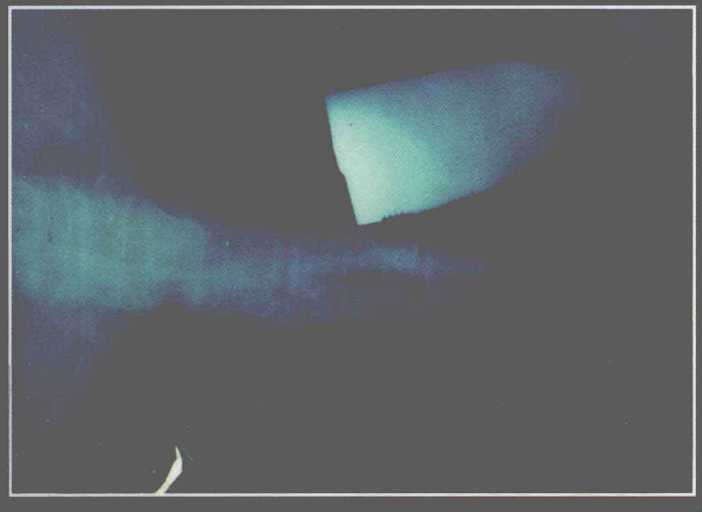
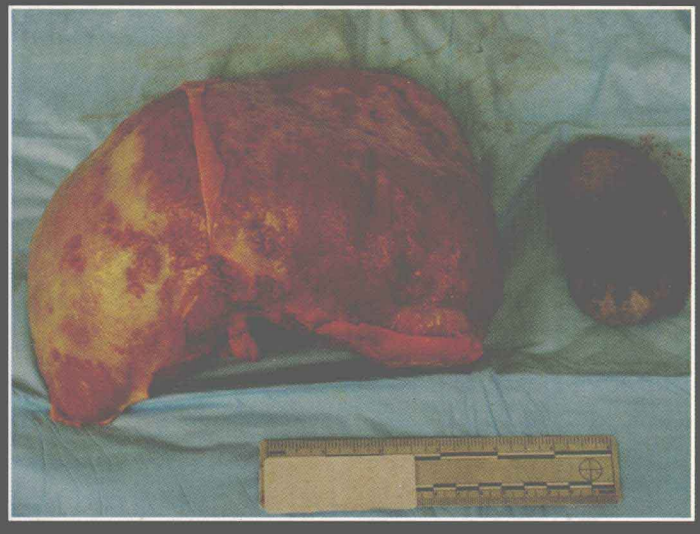
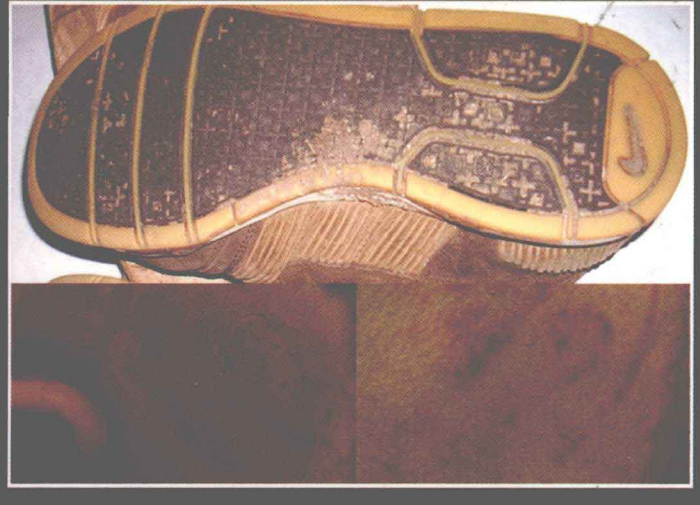
Color Atlas of Forensic Medicine and Pathology







Edited by
Charles A. Catanese

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Dedicated

This book is dedicated to the members of service who lost their lives as a result of the 9/11 attacks, and further dedicated to all those who suffered a loss associated with the aftermath.

Preface

While death investigation, and what we have come to understand as "forensic pathology," has been practiced in one way or another back to antiquity, official designation of forensic pathology as a subspecialty by the American Board of Pathology dates back only to 1956. The subspecialty comprises a small band of trainees in pathology and at any given time there are only 400 to 500 full-time practitioners of the specialty in the United States.

Furthermore, as a recognized subspecialty, forensic pathology is young enough that each of its full-time practitioners can trace his or her roots back to one of the six individuals who sat for that original examination back in 1956. For many of the authors of this volume, that family tree goes from Lester Adelson through Charles Hirsch and ultimately to us. We acknowledge and are proud of the fact that we stand on the shoulders of giants.

Our collective experiences as trainees and staff at the Office of the Chief Medical Examiner in New York City under the tutelage of Dr. Charles S. Hirsch has made us the forensic pathologists we are today. Our mentor's emphasis on precision and accuracy in description of findings, translation of these descriptions into language easily understood by a broad range of end users, and the public health importance of our work has left an indelible imprint that we desire to pass on to others. It is our hope that this atlas reflects these qualities.

With sincerest gratitude,

Thomas Andrew
Fellowship Class of 1992

Michael J. Caplan
Gerard Catanese
Bruce Levy
Fellowship Class of 1993

Thomas Gilson
Charles A. Catanese
Fellowship Class of 1995

Barbara K. Bollinger
Jonathan Lucas
Fellowship Class of 2000

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I would first like to thank my parents, S. John Catanese and Helen J. Amendola, and my grandparents, with a special thanks to my grandfather, Anthony J. Amendola, who taught me a strong work ethic and instilled a driving force to succeed while helping others. I would also like to thank my older brother, Anthony Catanese, MD, who first excited my interest in pursuing a career in medicine.

I would like to thank my judo sensei, Hank Kraff, who taught me how to defend myself, the nature of competition, and the value of pursuing an education.

I would like to thank my analytical chemistry professor at St. John's University, Richard E. Cover, PhD, whose kind nature and brilliant thought first excited my interest in the field of forensics.

I am grateful to Edward Laski, MD, PhD, who instilled in me an interest and understanding of psychiatry during medical school at SUNY Downstate, which led me to further understand different motivations leading to death.

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My fellowship training in forensic pathology was by Charles S. Hirsch, MD, at the New York City Office of the Chief Medical Examiner. My gratitude goes to Dr. Hirsch again for being such a great teacher and mentor.

I would also like to thank Maria Luz Alandy, MD, Joaquin Gutierrez Jr., MD, Thomas Andrew, MD, Gerard Catanese, MD, Kari Reiber, MD, Vernon Armbrustmacher MD, and Joseph Veress, MD for their patience in teaching and mentoring me as a junior attending in the New York City Medical Examiner system.

Thanks to Charles V. Wetli, MD, and Stewart Dawson, MD, who taught me many important concepts about forensic medicine while I worked for several years giving locums coverage at the Suffolk County (New York) Medical Examiner's Office.

I would like to thank all the NYC Medical Examiners, and the New York City Office of the Chief Medical Examiner for giving me access to many high quality academic images. More specifically, I thank Corinne Ambrosi, MD, Vernon Armbrustmacker, MD, Barbara Bollinger, MD, Stephen deRoux, MD, Thomas Gilson, MD, James Gill, MD, Lara Goldfeder, MD, Beverly Leffers, MD and Kristen Roman, MD.

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I would like to thank the Brooklyn District Attorney's Office and the New York City Police Department for always following through and maintaining the highest standard of work and professionalism in a harsh environment. Included in this is a special thanks to the Brooklyn North and South Homicide Divisions, with a very special thanks to Louis Savarese, Detective First Class, retired, and Terence Murnane, Detective First Class, retired.

Finally, I would like to thank the Orange County Executive, Edward Diana; the Orange County Commissioner of Health, Jean Hudson, MD; and the Orange County Deputy Commissioner of Health, Chris Dunleavy, for giving me the opportunity to serve the public and for helping me establish Orange County's first medical examiner system.

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, he 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 4000 autopsies, including more than 400 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 is currently the Chief Medical Examiner of Orange County New York, where he established and converted that office from a preexisting coroner system.

Contributors

Thomas Andrew, MD
Chief Medical Examiner
State of New Hampshire
Concord, New Hampshire

Barbara K. Bollinger, MD
Deputy Chief Medical Examiner
Orange County, New York

Michael J. Caplan, MD
Pediatric and Forensic Pathologist
Mercy Hospital-Cadillac
Cadillac, Michigan
and
Department of Pathology and Laboratory Management
Medical University of South Carolina
Charleston, South Carolina

Gerard Catanese, MD
Deputy Medical Examiner
Nassau County, New York
East Meadow, New York

Thomas Gilson, MD
Chief Medical Examiner
State of Rhode Island
Providence, Rhode Island

Laura M. Labay, PhD
Forensic Toxicologist
National Medical Services Laboratory
Willow Grove, Pennsylvania

Bruce Levy, MD
Chief Medical Examiner
President and CEO Forensic Medical
State of Tennessee
Nashville, Tennessee

Jonathan Lucas, MD
Deputy Medical Examiner
San Diego County, California
and
Associate Clinical Professor
Department of Pathology
University of California at San Diego
San Diego, California

Amy V. Rapkiewicz, MD
Director of Autopsy Service
Director, Anatomic Pathology
New York University/Bellevue Hospital
New York, New York

Research Assistants and Photo Imaging Contributors

Vanessa Cannone
Autopsy/Research Assistant
Orange County, New York

Jim Carlson
Department of Forensic Imagery and Radiology
New York City Office of the Chief Medical Examiner
New York, New York

Patrick Horan
Autopsy/Research Assistant
Orange County, New York

Salwa Jwayyed
New York City Police Department
New York, New York

Jaclyn McCarthy
Autopsy/Research Assistant
Orange County, New York

Jennifer McMahon
Medical Investigator
Orange County, New York

Tarin Romich, MPH
Osteopathic Medical Student
New York College of Osteopathic Medicine
Old Westbury, New York

Gina Marie Santucci
Department of Forensic Imagery
New York City Office of the Chief Medical Examiner
New York, New York

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Sudden Natural Death in a Forensic Setting

1

CHARLES CATANESE AND AMY V. RAPKIEWICZ

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 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 formaldehyde fixed state that can be used by the reader to compare with diseased organs.

Deaths under this category are often unexpected and sometimes unwitnessed. There is often 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, often 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, circumstance at 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 is defined in different ways. It may indicate a death that occurs within 24 hours of the onset of symptoms. It may also indicate the death occurred within 1 hour or even within seconds. There are not many diseases that can cause death within minutes of the onset of symptoms. Natural death means the manner of death is exclusively or 100% natural. If there is a 1% component of another manner of death, it is no longer natural. If there are multiple components of different manners of death commingled in a case investigation, the following rule will apply: a homicide overrides all, then an accident, then a therapeutic complication. A suicide requires the establishment of intent to do harm to oneself. 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 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 the 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 to create a lethal 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 life style.

Having 75% or greater blockage in any of the epicardial vessels is considered critical stenosis and is consistent with being alive one 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. Although hypertensive 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 myocardial irritability and fatal (tachy)arrythmias.

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 tamponode 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, particularly athletes, hypertrophic cardiomyopathy is not an uncommon cause of sudden death. These patients can be asymptomatic prior to the sudden event or may have past episodes of palpitations or syncope. Typically, 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.

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 and can be a clue to the underlying 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 disease 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.

S. 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 develops 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 infective endocarditis, 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 reentry 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 is suggesting that 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 congentially (uni)bicuspid valves. The mechanism for death in severe aortic stenosis (valve area <1cm2) 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 mucopolysacchrides.

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 take off, there maybe ridge like defect at the coronary ostea further decreasing blood flow in times of accelerated heart rates with increased oxygen demand. Myocardial tunneling is another anomalous coronary artery distribution that maybe 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 aneurism, 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's syndrome also make up a significant 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 to 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 subarachnoid hemorrhages 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 terms "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 to 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. Infiltration or edema formation into the key respiratory/cardiac centers of the brain with possible herniation are two mechanisms. 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 brochospasm 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 mucus 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 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 peaks approximately 15 to 60 minutes after the onset of anaphylaxis and then declines with a half-life of about 2 hours.

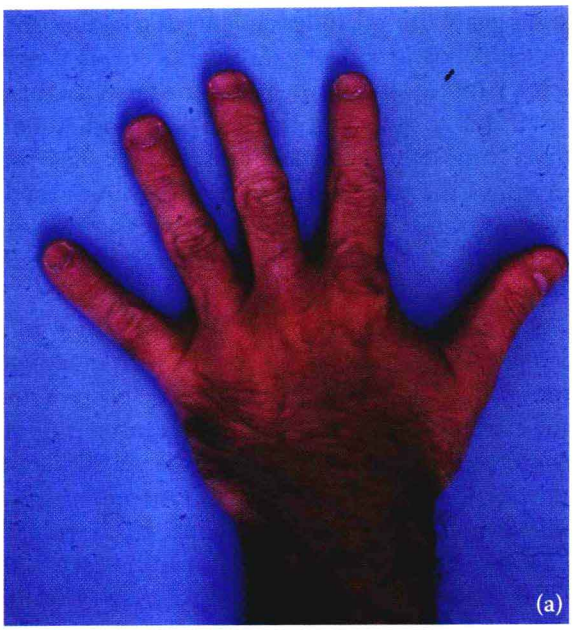
The mortality for gastrointestinal bleeding (GI) in the case of ruptured esophageal varices most commonly encountered in patients with portal hypertension is high. Intra-aortic balloon pumps are lifesaving procedures but only if the patient presents in a timely fashion. Other causes of fatal upper gastrointestinal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

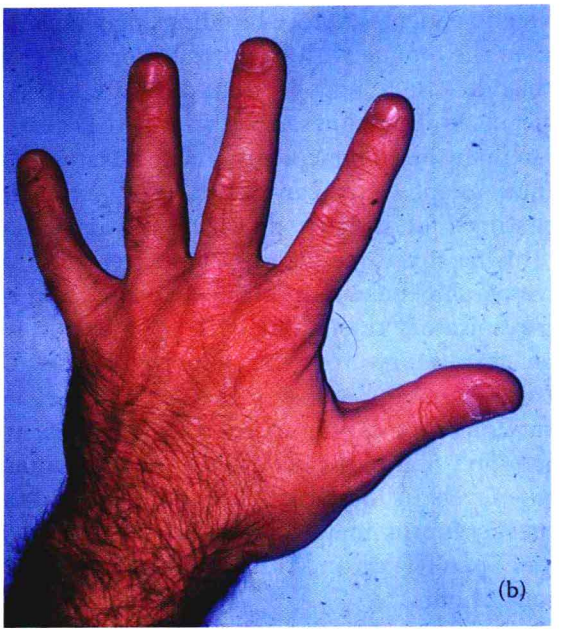
Mostly complications of morbid obesity are thought to underlie the association with sudden death. Hypertension, left ventricular hypertrophy, and cardiomegaly are all independent risk factors for sudden death. Postural asphyxia may occur as a result of obesity. Morbid obesity is a reasonable cause of 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.

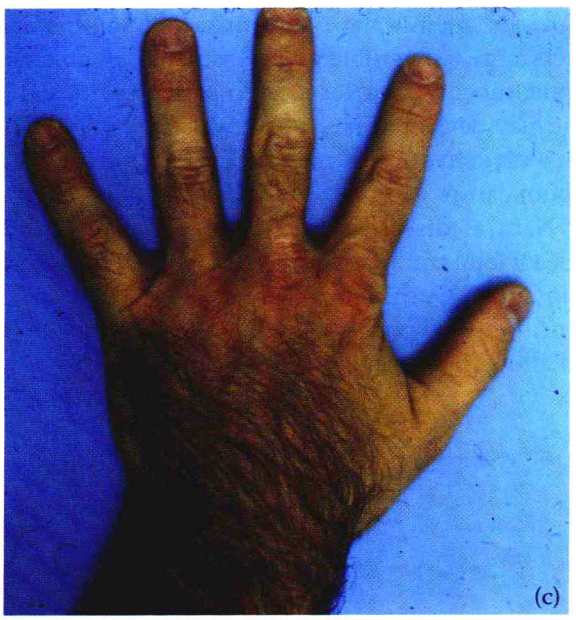
Waterhouse–Friderichsen syndrome was first described as occurring in patients with meningococcemia and is characterized by severe bacteremia and bilateral adrenal hemorrhages. This combination results in overwhelming shock and, if untreated, sudden death can occur. Organisms other than *N. meningitis,*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

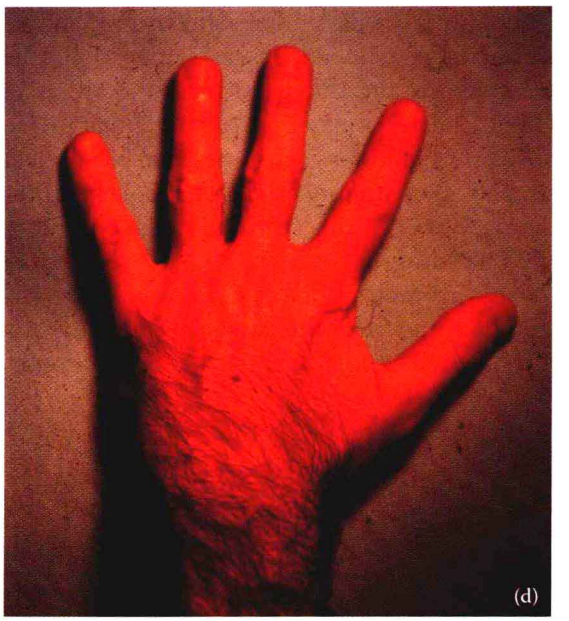
Multiorgan failure and death can be seen in sickle cell anemia patients with an acute crisis. Precipitants may include infection, dehydration, hypoxia, physical excretion, vaso-occlusion, or fat embolus following bone infarction. This acute hemolytic sickling crisis results in severe hypoxemia with end organ failure. Patients with sickle cell anemia have auto-infracted 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.

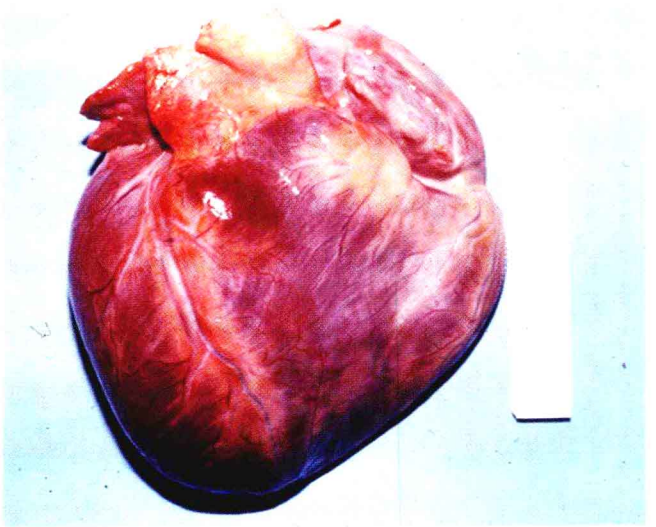




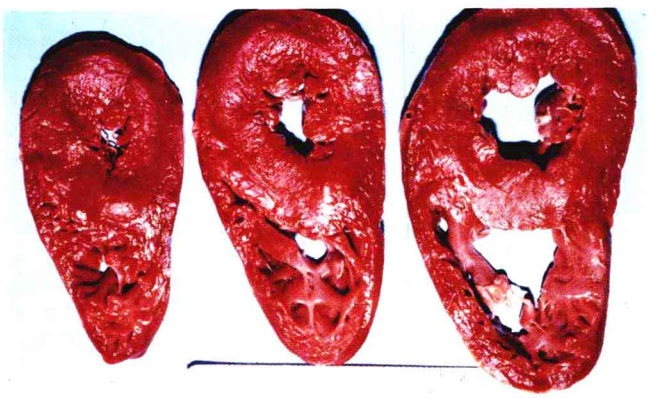




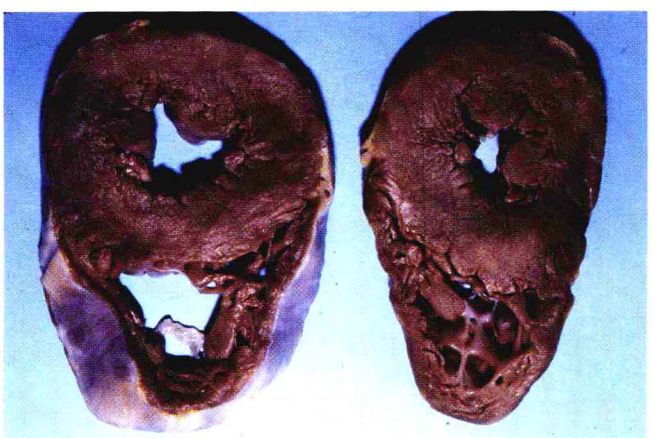
When examining photographic evidence it may be important to have knowledge of the type of camera, film, and lighting used when documenting different disease states. These four photos demonstrate different types of lighting causing variation in picture color. Figure (a) was taken in overcast sunlight, (b) with camera flash, (c) under fluorescent light, and (d) with a Tungsten filament regular light bulb. If your opinion is that a photographic image is not interpretable, it is perfectly acceptable to say that you cannot render an opinion based on this two-dimensional image.



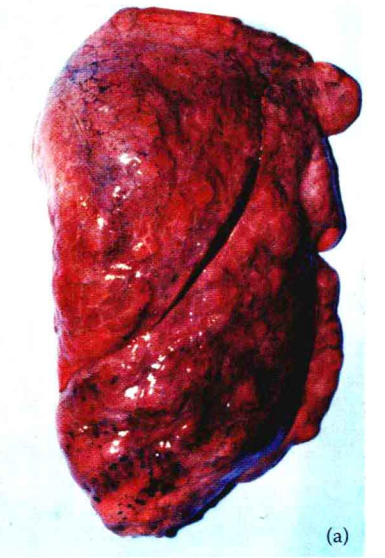
Normal fresh heart.

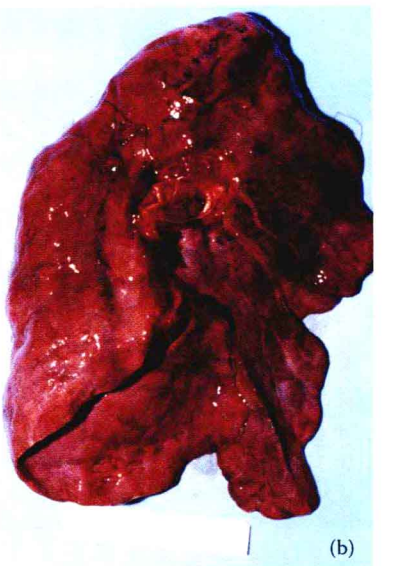


Sections of a normal fresh heart showing right and left ventricle.



Sections of normal right and left ventricle after formaldehyde fixation.





Normal fresh left lung (a) demonstrating two lobes. Right lungs (b) have three lobes.

Normal lung fixed in formaldehyde.