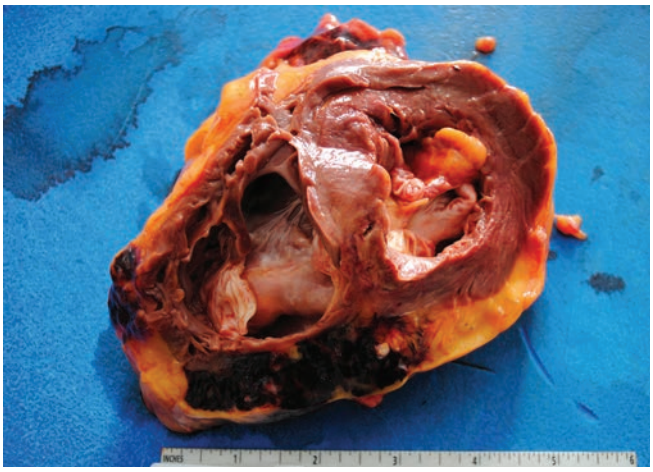
Figures 1.48–1.51 View of the thoracic cavity looking downward at the heart during autopsy. Note the purple discoloration of the pericardial sack due to underlying accumulation of blood. Note the two different examples with large blood clot encasing the heart after the pericardial sac was removed. This demonstrates a cardiac tamponade following an acute ruptured myocardial infarction.

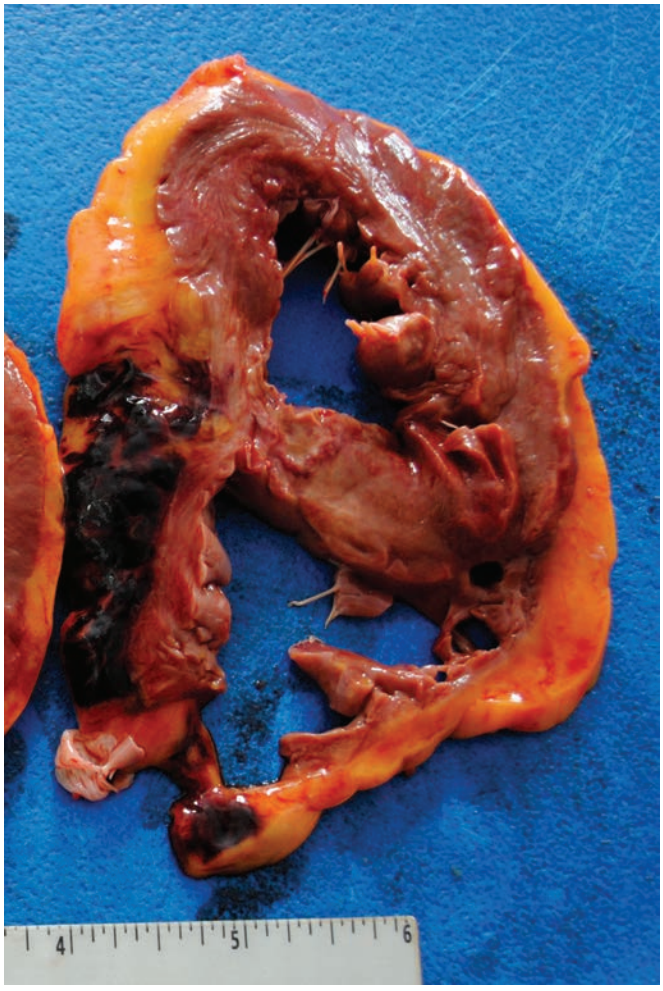




Figures 1.52 and 1.53 Two examples of hearts demonstrating acute ruptured myocardial infarction. Note the adjacent hemorrhage and perforation site. This resulted in cardiac tamponade and sudden death.







Figures 1.54–1.56 This individual had marked calcific atherosclerosis in all of his epicardial vessels with a ruptured acute myocardial infarction involving the posterior wall of the left ventricle, septum, and a large portion of the right ventricle. Note the hemorrhage to the right ventricle wall with the ruptured site indicated by the probe. Rupture of the right ventricle due to atherosclerosis is much less common than left ventricular rupture.

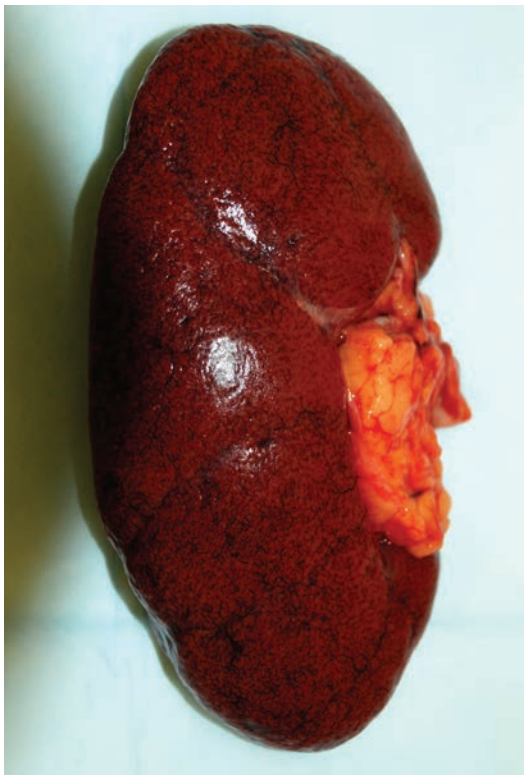


Figure 1.57 Early to moderate nephro-arteriosclerosis.

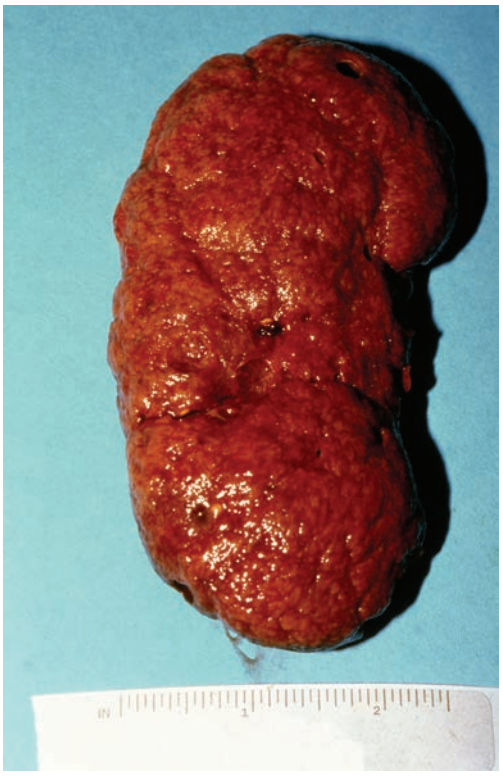


Figure 1.58 Moderate to marked nephro-arteriosclerosis.

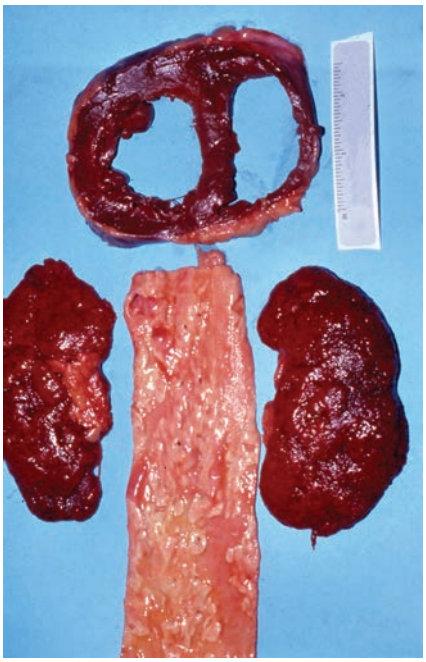


Figure 1.59 Nephro-arteriosclerosis with markedly granular subcapsular kidney surfaces and cortical scarring associated with hypertensive cardiovascular disease. Note the cardiac hypertrophy and biventricular dilatation in this failing heart. There is also moderate atherosclerosis of the aorta. Note the markedly granular subcapsular kidney surfaces and cortical scarring also associated with this process.

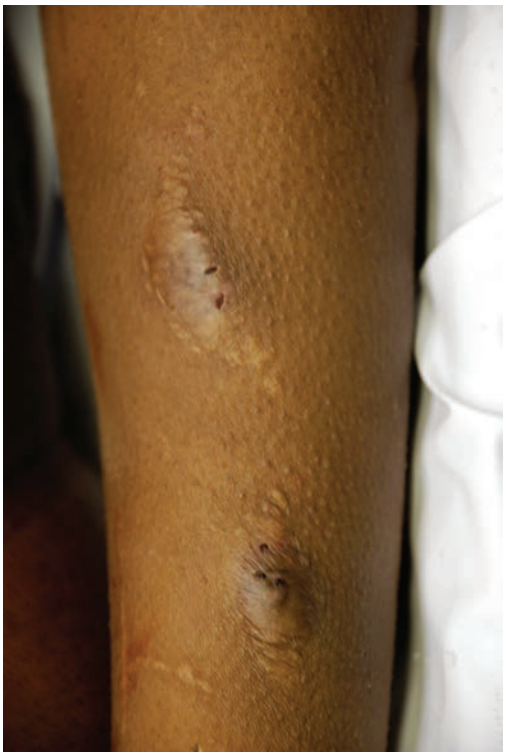


Figure 1.60 Arteriovenous hemodialysis grafts for treatment of chronic renal failure due to hypertensive cardiovascular disease. People receiving dialysis are more prone to hemorrhagic events during the time of treatment. Graphs may become infected and later rupture as well.

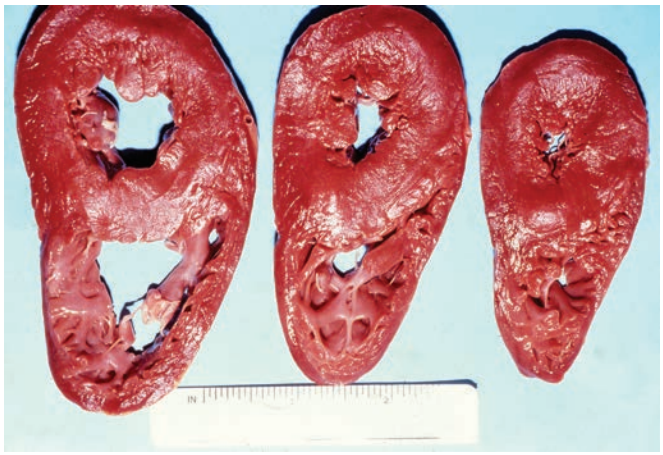


Figure 1.61 Normal cross sections of heart. Compare this image with the ones below.

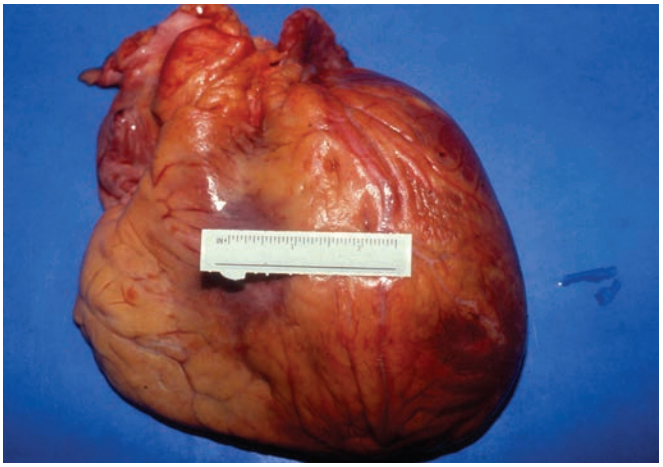


Figure 1.62 This is a markedly enlarged heart due to hypertensive cardiovascular disease. This heart is diffusely enlarged and shows cardiac hypertrophy.

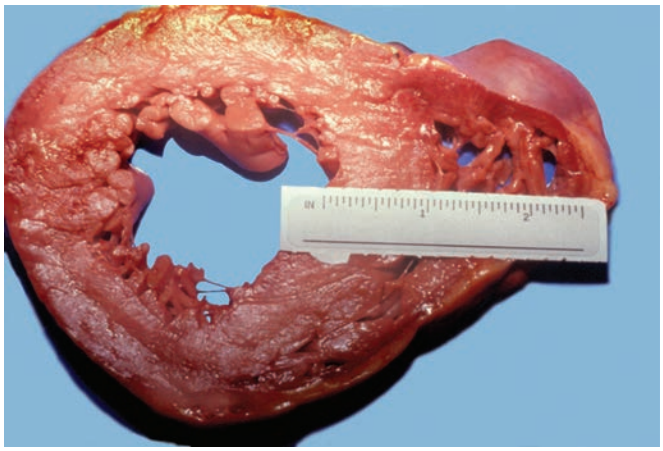


Figure 1.63 This is a markedly hypertrophied heart with extreme concentric left ventricle hypertrophy. This individual had severe hypertension.

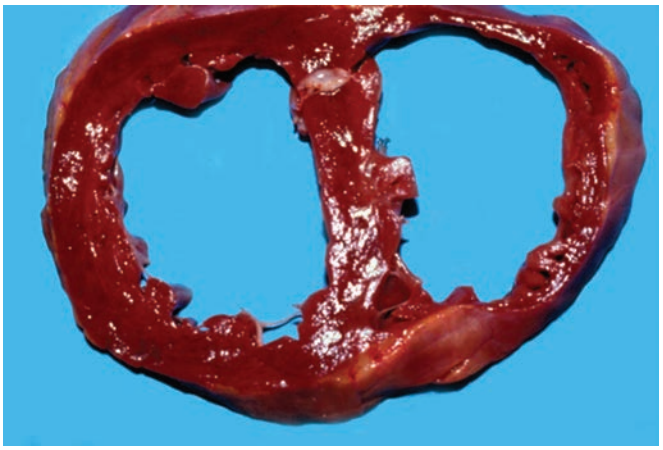


Figure 1.64 The right and left ventricles show marked dilatation. This individual died of a peripartum cardiomyopathy. A dilated cardiomyopathy or end-stage hypertensive cardiovascular disease with cardiac failure will appear the same grossly.

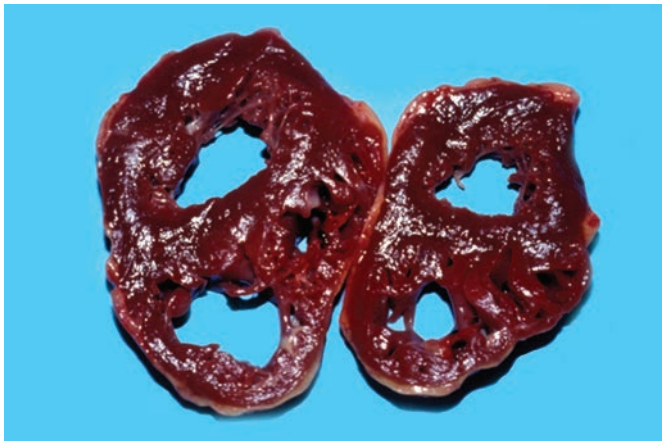


Figure 1.65 The sections of these ventricles reveal marked right ventricle hypertrophy. This individual had end-stage primary pulmonary fibrosis with cor pulmonale and cardiac failure.

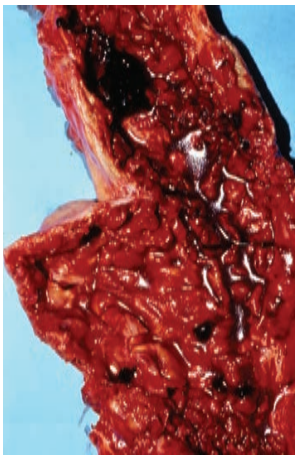


Figure 1.66 The intimal lining of an aorta with marked atherosclerosis in a decedent with a long-standing history of smoking, diabetes, high cholesterol, and high blood pressure.

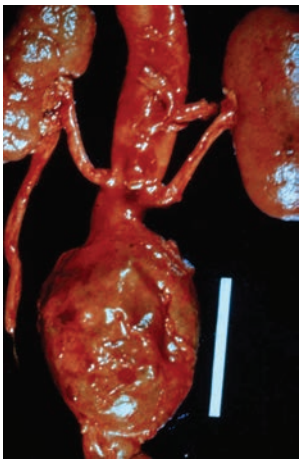


Figure 1.67 Abdominal aortic atherosclerotic aneurysm shown in its typical location inferior to the renal arteries and above the iliac bifurcation.

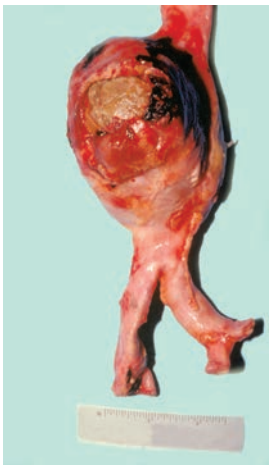


Figure 1.68 An abdominal atherosclerotic aneurysm with a rupture at its anterior aspect and visible thrombosis.

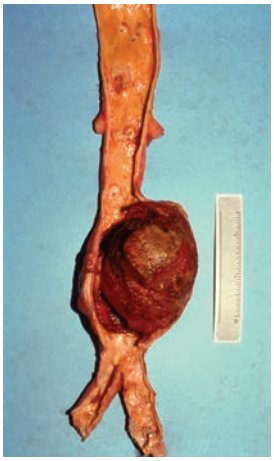


Figure 1.69 This abdominal aortic aneurysm has been opened to remove half of the vessel wall and show the underlying intimal surface with moderate atherosclerosis except for the region of the aneurysm that has marked atherosclerosis and a large overlying thrombus.

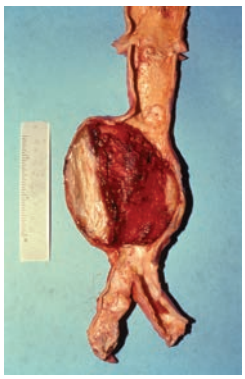


Figure 1.70 This abdominal aortic aneurysm has been cross sectioned to show the partial obstruction of the aneurysm by organizing thrombosis. The lumen is demonstrated by fresh red blood clot at its surface, and the thrombus is demonstrated by the light gray regions adjacent to the right and left wall.

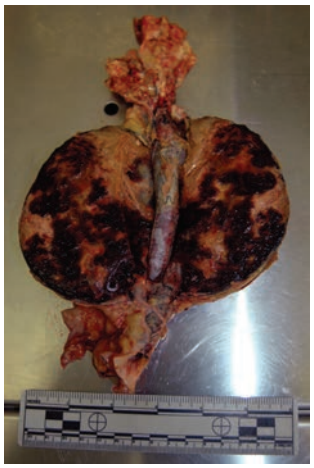


Figure 1.71 Abdominal aortic aneurysm with vascular graft repair.

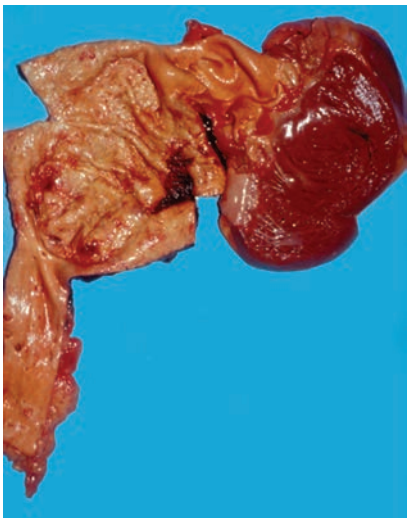


Figure 1.72 Thoracic aortic atherosclerotic aneurysm. This is not the typical location for such an aneurysm. Also note the aneurysm begins distal to the root of the aorta.

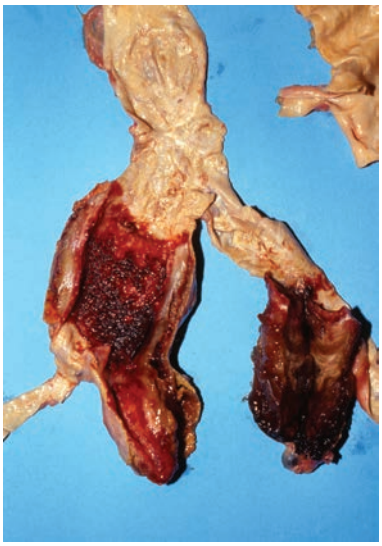


Figure 1.73 This demonstrates a decedent who had multiple atherosclerotic aneurysms including both iliac arteries.

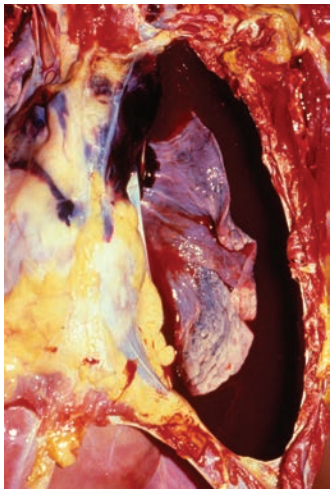


Figure 1.74 Hemothorax from a ruptured thoracic atherosclerotic aneurysm.

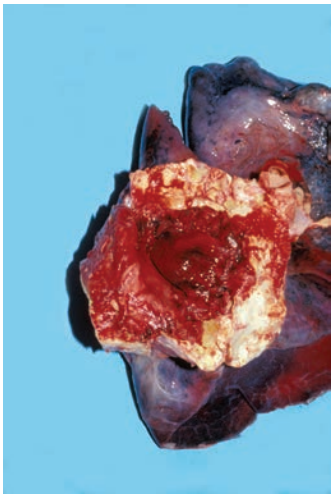


Figure 1.75 Thoracic atherosclerotic aortic aneurysm adherent to lung with rupture into the lung parenchyma causing massive hemoptysis. Note the second picture demonstrates the rupture site with adherent blood clot removed.

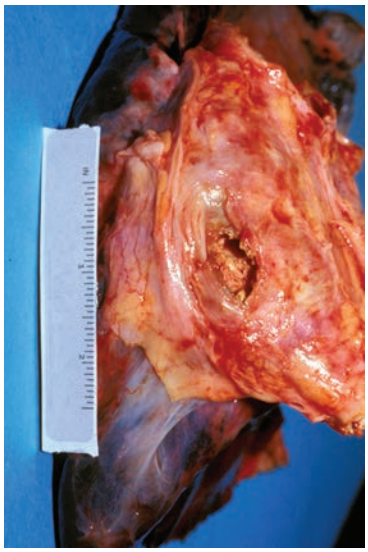


Figure 1.76 Thoracic atherosclerotic aortic aneurysm adherent to lung with rupture into the lung parenchyma causing massive hemoptysis. This picture demonstrates the rupture site with adherent blood clot removed.

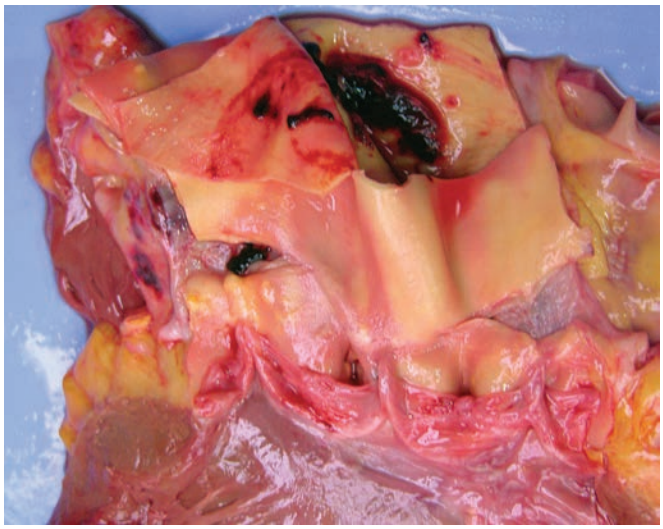


Figure 1.77 Intimal tear of the ascending aorta with dissection. Note blood tracking through the separated media.

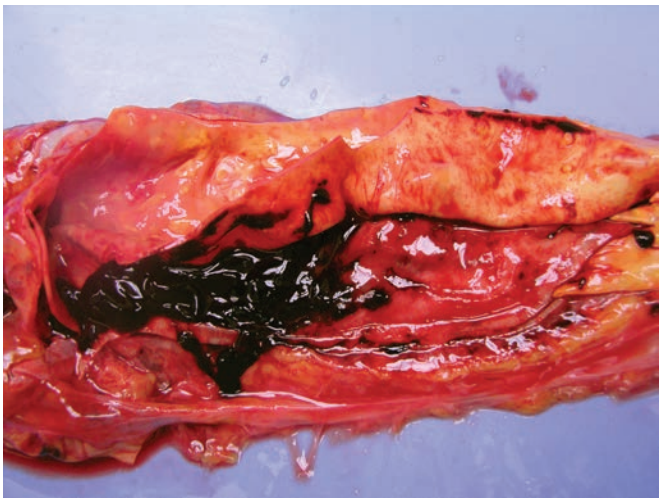


Figure 1.79 Aortic dissection with exposed separated media with blood clot in a person with Marfan syndrome.

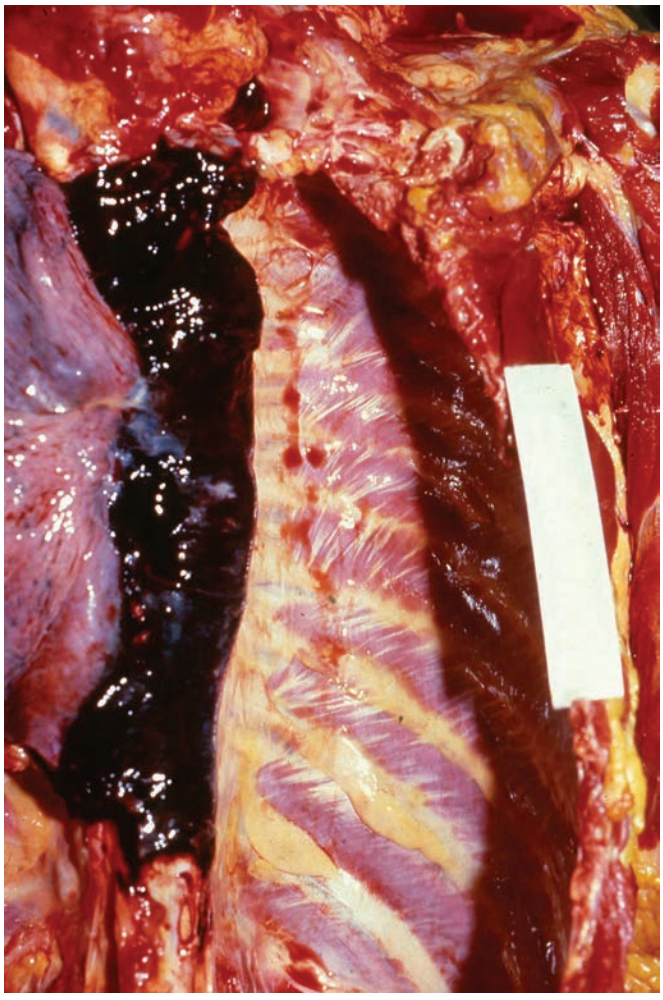


Figure 1.78 An in situ aortic dissection. Note the hemorrhage extending from the root of the aorta down the paravertebral region shown by dark red hemorrhagic discoloration. This decedent had severe hypertensive cardiovascular disease.

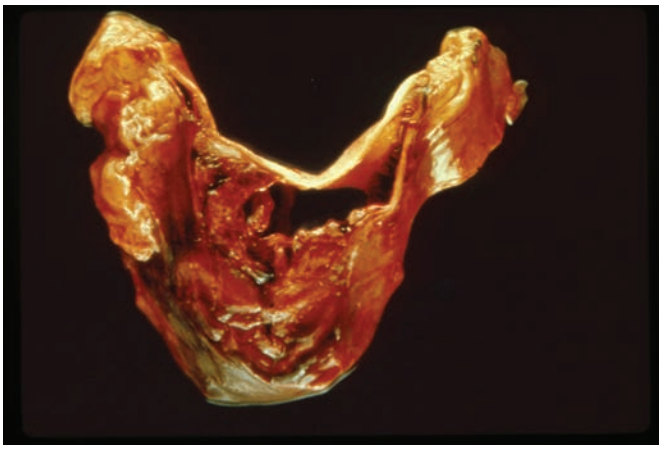
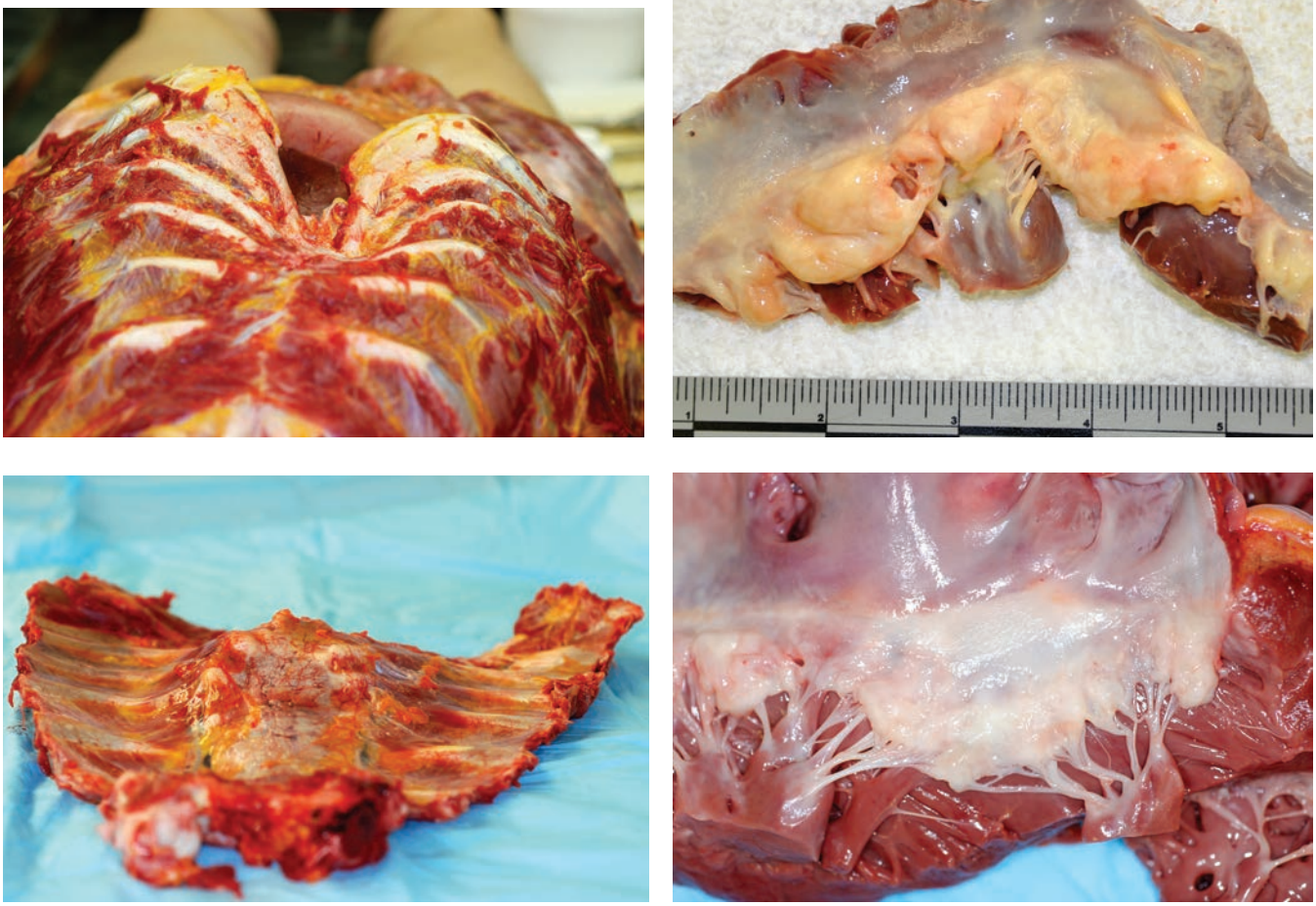


Figure 1.80 Cross section of aorta revealing a double barrel lumen. The superior aspect of this figure shows an opened aorta with the exposed lumen. Directly inferior to this is the separated media and adventitia with a second lumen that is partially thrombosed.



Figures 1.81–1.84 This demonstrates an individual with Marfan syndrome showing characteristics of pectus excavatum and mitral valve prolapse. Marfan syndrome is a connective-tissue disease associated with abnormality of the fibrillin gene on chromosome 15. This is also associated with aortic dissection. Mitral valve prolapse by itself may be associated with sudden cardiac death.

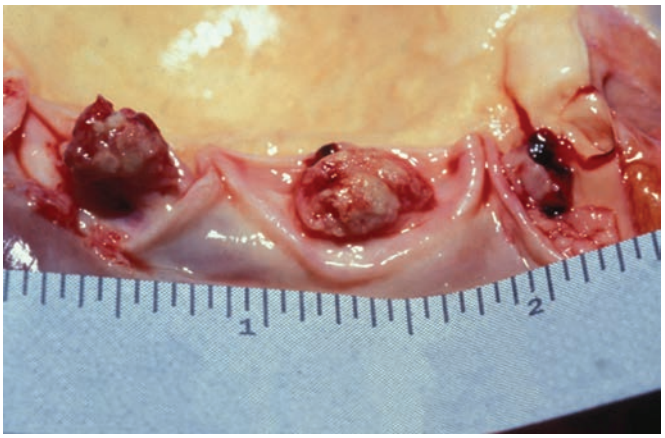


Figure 1.85 This endocardial surface shows large nonbacterial thrombotic endocarditis associated with a hypercoagulable state from metastatic adenocarcinoma. Special stains were negative for microorganisms.

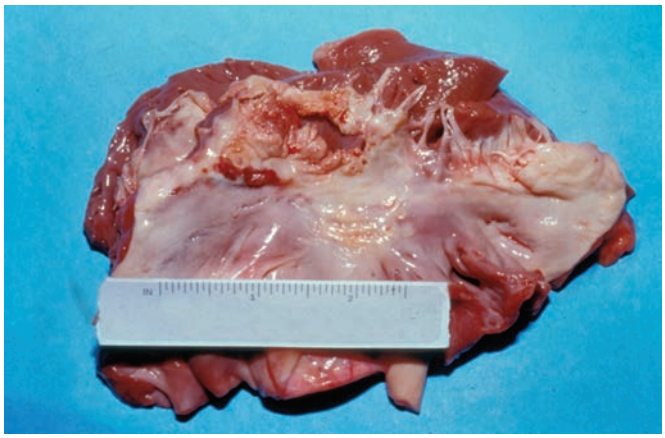


Figure 1.86 This perforated mitral valve is secondary to acute bacterial endocarditis. This individual first went to the emergency room approximately a day and a half before with the complaint of fever and chest pain. He was sent home with antibiotics and later returned with severe pulmonary edema and died shortly after.

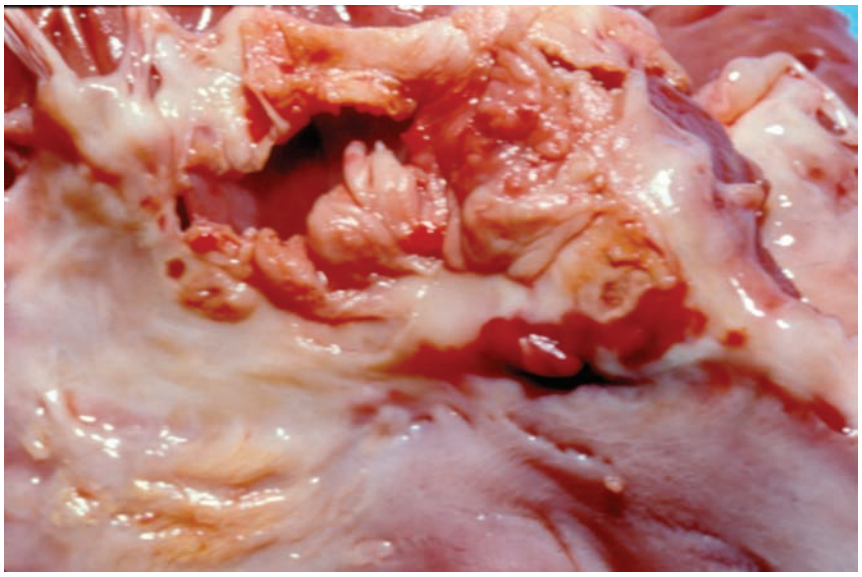


Figure 1.87 Close-up view of an acute infectious endocarditis with valve perforation. Gram stain revealed numerous Gram-positive organisms.

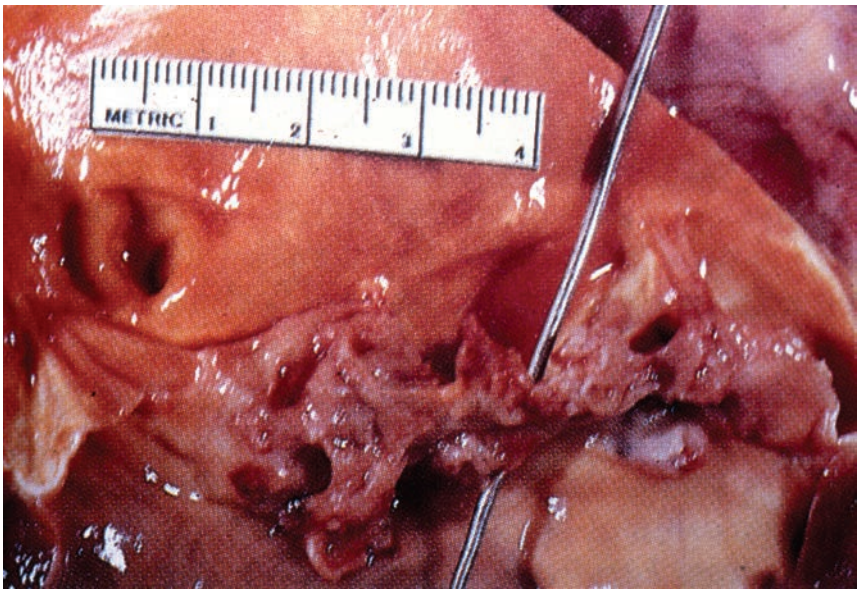


Figure 1.88 Acute infectious endocarditis with valve leaflet perforation.

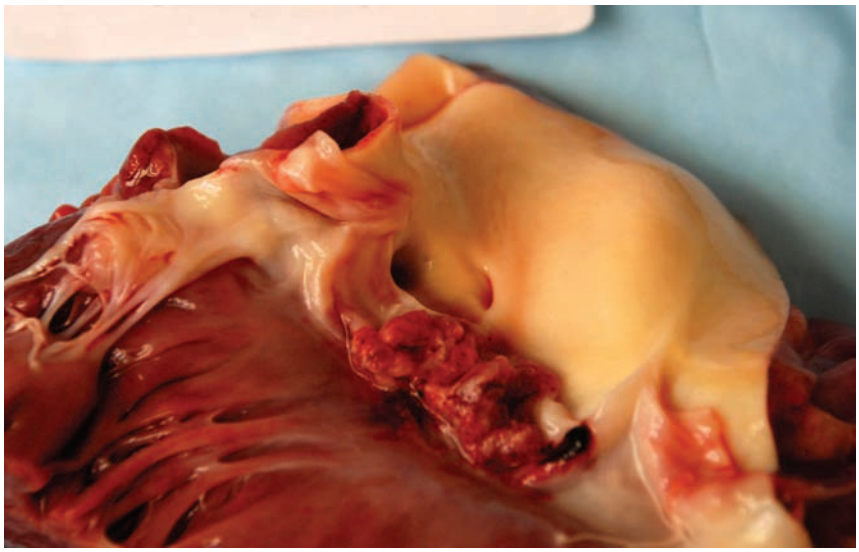


Figure 1.89 Note the partially fused central aortic cusps with inferior displacement and subacute bacterial endocarditis vegetations. Also note the anomalies distribution of coronary arteries. This coronary artery anomaly may be associated with sudden cardiac death by itself. Note the left coronary artery is displaced adjacent to the right coronary artery with an acute angle takeoff and pass between the aorta and pulmonary trunk.

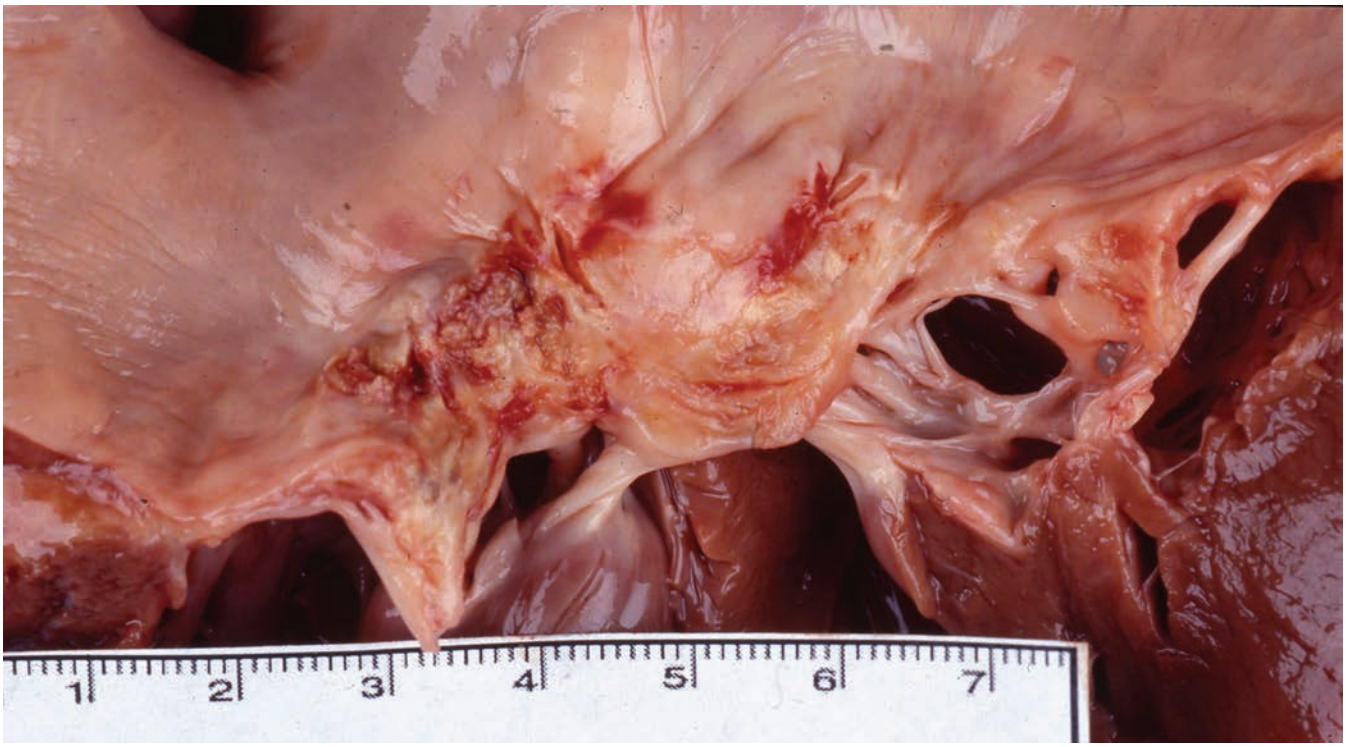
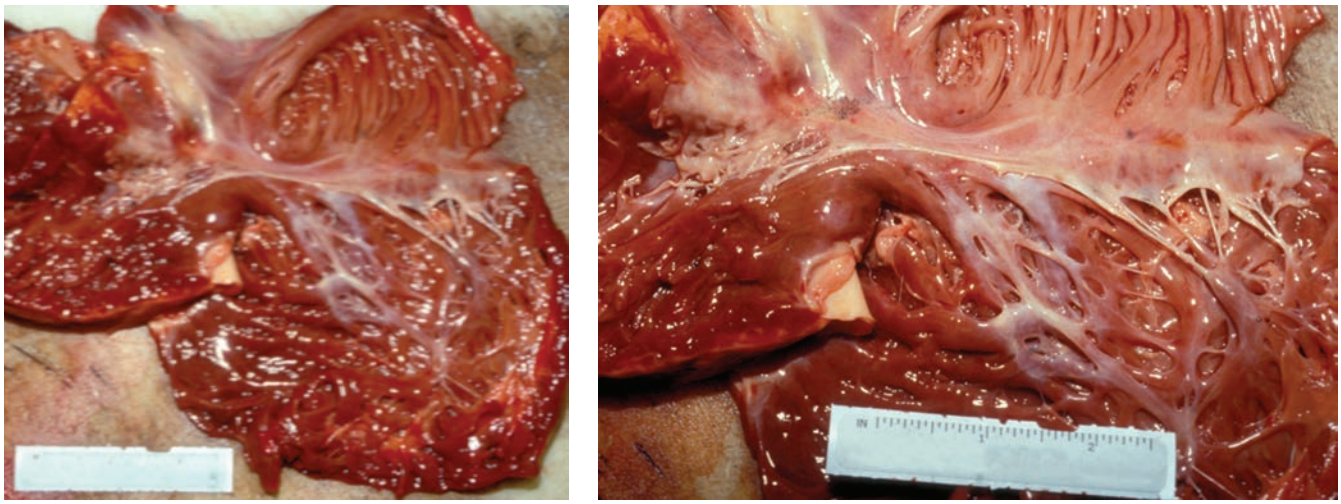
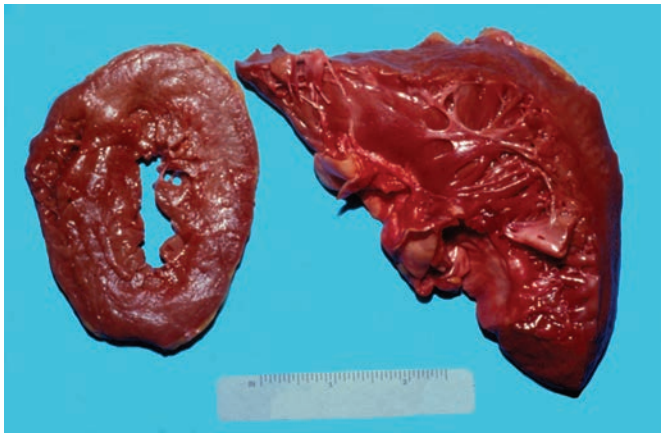
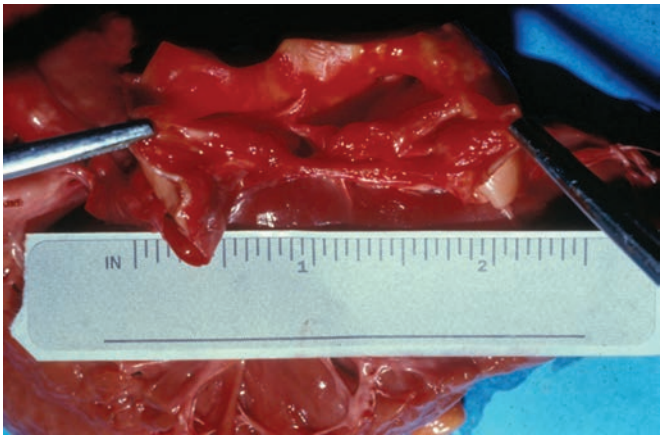


Figure 1.90 Remote cardiac valve damage from rheumatic fever.



Figures 1.91 and 1.92 These are views of a remotely damaged tricuspid valve secondary to chronic intravenous drug abuse and past endocarditis. Note the fibrosis of the adjacent endocardium secondary to regurgitative turbulent blood flow. The decedent was known to have a long-standing cardiac murmur.





Figures 1.93 and 1.94 Markedly hypertrophic heart with concentric left ventricle hypertrophy and a congenital subaortic band causing marked aortic stenosis and sudden cardiac death at age 42 years. The decedent decided years earlier not to have a valve replacement.

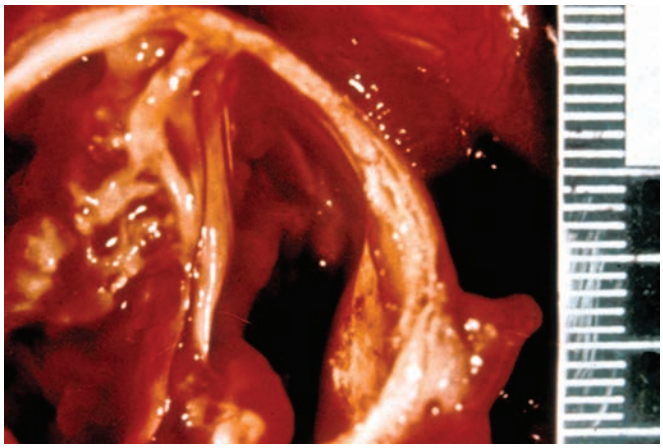


Figure 1.95 This is a bicuspid aortic valve. With advancing age and atherosclerosis, these valves may become markedly stenotic and increase one's risk for sudden cardiac death.

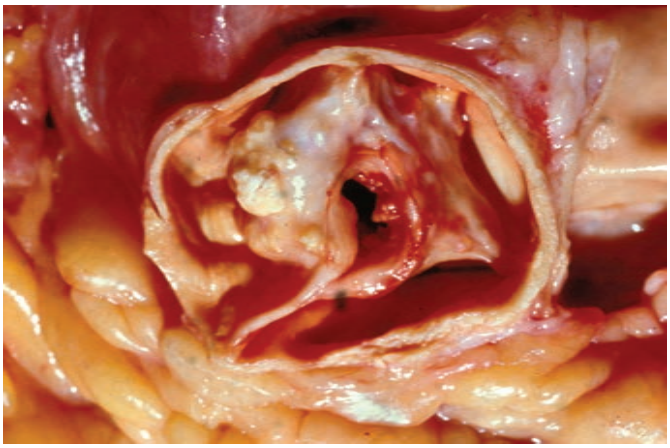


Figure 1.96 Markedly stenotic bicuspid aortic valve from an older individual with a long-standing history of atherosclerotic cardiovascular disease.

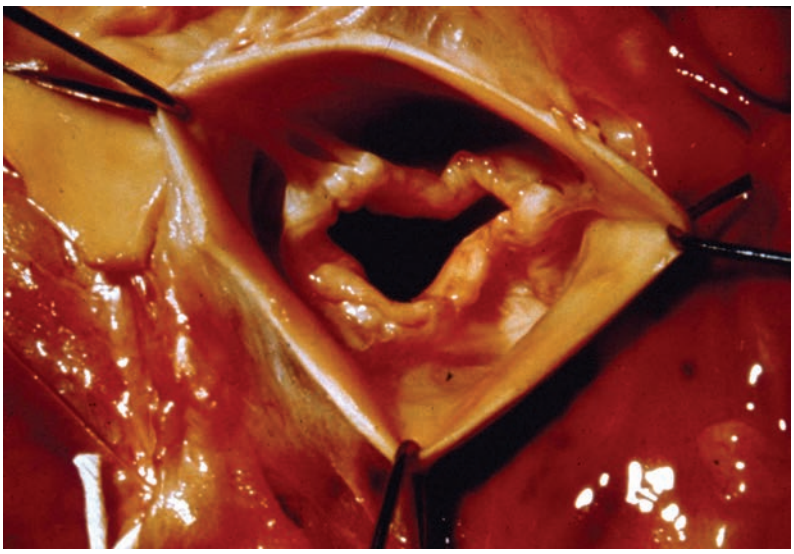


Figure 1.97 Severely stenotic and insufficient aortic valve associated with childhood rheumatic fever. Correction of aortic stenosis will decrease the risk of sudden cardiac death.

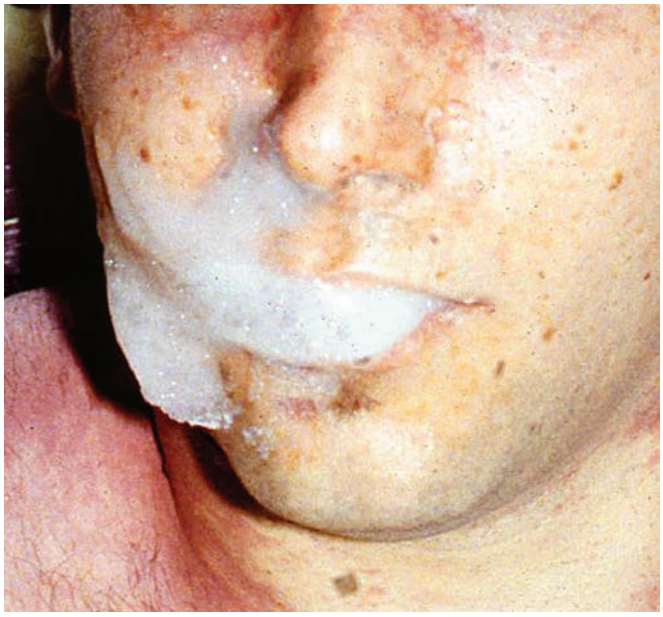


Figure 1.98 A fulminant pulmonary edema with foam extending from the mouth and nose due to congestive heart failure in this individual with past history of viral myocarditis. A foam cone in a younger individual should always first arouse the suspicion of opiate overdose in the absence of known heart disease.

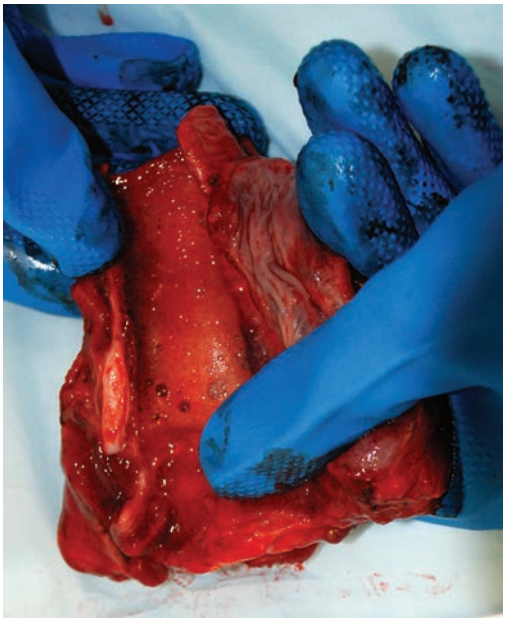


Figure 1.100 Note the frothy fluid from pulmonary edema extending into the laryngeal airway.

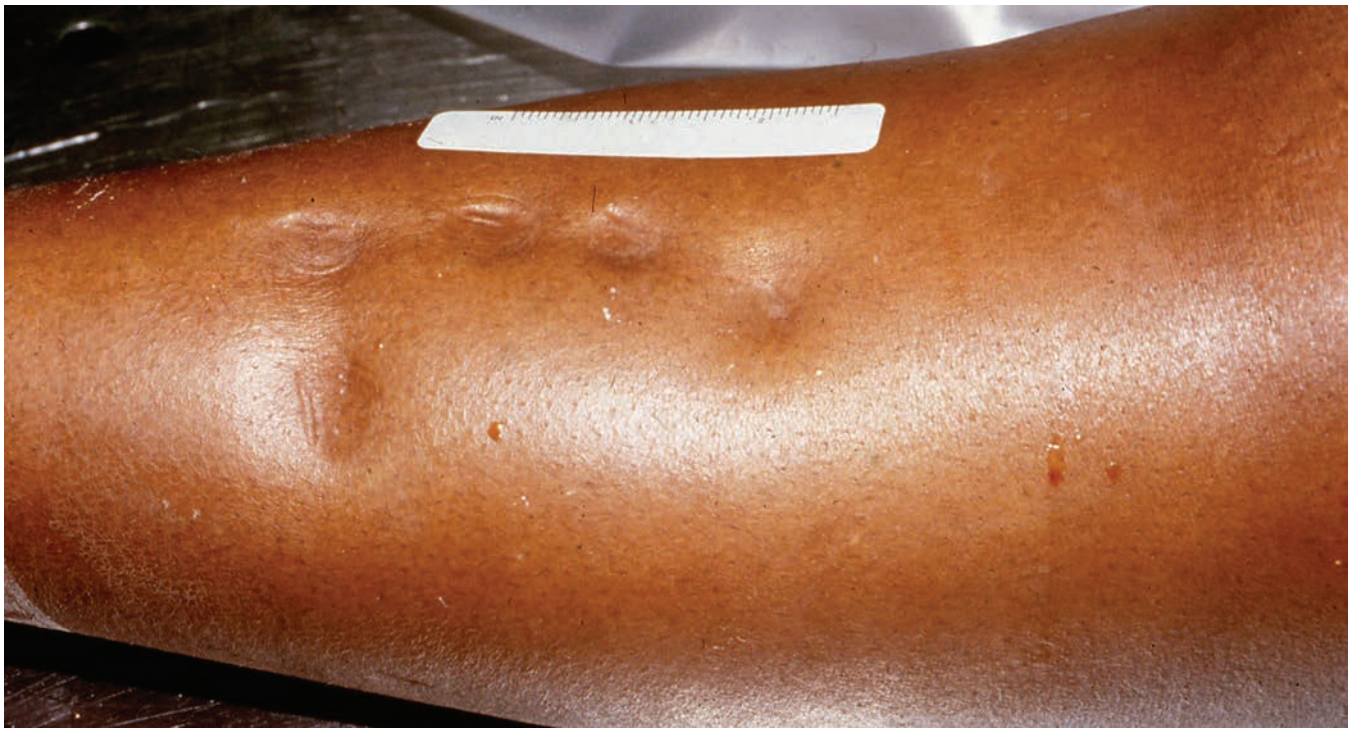


Figure 1.99 Pitting edema of the leg due to congestive heart failure.

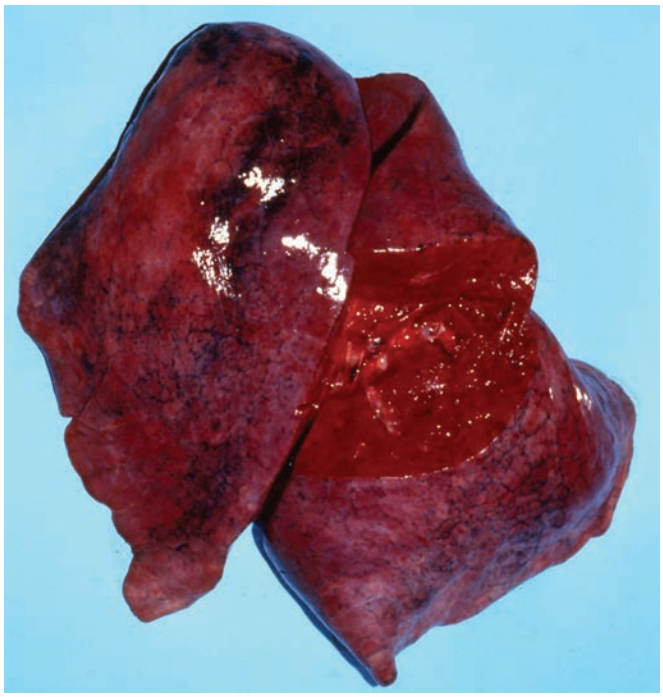


Figure 1.101 Lung with marked congestion and edema. Note the diffuse purple discoloration.

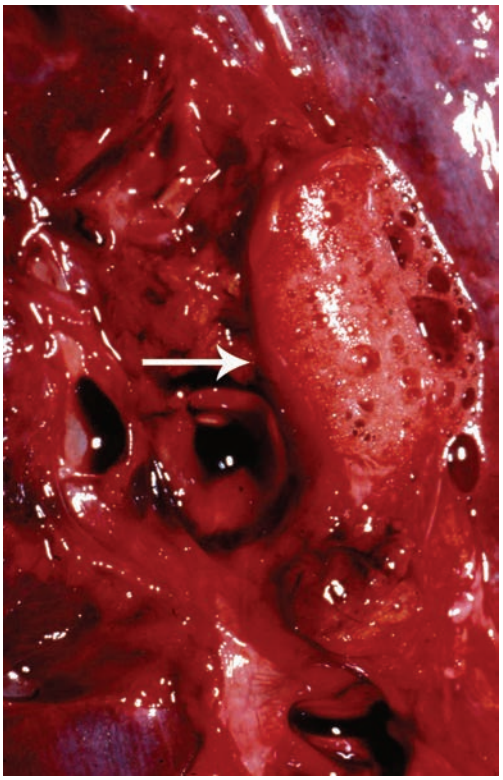


Figure 1.102 Marked pink to red frothy fluid extending from the cut parenchyma of a lung due to fulminant pulmonary edema.

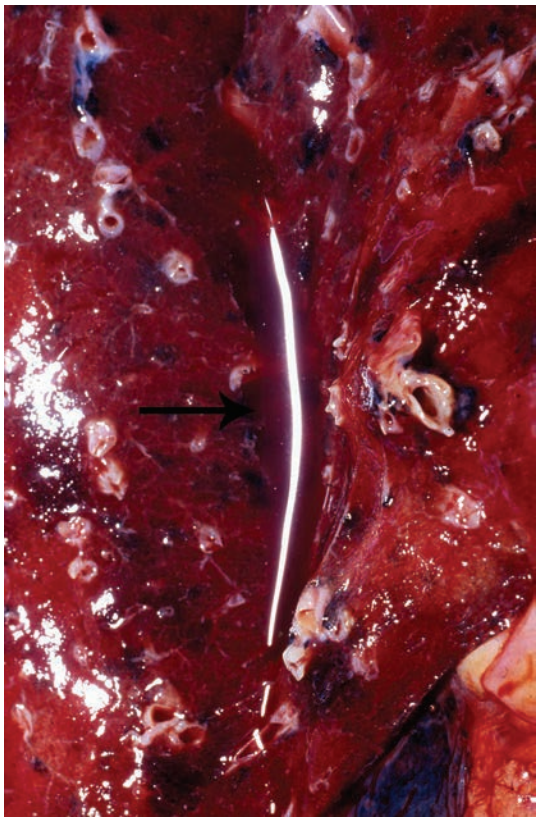


Figure 1.103 Cut section of a lung with pulmonary edema.

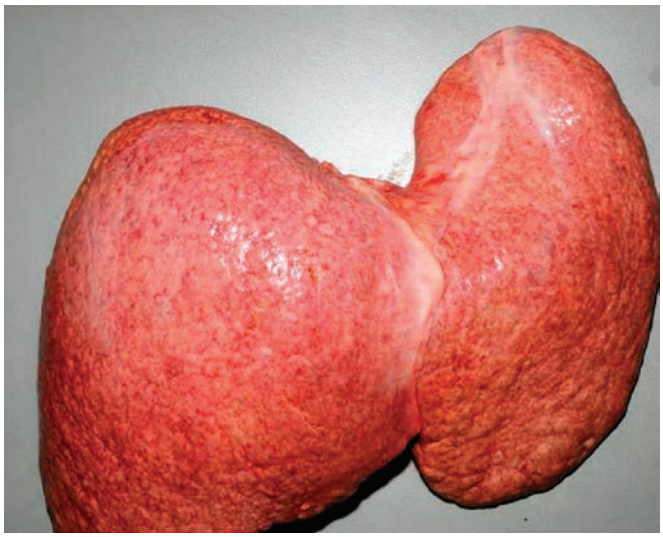


Figure 1.104 Cardiac sclerosis due to pulmonary hypertension and right-sided heart failure. The subcapsular and perivenular fibrosis mimics micronodular cirrhosis.

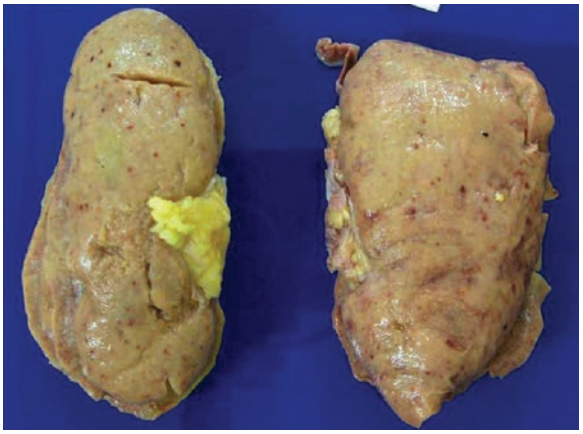
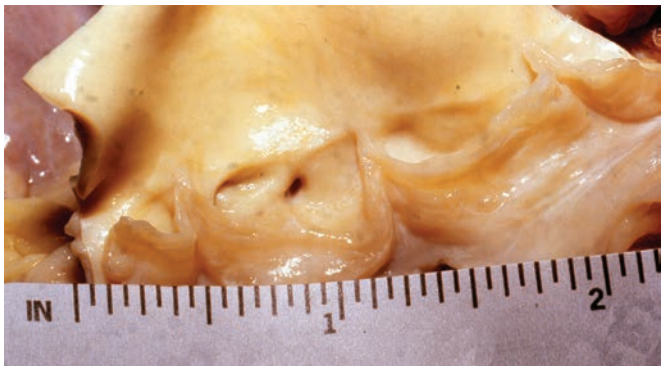
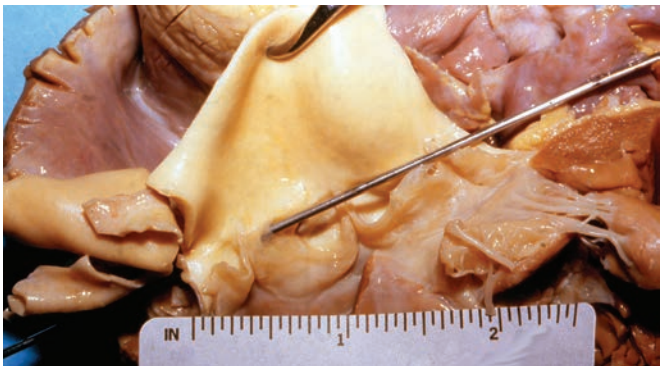
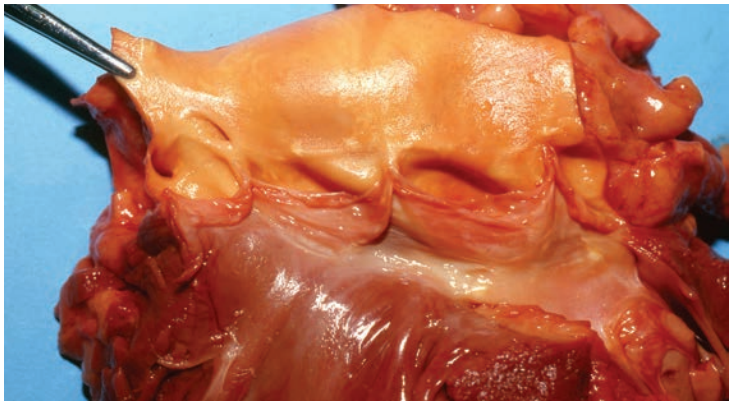


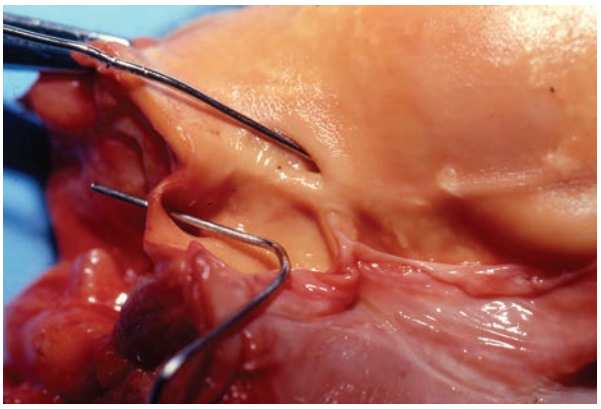
Figure 1.105 Amyloidosis, kidneys. The uniform pale waxy color of both kidneys is typical for organs involved by amyloidosis.





Figures 1.106 and 1.107 These figures demonstrate a coronary artery anomaly with acute angle takeoff and luminal narrowing in a 15-year-old who died suddenly during a basketball game. There was no history of blunt impacts to the chest during the game. There was no past history of syncopal episodes or chest discomfort.





Figures 1.108 and 1.109 A coronary artery anomaly with acute angle takeoff and luminal narrowing where both coronary ostia rise from the right aorta sinus. This individual died of a sudden cardiac death shortly after exertion.

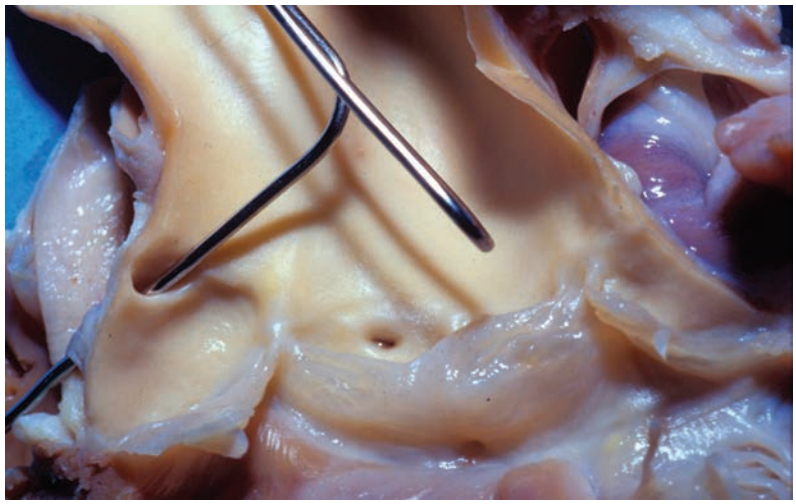


Figure 1.110 This coronary anomaly reveals a bicuspid aortic valve with superior displacement of one of the coronary ostia. This child died of trauma sustained in a motor vehicle accident and this finding was incidental.

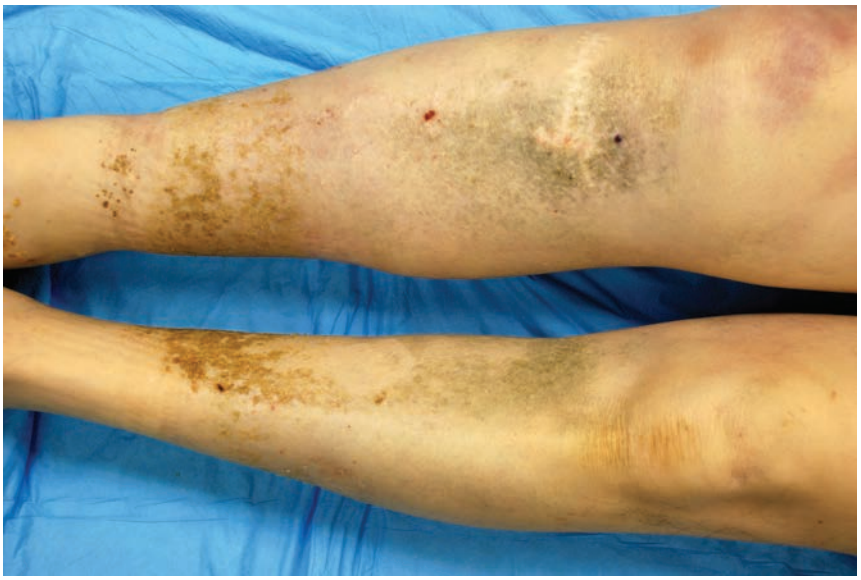
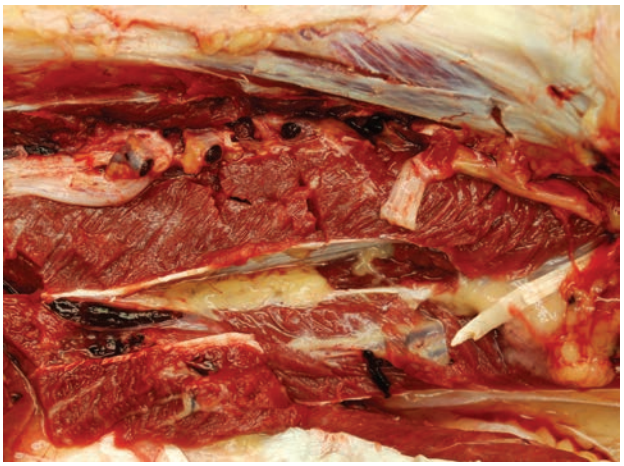
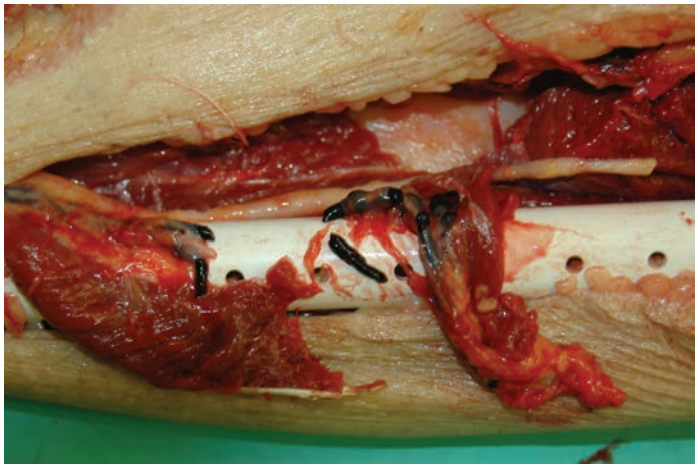
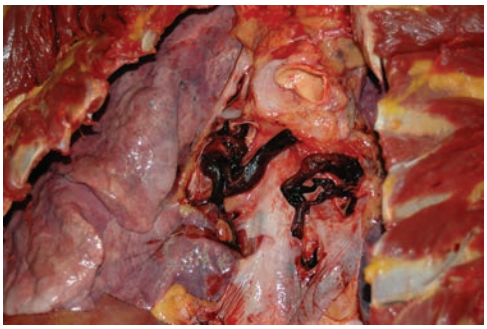


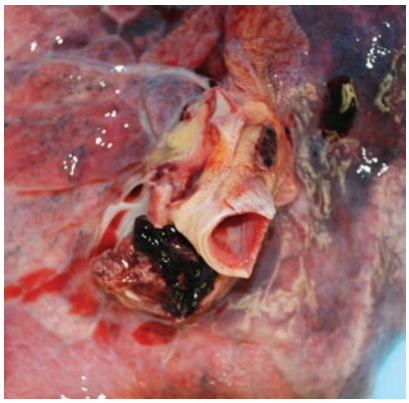
Figure 1.111 Note the large swelling of the right calf where deep venous thrombosis was found after incision.

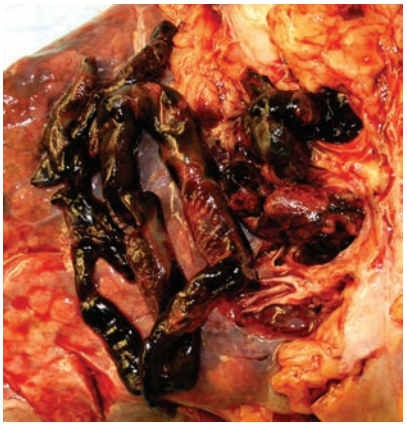




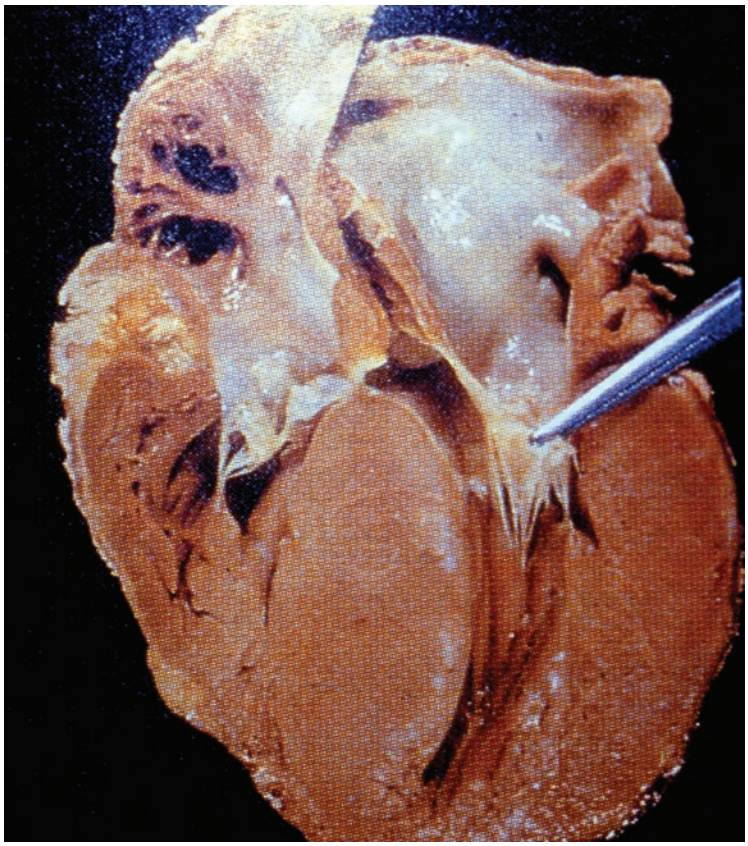
Figures 1.112 and 1.113 Incision of the lower leg with dissection of the gastrocnemius muscle demonstrating multiple deep venous thrombi from these individuals. Note the plastic tubing in Figure 1.113 associated with previous tissue donation with removal of bone and soft tissue.

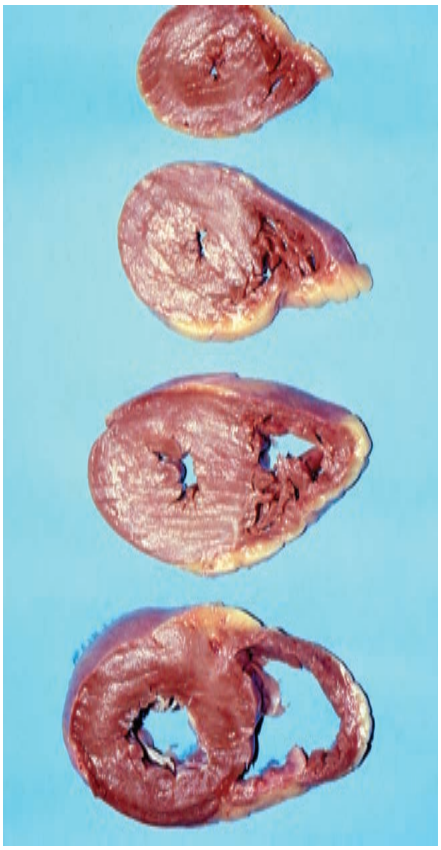


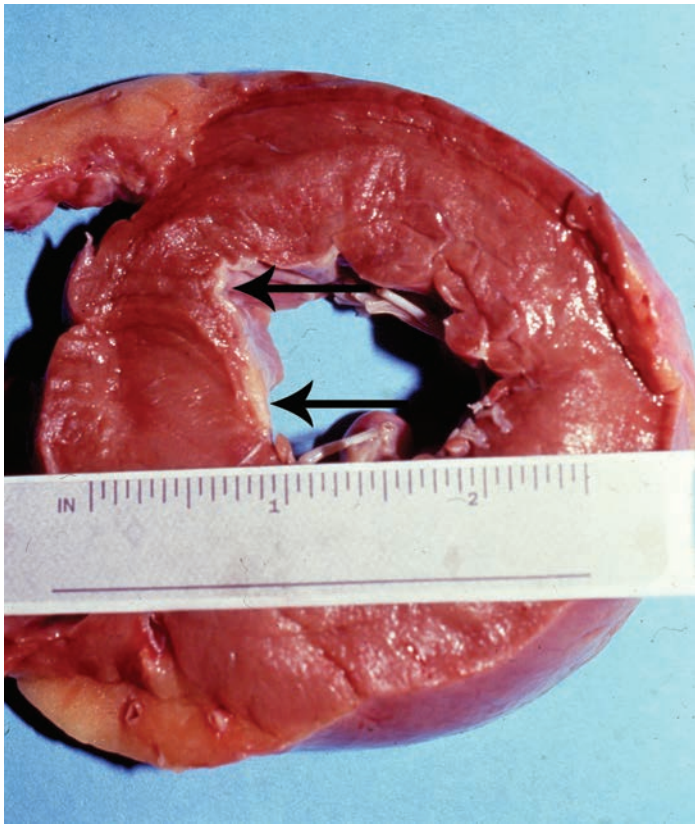




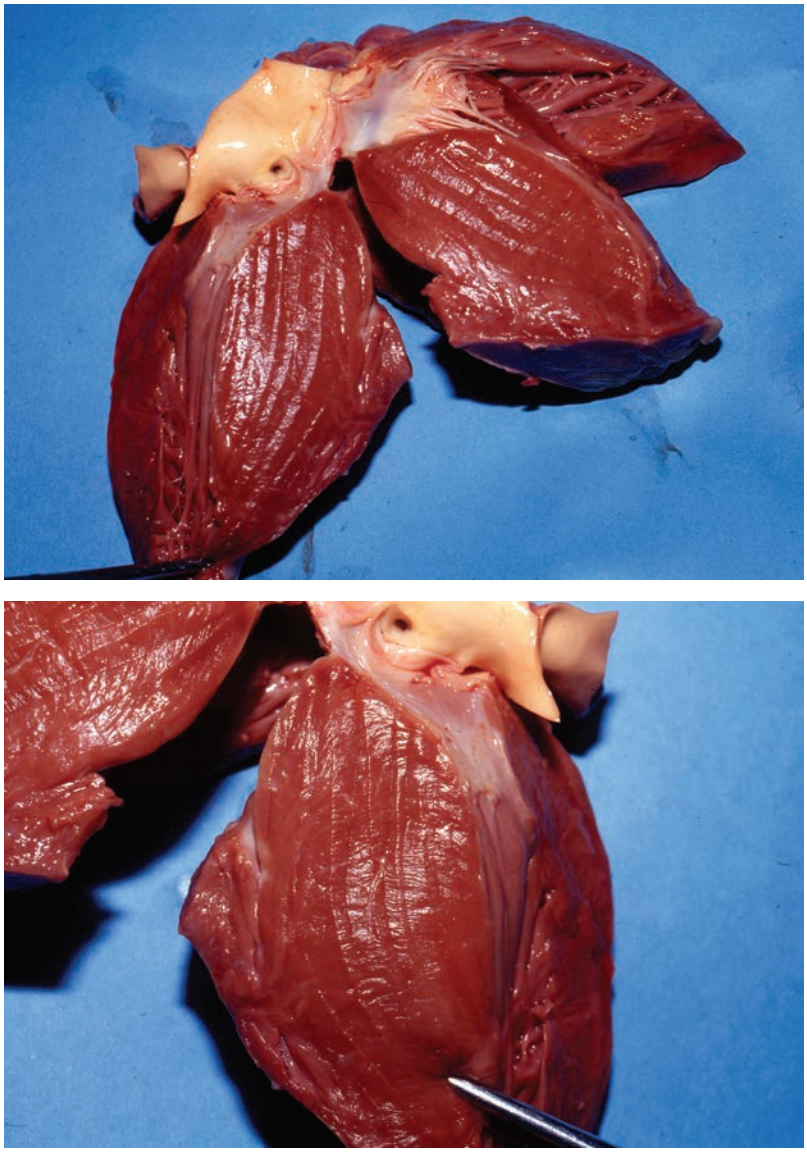
Figures 1.114–1.116 Pulmonary thromboembolus in an obese woman who was on birth control pills and smoked cigarettes. Note the dull granular tan to red overlapping thromboemboli that form branching casts of leg veins.







Figures 1.117–1.119 These are different views of two hearts with the classical type of hypertrophic cardiomyopathy. Note the large degree of asymmetric left ventricle hypertrophy.



Figures 1.120 and 1.121 Hypertrophic cardiomyopathy. Note the marked septal hypertrophy with asymmetry.

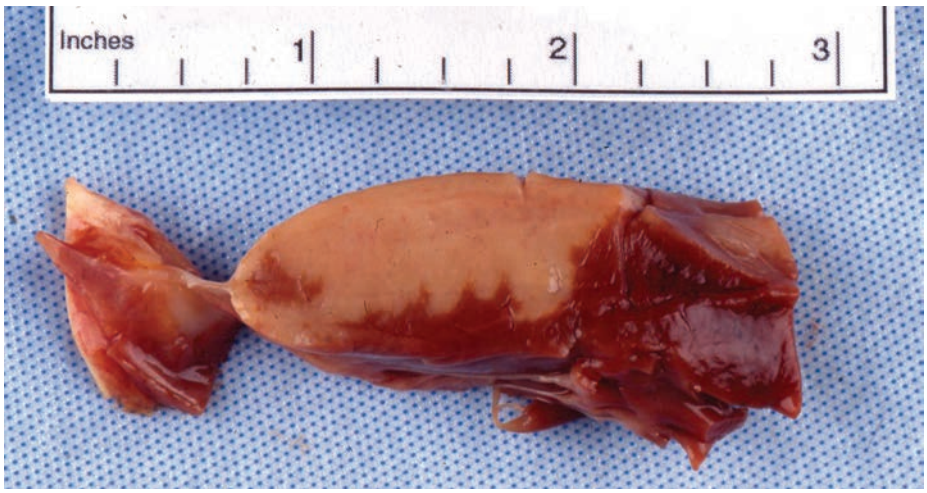
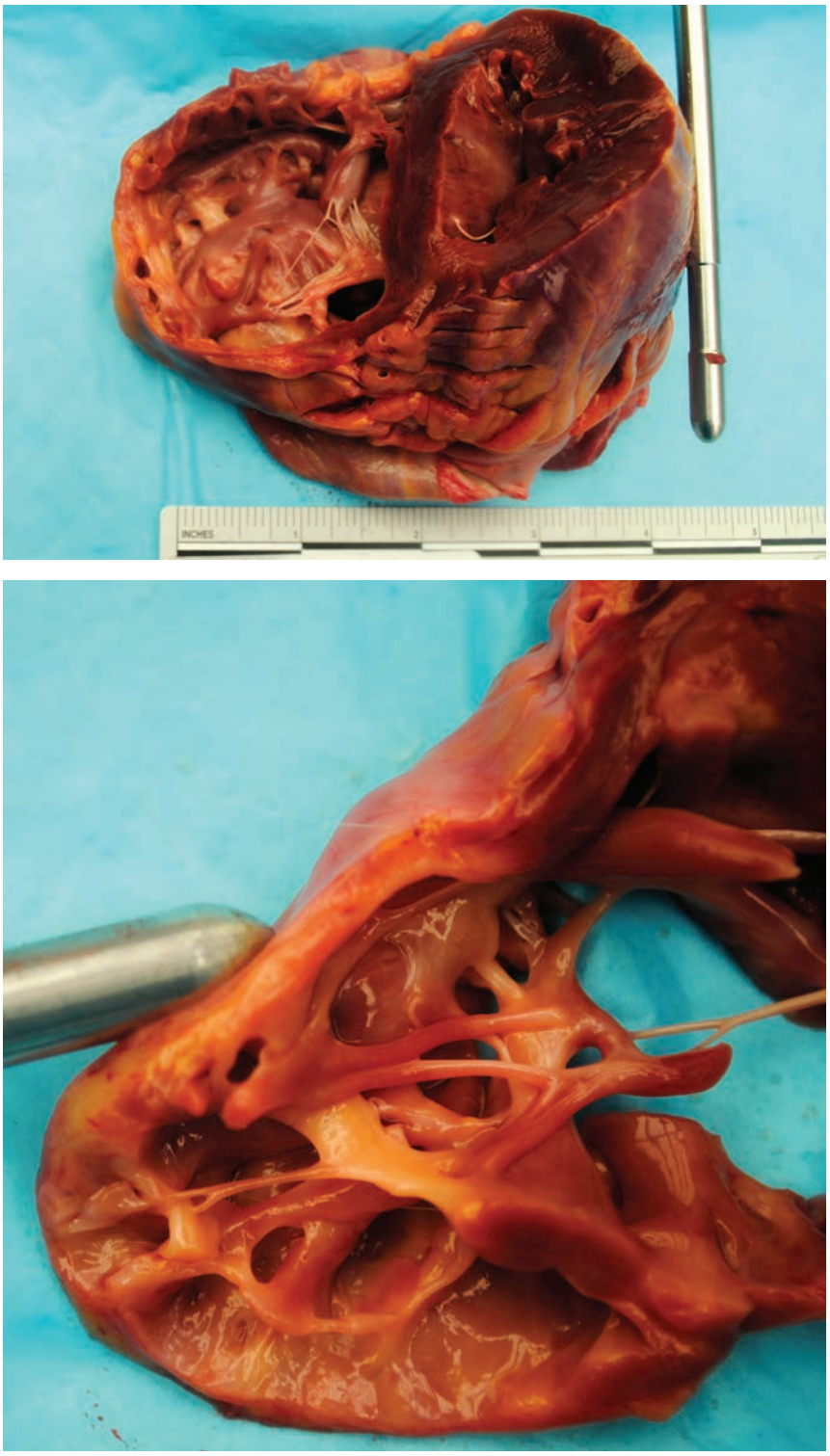
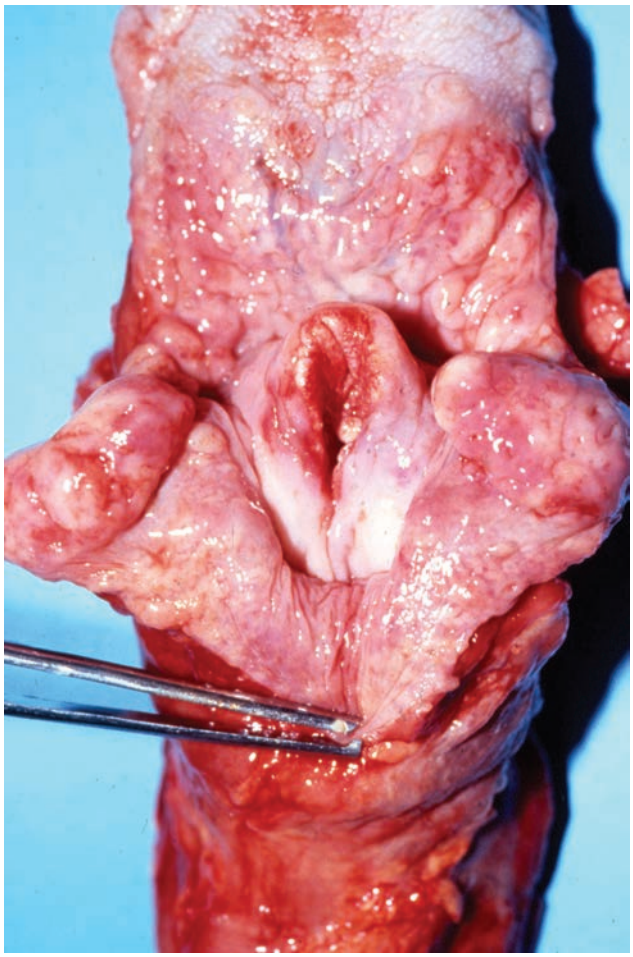
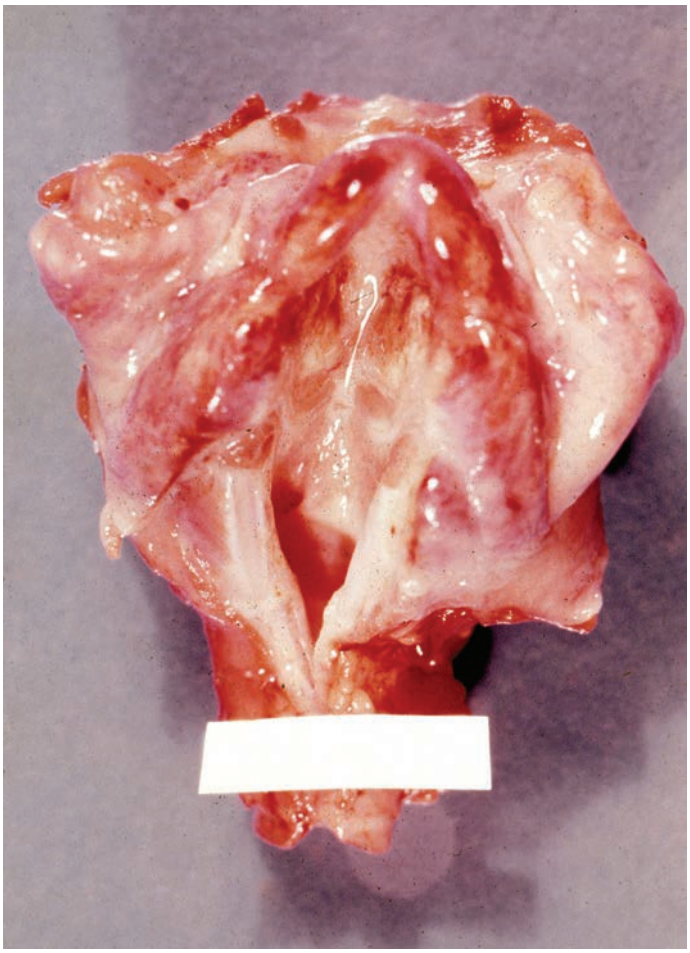


Figure 1.122 Gross autopsy section of myocardium from an individual who died of cardiac sarcoidosis.

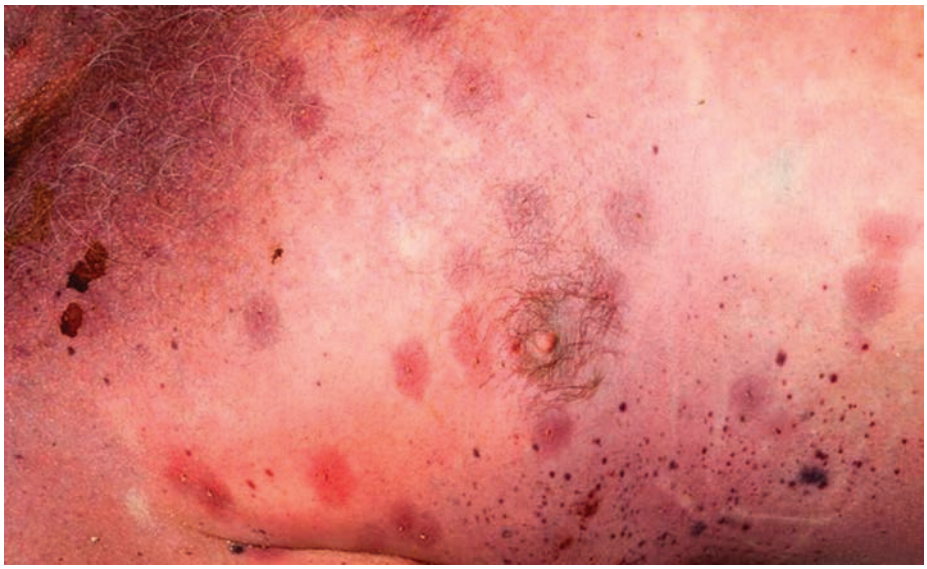


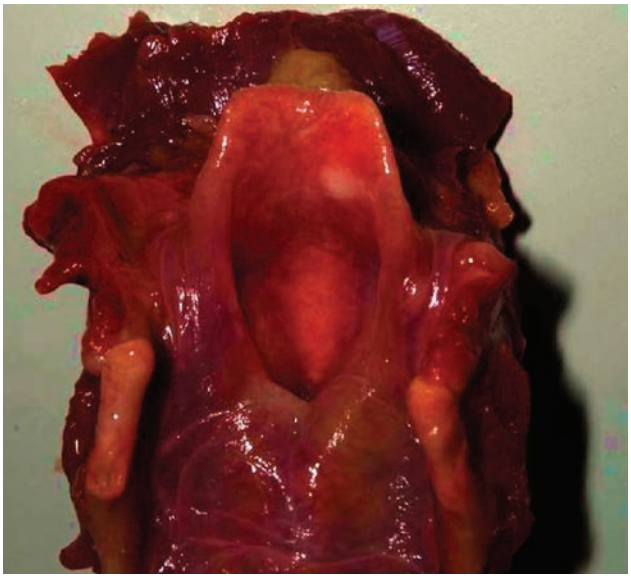
Figures 1.123 and 1.124 Arrhythmogenic right ventricular dysplasia. Note the transmural infiltration of fibro fatty tissue in regions of the right ventricle wall. This is a genetic defect of heart muscle involving desmosomes. This is usually an inherited autosomal dominant condition with variable expression that is associated with fatal arrhythmia.

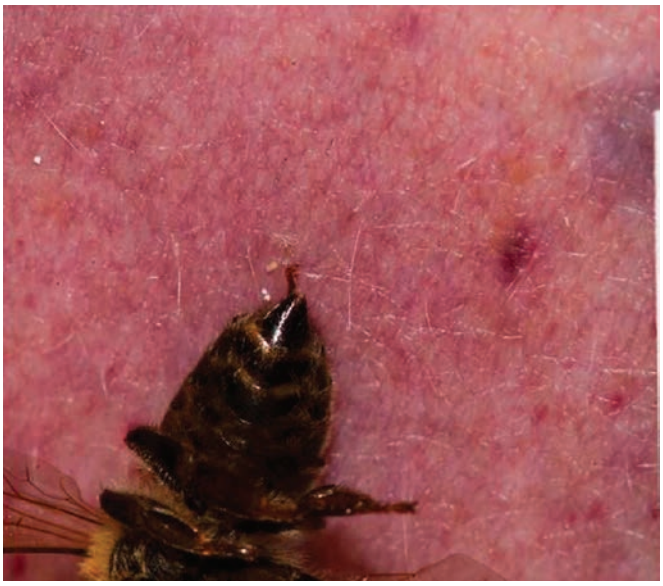




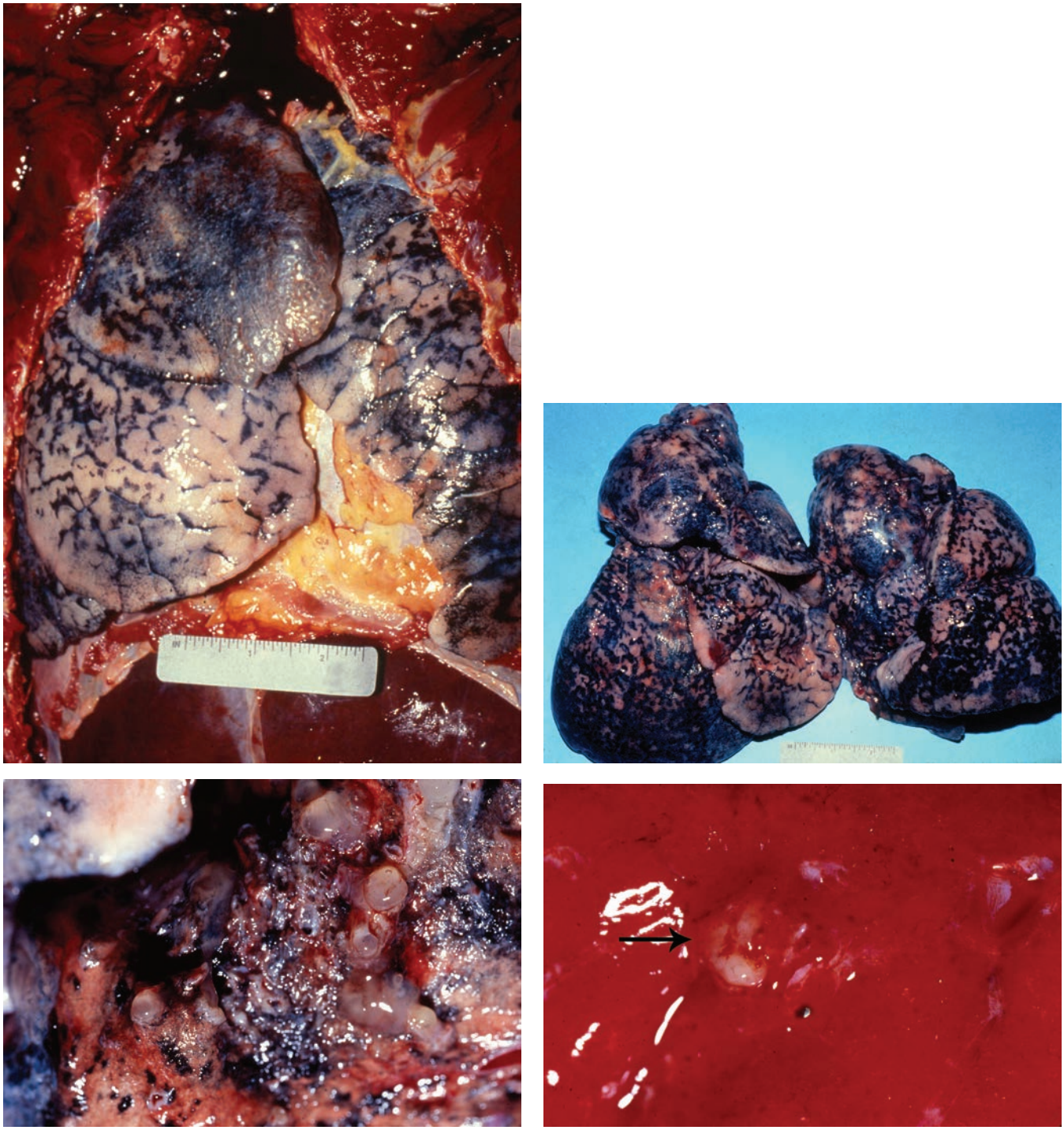
Figures 1.125 and 1.126 Acute epiglottis with airway obstruction due to *Haemophilus influenzae* type B infection. Note the swelling of the periepiglottic folds and the red/pink discoloration of the mucosa.



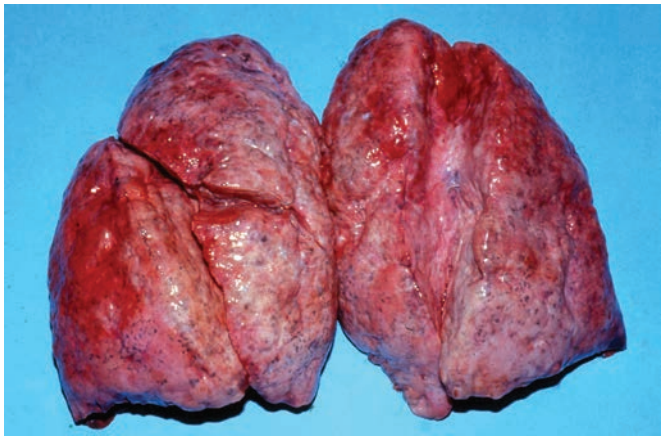


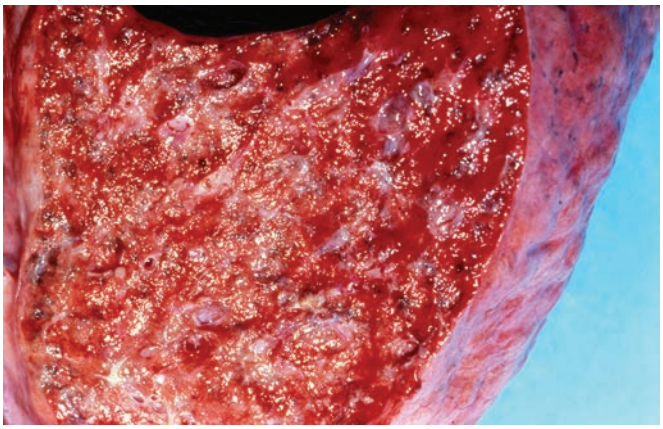


Figures 1.127–1.129 Bee envenomation associated with petechiae and laryngeal edema. Stung by over 100 bees, envenomation produced disseminated intravascular coagulation and anaphylaxis. Some of the bees had an "Africanized" profile.



Figures 1.130–1.133 These figures demonstrate acute and chronic bronchial asthma. Note the lungs within the thoracic cavity are hyperaerated and expand to overlie the pericardial sac. Upon removal of the lung, they appear markedly hyperaerated. If these lungs were placed on a water bath, they would float almost entirely on the surface. Cut section through the parenchyma reveals thick copious mucoid secretions within the bronchial distribution. Note Figure 1.133 shows darker red discoloration with congestion and edema besides mucous plugging in an individual who had an acute asthma attack with heart failure.





Figures 1.134 and 1.135 Chronic obstructive pulmonary disease (COPD) with hyperaeration and emphysematous change.

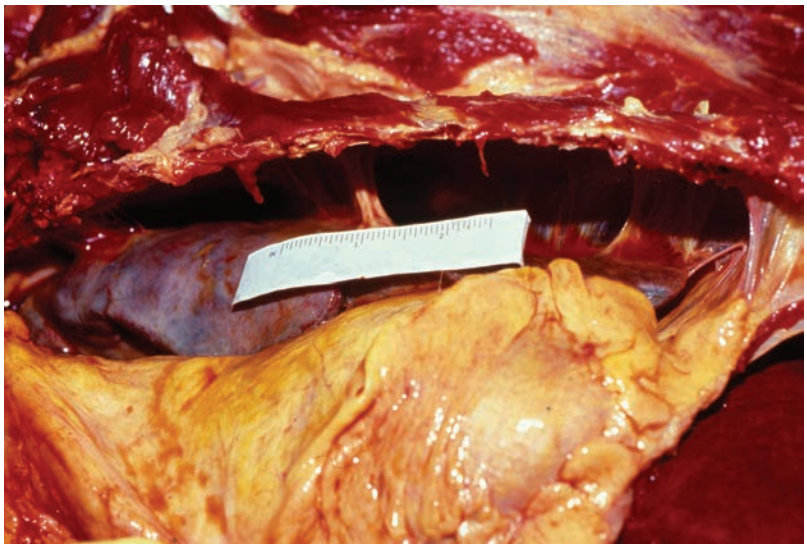


Figure 1.136 Pleural adhesions most likely due to past bouts of pneumonia.

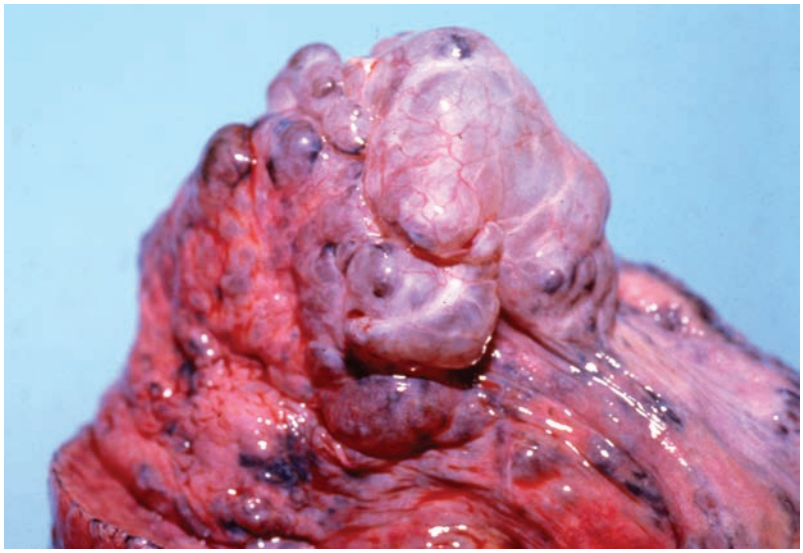


Figure 1.137 Bolus emphysema in a person with COPD. These bullae may occasionally rupture and cause a spontaneous pnuemothorax.

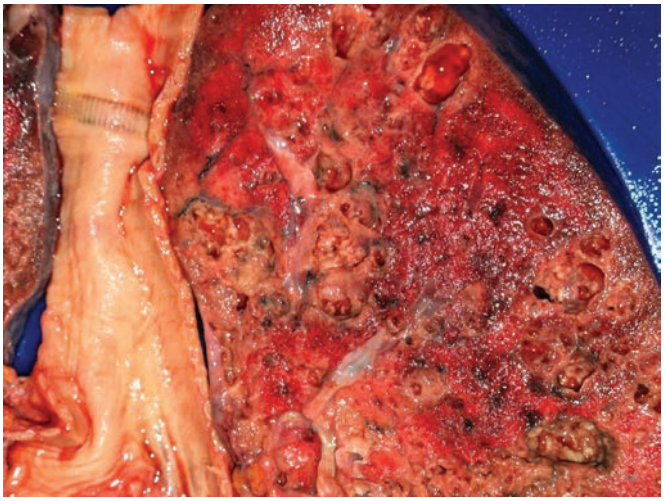


Figure 1.138 Acute or chronic *Pneumocystis* infection. The lung parenchyma is distorted by cysts filled with acute inflammation, fibrin, and organisms.

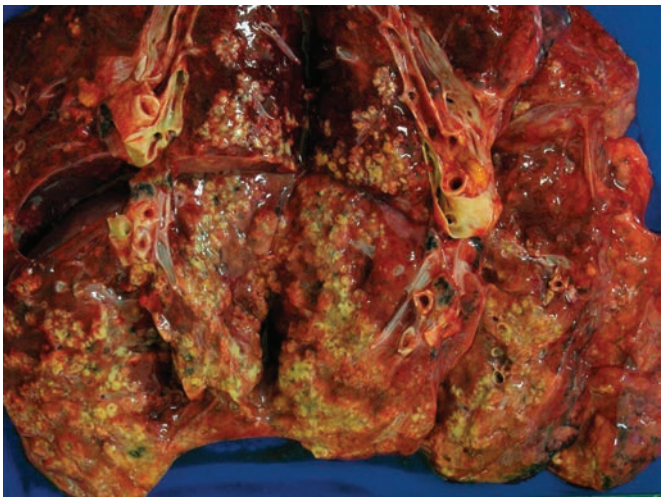


Figure 1.139 Disseminated tuberculosis, miliary pattern.

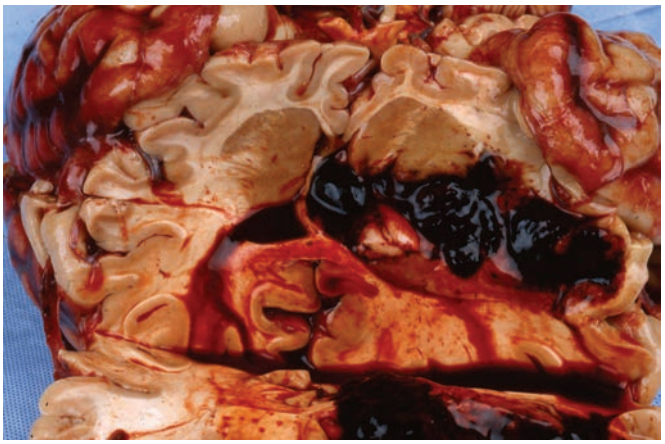


Figure 1.140 Hypertensive intracerebral hemorrhage. Mostly caused by rupture of a small intraparenchymal vessel.

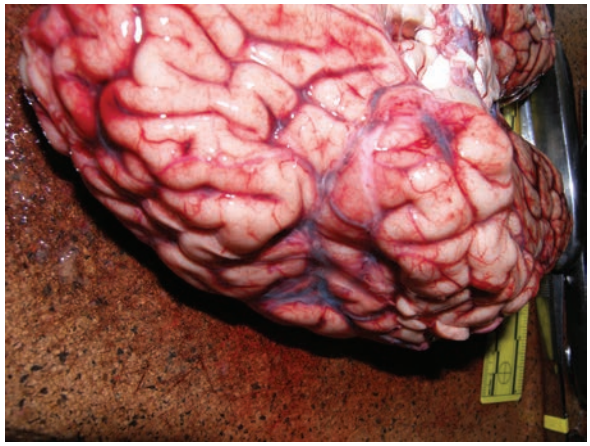


Figure 1.141 Old healed stroke. Note the indentation of the cerebral hemisphere.

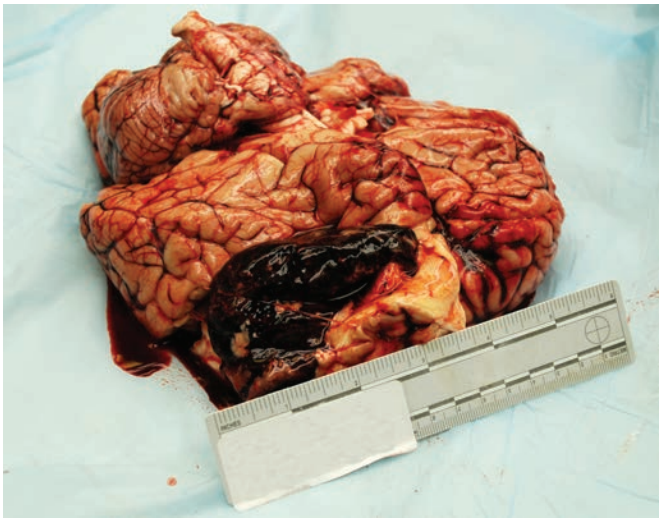


Figure 1.142 Intracerebral hemorrhage due to ruptured A-V malformation.

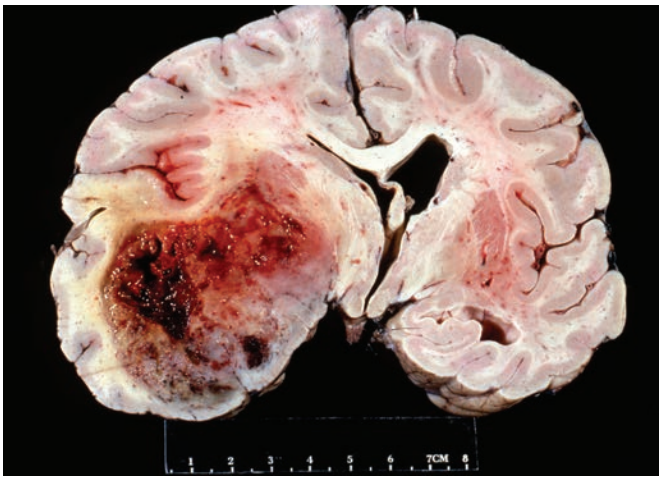
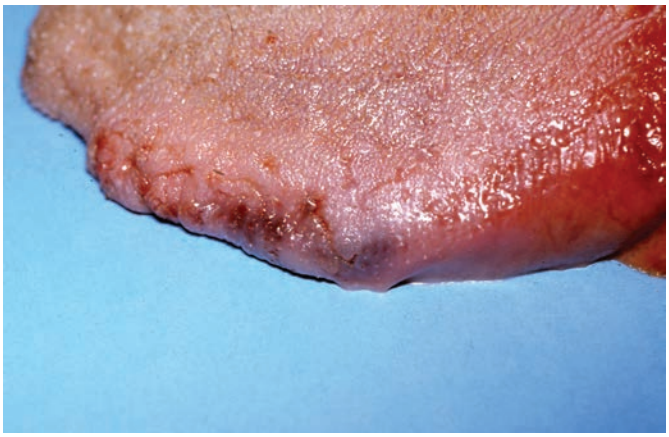
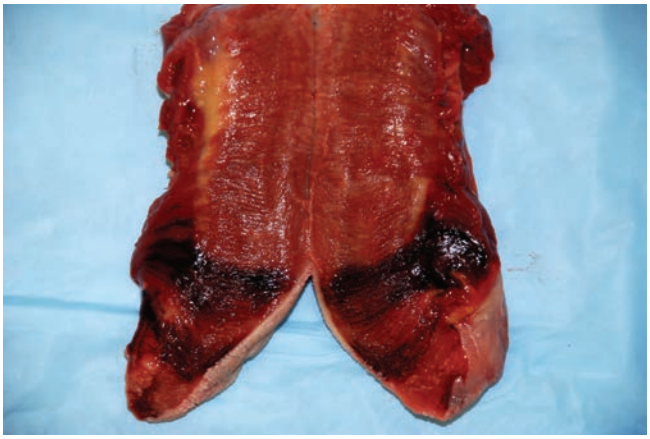
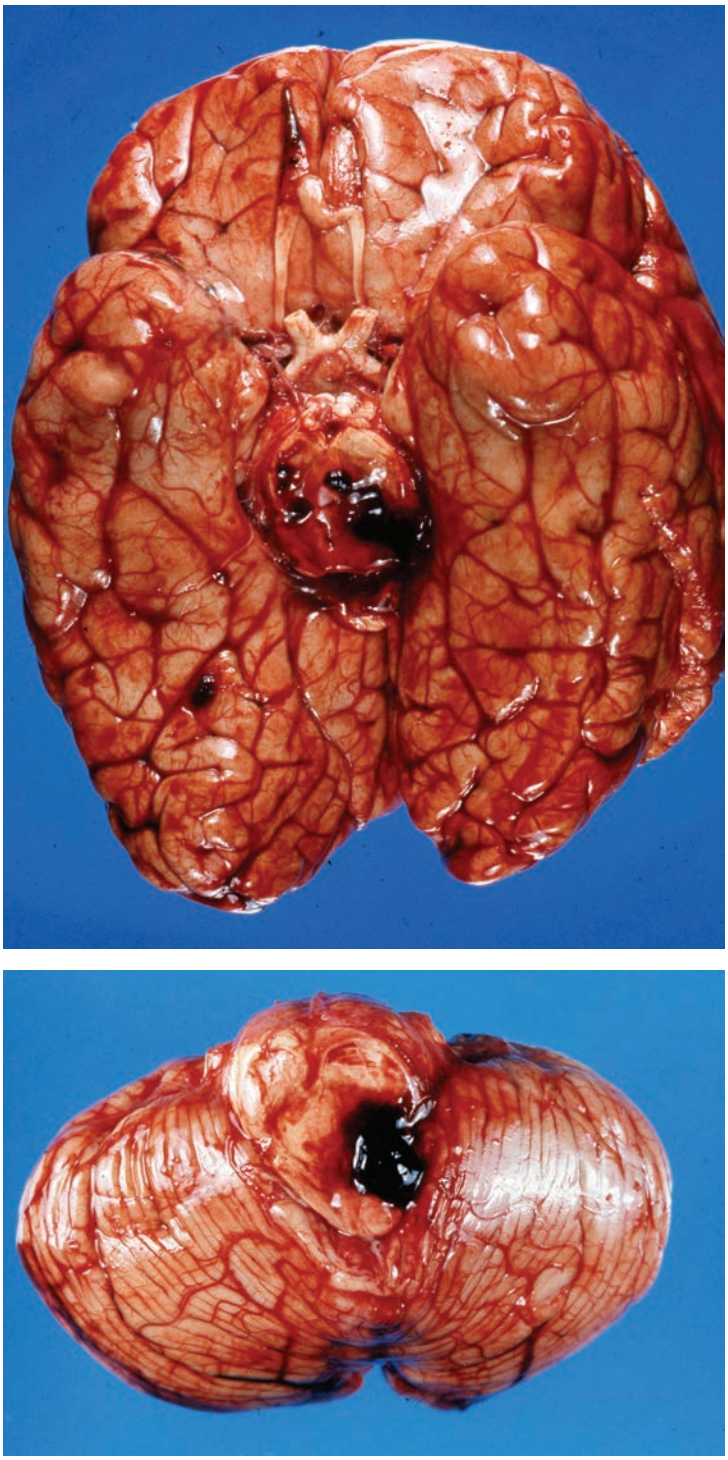


Figure 1.143 Astrocytoma causing significant compression of the surrounding structures.





Figures 1.144 and 1.145 Bite marks to tongue with hemorrhage from an individual who died of epilepsy. This was the only finding at autopsy.



Figures 1.146 and 1.147 Pontine hemorrhage due to hypertensive cardiovascular disease. This may be associated with locked-in syndrome where the person loses all voluntary motor control, particularly when the ventral pons are involved.

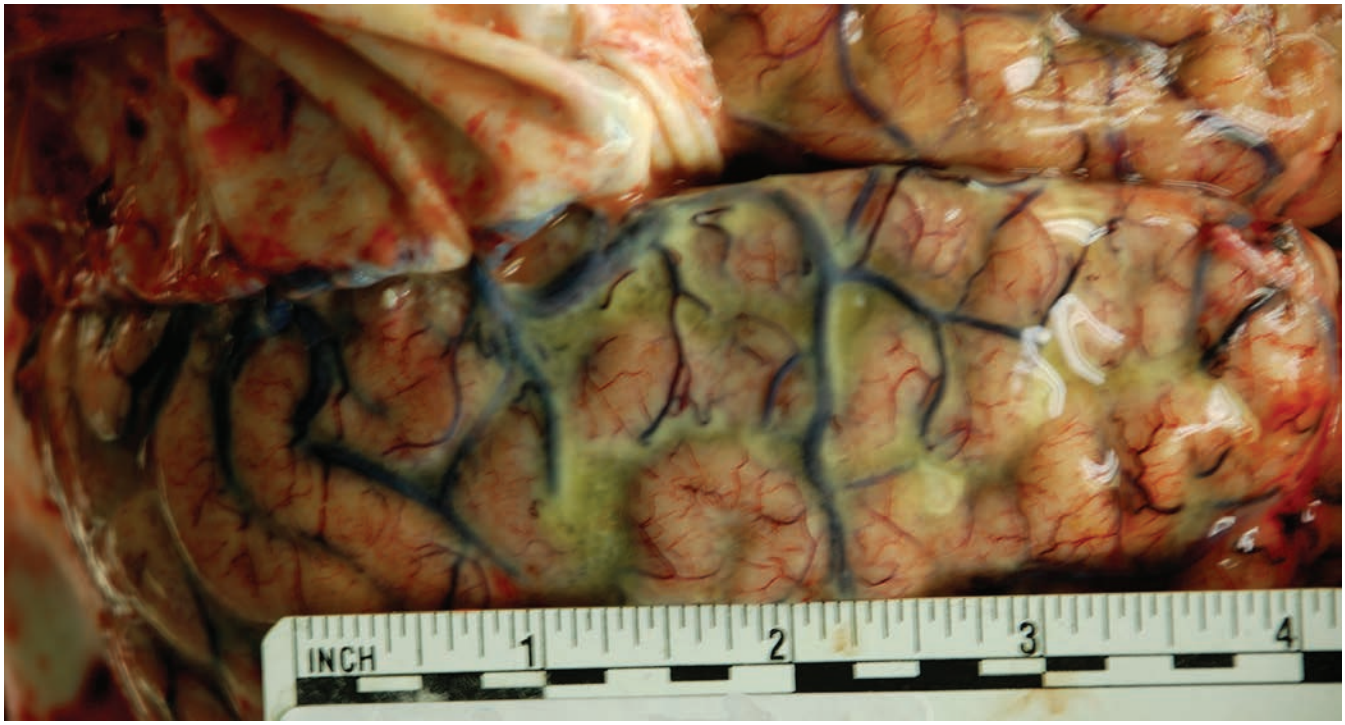


Figure 1.148 Bacterial meningitis. Note the yellow/green purulent exudate at the meninges.

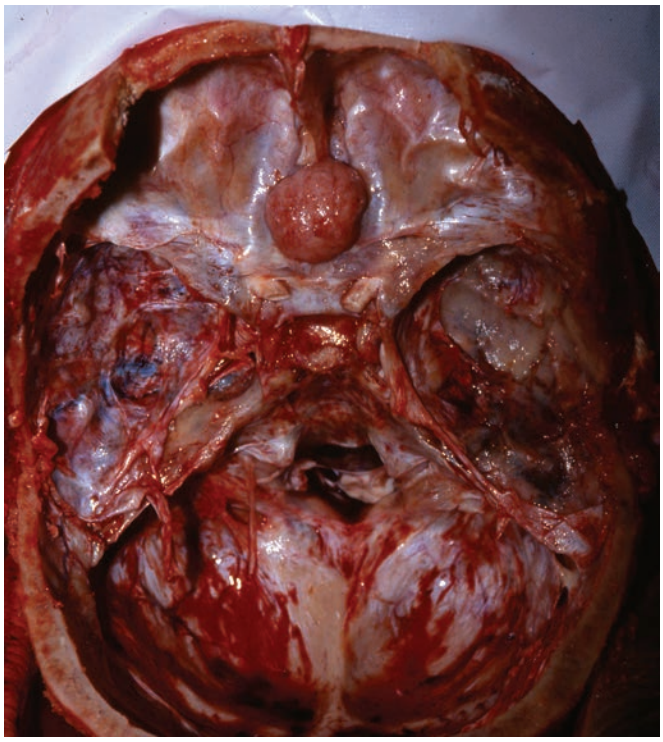


Figure 1.149 Anterior cranial fossa meningioma.

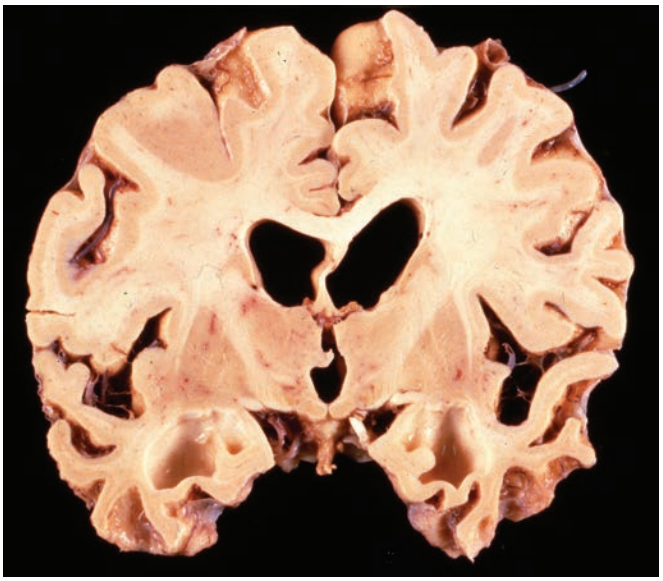
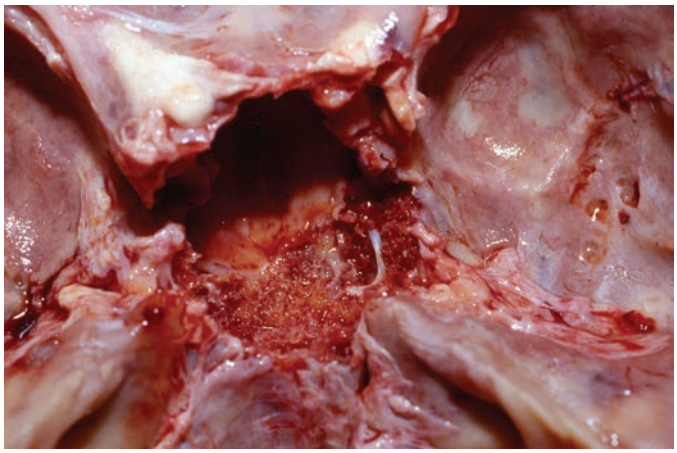
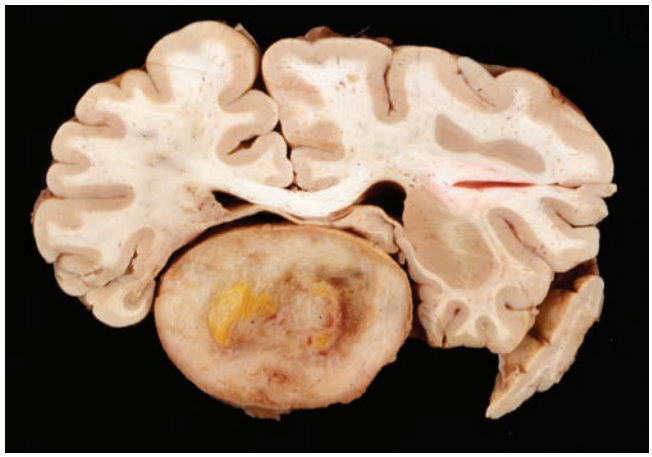
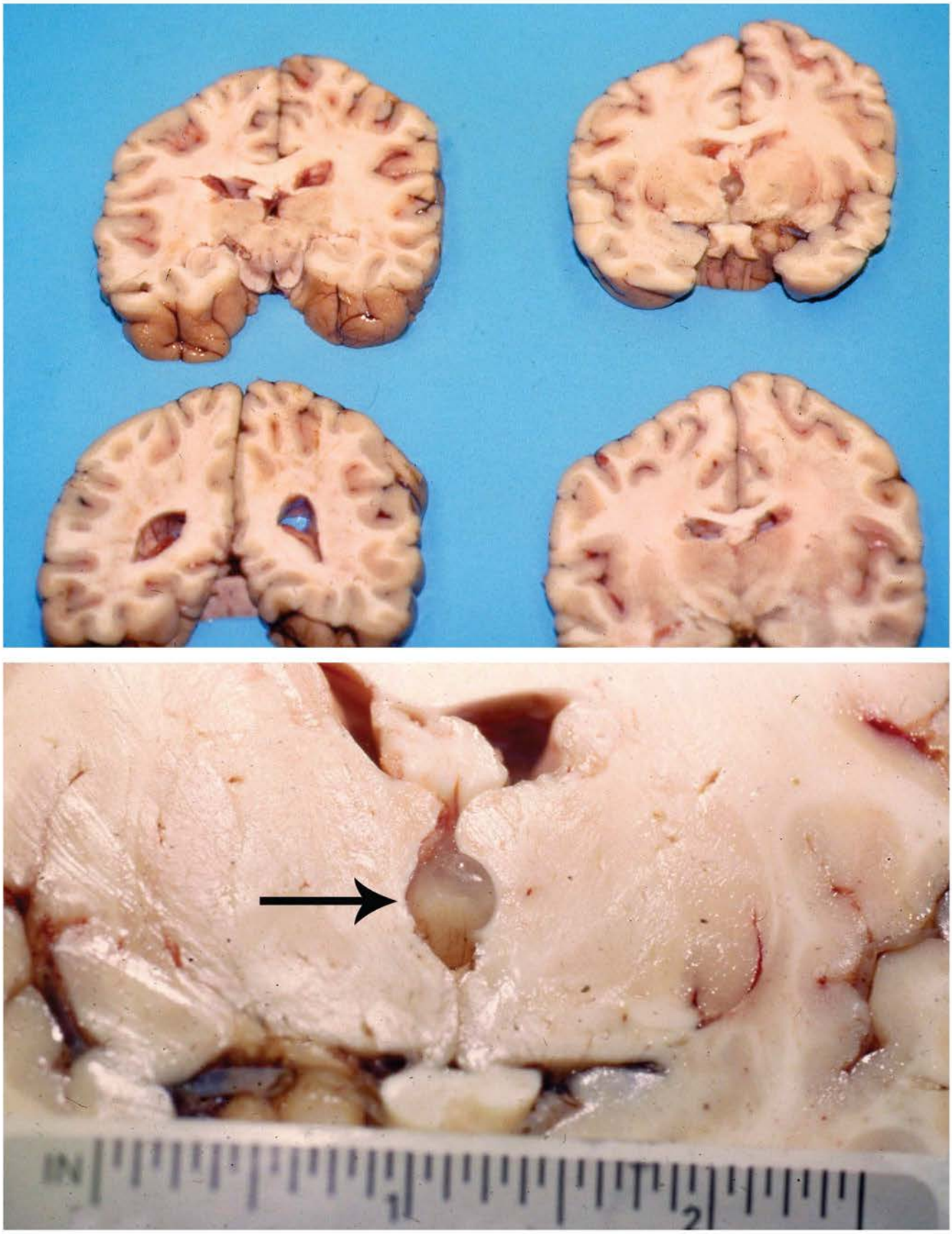


Figure 1.150 Pick disease. Note the asymmetric atrophy of the frontal, temporal, and parietal lobes. This form of chronic dementia occurs far less frequently than Alzheimer disease.

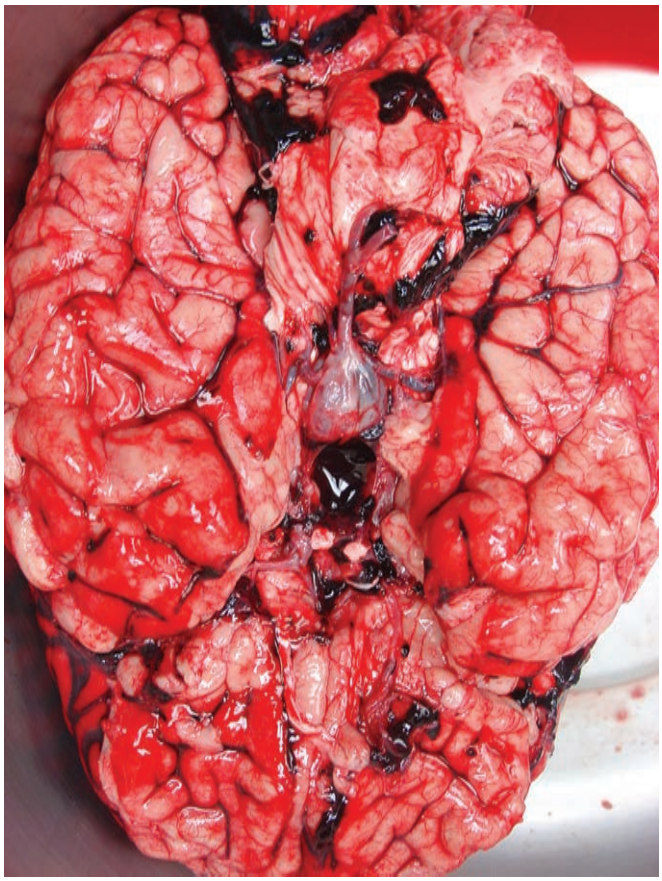


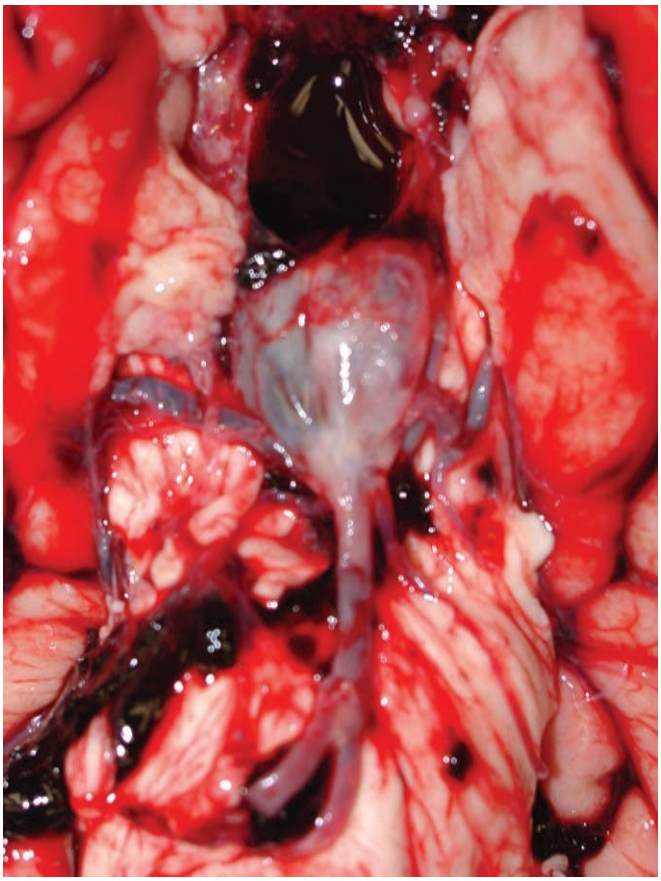


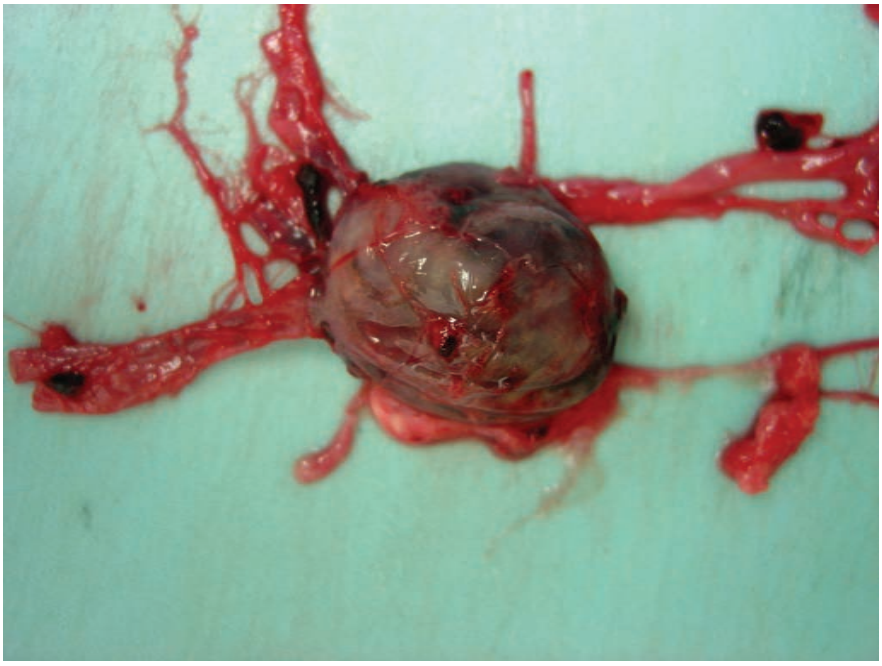
Figures 1.151 and 1.152 Two separate cases of pituitary adenoma. The first depicts a large erosion into the sella turcica. The second picture demonstrates a large adenoma viewed on a cross section of a cut formaldehyde-fixed brain. These adenomas may vary largely in size.



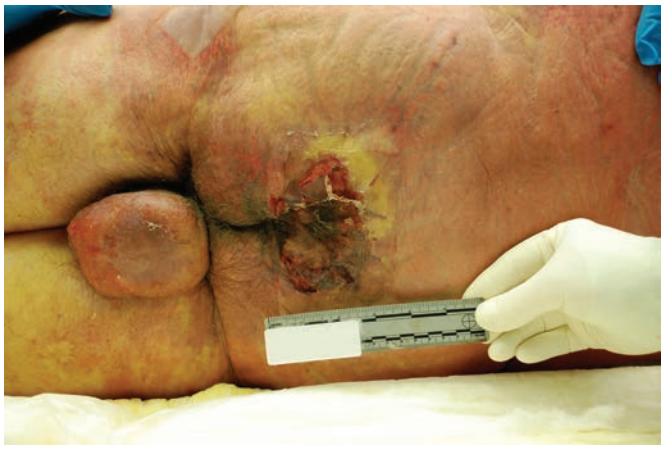
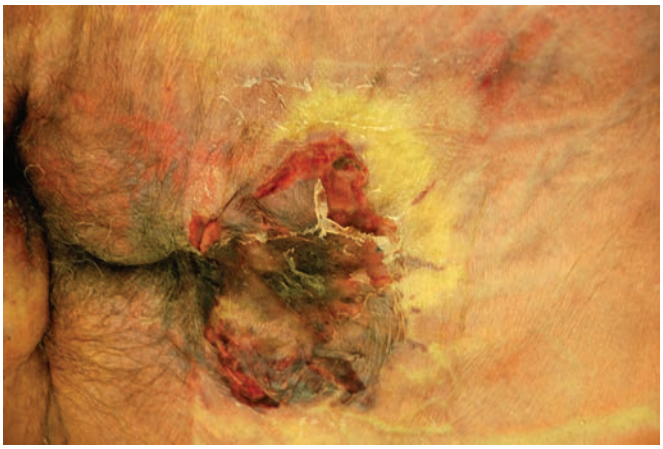
Figures 1.153 and 1.154 Colloid cyst of the third ventricle. This decedent had a history of severe headache with postural changes. This may be associated with sudden cardiac death following a buildup of cerebrospinal fluid pressures associated with central nervous system cardiac center disruption and fatal arrhythmia.







Figures 1.155–1.157 Sudden death associated with ruptured saccular cerebral artery aneurysm with subarachnoid hemorrhage. These are examples of giant berry aneurysms that are greater than 2.5 cm in greatest dimension. Small saccular aneurysms may rupture the same way. The jolt of increased pressure from arterial blood through the subarachnoid space at the base of the brain may disrupt the cardiac centers, causing fatal arrhythmia.

Figures 1.158 and 1.159 Decubital ulceration or pressure sore. This is often associated with poor nursing care but is also related to the degree of advancing disease associated with poor circulation and organ failure.

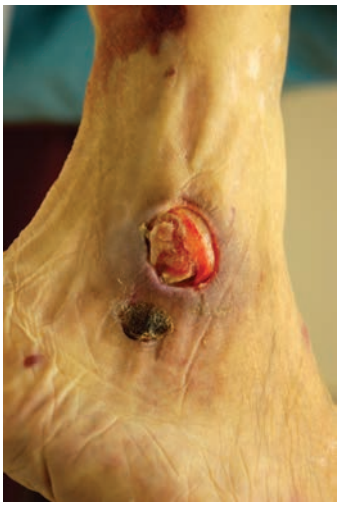
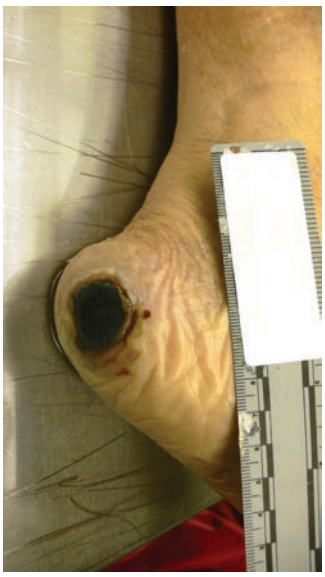
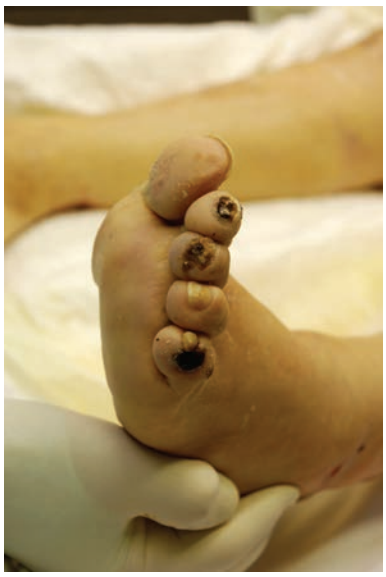
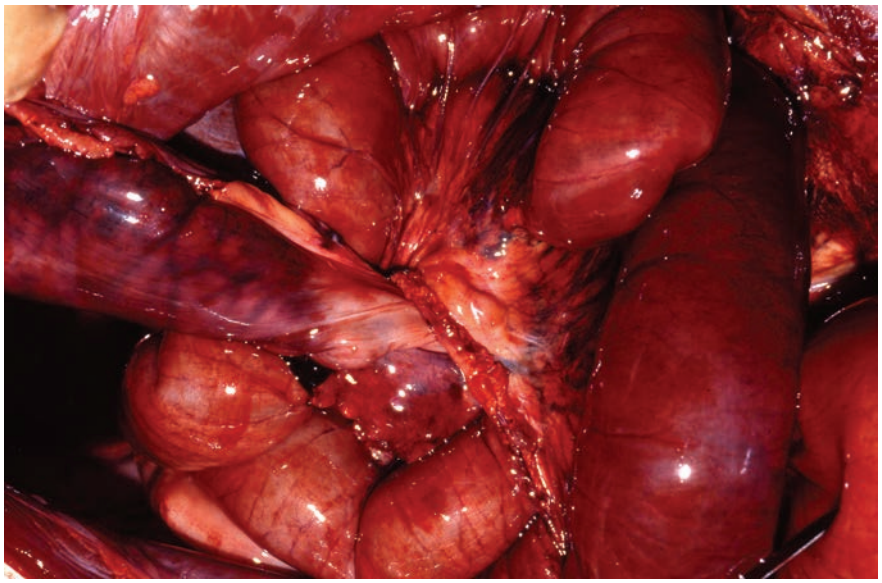
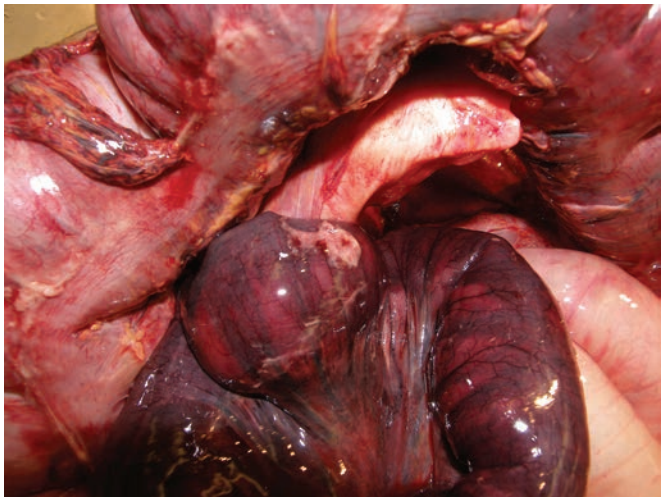


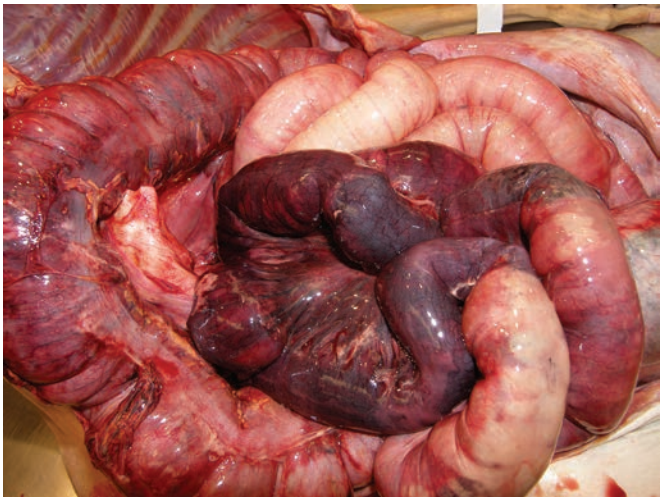
Figure 1.160 Healing ankle ulceration associated with peripheral vascular disease.

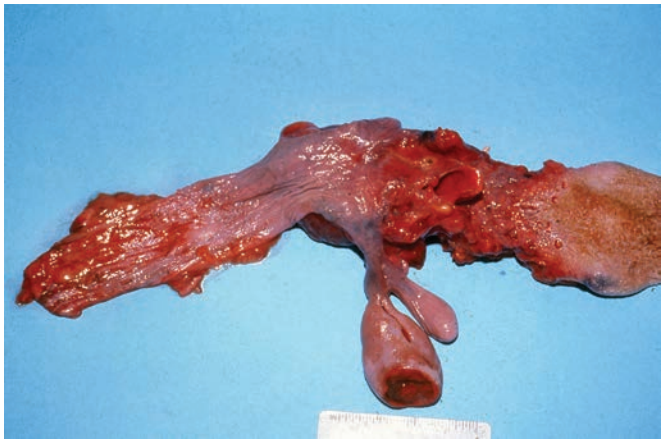
Figures 1.161 and 1.162 Dry gangrene associated with peripheral vascular disease due to long-term diabetes mellitus with pressure and/or rubbing from ill-fitting shoes or heavy blankets that further decrease blood flow.

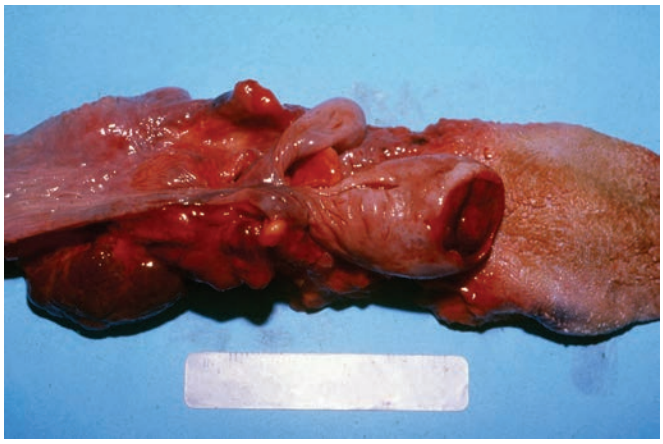




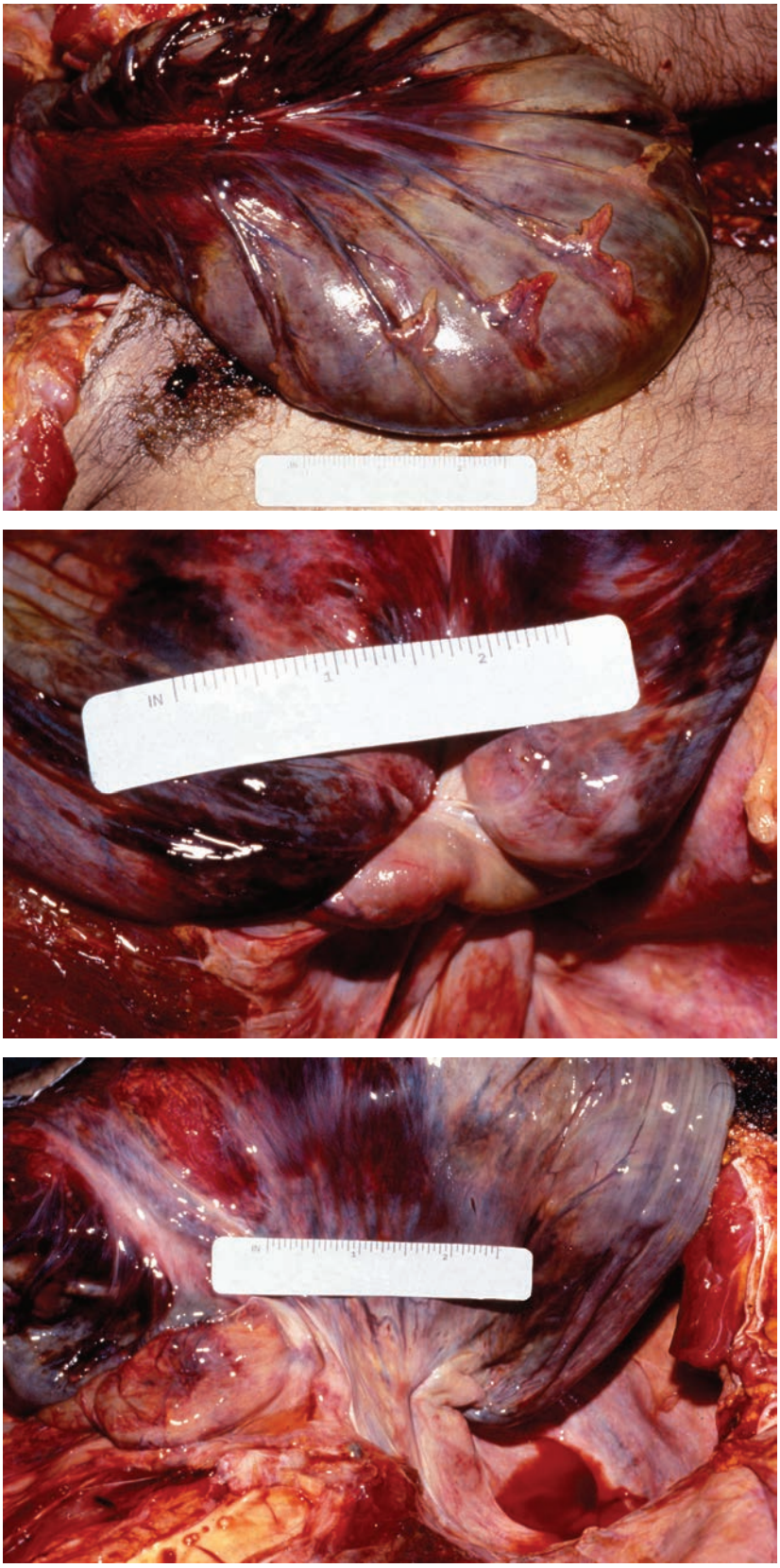


Figures 1.163–1.165 Ischemic bowel due to small intestine volvulus with vascular compromise. This segment of bowel twisted on itself and obstructed necessary blood flow. Note the early purulent exudate at the serosal surface.





Figures 1.166 and 1.167 This decedent had a long-standing history of difficulty swallowing. They were witnessed to gesture as though they could not breathe and then collapsed. Autopsy revealed a large benign esophageal polyp that obstructed the upper airway. Note the ulceration at the tip of the polyp from constant rubbing. During this last episode she was unable to clear the obstruction.



Figures 1.168–1.170 Cecal volvulus. Note the red discoloration of the serosal surface. Figure 1.168 shows cecal enlargement due to obstruction. Figure 1.169 shows the cecal volvulus with rotation and obstruction. Figure 1.170 shows this region untwisted.