SIDELINE EMERGENCIES: AN EVIDENCE-BASED APPROACH

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Introduction

Participation in athletics continues to increase at all age levels and as a result, the prevalence of on-field injuries and medical emergencies is increasing. Fortunately, catastrophic injury and death associated with sport participation is rare, however, the risk exists and events do occur. The field of sports medicine continues to advance over time and evidence-based interventions as well as on-field medical awareness and diagnostic skills have developed to help minimize these events. Increased involvement of athletic trainers, emergency medical technicians, and medical staff for coverage of events coupled with pre-prepared and practiced emergency action plans are the best defense against catastrophic outcomes. Regardless of pre-participation physical exams and protective outerwear, events will continue to occur. This article reviews the most catastrophic injuries and medical illnesses encountered in athletes and provides an evidence-based recommendation for immediate sideline evaluation and management.

Head Injuries

Clinical Vignette

A collegiate football player sustains a helmet-to-helmet hit and complains of headache with associated nausea and vomiting. He does not lose consciousness but is amnestic to the events. How should this athlete be evaluated? Does he need a CT scan? Can he return to play in this game? How can we safely determine when he may return to play?

Epidemiology

Head injuries are the leading cause of traumatic death in sports. An estimated 300,000 sports related brain injuries occur per year in the United States with 250,000 occurring in high school football. The majority of these are concussions, however, an average of 8-10 fatalities occur yearly in college and high school football related to more severe head injuries including epidural hematomas, subdural hematomas, and subarachnoid hemorrhages (1, 2). In a Level II prospective

observational cohort, Powell et al. (3)found football to have the highest injury rate among all sports with 63% of all concussions in high school athletes occurring in football. The football incidence was 0.59 events per 1000 athletic exposures, followed by wrestling (0.25), girls soccer (0.23) and boys soccer (0.18) (3). Guskiewicz noted 5% of high school and collegiate players sustain a concussion (4) with a collegiate incidence of 0.81 per 1000 athlete exposures (5). Pellman found the rate of concussion to be 0.41/game in the NFL (6). Most experts agree these numbers are underreported (1), and one recent survey revealed that 53% of high school athletes did not report their concussion symptoms (7).

Pathophysiology

The 3rd International Conference on Concussion in Sport recently defined a concussion as "a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces" (8). Giza and Hovda may have best explained the pathophysiology as a "metabolic mismatch" (9). Sufficient direct or indirect transmitted forces due to rotational and/or acceleration-deceleration forces to the brain cause changes at the cellular environment. There is an increase in the Na/K ATP pump requiring a higher energy demand and a decrease in cerebral blood flow in addition to intracellular calcium accumulation and impaired neurotransmission. These changes have been studied in animal models showing glucose metabolism and cerebral blood flow uncoupling lasting 30 minutes to 4 hours (10), intracellular calcium changes lasting up to 4 days (11), and impaired neurotransmission for >1 week (12). How well this data can be extrapolated to humans is unknown (13).

Diagnosis/Assessment

The clinical diagnosis of an acute concussion initially involves assessing for an array of signs and symptoms. Symptoms may include headaches, nausea, dizziness, photophobia, fatigue, and difficulty with concentration and memory. Outward signs include loss of consciousness, confusion, amnesia, loss of balance and coordination, and personality changes. Several Level II cohort studies

found headache was the most common symptom, occurring in 85% of cases (3, 5, 6). Loss of consciousness occurred in less than 10% of reported cases (3, 5, 6).

Management/Sideline Treatment

Players who exhibit signs or have symptoms of possible head injury must immediately be removed from play and medically evaluated. Unconscious players should be assumed to have a cervical spine injury and quick attention should be made to address their airway, breathing, and circulatory status while maintaining spinal immobilization. Unconscious and unresponsive players should immediately have their cervical spine stabilized and the facemask should be removed in helmeted players to gain access to the airway (Figures 4, 5, and 6).

Players should be referred to the emergency department (ED) for additional monitoring and a non-contrast head CT scan if they have a focal neurological deficit or if their status worsens. A Level II prospective cohort study by Saboori et al. evaluated 682 patients presenting to the ED with minor head injury with CT scans. All patients included in the study had a normal score of 15 on the Glascow Coma Scale (GCS) and 6.7% of patients had abnormal CT scans. On review of their signs and symptoms, all patients with abnormal CT scans had at least one of the following risk factors: obvious signs of skull fracture (Odds Ratio [OR] 8.9), vomiting (OR 8.3), age >60 (OR 4.9), confusion (OR 3.9), history of loss of consciousness (OR 2.4), and headache (OR 2.1). They concluded that patients with one risk factor should undergo CT scanning if available. If a CT scan is not available, they recommended close observation for several hours and if symptoms persisted or worsened, the patient should be transported for CT. In patients with more than one risk factor, the authors recommended transferring the patient to a facility if a CT was not readily available (14). Smits et al. similarly agreed that patients should undergo CT scan if they have vomiting (OR 2.4), posttraumatic amnesia >4 hrs (OR 7.5), GCS less than 15 (OR 3.9), signs of skull fracture (OR 10), and posttraumatic seizure (OR 2.3) (15).

In conscious patients, a quick survey of the ABCs should be followed by evaluation of spinal tenderness and a neurologic assessment of the upper and lower extremities. Unstable patients should be transported immediately to a medical facility for additional imaging and management. In more stable patients, a more thorough evaluation can be performed on the sideline. A full assessment of signs and symptoms should be followed by a thorough neurological exam. Symptomatic players should be withheld from play. Players should be evaluated and serially monitored for deterioration. Players should not be left alone and parents, teammates, and roommates should be informed of signs and symptoms to observe for deterioration.

Return to Play

The decision to allow a player to return to play following a concussion is a difficult decision to make for the team physician. All symptomatic players should not be allowed to return to play. Although many return-to-play guidelines have been published, none have been validated. Several Level II natural history cohort studies have followed athletes and their duration of symptoms following concussion. These studies consistently show that the majority of athletes have resolution of symptoms within 3-8 days, with 90% fully recovered at one week (3, 4, 6, 16). McCrea et al. followed concussed athletes using the Graded Symptom Checklist, Standardized Assessment of Concussion, the Balance Error Scoring System, and a neuropsychological test and found 91% returned to baseline by day 7, but not all players demonstrated the same pattern of recovery in symptoms, balance, and cognition (16). Tools such as the Standardized Assessment of Concussion and computerized neuropsychological tests have been shown to be useful adjuncts in the decision to return safely to play. The Standardized Assessment of Concussion has been shown in a Level II prospective cohort to be 95% sensitive and 76% specific when applied to concussed players, and may be a useful adjunct in players with minimal or resolved symptoms to help determine return to baseline. Similarly, several studies evaluating computerized neuropsychological tests have shown cognitive recovery largely overlaps with symptom recovery, though cognitive recovery may be

delayed until after symptoms have resolved (8, 17). Computerized testing may be able to detect abnormal cognitive findings in asymptomatic concussed individuals. Preseason normative values are helpful for comparison to scores following a concussion to determine a player's return to baseline. Computerized tests may be helpful in identifying asymptomatic athletes that may still be affected by a concussion, however, Broglio found retest reliability in non-concussed volunteers to have 20-38% false positive values indicating up to 38% may have been withheld from return to sport despite being asymptomatic (18). In 2005, Randolph et al. performed a Level III literature review on prospective controlled studies of neuropsychological testing and concluded "no neuropsychological tests have met necessary criteria to support a clinical application at this time" (19). Although these tests alone should not be the sole basis of management decisions, they may prove to be a useful adjunct in difficult return-to-play decisions (8).

The 3rd International Conference on Concussion in Sport recommends that all concussed players be removed from the contest and should not return to play that day (8). A graded return-to-play guideline should be applied to each athlete, allowing progression based on the individual. Each individual should be rested physically and cognitively until symptoms resolve. Upon resolution of symptoms, athletes can progress to light aerobic exercise, followed by sport-specific exercise, then non-contact drills, followed by full contact practice and eventual return to unrestricted play. Should the athlete develop symptoms during this progression, they should again be placed at rest until asymptomatic and then restart the protocol again in a stepwise fashion. Generally each step may take 24 hours, though some athletes may be able to progress more quickly under the watchful care of qualified medical staff (8, 20).

Return to Play Same Game

Studies have shown that up to 30% of high school and collegiate athletes returned to play same game, and up to 50% of NFL players have returned to play same game following concussions (4, 6, 21). Studies following athletes who returned to play same game following a concussion

showed up to 33% of players experienced delayed onset of additional symptoms that kept them out greater than 7 days. As noted above, asymptomatic post concussed players may still be affected as noted on neuropsychological tests and the SAC and may still be at risk. The 3rd International Conference on Concussion in Sport addressed the issue of some adult, elite athletes being able to return to play same day after thorough evaluation by team physicians experienced in concussion management, but noted they should still follow the same basic management principles and exhibit full clinical and cognitive recovery. Putukian reviewed the literature relating the difference in concussion management strategies between elite and non-elite athletes and concluded the elite athletes' access to physicians, neuropsychologists, and brain imaging may allow them more timely return to sport, however, she also reported that whether same day return to play is safe was a "contentious question that has not been fully answered in the published literature" (20). Experts still agree that younger athletes should be treated more conservatively and withheld from same game return to play (8).

Prevention

Several concussion prevention strategies have been proposed including properly fitted helmets, new helmet technology, use of mouth guards, neck strengthening and appropriate tackling techniques. To date, only limited data exists to support each strategy. Biomechanical studies have shown a reduction in impact forces to the brain with the use of head gear and football helmets (8). Newer helmet technology was shown to decrease concussion incidence (5.3% vs 7.6%, p=0.027) in a small Level III cohort study comparing the Revolution helmet and traditional football helmets in high school athletes (22). Helmet technology will likely continue to progress aiding in a reduction of force with head impacts. Withnall, in a Level IV case series, studied the use of head gear in soccer players and found no benefit in reduction of concussion rate for ball impacts, however, in head-to-head impact the headgear provided an overall 33% reduction in concussions (23). No

studies to date have shown the use of mouth guards to decrease risk of concussion; however, their use has been shown to significantly lower the rate of dental trauma (8, 24, 25).

Cervical Spine Injuries

Clinical Vignette

A high school football player remains in the prone position on the playing field after involvement in a collision with another player. From a sideline perspective, he is motionless without witnessed extremity movement, and his teammates are waving in a frantic fashion towards the medical staff. Upon initial assessment, he is alert and complaining of neck pain. What are the first steps in the evaluation of this athlete? Should he be rolled to a supine position? What are the indications for equipment removal? Does he need transport to an emergency facility?

Epidemiology

Spinal cord injury is a potentially catastrophic and life-changing event with an estimated 12,000 new cases each year. According to the National Spinal Cord Injury Statistical Center, sporting activities account for approximately 7.6% of these injuries (26). Due to the large numbers of participants in American football (estimated at 1.8 million in 2008), this sport remains the main focus of prevention strategies (2). Other high-risk sports such as ice hockey, downhill skiing, gymnastics, diving, and rugby have also been associated with spinal cord injury (27, 28). Despite attempts at prevention and education, spine injuries remain an important issue for the sideline physician.

Pathophysiology

The cervical spine consists of seven vertebral bodies with a natural lordotic curvature. Relative neck flexion converts the lordotic curve into a column decreasing the potential for the surrounding structures (ligaments, paraspinal muscles, and intervertebral discs) to dissipate force. In this position, axial loading can lead to fracture of the bony elements with potential for intrusion

upon the spinal cord (29). Leading with the helmet in American football has been implicated as a

risk factor for cervical spine injury. Spear tackling, defined as using the "helmet (including the face

mask) in an attempt to punish an opponent" was banned in 1976 leading to a decrease of 80% in

the rate of catastrophic cervical spine injuries over the course of the next decade (28, 29).

Prevention strategies aim to decrease axial loading situations and maintain the natural lordotic

curvature.

Diagnosis / Assessment

In reviewing the existing literature regarding effective options to maximize clinical outcome

following cervical injury, there is a paucity of high-level evidence to guide treatment. The

overwhelming majority of publications consist of EBM Level V manuscripts. In assessing the athlete

in an on-field setting, the initial possible clinical scenarios are broad ranging from generally self-

limiting conditions such as muscular strains, ligamentous sprains, and stingers/burners to more

alarming diagnoses such as transient quadriplegia, herniated discs, fractures, and dislocations with

or without associated spinal cord injury. With this in mind, the treating clinician must gain control

over the immediate situation and follow an ordered process to adequately diagnose, and if

necessary, stabilize the spine to prevent further injury until definite treatment can occur.

Management / Sideline Treatment

Management strategies actually begin prior to each season or event. The treating clinician

must first institute an emergency response plan. This includes reviewing the presence, location,

and maintenance of essential medical equipment and personnel (AED, spine board, airway and

ventilation equipment, paramedics, means of transportation, hospital services), defining roles for

each available member of the clinical team, and establishing a communication system in advance of

any clinical situations requiring a rapid and coordinated response (30).

Initial on-field evaluation consists of evaluating the ABCs (airway, breathing, and

circulation), and cervical spine injury should be presumed if an altered level of consciousness is

present (31). Immobilization is necessary to prevent further injury, and an athlete in the prone position represents a challenge. Log-rolling, a multi-person maneuver, is recommended to transfer the athlete to a supine position and involves manual in-line axial stabilization of the cervical spine (31) (Figure 1.) Attempt should be made to minimize manipulation of the injured athlete, and log-rolling onto a spine board will facilitate this goal if future transport is deemed necessary (Figure 2). In the setting of a supine patient needing placement upon a spine board, the log-roll technique is again a viable option to place the spine board under the athlete. An alternative consists of the five-person lift in which the player is lifted from the playing surface while maintaining in-line cervical axial stabilization, and the spine board is moved into position (Figure 3). Importance is again placed upon the multi-person requirements of these techniques, and pre-event practice is required to refine these techniques.

Once in the supine position on a spine board, the head should be immobilized to curtail future movement of the cervical spine. A combination of various accessory items can be utilized to accomplish this task including tape, binders, and bags. Facemask and mouthpiece removal are recommended to allow for immediate or future access to the airway, and removing these items generally allows adequate access to the airway without necessitating helmet removal. Numerous tools exist to accomplish facemask removal, and multiple options should be directly available in the urgent setting (32) (Figure 4). The clinician must become familiar with various helmet designs to determine the fashion in which the facemask is secured to the helmet in advance of an emergent situation (Figure 5). A power screwdriver and cutting tool for facemask clips should be available in case one technique fails (i.e. rusted or stripped screws in an older football helmet) (Figure 6).   In general, a helmet should be left in place, especially in the setting of a sport involving shoulder pads (football, lacrosse, hockey). If the helmet must be removed (poor fit hindering cervical spine immobilization, blocking access to airway, etc.) the shoulder pads should also be removed to avoid

unnecessary extension of the cervical spine (Figures 7, 8, and 9). Care must be taken to maintain in-line axial stabilization of the cervical spine throughout the equipment removal process.

Once initial assessment is complete (ABCs and cervical immobilization), the athlete is ready for further evaluation including rapid transport to an advanced treatment facility, if necessary. Indications for transport include midline tenderness in the cervical spine or the presence of neurological complaints/exam findings. Imaging studies to include radiographs and CT scan can be performed while the athlete remains immobilized to facilitate further evaluation and definitive diagnosis.

Prevention

At present, prevention strategies rely upon screening, technique modification, and protective equipment. During pre-participation physicals, focus attention upon history of previous neck injury or knowledge of congenital abnormalities. The presence of neurological symptoms or signs during examination warrants further evaluation with imaging studies (radiographs, magnetic resonance imaging, CT, and/or myelography) (27). Technique modification relies upon proper education of coaches and athletes as well as consistent rule enforcement by governing bodies. Penalization for spear tackling, use of excessive force, and blind-sided impacts combined with separation of athletes by age, size, and skill level can prevent injuries. Protective equipment includes helmet use in sports such as football, hockey, downhill skiing, and baseball in which high velocity impact may occur (28). Prevention is a multifactorial process that requires continual refinement and adaptation to minimize injury.

Sudden Cardiac Arrest

Clinical Vignette

During preseason conditioning drills, an 18-year-old freshman football player collapses. The athletic trainer responds immediately and finds the player unconscious and pulseless.   CPR is

initiated, 911 notified, and the athlete is transported by ambulance to the hospital. Despite efforts to revive the player, he is pronounced dead in the emergency department. What is the most likely cause of this athlete's death? What is the proper response to an athlete who collapses during play? What steps can be taken to prepare for and respond to such an event?

Epidemiology

Sudden cardiac death is the leading cause of death in high school and college-aged athletes (33). Although its true incidence is unknown in the United States, sudden cardiac death is estimated to occur in approximately 1:200,000 high school athletes and 1:90,000 college athletes (34, 35). Males are roughly nine times more likely than females to be affected (33, 35). Sudden cardiac death most often occurs in high school football, followed by high school basketball, then college football (33, 35).

Pathophysiology

Structural heart disease accounts for the majority of sudden cardiac death in athletes. In the United States, the foremost cause is hypertrophic cardiomyopathy, a relatively common genetic disease with an incidence of 1 in 500 in the general population, which leads to approximately 26% of these fatalities (36, 37). Coronary artery anomalies, especially wrong aortic sinus origins, lead to another 14% of cases (37). Other cardiac diseases associated with sudden cardiac death in athletes include ruptured aortic aneurysm associated with Marfan's syndrome, arrhythmogenic right ventricular dysplasia, valvular disease, dilated cardiomyopathies, and various conduction system and ion-channel disorders. In athletes over 35 years of age, atherosclerotic coronary artery disease accounts for over 80% of sudden cardiac death (38, 39).

Two other causes of sudden cardiac death in athletes that deserve mention are myocarditis and commotio cordis. Myocarditis is an inflammatory condition of the myocardium most commonly brought on by an acute viral infection, although numerous noninfectious causes such as exposure to cocaine or ethanol may also lead to the condition (40). While in most series it is felt to

cause less than 10% of sudden cardiac death in athletes, it was the leading cause in healthy Air Force recruits over a 20-year period (35, 37, 41). Commotio cordis occurs as a result of non-penetrating chest wall trauma, insufficient in force to cause structural damage to the sternum, ribs, or heart, that leads to ventricular fibrillation in an otherwise normal heart. This condition accounts for approximately 20% of sudden cardiac death in young athletes (37).

Whether from congenital or acquired heart disease, or from commotio cordis, the final common pathway leading to sudden cardiac death in athletes is most often ventricular fibrillation leading to asystole. The heart's inability to pump causes end-organ collapse in a period of minutes.

Initial Assessment and Treatment

A collapsed athlete requires rapid evaluation and immediate, orderly management. Although the differential diagnosis for non-trauma induced collapse is broad, and most athletes who collapse are not in cardiac arrest, the sideline physician should initially assume a cardiac etiology. Lightheadedness, dizziness, and even transient loss of consciousness immediately after exercise are usually benign but require both close monitoring until the athlete is fully recovered and further workup to ensure the absence of underlying heart disease. On the other hand, syncope that occurs during exercise is ominous and necessitates urgent investigation.

The American Heart Association has described 4 "links" in a "chain of survival" that are critical in responding to sudden cardiac collapse (42). These links are:

- Early recognition of the emergency and activation of emergency medical services
- Early cardiopulmonary resuscitation (CPR)
- Early defibrillation
- Rapid transition to advanced life support

When an athlete collapses and is unresponsive, emergency medical services should be notified immediately by calling 911. Early recognition of cardiac arrest requires a high index of suspicion. Collapsed athletes may display seizure-like myoclonic jerks and agonal breathing or

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gasping, both of which may be misinterpreted as signs of life. Pulses may be difficult to assess and should not be sought for more than 10 seconds before beginning chest compressions if no pulse is detectable (42).

The athlete who suffers cardiac collapse needs immediate CPR. CPR provides critical blood flow to the heart and brain, prolongs the time ventricular fibrillation is present, and increases the likelihood a shock will defibrillate the heart (42). Immediate CPR may double or triple the collapsed athlete's chance of surviving sudden cardiac arrest with ventricular fibrillation (43, 44). The American Heart Association released updated guidelines on CPR and emergency cardiovascular care in 2005 (45). These guidelines apply to any athlete older than 8 years of age. Figure 10 provides an algorithm for managing a collapsed athlete with suspected cardiac arrest.

The most critical factor affecting survival of cardiac arrest is the time interval between arrest and defibrillation (46). The automated external defibrillator (AED) is a computerized device that analyzes the victim's rhythm and prompts the rescuer to provide a shock if the victim is in ventricular fibrillation or rapid ventricular tachycardia. Provision of CPR and defibrillation within 3 to 5 minutes has achieved survival rates of 49% to 75% in community AED programs in this country (47-52).

Prevention and Preparedness

Vigorous exercise appears to be a trigger for lethal arrhythmias in athletes with occult heart disease (37). In up to 80% of cases of sudden cardiac death, the athlete is asymptomatic until the cardiac arrest (53). Ideally, athletes could be screened for potentially fatal cardiac conditions. In reality, the history and physical examination provide few clues to the discovery of these conditions. The American Heart Association has published recommendations for cardiovascular screening of athletes (54). These recommendations consist of 12 items, including 8 items in the personal and family history and 4 items in the physical examination. At the discretion of the examiner, a positive response or finding to any item may prompt a referral for more extensive cardiac evaluation. Due

to the poor sensitivity, high false-positive rate, and costs of implementation, universal screening of athletes with electrocardiography or echocardiography is not recommended.

In light of the limitations of screening for potentially lethal cardiac disease in athletes and the success of rapid response to cardiac arrest, an emergency action plan is critical to have in place when providing care to athletes. An interdisciplinary task force of representatives from 15 national organizations with special interest in sudden cardiac arrest in athletes has published recommendations on emergency preparedness (55). Essential elements of the plan include establishing an effective communication system, training anticipated responders in CPR and AED use, access to an AED for early defibrillation, acquisition of necessary equipment, coordination and integration of on-site responder and AED programs with local emergency medical services, and practice and review of the response plan (55).

Exertional Heat Illness

Clinical Vignette

A seventeen year old high school football player becomes fatigued and agitated during a water break of the second practice of the second day of two-a-days. It is 80 degrees Fahrenheit and 80% humidity. He refuses to be evaluated by the athletic trainer who is attending practice. He has to be restrained by the coaches in order to get a temperature measurement. Initial rectal temperature is 105 degrees Fahrenheit. What is the best way to cool a player with signs and symptoms of heat stroke? Should he be transported to the emergency department prior to cooling? When can he safely return to play?

Epidemiology

Exertional heat illness (EHI) is a common emergency that is faced on the sidelines of sporting events. The incidence varies between sports but is directly related to workout intensity, temperature, and humidity (56). Exertional heat illness includes heat stroke, heat exhaustion, and

heat related cramps.   It can occur in normal healthy athletes in mild conditions but is particularly dangerous in the sick and unfit in conditions of high heat and humidity.   If recognized and treated early heat illness can be quickly reversed. If left unrecognized and untreated, however, heat illness has a high morbidity and mortality.   In the US exertional heat illness is the third most common cause of deaths in young athletes (57).

Pathophysiology

Heat illness is a condition of hyperthermia (usually >102°F or 39°C) where the body can no longer cool itself or maintain normal body functions. In exercising athletes, heat is generated by working muscle. This heat is dissipated most effectively by the evaporation of sweat from the skin. If the temperature continues to rise despite the body's attempts at cooling, eventually organ function is affected.   Many organ systems can be affected; however, it is variable and does not follow a specific pattern.   Central nervous system involvement, typically presenting with altered mental status, is the hallmark finding for exertional heat stroke (temperature > 104°F or 40° C with symptoms).   The severity of multi-organ failure is directly related to the time the athlete stays in the hyperthermic range.

Diagnosis/Initial Patient Assessment

The symptoms and signs of heat illness are often nonspecific and a high index of suspicion must be maintained at all times to avoid missing the diagnosis.   CNS manifestations include disorientation, confusion, dizziness, irrational or unusual behavior, inappropriate comments, irritability, headache, inability to walk, loss of balance and muscle function resulting in collapse, profound fatigue, hyperventilation, vomiting, delirium, seizures, or coma.   Any athlete experiencing these symptoms should be suspected of having heat illness and have a core body temperature measurement taken.

Rectal temperature is considered the gold standard for measurement of core body temperature.   Although other temperature measurement methods are available including skin, ear,

oral, axillary, and gastrointestinal, only GI measurements compared accurately to rectal measurements in a Level II study by Casa et al. (58). There is some resistance from athletic trainers to the use of rectal monitoring because of inexperience, privacy concerns, and contamination. Practice and education can help overcome this hurdle. A rectal thermometer should be available for use on the sidelines of all practices and games. If a thermometer is not available, and a player presents with signs and symptoms concerning for heat illness, immediate cooling techniques should be started.

Management/Sideline Treatment

Once heat illness has been diagnosed, immediate cooling by any means possible is indicated. This includes removing the uniform and clothing as well as moving the player to a shaded and cool area. The most rapid whole body cooling rates (i.e., range 0.15–0.24°C per min) have been observed with cold water and ice water immersion therapy in a Level I study by McDermott et al. (59, 60). If cold water and ice immersion is not available, water mist with cooling fans, and using ice water soaked towels offers a slower but still effective means of cooling (59, 60). The goal is to cool the core temperature 0.3-0.5° F/minute down to 100-102° F. Athletes with heat stroke should immediately be transported to a medical facility for definitive care.

Return to play following exertional heat illness is unpredictable and depends on the length and severity of the hyperthermia. For heat stroke ACSM recommendations (Level 5) include no exercise for 7 days after the event, beginning exercise in a cool environment with gradual increase duration, intensity, and heat exposure over 2 weeks. Lab exercise-heat tolerance testing may be implemented if available and most players can be cleared to return to sport at 2-4 weeks if asymptomatic (56).

Prevention/Preparedness

All exertional heat illness is preventable provided that athletes, parents, and coaches understand what precautions to take before and during activity and what signs and symptoms to

look out for. Unfortunately, no evidence-based guidelines for heat illness prevention exist. Medical staff should educate their teams prior to the season and have heat illness policies and procedures in place.   Rectal thermometers and cooling procedures should be tested and be readily available before practice begins. Emergency action protocols should be practiced at least once yearly.

Acclimatization is a process where the body undergoes physiologic changes with gradual increase exposure to exercise in the heat.   Typically, acclimatization requires 4-7 sessions of 2-4 hours of exercising in the heat (61).   This results in increased heat loss and smaller rise in core temperature.   Changes seen include earlier sweating, increased sweat rate, less salt loss in sweat, and increased vasodilation.   This acclimatization should be required as a part of participation.   For college football, the NCAA allows one practice per day (less than three hours) for the first five days. The helmet is the only uniform allowed for the first two days, followed by helmet and shoulder pads for the next two days, and finally full pads on day five. After day five, two-a-day practices (less than 3 hours each) are allowed but not on consecutive days and the total practice time must be less than 5 hours per day (62).   Similar recommendations have been published for secondary school athletics (63).

It is important to identify and monitor at risk athletes closely.   Athletes at risk include those with previous heat illness, fever, dehydration, stimulant use or underlying medical conditions such as gastrointestinal or respiratory illness, poor nutrition, poor fitness, or obesity (56).   New research indicates that youth do not have less effective thermoregulatory ability and insufficient cardiovascular capacity compared to adults during exercise in the heat, when adequate hydration is maintained (64).

Asthma and Pulmonary Concerns

Clinical Vignette

A 15 year-old football player with no past medical history develops chest pain and difficulty catching his breath 10 minutes after performing wind sprints. He is alert and interactive but clearly anxious. He has difficulty completing a full sentence and poor aeration bilaterally on exam. What medicines should be provided immediately for this athlete? When can he safely return to play?

Epidemiology

Asthma is a chronic, episodic disease of the airways. An expert panel report released by the National Heart Lung and Blood Institute in 2007(65) (Level V) characterized asthma as having several integral features although it is not necessary for all items to be present. These features include recurrent episodes of respiratory symptoms, often irreversible variable airflow obstruction, the presence of airway hyperreactivity, and chronic airway inflammation. Specifically, exercise-induced asthma (EIA) is defined as episodic bronchoconstriction following exercise in asthmatic patients.

One of the most common health problems in the United States, more than 22 million people have the diagnosis of asthma. In fact, prevalence is on the rise from 3.1% in 1980 to 7.2% in 2004 (66) (Level III) with the largest subset increase being in those age 18 or younger. The prevalence of EIA in the general population has been recorded as 12-15%. A CDC analysis of the social and healthcare costs of asthma in 2004 (67) revealed 2 million ER visits/year and billions of dollars in treatment. In a Level IV case series, Becker (68) identified 263 athletes from 1993-2000 that may have died from asthma, confirming 63. Of these 263 athletes, 57% were considered elite and 10% occurred in those with no history of asthma. From Asthma in America: A Landmark Survey, Executive Summary (69), 48% of people with asthma report it limits their ability to participate in sports or recreation (LOE III).

Pathophysiology

The pathology of asthma centers on airflow obstruction and subsequent airway remodeling. A combination of smooth muscle constriction and hypertrophy, airway wall edema, intraluminal mucus hypersecretion, and submucosal inflammatory cell infiltration results in the physiologic consequence of reduced airway luminal diameter. This leads to the hallmarks of asthma— reversible bronchoconstriction, airway inflammation, and hyperresponsiveness to stimuli. A prospective cohort by Lange (70) (Level II) discovered a potential for accelerated loss of lung function with an average decline in FEV1 significantly greater in asthmatics versus control over a 15 year period.

Evaluation and Diagnosis

There are several patterns in the way in which asthma presents. These include intermittent attacks superimposed on an *asymptomatic* baseline, chronic symptoms punctuated by intermittent worsening and acute exacerbations which can be slow and progressive or may be sudden and without warning. A clear component of asthma is genetic in nature while other factors such as atopy and African-American race also increase the risk of developing asthma. In addition, infection, tobacco smoke, allergens, weather changes, environmental exposures and exercise have all been identified as just a few of the multiple triggers associated in the exacerbation of disease. In fact, 50-90% of asthmatics have been noted to have exercise as a trigger (71)(Level III), (72)(Level IV). Meanwhile, common signs and symptoms of asthma include cough, wheezing, shortness of breath, chest tightness or pain, fatigue, impaired performance, GI discomfort, decreased air entry, a prolonged expiratory phase, and an increased work of breathing. Caution must be used in the setting of athletes as the fear of losing playing time and the misinterpretation for poor conditioning can delay proper diagnosis.

Based on severity of disease, asthma can be classified into four distinct yet dynamic categories. These categories are mild intermittent, mild persistent, moderate persistent, and severe

persistent. Overall symptoms, number of exacerbations, nighttime symptoms, and lung function are the features that determine to which class an individual belongs.

Sideline Management and Treatment

The mainstay of treatment is pharmacologic. A Level III study by Anderson (73) revealed that the use of an albuterol metered-dose inhaler (MDI) (2 puffs) with spacer 15 minutes prior to exercise provides 80% protection for up to two hours. It is also strongly recommended to maximize control of the patient's baseline asthma. The underlying inflammatory process is ongoing and requires treatment attention, not just the acute attacks or exacerbations.

Treatment is guided by asthma severity and requires a stepwise approach with regular follow-up. It is important to understand that frequent rescue medication use is associated with deteriorating control. There are two main categories of medications, rescue (bronchodilators) and controller (immunomodulators). Bronchodilators relax airway smooth muscle and the most common is the inhaled beta-agonist, albuterol. Meanwhile, corticosteroids are the most potent anti-inflammatory agent for asthma (Level I) and can be delivered via a systemic or inhaled route. Multiple Level II studies support the regular use of inhaled budesonide in reducing symptom severity and improving performance in athletes with EIA (74-76).

Trigger control measures should also be employed except for the avoidance of exercise. In fact, some studies have shown the benefit of exercise for those with asthma. Emter showed an increase in FEV1 for those following a scheduled exercise program (77) (Level II) while athletes participating in studies by McKenzie (78) (Level III) and Mickleborough (79) (Level II) had a decrease in symptoms when performing a specified warm-up prior to competition.

Emergent treatment on the field again involves the use of albuterol at the recommended dose of 2 inhalations. However, if the athlete is not responsive, the continued use should be considered as transport to definitive care takes place. Other drugs of choice in an uncontrolled exacerbation include oxygen and epinephrine.

There are no validated guidelines for return to play of an athlete with EIA with all recommendations Level V in nature. Part of being prepared as a team physician is developing an appropriate differential diagnosis and being able to stabilize, treat, and transport as indicated. For athletes in which asthma is suspected, other respiratory disorders deserving consideration include vocal cord dysfunction, hyperventilation syndrome, foreign body aspiration, pneumothorax, and exercise-induced anaphylaxis.

Prevention and Preparedness

Trigger control is extremely important in an attempt to prevent or minimize symptoms. Sport- or athlete-specific triggers (i.e. pool chlorination) need to be taken into consideration beyond the standard control measures. A well-stocked sideline bag is also of utmost importance. In addition to the standard BLS tools, at a minimum, equipment should include an albuterol inhaler with spacer, an epi-pen for asthma treatment, and a 14 gauge, 1.75" angiocatheter for possible decompression of a tension pneumothorax.

Conclusions

Those affected by asthma are far-reaching and ever increasing, and athletes are no exception. The key elements of assessment and monitoring are severity, control, and responsiveness to treatment. Treatment involves a team approach based on education, prevention, and maintenance with goal of full athletic participation. Appropriate asthma management requires the proper use of long term and quick relief medications. The team physician must keep an open mind for nontraditional presentations or other potential etiologies.

Anaphylaxis

Clinical Vignette

During a softball game, an outfielder is seen wildly waving her arms. She begins running toward the dugout, but stops and kneels to the ground. Immediate assessment reveals facial swelling, including her tongue and lips, and stridor. What emergent care must be provided?

Epidemiology

Anaphylaxis is a serious allergic reaction that is rapid in onset and may cause death according to the US National Institute of Allergy and Infectious Diseases and the Food Allergy and Anaphylaxis Network (80). The lifetime individual risk of anaphylaxis is presumed to be 1-3% with a 1% mortality rate. Western countries, through a variety of approaches, estimate the incidence anywhere from 8-50 per 100,000 person-years. Prevalence estimates range from 1.21% to 15.04% in the United States and 0.002% for fatal events caused by food, drugs, latex, or insect stings.

Exercise Induced Anaphylaxis

Exercise Induced Anaphylaxis (EIAn) is anaphylaxis that occurs only in association with physical exertion. Intensity of exercise is believed to play a role and often at-risk individuals can tolerate low intensity activities, such as yard work, without symptoms. Symptoms begin to subside immediately upon cessation of exercise. EIAn is rare and more commonly seen in adolescents and young adults. A cross-sectional survey of school nurses in Japan (81) found a prevalence of 0.03% with equal numbers of males and females affected.

There is a subset of exercise induced anaphylaxis that is associated with ingestion of a trigger food referred to as food dependent exercise induced anaphylaxis (FDEIAn). This subset is even rarer and in the same cross-sectional survey was found to have a prevalence of 0.017% (81). Often it is one particular food, but in some individuals it is with the ingestion of any food and solids are more commonly triggers than liquids. The anaphylactic episode will generally occur during or

immediately after exercise that occurs within two hours of the ingested food. Patients may be able to exercise without anaphylactic symptoms after a fasting state of typically two hours. Several theories have been postulated including increased gastrointestinal permeability during exercise allowing intact allergens to enter into the blood stream.

Insect-venom Anaphylaxis

Insect-venom allergy is one of the most common triggers of anaphylaxis. It is estimated that up to 3% of the adult population will experience insect-venom anaphylaxis in the United States. Annually, it accounts for about 40-50 deaths. Hymenoptera envenomations, such as bees, account for the majority of clinically significant allergic reactions. The rate of anaphylaxis following insect stings is unknown, but is estimated to be between 0.3 and 3 percent of stings. Reactions may occur at any age, but are most common in individuals under age 20 with a 2:1 male predominance. Despite the higher incidence of anaphylaxis in young males, most fatalities occur in adults, especially those with cardiovascular comorbidities and poor tolerance for biochemical and physiologic stress (82). A history of atopy is a risk factor, but one-half of deaths from insect-venom anaphylaxis occur in individuals with no atopic history. Unlike food induced anaphylaxis, the skin is the most affected organ followed by respiratory symptoms. The risk of recurrent anaphylaxis is 60 percent (83).

Pathophysiology

Anaphylaxis is traditionally classified as either "immunologic" or "non-immunologic." Immunologic refers to IgE-mediated and immune complex/complement-mediated reactions. Non-immunologic anaphylaxis is caused by triggers that induce sudden, massive mast cell or basophil degranulation without antibody involvement.

In IgE-mediated anaphylaxis, mast cells, basophils, and eosinophils are activated, initiating degranulation causing release of preformed inflammatory mediators such as histamine, tryptase, chymase, heparin, histamine-releasing factor, and platelet activating factor. Systemic release of

histamine leads to cardiovascular and hemodynamic changes. Unlike the local release of histamine, systemic release is not associated with urticaria. In a study on the effects of histamine in normal individuals, the systemic effects of histamine were dose dependent (84). At low plasma levels, histamine was associated with a 30 percent increase in heart rate. Moderate levels caused flushing and headache. Higher levels were associated with a 30 percent increase in pulse pressure. This study also pretreated patients with H1 and H2 blockers to determine which receptors mediated each response. H1 and H2 receptors were responsible for flushing, hypotension, and headache. H1 receptors alone were responsible for tachycardia, bronchospasm, pruritis, and rhinorrhea.

Tryptase is a protease found mainly in mast cells. Similar to histamine, tryptase levels correlate with the severity of the anaphylactic episode. The exception to this is in food anapylaxis. It is not completely understood why food allergens are associated with minimal or no elevation of tryptase, but it is thought to be related to the subtype of the mast cell that is initially encountered by the antigen. Mast cells in the small intestine have significantly lower levels of tryptase than those contained in connective tissue. The mature form of tryptase, beta-tryptase, is enzymatically active and can activate the complement and coagulation pathways and the kallikerin-kinin contact system. Potential effects on the host include hypotension, angioedema, clotting and clot lysis leading to disseminated intravascular coagulation (85).

Anaphylaxis can involve any organ system, but the organs most commonly leading to shock and death are the heart and lungs. The most common effects on the cardiac system are myocardial depression, ischemia, and arrhythmias. The lungs can be affected in the lower airways with bronchoconstriction and mucous plugging or the upper airways with laryngeal edema. A retrospective chart review of 202 anaphylaxis fatalities occurring in the UK from 1992 to 2001 demonstrated an interval between onset of food anaphylaxis and death to be 25-30 minutes, in insect stings, 10-15 minutes, and drug mediated anaphylaxis averaged 5 minutes for patients in hospital, and 10-20 minutes for patients pre-hospital (82).

Diagnosis

Clinical diagnosis is based on probability and pattern recognition. In 2006 an expert panel was assembled for a second time from the National Institute of Allergy and Infectious Disease and Food Allergy and Anaphylaxis Network to create clinical criteria to facilitate the diagnosis of anaphylaxis. The panel created three criteria that would encompass 96% of reactions that are anaphylaxis. The first criteria identifies an acute onset of illness involving the skin and/or mucosal tissue in combination with respiratory symptoms (dyspnea, bronchospasm, wheezing) or cardiovascular symptoms (tachycardia, hypotension, syncope) as highly likely to be anaphylaxis. The second and third categories broaden the definition to include mild reactions to a known allergen that involve two or more of the following systems, skin, cardiac, pulmonary, and gastrointestinal or a drop in blood pressure alone (86).

Confirmation of the diagnosis with identification of the causative agent can only be confirmed by challenging the patient with the suspected agent. Allergen challenge is generally contraindicated due to ethical and safety concerns. The offending agent most commonly is confirmed historically from re-exposure causing anaphylaxis.

Sideline Management/Treatment

The initial treatment of anaphylaxis is the same, regardless of the type of anaphylaxis. After ensuring that you have a secure airway, you should remove the suspected trigger (i.e. remove stinger, stop infusion of a medication, etc), call for help, and initiate treatment with epinephrine. There are no absolute contraindications to epinephrine in the setting of anaphylaxis. Therapeutic recommendations for epinephrine administration are based primarily off results from clinical pharmacology studies, clinical observation, and animal models. There are few controlled clinical trials and no placebo controlled clinical trials due to the preponderance of data supporting the efficacy and need for immediate administration of epinephrine for survival in anaphylaxis (87). Efficacy comparisons of route of administration have not been performed during acute anaphylaxis.

A study looking at absorption of epinephrine in asymptomatic individuals demonstrated that there was more rapid and complete absorption with higher plasma levels with intramuscular (IM) administration into the anterolateral thigh of adults and children versus subcutaneous (SQ) administration (88). This study supports IM administration as the preferred route of initial administration.

Auto injection epinephrine delivery devices should be a part of every sideline medical kit. Epinephrine should be administered as soon a serious anaphylaxis event is recognized. Fatal outcomes have been reported when administration of epinephrine is delayed until hypotension or other end-organ symptoms are recognized. Epinephrine has a short half-life and additional doses should be administered IM every 5-15 minutes to control respiratory and cardiovascular symptoms as needed.

Auto-injectors come in 4 different types, two for pediatric dosing and two for adult dosing that differ based on containing a single dose of epinephrine versus a double dose that is delivered as two independent doses. The single adult dose auto-injector contains one 0.3 mg dose of epinephrine whereas the pediatric auto-injector contains a one-time dose of 0.15 mg epinephrine. The two dose auto-injector is able to deliver two independent doses of 0.3 mg for adults or 0.15 mg epinephrine for pediatric patients (defined as weight less than 25 kg).

If available, other adjunctive therapies may be helpful after the administration of epinephrine. These include placement of patient in a recumbent position with lower extremities elevated to maximize perfusion of vital organs, supplemental oxygen 6 to 8 liters by face mask, two large-bore IVs for aggressive fluid resuscitation due to the dramatic fluid shifts that may accompany anaphylaxis, H1 and H2 antihistamines, steroids, and bronchodilators. Little evidence exists to support the use of these adjunctive medications in acute anaphylaxis and the evidence that is available is low level. A systematic review of the use of H antihistamines in anaphylaxis failed to identify any Level I studies to support their use (89). Despite this, H1 antihistamines are the most

commonly administered medications in the treatment of acute anaphylaxis. H1 antihistamines relieve itching and hives, but much like the other adjunctive treatments, do not relieve any of the life threatening symptoms of anaphylaxis such as upper and lower airway obstruction, hypotension, and shock.   Therefore, these adjunctive medications should only be administered after IM epinephrine has been given.

Prevention

Identification of trigger is paramount in preventing anaphylaxis. The only definitive way to avoid a future episode is to avoid exposure with the trigger. A thorough history including any foods consumed in the 24 hours previous to the episode is important to identify potential triggers. For certain allergens skin testing or serum assays can be performed in follow-up with an allergist to help identify potential triggers.

Education of patients and their families about anaphylaxis, trigger avoidance, and the use of epinephrine auto-injectors that is reinforced annually reduces the recurrence of anaphylaxis (90). One intervention that concisely addresses this information is the development of an individualized emergency action plan. This intervention has been very effective in other life threatening chronic diseases such as asthma.

Prevention of EIAn and FDEIAn should also include a discussion on avoidance of eating the trigger food (or any solid food if no specific trigger identified) a minimum of 4 hours prior to physical activity initially and then may be gradually shortened to a minimum of 2 hours as tolerated by no further episodes of anaphylaxis. In addition, there is anecdotal evidence and case reports (90) that in some patients with EIAn that an antihistamine given 2 hours prior to physical activity or daily depending on the athlete's practice schedule decreases the frequency and severity of episodes. There is no evidence on which individuals may benefit from this intervention. Therefore, a trial of an H1 antihistamine is recommended, but may be discontinued if it does not demonstrate a benefit to the patient. Non-sedating second generation antihistamines, such as cetirizine (10 mg

1-2 times daily), fexofenadine (180 mg 1-2 times daily), or loratidine (10 mg 1-2 times daily) are used more commonly to minimize unwanted side effects.

Exercise should be resumed gradually as there seems to be an exertional threshold for most patients. EIAn is unlikely to progress beyond the early stage if exercise is immediately stopped. Patients should also not be allowed to exercise without supervision by an individual who is educated in the signs and symptoms of anaphylaxis and knows where the auto-injector is located and how to use it. The auto-injector should be with the athlete at all times as to not delay treatment. A phone should also be immediately available. A natural history study of a retrospective cohort of individuals with EIAn also demonstrated that patients who participated in sports that allowed for a gradual warm-up period before exercise reduced anaphylaxis episodes (91).

In insect-venom anaphylaxis venom immunotherapy lowers the risk of recurrent anaphylaxis from approximately 33 percent to 5 percent in children with a history of a moderate to severe reaction (92). Immunotherapy is therefore recommended in individuals with a history of systemic allergic reaction and a positive skin test. The risk of recurrence in adults is 60 percent and is also reduced with immunotherapy and is therefore recommended as well in patients with positive skin tests.

Prevention

In summary, anaphylaxis is a systemic allergic reaction that may be difficult to diagnose due to its varying presentations and can rapidly progress to death. Anaphylaxis is most commonly triggered by food, insect venom, medications, and exercise. It is an IgE mediated allergic reaction that can be confirmed by plasma histamine or tryptase levels. Prompt recognition and treatment with epinephrine is critical to prevent fatality, as death usually occurs within 30 minutes of exposure to the trigger. Once an anaphylactic episode is identified, management should include assessing the ABCs, summoning emergency medical personnel, placing the patient in a recumbent position with legs elevated, removing the inciting agent, and administration of IM epinephrine in

the lateral thigh. After the acute event has resolved, the offending trigger should be identified and then the family and the patient should be counseled on trigger avoidance, use of epinephrine auto-injectors, and provided with an anaphylaxis emergency action plan to prevent future episodes.

Lightning at Sporting Events

Clinical Vignette

A sixteen year old baseball player is struck by lightning as he stands on the mound preparing to throw a pitch. He lays motionless as the eight other players, who were knocked to the ground, are rolling around moaning with their hands on their ears. In this mass casualty scenario, who should be evaluated and treated first? Should CPR be initiated in the pulseless, apneic victim of a lighting strike when there are other victims with injuries?

Epidemiology

Lightning is estimated to kill about 1000 people worldwide every year, but most (about 70%) lightning injuries are not fatal (93). Lightning is a hazard during outdoor activities, especially for athletes during the summer months. The majority of lightning casualties in the US are reported during outdoor recreation and sporting activities (94). School and health care professionals taking care of athletic events should have lightning strike prevention and treatment protocols in place.

Pathophysiology

Lightning strikes can cause damage to the body via several mechanisms – direct strike, side splash (jumping from another object), contact injury (directly from another object), ground current or blunt trauma (being thrown) (93). Direct strikes cause the most damage because of the amount of energy passing directly through the body. While persons struck by lightning show evidence of multisystem derangement, the most dramatic effects involve the cardiovascular and central nervous systems. Cardiopulmonary arrest is the most common cause of death in lightning victims

(95).   There are many neurologic complications of lightning strike, some of which are temporary and benign, however, persistent symptoms of encephalopathy and myelopathy may require long-term neurorehabilitation (96). Other findings associated with lightning strikes are head injury, fractures, contusions, tympanic membrane rupture, pneumothorax and hematological abnormalities, such as disseminated intravascular coagulation.   Paralysis of the extremities caused by lightning (keraunoparalysis) is not typically the result of a neurological injury but is due to intense vasospasm which typically resolves within hours (93).

Diagnosis/initial patient assessment

Diagnosis of lightning strike is usually made by direct observation.   Victims laying still should immediately be evaluated using the ABCs as cardiopulmonary arrest is a potential result of lighting strike. Often the heart will start beating again on its own but the victim may still die because of hypoxia from respiratory arrest.   Early initiation of CPR may prevent this hypoxia and allow full resuscitation without the need for an AED (97). Victims may be found wandering around and not know what happened to them.   Care must be taken to look for the characteristic skin findings evidenced by ferning or Lichtenberg figures (not burns) (Figure 11).   Severity of skin findings does not correlate well with internal damage so all victims should be transported to the hospital for further assessment.

Management/Sideline Treatment

In the treatment of lightning strike victims the first concern must be for the safety of the rescuers.   Once the scene is determined to be safe, victims should be removed to safety.   There is no danger of residual charge to the rescuer by touching a lightning strike victim.   Each victim should be evaluated using the ABCs.   The term "reverse triage" has been used to determine who to treat first in the case of multiple victims from lightning strike. Unlike other triage scenarios, victims of lightning strike who are pulseless and apneic should be treated first.   Lightning typically produces cardiac standstill and due to cardiac automaticity, spontaneous return of normal heart

rhythm can occur, however recovery from respiratory arrest may be more prolonged and patients will benefit from rescue breathing (93).

Prevention/Preparedness Options

Most lightning injuries can be avoided if a storm monitoring system is in place and proper precautions are taken when storm conditions are present. Most thunderstorms occur in the summer months during the afternoon and evening hours. The majority of lightning strikes occur when the storm is approaching or passing. Storms should be monitored using computer weather maps or weather radios. Multiple types of lightning detectors are available but their accuracy is unknown. The flash to bang method is one means of deciding when it is necessary to clear the playing field (93). After the flash of the lightning is observed, seconds are counted until the bang of thunder is heard. Every five seconds between flash and bang represents one mile of distance between the lightning strike and where you are. The "30-30 rule" states that if the lightning is detected within 6 miles (30 seconds) the area should be cleared immediately for at least 30 minutes (94).

Safe areas are dwellings that have wiring or piping to ground the structure and vehicles with metal roofs (98). Once inside, keep away from windows and doors and off computers and phones. Dugouts, benches, rain shelters, tents and other ungrounded structures are not considered safe and should be evacuated (98). It should be determined ahead of time who is assigned to monitor for storms and who has the authority to clear the field.

If caught out in a lightning storm, stay away from highpoints, single trees, power lines, light posts, flag poles, water, and open areas. The crouch position should be adopted immediately if there is a sensation of hair "standing on end," crackling noises or a visible glow as this may indicate an imminent lightning strike. By squatting down on the balls of the feet, with the feet together and hands over the ears, the possible contact area is decreased should a lightning strike occur (93).

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Figure Legends

1   Facemask removal tools. (Left to right: Screwdriver, electric screwdriver, anvil/garden pruners, FM Xtractor, FM Xtractor, Dremel, push key for Riddell Revolution Speed.)

2   Types of Football Helmets. Players on the same team may have different helmets with varied facemask clips.   Medical staff should be prepared to cut a variety of clips. Examples from one team from left to right: (A) Schutt Pro Air II, (B) Riddell Revolution, (C) Riddell Revolution Speed.

3   Removing the facemask. Side clips should be removed first in order to gain quick access to the airway. (A) Clips may be removed by cutting, using a screwdriver, or depressing the button. (B) Extreme caution should be used as the flipped facemask can act as a lever arm causing rotation on the c-spine. (C) Front clips should be removed next.

4   Log-rolling the player. (A) The first responder should immediately stabilize the head and neck. (B-D) The responder stabilizing the neck should then direct additional responders to log-roll the player maintaining in-line spinal immobilization.

5   Log-rolling onto a spine board.

6   Five-man lift. One responder stabilizes the head and neck. On his call, two or more responders on each side of the player lift at the player's trunk, waist, and legs. The player should be lifted a few inches off the ground while another responder slides the spine board under the player from his feet up toward his head.

7 Removing the helmet. (A) The head and neck should be stabilized by one responder from the front of the player. The chin straps should be cut. (B) From the top, another responder should apply outward traction at the sides of the helmet (ear pieces may need to be removed) and the helmet should be removed. (C) Cervical stabilization must be continued until adequate padding has been placed under the head, or the shoulder pads have been removed.

8 Removing the shoulder pads. (A) While maintaining cervical stabilization, the straps in the front of the shoulder pads and around the arms should be cut. (B) The pads may then be removed from the player's head. Alternately, the shoulder pads may be removed by cutting the front straps, then half log-rolling the player and cutting the rear straps. Each half can then be removed while maintaining spinal immobilization.

9 Player fully packaged for transport.

10 Algorithm for managing a collapsed athlete with suspected cardiac arrest. (From *Circulation* 2005; 112(24 IV) Figure 2.)

11 Feathering, ferning, or Lichtenberg's flowers are pathognomonic of lightning injuries. (From *New England Journal of Medicine* 343:1536, November 23, 2000).
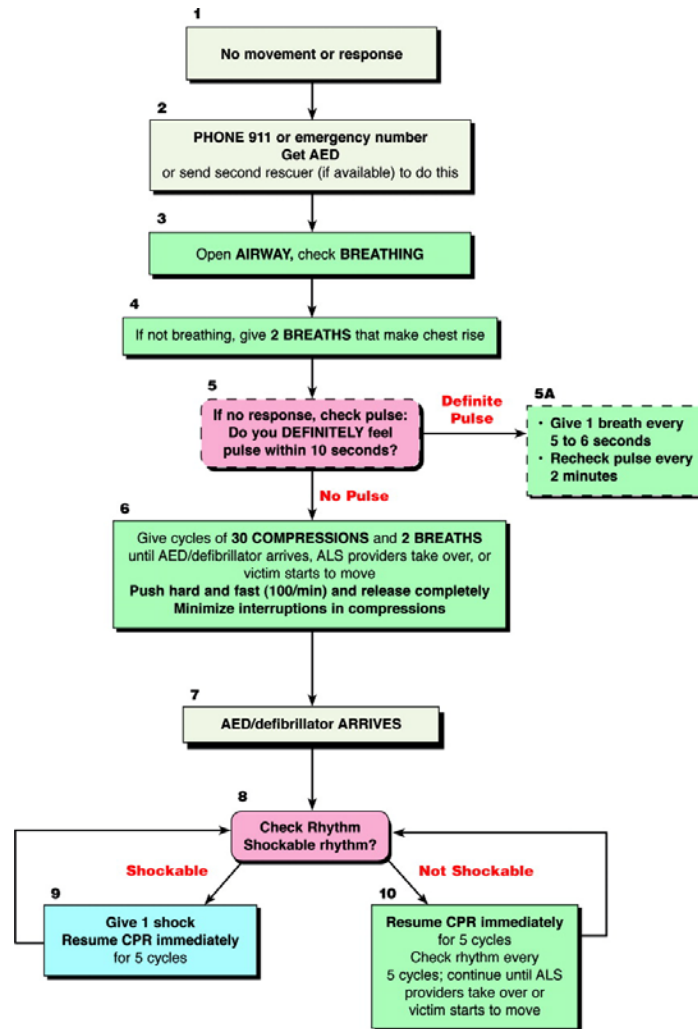
A

B

C

D

A   **B**

A   B

Adult BLS Healthcare Provider Algorithm

1 No movement or response

2 PHONE 911 or emergency number
Get AED
or send second rescuer (if available) to do this

3 Open AIRWAY, check BREATHING

4 If not breathing, give 2 BREATHS that make chest rise

5 If no response, check pulse: Do you DEFINITELY feel pulse within 10 seconds?

Definite Pulse

5A
· Give 1 breath every 5 to 6 seconds
· Recheck pulse every 2 minutes

No Pulse

6 Give cycles of 30 COMPRESSIONS and 2 BREATHS until AED/defibrillator arrives, ALS providers take over, or victim starts to move
Push hard and fast (100/min) and release completely
Minimize interruptions in compressions

7 AED/defibrillator ARRIVES

8 Check Rhythm Shockable rhythm?

Shockable

Not Shockable

9 Give 1 shock
Resume CPR immediately for 5 cycles

10 Resume CPR immediately for 5 cycles
Check rhythm every 5 cycles; continue until ALS providers take over or victim starts to move

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