Journal of Athletic Training 2011:47(1):1–24

www.nata.org/jat

National Athletic Trainers' Association Position Statement: Preventing Sudden Death in Sports

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Objective: To present recommendations for the prevention and screening, recognition, and treatment of the most common conditions resulting in sudden death in organized sports.

Background: Cardiac conditions, head injuries, neck injuries, exertional heat stroke, exertional sickling, asthma, and other factors (eg, lightning, diabetes) are the most common causes of death in athletes.

Recommendations: These guidelines are intended to provide relevant information on preventing sudden death in sports and to give specific recommendations for certified athletic trainers and others participating in athletic health care.

Key Words: asthma, cardiac conditions, diabetes, exertional heat stroke, exertional hyponatremia, exertional sickling, head injuries, neck injuries, lightning safety

Sudden death in sports and physical activity has a variety of causes. The 10 conditions covered in this position statement are

- Asthma
- Catastrophic brain injuries
- Cervical spine injuries
- Diabetes
- Exertional heat stroke
- Exertional hyponatremia
- Exertional sickling
- Head-down contact in football
- Lightning
- Sudden cardiac arrest

(Order does not indicate rate of occurrence.)

Recognizing the many reasons for sudden death allows us to create and implement emergency action plans (EAPs) that provide detailed guidelines for prevention, recognition, treatment, and return to play (RTP). Unlike collegiate and professional teams, which usually have athletic trainers (ATs) available, nearly half of high schools as well as numerous other athletic settings lack the appropriate medical personnel to put these guidelines into practice and instead rely on the athletic director, team coach, or strength and conditioning specialist to do so.

To provide appropriate care for athletes, one must be familiar with a large number of illnesses and conditions in order to properly guide the athlete, determine when emergency treatment is needed, and distinguish among similar signs and symptoms that may reflect a variety of potentially fatal circumstances. For the patient to have the best possible outcome, correct and prompt emergency care is critical; delaying care until the ambulance arrives may result in permanent disability or death. Therefore, we urgently advocate training coaches in first aid, cardiopulmonary resuscitation (CPR), and automated external defibrillator (AED) use, so that they can provide treatment until a medical professional arrives; however, such training is inadequate for the successful and complete care of the conditions described in this position statement. Saving the life of a young athlete should not be a coach's responsibility or liability.

For this reason, we also urge every high school to have an AT available to promptly take charge of a medical emergency. As licensed medical professionals, ATs receive thorough training in preventing, recognizing, and treating critical situations in the physically active. Each AT works closely with a physician to create and apply appropriate EAPs and RTP guidelines.

Throughout this position statement, each recommendation is labeled with a specific level of evidence based on the Strength of Recommendation Taxonomy (SORT).[1] This taxonomy takes into account the quality, quantity, and consistency of the evidence in support of each recommendation: Category A represents consistent good-quality evidence, B represents

inconsistent or limited-quality or limited-quantity evidence, and C represents recommendations based on consensus, usual practice, opinion, or case series.

The following rules apply to every EAP:

1. Every organization that sponsors athletic activities should have a written, structured EAP. *Evidence Category: B*
2. The EAP should be developed and coordinated with local EMS staff, school public safety officials, onsite first responders, school medical staff, and school administrators. *Evidence Category: B*
3. The EAP should be specific to each athletic venue. *Evidence Category: B*
4. The EAP should be practiced at least annually with all those who may be involved. *Evidence Category: B*

Those responsible for arranging organized sport activities must generate an EAP to directly focus on these items:

1. Instruction, preparation, and expectations of the athletes, parents or guardians, sport coaches, strength and conditioning coaches, and athletic directors.
2. Health care professionals who will provide medical care during practices and games and supervise the execution of the EAP with respect to medical care.
3. Precise prevention, recognition, treatment, and RTP policies for the common causes of sudden death in athletes.

The EAP should be coordinated and supervised by the onsite AT. A sports organization that does not have a medical supervisor, such as an AT, present at practices and games and as part of the medical infrastructure runs the risk of legal liability. Athletes participating in an organized sport have a reasonable expectation of receiving appropriate emergency care, and the standards for EAP development have also become more consistent and rigorous at the youth level. Therefore, the absence of such safeguards may render the organization sponsoring the sporting event legally liable.

The purpose of this position statement is to provide an overview of the critical information for each condition (prevention, recognition, treatment, and RTP) and indicate how this information should dictate the basic policies and procedures regarding the most common causes of sudden death in sports. Our ultimate goal is to guide the development of policies and procedures that can minimize the occurrence of catastrophic incidents in athletes. All current position statements of the National Athletic Trainers' Association (NATA) are listed in the Appendix.

ASTHMA

Recommendations

Prevention and Screening

1. Athletes who may have or are suspected of having asthma should undergo a thorough medical history and physical examination.[2] *Evidence Category: B*
2. Athletes with asthma should participate in a structured warmup protocol before exercise or sport activity to decrease reliance on medications and minimize asthmatic symptoms and exacerbations.[3] *Evidence Category: B*
3. The sports medicine staff should educate athletes with asthma about the use of asthma medications as prophylaxis before exercise, spirometry devices, asthma triggers, recognition of signs and symptoms, and compliance with monitoring the condition and taking medication as prescribed. *Evidence Category: C*

Recognition

4. The sports medicine staff should be aware of the major asthma signs and symptoms (ie, confusion, sweating, drowsiness, forced expiratory volume in the first second [FEV_1] of less than 40%, low level of oxygen saturation, use of accessory muscles for breathing, wheezing, cyanosis, coughing, hypotension, bradycardia or tachycardia, mental status changes, loss of consciousness, inability to lie supine, inability to speak coherently, or agitation) and other conditions (eg, vocal cord dysfunction, allergies, smoking) that can cause exacerbations.[4,5] *Evidence Category: A*
5. Spirometry tests at rest and with exercise and a field test (in the sport-specific environment) should be conducted on athletes suspected of having asthma to help diagnose the condition.[2,6] *Evidence Category: B*
6. An increase of 12% or more in the FEV_1 after administration of an inhaled bronchodilator also indicates reversible airway disease and may be used as a diagnostic criterion for asthma.[7]

Treatment

7. For an acute asthmatic exacerbation, the athlete should use a short-acting β_2-agonist to relieve symptoms. In a severe exacerbation, rapid sequential administrations of a β_2-agonist may be needed. If 3 administrations of medication do not relieve distress, the athlete should be referred promptly to an appropriate health care facility.[8] *Evidence Category: A*
8. Inhaled corticosteroids or leukotriene inhibitors can be used for asthma prophylaxis and control. A long-acting β_2-agonist can be combined with other medications to help control asthma.[9] *Evidence Category: B*
9. Supplemental oxygen should be offered to improve the athlete's available oxygenation during asthma attacks.[10] *Evidence Category: B*
10. Lung function should be monitored with a peak flow meter. Values should be compared with baseline lung volume values and should be at least 80% of predicted values before the athlete may participate in activities.[11] *Evidence Category: B*
11. If feasible, the athlete should be removed from an environment with factors (eg, smoke, allergens) that may have caused the asthma attack. *Evidence Category: C*
12. In the athlete with asthma, physical activity should be initiated at low aerobic levels and exercise intensity gradually increased while monitoring occurs for recurrent asthma symptoms. *Evidence Category: C*

Background and Literature Review

Definition, Epidemiology, and Pathophysiology. In 2009, asthma was thought to affect approximately 22 million people in the United States, including approximately 6 million children.[4] *Asthma* is a disease in which the airways become inflamed and airflow is restricted.[4] Airway inflammation, which

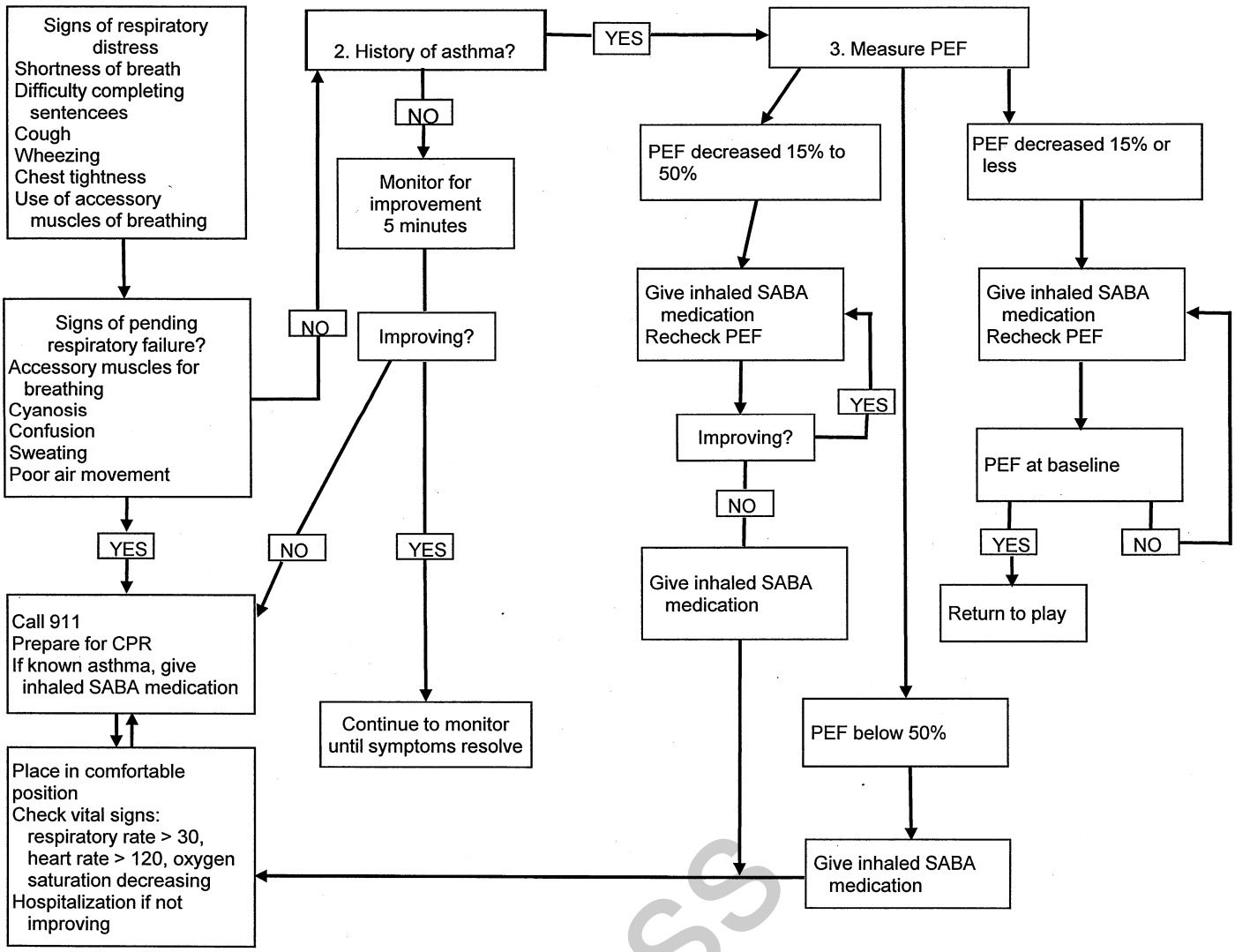

Figure 1. Asthma pharmacologic management. Abbreviations: CPR, cardiopulmonary resuscitation; PEF, peak expiratory flow; SABA, short-acting β_2-agonist. Casa DJ, *Preventing Sudden Death in Sport and Physical Activity*, 2012: Jones & Bartlett Learning, Sudbury, MA. www.jblearning.com. Reprinted with permission.

may lead to airway hyperresponsiveness and narrowing, is associated with mast cell production and activation and increased number of eosinophils and other inflammatory cells.[2,3] Cellular and mediator events cause inflammation, bronchial constriction via smooth muscle contraction, and acute swelling from fluid shifts. Chronic airway inflammation may cause remodeling and thickening of the bronchiolar walls.[12,13]

Clinical signs of asthma include confusion, sweating, drowsiness, use of accessory muscles for breathing, wheezing, coughing, chest tightness, and shortness of breath. Asthma may be present during specific times of the year, vary with the type of environment, occur during or after exercise, and be triggered by respiratory infections, allergens, pollutants, aspirin, nonsteroidal anti-inflammatory drugs, inhaled irritants, exposure to cold, and exercise.[5]

Prevention. Athletes suspected of having asthma should undergo a thorough health history examination and preparticipation physical examination. Unfortunately, the sensitivity and specificity of the medical history are not known, and this evaluation may not be the best method for identifying asthma.[14]

Performing warmup activities before sport participation can help prevent asthma attacks. With a structured warmup protocol, the athlete may experience a refractory period of as long as 2 hours, potentially decreasing the risk of an exacerbation or decreasing reliance on medications.[6] In addition, the sports medicine team should provide education to assist the athlete in recognizing asthma signs and symptoms, understanding how to use medication as prescribed (including potential adverse effects and barriers to taking medications, which can include failure to recognize the importance of controlling asthma, failure to recognize the potential severity of the condition, medication costs, difficulty obtaining medications, inability to integrate treatment of the disease with daily life, and distrust of the medical establishment), and using spirometry equipment correctly.[2,4,5]

Recognition. Athletes with asthma may display the following signs and symptoms: confusion, sweating, drowsiness, FEV_1 of less than 40%, low level of oxygen saturation, use of accessory muscles for breathing, wheezing, cyanosis, coughing, hypotension, bradycardia or tachycardia, mental status

changes, loss of consciousness, inability to lie supine, inability to speak coherently, or agitation.[2,4,5] Peak expiratory flow rates of less than 80% of the personal best or daily variability greater than 20% of the morning value indicate lack of control of asthma. The sports medicine staff should consider testing all athletes with asthma using a sport-specific and environment-specific exercise challenge protocol to assist in determining triggers of airway hyperresponsiveness.[6]

Treatment. Treatment for those with asthma includes recognition of exacerbating factors and the proper use of asthma medications (Figure 1). A short-acting β_2-agonist should be readily available; onset of action is typically 5 to 15 minutes, so the medication can be readministered 1 to 3 times per hour if needed.[10] If breathing difficulties continue after 3 treatments in 1 hour or the athlete continues to have any signs or symptoms of acute respiratory distress, referral to an acute or urgent care facility should ensue. For breathing distress, the sports medicine team should provide supplemental oxygen to help maintain blood oxygen saturation above 92%.[10]

Proper use of inhaled corticosteroids can decrease the frequency and severity of asthma exacerbations while improving lung function and reducing hyperresponsiveness and the need for short-acting β_2-agonists.[15,16] Leukotriene modifiers can be used to control allergen-, aspirin-, or exercise-induced bronchoconstriction and decrease asthma exacerbations.[17]

Return to Play. No specific guidelines describe RTP after an asthma attack in an athlete. However, in general, the athlete should first be asymptomatic and progress through graded increases in exercise activity. Lung function should be monitored with a peak flow meter and compared with baseline measures to determine when asthma is sufficiently controlled to allow the athlete to resume participation.[11] Where possible, the sports medicine staff should identify and treat asthmatic triggers, such as allergic rhinitis, before the athlete returns to participation.

CATASTROPHIC BRAIN INJURIES

Recommendations

Prevention

1. The AT is responsible for coordinating educational sessions with athletes and coaches to teach the recognition of concussion (ie, specific signs and symptoms), serious nature of traumatic brain injuries in sport, and importance of reporting concussions and not participating while symptomatic. *Evidence Category: C*
2. The AT should enforce the standard use of certified helmets while also educating athletes, coaches, and parents that although such helmets meet a standard for helping to prevent catastrophic head injuries, they do not prevent cerebral concussions. *Evidence Category: B*

Recognition

3. The AT should incorporate the use of a comprehensive objective concussion assessment battery that includes symptom, cognitive, and balance measures. Each of these represents only one piece of the concussion puzzle and should not be used in isolation to manage concussion. *Evidence Category: A*

Treatment and Management

4. A comprehensive medical management plan for acute care of an athlete with a potential intracranial hemorrhage or diffuse cerebral edema should be implemented. *Evidence Category: B*
5. If the athlete's symptoms persist or worsen or the level of consciousness deteriorates after a concussion, the patient should be immediately referred to a physician trained in concussion management. *Evidence Category: B*
6. Oral and written instructions for home care should be given to the athlete and to a responsible adult. *Evidence Category: C*
7. Returning an athlete to participation after a head injury should follow a graduated progression that begins once the athlete is completely asymptomatic. *Evidence Category: C*
8. The athlete should be monitored periodically throughout and after these sessions to determine whether any symptoms develop or increase in intensity. *Evidence Category: C*

Background and Literature Review

Definition, Epidemiology, and Pathophysiology. Cerebral concussion is classified as mild traumatic brain injury and often affects athletes in both helmeted and nonhelmeted sports.[18,19] The Centers for Disease Control and Prevention estimated that 1.6 to 3.8 million sport-related concussive injuries occur annually in the United States.[20] Although they are rare, severe catastrophic traumatic brain injuries, such as subdural and epidural hematomas and malignant cerebral edema (ie, second-impact syndrome), result in more fatalities from direct trauma than any other sport injury. When these injuries do occur, brain swelling or pooling of blood (or both) increases intracranial pressure; if this condition is not treated quickly, brainstem herniation and respiratory arrest can follow. Catastrophic brain injuries rank second only to cardiac-related injuries and illnesses as the most common cause of fatalities in football players.[21] However, the National Center for Catastrophic Sport Injury Research reported that fatal brain injuries have occurred in almost every sport, including baseball, lacrosse, soccer, track, and wrestling.[22] For a catastrophic brain injury such as second-impact syndrome, which has a mortality rate approaching 50% and a morbidity rate nearing 100%, prevention is of the utmost importance.

Prevention. Preventing catastrophic brain injuries in sports, such as skull fractures, intracranial hemorrhages, and diffuse cerebral edema (second-impact syndrome), must involve the following: (1) prevention and education about traumatic brain injury for athletes, coaches, and parents; (2) enforcing the standard use of sport-specific and certified equipment (eg, National Operating Committee on Standards for Athletic Equipment [NOCSAE] or Hockey Equipment Certification Council, Inc [HECC]–certified helmets); (3) use of comprehensive, objective baseline and postinjury assessment measures; (4) administration of home care and referral instructions emphasizing the monitoring and management of deteriorating signs and symptoms; (5) use of systematic and monitored graduated RTP progressions; (6) clearly documented records of the evaluation and management of the injury to help guide a sound RTP decision; and (7) proper preparedness for on-field medical management of a serious head injury.

Prevention begins with education. The AT is responsible for coordinating educational sessions with athletes and coaches to teach the recognition of concussion (ie, specific signs and

symptoms), serious nature of traumatic brain injuries in sport, and importance of reporting their injuries and not participating while symptomatic. During this process, athletes who are at risk for subsequent concussion or catastrophic injury should be identified and counseled about the risk of subsequent injury.

As recommended in the NATA position statement on management of sport-related concussion,[23] the AT should enforce the standard use of helmets for preventing catastrophic head injuries and reducing the severity of cerebral concussions in sports that require helmet protection (eg, football, men's lacrosse, ice hockey, baseball, softball). The AT should ensure that all equipment meets NOCSAE, HECC, or American Society for Testing and Materials (ASTM) standards. A poorly fitted helmet is limited in the amount of protection it can provide, and the AT must play a role in enforcing the proper fit and use of the helmet. Protective sport helmets are designed primarily to help prevent catastrophic injuries (eg, skull fractures and intracranial hematomas) and not concussions. A helmet that protects the head from a skull fracture does not adequately prevent the rotational and shearing forces that lead to many concussions,[24] a fact that many people misunderstand.

Recognition. The use of objective concussion measures during preseason and postinjury assessments helps the AT and physician accurately identify deficits associated with the injury and track recovery. However, neuropsychological testing is only one component of the evaluation process and should not be used as a standalone tool to diagnose or manage concussion or to make RTP decisions after concussion. Including objective measures of cognitive function and balance prevents premature clearance of an athlete who reports being symptom free but has persistent deficits that are not easily detected through the clinical examination. The concussion assessment battery should include a combination of tests for cognition, balance, and self-reported symptoms known to be affected by concussion. Because many athletes (an estimated 49% to 75%)[25,26] do not report their concussions, this objective assessment model is important. The sensitivity of this comprehensive battery, including a graded symptom checklist, computerized neuropsychological test, and balance test, reached 94%,[27] which is consistent with previous reports.[28,29]

Multiple concussion assessment tools are available, including low-technology and high-technology balance tests, brief paper-and-pencil cognitive tests, and computerized cognitive tests. As of 2010, the National Football League, National Hockey League, and National Collegiate Athletic Association require an objective assessment as part of a written concussion management protocol. By using objective measures, which were endorsed by the Third International Consensus Statement on Concussion in Sport (Zurich, 2008),[30,31] ATs and physicians are better equipped to manage concussion than by relying solely on subjective reports from the athlete. Additionally, the often hidden deficits associated with concussion and gradual deterioration that may indicate more serious brain trauma or postconcussion syndrome (ie, symptoms lasting longer than 4 weeks) may be detected with these tools.

Treatment. Once the athlete has been thoroughly evaluated and identified as having sustained a concussion, a comprehensive medical management plan should be implemented. This begins with making a determination about whether the patient should be immediately referred to a physician or sent home with specific observation instructions. Although this plan should include serial evaluations and observations by the AT (as outlined earlier), continued monitoring of postconcussion signs and symptoms by those with whom the athlete lives is both important and practical. If symptoms persist or worsen or the level of consciousness deteriorates after a concussion, the athlete should be immediately referred to a medical facility. To assist with this, oral and written instructions for home care should be given to the athlete and to a responsible adult (eg, parents or roommate) who will observe and supervise the athlete during the acute phase of the concussion while at home or in the dormitory. The AT and physician should agree on a standard concussion home instruction form similar to the one presented in the NATA position statement[23] and Zurich guidelines.[30,31]

The proper preparedness for on-field and sideline medical management of a head injury becomes paramount if the athlete has a more serious and quickly deteriorating condition. If the athlete presents with a Glasgow coma score of less than 8 or other indications of more involved brain or brainstem impairment appear (eg, posturing, altered breathing pattern), the AT or other members of the sports medicine team must be prepared to perform manual ventilations through either endotracheal intubation or bag-valve-mouth resuscitation. These procedures should be initiated if the athlete is not oxygenating well (ie, becoming dusky or blue, ventilating incompletely and slower than normal at 12 to 15 breaths per minute).[32] Additionally, the sports medicine team should aim to reduce intracranial pressure by elevating the head to at least 30° and ensuring that the head and neck are maintained in the midline position to optimize venous outflow from the brain. Hyperventilation and intravenous (IV) diuretics such as mannitol (0.5 to 1.0 g/kg) may also decrease intracranial pressure.[32] Obviously, being prepared for immediate transfer to a medical facility is extremely important under these conditions.

Return to Play. Once the athlete is asymptomatic, has been cleared by a physician with training in concussion management, and has returned to baseline on follow-up assessments, a graduated RTP protocol should begin (Table 1). If the exertional activities do not produce acute symptoms, he or she may progress to the next step. No more than 2 steps should be performed on the same day, which allows monitoring of both acute (during the activity) and delayed (within 24 hours after the activity) symptoms. The athlete may advance to step 5 and return to full participation once he or she has remained asymptomatic for 24 hours after step 4 of the protocol. The athlete should be monitored periodically throughout and after these sessions with objective assessment measures to determine whether an increase in intensity is warranted. If the athlete's symptoms return at any point during the RTP progression, at least 24 hours without symptoms must pass before the protocol is reintroduced, beginning at step 1.

Table 1. Graduated Return-to-Play Sample Protocol

Exertion Step	Activities
1.	20-min stationary bike at 10–14 mph (16–23 kph)
2.	Interval bike: 30-s sprint at 18–20 mph (29–32 kph), 30-s recovery × 10 repetitions; body weight circuit: squats, push-ups, sit-ups × 20 s × 3 repetitions
3.	60-yd (55-m) shuttle run × 10 repetitions with 40-s rest, plyometric workout: 10-yd (9-m) bounding, 10 medicine ball throws, 10 vertical jumps × 3 repetitions; noncontact, sport-specific drills × 15 min
4.	Limited, controlled return to practice with monitoring for symptoms
5.	Full sport participation in practice

The AT should document all pertinent information surrounding the evaluation and management of all suspected concussions, including (a) mechanism of injury; (b) initial signs and symptoms; (c) state of consciousness; (d) findings on serial testing of symptoms, neuropsychological function, and balance (noting any deficits compared with baseline); (e) instructions given to the athlete, parent, or roommate; (f) recommendations provided by the physician; (g) graduated RTP progression, including dates and specific activities involved in the athlete's return to participation; and (h) relevant information on the player's history of prior concussion and associated recovery patterns.[23] This level of detail can help prevent a premature return to participation and a catastrophic brain injury such as second-impact syndrome.

CERVICAL SPINE INJURIES

Recommendations

Prevention

1. Athletic trainers should be familiar with sport-specific causes of catastrophic cervical spine injury and understand the physiologic responses in spinal cord injury. *Evidence Category: C*
2. Coaches and athletes should be educated about the mechanisms of catastrophic spine injuries and pertinent safety rules enacted for the prevention of cervical spine injuries. *Evidence Category: C*
3. Corrosion-resistant hardware should be used in helmets, helmets should be regularly maintained throughout a season, and helmets should undergo regular reconditioning and recertification.[33] *Evidence Category: B*
4. Emergency department personnel should become familiar with proper athletic equipment removal, seeking education from sports medicine professionals regarding appropriate methods to minimize motion. *Evidence Category: C*

Recognition

5. During initial assessment, the presence of any of the following, alone or in combination, requires the initiation of the spine injury management protocol: unconsciousness or altered level of consciousness, bilateral neurologic findings or complaints, significant midline spine pain with or without palpation, or obvious spinal column deformity.[34–39] *Evidence Category: A*

Treatment and Management

6. The cervical spine should be in neutral position, and manual cervical spine stabilization should be applied immediately.[40,41] *Evidence Category: B*
7. Traction must not be applied to the cervical spine.[42,43] *Evidence Category: B*
8. Immediate attempts should be made to expose the airway. *Evidence Category: C*
9. If rescue breathing becomes necessary, the person with the most training and experience should establish an airway and begin rescue breathing using the safest technique.[44,45] *Evidence Category: B*
10. If the spine is not in a neutral position, rescuers should realign the cervical spine.[46,47] However, the presence or development of any of the following, alone or in combination, is a contraindication to realignment[45,48]: pain caused or increased by movement, neurologic symptoms, muscle spasm, airway compromise, physical difficulty repositioning the spine, encountered resistance, or apprehension expressed by the patient. *Evidence Category: B*
11. Manual stabilization of the head should be converted to immobilization using external devices such as foam head blocks.[47,49] Whenever possible, manual stabilization[50] is resumed after the application of external devices. *Evidence Category: B*
12. Athletes should be immobilized with a long spine board or other full-body immobilization device.[51,52] *Evidence Category: B*

Equipment-Laden Athletes

13. The primary acute treatment goals in equipment-laden athletes are to ensure that the cervical spine is immobilized in neutral and vital life functions are accessible. Removal of helmet and shoulder pads in any equipment-intensive sport should be deferred[53–56] until the athlete has been transported to an emergency medical facility except in 3 circumstances[57]: the helmet is not properly fitted to prevent movement of the head independent of the helmet, the equipment prevents neutral alignment of the cervical spine, or the equipment prevents airway or chest access.[53,54,58] *Evidence Category: C*
14. Full face-mask removal using established tools and techniques[59–61] is executed once the decision has been made to immobilize and transport. *Evidence Category: C*
15. If possible, a team physician or AT should accompany the athlete to the hospital. *Evidence Category: C*
16. Remaining protective equipment should be removed by appropriately trained professionals in the emergency department. *Evidence Category: C*

Background and Literature Review

Definition, Epidemiology, and Pathophysiology. A catastrophic cervical spinal cord injury occurs with structural distortion of the cervical spinal column and is associated with actual or potential damage to the spinal cord.[62] The spinal injury that carries the greatest risk of immediate sudden death for the athlete occurs when the damage is both severe enough and at a high enough level in the spinal column (above C5) to affect the spinal cord's ability to transmit respiratory or circulatory control from the brain.[63,64] The priority in these situations is simply to support the basic life functions of breathing and circulation. Unfortunately, even if an athlete survives the initial acute management phase of the injury, the risk of death persists because of the complex biochemical cascade of events that occurs in the injured spinal cord during the initial 24 to 72 hours after injury.[64] Because of this risk, efficient acute care, transport, diagnosis, and treatment are critical in preventing sudden death in a patient with a catastrophic cervical spine injury.

Treatment and Management. A high level of evidence (ie, prospective randomized trials) on this topic is rare, and technology, equipment, and techniques will continue to evolve, but

the primary goals offered in the NATA position statement on acute management of the cervical spine–injured athlete[65] remain the same: create as little motion as possible and complete the steps of the EAP as rapidly as is appropriate to facilitate support of basic life functions and prepare for transport to the nearest emergency treatment facility.

Additional complications can affect the care of the spine-injured athlete in an equipment-intensive sport when rescuers may need to remove protective equipment that limits access to the airway or chest. Knowing how to deal properly with protective equipment during the immediate care of an athlete with a potential catastrophic cervical spine injury can greatly influence the outcome. Regardless of the sport or the equipment, 2 principles should guide management of the equipment-laden athlete with a potential cervical spine injury:

1. Exposure and access to vital life functions (eg, airway, chest for CPR, or use of an AED) must be established or easily achieved in a reasonable and acceptable manner.
2. Neutral alignment of the cervical spine should be maintained while allowing as little motion at the head and neck as possible.

Return to Play. Return to play after cervical spine injury is highly variable and may be permitted only after complete tissue healing, neurologic recovery, and clearance by a physician. Factors considered for RTP include the level of injury, type of injury, number of levels fused for stability, cervical stenosis, and activity.[66]

DIABETES MELLITUS

Recommendations

Prevention

1. Each athlete with diabetes should have a diabetes care plan that includes blood glucose monitoring and insulin guidelines, treatment guidelines for hypoglycemia and hyperglycemia, and emergency contact information. *Evidence Category: C*
2. Prevention strategies for hypoglycemia include blood glucose monitoring, carbohydrate supplementation, and insulin adjustments. *Evidence Category: B*
3. Prevention strategies for hyperglycemia are described by the American Diabetes Association (ADA) and include blood glucose monitoring, insulin adjustments, and urine testing for ketone bodies.[67] *Evidence Category: C*

Recognition

4. Hypoglycemia typically presents with tachycardia, sweating, palpitations, hunger, nervousness, headache, trembling, or dizziness; in severe cases, loss of consciousness and death can occur. *Evidence Category: C*
5. Hyperglycemia can present with or without ketosis. Typical signs and symptoms of hyperglycemia without ketosis include nausea, dehydration, reduced cognitive performance, feelings of sluggishness, and fatigue. *Evidence Category: C*
6. Hyperglycemia with ketoacidosis may include the signs and symptoms listed earlier as well as Kussmaul breathing (abnormally deep, very rapid sighing respirations characteristic of diabetic ketoacidosis), fruity odor to the breath, unusual fatigue, sleepiness, loss of appetite, increased thirst, and frequent urination. *Evidence Category: C*

Treatment and Management

7. Mild hypoglycemia (ie, the athlete is conscious and able to swallow and follow directions) is treated by administering approximately 10–15 g of carbohydrates (examples include 4–8 glucose tablets or 2 tablespoons of honey) and reassessing blood glucose levels immediately and 15 minutes later. *Evidence Category: C*
8. Severe hypoglycemia (ie, the athlete is unconscious or unable to swallow or follow directions) is a medical emergency, requiring activation of emergency medical services (EMS) and, if the health care provider is properly trained, administering glucagon. *Evidence Category: C*
9. Athletic trainers should follow the ADA guidelines for athletes exercising during hyperglycemic periods. *Evidence Category: C*
10. Physicians should determine a safe blood glucose range to return an athlete to play after an episode of mild hypoglycemia or hyperglycemia. *Evidence Category: C*

Background and Literature Review

Definition, Epidemiology, and Pathophysiology. Diabetes mellitus is a chronic metabolic disorder characterized by hyperglycemia, caused by either absolute insulin deficiency or resistance to the action of insulin at the cellular level, which results in the inability to regulate blood glucose levels within the normal range of 70–110 mg/dL. Type 1 diabetes is an autoimmune disorder stemming from a combination of genetic and environmental factors. The autoimmune response is often triggered by an environmental event, such as a virus, and it targets the insulin-secreting beta cells of the pancreas. When beta cell mass is reduced by approximately 80%, the pancreas is no longer able to secrete sufficient insulin to compensate for hepatic glucose output.[67,68]

Prevention. Although the literature supports physical activity for people with type 1 diabetes, exercise training and competition can result in major disturbances to blood glucose management. Extreme glycemic fluctuations (severe hypoglycemia or hyperglycemia with ketoacidosis) can lead to sudden death in athletes with type 1 diabetes mellitus.[69–71] Prevention of these potentially life-threatening events begins with the creation of the diabetes care plan by a physician. The plan should identify blood glucose targets for practices and games, including exclusion thresholds; strategies to prevent exercise-associated hypoglycemia, hyperglycemia, and ketosis; a list of medications used for glycemic control; signs, symptoms, and treatment protocols for hypoglycemia, hyperglycemia, and ketosis; and emergency contact information.[72]

Preventing hypoglycemia relies on a 3-pronged approach of frequent blood glucose monitoring, carbohydrate supplementation, and insulin adjustments. The athlete should check blood glucose levels 2 or 3 times before, every 30 minutes during, and every other hour up to 4 hours after exercise. Carbohydrates should be eaten before, during, and after exercise; the quantity the athlete ingests depends on the prevailing blood

Table 2. Treatment Guidelines for Mild and Severe Hypoglycemia[76,77]

Mild Hypoglycemia	Severe Hypoglycemia
1. Give 10–15 g of fast-acting carbohydrate. Example: 4–8 glucose tablets, 2 Tbsp honey. 2. Measure blood glucose level. 3. Wait 15 min and remeasure blood glucose level. 4. If blood glucose level remains low, administer another 10–15 g of fast-acting carbohydrate. 5. Recheck blood glucose level in 15 min. 6. If blood glucose level does not return to normal after second dose of carbohydrate, activate EMS. 7. Once blood glucose level normalizes, provide a snack (eg, sandwich, bagel).	1. Activate EMS. 2. Prepare glucagon for injection, following directions in glucagon kit. 3. Once athlete is conscious and able to swallow, provide food.

Abbreviation: EMS, emergency medical services. Revised with permission from Jimenez CC, Corcoran MH, Crawley JT, et al. National Athletic Trainers' Association position statement: management of the athlete with type I diabetes mellitus. *J Athl Train.* 2007;42(4):536–545.

glucose level and exercise intensity. Finally, some athletes may use insulin adjustments to prevent hypoglycemia. These adjustments vary depending on the method of insulin delivery (insulin pump versus multiple daily injections), prevailing blood glucose level, and exercise intensity.[67,68,73,74]

Athletes with type 1 diabetes may also experience hyperglycemia, with or without ketosis, during exercise. Hyperglycemia during exercise is related to several factors, including exercise intensity[75,76] and the psychological stress of competition.[77] When the insulin level is adequate, these episodes of hyperglycemia are transient. However, when the insulin level is insufficient, ketosis can occur. Exercise is contraindicated when ketones are present in the urine. Athletic trainers should know the ADA guidelines for athletes exercising during an episode of hyperglycemia.[67] In addition, the athlete's physician should determine the need for insulin adjustments during hyperglycemic periods.

Recognition. Signs and symptoms of hypoglycemia typically occur when blood glucose levels fall below 70 mg/dL (3.9 mmol/L). Early symptoms include tachycardia, sweating, palpitations, hunger, nervousness, headache, trembling, and dizziness. These symptoms are related to the release of epinephrine and acetylcholine. As the glucose level continues to fall, symptoms of brain neuronal glucose deprivation occur, including blurred vision, fatigue, difficulty thinking, loss of motor control, aggressive behavior, seizures, convulsions, and loss of consciousness. If hypoglycemia is prolonged, severe brain damage and even death can occur. Athletic trainers should be aware that the signs and symptoms of hypoglycemia are individualized and be prepared to act accordingly.[78–80]

Although the signs and symptoms of hypoglycemia may vary from one athlete to another, they include nausea, dehydration, reduced cognitive performance, slowing of visual reaction time, and feelings of sluggishness and fatigue. The signs and symptoms of hyperglycemia with ketoacidosis may include those listed earlier as well as Kussmaul breathing, fruity odor to the breath, sleepiness, inattentiveness, loss of appetite, increased thirst, and frequent urination. With severe ketoacidosis, the level of consciousness may be reduced. Athletic trainers should also be aware that some athletes with type 1 diabetes intentionally train and compete in a hyperglycemic state (above 180 mg/dL [10 mmol/L]) to avoid hypoglycemia. Competing in a hyperglycemic state places the athlete at risk for dehydration, reduced athletic performance, and possibly ketosis.[67,81]

Treatment and Management. Treatment guidelines for mild and severe cases of hypoglycemia are shown in Table 2.[82,83] The ADA provides guidelines for exercise during hyperglycemic periods. If the fasting blood glucose level is ≥250 mg/dL (≥13.9 mmol/L), the athlete should test his or her urine for the presence of ketones. If ketones are present, exercise is contraindicated. If the blood glucose value is ≥300 mg/dL (≥16.7 mmol/L) and without ketones, the athlete may exercise with caution and continue to monitor blood glucose levels. Athletes should work with their physicians to determine the need for insulin adjustments for periods of hyperglycemia before, during, and after exercise.[67]

Return to Play. The literature does not address specific RTP guidelines after hypoglycemic or hyperglycemic events. Therefore, RTP for an athlete varies with the individual and becomes easier as the AT works with the athlete on a regular basis and learns how his or her blood glucose reacts to exercise and insulin and glucose doses. The athlete should demonstrate a stable blood glucose level that is within the normal range before RTP. Athletic trainers working with new athletes should seek guidance from the athlete, athlete's physician, and athlete's parents to gain insight on how the athlete has been able to best control the blood glucose level during exercise.

EXERTIONAL HEAT STROKE

Recommendations

Prevention

1. In conjunction with preseason screening, athletes should be questioned about risk factors for heat illness or a history of heat illness. *Evidence Category: C*
2. Special considerations and modifications are needed for those wearing protective equipment during periods of high environmental stress. *Evidence Category: B*
3. Athletes should be acclimatized to the heat gradually over a period of 7 to 14 days. *Evidence Category: B*
4. Athletes should maintain a consistent level of euhydration and replace fluids lost through sweat during games and practices. Athletes should have free access to readily available fluids at all times, not only during designated breaks. *Evidence Category: B*
5. The sports medicine staff must educate relevant personnel (eg, coaches, administrators, security guards, EMS staff, athletes) about preventing exertional heat stroke (EHS) and the policies and procedures that are to be followed in the event of an incident. Signs and symptoms of

a medical emergency should also be reviewed. *Evidence Category: C*

Recognition

6. The 2 main criteria for diagnosis of EHS are (1) core body temperature of greater than 104° to 105°F (40.0° to 40.5°C) taken via a rectal thermometer soon after collapse and (2) CNS dysfunction (including disorientation, confusion, dizziness, vomiting, diarrhea, loss of balance, staggering, irritability, irrational or unusual behavior, apathy, aggressiveness, hysteria, delirium, collapse, loss of consciousness, and coma). *Evidence Category: B*
7. Rectal temperature and gastrointestinal temperature (if available) are the only methods proven valid for accurate temperature measurement in a patient with EHS. Inferior temperature assessment devices should not be relied on in the absence of a valid device. *Evidence Category: B*

Treatment

8. Core body temperature must be reduced to less than 102°F (38.9°C) as soon as possible to limit morbidity and mortality. Cold-water immersion is the fastest cooling modality. If that is not available, cold-water dousing or wet ice towel rotation may be used to assist with cooling, but these methods have not been shown to be as effective as cold- water immersion. Athletes should be cooled first and then transported to a hospital unless cooling and proper medical care are unavailable onsite. *Evidence Category: B*
9. Current suggestions include a period of no activity, an asymptomatic state, and normal blood enzyme levels before the athlete begins a gradual return-to-activity progression under direct medical supervision. This progression should start at low intensity in a cool environment and slowly advance to high-intensity exercise in a warm environment. *Evidence Category: C*

Background and Literature Review

Definition, Epidemiology, and Pathophysiology. Exertional heat stroke is classified as a core body temperature of greater than 104° to 105°F (40.0° to 40.5°C) with associated CNS dysfunction.[84–87] The CNS dysfunction may present as disorientation, confusion, dizziness, vomiting, diarrhea, loss of balance, staggering, irritability, irrational or unusual behavior, apathy, aggressiveness, hysteria, delirium, collapse, loss of consciousness, and coma. Other signs and symptoms that may be present are dehydration, hot and wet skin, hypotension, and hyperventilation. Most athletes with EHS will have hot, sweaty skin as opposed to the dry skin that is a manifestation of classical EHS.[84,85,88,89]

Although it is usually among the top 3 causes of death in athletes, EHS may rise to the primary cause during the summer.[89] The causes of EHS are multifactorial, but the ultimate result is an overwhelming of the thermoregulatory system, which causes a buildup of heat within the body.[84,90–92]

Prevention. Exercise intensity can increase core body temperature faster and higher than any other factor.[85] Poor physical condition is also related to intensity. Athletes who are less fit than their teammates must work at a higher intensity to produce the same outcome. Therefore, it is important to alter exercise intensity and rest breaks when environmental conditions are dangerous.[93]

As air temperature increases, thermal strain increases, but if relative humidity increases as well, the body loses its ability to use evaporation as a cooling method (the main method used during exercise in the heat).[87,94–97] Adding heavy or extensive protective equipment also increases the potential risk, not only because of the extra weight but also as a barrier to evaporation and cooling. Therefore, extreme or new environmental conditions should be approached with caution and practices altered and events canceled as appropriate.

Acclimatization is a physiologic response to repeated heat exposure during exercise over the course of 10 to 14 days.[90,98] This response enables the body to cope better with thermal stressors and includes increases in stroke volume, sweat output, sweat rate, and evaporation of sweat and decreases in heart rate, core body temperature, skin temperature, and sweat salt losses.[90] Athletes should be allowed to acclimatize to the heat before stressful conditions such as full equipment, multiple practices within a day, or performance trials are implemented.[91,93]

Hydration can help reduce heart rate, fatigue, and core body temperature while improving performance and cognitive functioning.[96–98] Dehydration of as little as 2% of body weight has a negative effect on performance and thermoregulation.[87] Caution should be taken to ensure that athletes arrive at practice euhydrated (eg, having reestablished their weight since the last practice) and maintain or replace fluids that are lost during practice.

Assessment. The 2 main diagnostic criteria for EHS are CNS dysfunction and a core body temperature of greater than 104° to 105°F (40.0° to 40.5°C).[99–101] The only accurate measurements of core body temperature are via rectal thermometry or ingestible thermistors.[102] Other devices, such as oral, axillary, aural canal, and temporal artery thermometers, are inaccurate methods of assessing body temperature in an exercising person. A delay in accurate temperature assessment must also be considered during diagnosis and may explain body temperatures that are lower than expected. Lastly, in some cases of EHS, the patient has a lucid interval during which he or she is cognitively normal, followed by rapidly deteriorating symptoms.[86]

Due to policy and legal concerns in some settings, obtaining rectal temperature may not be feasible. Because immediate treatment is critical in EHS, it is important to not waste time by substituting an invalid method of temperature assessment. Instead, the practitioner should rely on other key diagnostic indicators (eg, CNS dysfunction, circumstances of the collapse). If EHS is suspected, cold-water immersion should be initiated at once. The evidence strongly indicates that in patients with suspected EHS, prompt determination of rectal temperature followed by aggressive, whole-body cold-water immersion maximizes the chances for survival. Practitioners in settings in which taking rectal temperature is a concern should consult with their administrators in advance. Athletic trainers, in conjunction with their supervising physicians, should clearly communicate to their administrators the dangers of skipping this important step and should obtain a definitive ruling on how to proceed in this situation.

Treatment. The goal for any EHS victim is to lower the body temperature to 102°F (38.9°C) or less within 30 minutes of collapse. The length of time body temperature is above the critical core temperature (~105°F [40.5°C]) dictates any morbidity and the risk of death from EHS.[103] Cold-water immersion is the most effective cooling modality for patients with EHS.[104,105] The water should be approximately 35°F (1.7°C) to

59°F (15.0°C) and continuously stirred to maximize cooling. The athlete should be removed when core body temperature reaches 102°F (38.9°) to prevent overcooling. If appropriate medical care is available, cooling should be completed before the athlete is transported to a hospital. If cold-water immersion is not available, other modalities, such as wet ice towels rotated and placed over the entire body or cold-water dousing with or without fanning, may be used but are not as effective. Policies and procedures for cooling athletes before transport to the hospital must be explicitly clear and shared with potential EMS responders, so that treatment by all medical professionals involved with a patient with EHS is coordinated.

Return to Play. Structured guidelines for RTP after EHS are lacking. The main considerations are treating any associated sequelae and, if possible, identifying the cause of the EHS, so that future episodes can be prevented. Many patients with EHS are cooled effectively and sent home the same day; they may be able to resume modified activity within 1 to 3 weeks. However, when treatment is delayed, patients may experience residual complications for months or years after the event. Most guidelines suggest that the athlete be asymptomatic with normal blood work (renal and hepatic panels, electrolytes, and muscle enzyme levels) before a gradual return to activity is initiated.[106] Unfortunately, no evidence-based tools are available to determine whether the body's thermoregulatory system is fully recovered. In summary, in all cases of EHS, after the athlete has completed a 7-day rest period and obtained normal blood work and physician clearance, he or she may begin a progression of physical activity, supervised by the AT, from low intensity to high intensity and increasing duration in a temperate environment, followed by the same progression in a warm to hot environment. The ability to progress depends largely on the treatment provided, and in some rare cases full recovery may not be possible. If the athlete experiences any side effects or negative symptoms with training, the progression should be slowed or delayed.

EXERTIONAL HYPONATREMIA

Recommendations

Prevention

1. Each physically active person should establish an individualized hydration protocol based on personal sweat rate, sport dynamics (eg, rest breaks, fluid access), environmental factors, acclimatization state, exercise duration, exercise intensity, and individual preferences. *Evidence Category: B*
2. Athletes should consume adequate dietary sodium at meals when physical activity occurs in hot environments. *Evidence Category: B*
3. Postexercise rehydration should aim to correct fluid loss accumulated during activity. *Evidence Category: B*
4. Body weight changes, urine color, and thirst offer cues to the need for rehydration. *Evidence Category: A*
5. Most cases of exertional hyponatremia (EH) occur in endurance athletes who ingest an excessive amount of hypotonic fluid. Athletes should be educated about proper fluid and sodium replacement during exercise. *Evidence Category: C*

Recognition

6. Athletic trainers should recognize EH signs and symptoms during or after exercise, including overdrinking, nausea, vomiting, dizziness, muscular twitching, peripheral tingling or swelling, headache, disorientation, altered mental status, physical exhaustion, pulmonary edema, seizures, and cerebral edema. *Evidence Category: B*
7. In severe cases, EH encephalopathy can occur and the athlete may present with confusion, altered CNS function, seizures, and a decreased level of consciousness. *Evidence Category: B*
8. The AT should include EH in differential diagnoses until confirmed otherwise. *Evidence Category: C*

Treatment and Management

9. If an athlete's mental status deteriorates or if he or she initially presents with severe symptoms of EH, IV hypertonic saline (3% to 5%) is indicated. *Evidence Category: B*
10. Athletes with mild symptoms, normal total body water volume, and a mildly altered blood sodium level (130 to 135 mEq/L; normal is 135 to 145 mEq/L) should restrict fluids and consume salty foods or a small volume of oral hypertonic solution (eg, 3 to 5 bouillon cubes dissolved in 240 mL of hot water). *Evidence Category: C*
11. The athlete with severe EH should be transported to an advanced medical facility during or after treatment. *Evidence Category: B*
12. Return to activity should be guided by a plan to avoid future EH episodes, specifically an individualized hydration plan, as described earlier. *Evidence Category: C*

Background and Literature Review

Definition, Epidemiology, and Pathophysiology. Exertional hyponatremia is a rare condition defined as a serum sodium concentration less than 130 mEq/L.[107] Although no incidence data are available from organized athletics, the condition is seen in fewer than 1% of military athletes[108] and up to 30% of distance athletes.[107,109] Signs and symptoms of EH include overdrinking, nausea, vomiting, dizziness, muscular twitching, peripheral tingling or swelling, headache, disorientation, altered mental status, physical exhaustion, pulmonary edema, seizures, and cerebral edema. If not treated properly and promptly, EH is potentially fatal because of the encephalopathy. Low serum sodium levels are identified more often in females than in males and during activity that exceeds 4 hours in duration.[107,110] Two common, often additive scenarios occur when an athlete ingests hypotonic beverages well beyond sweat losses (ie, water intoxication) or an athlete's sweat sodium losses are not adequately replaced.[111–114] Water intoxication causes low serum sodium levels because of a combination of excessive fluid intake and inappropriate body water retention. Insufficient sodium replacement causes low serum sodium levels when high sweat sodium content leads to decreased serum sodium levels (which may occur over 3 to 5 days). In both scenarios, EH causes intracellular swelling due to hypotonic intravascular and extracellular fluids. This, in turn, leads to potentially fatal neurologic and

physiologic dysfunction. When physically active people match fluid and sodium losses, via sweat and urine, with overall intake, EH is prevented.[94,115] Successful treatment of EH involves rapid sodium replacement in sufficient concentrations via foods containing high levels of sodium (minor cases) or hypertonic saline IV infusion (for moderate or severe cases).

Prevention. Exertional hyponatremia is most effectively prevented when individualized hydration protocols are used for the physically active, including hydration before, during, and after exercise.[94,115] This strategy should take into account sweat rate, sport dynamics (eg, rest breaks, fluid access), environmental factors, acclimatization state, exercise duration, exercise intensity, and individual preferences. The strategy should guide hydration before, during, and after activity to approximate sweat losses but ensure that fluids are not consumed in excess. This goal can be achieved by calculating individual sweat rates (sweat rate = pre-exercise body weight − postexercise body weight + fluid intake + urine volume/exercise time, in hours) for a representative range of environmental conditions and exercise intensities. Suggestions for expediting this procedure can be found in the NATA position statement on fluid replacement.[94] Sweat rate calculation is the most fundamental consideration when establishing a rehydration protocol. Average sweat rates from the scientific literature or other athletes vary from 0.5 L/h to more than 2.5 L/h.[115]

Dietary sodium is important for normal body maintenance of fluid balance and can help prevent muscle cramping, heat exhaustion, and EH.[91] The AT should encourage adequate dietary sodium intake, especially when athletes are training in a hot environment and as a part of daily meals.[116] Sport drinks generally contain low levels of sodium relative to blood and do little to attenuate decreases in whole-body sodium levels. Instead, athletes should consume foods that are high in sodium (eg, canned soups, pretzels) during meals before and after exercise. Including sodium in fluid-replacement beverages should be considered under the following conditions: inadequate access to meals, physical activity exceeding 2 hours in duration, and during the initial days to weeks of hot weather.[94,115] Under these conditions, adding salt in amounts of 0.3 to 0.7 g/L can offset salt losses in sweat and minimize medical events associated with electrolyte imbalances.

Postexercise hydration should aim to correct the fluid loss accumulated during activity.[94,115] Ideally completed within 2 hours, rehydration fluids should contain water, carbohydrates to replenish glycogen stores, and electrolytes to speed rehydration. When rehydration must be rapid (within 2 hours), the athlete should compensate for obligatory urine losses incurred during the rehydration process and drink about 25% more than sweat losses to ensure optimal hydration 4 to 6 hours after the event.[117] However, athletes should not drink enough to gain weight beyond pre-exercise measurements.[94,115,116]

Body weight changes, urine color, and thirst offer cues to the need for rehydration.[118] When preparing for an event, an athlete should know his or her sweat rate and pre-exercise hydration status and develop a rehydration plan (discussed in detail in the recommendations).[94,115] If the athlete's specific needs are unknown, the athlete should not drink beyond thirst.

Recognition. The AT should recognize and the physically active should be educated on EH signs and symptoms during exercise.[113,114,116] After an exercise bout or competition, symptoms of EH may appear immediately or gradually progress over several hours. The most efficient method of diagnosing EH onsite is the use of a handheld analyzer, which can identify the serum sodium concentration within minutes.[113,114] Athletic trainers should work with physicians and EMS to maximize access to these analyzers when EH is likely.

A collapsed, semiconscious, or unconscious athlete should be evaluated for all potential causes of sudden death in sport. The key to the differential diagnosis of EH is serum sodium assessment, which should be conducted when EH is suspected.[113,114] If a portable serum sodium analyzer is not available, it is then necessary to rule out other conditions that may warrant onsite treatment (eg, EHS) before emergency transport.[91]

Treatment. If the athlete's mental status deteriorates or if he or she initially presents with severe symptoms, IV hypertonic saline (3% to 5%) is indicated.[91,113,114] Intravenous hypertonic saline rapidly corrects the symptoms of EH and decreases intracellular fluid volume. Serial measures of blood sodium should be obtained throughout treatment (after every 100 mL of IV fluid). To avoid complications, hypertonic saline administration should be discontinued when the serum sodium concentration reaches 128 to 130 mEq/L.[114] Normal saline (0.9% NaCl) IV fluids should not be provided to patients without prior serum sodium assessment.[113,114] Ideally, the ATs have discussed with EMS in the off-season the importance of having a portable sodium analyzer available and being ready to administer hypertonic saline during transport.

Athletes with mild symptoms, normal total body water volume, and a mildly altered blood sodium concentration (130 to 135 mEq/L) should restrict fluids and consume salty foods or a small volume of oral hypertonic solution (eg, 3 to 5 bouillon cubes dissolved in 240 mL of hot water). This can be continued until diuresis and correction of the blood sodium concentration occur; such management may take hours to complete, but it is successful in stable patients.[114]

The patient with severe EH should be transported to an advanced medical facility during or after treatment. Once the patient arrives at the emergency department, a plasma osmolality assessment is performed to identify hypovolemia or hypervolemia. Patients with persistent hypovolemia despite normal serum sodium values should receive 0.9% NaCl IV until euvolemia is reached. The progress of symptoms and blood sodium levels determines the follow-up care.[119]

Return to Play. When EH is treated appropriately with IV hypertonic saline, chronic morbidity is rare. Literature documenting the expected time course of recovery after EH is lacking, but recovery seems to depend on the severity and duration of brain swelling. Rapid recognition and prompt treatment reduce the risk of CNS damage.[120]

Return to activity should be guided by a plan to avoid future EH episodes, specifically an individualized hydration plan (documented earlier).[94,115] This plan should also be based on the history and factors that contributed to the initial EH episode.

EXERTIONAL SICKLING

Recommendations

Prevention

1. The AT should educate coaches, athletes, and, as warranted, parents about complications of exertion in the athlete with sickle cell trait (SCT). *Evidence Category: C*

2. Targeted education and tailored precautions may provide a margin of safety for the athlete with SCT. *Evidence Category: C*
3. Athletes with known SCT should be allowed longer periods of rest and recovery between conditioning repetitions, be excluded from participation in performance tests such as mile runs and serial sprints, adjust work-rest cycles in the presence of environmental heat stress, emphasize hydration, control asthma (if present), not work out if feeling ill, and have supplemental oxygen available for training or competition when new to a high-altitude environment. *Evidence Category: B*

Recognition

4. Screening for SCT, by self-report, is a standard component of the preparticipation physical evaluation (PPE) monograph. Testing for SCT, when included in the PPE or conducted previously, confirms SCT status. *Evidence Category: A*
5. The AT should know the signs and symptoms of exertional sickling, which include muscle cramping, pain, swelling, weakness, and tenderness; inability to catch one's breath; and fatigue, and be able to differentiate exertional sickling from other causes of collapse. *Evidence Category: C*
6. The AT should understand the usual settings for and patterns of exertional sickling. *Evidence Category: C*

Treatment

7. Signs and symptoms of exertional sickling warrant immediate withdrawal from activity. *Evidence Category: C*
8. High-flow oxygen at 15 L/min with a nonrebreather face mask should be administered. *Evidence Category: C*
9. The AT should monitor vital signs and activate the EAP if vital signs decline. *Evidence Category: C*
10. Sickling collapse should be treated as a medical emergency. *Evidence Category: C*
11. The AT has a duty to make sure the athlete's treating physicians are aware of the presence of SCT and prepared to treat the metabolic complications of explosive rhabdomyolysis. *Evidence Category: B*

Background and Literature Review

Prevention. No contraindications to participation in sport exist for the athlete with SCT.[121–123] Recognition of the athlete's positive SCT status must be followed with targeted education and tailored precautions because deaths have been tied to lapses in education and inadequate precautions.[124] The athlete with SCT should be informed that SCT is consistent with a normal, healthy life span, although associated complications may occur. Education should include genetic considerations with respect to family planning and questioning about any past medical history of sickling events. Athletes and staff should be educated about the signs, symptoms, and settings of exertional sickling and precautions for the athlete with SCT.[123]

The premise behind the suggested precautions is that exertional sickling can be brought about through intense, sustained activity with modifiers that increase the intensity.[125] One precaution that can mitigate exertional sickling is a slow, paced training progression that allows longer periods of rest and recovery between repetitions.[123,125] Strength and conditioning programs may increase preparedness but must be sport specific. Athletes with SCT should be excluded from participation in performance tests, such as mile runs and serial sprints, because several deaths have occurred in this setting.[124] Cessation of activity with the onset of symptoms is essential to avoid escalating a sickling episode (eg, muscle cramping, pain, swelling, weakness, and tenderness; inability to catch one's breath; fatigue).[123,125] In general, when athletes with SCT set their own pace, they seem to do well.[123,125] Therefore, athletes with SCT who perform repetitive high-speed sprints, distance runs, or interval training that induces high levels of lactic acid as a component of a sport-specific training regimen should be allowed extended recovery between repetitions because this type of conditioning poses special risks to them.[123,125]

Factors such as ambient heat stress, dehydration, asthma, illness, and altitude predispose the athlete with SCT to a crisis during physical exertion, even when exercise is not all-out.[123,125] Extra precautions are warranted in these conditions. These precautions may include the following:

- Work-rest cycles should be adjusted for environmental heat stress.
- Hydration should be emphasized.
- Asthma should be controlled.
- The athlete with SCT who is ill should not work out.
- The athlete with SCT who is new to a high-altitude environment should be watched closely. Training should be modified and supplemental oxygen should be available for competitions.

One last precaution is to create an environment that encourages athletes with SCT to immediately report any signs or symptoms such as leg or low back cramping, difficulty breathing, or fatigue. Such signs and symptoms in an athlete with SCT should be assumed to represent sickling.[123]

Recognition. The PPE monograph[14] recommends screening for SCT with the question, "Do you or [does] someone in your family have SCT or disease?" Small numbers of affected athletes limit the collection of sufficient evidence to support testing for SCT in the PPE. However, because PPE medical history form answers are highly suspect[126] and deaths can be tied to a lack of awareness about SCT, the argument for testing to confirm trait status remains strong. The National Collegiate Athletic Association currently mandates testing for SCT. Irrespective of testing, the AT should educate staff, coaches, and athletes on the potentially lethal nature of this condition.[123] Education and precautions work best when targeted at the athletes most at risk. Incidence rates of SCT are approximately 8% in African Americans, 0.5% in Hispanics, and 0.2% in whites (but more common in those from the Mediterranean, the Middle East, and India).[127]

Not all athletes who experience sickling present the same way. The primary limiting symptoms are leg or low back cramps or spasms, weakness, debilitating low back pain,[128] difficulty recovering ("I can't catch my breath"), and fatigue. Sickling often lacks a prodrome, so these symptoms in an athlete with SCT should be treated as exertional sickling.[123]

Sickling collapse has been mistaken for cardiac collapse or heat illness.[129] However, unlike sickling collapse, cardiac collapse tends to be instantaneous, is not associated with cramping, and results in the athlete hitting the ground without any protective reflex mechanism and being unable to talk. Also unlike sickling collapse, heat illness collapse often occurs after a

moderate but still intense bout of exercise, usually more than 30 minutes in duration. In addition, the athlete will have a core body temperature >104°F (40.0°C). Alternatively, sickling collapse typically occurs within the first half hour on the field, and core temperature is not greatly elevated.[129,130]

Sickling is often confused with heat cramping but may be differentiated by the following:

- Heat cramping often has a prodrome of muscle twinges; sickling has none.
- Heat-cramping pain is more excruciating and can be pinpointed, whereas sickling cramping is more generalized but still strong.
- Those with heat cramps hobble to a halt with "locked-up" muscles, whereas sickling athletes slump to the ground with weak muscles. Many times, sickling athletes push through several instances of collapse before being unable to continue.
- Those with heat cramps writhe and yell in pain; their muscles are visibly contracted and rock hard. Those who are sickling lie fairly still, not yelling in pain, with muscles that look and feel normal to the observer.

Certain factors are common in severe or fatal exertional sickling collapses. These cases tend to be similar in setting and syndrome and are characterized by the following:

- Sickling athletes may be on the field only briefly before collapsing, sprinting only 800 to 1600 meters, often early in the season.
- Sickling can occur during repetitive running of hills or stadium steps, during intense, sustained strength training; if the tempo increases toward the end of intense 1-hour drills; and at the end of practice when athletes run "gassers." Sickling occurs rarely in competition, most often in athletes previously exhibiting symptoms in training for sport.[123]

Severe to fatal sickling cases are not limited to football players. Sickling collapse has occurred in distance racers and has killed or nearly killed several collegiate and high school basketball players (including 2 women) in training, typically during "suicide sprints" on the court, laps on a track, or a long training run.[123]

The harder and faster athletes with SCT work, the earlier and greater the sickling. Sickling can begin after only 2 to 3 minutes of sprinting—or any all-out exertion—and can quickly increase to grave levels if the athlete struggles on or is urged on by the coach.[124]

Athletes react in different ways. Some stoic athletes simply stop and say, "I can't go on." When the athlete rests, sickle red cells regain oxygen in the lungs; most sickle cells then revert to normal shape, and the athlete soon feels good again and ready to continue. This self-limiting feature surely saves lives.

Treatment. Complaints or evidence of exertional sickling signs and symptoms in a working athlete with SCT should be assumed to represent the onset of sickling and first managed by cessation of activity. A sickling collapse is treated as a medical emergency. Immediate action can save lives[123]:

1. Check vital signs.
2. Administer high-flow oxygen, 15 L/min (if available), with a nonrebreather face mask.
3. Cool the athlete if necessary.
4. If the athlete is obtunded or if vital signs decline, call 911, attach an AED, and quickly transport the athlete to the hospital.[125,129] Appropriate medical personnel should start an IV.
5. The AT should inform treating physicians of the athlete's trait status so that they are prepared to treat explosive rhabdomyolysis and associated metabolic complications.[124,125,129,131,132]
6. Proactively prepare by having an EAP and appropriate emergency equipment available.

Return to Play. After nonfatal sickling, the athlete may return to sport the same day or be disqualified from further participation. Athletes whose conditions are identified quickly and managed appropriately may return the same day as symptoms subside. Others have self-limiting myalgia from myonecrosis in moderate rhabdomyolysis and may need 1 to 2 weeks of recovery with serial assessments.[122] Patients with severe rhabdomyolysis necessitating dialysis and months of hospitalization[133] may not RTP due to diminished renal function, muscle lost to myonecrosis, or neuropathy from compartment syndrome.[121] As with any RTP after a potential deadly incident, it is imperative that the physician, AT, coach, and athlete work in concert to ensure the athlete's safety and minimize risk factors that may have caused the initial incident.

HEAD-DOWN CONTACT IN FOOTBALL

Recommendations

Prevention

1. Axial loading is the primary mechanism for catastrophic cervical spine injury. *Head-down contact,* defined as initiating contact with the top or crown of the helmet, is the only technique that results in axial loading. *Evidence Category: A*
2. *Spearing* is the intentional use of a head-down contact technique. Unintentional head-down contact is the inadvertent dropping of the head just before contact. Both head-down techniques are dangerous and may result in axial loading of the cervical spine and catastrophic injury. *Evidence Category: A*
3. Football helmets and other standard football equipment do not cause or prevent axial-loading injuries of the cervical spine. *Evidence Category: A*
4. Injuries that occur as a result of head-down contact are technique related and are preventable to the extent that head-down contact is preventable. *Evidence Category: C*
5. Making contact with the shoulder or chest while keeping the head up greatly reduces the risk of serious head and neck injury. With the head up, the player can see when and how impact is about to occur and can prepare the neck musculature. Even if head-first contact is inadvertent, the force is absorbed by the neck musculature, the intervertebral discs, and the cervical facet joints. This is the safest contact technique. *Evidence Category: C*
6. The game can be played as aggressively with the head up and with shoulder contact but with much less risk of serious injury (Figure 2). However, the technique must be learned, and to be learned, it must be practiced extensively. Athletes who continue to drop their heads just before contact need additional coaching and practice time. *Evidence Category: C*
7. Initiating contact with the face mask is a rule violation and must not be taught. If the athlete uses poor technique by lowering his head, he places himself in the head-

Figure 2. Initiating contact with the shoulder while keeping the head up reduces the risk of head and neck injuries.

down position and at risk of serious injury. *Evidence Category: C*

8. The athlete should know, understand, and appreciate the risk of head-down contact, regardless of intent. Formal team education sessions (conducted by the AT, team physician, or both with the support of the coaching staff) should be held at least twice per season. One session should be conducted before contact begins and the other at the midpoint of the season. Recommended topics are mechanisms of head and neck injuries, related rules and penalties, the incidence of catastrophic injury, the severity of and prognosis for these injuries, and the safest contact positions. The use of videos such as *Heads Up: Reducing the Risk of Head and Neck Injuries in Football*[134] and *Tackle Progression*[135] should be mandatory. Parents of high school athletes should be given the opportunity to view these videos. *Evidence Category: C*

Recognition

9. Attempts to determine a player's intent regarding intentional or unintentional head-down contact are subjective. Therefore, coaching, officiating, and playing techniques must focus on decreasing all head-down contact, regardless of intent. *Evidence Category: C*
10. Officials should enforce existing helmet contact rules to further reduce the incidence of head-down contact. A clear discrepancy has existed between the incidence of head-down or head-first contact and the level of enforcement of the helmet contact penalties. Stricter officiating would bring more awareness to coaches and players about the effects of head-down contact. *Evidence Category: B*

Background and Literature Review

Definition and Pathophysiology. Sudden death from a cervical spine injury is most likely to occur in football from a fracture-dislocation above C4. Axial loading is accepted as the primary cause of cervical spine fractures and dislocations in football players.[136,137] Axial loading occurs secondary to head-down contact, whether intentional or unintentional, when the cervical vertebrae are aligned in a straight column. Essentially, the head is stopped at contact, the trunk keeps moving, and the spine is crushed between the two. When maximum vertical compression is reached, the cervical spine fails,[138] resulting in damage to the spinal cord.

Although the football helmet has been successful in reducing the number of catastrophic brain injuries, it is neither the cause nor the solution for cervical spine fractures, primarily because with head-first impact, the head, neck, and torso decelerate nonuniformly. Even after the head is stopped, the body continues to accelerate, and no current football helmet can effectively manage the force placed on the cervical spine by the trunk.[139–141] As identified in the 1970s, contact technique remains the critical factor in preventing axial loading.

Prevention. Initiating contact with the shoulder while keeping the head up is the safest contact position.[142–148] With the head up, the athlete can see when and how impact is about to occur and can prepare the neck musculature accordingly. This guideline applies to all position players, including ball carriers. The game can be played just as aggressively with this technique but with much less risk of serious head or neck injury. Tacklers can still deliver a big hit, and ball carriers can still break tackles.[149]

A top priority for prevention is player education. Athletes have to know, understand, and appreciate the risks of head-first contact in football.[150,151] The videos *Heads Up: Reducing the Risk of Head and Neck Injuries in Football*[134] and *Tackle Progression*[135] are excellent educational tools. Parents of high school players should also be given the opportunity to view these videos. Coaches have a responsibility to spend adequate time teaching and practicing correct contact techniques with all position players. The goal should be not merely to discourage head-down contact but to eliminate it from the game.[139]

Recognition. Coaches have stated that although they have taught players to tackle correctly, the players still tended to lower their heads just before contact.[143,144] It seems that players have learned to approach contact with their head up, but they need to maintain this position during contact.[146,149] Instinctively, players protect their eyes and face from injury by lowering their heads at impact.[144,146,149] Therefore, coaches must allocate enough practice time to overcome this instinct. Players who drop their heads at the last instant are demonstrating that they need additional practice time with correct contact techniques in game-like situations. In addition to teaching correct contact in the beginning of the season, coaches should reinforce the technique regularly throughout the season.[144]

The increase in catastrophic cervical spine injuries in the early 1970s was attributed to coaches teaching players to initiate contact with their face masks.[136,150] Players did not execute maneuvers as they were taught, often unintentionally, and they lowered their heads just before impact, resulting in increased exposure to axial loading and cervical spine fractures. The

teaching of face-first contact remains a rule violation at the high school level and is a concern at all levels of football.

Adequate enforcement of the helmet contact rules will further reduce the risk of catastrophic injuries.[142–144,152–154] Both the National Collegiate Athletic Association and the National Federation of State High School Associations have changed their helmet contact penalties multiple times in the past 5 years[155] to resolve the dilemma for officials trying to distinguish between intentional and unintentional helmet contact. The current rules for both organizations are now more complete and concise.

A discrepancy has existed between enforcement of the helmet contact penalties and the incidence of head-down contact. Contact with the top of the helmet has been observed in 40% of plays[146] and 18% of helmet collisions in 2007.[156] In contrast, NCAA Division I officials called 1 helmet contact penalty in every 75 games in 2007.[157] If illegal helmet contact is not penalized, the message is that the technique is acceptable.[158] Therefore, football officials must continue to improve the enforcement of these penalties.

LIGHTNING SAFETY

Recommendations

Prevention

1. The most effective means of preventing lightning injury is to reduce the risk of casualties by remaining indoors during lightning activity. When thunder is heard or lightning seen, people should vacate to a previously identified safe location.[159–161] *Evidence Category: A*
2. Establish an EAP or policy specific to lightning safety.[161,162] *Evidence Category: C*
3. No place outdoors is completely safe from lightning, so alternative safe structures must be identified. Sites that are called "shelters" typically have at least one open side and therefore do not provide sufficient protection from lightning injury. These sites include dugouts; picnic, golf, or rain shelters; tents; and storage sheds.[160,163,164] Safe places to be while lightning occurs are structures with 4 substantial walls, a solid roof, plumbing, and electric wiring—structures in which people live or work.[160,164] *Evidence Category: B*
4. Buses or cars that are fully enclosed and have windows that are completely rolled up and metal roofs can also be safe places during a lightning storm.[165] *Evidence Category: B*
5. People should remain entirely inside a safe building or vehicle until at least 30 minutes have passed since the last lightning strike or the last sound of thunder.[166,167] *Evidence Category: A*
6. People injured by lightning strikes while indoors were touching electric devices or using a landline telephone or plumbing (eg, showering). Garages with open doors and rooms with open windows do not protect from the effects of lightning strikes.[159,161,168–170] *Evidence Category: B*

Treatment and Management

7. Victims are safe to touch and treat, but first responders must ensure their own safety by being certain the area is safe from imminent lightning strikes.[171,172] *Evidence Category: A*
8. Triage first lightning victims who appear to be dead. Most deaths are due to cardiac arrest.[171,173,174] Although those who sustain a cardiac arrest may not survive due to subsequent apnea, aggressive CPR and defibrillation (if indicated) may resuscitate these patients. *Evidence Category: A*
9. Apply an AED and perform CPR as warranted.[174] *Evidence Category: A*
10. Treat for concussive injuries, fractures, dislocations, and shock.[14,164] *Evidence Category: A*

Background and Literature Review

Definition. Lightning is a natural phenomenon that most people observe within their lifetimes. One of the most dangerous natural hazards encountered, it causes more than 60 fatalities and hundreds of injuries annually in the United States.[169,175] Lightning occurs with greater frequency in the southeastern United States, the Mississippi and Ohio river valleys, the Rocky Mountains, and the Southwest,[175] but no location is truly safe from the hazard of lightning. Lightning is most prevalent from May through September, with most fatalities and trauma reported in July.[169,175,176] Most deaths and injuries are recorded between 10:00 AM and 7:00 PM, when many people are engaged in outdoor activities.[159,169,177]

Lightning can occur from cloud to cloud or cloud to ground. Injuries and deaths are often attributed to cloud-to-ground lightning, but compared with cloud-to-cloud lightning, it occurs only 30% of the time. Negatively charged ionized gas builds up in clouds and seeks objects on the earth (eg, people, houses, trees) that have positively charged regions. When the 2 channels meet, lightning is produced, and an audible repercussion is created; we know this as thunder.[170,178] The lightning channel has an average peak current of 20000 A and is 5 times hotter than the surface of the sun.[170,178]

Prevention. Prevention of lightning injury is simple: Avoid the risk of trauma by staying completely indoors in a substantial building where people live and work.[160,162] A proactive lightning-specific safety policy is paramount to preventing lightning-specific injury. The policy should identify a weather watcher whose job is to look for deteriorating conditions. The weather watcher must have the unchallengeable authority to clear a venue when conditions are unsafe.[161] In addition to on-site observations for deteriorating conditions, use of federal weather monitoring Web sites is encouraged. Safe buildings must be identified before outdoor activity begins.[161,162,179] The lightning safety plan must allow sufficient time to safely move people to the identified building, and this time frame should be adjusted according to the number of people being moved. For example, moving a soccer team to safety takes less time than moving a football team. It is also critical to remain wholly within the safe building for at least 30 minutes after the last sighting of lightning and sound of thunder.[166,168]

Treatment. People who have been struck by lightning are safe to touch and treat and do not carry an electric charge. However, rescuers themselves are vulnerable to a lightning strike while treating victims during active thunderstorms. Treatment of lightning strike patients includes establishing and maintaining normal cardiorespiratory status.[161,162,171,173,174] Patients may present in asystole, pulseless, and with fixed and dilated pupils.

Therefore, CPR should be continued even when defibrillation with an AED is not indicated (eg, asystole). Advanced cardiac life support, medications, intubation, and continued CPR may resuscitate these victims. People with a Glasgow Coma Scale score as low as 5 have survived after aggressive resuscitation.[180] After a lightning strike, many patients present with symptoms resembling a concussion. Some may have temporary paralysis, hearing loss, or skin markings, yet true burns are rare. Patients should be assessed and treated for concussion, fractures, dislocations, and shock.[174]

Return to Play. Lightning strike patients are eligible to return to previous activities upon release by a qualified physician. Many never seek treatment and do not need hospitalization. If orthopaedic injuries are present, recovery follows the typical protocols. More often than not, however, patients experience neurologic sequelae and have difficulty returning to their preinjury levels.[175,181] They may never fully return to desired levels, and they need consistent and perhaps multidisciplinary medical and psychological follow-up.[174,181]

SUDDEN CARDIAC ARREST

Recommendations

Prevention

1. Access to early defibrillation is essential. A goal of less than 3–5 minutes from the time of collapse to delivery of the first shock is strongly recommended. *Evidence Category: B*
2. The preparticipation physical examination should include the completion of a standardized history form and attention to episodes of exertional syncope or presyncope, chest pain, a personal or family history of sudden cardiac arrest or a family history of sudden death, and exercise intolerance. *Evidence Category: C*

Recognition

3. Sudden cardiac arrest (SCA) should be suspected in any athlete who has collapsed and is unresponsive. A patient's airway, breathing, circulation, and heart rhythm (using the AED) should be assessed. An AED should be applied as soon as possible for rhythm analysis. *Evidence Category: B*
4. Myoclonic jerking or seizure-like activity is often present after collapse from SCA and should not be mistaken for a seizure. Occasional or agonal gasping should not be mistaken for normal breathing. *Evidence Category: B*

Management

5. Cardiopulmonary resuscitation should be provided while the AED is being retrieved, and the AED should be applied as soon as possible. Interruptions in chest compressions should be minimized by stopping only for rhythm analysis and defibrillation. Treatment should proceed in accordance with the updated American Heart Association guidelines,[182] which recommend that health care professionals follow a sequence of chest compressions (C), airway (A), and breathing (B). *Evidence Category: B*

Background and Literature Review

Definition, Epidemiology, and Pathophysiology. Sudden cardiac death (SCD) is the leading cause of death in exercising young athletes.[183,184] The underlying cause of SCD is usually a structural cardiac abnormality. Hypertrophic cardiomyopathy and coronary artery anomalies are responsible for approximately 25% and 14% of SCD, respectively, in the United States.[183] Commotio cordis accounts for approximately 20% of SCD in young athletes; caused by a blunt, nonpenetrating blow to the chest, it induces ventricular arrhythmia in an otherwise normal heart.[183] Other structural anomalies that can cause SCD include myocarditis, arrhythmogenic right ventricular dysplasia, Marfan syndrome, valvular heart disease, dilated cardiomyopathy, and atherosclerotic coronary artery disease. In 2% of athletes with SCD, a postmortem examination fails to identify a structural abnormality. These deaths may result from inherited arrhythmia syndromes and ion channel disorders or familial catecholaminergic polymorphic ventricular tachycardia.[183]

The incidence of SCD in high school athletes is estimated to be 1:100000 to 1:200000.[184,185] In collegiate athletes, this incidence is slightly higher, with estimates ranging from 1:65000 to 1:69000.[184,186] A recent report[185] described the incidence of SCD in National Collegiate Athletic Association student–athletes as 1:43000, with higher rates in black athletes (1:1700) and male basketball players (1:7000). Unfortunately, because we have no mandatory national reporting system, the true incidence of SCD is unknown and probably underestimated. The reports demonstrating the greatest incidence have estimated up to 110 deaths each year in young athletes, equating to 1 death every 3 days in the United States.[187]

Prevention. Preparticipation screening is one strategy available to prevent SCD, but the best protocol to screen athletes is highly debated, and some methods lack accuracy. As many as 80% of patients with SCD are asymptomatic until sudden cardiac arrest occurs,[188,189] suggesting that screening by history and physical examination alone may have limited sensitivity to identify athletes with at-risk conditions. Further research is needed to understand whether additional tests such as electrocardiograms and echocardiograms improve sensitivity and can be performed with acceptable cost-effectiveness and an acceptable false-positive rate. Detection of asymptomatic conditions should be improved with standardized history forms and attention to episodes of exertional syncope or presyncope, chest pain, a personal or family history of sudden cardiac arrest or a family history of sudden death, or exercise intolerance; selective use of electrocardiograms in high-risk athletes; and a stronger knowledge base for health care professionals.

In 2007, the American Heart Association released a helpful 12-point preparticipation cardiovascular screen for competitive athletes based on the medical history and physical examination (Table 3).

Emergency Preparedness. Preparation is the key to survival once SCA has occurred. Public access to AEDs and established EAPs greatly improve the likelihood of survival. All necessary equipment should be placed in a central location that is highly visible and accessible; multiple AEDs may be needed for larger facilities. An EAP should be in place and specific to each athletic venue and should include an effective communication system, training of likely first responders in CPR and AED use, acquisition of the necessary emergency equipment, a coordinated and practiced response plan, and access to early

Table 3. The 12-Element AHA Recommendations for Preparticipation Cardiovascular Screening of Competitive Athletes

Medical history[a]

Personal history

1. Exertional chest pain/discomfort
2. Unexplained syncope/near-syncope[b]
3. Excessive exertional and unexplained dyspnea/fatigue, associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure

Family history

6. Premature death (sudden and unexpected, or otherwise) before age 50 years due to heart disease, in ≥1 relative
7. Disability from heart disease in a close relative <50 years of age
8. Specific knowledge of certain cardiac conditions in family members: hypertrophic or dilated cardiomyopathy, long-QT syndrome or other ion channelopathies, Marfan syndrome, or clinically important arrhythmias

Physical examination

9. Heart murmur[c]
10. Femoral pulses to exclude aortic coarctation
11. Physical stigmata of Marfan syndrome
12. Brachial artery blood pressure (sitting position)[d]

[a] Parental verification is recommended for high school and middle school athletes.

[b] Judged not to be neurocardiogenic (vasovagal); of particular concern when related to exertion.

[c] Auscultation should be performed in both supine and standing positions (or with Valsalva maneuver), specifically to identify murmurs of dynamic left ventricular outflow tract obstruction.

[d] Preferably taken in both arms (Kaplan NM, Gidding SS, Pickering TG, Wright JT Jr. Task Force 5: systemic hypertension. *J Am Coll Cardiol*. 2005;45(8):1346–1348).

Reprinted with permission from Maron BJ, Thompson PD, Ackerman MJ, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update. *Circulation*. 2007;115(12):1643–1655.

defibrillation. It should identify the person or group responsible for documentation of personnel training, equipment maintenance, actions taken during the emergency, and evaluation of the emergency response.[192] The EAP should be coordinated with the local EMS agency and integrated into the local EMS system. It should also be posted at every venue and near appropriate telephones and include the address of the venue and specific directions to guide EMS personnel.

Assessment. Differential diagnosis of nontraumatic exercise-related syncope or presyncope includes sudden cardiac arrest, EHS, heat exhaustion, hyponatremia, hypoglycemia, exercise-associated collapse, exertional sickling, neurocardiogenic syncope, seizures, pulmonary embolus, cardiac arrhythmias, valvular disorders, coronary artery disease, cardiomyopathies, ion channel disorders, and other structural cardiac diseases. In any athlete who has collapsed in the absence of trauma, suspicion for sudden cardiac arrest should be high until normal airway, breathing, and circulation are confirmed. Agonal respiration or occasional gasping should not be mistaken for normal breathing and should be recognized as a sign of SCA[193]; myoclonic jerking or seizure-like activity shortly after collapse should also be treated as SCA until proven otherwise.[194,195] If no pulse is palpable, the patient should be treated for SCA, and CPR should be initiated.

Treatment. In any athlete who has collapsed and is unresponsive, SCA should be suspected. If normal breathing and pulse are absent, CPR should be started immediately and EMS activated. The CPR should be performed in the order of CAB (chest compressions, airway, breathing) by medical professionals (hands-only CPR is now recommended for lay responders) while waiting for arrival of the AED and stopped only for rhythm analysis and defibrillation. This should continue until either advanced life support providers take over or the victim starts to move.[193,194,196,197] Early detection, prompt CPR, rapid activation of EMS, and early defibrillation are vital to the athlete's survival. For any athlete who has collapsed and is unresponsive, an AED should be applied as soon as possible for rhythm analysis and defibrillation if indicated. The greatest factor affecting survival after SCA arrest is the time from arrest to defibrillation.[195,196] Survival rates have been reported at 41%–74% if bystander CPR is provided and defibrillation occurs within 3 to 5 minutes of collapse.[186,194,196–207]

Certain weather situations warrant special consideration. In a rainy or icy environment, AEDs are safe and do not pose a shock hazard. However, a patient lying on a wet surface or in a puddle should be moved. A patient lying on a metal conducting surface (eg, stadium bleacher) should be moved to a nonmetal surface. If lightning is ongoing, rescuers must ensure their safety by moving the patient indoors if possible.

ACKNOWLEDGMENTS

We gratefully acknowledge the efforts of Gianluca Del Rossi, PhD, ATC; Jonathan Drezner, MD; John MacKnight, MD; Jason Mihalik, PhD, ATC; Francis O'Connor, MD, MPH; and the Pronouncements Committee in the preparation of this document.

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Appendix. National Athletic Trainers' Association Statements[a]

Topic (Year)	Citation	URL
Safe weight loss and maintenance practices in sport and exercise (2011)	Sammarone Turocy P, DePalma BF, Horswill CA, et al. National Athletic Trainers' Association position statement: safe weight loss and maintenance practices in sport and exercise. *J Athl Train*. 2011;46(3):322–336.	http://www.nata.org/sites/default/files/JAT-46-3-16-turocy-322-336.pdf
Prevention of pediatric overuse injuries (2011)	Valovich McLeod TC, Decoster LC, Loud KJ, et al. National Athletic Trainers' Association position statement: prevention of pediatric overuse injuries. *J Athl Train*. 2011;46(2):206–220.	http://www.nata.org/sites/default/files/Pediatric-Overuse-Injuries.pdf
Skin diseases (2010)	Zinder SM, Basler RSW, Foley J, Scarlata C, Vasily DB. National Athletic Trainers' Association position statement: skin diseases. *J Athl Train*. 2010;45(4):411–428.	http://www.nata.org/sites/default/files/position-statement-skin-disease.pdf
Acute management of the cervical spine–injured athlete (2009)	Swartz EE, Boden BP, Courson RW, et al. National Athletic Trainers' Association position statement: acute management of the cervical spine–injured athlete. *J Athl Train*. 2009;44(3):306–331.	http://www.nata.org/sites/default/files/acutemgmtofcervicalspineinjuredathlete.pdf
Preventing, detecting, and managing disordered eating in athletes (2008)	Bonci CM, Bonci LJ, Granger LR, et al. National Athletic Trainers' Association position statement: preventing, detecting, and managing disordered eating in athletes. *J Athl Train*. 2008;43(1):80–108.	http://www.nata.org/sites/default/files/PreventingDetectingAndManagingDisorderedEating.pdf
Environmental cold injuries (2008)	Cappaert TA, Stone JA, Castellani JW, Krause BA, Smith D, Stephens BA. National Athletic Trainers' Association position statement: environmental cold injuries. *J Athl Train*. 2008;43(6):640–658.	http://www.nata.org/sites/default/files/EnvironmentalColdInjuries.pdf
Sickle cell trait and the athlete (2007)	Anderson S, Eichner ER. Consensus statement: sickle cell trait and the athlete.[b]	http://www.nata.org/sites/default/files?SickleCellTraitAndTheAthlete.pdf
Emergency preparedness and management of sudden cardiac arrest in high school and college athletic programs (2007)	Drezner JA, Courson RW, Roberts WO, Mosesso VN, Link MS, Maron BJ. Inter-Association Task Force Recommendations on emergency preparedness and management of sudden cardiac arrest in high school and college athletic programs: a consensus statement. *J Athl Train.* 2007;42(1):143–158.	http://www.nata.org/sites/default/files/sudden-cardiac-arrest-consensus-statement.pdf
Management of the athlete with type I diabetes mellitus (2007)	Jimenez CC, Corcoran MH, Crawley JT, et al. National Athletic Trainers' Association position statement: management of the athlete with type I diabetes mellitus. *J Athl Train*. 2007;42(4):536–545.	http://www.nata.org/sites/default/files/MgmtOfAthleteWithType1DiabetesMellitus.pdf
Management of asthma in athletes (2005)	Miller MG, Weiler JM, Baker R, Collins J, D'Alonzo G. National Athletic Trainers' Association position statement: management of asthma in athletes. *J Athl Train*. 2005;40(3):224–245.	http://www.nata.org/sites/default/files/MgmtOfAsthmaInAthletes.pdf
Head-down contact and spearing in tackle football (2004)	Heck JF, Clarke KS, Peterson TR, Torg JS, Weis MP. National Athletic Trainers' Association position statement: head-down contact and spearing in tackle football. *J Athl Train*. 2004;39(1):101–111.	http://www.nata.org/sites/default/files/HeadDownContactAndSpearingInTackleFB.pdf
Management of sport-related concussion (2004)	Guskiewicz KM, Bruce SL, Cantu RC, et al. National Athletic Trainers' Association position statement: management of sport-related concussion. *J Athl Train*. 2004;39(3):280–297.	http://www.nata.org/sites/default/files?MgmtOfSportRelatedConcussion.pdf
Emergency planning in athletics (2002)	Andersen JC, Courson RW, Kleiner DM, McLoda TA. National Athletic Trainers' Association position statement: emergency planning in athletics. *J Athl Train*. 2002;371(1):99–104.	http://www.nata.org/sites/default/files/EmergencyPlanningInAthletics.pdf
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Fluid replacement for athletes (2000)	Casa DJ, Armstrong LE, Hillman SK, et al. National Athletic Trainers' Association position statement: fluid replacement for athletes. *J Athl Train*. 2000; 35(2):212–224.	http://www.nata.org/sites/default/files/FluidReplacementsForAthletes.pdf
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[a] Updated position statements are posted at www.nata.org. Readers should check the Web site for the most current versions.

[b] Available online only.

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