Sports-Related Injuries and Deaths

26

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Abstract

Physical activity in children and adolescents should be strongly encouraged. While there is a very low risk of death associated with participation in athletics within this age group, the epidemic of childhood obesity and sedentary lifestyle

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must be combated to ensure the long-term health and quality of life of today's youth. Sports-related deaths due to trauma are usually readily identified; others require careful examination, adjunctive testing, and/or the expertise of consultants. A thorough investigation of circumstances surrounding the death, review of the medical records, and autopsy is mandated in these cases.

Introduction

The loss of a child is always tragic. When death comes to a young person in peak physical condition engaged in athletics, the fatality strikes a blow to an entire community and often attracts the attention of the national media. In some cases, screening studies or training modifications could have prevented the end result. In many others, however, these sudden, unexpected deaths are the result of conditions that cannot reasonably be anticipated or avoided.

The benefits of physical activity in young people are incontrovertible. In fact, a presidential initiative has focused on the necessity for solid nutrition and exercise among American youth. Physical activity is essential to well-being, and it must be encouraged in children. Childhood obesity has dire health consequences and creates a pattern that can result in significant morbidity and mortality in later life (see ▶ Chap. 33, "Childhood Obesity"). That being said, engagement in athletics can result in serious injury or death, albeit very infrequently.

Deaths secondary to trauma are fairly self-explanatory so only a brief overview is in order. Much of this chapter will focus on natural disease processes and pathologic conditions that can present as sudden death while a child or adolescent is involved in physical activity.

Definitions

A sports-related fatality is one in which death occurs while the participant is engaged in athletics. Within this broad category, death can be directly attributed to an injury received during the activity in which case the manner of death is best certified as an "accident" provided that the trauma was received in accordance with the rules of the sport. In the case of death due to trauma inflicted flagrantly outside of the rules of the sport or if the lethal injury was intentionally inflicted, a manner certification of "homicide" may be more appropriate. In many cases, sudden death may be the result of physical stress superimposed upon a natural disease process or pathologic condition, often involving the heart. In this setting, the manner of death should be certified "natural," analogous to myocardial infarction occurring in an older person engaged in physical exertion (Froede 2003).

Traumatic Death

Head and Neck Injuries

The sequelae of repetitive blows to the head have attracted much attention in recent years (Omalu et al. 2005, 2010). While the manifestation of repeated concussions usually appears in middle age or later, it is probable that the damage begins when the brain is first jarred with resultant alteration in mental status that is characteristic of a concussion. What is certain is that concussions must be treated as a serious medical condition, and vigilance is necessary to ensure the safety of participants who are in a post-concussive state. The clinical entity known as the "second impact syndrome" (SIS) can cause morbidity and mortality following head injury (Bey and Ostick 2009). SIS is comprised of two events: (1) a concussive head injury and (2) a second head injury within several weeks followed by cerebral edema, herniation, and death. Although the incidence is arguable and is yet to be firmly established, it is thought to be a rare outcome of head injury (Bey and Ostick 2009). In any event, any athlete who manifests concussive symptoms following a head injury (e.g., fatigue, confusion, headache, nausea, vomiting) should be closely observed and not be permitted to return to play for 7–14 days (Bey and Ostick 2009) (Table 26.1).

Most serious head injuries occur in the traditional contact sports. They run a spectrum that includes superficial lacerations, contusions, and abrasions to skull fractures, cerebral contusions, deep axonal injury (in the delicate white matter of the brain), and intracranial bleeding.

When a human head collides with another object, skull fractures may occur. Protective gear, such as helmets, minimizes this risk in many sports. In an unprotected head, skull fractures may be associated with tearing of arteries within the bones of the calvarium, including the middle meningeal artery, resulting in epidural hematomas (EDH) that may evolve rapidly and compress the underlying brain. Cerebral contusions may also occur at the fracture site. These types of injuries are surgical emergencies. With a more significant direct impact to the head, an open fracture may occur with resultant direct injury and extrusion of the brain. Impacts to the face can result in fractures with resultant compromise of the upper airways.

Deceleration injuries to the head can also be devastating. When a moving or falling head makes contact with a firm surface, injuries to the brain and intracranial bleeding may occur. In contradistinction to the cerebral contusions and/or fractures directly subjacent to the site of impact of a moving object with a stationary head, deceleration injuries may be associated with contrecoup cerebral contusions. These lesions are located opposite to the site of impact of a moving head with a stationary surface. Common locations for contrecoup cerebral contusions are the inferior aspects of the frontal lobes and the anterior temporal lobes. Contrecoup contusions may or may not be accompanied by basilar skull fractures. Rapid deceleration of the cranial contents can also result in diffuse axonal injury (DAI) in the white matter and intracranial hemorrhage. Subdural hematomas (SDH) secondary to venous

Table 26.1 Guidelines for the management of sports-related concussion (Adapted from Bey and Ostick 2009)

Symptoms	First concussion	Second concussion
Grade 1: no loss of consciousness, transient confusion, resolution of symptoms, and mental abnormalities in <15 min	Remove from play. Examine at 5-min intervals. May return to play if symptoms disappear and results of mental function exam return to normal within 15 min	Allow return to play after 1 week if there are no symptoms at rest or with exertion
Grade 2: as above, but with mental symptoms for >15 min	Remove from play for rest of day. Examine for signs of intracranial lesion at sidelines and obtain further examination by a trained person the same day. Allow return to play after 1 week if neurological examination is normal	Allow return to play after 2 weeks of no symptoms at rest or with exertion. Remove from play for season if imaging shows abnormality
Grade 3: any loss of consciousness	Perform thorough neurological exam in hospital and obtain imaging studies when indicated. Assess neurological status daily until post-concussive symptoms resolve or stabilize. Remove from play for 1 week if loss of consciousness lasts seconds and for 2 weeks if it lasts minutes; must be asymptomatic at rest and with exertion to return to play	Withhold from play until symptoms have been absent for at least 1 month

bleeding following rapid deceleration of the head can result in a potentially lethal increase in intracranial pressure that must be aggressively managed.

With a whiplash type of motion or significant hyperextension of the neck, severe injuries to the cervical spine and underlying spinal cord can occur (Watanabe et al. 2010) resulting in paralysis, respiratory arrest, hemodynamic instability, or death. This type of injury can be seen in violent collisions between bodies, after being ejected from a moving vehicle or animal, or upon impact with the ground while the body is tumbling or rolling. Violent impacts to the face, as seen in boxing, can also cause the head to snap back or rapidly rotated with laceration or dissection of the vertebral arteries and subsequent subarachnoid hemorrhage (Nedeltchev and Baumgartner 2005).

Special Techniques: Head and Neck Injuries

At autopsy, the pathologist should remove the brain, cerebellum, pons, and medulla and may choose to preserve the block in formalin to permit careful sectioning after 2 weeks. If cervical injuries are anticipated, the pathologist should employ anterior and posterior neck dissections and/or vertebral artery dissection for accurate evaluation.

Thoracoabdominal and Pelvic Injuries

Injuries to the ribs and internal organs with subsequent internal bleeding can occur with significant impact to the chest, back, or abdomen. While rib fractures can be debilitating and painful, they are not usually lethal unless there are associated vascular or visceral lacerations or collapse of the lung and pneumothorax.

Internal bleeding is most often associated with lacerations of the spleen or liver following an impact. The bleeding may occur over a matter of hours or days, so a careful history may be required to establish that the injury occurred during participation in a sport. The spleen is particularly prone to injury if enlarged due to infectious mononucleosis, so infection with Epstein-Barr virus should be considered if rupture occurs after relatively trivial impact. Direct impacts to the abdomen can also injure the mesentery, pancreas, or gastrointestinal tract. The kidneys, being relatively protected by their retroperitoneal position and the presence of a thick fat pad, are injured less frequently.

A well-established cause of death in athletes receiving a precordial impact is commotio cordis (Westrol et al. 2010; Geddes and Roeder 2005). Death is the result of a lethal dysrhythmia related to a blunt force impact to the chest occurring at a vulnerable phase in the cardiac cycle. Classically, the athlete is struck in the mid-chest by a projectile (e.g., a baseball) and collapses within a matter of seconds. Reconstruction of the events leading up to death is required to establish this diagnosis as there may be minimal or no anatomic signs to establish chest trauma and the mechanism of death is transient disruption of impulses within the cardiac conduction system.

Injuries to the pelvis are unusual in contact sports. However, a significant impact to the perineum, a situation that may occur during riding events (e.g., riding cycles, motorbikes, horses), may cause pelvic fractures, injuries to the genitourinary tract, and internal bleeding. Impacts to the scrotum and penis can also result in significant pain and morbidity, but they are rarely life threatening.

Injuries to Extremities

Fractures, sprains, strains, and dislocations are commonly encountered in sports, but these injuries are also rarely life threatening. While in a prolonged debilitated state during rehabilitation, however, thrombi may develop in the deep veins of the lower extremities which may embolize to the lungs resulting in sudden death. If a preexisting coagulation disorder is present, the risk for deep venous thrombosis is increased. Smokers and female athletes on birth control pills may be at greater risk for this complication.

Fractures can lead to embolization of marrow elements, predominantly fat, throughout the body (Fig. 26.1). In addition to the problems associated with physical obstruction of vessels by large emboli, disseminated intravascular coagulation (DIC) and activation of chemical mediators can result in death (Hofmann et al. 1995). The fat embolism syndrome typically occurs 24–72 h following a long bone fracture or

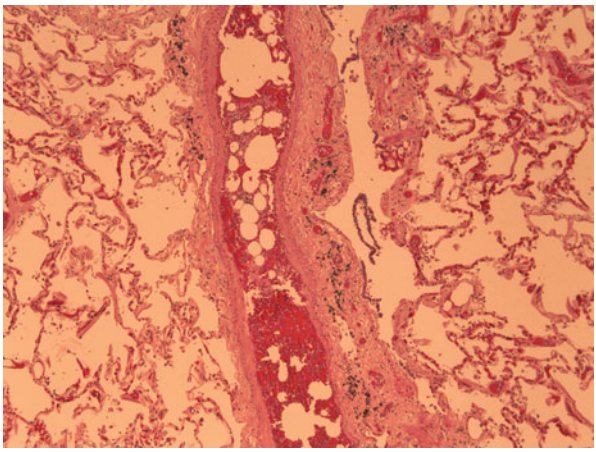

Fig. 26.1 Fat and bone marrow elements may embolize from fracture sites to the lungs. This may be a cause of death, an artifact of trauma, or secondary to cardiopulmonary resuscitation (Hematoxylin and Eosin, H&E × 10)

crush injury presenting as Adult Respiratory Distress Syndrome (ARDS). Deep trauma to adipose tissues can also cause fat embolization. Of note, fat and marrow emboli are often the result of cardiopulmonary resuscitation with associated rib and/or sternal fractures, so clinical correlation is required. Soft tissue injuries have also been associated with the development of "flesh eating" (Group A Streptococcus) bacterial infections, even with no breach of the integument (Chang et al. 2009). It should be remembered that even if death is due to a natural disease process, such as sepsis or pneumonia, the manner of death should be certified as "accident" if trauma initiated the chain of events that culminated in death.

Diet, Nutrition, and Drugs

Sport participants are often under intense pressure to perform at a high level, and they may be encouraged to maximize performance through the use of supplements and dietary modification. This is not only true of professionals but also of amateurs and individuals as young as preadolescents. When investigating the death of an athlete, a complete dietary history, including inquiry into the use of chemicals, vitamins, herbal supplements, and performance-enhancing drugs, should be obtained and any such substances procured.

Anabolic steroids have been historically used to facilitate strength and speed increases, muscle hypertrophy, and decreased recovery time and to generally improve athletic performance (Hartgens and Kuipers 2004). Pathologic changes manifested following anabolic steroid use may be appreciated on external examination and can include testicular atrophy, male pattern alopecia, male gynecomastia, masculinization, breast size and body fat decreases, clitoral enlargement, acne, and hirsutism in females (US Department of Health and Human Services National Institute on Drug Abuse 2001). Anabolic steroid use can result in peliosis of the liver, psychiatric instability, cardiomyopathy, and death

(Hartgens and Kuipers 2004). Recently, anabolic steroid use has been implicated in suicides and homicides (so-called "roid rage"). The long-term effects of other performance-enhancing substances, such as Human Growth Hormone (hGH) and creatine, have not been well established and are not recommended for children and adolescents. Testing for these "performance-enhancing substances" are commonly performed on urine and hair samples through the use of reference laboratories.

Stimulants and diet aids can also cause or contribute to sports-related deaths. Substances containing ephedrine and ephedra alkaloids have been linked to sudden death (Haller and Benowitz 2000). "Energy drinks" often contain agents that can contribute to cardiac deaths and hyperthermia as can certain antihistamines (Clauson et al. 2003; López-Barbeito et al. 2005). It goes without saying that illicit drugs, including cocaine and methamphetamine, can contribute to or directly cause the death of athletes.

Participants in sports requiring lean body mass are at risk for death related to dehydration or metabolic abnormalities associated with anorexia nervosa and bulimia (Warren 2011; Misra and Klibanski 2011). It should be stressed that these disorders afflict adolescents who are concerned about their body image as well as those participating in competitive sports. Investigative history consistent with these disorders are drastic weight loss, a history of using either prescription or over-the-counter medications to facilitate urination and defecation, use of appetite suppressants, a history of vomiting after eating, and exercising incessantly. Physical manifestations appreciable on external examination may include cachexia; calluses, scars, or abrasions on the hands if fingers are used to induce vomiting; dental caries or loss of tooth enamel from chronic exposure to gastric acid; and periorbital, conjunctival, or scleral petechiae from induced vomiting (Department of Health and Human Services Office on Women's Health 2009). Vitreous electrolyte analysis may shed light on deaths due to dehydration or self-imposed starvation or malnutrition, but it may not establish the cause of death in all such cases. Once again, a careful investigation may be required to establish this risk factor for sudden death (see ► Chap. 24, "Starvation, Malnutrition, Dehydration, and Fatal Neglect").

Natural Deaths in Sports

Cardiovascular

Cardiac disease is the leading cause of sudden death in athletes engaged in sports and strenuous activities. Until proven otherwise, a cardiovascular source of death should be sought when an athlete unexpectedly collapses and dies. This category of death can be broadly divided into infection, congenital conditions (molecular and structural), coronary artery anomalies, neoplasms, and progressive organic diseases.

Myocarditis is an inflammatory process involving the heart characterized microscopically by an inflammatory infiltrate in the myocardial interstitium accompanied by myocyte necrosis (Fig. 26.2). In the majority of cases, the inflammation is due to

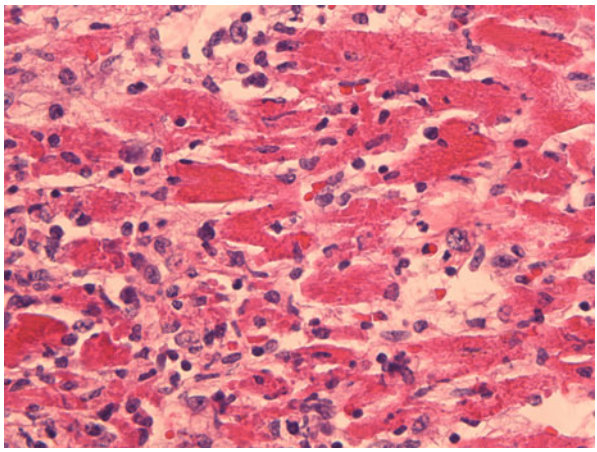

Fig. 26.2 Viral myocarditis is characterized by an inflammatory infiltrate, usually lymphocyte rich, accompanied by myocyte necrosis (Hematoxylin and Eosin, H&E × 40)

a viral infection (e.g., Coxsackie virus and Adenovirus), and a lymphocytic infiltrate will predominate. Clues to the diagnosis include a recent viral illness and a "floppy" heart upon gross examination. Although a viral etiology can be demonstrated in some cases through laboratory studies, in other cases the infectious agent will not be isolated. Other myocarditides are caused by bacteria, fungi, parasites, or autoimmune processes. Depending on the etiology of the process, the inflammatory infiltrate may consist of giant cells, eosinophils, histiocytes, or neutrophils. Histologic sections may require special stains (e.g., Brown and Hopps, silver, or Periodic Acid-Schiff stains) in order to better delineate microorganisms. Sarcoidosis, a granulomatous inflammation of the heart, may be the result of a postinfectious inflammatory response or of an autoimmune process, the etiology of which remains unclear. Special stains to rule out tuberculosis and fungi should be employed to support this diagnosis.

Congenital conditions may manifest themselves at a structural, cellular, or molecular level. There are a litany of metabolic diseases that may infect the heart, including Pompe disease and other storage disorders. These are beyond the scope of this chapter and will not be discussed in further detail, other than to say that they may be a cause of sudden death in childhood. Many of these diseases are symptomatic early in life (see ▶ Chap. 31, "Cardiac Channelopathies and the Molecular Autopsy," ▶ Chap. 32, "Other Pediatric Cardiac Conditions," and ▶ Chap. 34, "Pediatric Metabolic Diseases").

At the molecular level, two major considerations are Long QT Syndrome and Brugada Syndrome (Goldenberg et al. 2008; Escárcega et al. 2009). These cardiac ion channelopathies may result in sudden, unexpected death in apparently healthy individuals. There is an association with death during swimming with Long QT syndrome (Choi et al. 2004), which may be diagnosed by evaluation at reference laboratories if it is suspected and appropriate samples are obtained. These diagnoses can be made by retrospective analysis of electrocardiograms in some cases; however, in many young people, this antemortem study has never been performed.

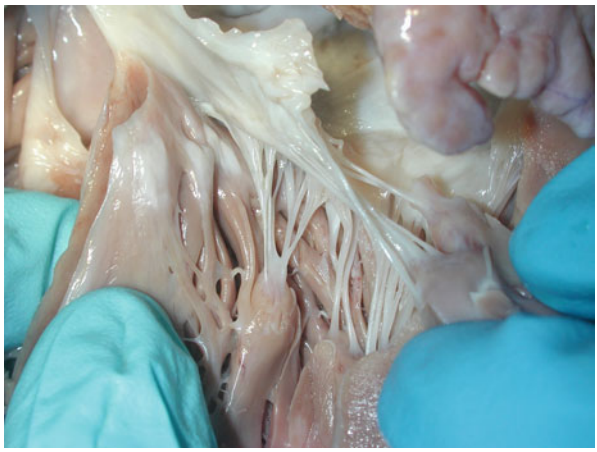

Fig. 26.3 In hypertrophic cardiomyopathy, a fibroelastotic "jet lesion" is often found on the endocardium subjacent to the aortic valve

These conditions cannot be diagnosed at the gross or microscopic level as they are rhythm disturbances.

Hypertrophic cardiomyopathy (formerly asymmetric septal hypertrophy, idiopathic hypertrophic subaortic stenosis) can be diagnosed grossly and microscopically. In classic cases, the interventricular septum will be markedly thickened when compared to the left ventricular free wall. In other cases, the left ventricle may show concentric hypertrophy; the right ventricle may also be thickened. Often, fibroelastosis of the endocardium below the aortic valve is seen as a "jet lesion" (Fig. 26.3). Microscopically, myocyte disarray with intervening fibrosis is the characteristic histologic finding (Fig. 26.4). This finding may be focal, and multiple microscopic sections of the heart with trichrome staining may assist in the diagnosis. This disease is caused by a protein abnormality in the heart resulting from a mutation in the genes encoding for the sarcomeric proteins (e.g., myosin heavy and light chains, myosin-binding protein C, troponins I and T, and tropomyosin) (Harris et al. 2011). As hypertrophic cardiomyopathy is an autosomal-dominant inheritable condition in approximately half of the victims, this diagnosis has implications for surviving family members (Cirino and Ho 2008).

Marfan Syndrome affects multiple sites in the body. The cardiovascular manifestation of this condition is cystic medial necrosis of the aorta. This may result in aortic dissection with rupture into the pleural spaces or pericardial sac with cardiac tamponade or dissection of the coronary arteries. Marfan syndrome should be suspected in the sudden collapse and death of tall athletes with long hands and feet (arachnodactyly), a desirable physique for basketball and volleyball players. This disease is caused by a mutation in the fibrillin-1 gene, the product of which is an extracellular matrix glycoprotein that maintains the structural integrity of connective tissues (Robinson et al. 2006).

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC) may be inherited in an autosomal-dominant pattern (Azaouagh et al. 2011). It is characterized by progressive replacement of the myocardium of the right ventricle by adipose tissue

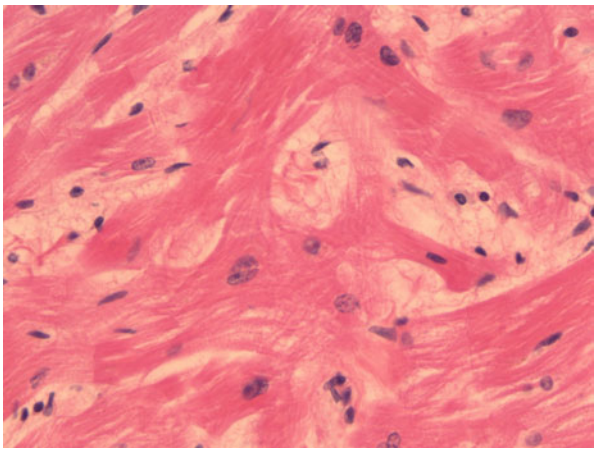

Fig. 26.4 Myocyte disarray with intervening fibrosis is characteristic of hypertrophic cardiomyopathy. It may be focal, and multiple heart sections should be examined if there is no apparent cause of death in an athlete following autopsy (Hematoxylin and Eosin, H&E × 100)

and fibrosis. Occasionally, there are a few scattered inflammatory cells. In advanced cases, the left ventricle may also be involved. This condition may be undiagnosed as the findings are subtle in the early stages, and the right ventricle is often undersampled for histologic analysis. This diagnosis can, at times, be difficult to make, and cardiovascular pathology consultation may prove beneficial.

Structural defects resulting in sudden death may or may not be grossly apparent. Valvular anomalies, septal defects, and transposition of the great vessels can be readily identified at autopsy. Deaths due to structural anomalies of the coronary arteries may be more subtle. Consultation with a cardiovascular pathologist may be helpful in identifying coronary arterial atresia, intramyocardial tunneling, or acute origin from the Sinus of Valsalva. These experts may also assist in identifying problems with the cardiac conduction system. These may be either aberrant neural pathways or stenoses of the arteries supplying the atrioventricular (AV) or sinoatrial (SA) nodes. A microscopic tumor of the AV node can also result in sudden death (Fig. 26.5).

Structural anomalies of other blood vessels may also lead to sudden death. Arteriovenous malformations, particularly within the central nervous system, may rupture with catastrophic results. “Berry” aneurysms of the cerebral vasculature may enlarge over time, and intense physical exertion with associated elevation of blood pressure (e.g., weightlifting) may precipitate bleeding. Aneurysms and pseudoaneurysms of large arteries cause massive internal hemorrhage.

Primary cardiac neoplasms are rare but they can lead to death. Tumors that affect the heart include atrial myxoma, fibroma, and rhabdomyoma, the latter associated with tuberous sclerosis. The heart may also be affected by lymphomas, angiosarcomas, and metastatic disease. The most common cancers metastatic to the heart are lung, breast, melanoma, and leukemia/lymphoma.

Adolescents are not immune to cardiovascular diseases that kill older individuals. Especially in the setting of familial hypercholesterolemia, atherosclerotic coronary artery disease may develop in the mid-teen years. Hypertension may also result in myocardial hypertrophy and lethal dysrhythmia; however, this must

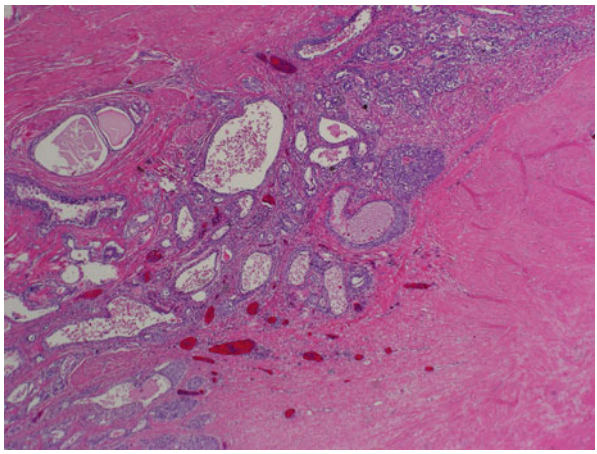

Fig. 26.5 Cystic tumor of the atrioventricular node (Hematoxylin and Eosin, H&E × 10)

be distinguished from hypertrophic cardiomyopathy, discussed above. Whereas hypertrophic cardiomyopathy commonly affects the septum on gross inspection and is associated with myocyte disarray, hypertension generally results in concentric thickening of the left ventricular chamber and enlarged, hypertrophic myocytes with hyperchromatic "box car" nuclei at the microscopic level. Lastly, morbid obesity has been associated with sudden death (see ▶ Chap. 33, "Childhood Obesity").

Special Techniques: Cerebrovascular Aneurysms

When faced with an unanticipated subarachnoid hemorrhage, the pathologist should remove the brain themselves with frequent photographic documentation of the process in order to capture occult lesions prior to onset of any removal artifact(s). Once removed, the brain, cerebellum, pons, and medulla should be copiously rinsed with water to remove adherent blood and clot. In lieu of water, hydrogen peroxide may be used to facilitate the lysis of adherent blood from the delicate vasculature so that it can be better examined. Care must be taken to avoid destruction of subtle vascular malformations and aneurysmal sacs.

Chronic Diseases

Sickle-cell disease may be diagnosed in childhood, and it can afflict participants in athletics and other strenuous activities. If the diagnosis of sickle-cell disease is known, recognition and treatment of an impending crisis can avert death. Many people with sickle-cell trait, however, are unaware of their condition. When subjected to intense physical exertion, high temperatures, and a component of dehydration, a crisis may ensue and death may rapidly follow

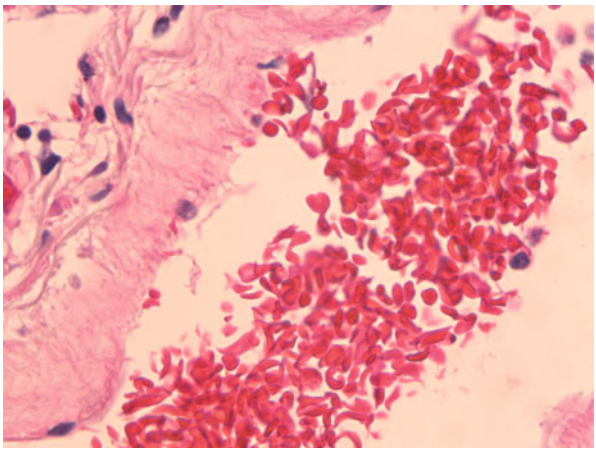

Fig. 26.6 Sickled erythrocytes in the pulmonary vessels in a case of sudden collapse and death in a person with sickle-cell trait (Hematoxylin and Eosin, H&E × 100)

(Scheinin and Wetli 2009; Manci et al. 2003). A recent viral illness could also be an exacerbating factor. This condition should be considered when an athlete of African or Mediterranean descent complains of joint pain, chest pain, fatigue, and weakness prior to collapse. It is often misdiagnosed as a heat-related illness. The diagnosis is made at the microscopic level wherein virtually all organs will be congested by sickled erythrocytes (Figs. 26.6 and 26.7). At the gross level, persons with sickle-cell disease may have fibrotic, atrophic spleens, whereas those with sickle-cell trait may have enlarged spleens, congested with sickled erythrocytes. The diagnosis can be confirmed with hemoglobin electrophoresis (blood best procured in a tube with anticoagulant/EDTA) and correlated with information obtained regarding prevalence and distribution of this disease within the family.

Asthma is a common disease among children and teens, and acute attacks may be precipitated by physical activity. In order to certify death due to asthma, the circumstances of death need to reflect a respiratory crisis. In many cases, an inhaler and/or a nebulizer will be found near the victim or with their personal belongings. Grossly, the lungs will be hyperinflated, often touching over the heart in the midline, with prominent mucus plugging of the airways. The microscopic findings of chronic asthma in the bronchioles (thickening of the basement membranes, smooth muscle hypertrophy, and mucus gland hyperplasia) will be accompanied by an eosinophil-rich inflammatory infiltrate that extends into the luminal mucus plugs (Figs. 26.8 and 26.9). A history of asthma should not be a default cause of death in these cases without the circumstances and scene findings supporting a respiratory catastrophe.

Epilepsy, like asthma, may be a cause of death, however, the circumstances should support the diagnosis. In the absence of a witnessed seizure, other causes of death must be excluded prior to attributing death to epilepsy. Further, intracranial trauma must be excluded as a cause of the seizure. Some epileptics will die suddenly and unexpectedly in the absence of a seizure (sudden unexpected death in an epileptic person or SUDEP), but this does not typically occur while the victim

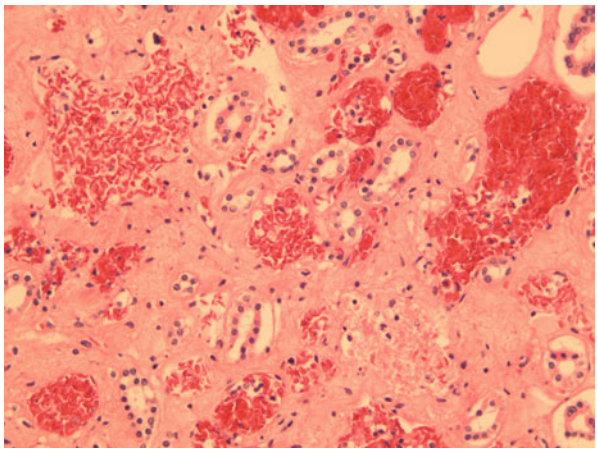

Fig. 26.7 Sickled erythrocytes are often readily identified in the renal vasculature in deaths due to sickle-cell disease or exacerbation of sickle-cell trait (Hematoxylin and Eosin, H&E × 20)

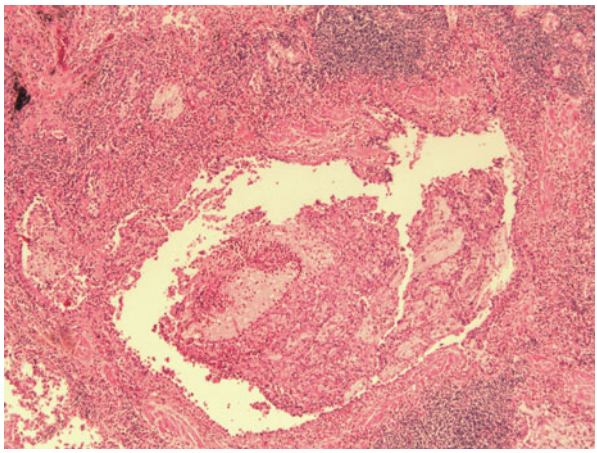

Fig. 26.8 Mucus plugs with admixed inflammatory cells in a person who died of status asthmaticus (Hematoxylin and Eosin, H&E × 10)

is engaged in sports. The autopsy may or, more commonly, may not identify the anatomic correlate of the seizure focus in the brain.

Spontaneous pneumothorax may occur during sports presumably due to increased shear forces at the apex of the lung (Abolnik et al. 1993). It may cause death if it progresses to a tension pneumothorax, a condition that results in both hypoxia and mechanical alterations of the cardiovascular system. This is a diagnosis that may be missed if it is not suspected.

Diabetes mellitus can kill children and adolescents engaged in sports. Activities which entail dietary restrictions may predispose those with the disease to ketoacidosis. Young people more often have type I diabetes and may be insulin dependent. As teens have a tendency toward denial and risk-taking behavior, they may not be fully compliant with their treatment regimens and therefore be prone to significant blood glucose fluxes. The gross findings at autopsy will be minimal in

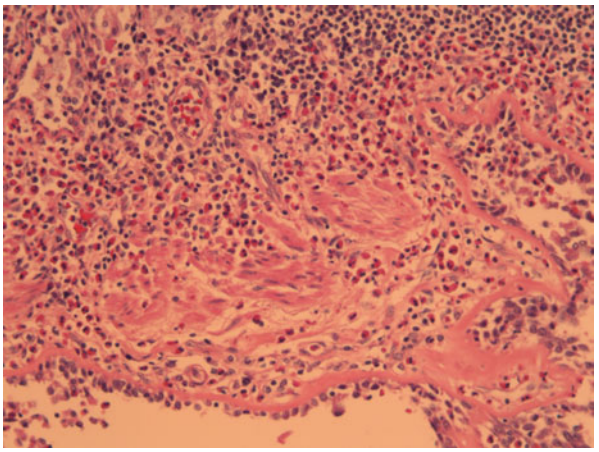

Fig. 26.9 The findings of chronic asthma (basement membrane thickening, smooth muscle hypertrophy, mucus gland hyperplasia) with an intense eosinophilic inflammatory response in a person who died during an asthma attack during exercise (Hematoxylin and Eosin, H&E × 20)

these cases. Microscopically, the islets of Langerhans in the pancreas may be infiltrated by lymphocytes ("insulitis"), or they may be diminished in number. Urine screens for glucose and ketones may be useful, but postmortem blood analysis is unreliable. The best sample for diagnosing diabetes mellitus and ketoacidosis postmortem is vitreous humor. The presence of ketones and significantly elevated glucose (> 500 mg/dL) in the vitreous humor is diagnostic of this condition (Chansky et al. 2009). Vitreous glucose levels drop significantly after death, however, so a lower ocular glucose level does not exclude hyperglycemia. Further, hypoglycemia cannot be diagnosed postmortem due to the aforementioned postmortem change. In the evaluation of a nontraumatic death occurring during sports, analysis of vitreous glucose, ketones, and electrolyte levels is recommended in all cases.

Special Techniques: Pneumothoraces

Prior to the examination, chest radiographs including lateral and seated views may illustrate free pleural air and displacement of the heart. At autopsy, care should be taken to reflect the skin and soft tissues of the chest without breaching the intercostal tissues or entering the chest cavities. A pocket which should be filled with water can be created using the reflecting chest tissues. The intercostal tissues can then be pierced below the water level to examine whether air bubbles emerge.

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