National Athletic Trainers’ Association
Position Statement: Emergency Planning in Athletics

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Objectives: To educate athletic trainers and others about the need for emergency planning, to provide guidelines in the development of emergency plans, and to advocate documentation of emergency planning.

Background: Most injuries sustained during athletics or other physical activity are relatively minor. However, potentially limb-threatening or life-threatening emergencies in athletics and physical activity are unpredictable and occur without warning. Proper management of these injuries is critical and should be carried out by trained health services personnel to minimize risk to the injured participant. The organization or institution and its personnel can be placed at risk by the lack of an emergency plan, which may be the foundation of a legal claim.

Recommendations: The National Athletic Trainers’ Association recommends that each organization or institution that sponsors athletic activities or events develop and implement a written emergency plan. Emergency plans should be developed by organizational or institutional personnel in consultation with local emergency medical services. Components of the emergency plan include identification of the personnel involved, specification of the equipment needed to respond to the emergency, and establishment of a communication system to summon emergency care. Additional components of the emergency plan are identification of the mode of emergency transport, specification of the venue or activity location, and incorporation of emergency service personnel into the development and implementation process. Emergency plans should be reviewed and rehearsed annually, with written documentation of any modifications. The plan should identify responsibility for documentation of actions taken during the emergency, evaluation of the emergency response, institutional personnel training, and equipment maintenance. Further, training of the involved personnel should include automatic external defibrillation, cardiopulmonary resuscitation, first aid, and prevention of disease transmission.

Key Words: policies and procedures, athletics, planning, catastrophic

Although most injuries that occur in athletics are relatively minor, limb-threatening or life-threatening injuries are unpredictable and can occur without warning.1 Because of the relatively low incidence rate of catastrophic injuries, athletic program personnel may develop a false sense of security over time in the absence of such injuries.1–4 However, these injuries can occur during any physical activity and at any level of participation. Of additional concern is the heightened public awareness associated with the nature and management of such injuries. Medicolegal interests can lead to questions about the qualifications of the personnel involved, the preparedness of the organization for handling these situations, and the actions taken by program personnel.5

Proper emergency management of limb- or life-threatening injuries is critical and should be handled by trained medical and allied health personnel.1–4 Preparation for response to emergencies includes education and training, maintenance of emergency equipment and supplies, appropriate use of personnel, and the formation and implementation of an emergency plan. The emergency plan should be thought of as a blueprint for handling emergencies. A sound emergency plan is easily understood and establishes accountability for the management of emergencies. Furthermore, failure to have an emergency plan can be considered negligence.5

POSITION STATEMENT

Based on an extensive survey of the literature and expert review, the following is the position of the National Athletic Trainers’ Association (NATA):

1. Each institution or organization that sponsors athletic activities must have a written emergency plan. The emergency plan should be comprehensive and practical, yet flexible enough to adapt to any emergency situation.
2. Emergency plans must be written documents and should be distributed to certified athletic trainers, team and at-
tending physicians, athletic training students, institutional and organizational safety personnel, institutional and organizational administrators, and coaches. The emergency plan should be developed in consultation with local emergency medical services personnel.

3. An emergency plan for athletics identifies the personnel involved in carrying out the emergency plan and outlines the qualifications of those executing the plan. Sports medicine professionals, officials, and coaches should be trained in automatic external defibrillation, cardiopulmonary resuscitation, first aid, and prevention of disease transmission.

4. The emergency plan should specify the equipment needed to carry out the tasks required in the event of an emergency. In addition, the emergency plan should outline the location of the emergency equipment. Further, the equipment available should be appropriate to the level of training of the personnel involved.

5. Establishment of a clear mechanism for communication to appropriate emergency care service providers and identification of the mode of transportation for the injured participant are critical elements of an emergency plan.

6. The emergency plan should be specific to the activity venue. That is, each activity site should have a defined emergency plan that is derived from the overall institutional or organizational policies on emergency planning.

7. Emergency plans should incorporate the emergency care facilities to which the injured individual will be taken. Emergency receiving facilities should be notified in advance of scheduled events and contests. Personnel from the emergency receiving facilities should be included in the development of the emergency plan for the institution or organization.

8. The emergency plan specifies the necessary documentation supporting the implementation and evaluation of the emergency plan. This documentation should identify responsibility for documenting actions taken during the emergency, evaluation of the emergency response, and institutional personnel training.

9. The emergency plan should be reviewed and rehearsed annually, although more frequent review and rehearsal may be necessary. The results of these reviews and rehearsals should be documented and should indicate whether the emergency plan was modified, with further documentation reflecting how the plan was changed.

10. All personnel involved with the organization and sponsorship of athletic activities share a professional responsibility to provide for the emergency care of an injured person, including the development and implementation of an emergency plan.

11. All personnel involved with the organization and sponsorship of athletic activities share a legal duty to develop, implement, and evaluate an emergency plan for all sponsored athletic activities.

12. The emergency plan should be reviewed by the administration and legal counsel of the sponsoring organization or institution.

BACKGROUND FOR THIS POSITION STAND

Need for Emergency Plans

Emergencies, accidents, and natural disasters are rarely predictable; however, when they do occur, rapid, controlled response will likely make the difference between an effective and an ineffective emergency response. Response can be hindered by the chaotic actions and increased emotions of those who make attempts to help persons who are injured or in danger. One method of control for these unpredictable events is an emergency plan that, if well designed and rehearsed, can provide responders with an organized approach to their reaction. The development of the emergency plan takes care and time to ensure that all necessary contingencies have been included. Lessons learned from major emergencies are also important to consider when developing or revising an emergency plan.

Emergency plans are applicable to agencies of the government, such as law enforcement, fire and rescue, and federal emergency management teams. Furthermore, the use of emergency plans is directly applicable to sport and fitness activities due to the inherent possibility of “an untoward event” that requires access to emergency medical services. Of course, when developing an emergency plan for athletics, there is one notable difference from those used by local, state, and federal emergency management personnel. With few exceptions, typically only one athlete, fan, or sideline participant is at risk at one time due to bleeding, internal injury, cardiac arrest, shock, or traumatic head or spine injury. However, emergency planning in athletics should account for an untoward event involving a game official, fan, or sideline participant as well as the participating athlete. Although triage in athletic emergency situations may be rare, this does not minimize the risks involved and the need for carefully prepared emergency care plans. The need for emergency plans in athletics can be divided into 2 major categories: professional and legal.

Professional Need. The first category for consideration in determining the need for emergency plans in athletics is organizational and professional responsibility. Certain governing bodies associated with athletic competition have stated that institutions and organizations must provide for access to emergency medical services if an emergency should occur during any aspect of athletic activity, including in-season and off-season activities. The National Collegiate Athletic Association (NCAA) has recommended that all member institutions develop an emergency plan for their athletic programs. The National Federation of State High School Associations has recommended the same at the secondary school level. The NCAA states, “Each scheduled practice or contest of an institution-sponsored intercollegiate athletics event, as well as out-of-season practices and skills sessions, should include an emergency plan.” The 1999–2000 NCAA Sports Medicine Handbook further outlines the key components of the emergency plan.

Although the 1999–2000 NCAA Sports Medicine Handbook is a useful guide, a recent survey of NCAA member institutions revealed that at least 10% of the institutions do not maintain any form of an emergency plan. In addition, more than one third of the institutions do not maintain emergency plans for the off-season strength and conditioning activities of the sports.

Personnel coverage at NCAA institutions was also found to be an issue. Nearly all schools provided personnel qualified to administer emergency care for high-risk contact sports, but fewer than two thirds of institutions provided adequate personnel to sports such as cross-country and track. In a memorandum dated March 25, 1999, and sent to key personnel at
all schools, the president of the NCAA reiterated the recommendations in the 1999–2000 NCAA Sports Medicine Handbook to maintain emergency plans for all sport activities, including skill instruction, conditioning, and the nontraditional practice seasons.8

A need for emergency preparedness is further recognized by several national organizations concerned with the delivery of health care services to fitness and sport participants, including the NATA Education Council,10 NATA Board of Certification, Inc,11 American College of Sports Medicine, International Health Racquet and Sports Club Association, American College of Cardiology, and Young Men’s Christian Association.12 The NATA-approved athletic training educational competencies for athletic trainers include several references to emergency action plans.10 The knowledge of the key components of an emergency plan, the ability to recognize and appraise emergency plans, and the ability to develop emergency plans are all considered required tasks of the athletic trainer.11 These responsibilities justify the need for the athletic trainer to be involved in the development and application of emergency plans as a partial fulfillment of his or her professional obligations.

In addition to the equipment and personnel involved in emergency response, the emergency plan must include consideration for the sport activity and rules of competition, the weather conditions, and the level of competition.13 The variation in these factors makes venue-specific planning necessary because of the numerous contingencies that may occur. For example, many youth sport activities include both new participants of various sizes who may not know the rules of the activity and those who have participated for years. Also, outdoor sport activities include the possibility of lightning strikes, excessive heat and humidity, and excessive cold, among other environmental concerns that may not be factors during indoor activities. Organizations in areas of the country in which snow may accumulate must consider provisions for ensuring that accessibility by emergency vehicles is not hampered. In addition, the availability of safety equipment that is necessary for participation may be an issue for those in underserved areas. The burden of considering all the possible contingencies in light of the various situations must rest on the professionals, who are best trained to recognize the need for emergency plans and who can develop and implement the venue-specific plans.

Legal Need. Also of significance is the legal basis for the development and application of an emergency plan. It is well known that organizational medical personnel, including certified athletic trainers, have a legal duty as reasonable and prudent professionals to ensure high-quality care of the participants. Of further legal precedence is the accepted standard of care by which allied health professionals are measured.14 This standard of care provides necessary accountability for the actions of both the practitioners and the governing body that oversees those practitioners. The emergency plan has been categorized as a written document that defines the standard of care required during an emergency situation.15 Herbert emphasized that well-formulated, adequately written, and periodically rehearsed emergency response protocols are absolutely required by sports medicine programs. Herbert further stated that the absence of an emergency plan frequently is the basis for claim and suit based on negligence.

One key indicator for the need for an emergency action plan is the concept of foreseeability. The organization administrators and the members of the sports medicine team must question whether a particular emergency situation has a reasonable possibility of occurring during the sport activity in question.14,15,17 For example, if it is reasonably possible that a catastrophic event such as a head injury, spine injury, or other severe trauma may occur during practice, conditioning, or competition in a sport, a previously prepared emergency plan must be in place. The medical and allied health care personnel must constantly be on guard for potential injuries, and although the occurrence of limb-threatening or life-threatening emergencies is not common, the potential exists. Therefore, prepared emergency responders must have planned in advance for the action to be taken in the event of such an emergency.

Several legal claims and suits have indicated or alluded to the need for emergency plans. In Gathers v Loyola Marymount University,18 the state court settlement included a statement that care was delayed for the injured athlete, and the plaintiffs further alleged that the defendants acted negligently and carelessly in not providing appropriate emergency response. These observations strongly support the need to have clear emergency plans in place, rehearsed, and carried out. In several additional cases,19–21 the courts have stated that proper care was delayed, and it can be reasoned that these delays could have been avoided with the application of a well-prepared emergency plan.

Perhaps the most significant case bearing on the need for emergency planning is Kleinknecht v Gettysburg College, which came before the appellate court in 1993.5,17 In a portion of the decision, the court stated that the college owed a duty to the athletes who are recruited to be athletes at the institution. Further, as a part of that duty, the college must provide “prompt and adequate emergency services while engaged in the school-sponsored intercollegiate athletic activity for which the athlete had been recruited.”17 The same court further ruled that reasonable measures must be ensured and in place to provide prompt treatment of emergency situations. One can conclude from these rulings that planning is critical to ensure prompt and proper emergency medical care, further validating the need for an emergency plan.5

Based on the review of the legal and professional literature, there is no doubt regarding the need for organizations at all levels that sponsor athletic activities to maintain an up-to-date, thorough, and regularly rehearsed emergency plan. Furthermore, members of the sports medicine team have both legal and professional obligations to perform this duty to protect the interests of both the participating athletes and the organization or institution. At best, failure to do so will inevitably result in inefficient athlete care, whereas at worst, gross negligence and potential life-threatening ramifications for the injured athlete or organizational personnel are likely.

Components of Emergency Plans

Organizations that sponsor athletic activities have a duty to develop an emergency plan that can be implemented immediately and to provide appropriate standards of health care to all sports participants.5,14,15,17 Athletic injuries may occur at any time and during any activity. The sports medicine team must be prepared through the formulation of an emergency plan, proper coverage of events, maintenance of appropriate emergency equipment and supplies, use of appropriate emergency medical personnel, and continuing education in the area of emergency medicine. Some potential emergencies may be averted through careful preparticipation physical

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Sample Venue-Specific Emergency Protocol

University Sports Medicine Football Emergency Protocol

1. Call 911 or other emergency number consistent with organizational policies
2. Instruct emergency medical services (EMS) personnel to “report to _______ and meet _______ at _______ as we have an injured student-athlete in need of emergency medical treatment.”
   University Football Practice Complex: _______ Street entrance (gate across street from _______ ) cross street: _______ Street
   University Stadium: Gate _______ entrance off _______ Road
3. Provide necessary information to EMS personnel:
   ● name, address, telephone number of caller
   ● number of victims; condition of victims
   ● first-aid treatment initiated
   ● specific directions as needed to locate scene
   ● other information as requested by dispatcher
4. Provide appropriate emergency care until arrival of EMS personnel: on arrival of EMS personnel, provide pertinent information (method of injury, vital signs, treatment rendered, medical history) and assist with emergency care as needed

Note:
   ● sports medicine staff member should accompany student-athlete to hospital
   ● notify other sports medicine staff immediately
   ● parents should be contacted by sports medicine staff
   ● inform coach(es) and administration
   ● obtain medical history and insurance information
   ● appropriate injury reports should be completed

Emergency Telephone Numbers
   _______ Hospital
   _______ Emergency Department
   _______ University Health Center
   _______ Campus Police

Emergency Signals
   Physician: arm extended overhead with clenched first
   Paramedics: point to location in end zone by home locker room and wave onto field
   Spine board: arms held horizontally
   Stretcher: supinated hands in front of body or waist level
   Splints: hand to lower leg or thigh

screenings, adequate medical coverage, safe practice and training techniques, and other safety measures. However, accidents and injuries are inherent with sports participation, and proper preparation on the part of the sports medicine team will enable each emergency situation to be managed appropriately.

The goal of the sports medicine team is the delivery of the highest possible quality health care to the athlete. Management of the emergency situation that occurs during athletic activities may involve certified athletic trainers and students, emergency medical personnel, physicians, and coaches working together. Just as with an athletic team, the sports medicine team must work together as an efficient unit to accomplish its goals. In an emergency situation, the team concept becomes even more critical, because time is crucial and seconds may mean the difference among life, death, and permanent disability. The sharing of information, training, and skills among the various emergency medical care providers helps reach the goal.

Implementation. Once the importance of the emergency plan is realized and the plan has been developed, the plan must be implemented. Implementation of the emergency plan requires 3 basic steps.

First, the plan must be committed to writing (Table) to provide a clear response mechanism and to allow for continuity among emergency team members. This can be accomplished by using a flow sheet or an organizational chart. It is also important to have a separate plan or to modify the plan for different athletic venues and for practices and games. Emergency team members, such as the team physician, who are present at games may not necessarily be present at practices. Moreover, the location and type of equipment and communication devices may differ among sports, venues, and activity levels.

The second step is education. It is important to educate all the members of the emergency team regarding the emergency plan. All personnel should be familiar with the emergency medical services system that will provide coverage to their venues and include their input in the emergency plan. Each team member, as well as institution or organization administrators, should have a written copy of the emergency plan that provides documentation of his or her roles and responsibilities in emergency situations. A copy of the emergency plan specific to each venue should be posted prominently by the available telephone.

Third, the emergency plan and procedures have to be rehearsed. This provides team members a chance to maintain their emergency skills at a high level of competency. It also provides an opportunity for athletic trainers and emergency medical personnel to communicate regarding specific policies and procedures in their particular region of practice. This rehearsal can be accomplished through an annual in-service meeting, preferably before the highest-risk sports season (eg, football, ice hockey, lacrosse). Reviews should be undertaken as needed throughout the sports season, because emergency medical procedures and personnel may change.
Personnel. In an athletic environment, the first person who responds to an emergency situation may vary widely\textsuperscript{22,24}; it may be a coach or a game official, a certified athletic trainer, an emergency medical technician, or a physician. This variation in the first responder makes it imperative that an emergency plan be in place and rehearsed. With a plan in place and rehearsed, these differently trained individuals will be able to work together as an effective team when responding to emergency situations.

The plan should also outline who is responsible for summoning help and clearing the uninjured from the area.

In addition, all personnel associated with practices, competitions, skills instruction, and strength and conditioning activities should have training in automatic external defibrillation and current certification in cardiopulmonary resuscitation, first aid, and the prevention of disease transmission.\textsuperscript{5,7}

Equipment. All necessary supplemental equipment should be at the site and quickly accessible.\textsuperscript{13,25} Equipment should be in good operating condition, and personnel must be trained in advance to use it properly. Improvements in technology and emergency training require personnel to become familiar with the use of automatic external defibrillators, oxygen, and advanced airways.

It is imperative that health professionals and organizational administrators recognize that recent guidelines published by the American Heart Association call for the availability and use of automatic external defibrillators and that defibrillation is considered a component of basic life support.\textsuperscript{26} In addition, these guidelines emphasize use of the bag-valve mask in emergency resuscitation and the use of emergency oxygen and advanced airways in emergency care. Personnel should consider receiving appropriate training for these devices and should limit use to devices for which they have been trained.

To ensure that emergency equipment is in working order, all equipment should be checked on a regular basis. Also, the use of equipment should be regularly rehearsed by emergency personnel, and the emergency equipment that is available should be appropriate for the level of training of the emergency medical providers and the venue.

Communication. Access to a working telephone or other telecommunications device, whether fixed or mobile, should be ensured.\textsuperscript{5,17,21} The communications system should be checked before each practice or competition to ensure proper working order. A back-up communication plan should be in effect in case the primary communication system fails. A listing of appropriate emergency numbers should be either posted by the communication system or readily available, as well as the street address of the venue and specific directions (cross streets, landmarks, and so on) (Table).

Transportation. The emergency plan should encompass transportation of the sick and injured. Emphasis should be placed on having an ambulance on site at high-risk events.\textsuperscript{15} Emergency medical services response time should also be factored in when determining on-site ambulance coverage. Consideration should be given to the level of transportation service that is available (eg, basic life support, advanced life support) and the equipment and training level of the personnel who staff the ambulance.\textsuperscript{23}

In the event that an ambulance is on site, a location should be designated with rapid access to the site and a cleared route for entering and exiting the venue.\textsuperscript{19} In the emergency evaluation, the primary survey assists the emergency care provider in identifying emergencies that require critical intervention and in determining transport decisions. In an emergency situation, the athlete should be transported by ambulance to the most appropriate receiving facility, where the necessary staff and equipment can deliver appropriate care.\textsuperscript{23}

In addition, a plan must be available to ensure that the activity areas are supervised if the emergency care provider leaves the site to transport the athlete.

Venue Location. The emergency plan should be venue specific, based on the size of the venue and the activity involved (Table). The plan for each venue should encompass accessibility to emergency personnel, communication system, equipment, and transportation.

At home sites, the host medical providers should orient the visiting medical personnel regarding the site, emergency personnel, equipment available, and procedures associated with the emergency plan.

At away or neutral sites, the coach or athletic trainer should identify, before the event, the availability of communication with emergency medical services and should verify service and reception, particularly in rural areas. In addition, the name and location of the nearest emergency care facility and the availability of an ambulance at the event site should be ascertained.

Emergency Care Facilities. The emergency plan should incorporate access to an emergency medical facility. In selection of the appropriate facility, consideration should be given to the location with respect to the athletic venue. Consideration should also include the level of service available at the emergency facility.

The designated emergency facility and emergency medical services should be notified in advance of athletic events. Furthermore, it is recommended that the emergency plan be reviewed with both medical facility administrators and in-service medical staff regarding pertinent issues involved in athlete care, such as proper removal of athletic equipment in the facility when appropriate.\textsuperscript{22,23,27}

Documentation. A written emergency plan should be reviewed and approved by sports medicine team members and institutions involved. If multiple facilities or sites are to be used, each will require a separate plan. Additional documentation should encompass the following\textsuperscript{15,16}:

1. Delineation of the person and/or group responsible for documenting the events of the emergency situation
2. Follow-up documentation on evaluation of response to emergency situation
3. Documentation of regular rehearsal of the emergency plan
4. Documentation of personnel training
5. Documentation of emergency equipment maintenance

It is prudent to invest organizational and institutional ownership in the emergency plan by involving administrators and sport coaches as well as sports medicine personnel in the planning and documentation process. The emergency plan should be reviewed at least annually with all involved personnel. Any revisions or modifications should be reviewed and approved by the personnel involved at all levels of the sponsoring organization or institution and of the responding emergency medical services.

SUMMARY

The purpose of this statement is to present the position of the NATA on emergency planning in athletics. Specifically,
professional and legal requirements mandate that organizations or institutions sponsoring athletic activities have a written emergency plan. A well-thought-out emergency plan consists of a number of factors, including, but not necessarily limited to, personnel, equipment, communication, transportation, and documentation. Finally, all sports medicine professionals, coaches, and organizational administrators share professional and legal duties to develop, implement, and evaluate emergency plans for sponsored athletic activities.

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REFERENCES
19. Mogabgab v Orleans Parish School Board, 239 So2d 456 (Court of Appeals, Los Angeles, 970).
20. Hanson v Kynast, 494 NE2d 1091 (Oh 1986).
Objective: To present recommendations to optimize the fluid-replacement practices of athletes.

Background: Dehydration can compromise athletic performance and increase the risk of exertional heat injury. Athletes do not voluntarily drink sufficient water to prevent dehydration during physical activity. Drinking behavior can be modified by education, increasing accessibility, and optimizing palatability. However, excessive overdrinking should be avoided because it can also compromise physical performance and health. We provide practical recommendations regarding fluid replacement for athletes.

Recommendations: Educate athletes regarding the risks of dehydration and overhydration on health and physical performance. Work with individual athletes to develop fluid-replacement practices that optimize hydration status before, during, and after competition.

Key Words: athletic performance, dehydration, heat illness, hydration protocol, hydration status, oral rehydration solution, rehydration

During exercise, evaporation is usually the primary mechanism of heat dissipation. The evaporation of sweat from the skin’s surface assists the body in regulating core temperature. If the body cannot adequately evaporate sweat from the skin’s surface, core temperature rises rapidly. A side effect of sweating is the loss of valuable fluids from the finite reservoir within the body, the rate being related to exercise intensity, individual differences, environmental conditions, acclimatization state, clothing, and baseline hydration status. Athletes whose sweat loss exceeds fluid intake become dehydrated during activity. Therefore, a person with a high sweat rate who undertakes intense exercise in a hot, humid environment can rapidly become dehydrated. Dehydration of 1% to 2% of body weight begins to compromise physiologic function and negatively influence performance. Dehydration of greater than 3% of body weight further disturbs physiologic function and increases an athlete’s risk of developing an exertional heat illness (ie, heat cramps, heat exhaustion, or heat stroke). This level of dehydration is common in sports; it can be elicited in just an hour of exercise or even more rapidly if the athlete enters the exercise session dehydrated. The onset of significant dehydration is preventable, or at least modifiable, when hydration protocols are followed to assure all athletes the most productive and the safest athletic experience.

The purpose of this position stand is to 1) provide useful recommendations to optimize fluid replacement for athletes, 2) emphasize the physiologic, medical, and performance considerations associated with dehydration, and 3) identify factors that influence optimal rehydration during and after athletic participation.

RECOMMENDATIONS

The National Athletic Trainers’ Association (NATA) recommends the following practices regarding fluid replacement for athletic participation:

1. Establish a hydration protocol for athletes, including a rehydration strategy that considers the athlete’s sweat rate, sport dynamics (eg, rest breaks, fluid access), environmental factors, acclimatization state, exercise duration, exercise intensity, and individual preferences (see Table 1 for examples of potential outcomes).
2. A proper hydration protocol considers each sport’s unique features. If rehydration opportunities are frequent (eg, baseball, football, track and field), the athlete can consume smaller volumes at a convenient pace based on sweat rate and environmental conditions. If rehydration must occur at specific times (eg, soccer, lacrosse, distance running), the athlete must consume fluids to maximize hydration within the sport’s confines and rules.

3. Fluid-replacement beverages should be easily accessible in individual fluid containers and flavored to the athlete’s preference. Individual containers permit easier monitoring of fluid intake. Clear water bottles marked in 100-mL (3.4-fl oz) increments provide visual reminders to athletes to drink beyond thirst satiation or the typical few gulps. Carrying water bottles or other hydration systems, when practical, during exercise encourages greater fluid volume ingestion.

4. Athletes should begin all exercise sessions well hydrated. Hydration status can be approximated by athletes and athletic trainers in several ways (Table 2). Assuming proper hydration, pre-exercise body weight should be relatively consistent across exercise sessions. Determine the percentage difference between the current body weight and the hydrated baseline body weight. Remember that body weight is dynamic. Frequent exercise sessions can induce nonfluid-related weight loss influenced by timing of meals and defecation, time of day, and calories expended in exercise. The simplest method is comparison of urine color (from a sample in a container) with a urine color chart (Figure). Measuring urine specific gravity (USG) with a refractometer (available for less than $150) is less subjective than comparing urine color and also simple to use. Urine volume is another indicator of hydration status but inconvenient to collect and measure. For color analysis or specific gravity, use midstream urine collection for consistency and accuracy. Remember that body weight changes during exercise give the best indication of hydration status. Because of urine and body weight dynamics, measure urine before exercise and check body weight (percentage of body weight change) before, during, and after exercise sessions to estimate fluid balance.

5. To ensure proper pre-exercise hydration, the athlete should consume approximately 500 to 600 mL (17 to 20 fl oz) of water or a sports drink 2 to 3 hours before exercise and 200 to 300 mL (7 to 10 fl oz) of water or a sports drink 10 to 20 minutes before exercise.

6. Fluid replacement should approximate sweat and urine losses and at least maintain hydration at less than 2% body weight reduction. This generally requires 200 to 300 mL (7 to 10 fl oz) every 10 to 20 minutes. Specific individual recommendations are calculated based on sweat rates, sport dynamics, and individual tolerance. Maintaining hydration status in athletes with high sweat rates, in sports with limited fluid access, and during high-intensity exercise can be difficult, and special efforts should be made to minimize dehydration. Dangerous hyperhydration is also a risk if athletes drink based on published recommendations and not according to individual needs.

7. Postexercise hydration should aim to correct any fluid loss accumulated during the practice or event. Ideally completed within 2 hours, rehydration should contain water to restore hydration status, carbohydrates to replenish glycogen stores, and electrolytes to speed rehydration. The primary goal is the immediate return of physiologic function (especially if an exercise bout will follow). When rehydration must be rapid, the athlete should compensate for obligatory urine losses incurred during the rehydration process and drink about 25% to 50% more than sweat losses to assure optimal hydration 4 to 6 hours after the event.

8. Fluid temperature influences the amount consumed. While individual differences exist, a cool beverage of 10°C to 15°C (50° to 59°F) is recommended.

9. The Wet Bulb Globe Temperature (WBGT) should be ascertained in hot environments. Very high relative humidity limits evaporative cooling; the air is nearly saturated with water vapor, and evaporation is minimized. Thus, dehydration associated with high sweat losses can induce a rapid core temperature increase due to the inability to dissipate heat. Measuring core temperature rectally allows the athlete’s thermal status to be accurately determined. See the NATA position statement on heat illnesses for expanded information on this topic.

10. In many situations, athletes benefit from including carbohydrates (CHO) in their rehydration protocols. Consuming CHO during the pre-exercise hydration session (2 to 3 hours pre-exercise), as in item 5, along with a normal daily diet increases glycogen stores. If exercise is intense, then consuming CHO about 30 minutes pre-exercise may also be beneficial. Include CHO in the rehydration beverage during exercise if the session lasts longer than 45 to 50 minutes or is intense. An ingestion rate of about 1 g/min (0.04 oz/min) maintains optimal carbohydrate metabolism: for example, 1 L of a 6% CHO drink per hour of exercise. CHO concentrations greater than 8% increase the rate of CHO delivery to the body but compromise the rate of fluid emptying from the stomach and absorbed from the intestine. Fruit juices, CHO gels, sodas, and some sports drinks have CHO concentrations greater than 8% and are not recommended during an exercise session as the sole beverage. Athletes should consume CHOs at least 30 minutes before the normal onset of fatigue and earlier if the environmental conditions are unusually extreme, although this may not apply for very intense short-term exercise, which may require earlier intake of CHOs. Most CHO forms (ie, glucose, sucrose, glucose polymers) are suitable, and the absorption rate is maximized when multiple forms are consumed simultaneously. Substances to be limited include fructose (which may cause gastrointestinal distress); those to be avoided include caffeine, alcohol (which may increase urine output and reduce fluid retention), and carbonated beverages (which may reduce voluntary fluid intake due to stomach fullness).

11. Those supervising athletes should be able to recognize the basic signs and symptoms of dehydration: thirst, irritability, and general discomfort, followed by headache, weakness, dizziness, cramps, chills, vomiting, nausea, head or neck heat sensations, and decreased performance. Early diagnosis of dehydration decreases the occurrence and severity of heat illness. A conscious, cognizant, dehydrated athlete without gastrointestinal distress can aggressively rehydrate orally, while one with mental compromise from dehydration or gastrointestinal distress should be transported to a medical facility for intravenous rehydration. For a complete description of heat illnesses and issues
related to hyperthermia, see the NATA position statement on heat illnesses.

12. Inclusion of sodium chloride in fluid-replacement beverages should be considered under the following conditions: inadequate access to meals or meals not eaten; physical activity exceeding 4 hours in duration; or during the initial days of hot weather. Under these conditions, adding modest amounts of salt (0.3 to 0.7 g/L) can offset salt loss in sweat and minimize medical events associated with electrolyte imbalances (eg, muscle cramps, hyponatremia). Adding a modest amount of salt (0.3 to 0.7 g/L) to all hydration beverages would be acceptable to stimulate thirst, increase voluntary fluid intake, and decrease the risk of hyponatremia and should cause no harm.

13. Calculate each athlete’s sweat rate (sweating rate = pre-exercise body weight − postexercise body weight + fluid intake − urine volume/exercise time in hours) for a representative range of environmental conditions, practices, and competitions (Table 3). This time-consuming task can be made easier by weighing a large number of athletes before an intense 1-hour practice session and then reweighing them at the end of the 1-hour practice. Sweat rate can now be easily calculated (do not allow rehydration or urination during this 1 hour when sweat rate is being determined to make the task even easier). This calculation is the most fundamental consideration when establishing a rehydration protocol. Average sweat rates from the scientific literature or other athletes can vary from 0.5 L/h to more than 2.5 L/h (0.50 to 2.50 kg/h) and are not ideal to use.

14. Heat acclimatization induces physiologic changes that may alter individual fluid-replacement considerations. First, sweat rate generally increases after 10 to 14 days of heat exposure, requiring a greater fluid intake for a similar bout of exercise. An athlete’s sweat rate should be reassessed after acclimatization. Second, moving from a cool environment to a warm environment increases the overall sweat rate for a bout of exercise. The athlete’s hydration status must be closely monitored for the first week of exercise in a warm environment. Third, increased sodium intake may be warranted during the first 3 to 5 days of heat exposure, since the increased thermal strain and associated increased sweat rate increase the sodium lost in sweat. Adequate sodium intake optimizes fluid palatability and absorption during the first few days and may decrease exercise-associated muscle cramping. After 5 to 10 days, the sodium concentration of sweat decreases, and normal sodium intake suffices.

15. All sports requiring weight classes (ie, wrestling, judo, rowing) should mandate a check of hydration status at weigh-in to ensure that the athlete is not dehydrated. A USG less than or equal to 1.020 or urine color less than or equal to 4 should be the upper range of acceptable on weigh-in. Any procedures used to induce dramatic dehydration (eg, diuretics, rubber suits, exercising in a sauna) are strictly prohibited.

16. Hyperhydration by ingesting a pre-exercise glycerol and water beverage has equivocal support from well-controlled studies. At this time, evidence is insufficient to endorse the practice of hyperhydration via glycerol. Also, a risk of side effects such as headaches and gastrointestinal distress exists when glycerol is consumed.

17. Consider modifications when working with prepubescent and adolescent athletes who exercise intensely in the heat and may not fully comprehend the medical and performance consequences of dehydration. Focus special attention on schedules and event modification to minimize environmental stress and maximize time for fluid replacement. Make available the most palatable beverage possible. Educate parents and coaches about rehydration and the signs of dehydration. Monitor and remove a child from activity promptly if signs or symptoms of dehydration occur.

18. Large-scale event management (eg, tournaments, camps) requires advance planning. Ample fluid and cups should be conveniently available. With successive practice sessions during a day or over multiple days (as in most summer sport camps), check hydration status daily before allowing continued participation. Be aware of unhealthy behaviors, such as eating disorders and dehydration in weight-class sports. Use extra caution with novice and unconditioned athletes, and remember, many athletes are not supervised on a daily basis. If the WBGT dictates, modify events (change game times or cancel) or change game dynamics (insert nonroutine water breaks, shorten game times). Recruit help from fellow athletic trainers in local schools, student athletic trainers, and athletes from other sports to ensure that hydration is maintained at all venues (ie, along a road race course, on different fields during a tournament). Be sure all assistants can communicate with the supervising athletic trainer at a central location. For successive-day events, provide educational materials on rehydration principles to inform athletes and parents of this critical component of athletic performance.

19. Implementing a hydration protocol for athletes will only succeed if athletes, coaches, athletic trainers, and team physicians realize the importance of maintaining proper hydration status and the steps required to accomplish this goal. Here are the most critical components of hydration education:

- Educate athletes on the effects of dehydration on physical performance.
- Inform athletes on how to monitor hydration status.
- Convince athletes to participate in their own hydration protocols based on sweat rate, drinking preferences, and personal responses to different fluid quantities.
- Encourage coaches to mandate rehydration during practices and competitions, just as they require other drills and conditioning activities.
- Have a scale accessible to assist athletes in monitoring weight before, during, and after activity.
- Provide the optimal oral rehydration solution (water, CHOs, electrolytes) before, during, and after exercise.
- Implement the hydration protocol during all practices and games, and adapt it as needed.
- Finally, encourage event scheduling and rule modifications to minimize the risks associated with exercise in the heat.

BACKGROUND AND LITERATURE REVIEW

Dehydration and Exercise

Physiologic Implications. All physiologic systems in the human body are influenced by dehydration.1,2 The degree of
The body attempts to balance endogenous heat production and exogenous heat accumulation by heat dissipation via conduction, convection, evaporation, and radiation. The relative contribution of each method depends on the ambient temperature, relative humidity, and exercise intensity. As ambient temperature rises, conduction and convection decrease markedly, and radiation becomes nearly insignificant. Heat loss from evaporation is the predominant heat-dissipating mechanism for the exercising athlete. In warm, humid conditions, evaporation may account for more than 80% of heat loss. In hot, dry conditions, evaporation may account for as much as 98% of sweating.

Water is the major component of the human body, accounting for approximately 73% of lean body mass. Body water is distributed within and between cells and in the plasma. At rest, approximately 30% to 35% of total body mass is intracellular fluid, 20% to 25% is interstitial fluid, and 5% is plasma. Water movement between compartments occurs due to hydrostatic pressure and osmotic-oncotic gradients. Because sweat is hypotonic relative to body water, the elevation of extracellular tonicity results in water movement from intracellular to extracellular spaces.

A major consequence of dehydration is an increase in core temperature during physical activity, with core temperature rising an additional 0.15 to 0.20°C for every 1% of body weight lost during activity. The added thermal strain occurs due to both impaired skin blood flow and altered sweating responses, which is best illustrated by the delayed onset of skin vasodilation and sweating when a dehydrated person begins to exercise. This thermo-regulatory changes may negate the physiologic advantages resulting from increased fitness and heat acclimatization. Additionally, heat tolerance is reduced and exercise time to exhaustion occurs at lower core temperatures with hypohydration.

Table 1. Sample Hydration Protocol Worksheet

<table>
<thead>
<tr>
<th>Parameter to Consider</th>
<th>Example A: College Soccer, Katie (60 kg)*</th>
<th>Example B: High School Basketball, Mike (80 kg)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) WBGT</td>
<td>28.3°C (83°F)</td>
<td>21.1°C (70°F)</td>
</tr>
<tr>
<td>2) Sweat rate†</td>
<td>1.7 L/h</td>
<td>1.2 L/h</td>
</tr>
<tr>
<td>3) Acclimatized</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>4) Length of activity</td>
<td>2 45-minute halves</td>
<td>4 10-minute quarters</td>
</tr>
<tr>
<td>5) Intensity</td>
<td>Game situation (maximal)</td>
<td>Game situation (maximal)</td>
</tr>
<tr>
<td>6) Properly prehydrated</td>
<td>No (began 2% body weight)</td>
<td>Yes</td>
</tr>
<tr>
<td>7) Individual container</td>
<td>Yes</td>
<td>No (just cups)</td>
</tr>
<tr>
<td>8) Type of beverage</td>
<td>5% to 7% CHO‡ solution</td>
<td>5% to 7% CHO solution</td>
</tr>
<tr>
<td>9) Assess hydration status</td>
<td>At halftime (with scale)</td>
<td>Normal hydration</td>
</tr>
<tr>
<td>10) Available breaks</td>
<td>Halftime</td>
<td>Quarters, half timeouts</td>
</tr>
<tr>
<td>11) Amount given</td>
<td>Maximal comfortable predetermined amount given at half time (about 700 to 1000 L)</td>
<td>200 mL at quarter breaks</td>
</tr>
<tr>
<td>12) End hydration status</td>
<td>−4.8% body weight</td>
<td>400 mL at half time</td>
</tr>
<tr>
<td>13) Hydrated body weight</td>
<td>60 kg</td>
<td>100 mL at 1 timeout/half</td>
</tr>
<tr>
<td>Pre-exercise body weight</td>
<td>58.8 kg</td>
<td>Normal hydration</td>
</tr>
<tr>
<td>Halftime body weight</td>
<td>57.5</td>
<td>80 kg</td>
</tr>
<tr>
<td>Postexercise body weight</td>
<td>57.1</td>
<td>80 kg</td>
</tr>
</tbody>
</table>
| *Assumptions: Both are starters and play a full game.†Sweat rate determined under similar parameters described in example (ie, acclimatization state, WBGT, intensity, etc) under normal game conditions (ie, no injury timeouts, overtime, etc). Note: Keep results on record for future reference.‡CHO, carbohydrate.

Table 2. Indexes of Hydration Status

<table>
<thead>
<tr>
<th>Condition</th>
<th>% Body Weight Change*</th>
<th>Urine Color</th>
<th>USG†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Well hydrated</td>
<td>+1 to −1</td>
<td>1 or 2</td>
<td>&lt;1.010</td>
</tr>
<tr>
<td>Minimal dehydration</td>
<td>−1 to −3</td>
<td>3 or 4</td>
<td>1.010–1.020</td>
</tr>
<tr>
<td>Significant dehydration</td>
<td>−3 to −5</td>
<td>5 or 6</td>
<td>1.021–1.030</td>
</tr>
<tr>
<td>Serious dehydration</td>
<td>&gt;5</td>
<td>&gt;6</td>
<td>&gt;1.030</td>
</tr>
</tbody>
</table>


See Figure for urine color chart and references. Please note that obtaining a urine sample may not be possible if the athlete is seriously dehydrated. These are physiologically independent entities, and the numbers provided are only general guidelines.

Dehydration indicates the extent of systemic compromise. Isolating the physiologic changes that contribute to decrements in performance is difficult, as any change in 1 system (ie, cardiovascular) influences the performance of other systems (ie, thermoregulatory, muscular).

The body attempts to balance endogenous heat production and exogenous heat accumulation by heat dissipation via conduction, convection, evaporation, and radiation. The relative contribution of each method depends on the ambient temperature, relative humidity, and exercise intensity. As ambient temperature rises, conduction and convection decrease markedly, and radiation becomes nearly insignificant. Heat loss from evaporation is the predominant heat-dissipating mechanism for the exercising athlete. In warm, humid conditions, evaporation may account for more than 80% of heat loss. In hot, dry conditions, evaporation may account for as much as 98% of sweating. If sufficient fluids are not consumed to offset the rate of water loss via sweating, progressive dehydration will occur. The sweating response is critical to body cooling during exercise in the heat. Therefore, any factor that limits evaporation (ie, high humidity, dehydration) will have profound effects on physiologic function and athletic performance.
deficit. For example, heart rate rises an additional 3 to 5 beats per minute for every 1% of body weight loss.14 The stroke-volume reduction seen with dehydration appears to be due to reduced central venous pressure, resulting from reduced blood volume and the additional hyperthermia imposed by dehydration.6,14,25,32–34

Both hypovolemia7,17,35,36 and hypertonicity7,35,37–39 have been suggested as mechanisms for the altered thermoregulatory and cardiovascular responses during dehydration. Manipulation of each factor independently has resulted in decreased blood flow to the skin and sweating responses.28,34 Some authors7,35 have argued that hypovolemia is primarily responsible for the thermoregulatory changes by reducing cardiac preload and may alter the feedback to the hypothalamus via the atrial pressure receptors (baroreceptors). The hypothalamic thermoregulatory centers may induce a decrease in the blood volume perfusing the skin in order to reestablish a normal cardiac preload. Some studies30,31 have provided support for this hypothesis, but it is clearly not the only variable influencing thermoregulation during hyponhydration. Two hypotheses explain the role of hyperosmolality on the thermoregulatory system. Peripheral regulation may occur via the strong osmotic pressure influence of the interstitium, limiting the available fluid sources for the eccrine sweat glands.42 However, while this peripheral influence is likely, it seems more feasible that central brain regulation plays the largest role.7 The neurons surrounding the thermoregulatory control centers in the hypothalamus are sensitive to osmolality.43,44 Changes in the plasma osmolality of the blood perfusing the hypothalamus affect body water regulation and the desire for fluid consumption.28,32,45 It is likely that both hypovolemia and hypertonicity contribute to body fluid regulation.

Potential changes at the level of the muscle tissue include a possible increased rate of glycogen degradation,18,46,47 elevated muscle temperature,48 and increased lactate levels.49 These changes may be caused by a decrease in blood perfusion of the muscle tissue during the recovery between contractions.50

The psychological changes associated with exercise in a dehydrated state should not be overlooked. Dehydration increases the rating of perceived exertion and impairs mental functioning.14,51 Dehydration also decreases the motivation to exercise and decreases the time to exhaustion, even in instances when strength is not compromised.52–54 These are important factors when considering the motivation required by high-level athletes to maintain maximal performance.

Performance Implications. Studies investigating the role of dehydration on muscle strength have generally shown decrements in performance at 5% or more dehydration.15,33,35–38 The greater the degree of dehydration, the more negative the impact on physiologic systems and overall athletic performance.

Most studies30,55,59–62 that address the influence of dehydration on muscle endurance show that dehydration of 3% to 4% elicits a performance decrement, but in 1 study,33 this finding was not supported. Interestingly, hypohydrated wrestlers who were working at maximal or near-maximal muscle activity for more than 30 seconds had a decrease in performance.63 The environmental conditions may also play an important role in muscle endurance.33,48

The research concerning maximal aerobic power and the physical work capacity for extended exercise is relatively consistent. Maximal aerobic power usually decreases with more than 3% hypohydration.6 In the heat, aerobic power decrements are exaggerated.33 Even at 1% to 2% hypohydration in a cool environment, loss of aerobic power is demonstrated. Two important studies have noted a decrease in physical work capacity with less than 2% dehydration during intense exercise in the heat.64,65 When the percentage of dehydration increased, physical work capacity decreased by as much as 35% to 48%,66,68 and physical work capacity often decreased even when maximal aerobic power did not change.33,46,48,65 Hypohydration of 2.5% of body weight results in significant performance decrements while exercising in the heat, regardless of fitness or heat acclimation status, although enhanced fitness and acclimation can lessen the effects of dehydration.69 Partial rehydration will enhance performance during an ensuing exercise session in the heat, which is important when faced with the reality of sports situations.39,70 The performance decrements noted with low to moderate levels of hypohydration may be due to an increased perception of fatigue.50

Rehydration and Exercise

Factors Influencing Rehydration. The degree of environmental stress is determined by temperature, humidity, wind speed, and radiant energy load, which induce physiologic changes that affect the rehydration process.71–73 Fluid intake
increases substantially when ambient temperature rises above 25°C; the rehydration stimulus can also be psychological. An athlete exercising in the heat will voluntarily ingest more fluid if it is chilled. Individual differences in learned behavior also play a role in the rehydration process. An athlete who knows that rehydrating enhances subsequent performance is more apt to consume fluid before significant dehydration occurs, so appropriate education of athletes is essential.

The physical characteristics of the rehydration beverage can dramatically influence fluid replacement. Salinity, color, sweetness, temperature, flavor, carbonation, and viscosity all affect how much an athlete drinks. Since most fluid consumed by athletes is with meals, the presence of ample fluid during meals and adequate amount of time to eat are critical to rehydration. When access to meals is limited, a CHO-electrolyte beverage will help maintain CHO and electrolyte intake along with hydration status.

Other factors that contribute to fluid replacement include the individual’s mood (calmness is associated with enhanced rehydration) and the degree of concentration required by the task. For example, industrial laborers need frequent breaks to rehydrate because they must remain focused on a specific task. This need for concentration may explain why many elite mountain bikers use a convenient back-mounted hydration system instead of the typical rack-mounted water bottle. The back-mounted water reservoir may allow the cyclist to enhance rehydration while remaining focused on terrain, speed, gears, braking, and exertion. Accessibility to a fluid and ease of drinking may explain why athletes consume more fluid while cycling compared with running in a simulated duathlon.

Hydration before Exercise. An athlete should begin exercising well hydrated. Many athletes who perform repeated bouts of exercise on the same day or on consecutive days can become chronically dehydrated. When a hypohydrated athlete begins to exercise, physiologic mechanisms are compromised and the extent of the dysfunction is related to the degree of thermal stress experienced by the athlete. Athletes may require substantial assistance in obtaining fluids as evidenced by the phenomena of voluntary (when individuals drink insufficient quantities to replace fluid losses) and involuntary dehydration.

Athletes should ingest 500 mL of fluid 2 hours before the event (which allows ample time to urinate excess fluid) to ensure proper hydration and physiologic function at the onset of exercise. Mandatory pre-exercise hydration is physiologically advantageous and more effective than hydration dictated by often insufficient personal preference. Ingesting a nutritionally balanced diet and fluids during the 24 hours before an exercise session is also crucial. Increasing CHO intake before endurance activity may be beneficial for performance and may even enhance performance for activities as short as 10 minutes, but it may have a limited effect on resistance exercise.

There has been recent interest in potential benefits of purposefully overhydrating before exercise to postpone the onset of water deficit. While an enhanced hydration state is often reported with glycerol use, this does not always translate into a performance improvement. A recent study found increased exercise time and plasma volume during exercise to exhaustion in the heat when subjects were rehydrated with water and glycerol before exercise as compared with rehydration using an equal volume of water without glycerol. However, another study found no benefits of glycerol ingestion when the ensuing exercise took place in a thermoneutral environment. Hyperhydrating before exercise, even without glycerol, may enhance thermoregulatory function and limit the performance decrements normally noted with dehydration while exercising in the heat (WBGT > 25°C). A key point is that the benefits associated with glycerol use seem to be negated when proper hydration status is maintained during exercise. However, many athletes are unable to maintain hydration, so hyperhydration may be beneficial in extreme conditions when fluid intake cannot match sweat loss.

Rehydration during Exercise. Proper hydration during exercise will influence cardiovascular function, thermoregulatory function, muscle functioning, fluid volume status, and exercise performance. This topic has been extensively reviewed through the years, but some recent compilations are especially notable. Proper hydration during exercise enhances heat dissipation (increased skin blood flow and sweating rate), limits plasma hypertonicity, and helps sustain cardiac output. The enhanced evaporative cooling that can occur (due to increased skin blood flow and maintained perfusion of working muscles) is the result of sustained cardiac filling pressure. Rehydration during exercise conserves the centrally circulating fluid volume and allows maximal physiologic responses to intense exercise in the heat.

Two important purposes of rehydration are to decrease the rate of hyperthermia and to maintain athletic performance. A classic study showed that changes in rectal temperature during exercise depended on the degree of fluid intake. When water intake equaled sweat loss, rise in core temperature was slowest when compared with ad libidum water and no-water groups. This benefit of rehydration on thermoregulatory function is likely due to increased blood volume, reduced hyperosmolality, reduced cellular dehydration, and improved maintenance of extravascular fluid volume. Some studies have not shown a physiologic or performance benefit when rehydration occurred during a 1-hour intense exercise session in mild environmental conditions. The likely reason for a lack of benefit in these studies was the fact that the exercise session did not elicit enough sweat loss to cross the physiologic threshold of percentage of body weight loss (eg, −2%) that would negatively influence performance and physiologic function. For example, in 1 of the studies, the subjects had only lost 1.5% of body weight at the completion of the exercise session.

Athletes generally do not rehydrate to pre-exercise levels during exercise due to personal choice, fluid availability, the circumstances of competition, or a combination of these factors. Athletes should aim to drink quantities equal to sweat and urine losses, and while they rarely meet this goal, athletes can readily handle these large volumes (> 1 L/h). Additionally, athletes may not need to exactly match fluid intake with sweat loss to maintain water balance given the small contribution of water from metabolic processes. Appealing to individual taste preferences may encourage athletes to drink more fluids. In addition, including CHOs and electrolytes (especially sodium and potassium) in the rehydration drink can maintain blood glucose, CHO oxidation, and electrolyte balance and can maintain performance.

if the exercise session exceeds about 50 minutes in duration. Also, recent evidence indicates that athletes performing extremely intense intermittent activity with total exercise times of less than 50 minutes may benefit from ingestion of CHOs in the rehydration beverage.

Rates of gastric emptying and intestinal absorption should also be considered. Fluid volume, fluid calorie content, fluid osmolality, exercise intensity, environmental stress, and fluid temperature are some of the most important factors in determining the rates of gastric emptying and small intestine absorption (the small intestine is the primary site of fluid absorption). The single most important variable may be the volume of fluid in the stomach. Maintaining 400 to 600 mL of fluid in the stomach (or the maximum tolerated) will optimize gastric emptying. If CHOs are included in the fluid, the concentration should be 4% to 8%. Concentrations higher than 8% slow the rate of fluid absorption. Intense exercise (>80% of VO₂ max) may also decrease the rate of gastric emptying. Frequent ingestion (every 15 to 20 minutes) of a moderate fluid volume (200 mL) may be ideal, but it is not feasible in sports with extended periods between breaks. The rates of gastric emptying and intestinal absorption likely influence the speed of movement of the ingested fluids into the plasma volume. Since the gastric emptying and intestinal absorption rates are not compromised with the addition of a 6% carbohydrate solution as compared with water, fluid replacement and energy replenishment are equally achievable. The rate of gastric emptying is slowed by significant dehydration (>4%), which complicates rehydration and may increase gastrointestinal discomfort. Regardless, rehydration will still benefit the athlete’s hydration status.

Rehydration during exercise is also influenced by the state of acclimatization of the athlete. Heat acclimatization is achieved after 5 to 10 days of training in a hot environment and will increase sweat rate, decrease electrolyte losses in the sweat, and allow athletes to better tolerate exercise in the heat. Heat acclimatization modestly increases rehydration needs due to greater sweating. Fortunately, an athlete who is heat acclimatized has fewer deficits associated with dehydration and tends to be a “better” voluntary drinker (ingests fluid earlier and more often).

An athlete who exercises for more than 4 hours and hydrates excessively (well beyond sweat loss) only with water or low-solute beverages may be susceptible to a relatively rare condition known as symptomatic hyponatremia (also known as water intoxication). Ultimately, the body cannot excrete the consumed fluid rapidly enough to prevent intracellular swelling, which is sufficient to produce neuropsychological manifestations. Patients present with serum sodium levels below 130 to 135 mmol/L, and the sequelae of hyponatremia can result in death if not treated. The condition can most likely be avoided if sodium is consumed with the rehydration beverage and if fluid intake does not exceed sweat losses.

Every athlete will benefit from attempting to match intake with sweating rate and urine losses. Individual differences exist for gastric emptying and availability of fluids during particular sports. Rehydration procedures should be tested in practice and individually modified to maximize performance in competition.

**Rehydration after Exercise.** Replenishing fluid volume and glycogen stores is critical in the recovery of many body processes, including the cardiovascular, thermoregulatory, and metabolic activities.

Based on volume and osmolality, the best fluid to drink after exercise to replace the fluids that are lost via sweating may not be water. Consuming water alone decreases osmolality, which limits the drive to drink and slightly increases urine output. Including sodium in the rehydration beverage (or diet) allows fluid volume to be better conserved and increases the drive to drink. Including CHOs in the rehydration solution may improve the rate of intestinal absorption of sodium and water and replenishes glycogen stores. Replenishing glycogen stores can enhance performance in subsequent exercise sessions and may enhance immune function. While a normal diet commonly restores proper electrolyte concentrations, many athletes are forced to rehydrate between exercise sessions in the absence of meals. In addition, some athletes’ meals are eaten as long as 6 hours after an exercise session, which may compromise electrolyte availability during rehydration after intense exercise in hot conditions.

While replenishing fluid to equal sweating losses is often recommended, this formula does not replace urine losses. Ingestion equal to 150% of weight loss resulted in optimal rehydration 6 hours after exercise.

**Assessment of Hydration Status.** Body weight changes, urine color, subjective feelings, and thirst, among other indicators, offer cues to the need for rehydration. When preparing for an event, an athlete should know the sweat rate, assess current hydration status, and develop a rehydration plan. Determinations of sweat rate can be made. Hydration status can be assessed by measuring body weight before and after exercise sessions; monitoring urine color, USG, or urine volume; or using a combination of these factors. A urine color chart is included in this manuscript (Figure). The general indexes of hydration status are provided in Table 3. A refractometer offers a precise reading of USG and can be used as a general indicator of hydration state. A reading of less than 1.010 reflects a well-hydrated condition, while a reading of more than 1.020 reflects dehydration. Urine osmolality and urine conductivity may also be useful tools in assessing hydration status.

The hydration plan should take into account the length of the event, the individual’s sweat rate, exercise intensity, the temperature and humidity, and the availability of fluids (is fluid constantly available, as in cycling, or is it consumed in a large bolus during a break?). Habits of the coach or athlete, or both, may need to be altered in order to maximize the hydration process. Any plan for rehydrating during competition should be instituted and perfected during practice sessions; it should also be individually implemented, given the large variation among people in what constitutes a “comfortable” amount of rehydration.

**Composition of Rehydration Fluid.** During exercise, the body uses 30 to 60 g of CHOs per hour that need to be replaced to maintain CHO oxidation and delay the onset of glycogen depletion fatigue. Thus, including 60 g of CHOs in 1 L of fluid will not hinder fluid absorption and provides an adequate supply of CHOs during or while recovering from an exercise bout. The CHO concentration in the ideal fluid-replacement solution should be in the range of to 6% to 8% (g/100 mL). The simple sugars, glucose or sucrose in simple or polymer form, are the best additives to the replacement
fluid. Absorption is maximized if multiple forms of CHO are ingested simultaneously (ie, fluid is absorbed more quickly from the intestine if both glucose and fructose are present than if only glucose is present).107,114,206 The amount of fructose in the beverage should be limited to about 2% to 3% (2 to 3 g/100 mL of the beverage), since larger quantities may play a role in decreasing rates of absorption and oxidation and causing gastrointestinal distress.107,207 Ultimately, CHO composition depends on the relative need to replace fluids or CHOs. During events, when a high rate of fluid intake is necessary to sustain hydration, the CHO composition should be kept low (eg, <7%) to optimize gastric emptying and fluid absorption. During conditions when high rates of fluid replacement are not as necessary (ie, during recovery from an exercise session, mild environmental conditions, etc), the carbohydrate concentration can be increased to optimize CHO delivery with minimal risk of jeopardizing the hydration status.

Small quantities of sodium may enhance palatability and retention, stimulate thirst, and prevent hyponatremia in a susceptible individual.* Sodium concentration should be approximately 0.3 to 0.7 g/L.72,80,108,157,208 Other valuable sources of practical information concerning the composition of rehydration beverages and rehydration in general are available.†

Recognizing Dehydration in Athletes. The early signs and symptoms of dehydration include thirst and general discomfort and complaints. These are followed by flushed skin, weariness, cramps, and apathy. At greater water deficits, dizziness, headache, vomiting, nausea, heat sensations on the head or neck, chills, decreased performance, and dyspnea may be present.5,79,211,212 The degree of dehydration, the mental status, and the general medical condition of the athlete will dictate the mode, amount, type, and rate of rehydration. Identifying the early signs of dehydration can limit the onset or degree of an exertional heat illnesses.5,79,211,212 A comprehensive review of the prevention, identification, and treatment of the exertional heat illness can be found in the position stands by the NATA and the American College of Sports Medicine.211,213

Event Management. Some events are conducted under environmental conditions that are extreme and force the athlete to reduce intensity or risk a heat illness. These hazardous heat stresses can be avoided by scheduling athletic events during the coolest part of the day or a cooler time of the year.211,212 The reality of sport administration is that many events take place regardless of the environmental conditions. Individuals supervising an event in a hot humid environment must ensure that athletes have ample access to fluids, are encouraged to match fluid intakes with sweat losses, and are monitored for dehydration and exertional heat illness. Whenever possible, minimize the exercise intensity of athletes in the extreme heat, since this is the largest contributor to dehydration and heat illness. When successive exercise sessions occur on the same day or on ensuing days, hydration status, sleep, meals, and other factors that maximize performance and enhance safety should be maintained. Given the variety of events an athletic trainer may supervise, we cannot formulate an event management recommendation for all sports. However, the general concepts are interchangeable across sports and venues. For example, game modifications such as decreasing the length of play or inserting nontraditional water breaks (especially in youth sports and practice situations) will reduce the rate of heat illness. Closely monitoring environmental conditions via the WBGT or the heat index will allow an informed approach to hydration and sweat modification. Athletes who are educated on how to prevent and recognize dehydration are empowered to participate actively in implementing their own hydration protocols, thereby enhancing both performance and safety. The person responsible for the medical supervision of an event should have a detailed plan to address facilities, equipment, supplies, staffing, communication systems, education, and implementation of event policy.213,215–220

ACKNOWLEDGMENTS

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65. Caldwell JE, Ahonen E, Nousiainen U. Differential effects of sauna-,


Objective: To present recommendations for the prevention, recognition, and treatment of exertional heat illnesses and to describe the relevant physiology of thermoregulation.

Background: Certified athletic trainers evaluate and treat heat-related injuries during athletic activity in "safe" and high-risk environments. While the recognition of heat illness has improved, the subtle signs and symptoms associated with heat illness are often overlooked, resulting in more serious problems for affected athletes. The recommendations presented here provide athletic trainers and allied health providers with an integrated scientific and practical approach to the prevention, recognition, and treatment of heat illnesses. These recommendations can be modified based on the environmental conditions of the site, the specific sport, and individual considerations to maximize safety and performance.

Recommendations: Certified athletic trainers and other allied health providers should use these recommendations to establish on-site emergency plans for their venues and athletes. The primary goal of athlete safety is addressed through the prevention and recognition of heat-related illnesses and a well-developed plan to evaluate and treat affected athletes. Even with a heat-illness prevention plan that includes medical screening, acclimatization, conditioning, environmental monitoring, and suitable practice adjustments, heat illness can and does occur. Athletic trainers and other allied health providers must be prepared to respond in an expedient manner to alleviate symptoms and minimize morbidity and mortality.

Key Words: heat cramps, heat syncope, heat exhaustion, heat stroke, hyponatremia, dehydration, exercise, heat tolerance.

Heat illness is inherent to physical activity and its incidence increases with rising ambient temperature and relative humidity. Athletes who begin training in the late summer (eg, football, soccer, and cross-country athletes) experience exertional heat-related illness more often than athletes who begin training during the winter and spring.1-5 Although the hot conditions associated with late summer provide a simple explanation for this difference, we need to understand what makes certain athletes more susceptible and how these illnesses can be prevented.

PURPOSE

This position statement provides recommendations that will enable certified athletic trainers (ATCs) and other allied health providers to (1) identify and implement preventive strategies that can reduce heat-related illnesses in sports, (2) characterize factors associated with the early detection of heat illness, (3) provide on-site first aid and emergency management of athletes with heat illnesses, (4) determine appropriate return-to-play procedures, (5) understand thermoregulation and physiologic responses to heat, and (6) recognize groups with special concerns related to heat exposure.

ORGANIZATION

This position statement is organized as follows:
1. Definitions of exertional heat illnesses, including exercise-associated muscle (heat) cramps, heat syncope, exercise (heat) exhaustion, exertional heat stroke, and exertional hyponatremia;
2. Recommendations for the prevention, recognition, and treatment of exertional heat illnesses;
3. Background and literature review of the diagnosis of exertional heat illnesses; risk factors; predisposing medical conditions; environmental risk factors; thermoregulation, heat acclimatization, cumulative dehydration, and cooling therapies;
Table 1. Signs and Symptoms of Exertional Heat Illnesses

<table>
<thead>
<tr>
<th>Condition</th>
<th>Sign or Symptom*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercise-associated muscle (heat) cramps</td>
<td>Thirst, Sweating</td>
</tr>
<tr>
<td>Dehydration</td>
<td></td>
</tr>
<tr>
<td>Heat syncope</td>
<td>Tunnel vision, Pale or sweaty skin, Decreased pulse rate, Dizziness, Lightheadedness</td>
</tr>
<tr>
<td>Dehydration</td>
<td></td>
</tr>
<tr>
<td>Heat exhaustion</td>
<td>Normal or elevated body-core temperature, Dizziness, Lightheadedness, Syncope, Headache, Nausea, Anorexia, Diarrhea</td>
</tr>
<tr>
<td>Dehydration</td>
<td></td>
</tr>
<tr>
<td>Exercise (heat) exhaustion</td>
<td></td>
</tr>
<tr>
<td>High body-core temperature (&gt;40°C [104°F])</td>
<td></td>
</tr>
<tr>
<td>Central nervous system changes</td>
<td>Dizziness, Drowsiness, Irrational behavior, Confusion, Irritability, Emotional instability, Hysteria, Apathy, Aggressiveness, Delirium, Disorientation, Staggering, Seizures, Loss of consciousness, Coma, Dehydration, Weakness, Hot and wet or dry skin, Tachycardia (100 to 120 beats per minute), Hypotension, Hyperventilation, Vomiting, Diarrhea, Exertional hyponatremia</td>
</tr>
<tr>
<td>Exertional heat stroke</td>
<td></td>
</tr>
</tbody>
</table>

*Not every patient will present with all the signs and symptoms for the suspected condition.

4. Special concerns regarding exertional heat illnesses in prepubescent athletes, older athletes, and athletes with spinal-cord injuries;
5. Hospitalization and recovery from exertional heat stroke and resumption of activity after heat-related collapse; and
6. Conclusions.

DEFINITIONS OF EXERTIONAL HEAT ILLNESSES

The traditional classification of heat illness defines 3 categories: heat cramps, heat exhaustion, and heat stroke. However, this classification scheme omits several other heat- and activity-related illnesses, including heat syncope and exertional hyponatremia. The signs and symptoms of the exertional heat illnesses are listed in Table 1.

Heat illness is more likely in hot, humid weather but can occur in the absence of hot and humid conditions.

Exercise-Associated Muscle (Heat) Cramps

Exercise-associated muscle (heat) cramps represent a condition that presents during or after intense exercise sessions as an acute, painful, involuntary muscle contraction. Proposed causes include fluid deficiencies (dehydration), electrolyte imbalances, neuromuscular fatigue, or any combination of these factors.

Heat Syncope

Heat syncope, or orthostatic dizziness, can occur when a person is exposed to high environmental temperatures. This condition is attributed to peripheral vasodilation, postural pooling of blood, diminished venous return, dehydration, reduction in cardiac output, and cerebral ischemia. Heat syncope usually occurs during the first 5 days of acclimatization, before the blood volume expands, or in persons with heart disease or those taking diuretics. It often occurs after standing for long periods of time, immediately after cessation of activity, or after rapid assumption of upright posture after resting or being seated.

Exercise (Heat) Exhaustion

Exercise (heat) exhaustion is the inability to continue exercise associated with any combination of heavy sweating, dehydra-
tion, sodium loss, and energy depletion. It occurs most frequently in hot, humid conditions. At its worst, it is difficult to distinguish from exertional heat stroke without measuring rectal temperature. Other signs and symptoms include pallor, persistent muscular cramps, urge to defecate, weakness, fainting, dizziness, headache, hyperventilation, nausea, anorexia, diarrhea, decreased urine output, and a body-core temperature that generally ranges between 36°C (97°F) and 40°C (104°F).6,9,10,13,19

Exertional Heat Stroke

Exertional heat stroke is an elevated core temperature (usually >40°C [104°F]) associated with signs of organ system failure due to hyperthermia. The central nervous system neurologic changes are often the first marker of exertional heat stroke. Exertional heat stroke occurs when the temperature regulation system is overwhelmed due to excessive endogenous heat production or inhibited heat loss in challenging environmental conditions and can progress to complete thermoregulatory system failure.19,21 This condition is life threatening and can be fatal unless promptly recognized and treated. Signs and symptoms include tachycardia, hypotension, sweating (although skin may be wet or dry at the time of collapse), hyperventilation, altered mental status, vomiting, diarrhea, seizures, and coma.6,10,14 The risk of morbidity and mortality is greater the longer an athlete’s body temperature remains above 41°C (106°F) and is significantly reduced if body temperature is lowered rapidly.22–24

Unlike classic heat stroke, which typically involves prolonged heat exposure in infants, elderly persons, or unhealthy, sedentary adults in whom body heat-regulation mechanisms are inefficient,25–27 exertional heat stroke occurs during physical activity.28 The pathophysiology of exertional heat stroke is due to the overheating of organ tissues that may induce malfunction of the temperature-control center in the brain, circulatory failure, or endotoxemia (or a combination of these).29,30 Severe lactic acidosis (accumulation of lactic acid in the blood), hyperkalemia (excessive potassium in the blood), acute renal failure, rhabdomyolysis (destruction of skeletal muscle that may be associated with strenuous exercise), and disseminated intravascular coagulation (a bleeding disorder characterized by diffuse blood coagulation), among other medical conditions, may result from exertional heat stroke and often cause death.25

Exertional Hyponatremia

Exertional hyponatremia is a relatively rare condition defined as a serum-sodium level less than 130 mmol/L. Low serum-sodium levels usually occur when activity exceeds 4 hours.19 Two, often-additive mechanisms are proposed: an athlete ingests water or low-solute beverages well beyond sweat losses (also known as water intoxication), or an athlete’s sweat sodium losses are not adequately replaced.15–18 The low blood-sodium levels are the result of a combination of excessive fluid intake and inappropriate body water retention in the water-intoxication model and insufficient fluid intake and inadequate sodium replacement in the latter. Ultimately, the intravascular and extracellular fluid has a lower solute load than the intracellular fluids, and water flows into the cells, producing intracellular swelling that causes potentially fatal neurologic and physiologic dysfunction. Affected athletes present with a combination of disorientation, altered mental status, headache, vomiting, lethargy, and swelling of the extremities (hands and feet), pulmonary edema, cerebral edema, and seizures. Exertional hyponatremia can result in death if not treated properly. This condition can be prevented by matching fluid intake with sweat and urine losses and by rehydrating with fluids that contain sufficient sodium.31,32

RECOMMENDATIONS

The National Athletic Trainers’ Association (NATA) advocates the following prevention, recognition, and treatment strategies for exertional heat illnesses. These recommendations are presented to help ATCs and other allied health providers maximize health, safety, and sport performance as they relate to these illnesses. Athletes’ individual responses to physiologic stimuli and environmental conditions vary widely. These recommendations do not guarantee full protection from heat-related illness but should decrease the risk during athletic participation. These recommendations should be considered by ATCs and allied health providers who work with athletes at risk for exertional heat illnesses to improve prevention strategies and ensure proper treatment.

Prevention

1. Ensure that appropriate medical care is available and that rescue personnel are familiar with exertional heat illness prevention, recognition, and treatment. Table 2 provides general guidelines that should be considered.7 Ensure that ATCs and other health care providers attending practices or events are allowed to evaluate and examine any athlete who displays signs or symptoms of heat illness and have the authority to restrict the athlete from participating if heat illness is present.

2. Conduct a thorough, physician-supervised, preparticipation medical screening before the season starts to identify athletes predisposed to heat illness on the basis of risk factors and those who have a history of exertional heat illness.

3. Adapt athletes to exercise in the heat (acclimatization) gradually over 10 to 14 days. Progressively increase the intensity and duration of work in the heat with a combination of strenuous interval training and continuous exercise.6,9,14,33,37–44 Well-acclimatized athletes should train for 1 to 2 hours under the same heat conditions that will be present for their event.6,45,46 In a cooler environment, an athlete can wear additional clothing during training to induce or maintain heat acclimatization. Athletes should maintain proper hydration during the heat-acclimatization process.47

4. Educate athletes and coaches regarding the prevention, recognition, and treatment of heat illnesses and the risks associated with exercising in hot, humid environmental conditions.

5. Educate athletes to match fluid intake with sweat and urine losses to maintain adequate hydration.* (See the “National Athletic Trainers’ Association Position Statement: Fluid Replacement in Athletes.”52) Instruct athletes to drink sodium-containing fluids to keep their urine clear to light yellow to improve hydration and to replace fluids between practices on the same day and on successive days to maintain less than 2% body-weight change. These strategies will lessen the risk of acute and chronic dehydration and decrease the risk of heat-related events.

*References 9, 29, 37, 38, 40, 41, 43, 52–66.
Table 2. Prevention Checklist for the Certified Athletic Trainer*

1. Pre-event preparation
   - Am I challenging unsafe rules (e.g., ability to receive fluids, modify game and practice times)?
   - Am I encouraging athletes to drink before the onset of thirst and to be well hydrated at the start of activity?
   - Am I familiar with which athletes have a history of a heat illness?
   - Am I discouraging alcohol, caffeine, and drug use?
   - Am I encouraging proper conditioning and acclimatization procedures?

2. Checking hydration status
   - Do I know the preexercise weight of the athletes (especially those at high risk) with whom I work, particularly during hot and humid conditions?
   - Are the athletes familiar with how to assess urine color? Is a urine color chart accessible?
   - Do the athletes know their sweat rates and, therefore, know how much to drink during exercise?
   - Is a refractometer or urine color chart present to provide additional information regarding hydration status in high-risk athletes when baseline body weights are checked?

3. Environmental assessment
   - Am I regularly checking the wet-bulb globe temperature or temperature and humidity during the day?
   - Am I knowledgeable about the risk categories of a heat illness based on the environmental conditions?
   - Are alternate plans made in case risky conditions force rescheduling of events or practices?

4. Coaches’ and athletes’ responsibilities
   - Are coaches and athletes educated about the signs and symptoms of heat illnesses?
   - Am I double checking to make sure coaches are allowing ample rest and rehydration breaks?
   - Are modifications being made to reduce risk in the heat (e.g., decrease intensity, change practice times, allow more frequent breaks, eliminate double sessions, reduce or change equipment or clothing requirements, etc.)?
   - Are rapid weight-loss practices in weight-class sports adamantly disallowed?

5. Event management
   - Have I checked to make sure proper amounts of fluids will be available and accessible?
   - Are carbohydrate-electrolyte drinks available at events and practices (especially during twice-a-day practices and those that last longer than 50 to 60 minutes or are extremely intense in nature)?
   - Am I aware of the factors that may increase the likelihood of a heat illness?
   - Am I promptly rehydrating athletes to preexercise weight after an exercise session?
   - Are shaded or indoor areas used for practices or breaks when possible to minimize thermal strain?

6. Treatment considerations
   - Am I familiar with the most common early signs and symptoms of heat illnesses?
   - Do I have the proper field equipment and skills to assess a heat illness?
   - Is an emergency plan in place in case an immediate evacuation is needed?
   - Is a kiddy pool available in situations of high risk to initiate immediate cold-water immersion of heat-stroke patients?
   - Are ice bags available for immediate cooling when cold-water immersion is not possible?
   - Have shaded, air-conditioned, and cool areas been identified to use when athletes need to cool down, recover, or receive treatment?
   - Are fans available to assist evaporation when cooling?
   - Am I properly equipped to assess high core temperature (i.e., rectal thermometer)?

7. Other situation-specific considerations

*Adapted with permission from Casa.*

Table 3. Wet-Bulb Globe Temperature Risk Chart62–67

<table>
<thead>
<tr>
<th>WBGT</th>
<th>Flag Color</th>
<th>Level of Risk</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;18°C (&lt;65°F)</td>
<td>Green</td>
<td>Low</td>
<td>Risk low but still exists on the basis of risk factors</td>
</tr>
<tr>
<td>18–23°C (65–73°F)</td>
<td>Yellow</td>
<td>Moderate</td>
<td>Risk level increases as event progresses through the day</td>
</tr>
<tr>
<td>23–28°C (73–82°F)</td>
<td>Red</td>
<td>High</td>
<td>Everyone should be aware of injury potential; individuals at risk should not compete</td>
</tr>
<tr>
<td>&gt;28°C (82°F)</td>
<td>Black</td>
<td>Extreme or hazardous</td>
<td>Consider rescheduling or delaying the event until safer conditions prevail; if the event must take place, be on high alert</td>
</tr>
</tbody>
</table>

*Adapted with permission from Roberts.*

6. Encourage athletes to sleep at least 6 to 8 hours at night in a cool environment,41,35,50 eat a well-balanced diet that follows the Food Guide Pyramid and United States Dietary Guidelines,56–58 and maintain proper hydration status. Athletes exercising in hot conditions (especially during twice-a-day practices) require extra sodium from the diet or rehydration beverages or both.

7. Develop event and practice guidelines for hot, humid weather that anticipate potential problems encountered based on the wet-bulb globe temperature (WBGT) (Table 3) or heat and humidity as measured by a sling psychrometer (Figure 1), the number of participants, the nature of the activity, and other predisposing risk factors.14,51 If the WBGT is greater than 28°C (82°F), or “very high” as indicated in Table 3, Figure 1), an athletic event should be delayed, rescheduled, or moved into an air-conditioned space, if possible.69–74 It is important to note that these measures are based on the risk of environmental stress for athletes wearing shorts and a T-shirt; if an
athlete is wearing additional clothing (ie, football uniform, wetsuit, helmet), a lower WBGT value could result in comparable risk of environmental heat stress (Figure 2). If the event or practice is conducted in hot, humid conditions, then use extreme caution in monitoring the athletes and be proactive in taking preventive steps. In addition, be sure that emergency supplies and equipment are easily accessible and in good working order. The most important factors are to limit intensity and duration of activity, limit the amount of clothing and equipment worn, increase the number and length of rest breaks, and encourage proper hydration.

Modify activity under high-risk conditions to prevent exertional heat illnesses. Identify individuals who are susceptible to heat illnesses. In some athletes, the prodromal signs and symptoms of heat illnesses are not evident before collapse, but in many cases, adept medical supervision will allow early intervention.

8. Check the environmental conditions before and during the activity, and adjust the practice schedule accordingly.

Schedule training sessions to avoid the hottest part of the day (10 AM to 5 PM) and to avoid radiant heating from direct sunlight, especially in the acclimatization during the first few days of practice sessions.9,29,33,34,38,40,50,60

9. Plan rest breaks to match the environmental conditions and the intensity of the activity. Exercise intensity and environmental conditions should be the major determinants in deciding the length and frequency of rest breaks. If possible, cancel or postpone the activity or move it indoors (if air conditioned) if the conditions are “extreme or hazardous” (see Table 3) or “very high” (see Figure 1) or to the right of the circled line (see Figure 2). General guidelines during intense exercise would include a work:rest ratio of 1:1, 2:1, 3:1, and 4:1 for “extreme or hazardous” (see Table 3) or “very high” (see Figure 1), “high,” “moderate,” or “low” environmental risk, respectively. For activities such as football in which equipment must be considered, please refer to Figure 2 for equipment modifications and appropriate work:rest ratios for various environmental conditions. Rest breaks should occur in the shade if possible, and hydration during rest breaks should be encouraged.

10. Implement rest periods at mealtime by allowing 2 to 3 hours for food, fluids, nutrients, and electrolytes (sodium and potassium) to move into the small intestine and bloodstream before the next practice.34,50,77

11. Provide an adequate supply of proper fluids (water or sports drinks) to maintain hydration and institute a hydration protocol that allows the maintenance of hydration status. Fluids should be readily available and served in containers that allow adequate volumes to be ingested with ease and with minimal interruption of exercise. The goal should be to lose no more than 2% to 3% of body weight during the practice session (due to sweat and urine losses). See the “National Athletic Trainers’ Association Position Statement: Fluid Replacement in Athletes.”

12. Weigh high-risk athletes (in high-risk conditions, weigh all athletes) before and after practice to estimate the amount of body water lost during practice and to ensure a return to prepractice weight before the next practice. Following exercise athletes should consume approximately 1-1.25 L (16 oz) of fluid for each kilogram of body water lost during exercise.†

†References 6, 9, 29, 33, 38, 40, 49, 60, 77, 83.
13. Minimize the amount of equipment and clothing worn by the athlete in hot or humid (or both) conditions. For instance, a full football uniform prevents sweat evaporation from more than 60% of the body. Consult Figure 2 for possible equipment and clothing recommendations. When athletes exercise in the heat, they should wear loose-fitting, absorbent, and light-colored clothing; mesh clothing and new-generation cloth blends have been specially designed to allow more effective cooling.

14. Minimize warm-up time when feasible, and conduct warm-up sessions in the shade when possible to minimize the radiant heat load in “high” or “very high” or “extreme or hazardous” (see Table 3, Figure 1) conditions. Consult Figure 2 for recommendations regarding the appropriate composition of rehydration beverages based on the length and intensity of the activity.

15. Allow athletes to practice in shaded areas and use electric or cooling fans to circulate air whenever feasible.

16. Include the following supplies on the field, in the locker room, and at various other stations:

- A supply of cool water or sports drinks or both to meet the participants’ needs (see the “National Athletic Trainers’ Association Position Statement: Fluid Replacement in Athletes” for recommendations regarding the composition of rehydration beverages based on the length and intensity of the activity).
- Ice for active cooling (ice bags, tub cooling) and to keep beverages cool during exercise.
- Rectal thermometer to assess body-core temperature.
- Telephone or 2-way radio to communicate with medical personnel and to summon emergency medical transportation.
- Tub, wading pool, kiddy pool, or whirlpool to cool the trunk and extremities for immersion cooling therapy.
- Progressive cooling is the most critical factor in the treatment of exertional heat stroke. Circulation of the tub water may enhance cooling.

17. Notify local hospital and emergency personnel before mass participation events to inform them of the event and the increased possibility of heat-related illnesses.

18. Mandate a check of hydration status at weigh-in to ensure athletes in sports requiring weight classes (eg, wrestling, judo, rowing) are not dehydrated. Any procedures used to induce dramatic dehydration (eg, diuretics, rubber suits, exercising in a sauna) are strictly prohibited. Dehydrated athletes exercising at the same intensity as euvhydrated athletes are at increased risk for thermoregulatory strain (see the “National Athletic Trainers’ Association Position Statement: Fluid Replacement in Athletes”).

**Recognition and Treatment**

19. Exercise-associated muscle (heat) cramps:

- An athlete showing signs or symptoms including dehydration, thirst, sweating, transient muscle cramps, and fatigue is likely experiencing exercise-associated muscle (heat) cramps.
- To relieve muscle spasms, the athlete should stop activity, replace lost fluids with sodium-containing fluids, and begin mild stretching with massage of the muscle spasm.
- Fluid absorption is enhanced with sports drinks that contain sodium. A high-sodium sports product may be added to the rehydration beverage to prevent or relieve cramping in athletes who lose large amounts of sodium in their sweat. A simple salted fluid consists of two 10-grain salt tablets dissolved in 1 L (34 oz) of water. Intravenous fluids may be required if nausea or vomiting limits oral fluid intake; these must be ordered by a physician.
- A recumbent position may allow more rapid redistribution of blood flow to cramping leg muscles.

20. Heat syncope:

- If an athlete experiences a brief episode of fainting associated with dizziness, tunnel vision, pale or sweaty skin, and a decreased pulse rate but has a normal rectal temperature (for exercise, 36°C to 40°C [97°F to 104°F]), then heat syncope is most likely the cause.
- Move the athlete to a shaded area, monitor vital signs, elevate the legs above the level of the head, and rehydrate.

21. Exercise (heat) exhaustion:

- Cognitive changes are usually minimal, but assess central nervous system function for bizarre behavior, hallucinations, altered mental status, confusion, disorientation, or coma (see Table 1) to rule out more serious conditions.
- If feasible, measure body-core temperature (rectal temperature) and assess cognitive function (see Table 1) and vital signs. Rectal temperature is the most accurate method possible in the field to monitor body-core temperature. The ATC should not rely on the oral, tympanic, or axillary temperature for athletes because these are inaccurate and ineffective measures of body-core temperature during and after exercise.
- If the athlete’s temperature is elevated, remove his or her excess clothing to increase the evaporative surface and to facilitate cooling.
- Cool the athlete with fans, ice towels, or ice bags because these may help the athlete with a temperature of more than 38.8°C (102°F) to feel better faster.
- Remove the athlete to a cool or shaded environment if possible.
- Start fluid replacement.
- Transfer care to a physician if intravenous fluids are needed or if recovery is not rapid and uneventful.

22. Exertional heat stroke:

- Measure the rectal temperature if feasible to differentiate between heat exhaustion and heat stroke. With heat stroke, rectal temperature is elevated (generally higher than 40°C [104°F]).
- Assess cognitive function, which is markedly altered in exertional heat stroke (see Table 1).
- Lower the body-core temperature as quickly as possible. The fastest way to decrease body temperature is to remove clothes and equipment and immerse the body (trunk and extremities) into a pool or tub of cold water (approximately 1°C to 15°C [35°F to 59°F]). Aggressive cooling is the most critical factor in the treatment of exertional heat stroke. Circulation of the tub water may enhance cooling.
- Monitor the temperature during the cooling therapy and recovery (every 5 to 10 minutes). Once the athlete’s rectal temperature reaches approximately 38.3°C to 38.9°C (101°F to 102°F), he or she should be removed from the pool or tub to avoid overcooling.
- If a physician is present to manage the athlete’s medical care on site, then initial transportation to a medical facility may not be necessary so immersion can continue uninterrupted.

†References 8, 9, 29, 33, 38, 40, 53, 59, 84–86.
If a physician is not present, aggressive first-aid cooling should be initiated on site and continued during emergency medical system transport and at the hospital until the athlete is normothermic.

- Activate the emergency medical system.
- Monitor the athlete’s vital signs and other signs and symptoms of heat stroke (see Table 1).34,95
- During transport and when immersion is not feasible, other methods can be used to reduce body temperature: removing the clothing; sponging down the athlete with cool water and applying cold towels; applying ice bags to as much of the body as possible, especially the major vessels in the armpit, groin, and neck; providing shade; and fanning the body with air.19
- In addition to cooling therapies, first-aid emergency procedures for heat stroke may include airway management. Also a physician may decide to begin intravenous fluid replacement.87
- Monitor for organ-system complications for at least 24 hours.

23. Exertional hyponatremia:

- Attempt to differentiate between hyponatremia and heat exhaustion. Hyponatremia is characterized by increasing headache, significant mental compromise, altered consciousness, seizures, lethargy, and swelling in the extremities. The athlete may be dehydrated, normally hydrated, or overhydrated.19
- Attempt to differentiate between hyponatremia and heat stroke. In hyponatremia, hyperthermia is likely to be less (rectal temperature less than 40°C [104°F]).19 The plasma-sodium level is less than 130 mEq/L and can be measured with a sodium analyzer on site if the device is available.
- If hyponatremia is suspected, immediate transfer to an emergency medical center via the emergency medical system is indicated. An intravenous line should be placed to administer medication as needed to increase sodium levels, induce diuresis, and control seizures.
- An athlete with suspected hyponatremia should not be administered fluids until a physician is consulted.

24. Return to activity

In cases of exercise-associated muscle (heat) cramps or heat syncope, the ATC should discuss the athlete’s case with the supervising physician. The cases of athletes with heat exhaustion who were not transferred to the physician’s care should also be discussed with the physician. After exertional heat stroke or exertional hyponatremia, the athlete must be cleared by a physician before returning to athletic participation.92 The return to full activity should be gradual and monitored.8,87

BACKGROUND AND LITERATURE REVIEW

Diagnosis

To differentiate heat illnesses in athletes, ATCs and other on-site health care providers must be familiar with the signs and symptoms of each condition (see Table 1). Other medical conditions (eg, asthma, status epilepticus, drug toxicities) may also present with similar signs and symptoms. It is important to realize, however, that an athlete with a heat illness will not exhibit all the signs and symptoms of a specific condition, increasing the need for diligent observation during athletic activity.

Nonenvironmental Risk Factors

Athletic trainers and other health care providers should be sensitive to the following nonenvironmental risk factors, which could place athletes at risk for heat illness.

- **Dehydration.** Sweating, inadequate fluid intake, vomiting, diarrhea, certain medications,89,101–103 and alcohol104,105 or caffeine106 use can lead to fluid deficit. Body-weight change is the preferred method to monitor for dehydration in the field, but a clinical refractometer is another accurate method (specific gravity should be no more than 1.020).34,49,107–110 Dehydration can also be identified by monitoring urine color or body-weight changes before, during, and after a practice or an event and across successive days.53,54

The signs and symptoms of dehydration are thirst, general discomfort, flushed skin, weariness, cramps, apathy, dizziness, headache, vomiting, nausea, heat sensations on the head or neck, chills, decreased performance, and dyspnea.32 Water loss that is not regained by the next practice increases the risk for heat illness.110

- **Barriers to Evaporation.** Athletic equipment and rubber or plastic suits used for “weight loss” do not allow water vapor to pass through and inhibit evaporative, convective, and radiant heat loss.111,112 Participants who wear equipment that does not allow for heat dissipation are at an increased risk for heat illness.113 Helmets are also limiting because a significant amount of heat is dissipated through the head.

- **Illness.** Athletes who are currently or were recently ill may be at an increased risk for heat illness because of fever or dehydration.114–116

- **History of Heat Illness.** Some individuals with a history of heat illness are at greater risk for recurrent heat illness.8,117

- **Increased Body Mass Index (Thick Fat Layer or Small Surface Area).** Obese individuals are at an increased risk for heat illness because the fat layer decreases heat loss.118 Obese persons are less efficient and have a greater metabolic heat production during exercise. Conversely, muscle-bound individuals have increased metabolic heat production and a lower ratio of surface area to mass, contributing to a decreased ability to dissipate heat.119–121

- **Hot-Bulb Globe Temperature on Previous Day and Night.** When the WBGT is high to extreme (see Table 3), the risk of heat-related problems is greater the next day; this appears to be one of the best predictors of heat illness.121 Athletes who sleep in cool or air-conditioned quarters are at less risk.

- **Poor Physical Condition.** Individuals who are untrained are more susceptible to heat illness than are trained athletes. As the VO_{2}max of an individual improves, the ability to withstand heat stress improves independent of acclimatization and heat adaptation.122 High-intensity work can easily produce 1000 kcal/h and elevate the core temperature of at-risk individuals (those who are unfit, overweight, or unacclimatized) to dangerous levels within 20 to 30 minutes.123

- **Excessive or Dark-Colored Clothing or Equipment.** Excessive clothing or equipment decreases the ability to thermoregulate, and dark-colored clothing or equipment may cause a greater absorption of heat from the environment. Both should be avoided.113

- **Overzealousness.** Overzealous athletes are at a higher risk for heat illness because they override the normal behavioral adaptations to heat and decrease the likelihood of subtle cues being recognized.
Lack of Acclimatization to Heat. An athlete with no or minimal physiologic acclimatization to hot conditions is at an increased risk of heat-related illness. 8,37,83,124

Medications and Drugs. Athletes who take certain medications or drugs, particularly medications with a dehydrating effect, are at an increased risk for a heat illness. 101–106,125–136 Alcohol, caffeine, and theophylline at certain doses are mild diuretics. 106,137,138 Caffeine is found in coffee, tea, soft drinks, chocolate, and several over-the-counter and prescription medications. 139 Theophylline is found mostly in tea and anti-asthma medications. 140

Electrolyte Imbalance. Electrolyte imbalances do not usually occur in trained, acclimatized individuals who engage in physical activity and eat a normal diet. 141 Most sodium and chloride losses in athletes occur through the urine, but athletes who sweat heavily, are salty sweaters, or are not heat acclimatized can lose significant amounts of sodium during activity. 142 Electrolyte imbalances often contribute to heat illness in older athletes who use diuretics. 143,144

Predisposing Medical Conditions

The following predisposing medical conditions add to the risk of heat illness.

Malignant Hyperthermia. Malignant hyperthermia is caused by an autosomal dominant trait that causes muscle rigidity, resulting in elevation of body temperature due to the accelerated metabolic rate in the skeletal muscle. 145–147

Neuroleptic Malignant Syndrome. Neuroleptic malignant syndrome is associated with the use of neuroleptic agents and antipsychotics and an unexpected idopathic increase in core temperature during exercise. 148–151

Arteriosclerotic Vascular Disease. Arteriosclerotic vascular disease compromises cardiac output and blood flow through the vascular system by thickening the arterial walls. 115,152

Scleroderma. Scleroderma is a skin disorder that decreases sweat production, thereby decreasing heat transfer. 149,153

Cystic Fibrosis. Cystic fibrosis causes increased salt loss in sweat and can increase the risk for hyponatremia. 154,155

Sickle Cell Trait. Sickle cell trait limits blood flow distribution and decreases oxygen-carrying capacity. The condition is exacerbated by exercise at higher altitudes. 156,157

Environmental Risk Factors

When the environmental temperature is above skin temperature, athletes begin to absorb heat from the environment and depend entirely on evaporation for heat loss. 113,158,159 High relative humidity inhibits heat loss from the body through evaporation. 61

The environmental factors that influence the risk of heat illness include the ambient air temperature, relative humidity (amount of water vapor in the air), air motion, and the amount of radiant heat from the sun or other sources. 2,9,41 The relative risk of heat illness can be calculated using the WBGT equation. 2,43,50,69,77,160,161 Using the WBGT index to modify activity in high-risk settings has virtually eliminated heat-stroke deaths in United States Marine Corps recruits. 159 Wet-bulb globe temperature is calculated using the wet-bulb (wb), dry-bulb (db), and black-globe (bg) temperature with the following equation 59,62,85,162,163:

\[ \text{WBGT} = 0.7T_{wb} + 0.2T_{bg} + 0.1T_{db} \]

When there is no radiant heat load, \( T_{db} = T_{bg} \), and the equation is reduced 62 to

\[ \text{WBGT} = 0.7T_{wb} + 0.3T_{db} \]

This equation is used to estimate risk as outlined in Table 3. 11,35,40,50,61,85 This index was determined for athletes wearing a T-shirt and light pants. 158 The WBGT calculation can be performed using information obtained from electronic devices 42 or the local meteorologic service, but conversion tables for relative humidity and \( T_{db} \) are needed to calculate the wet-bulb temperature. 50,162 The predictive value from the meteorologic service is not as accurate as site-specific data for representing local heat load but will suffice in most situations. When WBGT measures are not possible, environmental heat stress can be estimated using a sling psychrometer (see Figures 1, 2).

Several recommendations have been published for distance running, but these can also be applied to other continuous activity sports. The Canadian Track and Field Association recommended that a distance race should be cancelled if the WBGT is greater than 26.7°C (80°F). 39 The American College of Sports Medicine guidelines from 1996 recommended that a race should be delayed or rescheduled when the WBGT is greater than 27.8°C (82°F). 31,72,73 In some instances, the event will go on regardless of the WBGT; ATCs should then have an increased level of suspicion for heat stroke and focus on hydration, emergency supplies, and detection of exertional heat illnesses.

Thermoregulation

Thermoregulation is a complex interaction among the central nervous system (CNS), the cardiovascular system, and the skin to maintain a body-core temperature of 37°C. 9,43,51,164 The CNS temperature-regulation center is located in the hypothalamus and is the site where the core temperature setpoint is determined. 9,43,82,158,164–166 The hypothalamus receives information regarding body-core and shell temperatures from peripheral skin receptors and the circulating blood; body-core temperature is regulated through an open-ended feedback loop similar to that in a home thermostat. 158,165,167,168 Body responses for heat regulation include cutaneous vasodilation, increased sweating, increased heart rate, and increased respiratory rate. 38,43,51,164,165

Body-core temperature is determined by metabolic heat production and the transfer of body heat to and from the surrounding environment using the following heat-production and heat-storage equation 166,167:

\[ S = M + R \pm K \pm Cv - E \]

where \( S \) is the amount of stored heat, \( M \) is the metabolic heat production, \( R \) is the heat gained or lost by radiation, \( K \) is the conductive heat lost or gained, \( Cv \) is the convective heat lost or gained, and \( E \) is the evaporative heat lost.

Basal metabolic heat production fasting and at absolute rest is approximately 60 to 70 kcal/h for an average adult, with 50% of the heat produced by the internal organs. Metabolic heat produced by intense exercise may approach 1000 kcal/h. 51,164 with greater than 90% of the heat resulting from muscle metabolism. 9,40,42,166

Heat is gained or lost from the body by one or more of the following mechanisms 9,85:
Radiation. The energy is transferred to or from an object or body via electromagnetic radiation from higher to lower energy surfaces.9,43,51,85,166

Conduction. Heat transfers from warmer to cooler objects through direct physical contact.9,43,51,85,166 Ice packs and cold-water baths are examples of conductive heat exchange.

Convection. Heat transfers to or from the body to surrounding moving fluid (including air).9,43,51,85,166 Moving air from a fan, cycling, or windy day produces convective heat exchange.

Evaporation. Heat transfers via the vaporization of sweat§ and is the most efficient means of heat loss.51,158,169 The evaporation of sweat from the skin depends on the water saturation of the air and the velocity of the moving air.170–172 The effectiveness of this evaporation for heat loss from the body diminishes rapidly when the relative humidity is greater than 60%.9,20,164

Cognitive performance and associated CNS functions deteriorate when brain temperature rises. Signs and symptoms include dizziness, confusion, behavior changes, coordination difficulties, decreased physical performance, and collapse due to hyperthermia.168,173 The residual effects of elevated brain temperature depend on the duration of the hyperthermia. Heat stroke rarely leads to permanent neurologic deficits; however, some sporadic symptoms of frontal headache and sleep disturbances have been noted for up to 4 months.168,174,175 When permanent CNS damage occurs, it is associated with cerebellar changes, including ataxia, marked dysarthria, and dysmetria.174

Heat Acclimatization

Heat acclimatization is the physiologic response produced by repeated exposures to hot environments in which the capacity to withstand heat stress is improved.14,43,75,176,177 Physiologic responses to heat stress are summarized in Table 4. Exercise heat exposure produces progressive changes in thermoregulation that involve sweating, skin circulation, thermoregulatory setpoint, cardiovascular alterations, and endocrine adjustments.29,43,178 Individual differences affect the onset and decay of acclimatization.29,45,179 The rate of acclimatization is related to aerobic conditioning and fitness; more conditioned athletes acclimatize more quickly.43,45,180 The acclimatization process begins with heat exposure and is reasonably protective after 7 to 14 days, but maximum acclimatization may take 2 to 3 months.45,181,182 Heat acclimatization diminishes by day 6 when heat stress is no longer present.180,183 Fluid replacement improves the induction and effect of heat acclimatization.184–187 Extra salt in the diet during the first few days of heat exposure also improves acclimatization; this can be accomplished by encouraging the athlete to eat salty foods and to use the salt shaker liberally during meals.

Cumulative Dehydration

Cumulative dehydration develops insidiously over several days and is typically observed during the first few days of a season during practice sessions or in tournament competition. Cumulative dehydration can be detected by monitoring daily prepractice and postpractice weights. Even though a small decrease in body weight (less than 1%) may not have a detrimental effect on the individual, the cumulative effect of a 1% fluid loss per day occurring over several days will create an increased risk for heat illness and a decrease in performance.110

During intense exercise in the heat, sweat rates can be 1 to 2.5 L/h (about 1 to 2.25 kilograms [2 to 5 pounds] of body weight per hour) or more, resulting in dehydration. Unfortunately, the volume of fluid that most athletes drink voluntarily during exercise replaces only about 50% of body-fluid losses.188 Ideally, rehydration involves drinking at a rate sufficient to replace all of the water lost through sweating and urination.60,77 If the athlete is not able to drink at this rate, he or she should drink the maximum tolerated. Use caution to ensure that athletes do not overhydrate and put themselves at risk for the development of hyponatremia. However, hydration before an event is essential to help decrease the incidence of heat illnesses. For more information on this topic, see the “National Athletic Trainers’ Association Position Statement: Fluid Replacement in Athletes.”52

Cooling Therapies

The fastest way to decrease body-core temperature is immersion of the trunk and extremities into a pool or tub filled with cold water (between 1°C [35°F] and 15°C [59°F]).39,88,91,97 Conditions that have been associated with immersion therapy include shivering and peripheral vasoconstriction; however, the potential for these should not deter the medical staff from using immersion therapy for rapid cooling. Shivering can be prevented if the athlete is removed from the water once rectal temperature reaches 38.3°C to 38.9°C (101°F to 102°F). Peripheral vasoconstriction may occur, but the powerful cooling potential of immersion outweighs any potential concerns. Cardiogenic shock has also been a proposed consequence of immersion therapy, but this connection has not been proven in cooling heat-stroke patients.39 Cold-water immersion therapy was associated with a zero percent fatality rate in 252 cases of exertional heat stroke in the military.89 Other forms of cooling (water spray; ice packs covering the body; ice packs on axillae, groin, and neck; or blowing air) decrease body-core temperature at a slower rate compared with cold-water im-
mersion. If immersion cooling is not being used, cooling with ice bags should be directed to as much of the body as possible, especially the major vessels in the armpit, groin, and neck regions (and likely the hands and feet), and cold towels may be applied to the head and trunk because these areas have been demonstrated on thermography to have the most rapid heat loss.

**SPECIAL CONCERNS**

Most research related to heat illness has been performed on normal, healthy adults. Child athletes, older athletes, and athletes with spinal-cord injuries have been studied less frequently. The following are suggestions for special populations or those with special conditions.

**Children (Prepubescents)**

Exercise in hot environments and heat tolerance are affected by many physiologic factors in children. These include decreased sweat gland activity, higher skin temperatures, decreased cardiac output (increased heart rate and lower stroke volume) due to increased peripheral circulation, decreased exercise economy, decreased ability to acclimatize to heat (slower and takes longer), smaller body size (issues related to body surface-to-mass ratio), maturational differences, and predisposing conditions (obesity, hypohydration, childhood illnesses, and other disease states).

- Decrease the intensity of activities that last longer than 30 minutes and have the athlete take brief rests if the WBGT is between 22.8°C and 27.8°C (73°F and 82°F); cancel or modify the activity if the WBGT is greater than 27.8°C (82°F). Modification could involve longer and more frequent rest breaks than are usually permitted within the rules of the sport.
- Encourage children to ingest some fluids at least every 15 to 30 minutes during activity to maintain hydration, even if they are not thirsty.
- Use similar precautions as listed earlier for adults.

**Older Athletes (≥50 Years Old)**

The ability of the older athlete to adapt is partly a function of age and also depends on functional capacity and physiologic health status.

- The athlete should be evaluated by a physician before exercise, with the potential consequences of predisposing medical conditions and illnesses addressed. An increase has been shown in the exercise heart rate of 1 heat per minute for each 1°C (1.8°F) increase in ambient temperature above neutral (23.9°C [75°F]). Athletes with known or suspected heart disease should curtail activities at lower temperatures than healthy athletes and should have cardiovascular stress testing before participating in hot environments.
- Older athletes have a decreased ability to maintain an adequate plasma volume and osmolality during exercise, which may predispose them to dehydration. Regular fluid intake is critical to avoid hyperthermia.

**Athletes with Spinal-Cord Injuries**

As sport participation for athletes with spinal-cord injuries increases from beginner to elite levels, understanding the disability, training methods, and causes of heat injury will help make competition safer. For example, the abilities to regulate heart rate, circulate the blood volume, produce sweat, and transfer heat to the surface vary with the level and severity of the spinal-cord lesion.

- Monitor these athletes closely for heat-related problems. One technique for determining hyperthermia is to feel the skin under the arms of the distressed athlete. Rectal temperature may not be as accurate for measuring core temperature as in other athletes due to decreased ability to regulate blood flow beneath the spinal-cord lesion.
- If the athlete is hyperthermic, provide more water, lighter clothing, or cooling of the trunk, legs, and head.

**HOSPITALIZATION AND RECOVERY**

After an episode of heat stroke, the athlete may experience impaired thermoregulation, persistent CNS dysfunction, hepatic insufficiency, and renal insufficiency. For persons with exertional heat stroke and associated multisystem tissue damage, the rate of recovery is highly individualized, ranging up to more than 1 year. In one study, 9 of 10 patients exhibited normal heat-acclimatization responses, thermoregulation, whole-body sodium and potassium balance, sweat-gland function, and blood values about 2 months after the heat stroke. Transient or persistent heat intolerance was found in a small percentage of patients. Some athletes, a history of exertional heat stroke increases the chance of experiencing subsequent episodes.

An athlete who experiences heat stroke may have compromised heat tolerance and heat acclimatization after physician clearance. Decreased heat tolerance may affect 15% to 20% of persons after a heat-stroke-related collapse and in a few individuals, decreased heat tolerance has persisted up to 5 years. Additional heat stress may reduce the athlete’s ability to train and compete due to impaired cardiovascular and thermoregulatory responses. After recovery from an episode of heat stroke or hypohydremia, an athlete’s physical activity should be restricted and the gradual return to sport individualized by his or her physician. The athlete should be monitored on a daily basis by the ATC during exercise. During the return-to-exercise phase, an athlete may experience some detaining and deconditioning not directly related to the heat exposure. Evaluate the athlete over time to determine whether there has been a complete recovery of exercise and heat tolerance.

**CONCLUSIONS**

Athletic trainers and other allied health providers must be able to differentiate exercise-associated muscle (heat) cramps, heat syncope, exercise (heat) exhaustion, exertional heat stroke, and exertional hypohydremia in athletes.

This position statement outlines the NATA’s current recommendations to reduce the incidence, improve the recognition, and optimize treatment of heat illness in athletes. Education and increased awareness will help to reduce both the frequency and the severity of heat illness in athletes.

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National Athletic Trainers’ Association Position Statement: Environmental Cold Injuries

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Objective: To present recommendations for the prevention, recognition, and treatment of environmental cold injuries.

Background: Individuals engaged in sport-related or work-related physical activity in cold, wet, or windy conditions are at risk for environmental cold injuries. An understanding of the physiology and pathophysiology, risk management, recognition, and immediate care of environmental cold injuries is an essential skill for certified athletic trainers and other health care providers working with individuals at risk.

Recommendations: These recommendations are intended to provide certified athletic trainers and others participating in athletic health care with the specific knowledge and problem-solving skills needed to address environmental cold injuries. Each recommendation has been graded (A, B, or C) according to the Strength of Recommendation Taxonomy criterion scale.

Key Words: environmental physiology, hypothermia, frostbite, frostnip, chilblain, pernio, immersion foot, trench foot

PURPOSES

This position statement includes a review of available literature, definitions of cold injuries, and a set of recommendations that will allow certified athletic trainers (ATs) and other allied health and medical providers to

1. Identify and employ prevention strategies to reduce cold-related injuries and illnesses in the physically active.
2. Describe factors associated with cold-related injuries and illnesses.
3. Provide on-site first aid and immediate care of cold-related injuries and illnesses.
4. Understand the thermoregulatory and physiologic responses to cold.
5. Identify groups with unique concerns related to cold exposure.

DEFINITIONS OF COLD INJURIES

Cold injuries are classified into 3 categories: decreased core temperature (hypothermia), freezing injuries of the extremities, and nonfreezing injuries of the extremities. Each scenario and its characteristic condition(s) will be described. A summary of the signs and symptoms of these injuries and illnesses is found in Table 1, with images of the skin conditions displayed in Figures 1 through 3.

Hypothermia

Traditionally, hypothermia is defined as a decrease in core body temperature below 95°F (35°C). Hypothermia is
<table>
<thead>
<tr>
<th>Condition</th>
<th>Sign or Symptom[^a]</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypothermia</strong>[^1,5,7–9]</td>
<td>Core temperature 98.6°F to 95°F (37°C–35°C) Amnesia, lethargy Vigorous shivering Impaired fine motor control Cold extremities Polyuria Pallor Rhinorrhea Typically conscious Blood pressure within normal limits</td>
</tr>
<tr>
<td><strong>Mild</strong></td>
<td>Core temperature 94°F to 90°F (34°C–32°C) Depressed respiration and pulse Cardiac arrhythmias Cyanosis Cessation of shivering Impaired mental function Slurred speech Impaired gross motor control Loss of consciousness Muscle rigidity Dilated pupils Blood pressure decreased or difficult to measure</td>
</tr>
<tr>
<td><strong>Moderate</strong></td>
<td>Core temperature below 90°F (32°C) R rigidity Bradycardia Severely depressed respiration Hypotension, pulmonary edema Spontaneous ventricular fibrillation or cardiac arrest Usually comatose</td>
</tr>
<tr>
<td><strong>Frostbite</strong>[^1,5,8–13]</td>
<td><strong>Mild/superficial</strong></td>
</tr>
<tr>
<td><strong>Deep</strong></td>
<td>Skin is hard and cold Skin may be waxy and immobile Skin color is white, gray, black, or purple Vesicles present Burning aching, throbbing, or shooting pain Poor circulation in affected area Progressive tissue necrosis Neurapraxia Hemorrhagic blistering develops within 36 to 72 hours Muscle, peripheral nerve, and joint damage likely</td>
</tr>
<tr>
<td><strong>Chilblain/pernio</strong>[^5,13,14]</td>
<td>Red or cyanotic lesions Swelling Increased temperature Tenderness Itching, numbness, burning, or tingling Skin necrosis Skin sloughing</td>
</tr>
<tr>
<td><strong>Immersion (trench) foot</strong>[^5,8,14]</td>
<td>Burning, tingling, or itching Loss of sensation Cyanotic or blotchy skin Swelling Pain/sensitivity Blisters Skin fissures or maceration</td>
</tr>
</tbody>
</table>

[^a]: Not all patients will display all signs and symptoms of the condition.
classified as mild, moderate, or severe, depending upon measured core temperature. Information in the literature varies slightly as to which core temperatures are assigned to which degree of hypothermia, but in this paper, we will use the following definitions. Mild hypothermia is a core temperature of 95°F (35°C) to 98.6°F (37°C). Moderate hypothermia is a core temperature of 90°F (32°C) to 94°F (34°C). Severe hypothermia is a core temperature below 90°F (32°C). Each level of hypothermia has characteristic signs and symptoms, although individuals respond differently, and not every hypothermic person exhibits all signs and symptoms. Therefore, a detailed assessment is appropriate in all cases of potential cold injury. Hypothermia is most likely to occur with prolonged exposure to cold, wet, or windy conditions (or a combination of these) experienced during endurance events, outdoor team sports (eg, soccer, football), mountaineering, hiking, and military maneuvers and in occupations that require long periods outdoors or in unheated spaces (eg, public safety, building trades, transportation).

Frostbite and Frostnip

Frostbite is actual freezing of body tissues. It is a localized response to a cold, dry environment, yet moisture from sweating may exacerbate frostbite due to increased tissue cooling. Similar to hypothermia, frostbite has stages, delineated by the depth of tissue freezing and resulting in frostnip, mild frostbite, or severe frostbite. Frostbite develops as a function of the body’s protective mechanisms to maintain core temperature. Warm blood is shunted from cold peripheral tissues to the core by vasoconstriction of arterioles, which supply capillary beds and venules to the extremities and face, especially the nose and ears. Frostbite progresses from distal to proximal and from superficial to deep. As the temperature of these areas continues to decrease, cells begin to freeze. Damage to the frostbitten tissue is due to electrolyte concentration changes within the cells, resulting in water crystallization within the tissue. For cells to freeze, the tissue temperature must be below 28°F (−2°C).

Frostnip, the mildest form of cold injury to the skin, is a precursor to frostbite. It can occur with exposure of the skin to very cold temperatures, often in combination with windy conditions. It can also occur from skin contact with cold surfaces (eg, metal, equipment, liquid). With frostnip, only the superficial skin is frozen; the tissues are not permanently damaged, although they may be more sensitive to cold and more likely, with repeated exposures, to develop frostnip or frostbite. Mild frostbite involves freezing of the skin and adjacent subcutaneous tissues; extracellular water freezes first, followed by cell freezing. Severe frostbite is freezing of the tissues below the skin and the adjacent tissues, which can include muscle, tendon, and bone.

Chilblain (Pernio)

Chilblain, also known as pernio, is an injury associated with extended exposure (1–5 hours) to cold, wet conditions. Chilblain is an exaggerated or uncharacteristic inflammatory response to cold exposure. Prolonged constriction of the skin blood vessels results in hypoxemia and vessel wall inflammation; edema in the dermis may also be present. Chilblain can occur with or without
factors. Therefore, these recommendations do not guarantee insulation, exposure time, and other nonenvironmental responses to cold vary physiologically with combinations and provide effective immediate care when needed. Individual-of physically active individuals exposed to cold environments

**Prevention**

1. Perform a comprehensive, physician-supervised, preparticipation medical screening to identify athletes with a previous history of cold injury and athletes predisposed to cold injury based upon known risk factors (Table 3). This preparticipation examination should include questions pertaining to a history of cold injury and problems with cold exposure and should be performed before planned exposures to conditions that may lead to cold injury. 

2. Identify participants who present with known risk factors (Table 3) for cold injury and provide increased monitoring of these individuals for signs and symptoms.

3. Ensure that appropriately trained personnel are available on-site at the event and are familiar with cold injury prevention, recognition, and treatment approaches.

4. Educate athletes and coaches concerning the prevention, recognition, and treatment of cold injury and the risks associated with activity in cold environments.

5. Educate and encourage athletes to maintain proper hydration and eat a well-balanced diet. These guidelines are especially imperative for activities exceeding 2 hours. Consistent fluid intake during low-intensity exercise is necessary to maintain hydration in the presence of typical cold-induced diuresis. Athletes should be encouraged to hydrate even if they are not thirsty, as evidence suggests the normal thirst mechanism is blunted with cold exposure.

6. Develop event and practice guidelines that include recommendations for managing athletes participating in cold, windy, and wet conditions. The influence of air temperature and wind speed conditions should be taken into account by using wind-chill guidelines. Risk management guidelines (Table 3, Figure 4) can be used to make participation decisions based upon the prevailing conditions.
decisions depend upon the length of anticipated exposure and availability of facilities and interventions for rewarming if needed. Modify activity in high-risk conditions to prevent cold injury. Monitor athletes for signs and symptoms and be prepared to intervene with basic treatment. Also monitor environmental conditions before and during the activity and adjust activities if weather conditions change or degenerate.28,29 Evidence Category: C

7. Clothing should provide an internal layer that allows evaporation of sweat with minimal absorption, a middle layer that provides insulation, and a removable external layer that is wind and water resistant and allows for evaporation of moisture. Examples of various clothing ensembles are found in Table 4. Toes, fingers, ears, and skin should be protected when wind-chill temperatures are in the range at which frostbite is possible in 30 minutes or less. Remove wet clothing as soon as practical and replace with dry, clean items.30–32 Evidence Category: C

8. Provide the opportunity for athletes to rewarm, as needed, during and after activity using external heaters, a warm indoor environment, or the addition of clothing. After water immersion, rewarming should begin quickly and the athlete should be monitored for afterdrop, in which the core temperature actually decreases during rewarming.33–35 Evidence Category: C

9. Include the following supplies on the field, in the locker room, or at convenient aid stations for rewarming purposes:

- A supply of water or sports drinks for rehydration purposes as well as warm fluids for possible rewarming purposes. Fluids that may freeze during events in subfreezing temperatures may need to be placed in insulated containers or replaced intermittently.
- Heat packs, blankets, additional clothing, and external heaters, if feasible, for active rewarming.
- Flexible rectal thermometer probe to assess core body temperature. Rectal temperature has been identified as the best combination of practicality and accuracy for assessing core temperature in the field.36 Other measurements ( tympanic, aural, and esophageal) are problematic or difficult to obtain. The rectal thermometer used should be a low-reading thermometer (ie, capable of measuring temperatures below 95°F [35°C]).
- Telephone or 2-way radio to communicate with additional medical personnel and to summon emergency medical transportation.
- Tub, wading pool, or whirlpool for immersion warming treatments (including a thermometer and additional warm water to maintain required temperatures). Evidence Category: C

10. Notify area hospital and emergency personnel before large events to inform them of the potential for cold-related injuries. Evidence Category: C

Recognition and Treatment

Hypothermia (Mild)

11. Be aware of the signs and symptoms of hypothermia, which include vigorous shivering, increased blood pressure, rectal temperature less than 98.6°F (37°C) but greater than 95°F (35°C), fine motor skill impairment, lethargy, apathy, and mild amnesia (Table 1). Evidence Category: A

12. Rectal temperature obtained using a thermometer (digital or mercury) that can read below 94°F (34°C) is the preferred method for assessing core temperature in persons suspected of being hypothermic, even though procuring rectal temperature in the field can be a challenge. Using tympanic, axillary, or oral temperatures instead of rectal temperature is faulty due to environmental concerns, such as exposure to air temperatures; however, if either axillary or oral temperature is above 95°F (35°C), the person is not hypothermic.1,7–9 Figure 5 provides a treatment algorithm for hypothermia. Evidence Category: B

13. Begin by removing wet or damp clothing; insulating the athlete with warm, dry clothing or blankets (including covering the head); and moving the athlete to a warm environment with shelter from the wind and rain. Evidence Category: C

14. When rewarming, apply heat only to the trunk and other areas of heat transfer, including the axilla, chest wall, and groin.37–39 Rewarming the extremities can produce afterdrop, which is caused by dilation of peripheral vessels in the arms and legs when warmed. This dilation sends cold blood, often with a high level of acidity and metabolic byproducts, from the periphery to the core. This blood cools the core, leading to a drop in core temperature, and may result in cardiac arrhythmias and death.40,41 Evidence Category: C

15. Provide warm, nonalcoholic fluids and foods containing 6% to 8% carbohydrates to help sustain shivering and maintain metabolic heat production. Evidence Category: C

16. Avoid applying friction massage to tissues, as this may increase damage if frostbite is present.10 Evidence Category: A

Hypothermia (Moderate/Severe)

17. Be aware of the signs and symptoms of moderate and severe hypothermia, which may include cessation of shivering, very cold skin upon palpation, depressed vital signs, rectal temperature between 90°F (32°C) and 95°F (35°C) for moderate hypothermia or below 90°F (32°C) for severe hypothermia, impaired mental function, slurred speech, unconsciousness, and gross motor skill impairment (Table 1).1,7–9 Evidence Category: A

18. If an athlete with suspected hypothermia presents with signs of cardiac arrhythmia, he or she should be
moved very gently to avoid causing paroxysmal ventricular fibrillation.\textsuperscript{7–9} Evidence Category: B

19. Begin with a primary survey to determine the necessity of cardiopulmonary resuscitation (CPR) and activation of the emergency medical system. Remove wet or damp clothing; insulate the athlete with warm, dry clothing or blankets (including covering the head); and move the athlete to a warm environment with shelter from the wind and rain. Evidence Category: C

20. When rewarming, apply heat only to the trunk and other areas of heat transfer, including the axilla, chest wall, and groin.\textsuperscript{37–39} Evidence Category: C

21. If a physician is not present during the treatment phase, initiate rewarming strategies immediately and continue rewarming during transport and at the hospital. Evidence Category: C

22. During the treatment and/or transport, continually monitor vital signs and be prepared for airway management. A physician may order more aggressive rewarming procedures, including inhalation rewarming, heated intravenous fluids, peritoneal lavage, blood rewarming, and use of antiarrhythmia drugs.\textsuperscript{41–46} Evidence Category: C

23. When immediate management is complete, monitor for postrewarming complications, including infection and renal failure.\textsuperscript{47} Evidence Category: A

**Frostbite (Superficial)**

24. Be aware of signs and symptoms of superficial frostbite, which include edema, redness or mottled gray skin appearance, stiffness, and transient tingling or burning (Table 1, Figure 1). Evidence Category: A
25. Rule out the presence of hypothermia by evaluating observable signs and symptoms and measuring core temperature. Evidence Category: C

26. The decision to rewarm an athlete is contingent upon resources available and likelihood of refreezing. Rewarming can occur at room temperature or by placing the affected tissue against another person’s warm skin. Rewarming should be performed slowly, and water temperatures greater than 98°F to 104°F (37°C–40°C) should be avoided. Evidence Category: C

27. If rewarming is not undertaken, protect the affected area from additional damage and further tissue temperature decreases and consult with a physician or transport to a medical facility. Evidence Category: C

28. Avoid applying friction massage to tissues and leave any vesicles (fluid-filled blisters) intact. Evidence Category: C

29. Once rewarming has begun, it is imperative that affected tissue not be allowed to refreeze, as tissue necrosis usually results. Evidence Category: C

30. Athletes should avoid consuming alcohol and using nicotine. Evidence Category: C

Frostbite (Deep)

31. Be aware of signs and symptoms of deep frostbite, which include edema, mottled or gray skin appearance, tissue that feels hard and does not rebound, vesicles, and numbness or anesthesia (Table 1). Evidence Category: A

32. Rule out the presence of hypothermia by assessing observable signs and symptoms and measuring core temperature. Evidence Category: C

33. To rewarm, the affected tissue should be immersed in a warm (98°F–104°F [37°C–40°C]) water bath. Water temperature should be monitored and maintained. Remove any constrictive clothing and submerge the entire affected area. The water will need to be gently circulated, and the area should be immersed for 15 to 30 minutes. Thawing is complete when the tissue is pliable and color and sensation have returned. Rewarming can result in significant pain, so a physician may prescribe appropriate analgesic medication. Evidence Category: C

34. If rewarming is not undertaken, the affected area should be protected from additional damage and further tissue temperature decreases. Consult with a physician or transport the athlete to a medical facility. Evidence Category: C

35. Tissue plasminogen activators (tPA) may be administered to improve tissue perfusion. These agents have been shown to limit the need for subsequent amputation due to tissue death. Evidence Category: C

36. Do not use dry heat or steam to rewarm affected tissue. Evidence Category: C

37. Avoid friction massage or vigorous rubbing to the affected tissues and leave any vesicles or fluid-filled blisters intact. If vesicles rupture, they should be treated to prevent infection. Evidence Category: C

38. Once rewarming has begun, it is imperative that the affected tissue not be allowed to refreeze, as tissue necrosis usually results. Also, weight bearing should be avoided when feet are affected. If the possibility of refreezing exists, rewarming should be delayed until advanced medical care can be obtained. Evidence Category: C

39. Athletes should avoid using alcohol and nicotine. Evidence Category: C

40. If tissue necrosis occurs and tissue sloughs off, debridement and infection control measures are appropriate. Evidence Category: C

Chilblain

41. Be aware of the signs and symptoms of chilblain, which include exposure to cold, wet conditions for more than 60 minutes at temperatures less than 50°F (16°C) and the presence of small erythematous papules, with edema, tenderness, itching, and pain (Table 1, Figure 2). Upon rewarming, the skin may exhibit inflammation, redness, swelling, itching, or burning and increased temperature. Evidence Category: A

42. Remove wet or constrictive clothing, wash and dry the area gently, elevate the area, and cover with warm, loose, dry clothing or blankets. Evidence Category: C

43. Do not disturb blisters, apply friction massage, apply creams or lotions, use high levels of heat, or allow weight bearing on the affected area. Evidence Category: C

44. During treatment, continually monitor the affected area for return of circulation and sensation. Evidence Category: C

Immersion (Trench) Foot

45. Be aware of the signs and symptoms of immersion (trench) foot, which include exposure to cold, wet environments for 12 hours to 3 or 4 days, burning, tingling or itching, loss of sensation, cyanotic or blotty skin, swelling, pain or sensitivity, blisters, and skin fissures or maceration (Table 1, Figure 3). Evidence Category: A

46. To prevent immersion foot, encourage athletes to maintain a dry environment within the footwear, which includes frequent changes of socks or footwear (or both), the use of moisture-wicking sock material, controlling excessive foot perspiration, and allowing the feet to dry if wearing footwear that does not allow moisture evaporation (eg, vinyl, rubber, vapor-barrier shoes or boots). Evidence Category: C

47. For treatment, thoroughly clean and dry the feet, and then treat the affected area by applying warm packs or soaking in warm water (102°F–110°F [39°C–43°C])
for approximately 5 minutes. Replace wet socks with a clean, dry pair, and rotate footwear or allow footwear to dry before reusing.\textsuperscript{8,14} 

Evidence Category: C

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**Wind Chill Chart**

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**Frostbite Times**

- 30 minutes
- 10 minutes
- 5 minutes

**Wind Chill (°F) = 35.74 + 0.6215T − 35.75(V^{0.16}) + 0.4275V^{0.16}**

Where, \(T\) = Air Temperature (°F) \(V\) = Wind Speed (mph)

Effective 1/1/07

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48. Use a risk management process that includes strategies for preventing, recognizing, and treating cold injuries during events. These strategies can then be used for

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\textsuperscript{8,14}
Thermoregulation

During cold stress, a normal body temperature is maintained through a complex regulatory system. A summary of the interactions among various physiologic influences and controls is found in Figure 6. Maintaining a normothermic body temperature depends on a dynamic balance between heat gained from metabolic heat production and heat lost to the environment. The body’s basic responses to cold stress in order to maintain normothermia are an increase in metabolic heat production, or thermogenesis, and peripheral vasoconstriction at the skin surface to prevent heat loss to the environment.

An increase in metabolic heat production can be accomplished in 2 ways: nonshivering thermogenesis and shivering thermogenesis. Nonshivering thermogenesis is defined as an increase in metabolic heat production from sources other than muscle contraction and is not considered a major source of metabolic heat in adult humans. Shivering thermogenesis is defined as an increase in heat production due to involuntary muscle contraction. The onset and intensity of shivering are mitigated by the local temperature of the hypothalamic thermoregulatory center itself.

A decrease in peripheral blood flow due to blood vessel constriction prevents loss of heat to the environment by reducing the thermal gradient (ie, the temperature difference between the skin and the environment). The reduction in peripheral blood flow is highly specific and most pronounced in the extremities. Neural control of peripheral blood flow is under the influence of skin temperature, core temperature, and baroreflexes. The vasoconstriction response typically begins at a skin temperature of less than 93.2°F to 95.0°F (34°C–35°C) and is maximized at skin temperatures of 87.8°F (31°C) and below. The magnitude and duration of the vasoconstriction is modulated by a cold-induced vasodilation (CIVD). The CIVD appears to limit the duration of the vasoconstriction and protect the affected area from local cold injury. Periodic fluctuations in blood flow create fluctuations in skin temperature after the initial drop in skin blood flow and temperature with cold exposure. This fluctuation between minimal and greater-than-normal flow continues throughout the cold exposure. The CIVD appears to be mediated by the cessation of norepinephrine release after initial release. It may also occur due to decreases in tissue temperature, which diminish sympathetic nerve conduction and stop the release of norepinephrine. The resulting blood flow increase rewarms the tissue, nerve activity is reestablished, norepinephrine is released, and vasoconstriction is reinitiated. When the CIVD is absent (which can result from certain drugs; see “Nonenvironmental Risk Factors”) and fails to provide protection, the risk of nonfreezing cold injury may increase.

Heat Loss Mechanisms

The human body loses heat to the environment through 4 mechanisms: radiation, convection, conduction, and evaporation. By manipulating environment or clothing (microenvironment) or both, humans can minimize heat...
Figure 5. Algorithm for patient with hypothermia.
losses. However, with inappropriate choices or when proper choices are unavailable, heat losses are exacerbated, with potentially fatal consequences.

Radiation is heat lost directly to the environment by long-wave (infrared) radiation. Wind, wetness, and other factors do not affect it. Radiative heat loss is greatest at night, especially with the absence of the moon or cloud cover. Uncovered surfaces of the body, especially the head, face, neck, and hands, also increase radiative heat loss. Heat lost through uncovered skin is perceived to be greater because skin temperature is lower and can account for 50% to 65% of all heat losses from the human body in a resting state.

Convection is heat lost through the movement of air or water across the skin. The human body maintains a thin layer of warm air adjacent to the skin, called the boundary layer. Convection enhances the rate of evaporative heat loss. Air moving across the skin removes this warm layer, replacing it with a cold layer of air that must then be warmed. Air movement may be from wind or a person moving through the air, as in running, skiing, cycling, etc. Depending upon the speed of the air moving across the skin, convective heat losses may be small or large. An estimate of convective heat loss is the wind-chill factor. The amount and insulative properties of clothing worn may reduce or intensify convective heat losses.

Conduction is heat lost by direct contact with a cold surface. It is exacerbated by moisture, either in the environment (rain, snow, or water immersion) or in wet clothing. Conduction can increase heat loss by up to 5 times with wet clothing and up to 25 times with water immersion. Proper selection of clothing, footwear, layering, and activity level can drastically reduce conductive heat losses. Subcutaneous fat stores also help to reduce conductive heat loss. Conductive and convective heat losses usually account for approximately 15% of all heat losses; however, high air speed, inadequate amounts of dry clothing, wet clothing, and water immersion all drastically increase conductive and convective heat losses.

Evaporative heat losses account for 15% to 25% of the total and occur through respiration and exposed skin. Little can be done to reduce evaporative heat losses from respiration, because ambient air must be warmed to core temperature and humidified to 100% as it moves into the respiratory tract. However, like conductive heat losses, losses from the skin and sweating can be controlled through proper selection of clothing and activity level.

The amount of heat lost from the body can be calculated using the classical heat balance equation. The equation refers to the balancing of heat being produced by metabolism (thermogenesis) and the rate at which it is being lost to the environment via radiation, convection, conduction, and evaporation (thermolysis). This equation provides a conceptual understanding of the interplay between the production of heat within the body and the loss of heat to the environment. When an imbalance develops between the rates of heat production and heat loss, total body heat content changes. For example, when an athlete ceases exercise, the level of heat production diminishes, while the level of heat loss remains unchanged. This leads to an imbalance between the rates of heat production and heat loss.

$$S = M - R - C - K - E$$
$$S = \text{heat gain/loss}$$
$$M = \text{metabolic heat production}$$
$$R = \text{radiative heat loss}$$
$$C = \text{convective heat loss}$$
$$K = \text{conductive heat loss}$$
E=evaporative heat loss

Pathophysiology of Cold Injury

Hypothermia. Hypothermia (defined as a decrease in core body temperature below 95°F [35°C]) can develop in cold and dry or cold and wet conditions and can arise either slowly over many hours or quite suddenly. Generally, slow-onset hypothermia occurs on land, whereas sudden hypothermia results from cold-water immersion or exposure to cold rain. The body loses heat faster than it can generate heat and core temperature begins to drop. Although we associate hypothermia with temperatures below freezing, it also occurs at temperatures as high as 50°F to 60°F (10°C–16°C). For example, at the 1983 Bostonfest Marathon, 20% of the runners presenting for treatment at the finish-line medical tent were diagnosed with hypothermia despite an ambient temperature of approximately 50°F (10°C). During the 1985 Boston Marathon, 75 runners (1.3% of entrants) were treated for hypothermia, even though the ambient temperature was 76°F (24°C).

Hypothermia is caused by the body’s inability to maintain a normal core temperature, with resulting changes in the function of the nervous system, cardiovascular system, respiratory system, and renal system. The central nervous system is susceptible to depression during cold exposure. This depressed activity is typically manifested as changes in motor function (eg, clumsiness, loss of finger dexterity, slurred speech), cognition (eg, confusion, poor decision making, memory loss) and level of consciousness. Significant changes in central nervous system function occur as core temperature drops below 95°F (35°C), and show a linear decrease as core temperature continues to drop. Brain function becomes measurably abnormal below 92.3°F (34°C) and ceases at 66°F to 68°F (19°C–20°C).

Cardiovascular system functional change is manifested as an initial tachycardia and then a progressive bradycardia that decreases the resting heart rate by 50%. This drop in heart rate is due to decreased depolarization of the heart pacemaker cells. Other changes include increased myocardial oxygen demand and decreased arterial pressure and cardiac output. The conduction system changes are apparent during an electrocardiogram as prolonged PR, QRS, and QT intervals. Additional arrhythmias may include ventricular fibrillation and asystole, which can occur when the core temperature is below 77°F (25°C), and atrial fibrillation, which is common at core temperatures below 89.6°F (32°C).

The renal system responds to cold exposure by excreting large amounts of glomerular filtrate. This cold-induced diuresis appears to be due to a large increase in central volume secondary to peripheral vasoconstriction. Diuresis may occur to balance fluid levels as the central circulation becomes overloaded. The urine produced is very dilute, regardless of hydration status. Cold-water immersion can further increase urine output by 3.5 times normal. These renal responses occur during rest and light activity.

The respiratory system reacts to cold exposure by initially producing a hyperventilation response. After the initial response, however, the ventilation rate progressively decreases as core temperature drops, reaching 5 to 10 breaths per minute below a core temperature of 86°F (30°C). Carbon dioxide production also decreases by up to 50%, which can lead to respiratory acidosis. These respiratory responses occur during rest and light activity.

Frostbite. The pathophysiology of deep frostbite (defined as actual freezing of body tissues) typically consists of 3 distinct phases: frostnip, mild frostbite, and deep frostbite. The frostnip, or prefreeze, phase occurs with superficial skin cooling below 50°F (10°C), resulting in loss of sensation, constriction of the microvasculature, plasma leakage, and increased viscosity of vascular contents.

Mild frostbite, or freeze-thaw phase, begins as skin temperature drops below 28°F (−2°C) and extracellular ice crystals form. The location and rate of crystal formation depends on the severity of the cold stress (combination of air temperature, moisture presence, and wind). Water migrates across the cell membrane, resulting in intracellular dehydration and increased intracellular electrolyte concentrations. As the cell volume decreases, the cell eventually collapses, the membrane ruptures, and cell death occurs. As crystallization progresses, surrounding cells and vascular structures are compressed.

The third phase, or severe frostbite, results in microvascular collapse at the arteriole and venule levels. As microvascular tissue fails, blood viscosity increases, resulting in microthrombi, plasma leakage, increased tissue pressure, ischemia, and tissue death. Nerve and muscle tissue may also be affected. As edema resolves over approximately 72 hours from onset, the most noticeable sign of frostbite is gangrenous tissue.

Blood flow to the skin of the extremities (acral skin) (eg, fingers, toes, tip of nose) is under much stronger local control of vasoconstriction and vasodilation than nonacral skin and is very sensitive to local changes in temperature. This local control is independent of the central nervous system and produces significant changes in blood flow to skin that is cooled, regardless of changes in core temperature. Nonacral skin blood flow is controlled by the central nervous system, and changes in flow are likely due to changes in core temperature. These differences in control of blood flow produce the scenario of potential freezing injury to acral skin without a significant drop in core body temperature.

Chilblain (Pernio) and Immersion (Trench) Foot. Non-freezing cold injuries, such as chilblain and immersion foot, appear to share a common pathophysiology and generally result in cellular damage without ice crystal formation. The abnormal inflammatory response typically leads to microvascular collapse and extracellular fluid build-up. Increased extracellular fluid results in damage to neurologic and vascular tissue. Classic symptoms of swelling, numbness, and itching, followed by hypersensitivity to cold after rewarming, appear related to a dermal edema, migration of lymph fluid, microvascular damage, and vascular and neurologic hypersensitivity to cold exposure.

Nonenvironmental Risk Factors

Health care providers should be aware of the following nonenvironmental risk factors, which may make athletes more susceptible to cold injury and may affect normal physiologic responses to cold exposure.
Previous Cold Injuries. Having sustained a previous cold injury increases the chance of subsequent cold injuries by 2 to 4 times, even if prior injuries were not debilitating or resolved with no or minimal medical care. For example, an athlete who sustained frostnip or frostbite is 2 to 4 times more likely to develop frostbite in the same area again, given similar environmental conditions.

Low Caloric Intake, Dehydration, and Fatigue. Low caloric intake (less than 1200 to 1500 kcal/day) or hypoglycemia (or both) directly decreases metabolism and concomitant heat production, contributing to the inability to maintain body temperature balance through physical activity. Dehydration does not negatively affect peripheral vasoconstriction or shivering and, therefore, does not appear to increase susceptibility to cold injury. Fatigue associated with hypoglycemia is linked to impaired peripheral vasoconstriction and shivering responses and can lead to faulty decision making and inadequate preparations, indirectly resulting in cold injuries.

Race. Black individuals have been shown to be 2 to 4 times more likely than individuals from other racial groups to sustain cold injuries. These differences may be due to cold weather experience, but are likely due to anthropometric and body composition differences, including less pronounced CVRD, increased sympathetic response to cold exposure, and thinner, longer digits.

Nicotine, Alcohol, and Drug Use. Nicotine inhaled through smoking causes a reflex peripheral vasoconstriction, possibly negating the CVRD and later enhancing the cold-induced vasoconstriction to maintain core temperature. Alcohol reduces the glucose concentration in the blood, which tends to decrease the shivering response. Alcohol also may lead to faulty decision making due to its effects on the central nervous system. Drugs with a depressive effect may impair the thermoregulatory system and so inhibit the body’s reaction to cold by blunting the peripheral vasoconstriction and shivering responses. As with alcohol, they may also lead to faulty decision making.

Body Size and Composition. Body fat and muscle mass appear to be instrumental in providing protection for maintaining core body temperature with exposure to cold air and water. This effect appears in both males and females regardless of the amount of clothing worn. Strong evidence suggests that percentage of body fat (>25%, approximately) and amount of muscle mass are reliable predictors of the ability to maintain and protect core body temperature during prolonged exposure to a wide range of cold air and water temperatures. The greater the level of body fat and muscle mass, the better the ability to protect core body temperature through passive (eg, insulative properties of fat) and active (eg, shivering thermogenesis) mechanisms.

Aerobic Fitness Level and Training. Overall, physical training and fitness level appear to have only minimal influence on thermoregulatory responses to cold. Most cross-sectional comparisons of aerobically fit and less-fit persons show no relationship between maximal aerobic exercise capability and temperature regulation in the cold. In those studies purportedly demonstrating a relationship, differences in thermoregulation appear more likely attributable to anthropometric (body size and composition) differences between aerobically fit and less-fit participants, rather than an effect of maximal aerobic exercise capabilities per se. The primary thermoregulatory advantage provided by increased endurance resulting from physical training is that a fitter individual can sustain higher-intensity, longer-duration voluntary activity than a less-fit person and, thus, maintain higher sustained rates of metabolic heat production, keeping the former warmer. In addition, exercise training has been suggested to enhance the peripheral vasoconstriction response, which would conserve body heat but possibly increase peripheral cold injury risks.

Sex. The hypothermia injury rate for females is 2 times higher than for males. Sex differences in thermoregulatory responses during cold exposure are influenced by interactions among total body fat content, subcutaneous fat thickness, amount of muscle mass, and surface area-to-mass ratio. For example, among men and women with equivalent total body mass and surface areas, women’s greater fat content enhances insulation and reduces the fall in core temperature. In women and men of equivalent subcutaneous fat thickness, women typically have a greater surface area but smaller total body mass and smaller muscle mass (thus, lower total body heat content) than men and lose heat at a greater rate. Women’s thermogenic response to cold exposure also appears less able to generate metabolic heat than men of similar body composition due to less total muscle mass. Therefore, total heat loss is greater in women due to the larger surface area for convective heat loss, and body temperature tends to fall more rapidly for any given cold stress. As a result, the prevention and recognition recommendations in this position statement should be interpreted and used more conservatively for female athletes than male athletes.

Clothing. The role of clothing in preventing cold injuries lies in its ability to reduce heat loss to the environment by trapping warm air. Cold-weather clothing typically has an internal layer that allows evaporation of sweat without absorption, a middle layer that provides insulation, and an external layer that is wind and water resistant and allows evaporation of moisture. The internal layer is in direct contact with the skin and uses a moisture-wicking material such as polyester or polypropylene. This layer should not retain moisture but should transfer the moisture to other layers, from which it can evaporate. The middle layer provides the primary insulation against heat loss and can be a fleece or wool material. The outer layer should have venting abilities (eg, zippers or mesh in the armpits or low back area) to allow moisture transfer to the environment.

Clothing requirements for cold environment exercise depend on ambient temperature; presence of wind, rain, and water; and activity intensity. Generally speaking, as exercise intensity increases at any given air temperature, the amount of clothing insulation needed to maintain body temperature equilibrium decreases. The insulative protection offered by different clothing combinations is represented in units of clo. One clo of insulation is the clothing needed to permit a person to rest comfortably when the air temperature is 70°F (21°C). See Table 4 for a list of the clo values of common clothing combinations. Interactions between exercise intensity and ambient conditions dictate the selection of clothing based upon clo values. For example, if the ambient temperature is 20°F (−7°C) and the person is at rest (1 metabolic equivalent of exercise...
Exercise-Induced Bronchospasm. Exercise-induced bronchospasm (EIB), also called exercise-induced asthma or airway hyperresponsiveness, is a narrowing of the respiratory tract airways. It is exacerbated by exposure to cold, dry air. Predisposing factors include asthma and allergies. However, not all individuals with EIB have asthma or allergies, and not all individuals with asthma or allergies have EIB. This condition can affect any individual, from a small child at play to an elite athlete. Authors have noted a high prevalence of EIB in cold-weather athletes, in women, and in athletes walking at 3 mph (1.3 m·s⁻¹) in various combinations of low air temperature; humidity, rain, or immersion; and wind. This index estimates the danger of extreme cooling of exposed skin (i.e., the risk of frostbite) while walking at 3 mph (1.3 m·s⁻¹) in various combinations of low air temperature; humidity, rain, or immersion; and wind. This index estimates the danger of extreme cooling of exposed skin (i.e., the risk of frostbite) while walking at 3 mph (1.3 m·s⁻¹) in various combinations of weather conditions.

Raynaud Syndrome. As with EIB, Raynaud syndrome is caused by cold exposure and characterized by intermittent vasoconstriction of the digits. This vasospasm significantly reduces blood flow to the extremities. The affected area may present with tingling, swelling, or a throbbing pain. The skin may turn a shade of white, then possibly blue, and then becomes red upon rewarming. Raynaud syndrome describes a spectrum of disorders whose causes are usually idiopathic, although infrequently, autonomic dysfunction or an underlying condition such as thoracic outlet syndrome or collagen vascular disease may be responsible.

Anorexia Nervosa. Anorexia nervosa results in a deficiency of body fat stores, potential malnutrition, decreased metabolic rate, and peripheral vasoconstriction. These changes limit the ability to maintain a normal core temperature.

Cold Urticaria. Cold urticaria may be the most common form of urticaria. The condition has a rapid onset, presenting with wheals (hives) that may be local or generalized, redness, itching, and edema. Other symptoms may include fatigue, headache, dyspnea, and in rare cases, anaphylactic shock. Two forms of the condition, primary acquired and secondary acquired (hereditary), have been identified and differ in speed of onset: within minutes or 24 to 48 hours after cold exposure, respectively.

Cardiovascular Disease. Individuals with cardiovascular disease are sensitive to increased demands on the myocardium and increases in blood pressure, as well as having potentially decreased flow to cutaneous and subcutaneous tissues. Cold exposure coupled with exercise increases the demand on the cardiovascular system by increased sympathetic nervous system activity, peripheral resistance, blood pressure, and myocardial oxygen demands. This increased stress is in contrast to the demands of rest and exercise in warm environments. Therefore, individuals with diminished cardiovascular system function should be cautious when exercising in the cold and should be monitored closely for symptoms associated with a myocardial infarction.

Environmental Risk Factors

Environmental cold stress results from a combination of low air temperature; humidity, rain, or immersion; and little thermal radiation and air movement. An index of cold stress is the wind-chill temperature index (WCT) (Figure 4). This index gives an indication of how cold a person feels when exposed to a combination of cold air and wind. This index estimates the danger of extreme cooling of exposed skin (i.e., the risk of frostbite) while walking at 3 mph (1.3 m·s⁻¹) in various combinations of weather. When the WCT is below −18°F (−27°C), the risk of developing frostbite in exposed skin in less than 30 minutes increases, warranting closer observation. However, the environmental wind speed in the WCT does not account for wind produced during movement. Biking or running produce wind across the body at the same rate as body movement and should be taken into account when estimating risk. For example, if a light wind is present (less than 5 mph [2.24 m·s⁻¹]) with cold temperatures (25°F [−4°C]), the risk of frostbite for most people is low. Yet if the athlete is cycling at 15 mph (16.71 m·s⁻¹), the relative risk of developing frostbite increases. Therefore, the effect of air movement produced by the body should be taken into account when using the wind-chill recommendations. The WCT is calculated using the following formula:

\[ \text{Wind chill (°F)} = 35.74 + 0.6215T - 35.75V^{0.16} + 0.4275T(V^{0.16}) \]

Where \( T \) = Air temperature (°F), and \( V \) = Wind speed (mph)

This index is a useful tool to monitor the potential thermal stress athletes must deal with when exposed to cold temperatures.
Influence of Wind, Rain, and Immersion

Exercise during windy or rainy conditions or water immersion poses several unique challenges to the body’s ability to maintain a normal body temperature. The transfer of body heat in water may be 70 times greater than in air. This transfer can lead to a significant loss of body heat for those exercising in rainy conditions or in the water. Maintaining normal body temperature in those conditions depends on several factors, including exercise intensity, exercise mode, anthropometric factors, insulative properties of clothing and equipment, and the magnitude of heat loss caused by wind speed, amount of rain, or water temperature.

During exercise in cold and wet conditions, the ability to generate adequate metabolic heat to maintain body temperature depends on exercise intensity and mode. More heat is lost from the arms and legs due to smaller diameter and shorter distance from the limb center to the surface, which allows rapid heat conduction compared with the trunk. This increased conduction is outweighed when exercise intensity is greater than 75% of maximal oxygen uptake ($V_{\text{O}}_2\text{max}$). Exercise mode also affects the ability to generate metabolic heat. The more muscle mass involved in performing the exercise, the greater the heat generated. Therefore, exercise involving only the lower body generates more heat than exercise involving only the upper body, and whole-body exercise generates more heat than lower or upper body exercise alone.

The combination of windy and wet conditions can also affect body temperature maintenance. Light to moderate exercise in the rain leads to decreases in core temperature compared with resting conditions. When air temperature is $-15.0^\circ\text{F}$ ($-26.1^\circ\text{C}$) and clothes are wet, heat losses may double those observed in dry conditions. Decreases in core temperature have been observed with the addition of wind during light-intensity exercise. With high-intensity exercise (greater than 60% of peak oxygen consumption), body temperature can be maintained in cold, windy, and wet conditions.

The influence of water temperature and amount of wet or immersed skin also influences heat loss. The lower the water temperature and the larger the surface area of the body immersed in or in contact with water, the greater the heat loss and the more rapid the decreases in core temperature.

The best survival strategy for an individual exposed to accidental, prolonged cold-water immersion (eg, boating or water craft accident) has been debated: try to swim for as long as possible. Complete any tasks that require the use of your hands, such as tying knots or turning on flares, as soon as possible. As your hands cool, they lose dexterity.

Stick to your decision; don’t change your mind midway. After 30 minutes in cold water, you may become hypothermic, and you won’t make the best decisions.

After immersion in cold water, athletes should be monitored for the phenomenon of afterdrop. Once physical activity concludes, the body still dissipates significant amounts of heat but no longer generates metabolic heat through activity. This imbalance between heat loss and heat production could lead to drops in core temperature during the rewarming process. Afterdrop is commonly found in individuals after prolonged water immersion in which core temperature has already decreased.

Role of Cold Acclimatization. Cold acclimatization may play a role in tolerance of cold exposure, but observed adjustments are modest and rely upon the severity of previous exposure. Adjustments found in persons with recurring exposure to cold are habituation, metabolic acclimatization, and insulative acclimatization.

Cold-induced habituation manifests itself as a decreased shivering and vasoconstriction response during cold exposure compared with nonacclimatized exposures. Some individuals may also have a greater decrease in core temperature than nonacclimatized persons; this is known as hypothermic habituation. Short, intense exposures that occur a few times per week appear to elicit habituation. Hypothermic habituation, however, occurs with longer exposures in moderate temperatures during consecutive days over a period greater than 2 weeks.

Metabolic acclimatization is characterized by a more pronounced shivering response to cold and typically occurs after long-term exposures. Insulative acclimatization is produced by greater conservation of heat during exposure to cold. This includes a larger and more rapid decline in skin temperature, resulting in less heat conduction at the skin. Another response may be improved convective heat loss due to a circulatory countercurrent heat exchange mechanism.

In comparison with the acclimatization observed with repeated environmental heat exposure, adjustment to the physiologic response during cold exposure appears to be more difficult to acquire, varies more from individual to individual, develops more slowly, and has less of a preventive effect.

SPECIAL CONCERNS

Children (Prepubescents)

Exposure to cold environments poses unique challenges for young athletes due to their higher surface area-to-mass ratios and smaller adipose tissue deposits. These factors result in a faster cooling rate than for adults in water and similar cooling rates as adults in cold air. These rates
appear to be a function of a higher level of metabolic heat production and a stronger peripheral vasoconstriction response. Therefore, children should take similar preventive measures as those suggested for adults, but they should be encouraged to take more frequent breaks from a cold environment, especially water immersion.

Older Individuals (More Than 50 Years Old)

As one ages, the ability to tolerate cold decreases and risk of hypothermia increases. This increased risk for hypothermia appears to be due to diminished sympathetic nervous system-mediated reflex vasoconstriction, which allows greater heat loss. Additionally, an older individual is more likely to have health concerns such as diabetes, hypothyroidism, hypopituitarism, or hypertension, which increase the likelihood of cold injury. As older individuals continue an active lifestyle and as advances in medical and surgical treatments extend life, more persons with histories of myocardial infarction and stroke are becoming active in outdoor environments. Thus, ATs working with older athletes should apply prevention and recognition recommendations more conservatively in this population.

Spinal Cord Injuries

The presence of a spinal cord injury (SCI) resulting in some form of paralysis is associated with an increased risk for hypothermia during cold exposure. Athletes with SCI tend to be less sensitive to the sensation of cold on the skin surface and have a diminished perception of skin temperature change. Overall, individuals with SCI have a diminished capability to stabilize core temperature; specifically, they have a diminished autonomic response to cold, which results in a decreased vasoconstriction response and a decreased effector response to the muscle to generate metabolic heat. Those with SCI also have abnormal blood pressure responses to cold exposure, as well as increased complaints of muscle spasticity, pain, and numbness. Consequently, athletes with SCI should be monitored closely for both core temperature changes and skin changes associated with nonfreezing cold injury.

CONCLUSIONS

Certified athletic trainers and other health care providers must be able to identify the signs and symptoms of hypothermia, frostbite, chilblains, and immersion (trench) foot in athletes. This position statement outlines the current recommendations to prevent the occurrence and improve the recognition and treatment of cold injury in athletes.

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REFERENCES


National Athletic Trainers’ Association Position Statement: Lightning Safety for Athletics and Recreation

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**Objective:** To educate athletic trainers and others about the dangers of lightning, provide lightning-safety guidelines, define safe structures and locations, and advocate prehospital care for lightning-strike victims.

**Background:** Lightning may be the most frequently encountered severe-storm hazard endangering physically active people each year. Millions of lightning flashes strike the ground annually in the United States, causing nearly 100 deaths and 400 injuries. Three quarters of all lightning casualties occur between May and September, and nearly four fifths occur between 10:00 AM and 7:00 PM, which coincides with the hours for most athletic or recreational activities. Additionally, lightning casualties from sports and recreational activities have risen alarmingly in recent decades.

**Recommendations:** The National Athletic Trainers’ Association recommends a proactive approach to lightning safety, including the implementation of a lightning-safety policy that identifies safe locations for shelter from the lightning hazard. Further components of this policy are monitoring local weather forecasts, designating a weather watcher, and establishing a chain of command. Additionally, a flash-to-bang count of 30 seconds or less should be used as a minimal determinant of when to suspend activities. Waiting 30 minutes or longer after the last flash of lightning or sound of thunder is recommended before athletic or recreational activities are resumed. Lightning-safety strategies include avoiding shelter under trees, avoiding open fields and spaces, and suspending the use of land-line telephones during thunderstorms. Also outlined in this document are the prehospital care guidelines for triaging and treating lightning-strike victims. It is important to evaluate victims quickly for apnea, asystole, hypothermia, shock, fractures, and burns. Cardiopulmonary resuscitation is effective in resuscitating pulseless victims of lightning strike. Maintenance of cardiopulmonary resuscitation and first-aid certification should be required of all persons involved in sports and recreational activities.

**Key Words:** lightning, policies and procedures, lightning casualties, severe-storm hazards, environmental hazards, emergency action plan, thunderstorms, lightning-safety policy, athletics, recreation

Over the past century, lightning has consistently been one of the top 3 causes of weather-related deaths in this country.1,2 It kills approximately 100 people and injures hundreds more each year.2-5 Lightning is an enormous and widespread danger to the physically active population, due in part to the prevalence of thunderstorms in the afternoon to early evening during the late spring to early fall and a societal trend toward outdoor physical activities.2,3,6 Certain areas of the United States have higher propensities for thunderstorm activity, and thus, higher casualty rates: the Atlantic seaboard, southwest, southern Rocky Mountains, and southern plains states.2,7

Worldwide, approximately 2000 thunderstorms and 50 to 100 lightning flashes occur every second.8 In 1997, the National Lightning Detection Network recorded nearly 27 000 000 cloud-to-ground lightning strikes in the United States (Christoph Zimmerman, Global Atmospherics, Inc, Tucson, AZ, unpublished data). Many of these strikes caused fires, power outages, property damage, loss of life, and disabling injuries. Property damage from lightning is estimated to cost $5 000 000 000 to $6 000 000 000 annually in this country.9 While print and television news reports of lightning-strike incidents to recreational athletes are frequent during the thunderstorm season, many people are unsure about what to do and where to go to improve their safety during thunderstorms. It is incumbent on all individuals, particularly those who are leaders in athletics and recreation, to appreciate the lightning hazard, learn the published lightning-safety guidelines, and act prudently, wisely, and in a spirit that will encourage safe behavior in others.

The guidelines presented in this article govern all outdoor activities, as well as indoor swimming-pool activities. The purpose of this position statement is to recommend lightning-safety policy guidelines and strategies and to educate athletic trainers and others involved with athletic or recreation activities about the hazards of lightning.
RECOMMENDATIONS

1. Formalize and implement a comprehensive, proactive lightning-safety policy or emergency action plan specific to lightning safety. The components of this policy should include the following:
   A. An established chain of command that identifies who is to make the call to remove individuals from the field or an activity.
   B. A designated weather watcher (i.e., a person who actively looks for the signs of threatening weather and notifies the chain of command if severe weather becomes dangerous).
   C. A means of monitoring local weather forecasts and warnings.
   D. A listing of specific safe locations (for each field or site) from the lightning hazard.
   E. The use of specific criteria for suspension and resumption of activities (refer to recommendations 4, 5, and 6).
   F. The use of the recommended lightning-safety strategies (refer to recommendations 7, 8, and 9).

2. The primary choice for a safe location from the lightning hazard is any substantial, frequently inhabited building. The electric and telephone wiring and plumbing pathways aid in grounding a building, which is why buildings are safer than remaining outdoors during thunderstorms. It is important not to be connected to these pathways while inside the structure during ongoing thunderstorms.

3. The secondary choice for a safer location from the lightning hazard is a fully enclosed vehicle with a metal roof and the windows closed. Convertible cars and golf carts do not provide protection from lightning danger. It is important not to touch any part of the metal framework of the vehicle while inside it during ongoing thunderstorms.

4. Seeking a safe structure or location at the first sign of lightning or thunder activity is highly recommended. By the time the flash-to-bang count approaches 30 seconds (or is less than 30 seconds), all individuals should already be inside or should immediately seek a safe structure or location. To use the flash-to-bang method, the observer begins counting when a lightning flash is sighted. Counting is stopped when the associated bang (thunder) is heard. Divide this count by 5 to determine the distance to the lightning flash (in miles). For example, a flash-to-bang count of 30 seconds equals a distance of 6 miles (9.66 km).

5. Postpone or suspend activity if a thunderstorm appears imminent before or during an activity or contest (regardless of whether lightning is seen or thunder heard) until the hazard has passed. Signs of imminent thunderstorm activity are darkening clouds, high winds, and thunder or lightning activity.

6. Once activities have been suspended, wait at least 30 minutes after the last sound of thunder or lightning flash before resuming an activity or returning outdoors. A message should be read over the public address system and lightning-safety tips should be placed in game programs alerting spectators and competitors about what to do and where to go to find a safer location during thunderstorm activity.

7. Extremely large athletic events are of particular concern with regard to lightning safety. Consider using a multidisciplinary approach to lessen lightning danger, such as integrating weather forecasts, real-time thunderstorm data, a weather watcher, and the flash-to-bang count to aid in decision making.

8. Avoid being in contact with, or in proximity to, the highest point of an open field or on the open water. Do not take shelter under or near trees, flag poles, or light poles.

9. Avoid taking showers and using plumbing facilities (including indoor and outdoor pools) and land-line telephones during thunderstorm activity. Cordless or cellular telephones are safer to use when emergency help is needed.

10. Individuals who feel their hair stand on end or skin tingle or hear crackling noises should assume the lightning-safe position (i.e., crouched on the ground, weight on the balls of the feet, feet together, head lowered, and ears covered). Do not lie flat on the ground.

11. Observe the following basic first-aid procedures, in order, to manage victims of lightning strike:
   A. Survey the scene for safety. Ongoing thunderstorms may still pose a threat to emergency personnel responding to the situation.
   B. Activate the local emergency management system.
   C. Move the victim carefully to a safer location, if needed.
   D. Evaluate and treat for apnea and asystole.
   E. Evaluate and treat for hypothermia and shock.
   F. Evaluate and treat for fractures.
   G. Evaluate and treat for burns.

12. All persons should maintain current cardiopulmonary resuscitation (CPR) and first-aid certification.

13. All individuals should have the right to leave an athletic site or activity, without fear of repercussion or penalty, in order to seek a safe structure or location if they feel they are in danger from impending lightning activity.

BACKGROUND

Lightning-Flash Development

Within a developing thunderstorm cloud, updrafts promote the collision of rising and descending ice and water particles, and the positive and negative charges are separated into distinct layers. Positive charges are taken via updrafts to the top of the cloud, while negative charges accumulate in the bottom of the cloud, creating the equivalent of a giant atmospheric battery.

A cloud-to-ground lightning flash is the product of the buildup and discharge of static electric energy between the charged regions of the cloud and the earth. The negatively charged lower region of the cloud induces a positive charge on the ground below. The tremendous electric forces between these 2 opposite charges initiate the lightning flash, which begins as a barely visible step leader moving in a series of steps downward from the cloud. Various objects on the ground (trees, chimneys, people, etc) can produce positively charged, upward streamers. The connection of the step leader with an upward streamer determines the connection point on the ground. After contact, a bright return stroke propagates upward from the ground, while electrons move downward toward the earth. This entire phenomenon happens in less than a fraction of a second, but a large amount of charge is transferred to the earth from the cloud.

Most lightning flashes have several return strokes, separated by only 0.004 to 0.005 seconds. The human eye can barely
resolve the intervals between the strokes that cause the lightning flash to appear to flicker. A lightning flash is essentially a brief spark, similar to that received from touching a doorknob after walking across a carpeted room. The lightning channel is approximately 2.54 cm (1 inch) in diameter and averages 4.83 to 8.05 km (3 to 5 miles) in vertical height but can be 9.66 km (6 miles) or higher. Cloud-to-ground lightning flashes typically have peak currents ranging from 10 000 to 200 000 Å, and the electric potential between the cloud and ground can be 10 000 000 to 100 000 000 V.

Thunder is created when lightning quickly heats the air around it, sometimes to temperatures greater than approximately 27 800°F (50 000°C), which is about 5 times hotter than the surface of the sun. The rapidly heated air around a lightning channel explodes, which in turn creates the sound we hear as a clap of thunder. The audible range of thunder is about 16.09 km (10 miles) but can be more or less depending on local conditions. Heat lightning is intracloud or intercloud lightning that is too distant for the accompanying thunder to be heard. Although it is possible to have lightning without thunder, thunder never occurs in the absence of lightning.

Lightning Casualty Demographics

On average, lightning kills approximately 100 people each year in this country, while many hundreds more are injured. The death toll from lightning for 1940 to 1973 was greater than that from tornadoes and hurricanes combined. Ninety-two percent of lightning casualties occur between May and September, while July has the greatest number of casualties. Furthermore, 45% of the deaths and 80% of the casualties occurred in these months between 10:00 AM and 7:00 PM, which coincides with the most likely time period for athletic or recreational events. For these reasons, it is accurate to say that lightning is the most dangerous and frequently encountered severe-storm hazard that most people experience each year.

The statistics on lightning casualty demographics compiled from the National Oceanographic and Atmospheric Administration publication Storm Data for the state of Colorado over the last few decades demonstrate an increase in the number of lightning casualties in persons involved in sports and outdoor recreation. Fifty-two percent of lightning casualties were people involved in outdoor recreation. In addition, these authors noted that the highest number of casualties from lightning was recorded in recreational and sports activities for each year of the study. During the 1960s, more than 30% of lightning casualties occurred during outdoor recreation activities; during the 1970s, that figure rose to 47%. Furthermore, the rate of increase of lightning casualties during sports was higher than the general United States population rate of increase during the same time period.

Lightning casualty statistics from Colorado demonstrate that the most common sites for fatalities were open fields (27%), near trees (16%), and close to water (13%). Statistics from the country as a whole mimic the numbers from Colorado. Open fields, ballparks, and playgrounds accounted for nearly 27% of casualties, and under trees (14%), water-related (8%), and golf-related (5%) deaths associated with lightning followed. All these fatalities had 1 common denominator: being near the highest object or being the tallest object in the immediate area. This single factor accounted for 56% of all fatalities from Colorado. Thus, it is imperative to avoid high ridges and high points on the terrain, and conversely, it is important to seek low-lying points on the terrain.

The height above ground has been demonstrated to play a prominent role in determining the strike probability. Therefore, it is important to understand why minimizing vertical height is critical in decreasing the chances of becoming a victim of lightning. Warning signs of a high electromagnetic field and imminent lightning strike include hair standing on end and sounds similar to bacon sizzling or cloth tearing. If these conditions occur, a cloud-to-ground lightning flash could strike in the immediate area. Therefore, one should immediately crouch in the lightning-safe position: feet together, weight on the balls of the feet, head lowered, and ears covered. This position is intended to minimize the probability of a direct strike by both lowering the person’s height and minimizing the area in contact with the surface of the ground. Taller objects are more likely to be struck (but not always) because their upward streamer occurs first, so that it is closer in proximity to the step leader coming downward from the cloud.

The ultimate message is that individuals in dangerous lightning situations should never wait to seek a safe location and pursue safety measures. It is important to be proactive by having all individuals inside a safe structure or location long before the lightning is close enough to be threatening.

Mechanisms of Lightning Injury

Injury from lightning can occur via 5 mechanisms. A direct strike most commonly occurs to the head, and lightning current enters the orifices. This mechanism explains why eye and ear injuries in lightning-strike victims are abundantly reported in the literature. The shock wave created by the lightning channel can also produce injuries, such as rupture of the tympanic membrane, a common clinical presentation in the lightning-strike victim. Recommending that individuals cover their ears while in the lightning-safe position may help to mitigate this type of injury.

The second mechanism, contact injury, occurs when the lightning victim is touching an object that is in the pathway of a lightning current. Side flash, the third mechanism, occurs when lightning strikes an object near the victim and then jumps from that object to the victim. This is the main danger to a person who is sheltered under an isolated, tall tree. An upward streamer is triggered by the tree but when this connects with the step leader, the resulting stroke jumps to the victim, who represents an additional pathway to ground.

The fourth mechanism, a step voltage or ground current, occurs when the lightning current flowing in the ground radiates outward in waves from the strike point. If 1 of the individual’s feet is closer to the strike than the other, a step voltage is created. Humans are primarily salt minerals in an aqueous solution, and a lightning current preferentially travels up from the earth through this solution (that is, the person) rather than through the ground. The greater the differential step voltage (ie, the greater the distance between the 2 feet), the greater the likelihood of injury. Placing one’s feet close together while in the crouched position and not lying flat on the ground are crucial in reducing the likelihood of injury from a step voltage or ground current.

Blunt injury is the fifth mechanism for lightning-strike injuries. Lightning current can cause violent muscular contractions that throw its victims many meters from the strike point.
Explosive and implosive forces created by the rapid heating and cooling by the lightning current are also enough to produce traumatic injuries.\textsuperscript{15}

**Common Effects of Lightning Injury**

While lightning kills nearly 100 people annually in this country, the protracted suffering of the survivors should not be underestimated. Although the only acute cause of death from lightning injury is cardiac arrest,\textsuperscript{20} the anoxic brain damage that can occur if the person is not rapidly resuscitated can be devastating. In addition, even for the survivor who did not sustain a cardiac arrest, permanent sequelae can include common brain-injury symptoms such as deficits in short-term memory and processing of new information, as well as severe and ongoing headaches, hyperirritability, sleep disturbances, and distractibility.\textsuperscript{21,22} Others may develop chronic pain syndromes or absence-type seizures. Frequently, survivors are unable to return to their previous level of function. They may not be able to continue in their jobs or in their educational pursuits and may be permanently disabled.

**Components of a Lightning-Safety Policy**

The purpose of formalizing a policy on lightning safety is to provide written guidelines for safety during lightning storms. Ninety-two percent of National Collegiate Athletic Association Division I athletics departments responding to a survey did not have a formal, written lightning-safety policy.\textsuperscript{12} The best means of reducing the lightning hazard to people is to be proactive. Athletic and recreational personnel should formalize and implement an emergency action plan specific to lightning safety before the thunderstorm season.\textsuperscript{1,11,13–15} Dissemination of the plan is paramount, so that all persons will know what to do and where to go to improve their own safety during thunderstorms. The 6 components of a lightning-safety policy or emergency action plan for lightning are discussed in the following paragraphs.

The first component in an emergency action plan or policy for lightning safety is the establishment of a specific chain of command that identifies the person who has the authority to remove participants from athletic venues or activities. The second is to appoint a weather watcher who actively looks for signs of developing local thunderstorms, such as high winds, darkening clouds, and any lightning or thunder.

The third element of a lightning-safety policy is the stipulation for monitoring local weather forecasts. One method is to use weather radios that broadcast information on daily forecasts and approaching storm systems. Weather radios are an excellent informational tool for general storm movement and strength. While this information is extremely important in decision making, the National Weather Service does not broadcast information on specific storm cells or lightning. Therefore, in addition to monitoring weather radios, it is essential that the weather watcher be on constant lookout for conditions in the immediate vicinity of the athletic event and compare these conditions with the weather radio information.

When a local area is placed under a severe-storm watch or warning by the National Weather Service, weather radios can be programmed to give audible alert tones. A watch indicates conditions are favorable for severe weather; a warning means severe weather has been detected in the locale, and all persons should take the necessary precautions to preserve their own safety. If severe storms are in the vicinity, all individuals should more intently monitor thunderstorm activity, such as severity and direction of movement of the storm. It may also mean that steps should be taken to remove athletes from the field or perhaps to postpone or suspend athletic or recreational activities during the event or before the storm begins.

**Safe Locations**

The fourth aspect of a lightning-safety policy, defining and listing safe structures or locations to evacuate to in the event of lightning, is of utmost importance. While there are reports of people being injured by lightning inside buildings,\textsuperscript{8} evacuating to a substantial building can considerably lower the risks of lightning injury compared with those of remaining outside during the thunderstorm. The lightning-safety policy should identify the safe structure or location specific to each venue. This information will enable individuals to know where to go in advance of any thunderstorm situation and appreciate how long it takes to get to the specific safe location from each field or event site.

The primary choice for a safe structure is any fully enclosed, substantial building.\textsuperscript{1,3,8,13–15} Ideally, the building should have plumbing, electric wiring, and telephone service. The lightning current is more likely to follow these pathways to ground, which aids in electrically grounding the structure.\textsuperscript{8} If a substantial building is not available, a fully enclosed vehicle with a metal roof and the windows completely closed is a reasonable alternative.\textsuperscript{1,3,13–15} It is not the rubber tires that make the vehicle safe but the metal enclosure that guides the lightning current around the passengers, rather than through them. Do not touch any part of the metal framework while inside the vehicle.\textsuperscript{8} Convertible vehicles and golf carts do not provide a high level of protection and cannot be considered safe from lightning.

**Unsafe Locations**

Unfortunately, those properties that serve to define a safe structure and improve the safety of its inhabitants also present a potential risk. Lightning current can enter a building via the electric or telephone wiring. It can also enter via a ground current through the incoming plumbing pipelines. This condition makes locker-room shower areas, swimming pools (indoor and outdoor), telephones, and electric appliances unsafe during thunderstorms because of the possible contact with current-carrying conduction. While such reports are rare, people have been killed or injured by lightning in their homes while talking on the telephone, taking a shower, or standing near household appliances such as dishwashers, stoves, or refrigerators.\textsuperscript{1,3,8,13–15}

From 1959 through 1965, lightning killed 4 people and injured 36 others while they were talking on the telephone. These numbers comprised 0.42\% (n = 960) of deaths and 2.1\% (n = 1736) of injuries for the period.\textsuperscript{5} Studying reports from Storm Data, researchers found that between 1959 and 1994, 2.4\% of lightning casualties were telephone related.\textsuperscript{2} Because they are not connected directly to a land-line phone, cellular and cordless telephones are reasonably safe alternatives for summoning help during a thunderstorm. It should be noted that injury from acoustic damage can occur via explosive static from the earpiece caused by a nearby lightning strike.
Even though a swimming pool may be indoors and apparently safe, it can be a dangerous location during thunderstorms.\textsuperscript{25} The current can be propagated through plumbing and electric connections via the underwater lights and drains of most swimming pools. Lightning current can also enter the building, either into the electric wiring inside the building or through underground plumbing pipelines that enter the building.\textsuperscript{8} If lightning strikes the building or ground nearby, the current will most likely follow these pathways to the swimmers through the water. Thus, indoor-pool activities are potentially dangerous and should be avoided during thunderstorms.\textsuperscript{25}

Small structures, such as rain or picnic shelters or athletic storage sheds, are generally not properly protected and should be avoided during thunderstorms. These locations may actually increase the risk of lightning strike via a side flash and cause injury to the occupants.

Criteria for Postponement and Resumption of Activities

The fifth component of any lightning-safety policy is to clearly describe criteria for both the suspension and resumption of athletic or recreational activities. Various technologies currently on the market propose to assist in determining when lightning is in the immediate area. Within the developing area of this lightning technology, data-based research is insufficient to either support or dispute companies’ claims regarding establishing when one is in danger of a lightning strike. Therefore the National Athletic Trainers’ Association promotes the flash-to-bang standard to warn people of imminent lightning danger. The flash-to-bang method is the easiest and most convenient means for determining the distance to a lightning flash and can also be used to determine when to suspend or postpone activities. The flash-to-bang method is based on the fact that light travels faster than sound, which travels at a speed of approximately 1.61 km (1 mile) every 5 seconds.\textsuperscript{1,8,13,14} To use the flash-to-bang method, begin counting on the lightning flash, and stop counting when the associated clap of thunder is heard. When storms have a high flash rate, it is important to correlate a specific flash with the thunder it produced. Divide the time to thunder (in seconds) by 5 to determine the distance (in miles) to the lightning flash.\textsuperscript{1,8,13,14} For example, an observer obtains a count of 30 seconds from the time he or she spots the flash to when the thunder is heard. Thirty divided by 5 equals 6; therefore, that lightning flash was 6 miles (9.66 km) from the observer.

The 30-second rule is not an arbitrary guideline. López and Holle\textsuperscript{26} studied storms in Oklahoma, Colorado, and Florida and found that in larger thunderstorms, the distance between successive flashes can be up to 6 miles (9.66 km) (ie, a flash-to-bang count of 30 seconds) in approximately 80% of the flash pairs. The authors also found the distance between successive flashes may be as great as 9 miles (14.48 km) or more, depending on local geography and atmospheric conditions. If a flash-to-bang count of 30 seconds is observed, the next flash could conceivably be at the observer’s location.

Another important factor to consider when using the flash-to-bang method is that, although a relatively rare occurrence, lightning has been reported to strike 16.09 km (10 miles) or more from where it is raining.\textsuperscript{1} Therefore, a flash-to-bang count of at least 30 seconds is strongly recommended as a determinant of when to suspend or postpone athletic or recreational activities.\textsuperscript{13–15} As the flash-to-bang count approaches 30 seconds, all persons should be seeking, or already inside, a safe structure or location. This is the minimal guideline when using the flash-to-bang method to halt athletic or recreational activities. Seeking a safe location at the first sign of thunder or lightning activity is also highly recommended.

Another facet of the lightning-safety policy is embodied in the “30–30 rule” (Table 1), which relies on the flash-to-bang method. If a game, practice, or other activity is suspended or postponed due to lightning activity, it is important to establish strict criteria in the lightning-safety policy for resumption of activities. Waiting at least 30 minutes after the last lightning flash or sound of thunder is recommended.\textsuperscript{13–15} When storm reports and flash data at the time of death or injury were compared, researchers found that the end of the storm, when the flash-rate frequency began to decline, was as deadly as the middle of the storm, when the lightning flash rate was at its peak. The authors postulated that once the flash rate begins to decline, people do not perceive the thunderstorm as dangerous and are struck by lightning when they return outdoors prematurely.\textsuperscript{1} An important adage for athletic trainers, coaches, and officials to remember is, “if you see it (lightning) flee it, if you hear it (thunder), clear it.”

The 30-minute rule can also be explained in another way. A typical thunderstorm moves at a rate of approximately 40.23 km (25 miles) per hour. Experts believe that 30 minutes allow the thunderstorm to be about 16.09 to 19.31 km (10 to 12 miles) from the area, minimizing the probability of a nearby, and therefore dangerous, lightning strike.\textsuperscript{15} Blue sky in the local area or a lack of rainfall are not adequate reasons to breach the 30-minute return-to-play rule. Lightning can strike far from where it is raining, even when the clouds begin to clear and show evidence of blue sky.\textsuperscript{1} This situation is often referred to as a “bolt out of the blue.” Each time lightning is observed or thunder is heard, the 30-minute clock should be reset.

Obligation to Warn

The recommendation for reading lightning-safety messages over public address systems and placing placards conspicuously around each venue resulted from a fatal lightning strike in Washington, DC, in May 1991.\textsuperscript{27} During a high school lacrosse game, a dangerous thunderstorm swept into the local area, and the game was suspended. Lightning killed 1 young person and injured 10 others who sought refuge under a tree. Many people stated that they did not know what to do or where to go to protect themselves from the dangers of lightning.

According to the basic principles of tort law, an individual has a duty to warn others of dangers that may not be obvious to a guest or subordinate of that person.\textsuperscript{28} Black et al\textsuperscript{29} defined the legal principle of “foreseeability” as “the ability to see or

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know in advance, eg, reasonable anticipation, that harm or injury is a likely result from certain acts or omissions.” With regard to dangerous lightning situations, it could be argued that an institution (or athletic department) has the duty to warn spectators, invited guests, and participants if conditions are such that lightning activity may be an imminent danger in the immediate area. Whereas lightning is understood by all to be a dangerous phenomenon, the importance of seeking safe shelter and the specific time that one should vacate to safety are generally not known. Based on research presented in this article regarding the number of lightning casualties resulting from the erroneous tendency of people to seek shelter under trees, it would be wise for an organization to promote lightning safety to its clientele and participants, including a list of specific safe locations or structures.

Warnings should be commensurate with the age and understanding of those involved. Announcements should be repeated over the public address system and colorful notices and safety instructions both placed in the event programs and posted in visible, high-traffic areas. Safety instructions should include the location of the nearest safe shelter, similar to airline pocket diagrams of nearest emergency exits. Being proactive with regard to the lightning threat demands not putting individuals at risk if a hazardous situation could have been prevented. If thunderstorm activity looks menacing before or during an event, consider canceling or postponing the event until the complete weather situation can be ascertained and determined to no longer be a threat. The first lightning flash from the thunderstorm cloud and storms that produce only a few flashes still pose a potential threat and should be treated as such. Every cloud-to-ground lightning flash is dangerous and potentially deadly and should not be taken lightly or viewed complacently. Therefore, it is the recommendation of the National Athletic Trainers’ Association to postpone or suspend athletic and recreational activities before their onset, if thunderstorm activity appears imminent.

**Prehospital Care of Victims**

If a lightning-strike victim presents in asystole or respiratory arrest, it is critical to initiate CPR as soon as safely possible.23 Because lightning-strike victims do not remain connected to a power source, they do not carry an electric charge and are safe to assess.30 However, during an ongoing thunderstorm, lightning activity in the local area still poses a deadly hazard for the medical team responding to the incident. The athletic trainer or other medical personnel should consider his or her own personal safety before venturing into a dangerous situation to render care.

If medical personnel assume the risk of entering a dangerous lightning situation to render care, the first priority should be to move the victim to a safe location. In this way, a hazardous situation can be neutralized for the athletic trainer, as well as the victim. It is unlikely that moving a victim to an area of greater safety for resuscitation will cause any serious injury to the victim.16 The primary and secondary survey of the victim’s condition can then be conducted once safety is reached.

It is not uncommon to find a lightning-strike victim unconscious, with fixed and dilated pupils and cold extremities and in cardiopulmonary arrest. Case studies of individuals with prolonged apnea and asystole after a lightning strike have demonstrated successful resuscitations using CPR.23,24,31 Once stopped, the heart will most likely spontaneously restart, but breathing centers in the brain may be damaged. Respiratory arrest lasts longer than cardiac arrest, leading to secondary asystole from hypoxia.16 Therefore, the basic principle of triage, “treat the living first,” should be reversed in cases involving casualties from a lightning strike. It is imperative to treat those persons who are “apparently dead” first by promptly initiating CPR. See Table 2 for quick-reference guidelines in evaluating and treating victims of lightning strike.

**CONCLUSIONS**

Due to its pervasiveness during the times that most athletic events occur, lightning is a significant hazard to the physically active population. Lightning-casualty statistics show an alarming rise in the number of lightning casualties in recreational and sports settings in recent decades.2,3,9 Each person must take responsibility for his or her own personal safety during thunderstorms.10 However, because people are often under the direction of others, whether they are children or adults participating in organized athletics, athletic trainers, coaches, teachers, and game officials must receive education about the hazards of lightning and become familiar with proved lightning-safety strategies. A policy is only as good as its compliance and unwavering, broad-based enforcement.

It is important to be much more wary of the lightning threat than the rain. Lightning can strike in the absence of rain, as well as from apparently clear blue skies overhead, even though a thunderstorm may be nearby. The presence of lightning or thunder should be the determining factor in postponing or suspending games and activities, not the amount of rainfall on the playing field. Lightning should be the only critical factor in decision making for athletic trainers, umpires, officials, referees, and coaches.

Athletic trainers, umpires, officials, referees, coaches, teachers, and parents can make a difference in reducing the number of lightning casualties if they (1) formalize and implement a lightning-safety policy or emergency action plan specific to lightning safety; (2) understand the qualifications of safe structures or locations, in addition to knowing where they are in relation to each athletic field or activity site; (3) understand the 30–30 rule as a minimal determinant of when to suspend activities and follow it; being conservative and suspending activities at the first sign of lightning or thunder activity is also prudent and wise; (4) practice and follow the published lightning-safety guidelines and strategies; (5) and maintain CPR and standard first-aid certification.

**ACKNOWLEDGMENTS**

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REFERENCES

Sport in today’s society is more popular than probably ever imagined. Large numbers of athletes participate in a variety of youth, high school, collegiate, professional, and recreational sports. As sport becomes more of a fixture in the lives of Americans, a burden of responsibility falls on the shoulders of the various organizations, coaches, parents, clinicians, officials, and researchers to provide an environment that minimizes the risk of injury in all sports. For example, the research-based recommendations made for football between 1976 and 1980 resulted in a significant reduction in the incidence of fatalities and nonfatal catastrophic injuries. In 1968, 36 brain and cervical spine fatalities occurred in high school and collegiate football. The number had dropped to zero in 1990 and has averaged about 5 per year since then.¹ This decrease was attributed to a variety of factors, including (1) rule changes, which have outlawed spearing and butt blocking, (2) player education about the rule changes and the consequences of not following the rules, (3) implementation of equipment standards, (4) availability of alternative assessment techniques, (5) a marked reduction in physical contact time during practice sessions, (6) a heightened awareness among clinicians of the dangers involved in returning an athlete to competition too early, and (7) the athlete’s awareness of the risks associated with concussion.

Research in the area of sport-related concussion has provided the athletic training and medical professions with valuable new knowledge in recent years. Certified athletic trainers, who on average care for 7 concussive injuries per year,² have been forced to rethink how they manage sport-related concussion. Recurrent concussions to several high-profile athletes, some of whom were forced into retirement as a result, have increased awareness among sports medicine personnel and the general public. Bridging the gap between research and clinical practice is the key to reducing the incidence and severity of sport-related concussion and improving return-to-play decisions. This position statement should provide valuable information and recommendations for certified athletic trainers (ATCs), physicians, and other medical professionals caring for athletes at the youth, high school, collegiate, and elite levels. The following recommendations are derived from the most recent scientific and clinic-based literature on sport-related concussion. The justification for these recommendations is presented in the summary statement following the recommendations. The summary statement is organized into the following sections: “Defining and Recognizing Concussion,” “Evaluating and Making the Return-to-Play Decision,” “Concussion Assessment Tools,” “When to Refer an Athlete to a Physician After Concussion,” “When to Disqualify an Athlete,” “Special Considerations for the Young Athlete,” “Home Care,” and “Equipment Issues.”

RECOMMENDATIONS

Defining and Recognizing Concussion

1. The ATC should develop a high sensitivity for the various mechanisms and presentations of traumatic brain injury (TBI), including mild, moderate, and severe cerebral concussion, as well as the more severe, but less common, head injuries that can cause damage to the brain stem and other vital centers of the brain.

2. The colloquial term “ding” should not be used to describe a sport-related concussion. This stunned confusional state is a concussion most often reflected by the athlete’s initial confusion, which may disappear within minutes, leaving
Evaluating and Making the Return-to-Play Decision

6. Working together, ATCs and team physicians should agree on a philosophy for managing sport-related concussion before the start of the athletic season. Currently 3 approaches are commonly used: (1) grading the concussion at the time of the injury, (2) deferring final grading until all symptoms have resolved, or (3) not using a grading scale but rather focusing on attention to the athlete’s recovery via symptoms, neurocognitive testing, and postural-stability testing. After deciding on an approach, the ATC-physician team should be consistent in its use regardless of the athlete, sport, or circumstances surrounding the injury.

7. For athletes playing sports with a high risk of concussion, baseline cognitive and postural-stability testing should be considered. In addition to the concussion injury assessment, the evaluation should also include an assessment of the cervical spine and cranial nerves to identify any cervical spine or vascular intracerebral injuries.

8. The ATC should record the time of the initial injury and document serial assessments of the injured athlete, noting the presence or absence of signs and symptoms of injury. The ATC should monitor vital signs and level of consciousness every 5 minutes after a concussion until the athlete’s condition improves. The athlete should also be monitored over the next few days after the injury for the presence of delayed signs and symptoms and to assess recovery.

9. Concussion severity should be determined by paying close attention to the severity and persistence of all signs and symptoms, including the presence of amnesia (retrograde and anterograde) and loss of consciousness (LOC), as well as headache, concentration problems, dizziness, blurred vision, and so on. It is recommended that ATCs and physicians consistently use a symptom checklist similar to the one provided in Appendix A.

10. In addition to a thorough clinical evaluation, formal cognitive and postural-stability testing is recommended to assist in objectively determining injury severity and readiness to return to play (RTP). No one test should be used solely to determine recovery or RTP, as concussion presents in many different ways.

11. Once symptom free, the athlete should be reassessed to establish that cognition and postural stability have returned to normal for that player, preferably by comparison with preinjury baseline test results. The RTP decision should be made after an incremental increase in activity with an initial cardiovascular challenge, followed by sport-specific activities that do not place the athlete at risk for concussion. The athlete can be released to full participation as long as no recurrent signs or symptoms are present.

Concussion Assessment Tools

12. Baseline testing on concussion assessment measures is recommended to establish the individual athlete’s “normal” preinjury performance and to provide the most reliable benchmark against which to measure postinjury recovery. Baseline testing also controls for extraneous variables (eg, attention deficit disorder, learning disabilities, age, and education) and for the effects of earlier concussion while also evaluating the possible cumulative effects of recurrent concussions.

13. The use of objective concussion assessment tools will help ATCs more accurately identify deficits caused by injury and postinjury recovery and protect players from the potential risks associated with prematurely returning to competition and sustaining a repeat concussion. The concussion assessment battery should include a combination of tests for cognition, postural stability, and self-reported symptoms known to be affected by concussion.

14. A combination of brief screening tools appropriate for use on the sideline (eg, Standardized Assessment of Concussion [SAC], Balance Error Scoring System [BESS], symptom checklist) and more extensive measures (eg, neuropsychological testing, computerized balance testing) to more precisely evaluate recovery later after injury is recommended.

15. Before instituting a concussion neuropsychological testing battery, the ATC should understand the test’s user requirements, copyright restrictions, and standardized instructions for administration and scoring. All evaluators should be appropriately trained in the standardized instructions for test administration and scoring before embarking on testing or adopting an instrument for clinical use. Ideally, the sports medicine team should include a neuropsychologist, but in reality, many ATCs may not have access to a neuropsychologist for interpretation and consultation, nor the financial resources to support a neuropsychological testing program. In this case, it is recommended that the ATC use screening instruments (eg, SAC, BESS, symptom checklist) that have been developed specifically for use by sports medicine clinicians without extensive
training in psychometric or standardized testing and that
do not require a special license to administer or interpret.

16. Athletic trainers should adopt for clinical use only those
neuropsychological and postural stability measures with
population-specific normative data, test-retest reliability,
clinical validity, and sufficient sensitivity and specificity
established in the peer-reviewed literature. These stan-
dards provide the basis for how well the test can distin-
guish between those with and without cerebral dysfunc-
tion in order to reduce the possibility of false-positive and
false-negative errors, which could lead to clinical deci-
dence-making errors.

17. As is the case with all clinical instruments, results from
assessment measures to evaluate concussion should be in-
tegrated with all aspects of the injury evaluation (eg, phys-
ical examination, neurologic evaluation, neuroimaging,
and player’s history) for the most effective approach to
injury management and RTP decision making. Decisions
about an athlete’s RTP should never be based solely on
the use of any one test.

When to Refer an Athlete to a Physician After
Concussion

18. The ATC or team physician should monitor an athlete
with a concussion at 5-minute intervals from the time of
the injury until the athlete’s condition completely clears
or the athlete is referred for further care. Coaches should
be informed that in situations when a concussion is sus-
pected but an ATC or physician is not available, their
primary role is to ensure that the athlete is immediately
seen by an ATC or physician.

19. An athlete with a concussion should be referred to a phy-
sician on the day of injury if he or she lost consciousness,
experienced amnesia lasting longer than 15 minutes, or
meets any of the criteria outlined in Appendix B.

20. A team approach to the assessment of concussion should
be taken and include a variety of medical specialists. In
addition to family practice or general medicine physician
referrals, the ATC should secure other specialist referral
sources within the community. For example, neurologists
are trained to assist in the management of patients expe-
riencing persistent signs and symptoms, including sleep
disturbances. Similarly, a neuropsychologist should be
identified as part of the sports medicine team for assisting
athletes who require more extensive neuropsychological
testing and for interpreting the results of neuropsycholog-
cal tests.

21. A team approach should be used in making RTP decisions
after concussion. This approach should involve input from
the ATC, physician, athlete, and any referral sources. The
assessment of all information, including the physical ex-
amination, imaging studies, objective tests, and exertional
tests, should be considered prior to making an RTP de-
cision.

When to Disqualify an Athlete

22. Athletes who are symptomatic at rest and after exertion
for at least 20 minutes should be disqualified from return-
ing to participation on the day of the injury. Exertional
exercises should include sideline jogging followed by
sprinting, sit-ups, push-ups, and any sport-specific, non-
contact activities (or positions or stances) the athlete might
need to perform on returning to participation. Athletes
who return on the same day because symptoms resolved
quickly (<20 minutes) should be monitored closely after
they return to play. They should be repeatedly reevaluated
on the sideline after the practice or game and again at 24
and 48 hours postinjury to identify any delayed onset of
symptoms.

23. Athletes who experience LOC or amnesia should be dis-
qualified from participating on the day of the injury.

24. The decision to disqualify from further participation on
the day of a concussion should be based on a comprehen-
sive physical examination; assessment of self-reported
postconcussion signs and symptoms; functional impair-
ments, and the athlete’s past history of concussions. If
assessment tools such as the SAC, BESS, neuropsycholog-
ical test battery, and symptom checklist are not used,
a 7-day symptom-free waiting period before returning to
participation is recommended. Some circumstances, how-
ever, will warrant even more conservative treatment (see
recommendation 25).

25. Athletic trainers should be more conservative with athletes
who have a history of concussion. Athletes with a history
of concussion are at increased risk for sustaining subse-
quent injuries as well as for slowed recovery of self-re-
ported postconcussion signs and symptoms, cognitive dys-
function, and postural instability after subsequent injuries.
In athletes with a history of 3 or more concussions and
experiencing slowed recovery, temporary or permanent
disqualification from contact sports may be indicated.

Special Considerations for the Young Athlete

26. Athletic trainers working with younger (pediatric) athletes
should be aware that recovery may take longer in
older athletes. Additionally, these younger athletes are ma-
turing at a relatively fast rate and will likely require more
frequent updates of baseline measures compared with old-
er athletes.

27. Many young athletes experience sport-related concussion.
Athletic trainers should play an active role in helping to
educate young athletes, their parents, and coaches about
the dangers of repeated concussions. Continued research
into the epidemiology of sport-related concussion in
young athletes and prospective investigations to determine
the acute and long-term effects of recurrent concussions
in younger athletes are warranted.

28. Because damage to the maturing brain of a young athlete
can be catastrophic (ie, almost all reported cases of sec-
ond-impact syndrome are in young athletes), athletes un-
der age 18 years should be managed more conservatively,
using stricter RTP guidelines than those used to manage
concussion in the more mature athlete.

Home Care

29. An athlete with a concussion should be instructed to avoid
taking medications except acetaminophen after the injury.
Acetaminophen and other medications should be given
DEFINING AND RECOGNIZING CONCUSSION

Perhaps the most challenging aspect of managing sport-related concussion is recognizing the injury, especially in athletes with no obvious signs that a concussion has actually occurred. The immediate management of the head-injured athlete depends on the nature and severity of the injury. Several terms are used to describe this injury, the most global being TBI, which can be classified into 2 types: focal and diffuse. Focal or posttraumatic intracranial mass lesions include subdural hematomas, epidural hematomas, cerebral contusions, and intracerebral hemorrhages and hematomas. These are considered uncommon in sport but are serious injuries; the ATC must be able to detect signs of clinical deterioration or worsening symptoms during serial assessments. Signs and symptoms of these focal vascular emergencies can include LOC, cranial nerve deficits, mental status deterioration, and worsening symptoms. Concern for a significant focal injury should also be raised if these signs or symptoms occur after an initial lucid period in which the athlete seemed normal.

Diffuse brain injuries can result in widespread or global disruption of neurologic function and are not usually associated with macroscopically visible brain lesions except in the most severe cases. Most diffuse injuries involve an acceleration-deceleration motion, either within a linear plane or in a rotational direction or both. In these cases, lesions are caused by the brain being shaken within the skull.5,5 The brain is suspended within the skull in cerebrospinal fluid (CSF) and has several dural attachments to bony ridges that make up the inner contours of the skull. With a linear acceleration-deceleration mechanism (side to side or front to back), the brain experiences a sudden momentum change that can result in tissue damage. The key elements of injury mechanism are the velocity of the head before impact, the time over which the force is applied, and the magnitude of the force.4,5 Rotational acceleration-deceleration injuries are believed to be the primary injury mechanism for the most severe diffuse brain injuries. Structural diffuse brain injury (diffuse axonal injury [DAI]) is the most severe type of diffuse injury because axonal disruption occurs, typically resulting in disturbance of cognitive functions, such as concentration and memory. In its most severe form, DAI can disrupt the brain-stem centers responsible for breathing, heart rate, and wakefulness.5,5

Cerebral concussion, which is the focus of this position statement, can best be classified as a mild diffuse injury and is often referred to as mild TBI (MTBI). The injury involves an acceleration-deceleration mechanism in which a blow to the head or the head striking an object results in 1 or more of the following conditions: headache, nausea, vomiting, dizziness, balance problems, feeling “slowed down,” fatigue, trouble sleeping, drowsiness, sensitivity to light or noise, LOC, blurred vision, difficulty remembering, or difficulty concentrating.6 In 1966, the Congress of Neurological Surgeons proposed the following consensus definition of concussion, subsequently endorsed by a variety of medical associations: “Concussion is a clinical syndrome characterized by immediate and transient impairment of neural functions, such as alteration of consciousness, disturbance of vision, equilibrium, etc. due to mechanical forces.”17 Although the definition received widespread consensus in 1966, more contemporary opinion (as concluded at the First International Conference on Concussion in Sport, Vienna, 2001)18 was that this definition fails to include many of the predominant clinical features of concussion, such as headache and nausea. It is often reported that there is no universal agreement on the standard definition or nature of concussion; however, agreement does exist on several features that incorporate clinical, pathologic, and biomechanical injury constructs associated with head injury:
1. Concussion may be caused by a direct blow to the head or elsewhere on the body from an “impulsive” force transmitted to the head.
2. Concussion may cause an immediate and short-lived impairment of neurologic function.
3. Concussion may cause neuropathologic changes; however, the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury.
4. Concussion may cause a gradient of clinical syndromes that may or may not involve LOC. Resolution of the clinical and cognitive symptoms typically follows a sequential course.
5. Concussion is most often associated with normal results on conventional neuroimaging studies.

Occasionally, players sustain a blow to the head resulting in a stunned confusional state that resolves within minutes. The colloquial term “ding” is often used to describe this initial state. However, the use of this term is not recommended because this stunned confusional state is still considered a concussion resulting in symptoms, although only very short in duration, that should not be dismissed in a cavalier fashion. It is essential that this injury be reevaluated frequently to determine if a more serious injury has occurred, because often the evolving signs and symptoms of a concussion are not evident until several minutes to hours later.

Although it is important for the ATC to recognize and eventually classify the concussive injury, it is equally important for the athlete to understand the signs and symptoms of a concussion as well as the potential negative consequences (eg, second-impact syndrome and predisposition to future concussions) of not reporting a concussive injury. Once the athlete has a better understanding of the injury, he or she can provide a more accurate report of the concussion history.

Mechanisms of Injury

A forceful blow to the resting, movable head usually produces maximum brain injury beneath the point of cranial impact (coup injury). A moving head hitting an unyielding object usually produces maximum brain injury opposite the site of cranial impact (contrecoup injury) as the brain shifts within the cranium. When the head is accelerated before impact, the brain lags toward the trailing surface, thus squeezing away the CSF and creating maximal shearing forces at this site. This brain lag actually thickens the layer of CSF under the point of impact, which explains the lack of coup injury in the moving head. Alternatively, when the head is stationary before impact, neither brain lag nor disproportionate distribution of CSF occurs, accounting for the absence of contrecoup injury and the presence of coup injury.

No scientific evidence suggests that one type of injury (coup or contrecoup) is more serious than the other or that symptoms present any differently. Many sport-related concussions are the result of a combined coup-contrecoup mechanism, involving damage to the brain on both the side of initial impact and the opposite side of the brain due to brain lag. Regardless of whether the athlete has sustained a coup, contrecoup, or combined coup-contrecoup injury, the ATC should manage the injury the same.

Three types of stresses can be generated by an applied force to injure the brain: compressive, tensile, and shearing. Compression involves a crushing force in which the tissue cannot absorb any additional force or load. Tension involves pulling or stretching of tissue, whereas shearing involves a force that moves across the parallel organization of the tissue. Brief, uniform compressive stresses are fairly well tolerated by neural tissue, but tension and shearing stresses are very poorly tolerated.

Neuroimaging of Cerebral Concussion

Traditionally, computed tomography (CT) and magnetic resonance imaging (MRI) have been considered useful in identifying certain types of brain lesions; however, they have been of little value in assessing less severe head injuries, such as cerebral concussion, and contributing to the RTP decision. A CT scan is often indicated emergently if a focal injury such as an acute subdural or epidural bleed is suspected; this study easily demonstrates acute blood collection and skull fracture, but an MRI is superior at demonstrating an isodense subacute or chronic subdural hematoma that may be weeks old. Newer structural MRI modalities, including gradient echo, perfusion, and diffusion-weighted imaging, are more sensitive for structural abnormalities (eg, vascular shearing) compared with other diagnostic imaging techniques. Functional imaging technologies (eg, positron emission tomography [PET], single-photon emission computerized tomography [SPECT], and functional MRI [fMRI]) are also yielding promising early results and may help define concussion recovery. Presently, no neuroanatomic or physiologic measurements can be used to determine the severity of a concussion or when complete recovery has occurred in an individual athlete after a concussion.

EVALUATING AND MAKING THE RETURN-TO-PLAY DECISION

Clinical Evaluation

Results from a thorough clinical examination conducted by both the ATC and the physician cannot be overlooked and should be considered very important pieces of the concussion puzzle. These evaluations should include a thorough history (including number and severity of previous head injuries), observation (including pupil responses), palpation, and special tests (including simple tests of memory, concentration, and coordination and a cranial nerve assessment). In many situations, a physician will not be present at the time of the concussion, and the ATC will be forced to act on behalf of the sports medicine team. More formal neuropsychological testing and postural-stability testing should be viewed as adjuncts to the initial clinical and repeat evaluations (see “Concussion Assessment Tools”). The ATC-physician team must also consider referral options to specialists such as neurologists, neurosurgeons, neuropsychologists, and neuro-otologists, depending on the injury severity and situation. Referrals for imaging tests such as CT, MRI, or electroneystagmography are also options that sometimes can aid in the diagnosis and/or management of sport-related concussion but are typically used only in cases involving LOC, severe amnesia, abnormal physical or neurologic findings, or increasing or intensified symptoms.

Determining Injury Severity

The definition of concussion is often expanded to include mild, moderate, and severe injuries. Several early grading scales and RTP guidelines early were proposed for classifying
and managing cerebral concussions.6,13–20 None of the scales have been universally accepted or followed with much consistency by the sports medicine community. In addition, most of these classification systems denote the most severe injuries as associated with LOC, which we now know is not always predictive of recovery after a brain injury.21,22 It is important for the ATC and other health care providers to recognize the importance of identifying retrograde amnesia and anterograde amnesia, LOC, and other signs and symptoms present and to manage each episode independently.

The ATC must recognize that no 2 concussions are identical and that the resulting symptoms can be very different, depending on the force of the blow to the head, the degree of metabolic dysfunction, the tissue damage and duration of time needed to recover, the number of previous concussions, and the time between injuries. All these factors must be considered when managing an athlete suffering from cerebral concussion.3 The 2 most recognizable signs of a concussion are LOC and amnesia; yet, as previously mentioned, neither is required for an injury to be classified as a concussion. A 2000 study of 1003 concussions sustained by high school and collegiate football players revealed that LOC and amnesia presented infrequently, 9% and 27% of all cases, respectively, whereas other signs and symptoms, such as headache, dizziness, confusion, disorientation, and blurred vision, were much more common.23 After the initial concussion evaluation, the ATC should determine whether the athlete requires more advanced medical intervention on an emergent basis or whether the team physician should be contacted for an RTP decision (Appendix B). It may be helpful if the injury is graded throughout the process, but this grading is likely to be more important for treating subsequent injuries than the current injury.

Most grading systems rely heavily on LOC and amnesia as indicators of injury severity. Recent research, however, suggests that these 2 factors, either alone or in combination, are not good predictors of injury severity. A number of authors have documented no association between brief (<1 minute) LOC and abnormalities on neuropsychological testing at 48 hours, raising concern for brief LOC as a predictor of recovery after concussion.8,22,24–27 Studies involving high school and collegiate athletes with concussion revealed no association between (1) LOC and duration of symptoms or (2) LOC and neuropsychological and balance tests at 3, 24, 48, 72, and 96 hours postinjury.21,28,29 In other words, athletes experiencing LOC were similar to athletes without LOC on these same injury-severity markers.

With respect to amnesia, the issue is more clouded because findings have been inconsistent. Several studies of nonathletes30–37 suggest that the duration of posttraumatic amnesia correlates with the severity and outcome of severe TBI but not with mild TBI or concussion.38–40 More contemporary studies of athletes with concussion are also clouded. Two unrelated, prospective studies of concussion suggest that the presence of amnesia best correlates with abnormal neuropsychological testing at 48 hours and with the duration and number of other postconcussion signs and symptoms.24,41 However, more recently, investigations of high school and collegiate athletes with concussion revealed no association between (1) amnesia and duration of symptoms or (2) amnesia and neuropsychological and balance tests at 3, 24, 48, 72, and 96 hours postinjury.21,28,29 Of importance in these studies is the significant association between symptom-severity score (within the initial 3 hours postinjury) and the total duration of symptoms (measured until asymptomatic). Although these findings suggest that initial symptom severity is probably a better indicator than either LOC or amnesia in predicting length of recovery, amnesia was recently found to predict symptom and neurocognitive deficits at 2 days postinjury.42 More research is needed in this area to help improve clinical decision making.

It has been suggested that LOC and amnesia, especially when prolonged, should not be ignored,43,44 but evidence for their usefulness in establishing RTP guidelines is scarce. Loss of consciousness, whether it occurs immediately or after an initially lucid interval, is important in that it may signify a more serious vascular brain injury. Other postconcussion signs and symptoms should be specifically addressed for presence and duration when the ATC is evaluating the athlete. Determining whether a cervical spine injury has occurred is also of major importance because it is often associated with head injury and should not be missed. If the athlete complains of neck pain or has cervical spine tenderness, cervical spine immobilization should be considered. If a cervical spine injury is ruled out and the athlete is taken to the sideline, a thorough clinical examination should follow, including a complete neurologic examination and cognitive evaluation. The ATC must note the time of the injury and then maintain a timed assessment form to follow the athlete’s symptoms and examinations serially. It is often difficult to pay attention to the time that has passed after an injury. Therefore, it is important for one member of the medical team to track time during the evaluation process and record all pertinent information. After an initial evaluation, the clinician must determine whether the injured athlete requires more advanced medical intervention and eventually grade the injury and make an RTP decision that can occur within minutes, hours, days, or weeks of the injury.

There are currently 3 approaches to grading sport-related concussion. One approach is to grade the concussion at the time of the injury on the basis of the signs and symptoms present at the time of the concussion and within the first 15 minutes after injury. The American Academy of Neurology Concussion Grading Scale (Table 1)6 has been widely used with this approach. It permits the ATC to grade the injury primarily on the basis of LOC and to provide the athlete, coach, and parent with an estimation of injury severity. A disadvantage to this approach is that many injuries behave differently than expected on initial evaluation, potentially creating more difficulties with the athlete, coach, or parent and making the RTP decision more challenging. Another approach is to grade the concussion on the basis of the presence and overall duration of symptoms. This approach is best addressed using the Cantu Evidence-Based Grading Scale (Table 2)43 which guides the ATC to grade the injury only after all concussion signs and symptoms have resolved. This scale places less emphasis on LOC as a potential predictor of subsequent impairment and additional weight on overall symptom dura-

| Grade 1 (mild) | Transient confusion; no LOC*; symptoms and mental status abnormalities last <15 min |
| Grade 2 (moderate) | Transient confusion; no LOC; symptoms and mental status abnormalities last >15 min |
| Grade 3 (severe) | Any LOC |

*LOC indicates loss of consciousness.
Table 2. Cantu Evidence-Based Grading System for Concussion

<table>
<thead>
<tr>
<th>Grade 1 (mild)</th>
<th>Grade 2 (moderate)</th>
<th>Grade 3 (severe)</th>
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<tbody>
<tr>
<td>No LOC*, PTA† &lt;30 min, PCSS‡ &lt;24 h</td>
<td>LOC &lt;1 min or PTA ≥30 min or PCSS ≥24 h or PTA ≥24 h or PCSS ≥7 d</td>
<td>LOC ≥1 min or PTA ≥24 h or PCSS ≥7 d</td>
</tr>
</tbody>
</table>

*LOC indicates loss of consciousness.  †PTA indicates posttraumatic amnesia (anterograde/retrograde).  ‡PCSS indicates postconcussion signs and symptoms other than amnesia.

Making the Return-to-Play Decision

The question raised most often regarding the concussion grading and RTP systems is one of practicality in the sport setting. Many clinicians believe that the RTP guidelines are too conservative and, therefore, choose to base decisions on clinical judgment of individual cases rather than on a general recommendation. It has been reported that 30% of all high school and collegiate football players sustaining concussions return to competition on the same day of injury; the remaining 70% average 4 days of rest before returning to participation. Many RTP guidelines call for the athlete to be symptom free for at least 7 days before returning to participation after a grade 1 or 2 concussion. Although many clinicians deviate from these recommendations and are more liberal in making RTP decisions, recent studies by Guskiewicz and McCrea et al suggest that perhaps the 7-day waiting period can minimize the risk of recurrent injury. On average, athletes required 7 days to fully recover after concussion. Same-season repeat injuries typically take place within a short window of time, 7 to 10 days after the first concussion, supporting the concept that there may be increased neuronal vulnerability or blood-flow changes during that time, similar to those reported by Giza, Hovda, et al in animal models.

Returning an athlete to participation should follow a progression that begins once the athlete is completely symptom free. All signs and symptoms should be evaluated using a graded symptom scale or checklist (described in “Concussion Assessment Tools”) when performing follow-up assessments and should be evaluated both at rest and after exertional maneuvers such as biking, jogging, sit-ups, and push-ups. Baseline measurements of neuropsychological and postural stability are strongly recommended for comparing with postinjury measurements. If these exertional tests do not produce symptoms, then the athlete can then participate in sport-specific skills that allow return to practice but should remain out of any activities that put him or her at risk for recurrent head injury. For the basketball player, this may include shooting baskets or participating in walk-throughs, and for the soccer player, this may include dribbling or shooting drills or other sport-specific activities. These restricted and monitored activities should be continued for the first few days after becoming asymptomatic. The athlete should be monitored periodically and after these sessions to determine if any symptoms develop or increase in intensity. Before returning to full contact participation, the athlete should be reassessed using neuropsychological and postural-stability tests if available. If all scores have returned to baseline or better, return to full participation can be considered after further clinical evaluation. It is strongly recommended that after recurrent injury, especially within-season repeat injuries, the athlete be withheld for an extended period of time (approximately 7 days) after symptoms have resolved.

CONCUSSION ASSESSMENT TOOLS

Sports medicine clinicians are increasingly using standardized methods to obtain a more objective measurement of postconcussion signs and symptoms, cognitive dysfunction, and postural instability. These methods allow the clinician to quantify the severity of injury and measure the player’s progress over the course of postinjury recovery. An emerging model of sport concussion assessment involves the use of brief screening tools to evaluate postconcussion signs and symptoms, cognitive functioning, and postural stability on the sideline immediately after a concussion and neuropsychological testing to track recovery further out from the time of injury. Ultimately, these tests, when interpreted with the physical examination and other aspects of the injury evaluation, assist the ATC and other sports medicine professionals in the RTP decision-making process.

Data from objective measures of cognitive functioning, postural stability, and postconcussion signs and symptoms are most helpful in making a determination about severity of injury and postinjury recovery when preinjury baseline data for an individual athlete are available. Baseline testing provides an indicator of what is “normal” for that particular athlete while also establishing the most accurate and reliable benchmark against which postinjury results can be compared. It is important to obtain a baseline symptom assessment in addition to baseline cognitive and other ability testing. Without baseline measures, the athlete’s postinjury performance on neuropsychological testing and other concussion assessment measures must be interpreted by comparison with available population normative values, which ideally are based on a large sample of the representative population.Normative data for competitive athletes on conventional (ie, paper-and-pencil) and computerized neuropsychological tests and other concussion assessment measures are now more readily available from large-scale research studies, but baseline data on an individual athlete still provide the greatest clinical accuracy in interpreting postinjury test results. When performing baseline testing, a suitable testing environment eliminates all distractions that could alter the baseline performance and enhances the likelihood that all athletes are providing maximal effort. Most important, all evaluators should be aware of a test’s user requirements and be appropriately trained in the standardized instructions for test administration and scoring before embarking on baseline testing or adopting a concussion testing paradigm for clinical use.

Several models exist for implementing baseline testing. Ide-
ally, preseason baseline testing is conducted before athletes are exposed to the risk of concussion during sport participation (eg, before contact drills during football). Some programs choose to conduct baseline testing as part of the preparticipation physical examination process. In this model, stations are established for various testing methods (eg, history collection, symptom assessment, neuropsychological testing, and balance testing), and athletes complete the evaluation sequence after being seen by the attending physician or ATC. This approach has the advantage of testing large groups of athletes in 1 session, while they are already in the mindset of undergoing a preseason physical examination. When preseason examinations are not conducted in a systematic group arrangement, alternative approaches can be considered. In any case, it is helpful to conduct all modules of baseline testing on players in 1 session to limit the complications of scheduling multiple testing times and to keep testing conditions constant for the athletes. One should allow adequate planning time (eg, 3 months) to implement a baseline testing module. Often this equates to conducting baseline testing for fall sports during the spring semester, before school is recessed for the summer. The benefits of interpreting postinjury data for an athlete after a concussion far outweigh the considerable time and human resources dedicated to baseline testing.

Collecting histories on individual athletes is also a vital part of baseline testing, especially in establishing whether the athlete has any history of concussion, neurologic disorder, or other remarkable medical conditions. Specifically with respect to concussion, it is important to establish (1) whether the player has any history of concussions and, if so, how many and (2) injury characteristics of previous concussions (eg, LOC, amnesia, symptoms, recovery time, time lost from participation, and medical treatment). For athletes with a history of multiple concussions, it is also important to clarify any apparent pattern of (1) concussions occurring as a result of lighter impacts, (2) concussions occurring closer together in time, (3) a lengthier recovery time with successive concussions, and (4) a less complete recovery with each injury. Documenting a history of attentional disorders, learning disability, or other cognitive development disorders is also critical, especially in interpreting an individual player’s baseline and postinjury performance on neuropsychological testing. If resources do not allow for preseason examinations in all athletes, at least a concerted effort to evaluate those athletes with a previous history of concussion should be made because of the awareness of increased risk for subsequent concussions in this group.

Postconcussion Symptom Assessment

Self-reported symptoms are among the more obvious and recognizable ways to assess the effects of concussion. Typical self-reported symptoms after a concussion include but are not limited to headache; dizziness; nausea; vomiting; feeling “in a fog”; feeling “slowed down”; trouble falling asleep; sleeping more than usual; fatigue; drowsiness; sensitivity to light or noise; unsteadiness or loss of balance; feeling “dinged,” dazed, or stunned; seeing stars or flashing lights; ringing in the ears; and double vision. Self-reported symptoms are referenced by many of the concussion grading scales. The presence of self-reported symptoms serves as a major contraindication for RTP, and, based on current recommendations, the athlete should be fully symptom free for at least 7 days at rest and during exertion before returning to play.

A number of concussion symptom checklists and scales have been used in both research and clinical settings. A symptom checklist that provides a list of concussion-related symptoms allows the athlete to report whether the symptom is present by responding either “yes” (experiencing the symptom) or “no” (not experiencing the symptom). A symptom scale is a summative measure that allows the athlete to describe the extent to which he or she is experiencing the symptom. These instruments commonly incorporate a Likert-type scale that allows the player to rate the severity or frequency of postconcussion symptoms. These scores are then summed to form a composite score that yields a quantitative measure of overall injury severity and a benchmark against which to track postinjury symptom recovery. Initial evidence has been provided for the structural validity of a self-report concussion symptom scale. Obtaining a baseline symptom score is helpful to establish any preexisting symptoms attributable to factors other than the head injury (eg, illness, fatigue, or somatization). Serial administration of the symptom checklist is the recommended method of tracking symptom resolution over time (see Appendix A).

Mental Status Screening

Cognitive screening instruments similar to the physician’s mini mental status examination objectify what is often a subjective impression of cognitive abnormalities. Various methods have been suggested for a systematic survey of mental status and cognitive function in the athlete with a concussion. The SAC was developed to provide sports medicine clinicians with a brief, objective tool for assessing the injured athlete’s mental status during the acute period after concussion (eg, sport sideline, locker room, and clinic). The SAC includes measures of orientation, immediate memory, concentration, and delayed recall that sum to 30 points. Lower scores on the SAC indicate more severe cognitive impairment. The SAC also includes assessments of strength, sensation, and coordination and a standard neurologic examination but should not replace the clinician’s thorough physical examination or referral for more extensive neuropsychological evaluation when indicated. Information about the occurrence and duration of LOC and amnesia is also recorded on the SAC. Alternate forms of the SAC are available to minimize the practice effects during retesting. The SAC takes about 5 minutes to administer and should be used only after the clinician’s thorough review of the training manual and instructional video on the administration, scoring, and interpretation of the instrument.

The SAC has demonstrated reliability and validity in detecting mental status changes after a concussion. Recent evidence suggests that a decline of 1 point or more from baseline classified injured and uninjured players with a level of 94% sensitivity and 76% specificity. The SAC is also sensitive to detecting more severe neurocognitive changes in injured athletes with LOC or amnesia associated with their concussions. The SAC is most useful in the assessment of acute cognitive dysfunction resulting from concussion, with sensitivity and specificity comparable with extensive neuropsychological testing batteries during the initial 2 to 3 days after concussion. As with neuropsychological testing, sensitivity and specificity of the SAC in concussion assessment are maximized when individual baseline test data are available.
Postural-Stability Assessment

A number of postural-stability tests have been used to assess the effects of concussion in the clinical and laboratory settings. The Romberg and stork stand were basic tests used to assess balance and coordination. Riemann et al. developed the Balance Error Scoring System (BESS) based on existing theories of posturography. The BESS uses 3 stance positions and tests on both a firm and a foam surface with the eyes closed (for a total of 6 trials). The administration and scoring procedures are found in several publications. The BESS has established good test-retest reliability and good concurrent validity when compared with laboratory forceplate measures and significant group differences, with an increased number of errors for days 1, 3, and 5 postinjury when compared with controls. Thus, the BESS can be used as a clinical measure in identifying balance impairment that could indicate a neurologic deficit.

The NeuroCom Smart Balance Master System (NeuroCom International, Clackamas, OR) is a forceplate system that measures vertical ground reaction forces produced by the body’s center of gravity moving around a fixed base of support. The Sensory Organization Test (SOT, NeuroCom International) is designed to disrupt various sensory systems, including the visual, somatosensory, and vestibular systems. The SOT consists of 6 conditions with 3 trials per condition, for a total of 18 trials, with each trial lasting 20 seconds. The complete administration has been described previously. The SOT has produced significant findings related to the assessment of concussion recovery. In a sample of 36 athletes with concussion, the mean stability (composite score) and vestibular and visual ratios demonstrated deficits for up to 5 days postinjury. The greatest deficits were seen 24 hours postinjury, and the athletes with concussion demonstrated a gradual recovery during the 5-day period to within 6% of baseline scores. These results were confirmed by Peterson et al. who found that these deficits continued for up to 10 days after concussion. These findings reveal a sensory interaction problem from the effects of concussion with measurable changes in overall postural stability.

Neuropsychological Testing

Neuropsychological testing has historically been used to evaluate various cognitive domains known to be preferentially susceptible to the effects of concussion and TBI. In recent years, neuropsychological testing to evaluate the effects of sport-related concussion has gained much attention in the sport concussion literature. The work of Barth et al., who studied more than 2000 collegiate football players from 10 universities, was the first project to institute baseline neuropsychological testing. Similar programs are now commonplace among many collegiate and professional teams, and interest is growing at the high school level. Several recent studies have supported the use of neuropsychological testing as a valuable tool to evaluate the cognitive effects and recovery after sport-related concussion, but its feasibility for sideline use is not likely realistic. As is the case with other concussion assessment tools, baseline neuropsychological testing is recommended, when possible, to establish a normative level of neurocognitive functioning for individual athletes. Baseline neuropsychological testing typically takes 20 to 30 minutes per athlete.

Before implementing a neuropsychological testing program, the ATC must consider several issues, including test-specific training requirements and methodologic issues, the practicality of baseline testing, the reliability and validity of individual tests comprising the test battery, and the protocol for interpretation of the postinjury test results. Barr provided an excellent review on the methodologic and professional issues associated with neuropsychological testing in sport concussion assessment. Most states require advanced training and licensure to purchase and use neuropsychological tests for clinical purposes. Neuropsychological tests are also copyright protected to prevent inappropriate distribution or use by unqualified professionals. At present, these requirements necessitate that a licensed psychologist, preferably one Board certified in clinical neuropsychology or with clinical experience in the evaluation of sport-related concussion, oversee and supervise the clinical application of neuropsychological testing for sport concussion assessment. These factors likely restrict how widely neuropsychological testing can be used to assess sport-related concussion, especially at the high school level and in rural areas where neuropsychologists are not readily available for consultation.

Neuropsychologists, ATCs, and sports medicine clinicians are faced with the challenge of designing a model that jointly upholds the testing standards of neuropsychology and meets the clinical needs of the sports medicine community without undue burden. The cost of neuropsychological testing, either conventional or computerized, is also a factor in how widely this method can be implemented, especially at the high school level. Consultation fees for the neuropsychologist can be considerable if work is not done on a pro bono basis, and some computerized testing companies charge a consulting fee for interpreting postinjury test results by telephone.

Although no clear indications exist as to which are the best individual neuropsychological tests to evaluate sport concussion, the use of multiple instruments as a “test battery” offers clinicians greater potential for recognizing any cognitive deficits incurred from the injury. A number of neuropsychological tests and test batteries have been used to assess sport-related concussion. Table 3 provides a brief description of the paper-and-pencil neuropsychological tests commonly used by neuropsychologists in the assessment of sport concussion. Sport concussion batteries should include measures of cognitive abilities most susceptible to change after concussion, including attention and concentration, cognitive processing (speed and efficiency), learning and memory, working memory, executive functioning, and verbal fluency. Tests of attention and concentration, memory function testing, and memory function testing have been reported as the most sensitive to the acute effects of concussion. The athlete’s age, sex, primary language, and level of education should be considered when selecting a test battery.

Computerized Neuropsychological Tests. Recently, a number of computerized neuropsychological testing programs have been designed for the assessment of athletes after concussion. The Automated Neuropsychological Assessment Metrics (ANAM), CogSport, Concussion Resolution Index, and Immediate Postconcussion Assessment and Cognitive Testing (ImPACT) are all currently available and have shown promise for reliable and valid concussion assessment (Table 4). The primary advantages to computerized testing are the ease of administration, ability to baseline test a large number of athletes in a short period of time, and multiple forms used within the testing paradigm to reduce the
practice effects. Collie et al.\(^71\) summarized the advantage and disadvantages of computerized versus traditional paper-and-pencil testing.

As outlined, in the case of conventional neuropsychological testing, several of the same challenges must be addressed before computerized testing becomes a widely used method of sport concussion assessment. Issues requiring further consideration include demonstrated test reliability; validity, sensitivity, and specificity in the peer-reviewed literature; required user training and qualifications; the necessary role of the licensed psychologist for clinical interpretation of postinjury test results; hardware and software issues inherent to computerized testing; and user costs.\(^71\) Progress is being made on many of these issues, but further clinical research is required to provide clinicians with the most effective neuropsychological assessment tools and maintain the testing standards of neuropsychology.

**Neuropsychological Testing Methods.** Neuropsychological testing is not a tool that should be used to diagnose the injury (ie, concussion); however, it can be very useful in measuring recovery once it has been determined that a concussion has occurred. The point(s) at which postinjury neuropsychological testing should occur has been a topic of debate. A variety of testing formats has been used to evaluate short-term recovery from concussion.\(^24,41,50,73,75,82\) Two approaches are most common. The first incorporates neuropsychological testing only after the injured player reports that his or her symptoms are completely gone. This approach is based on the conceptual foundation that an athlete should not participate while symptomatic, regardless of neuropsychological test performance. Unnecessary serial neuropsychological testing, in addition to being burdensome and costly to the athlete and medical staff, also introduces practice effects that may confound the interpretation of performance in subsequent postinjury testing sessions.\(^85\) The second approach incorporates neuropsychological testing at fixed time points (eg, postinjury day 1, day 7, and so on) to track postinjury recovery. This approach is often appropriate for prospective research protocols but is unnecessary in a clinical setting when the player is still symptomatic and will be withheld from competition regardless of the neuropsychological test results. In this model, serial testing can be used until neuropsychological testing returns to normal, preinjury levels and the player is completely symptom free.

Measuring “recovery” on neuropsychological tests and other clinical instruments is often a complex statistical matter, further complicated by practice effects and other psychometric dynamics affected by serial testing, even when preinjury baseline data are available for individual athletes. The use of statistical models that empirically identify meaningful change while controlling for practice effects on serial testing may provide the clinician with the most precise benchmark in deter-

### Table 3. Common Neuropsychological Tests Used in Sport Concussion Assessment

<table>
<thead>
<tr>
<th>Neuropsychological Test</th>
<th>Cognitive Domain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controlled Oral Word Association Test</td>
<td>Verbal fluency</td>
</tr>
<tr>
<td>Hopkins Verbal Learning Test</td>
<td>Verbal learning, immediate and delayed memory</td>
</tr>
<tr>
<td>Trail Making: Parts A and B</td>
<td>Visual scanning, attention, information processing speed, psychomotor speed</td>
</tr>
<tr>
<td>Wechsler Letter Number Sequencing Test</td>
<td>Verbal working memory</td>
</tr>
<tr>
<td>Wechsler Digit Span: Digits Forward and Digits Backward</td>
<td>Attention, concentration</td>
</tr>
<tr>
<td>Wechsler Digit Symbol Test</td>
<td>Psychomotor speed, attention, concentration</td>
</tr>
<tr>
<td>Symbol Digit Modalities Test</td>
<td>Psychomotor speed, attention, concentration</td>
</tr>
<tr>
<td>Paced Auditory Serial Addition Test</td>
<td>Attention, concentration</td>
</tr>
<tr>
<td>Stroop Color Word Test</td>
<td>Attention, information processing speed</td>
</tr>
</tbody>
</table>

### Table 4. Computerized Neuropsychological Tests

<table>
<thead>
<tr>
<th>Neuropsychological Test</th>
<th>Developer (Contact Information)</th>
<th>Cognitive Domains</th>
</tr>
</thead>
<tbody>
<tr>
<td>Automated Neuropsychological Assessment Metrics (ANAM)</td>
<td>National Rehabilitation Hospital Assistive Technology and Neuroscience Center, Washington, DC(^44) (<a href="mailto:jsb2@mhg.edu">jsb2@mhg.edu</a>)</td>
<td>Simple Reaction Metrics, Sternberg Memory, Math Processing, Continuous Performance, Matching to Sample, Spatial Processing, Code Substitution</td>
</tr>
<tr>
<td>CogSport</td>
<td>CogState Ltd, Victoria, Australia (<a href="http://www.cogsport.com">www.cogsport.com</a>)</td>
<td>Simple Reaction Time, Complex Reaction Time, One-Back, Continuous Learning</td>
</tr>
<tr>
<td>Immediate Postconcussion Assessment and Cognitive Testing (ImPACT)</td>
<td>University of Pittsburgh Medical Center, Pittsburgh, PA (<a href="http://www.impacttest.com">www.impacttest.com</a>)</td>
<td>Verbal Memory, Visual Memory, Information Processing Speed, Reaction Time, Impulse Control</td>
</tr>
</tbody>
</table>
mining postinjury recovery, above and beyond the simple conclusion that the player is “back to baseline.” The complexity of this analysis is the basis for the neuropsychologist overseeing the clinical interpretation of test data to determine injury severity and recovery. Further research is required to clarify the guidelines for determining and tracking recovery on specific measures after concussion. The clinician should also be aware that any concussion assessment tool, either brief screening instruments or more extensive neuropsychological testing, comes with some degree of risk for false negatives (eg, a player performs within what would be considered the normal range on the measure before actually reaching a complete clinical recovery after concussion). Therefore, test results should always be interpreted in the context of all clinical information, including the player’s medical history. Also, caution should be exercised in neuropsychological test interpretation when pre-injury baseline data do not exist. Numerous factors apart from the direct effects of concussion can influence test performance (Table 5).

**WHEN TO REFER AN ATHLETE TO A PHYSICIAN AFTER CONCUSSION**

Although most sport-related concussions are considered mild head injuries, the potential exists for complications and life-threatening injuries. Each ATC should be concerned about the potential for the condition of an athlete with a concussion to deteriorate. This downward trend can occur immediately (minutes to hours) or over several days after the injury. As discussed earlier, the spectrum of sport-related head injuries includes more threatening injuries, such as epidural and subdural hematomas and second-impact syndrome. Postconcussion syndrome, however, is a more likely consequence of a sport-related concussion. Not every sport-related concussion warrants immediate physician referral, but ATCs must be able to recognize those injuries that require further attention and provide an appropriate referral for advanced care, which may include neuroimaging. Serial assessments and physician follow-up are important parts of the evaluation of the athlete with a concussion. Referrals should be made to medical personnel with experience managing sport-related concussion. The ATC should monitor vital signs and level of consciousness every 5 minutes after a concussion until the athlete’s condition stabilizes and improves. The athlete should also be monitored over the next few hours and days after the injury for delayed signs and symptoms and to assess recovery. Appendix B outlines scenarios that warrant physician referral or, in many cases, transport to the nearest hospital emergency department.

**WHEN TO DISQUALIFY AN ATHLETE**

Return to participation after severe or repetitive concussive injury should be considered only if the athlete is completely symptom free and has a normal neurologic examination, normal neuropsychological and postural-stability examinations, and, if obtained, normal neuroimaging studies (ie, MRI with gradient echo). It may not be practical or even possible to use all these assessments in all athletes or young children, but a cautious clinical judgment should take into account all evaluation options. Each injured athlete should be considered individually, with consideration for factors including age, level of participation, nature of the sport (high risk versus low risk), and concussion history.

Standardized neuropsychological testing, which typically assesses orientation, immediate and delayed memory recall, and concentration may assist the ATC and physician in determining when to disqualify an athlete from further participation.60 Balance testing may provide additional information to assist the clinician in the decision-making process of whether to disqualify an individual after a concussion.52 When to disqualify the athlete is one of the most important decisions facing the ATC and team physician when dealing with an athlete suffering from a concussion. This includes not only when to disqualify for a single practice or event but also when to disqualify for the season or for a career.

**Disqualifying for the Game or Practice**

The decision to disqualify an individual from further participation on the day of the concussive episode is based on the sideline evaluation, the symptoms the athlete is experiencing, the severity of the apparent symptoms, and the patient’s past history.56 The literature is clear: any episode involving LOC or persistent symptoms related to concussion (headache, dizziness, amnesia, and so on), regardless of how mild and transient, warrants disqualification for the remainder of that day’s activities.8,9,13,19,43,60,87 More recent studies of high school and collegiate athletes underscore the importance of ensuring that the athlete is symptom free before returning to participation on the same day; even when the player is symptom free within 15 to 20 minutes after the concussive episode, he or she may still demonstrate delayed symptoms or depressed neurocognitive levels. Lovell et al88 found significant memory deficits 36 hours postinjury in athletes who were symptom free within 15 minutes of a mild concussion. Guskiewicz et al21 found that 33% (10/30) of the players with concussion who returned on the same day of injury experienced delayed onset of symptoms at 3 hours postinjury, as compared with only 12.6% (20/158) of those who did not return to play on the same day of injury. Although more prospective work is needed in this area, these studies raise questions as to whether the RTP criteria for grade 1 (mild) concussions are conservative enough.
Disqualifying for the Season

Guidelines from Cantu43 and the American Academy of Neurology6 both recommend termination of the season after the third concussion within the same season. The decision is more difficult if one of the injuries was more severe or was a severe injury resulting from a minimal blow, suggesting that the athlete’s brain may be at particular risk for recurrent injury. In addition, because many athletes participate in year-round activities, once they are disqualified for the “season,” it may be difficult to determine at what point they can resume contact play. Other issues without clear-cut answers in the literature are when to disqualify an athlete who has not been rendered unconscious and whose symptoms cleared rapidly or one who suffered multiple mild to moderate concussions throughout the career and whether youth athletes should be treated differently for initial and recurrent concussive injuries.

Disqualifying for the Career

When to disqualify an athlete for a career is a more difficult question to answer. The duration of symptoms may be a better criterion as to when to disqualify an athlete for the season or longer. Merrill Hoge, Eric Lindros, Chris Miller, Al Toon, and Steve Young provide highly publicized cases of athletes sustaining multiple concussions with recurrent or postconcussion signs and symptoms that lasted for lengthy periods of time.43

Once an athlete has suffered a concussion, he or she is at increased risk for subsequent head injuries.21,43,82 Gusiewicz et al21,23 found that collegiate athletes had a 3-fold greater risk of suffering a concussion if they had sustained 3 or more previous concussions in a 7-year period and that players with 2 or more previous concussions required a longer time for total symptom resolution after subsequent injuries.21 Players also had a 3-fold greater risk for subsequent concussions in the same season,23 whereas recurrent, in-season injuries occurred within 10 days of the initial injury 92% of the time.21 In a similar study of high school athletes, Collins et al82 found that athletes with 3 or more prior concussions were at an increased risk of experiencing LOC (8-fold greater risk), anterograde amnesia (5.5-fold greater risk), and confusion (5.1-fold greater risk) after subsequent concussion. Despite the increasing body of literature on this topic, debate still surrounds the question of how many concussions are enough to recommend ending the player’s career. Some research suggests that the magic number may be 3 concussions in a career.21,23,82 Although these findings are important, they should be carefully interpreted because concussions present in varying degrees of severity, and all athletes do not respond in the same way to concussive insults. Most important is that these data provide evidence for exercising caution when managing younger athletes with concussion and athletes with a history of previous concussions.

SPECIAL CONSIDERATIONS FOR THE YOUNG ATHLETE

Many epidemiologic studies on concussion have focused on professional or collegiate athletes. However, this focus seems to now be shifting to the high school level and even to youth sports. Special consideration must be given to the young athlete. The fact that the brain of the young athlete is still developing cannot be ignored, and the effect of concussion on the developing brain is still not entirely understood. Even sub-

tle damage may lead to deficits in learning that adversely influence development. Therefore, it has been suggested that pediatric athletes suffering a concussion should be restricted from further participation for the day and that additional consideration should be given as to when to return these individuals to activity.46

Recent epidemiologic investigations of head-injury rates in high school athletes have shown that 13.3% of all reported injuries in high school football affect the head and neck, whereas the numbers in other sports range from 1.9% to 9.5% in baseball and wrestling, respectively.59 Gusiewicz et al23 prospectively examined concussion incidence in high school and collegiate football players and found that the greatest incidence was at the high school level (5.6%), compared with the National Collegiate Athletic Association Division I (4.4%), Division II (4.5%), and Division III (5.5%).

Authors who have tracked symptoms and neuropsychological function after concussion suggest that age-related differences exist between high school and collegiate athletes with regard to recovery. Lovell et al41 reported that the duration of on-field mental status changes in high school athletes, such as retrograde amnesia and posttraumatic confusion, was related to the presence of memory impairment at 36 hours, 4 days, and 7 days postinjury as well as slower resolution of self-reported symptoms. These findings further emphasize the need to collect these on-field measures after concussion and to use the information wisely in making RTP decisions, especially when dealing with younger athletes. Field et al90 found that high school athletes who sustained a concussion demonstrated prolonged memory dysfunction compared with collegiate athletes who sustained a concussion. The high school athletes performed significantly worse on select tests of memory than age-matched control subjects at 7 days postinjury when compared with collegiate athletes and their age-matched control subjects. We hope these important studies and others will eventually lead to more specific guidelines for managing concussions in high school athletes.

Very few investigators have studied sport-related injuries in the youth population, and even fewer focused specifically on sport-related concussion. One group91 reported that 15% of the children (mean = 8.34 ± 5.31 years) who were admitted to hospitals after MTBI suffered from a sport-related mechanism of injury. Another group92 found that sport-related head injury accounted for 3% of all sport-related injuries and 24% of all serious head injuries treated in an emergency department. Additionally, sport-related concussion represented a substantial percentage of all head injuries in children under the age of 10 years (18.2%) and 10- to 14-year-old (53.4%) and 15- to 19-year-old (42.9%) populations.92 Thus, sport-related head injury has a relatively high incidence rate and is a significant public health concern in youth athletes, not just participants at higher competitive levels.

Although no prospective investigations in younger athletes (younger than 15 years old) have been undertaken regarding symptom resolution and cognitive or postural-stability recovery, Valovich McLeod et al93 recently determined the reliability and validity of brief concussion assessment tools in a group of healthy young athletes (9–14 years old). The SAC is valid within 48 hours of injury and reliable for testing of youths above age 5 years, but younger athletes score slightly below high school and collegiate athletes.59 This issue is remedied, however, if preseason baseline testing is conducted for all players and a preinjury baseline score established for each
athlete against which changes resulting from concussion can be detected and other factors that affect test performance can be controlled. Users of standardized clinical tools should be aware of the effects of age and education on cognitive test performance and make certain to select the appropriate normative group for comparison when testing an injured athlete at a specific competitive level. Uncertainties about the effects of concussion on young children warrant further study.

**HOME CARE**

Once the athlete has been thoroughly evaluated and determined to have sustained a concussion, a comprehensive medical management plan should be implemented. This plan should include frequent medical evaluations and observations, continued monitoring of postconcussion signs and symptoms, and postinjury cognitive and balance testing. If symptoms persist or worsen or the level of consciousness deteriorates at all after a concussion, neuroimaging should be performed. Although scientific evidence for the evaluation and resolution of the concussion is ample, specific management advice to be given to the athlete on leaving the athletic training room is lacking. Athletic trainers and hospital emergency rooms have created various home instruction forms, but minimal scientific evidence supports these instructions. However, despite these limitations, a concussion instruction form (Appendix C) should be given to the athlete and a responsible adult who will have direct contact with the athlete for the initial 24 hours after the injury. This form helps the companion to know what signs and symptoms to watch for and provides useful recommendations on follow-up care.

**Medications**

At this time, the clinician has no evidence-based pharmacologic treatment options for an athlete with a concussion. Most pharmacologic studies have been performed in severely head-injured patients. It has been suggested that athletes with concussion avoid medications containing aspirin or nonsteroidal anti-inflammatories, which decrease platelet function and potentially increase intracranial bleeding, mask the severity and duration of symptoms, and possibly lead to a more severe injury. It is also recommended that acetaminophen (Tylenol, McNeil Consumer & Specialty Pharmaceuticals, Fort Washington, PA) be used sparingly in the treatment of headache-like symptoms in the athlete with a concussion. Other substances to avoid during the acute postconcussion period include those that adversely affect central nervous function, in particular alcohol and narcotics.

**Wake-Ups and Rest**

Once it has been determined that a concussion has been sustained, a decision must be made as to whether the athlete can return home or should be considered for overnight observation or admission to the hospital. For more severe injuries, the athlete should be evaluated by the team physician or emergency room physician if the team physician is not available. If the athlete is allowed to return home or to the dormitory room, the ATC should counsel a friend, teammate, or parent to closely monitor the athlete. Traditionally, part of these instructions included a recommendation to wake up the athlete every 3 to 4 hours during the night to evaluate changes in symptoms and rule out the possibility of an intracranial bleed, such as a subdural hematoma. This recommendation has raised some debate about unnecessary wake-ups that disrupt the athlete’s sleep pattern and may increase symptoms the next day because of the combined effects of the injury and sleep deprivation. It is further suggested that the concussed athlete have a teammate or friend stay during the night and that the athlete not be left alone. No documented evidence suggests what severity of injury requires this treatment. However, a good rule to use is if the athlete experienced LOC, had prolonged periods of amnesia, or is still experiencing significant symptoms, he or she should be awakened during the night. Both oral and written instructions should be given to both the athlete and the caregiver regarding waking. The use of written and oral instructions increases the compliance to 55% for purposeful waking in the middle of the night. In the treatment of concussion, complete bed rest was ineffective in decreasing post-concussion signs and symptoms. The athlete should avoid activities that may increase symptoms (eg, staying up late studying and physical education class) and should resume normal activities of daily living, such as attending class and driving, once symptoms begin to resolve or decrease in severity. As previously discussed, a graded test of exertion should be used to determine the athlete’s ability to safely return to full activity.

**Diet**

Evidence is limited to support the best type of diet for aiding in the recovery process after a concussion. A cascade of neurochemical, ionic, and metabolic changes occur after brain injury. Furthermore, some areas of the brain demonstrate glycolytic increases and go into a state of metabolic depression as a result of decreases in both glucose and oxidative metabolism with a reduction in cerebral blood flow. Severely brain-injured subjects ate larger meals and increased their daily caloric intake when compared with controls. Although limited information is available regarding the recommended diet for the management of concussion, it is well accepted that athletes should be instructed to avoid alcohol, illicit drugs, and central nervous system medications that may interfere with cognitive function. A normal, well-balanced diet should be maintained to provide the needed nutrients to aid in the recovery process from the injury.

**EQUIPMENT ISSUES**

**Helmets and Headgear**

Although wearing a helmet will not prevent all head injuries, a properly fitted helmet for certain sports reduces the risk of such injuries. A poorly fitted helmet is limited in the amount of protection it can provide, and the ATC must play a role in enforcing the proper fitting and use of the helmet. Protective sport helmets are designed primarily to prevent catastrophic injuries (ie, skull fractures and intracranial hematomas) and are not designed to prevent 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.

The National Collegiate Athletic Association requires helmets to be worn for the following sports: baseball, field hockey (goalkeepers only), football, ice hockey, women’s lacrosse (goalkeepers only), men’s lacrosse, and skiing. Helmets are
Mouth Guards

The wearing of a mouth guard is thought by some to provide additional protection for the athlete against concussion by either reducing the risk of injury or reducing the severity of the injury itself. Mouth guards aid in the separation between the head of the condyle of the mandible and the base of the skull. It is thought that wearing an improperly fitted mouth guard or none at all increases this contact point. This theory, which is based on Newtonian laws of physics, suggests that the increased separation between adjacent structures increases the time to contact, thus decreasing the amount of contact and decreasing the trauma done to the brain. However, no biomechanical studies support the theory that the increased separation results in less force being delivered to the brain.

High school football and National Collegiate Athletic Association football rules mandate the wearing of a mouth guard, but the National Football League rulebook does not require players to wear a mouth guard. The National Collegiate Athletic Association requires mouth guards to be worn by all athletes in football, field hockey, ice hockey, and lacrosse. ATCs and coaches should mandate the regular use of mouth guards because a properly fitted mouth guard, with no alterations such as cutting off the back part, is of great value in protecting the teeth and preventing fractures and avulsions that could require many years of expensive dental care.

ACKNOWLEDGMENTS

We gratefully acknowledge the efforts of Kent Struebe, PhD, ATC; Scott Anderson, MS, ATC; Michael Collins, PhD; Vito A. Perriello, Jr, MD, PhD; Karen Johnston, MD, PhD; and the Pronouncements Committee in the preparation of this document.

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## Graded Symptom Checklist (GSC)

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Time of injury</th>
<th>2-3 Hours postinjury</th>
<th>24 Hours postinjury</th>
<th>48 Hours postinjury</th>
<th>72 Hours postinjury</th>
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<tbody>
<tr>
<td>Blurred vision</td>
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<td>Dizziness</td>
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<td>Drowsiness</td>
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<td>Excess sleep</td>
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<td>Easily distracted</td>
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<tr>
<td>Fatigue</td>
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<tr>
<td>Feel “in a fog”</td>
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<tr>
<td>Feel “slowed down”</td>
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<td>Headache</td>
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<td>Inappropriate emotions</td>
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<td>Irritability</td>
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<tr>
<td>Loss of consciousness</td>
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<td>Loss or orientation</td>
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<td>Memory problems</td>
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<tr>
<td>Nausea</td>
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<td>Nervousness</td>
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<td>Personality change</td>
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<tr>
<td>Poor balance/coordination</td>
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<td>Poor concentration</td>
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<td>Ringing in ears</td>
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<td>Sadness</td>
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<tr>
<td>Seeing stars</td>
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<tr>
<td>Sensitivity to light</td>
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<td>Sensitivity to noise</td>
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<tr>
<td>Sleep disturbance</td>
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<td>Vacant stare/glassy eyed</td>
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<tr>
<td>Vomiting</td>
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**NOTE:** The GSC should be used not only for the initial evaluation but for each subsequent follow-up assessment until all signs and symptoms have cleared at rest and during physical exertion. In lieu of simply checking each symptom present, the ATC can ask the athlete to grade or score the severity of the symptom on a scale of 0-6, where 0=not present, 1=mild, 3=moderate, and 6=most severe.
Appendix B. Physician Referral Checklist

Day-of-injury referral

1. Loss of consciousness on the field
2. Amnesia lasting longer than 15 min
3. Deterioration of neurologic function
4. Decreasing level of consciousness
5. Decrease or irregularity in respirations
6. Decrease or irregularity in pulse
7. Increase in blood pressure
8. Unequal, dilated, or unreactive pupils
9. Cranial nerve deficits
10. Any signs or symptoms of associated injuries, spine or skull fracture, or bleeding
11. Mental status changes: lethargy, difficulty maintaining arousal, confusion, or agitation
12. Seizure activity
13. Vomiting
14. Motor deficits subsequent to initial on-field assessment
15. Sensory deficits subsequent to initial on-field assessment
16. Balance deficits subsequent to initial on-field assessment
17. Cranial nerve deficits subsequent to initial on-field assessment
18. Postconcussion symptoms that worsen
19. Additional postconcussion symptoms that worsen as compared with those on the field
20. Athlete is still symptomatic at the end of the game (especially at high school level)

Delayed referral (after the day of injury)

1. Any of the findings in the day-of-injury referral category
2. Postconcussion symptoms worsen or do not improve over time
3. Increase in the number of postconcussion symptoms reported
4. Postconcussion symptoms begin to interfere with the athlete’s daily activities (ie, sleep disturbances or cognitive difficulties)

*Requires that the athlete be transported immediately to the nearest emergency department.

Appendix C. Concussion Home Instructions

I believe that ____________________________ sustained a concussion on ____________________________. To make sure he/she recovers, please follow the following important recommendations:

1. Please remind ____________________________ to report to the athletic training room tomorrow at __________ for a follow-up evaluation.

2. Please review the items outlined on the enclosed Physician Referral Checklist. If any of these problems develop prior to his/her visit, please call ____________________________ at ____________________________ or contact the local emergency medical system or your family physician. Otherwise, you can follow the instructions outlined below.

It is OK to:
- Use acetaminophen (Tylenol) for headaches
- Use ice pack on head and neck as needed for comfort
- Eat a light diet
- Return to school
- Go to sleep
- Rest (no strenuous activity or sports)

There is NO need to:
- Check eyes with flashlight
- Wake up every hour
- Test reflexes
- Stay in bed

Do NOT:
- Drink alcohol
- Eat spicy foods

Specific recommendations:

Recommendations provided to: ____________________________

Recommendations provided by: ____________________________ Date: __________ Time: __________

Please feel free to contact me if you have any questions. I can be reached at: ____________________________

Signature: ____________________________ Date: ____________________________
National Athletic Trainers’ Association Position Statement: Prevention of Pediatric Overuse Injuries

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Objective: To provide certified athletic trainers, physicians, and other health care professionals with recommendations on best practices for the prevention of overuse sports injuries in pediatric athletes (aged 6–18 years).

Background: Participation in sports by the pediatric population has grown tremendously over the years. Although the health benefits of participation in competitive and recreational athletic events are numerous, one adverse consequence is sport-related injury. Overuse or repetitive trauma injuries represent approximately 50% of all pediatric sport-related injuries. It is speculated that more than half of these injuries may be preventable with simple approaches.

Recommendations: Recommendations are provided based on current evidence regarding pediatric injury surveillance, identification of risk factors for injury, preparticipation physical examinations, proper supervision and education (coaching and medical), sport alterations, training and conditioning programs, and delayed specialization.

Key Words: adolescents, children, chronic injuries, microtrauma, growth, development

Overuse injuries in the pediatric population represent a significant health care concern. Some reports and clinical observations indicate that 50% of pediatric patients present to sports medicine clinics for chronic injuries. In addition to their costs (direct and indirect medical expenditures), these injuries also result in lost participation time, numerous physician visits, and lengthy and often recurring rehabilitation. Furthermore, athletes who sustain recurrent overuse injuries may stop participating in sports and recreational activities, thus potentially adding to the already increasing number of sedentary individuals and the obesity epidemic.

In the pediatric population, overuse injuries can include growth-related disorders and those resulting from repeated microtrauma. Growth-related disorders include Osgood-Schlatter disease, Sever disease, and other apophyseal injuries. Overuse injuries resulting from repetitive microtrauma and chronic submaximal loading of tissues include stress fractures, similar to those described in adult athletes. However, overlap exists between broad classifications; some growth-related disorders may occur in sedentary children but much less often than in their active peers. Regardless of the cause, these injuries can result in significant pain and disability. Although little research has identified causative factors for overuse injuries in children and adolescents, these injuries may be caused by training errors, improper technique, excessive sports training, inadequate rest, muscle weakness and imbalances, and early specialization. More than half of all reported overuse injuries are speculated to be preventable, but few empirical data support this statistic.

The purpose of this position statement is to provide certified athletic trainers, physicians, and other health care professionals with current best practice recommendations regarding the prevention of overuse injuries in pediatric athletes, including children (aged 6–12 years) and adolescents (aged 13–18 years). Even though specific age ranges have been identified, it is important to note that the occurrence of puberty, followed by skeletal maturity, is a far more important marker of maturity than chronologic age when managing pediatric overuse injuries. In particular, this position statement will provide recommendations based on current evidence (Table 1) pertaining to injury surveillance (eg, incidence, prevalence), identification of risk factors for injury, preparticipation physical examinations (PPEs), proper supervision and education (coaching and medical), sport alterations, training and conditioning programs, and delayed specialization.

RECOMMENDATIONS

Injury Surveillance

1. Research should be devoted to improved understanding of the prevalence, incidence, and economic cost of overuse injuries among pediatric athletes in the United States and should focus on prevention and treatment of these overuse injuries.

Evidence Category: C

2. Funding and support for research into the prevalence, incidence, prevention, and treatment of pediatric overuse injuries should be increased. Evidence Category: C

3. All athletic health care providers should participate in injury-surveillance efforts, including accurate documentation in keeping with good clinical practice, and Web-based and other registries. Evidence Category: C

4. Resources and training for athletic health care providers (eg, certified athletic trainer, physician, physical therapist) to collect high-quality injury data must be developed. Evidence Category: C

Preparticipation Physical Examination

1. The PPE should be used as a means to screen each athlete for potential risk factors, including injury history, stature, maturity, joint stability, strength, and flexibility, which may be important for preventing recurrent injuries. Evidence Category: C

2. Children and adolescents with noted deficits on the PPE should be referred to appropriate medical specialists and health care providers (eg, physician specialist, certified athletic trainer, physical therapist) for further evaluation and corrective rehabilitation. Evidence Category: C

3. Robust documentation and injury-surveillance systems need to be developed to link PPE findings with injury, thereby identifying which measured factors may confer increased risk. Evidence Category: C

4. More research is needed to improve the effectiveness of the PPE, including strategies to implement the beneficial components more consistently and efficiently in the context of broader health-supervision and morbidity-prevention efforts for adolescents. Evidence Category: C

Identification of Risk Factors

1. Arm pain, fatigue, and decreased throwing performance should be recognized by athletes, coaches, parents, and medical personnel as early warning signs of potential overuse injuries in pediatric throwers. Evidence Category: A

2. Decreasing the volume of pitches as a means to prevent overuse injuries in throwing athletes is recommended. Evidence Category: A

3. Health care professionals should recognize that certain anatomic factors may predispose the athlete to overuse injury, including leg-length discrepancies, genu valgus, genu varus, pelvic rotation, and generalized joint hypermobility. Evidence Category: C

Coach Education and Medical Supervision

1. Pediatric athletes, parents, and coaches should be educated about the signs and symptoms of overuse injuries, and athletes should be instructed to notify an adult when such symptoms occur. Evidence Category: A

2. Coaches of youth and high school sports should have certifications or credentials identifying specific knowledge in areas related to sports safety, sports techniques and skills, psychosocial aspects of childhood and adolescence, growth and development, and common health and medical concerns. Evidence Category: C

3. Organized youth and interscholastic sports should be supervised by adults, ideally those with knowledge and training in monitoring for overuse injuries. Evidence Category: C

4. Medical personnel with training and education in pediatric sports injuries should be identified as referral sources to recognize, evaluate, and rehabilitate suspected overuse injuries. Evidence Category: C

Sport Alterations

1. Emerging evidence indicates that the sheer volume of sport activity, whether measured as number of throwing repetitions or quantity of time participating, is the most consistent predictor of overuse injury. Efforts should be made to limit the total amount of repetitive sport activity engaged in by pediatric athletes to prevent or reduce overuse injuries. Evidence Category: B

2. Although injury thresholds are yet to be determined for specific activities and age ranges, some data suggest a general guideline of no more than 16 to
Evidence Category: B

3. Alterations or modifications to the existing rules for adult sports may help to prevent overuse injuries in pediatric athletes and should be considered by coaches and administrators for sports in which youth rules are lacking.5,13,31 Evidence Category: C

4. Adults should ensure that pediatric athletes play only 1 overhead throwing sport at a time, avoid playing that sport year-round, and use caution when combining pitching with other demanding throwing positions (eg, pitch 1 day and catch the next day) to ensure adequate time for recovery.7,28,32 Evidence Category: C

5. Parents and coaches should restrict the use of breaking pitches in order to prevent pitching-related arm injuries.20 If an individual pitcher can throw breaking pitches on a limited basis and remain symptom free, then it may be allowed; however, if the use of this pitch precedes the development of any throwing-related symptoms, it should be immediately terminated and the athlete should seek medical attention. Evidence Category: C

6. Pitching limits should be established for players 9 to 14 years old: full-effort throwing (ie, in competition) should be limited to 75 pitches per game, 600 pitches per season, and 2000 to 3000 pitches per year.20,33 Evidence Category: B

7. Pitchers between 15 and 18 years of age should throw no more than 90 pitches per game and pitch no more than 2 games per week.21,33 Evidence Category: C

Training and Conditioning Programs

1. Preseason and in-season preventive training programs focusing on neuromuscular control, balance, coordination, flexibility, and strengthening of the lower extremities are advocated for reducing overuse injury risk, especially among pediatric athletes with a previous history of injury.34–36 Evidence Category: A

2. All pediatric athletes should begin participating in a general fitness program, emphasizing endurance, flexibility, and strengthening, at least 2 months before the sport season starts.5,9,37,38 Evidence Category: C

3. Pediatric athletes should have at least 1 to 2 days off per week from competitive practices, competitions, and sport-specific training.12,31 Coaches and administrators should consider these required days off when organizing season schedules. Evidence Category: C

4. Pediatric athletes should participate on only 1 team of the same sport per season when participation on 2 or more teams of the same sport (eg, high school and club) would involve practices or games (or both) more than 5 days per week.31 Evidence Category: C

5. Progression of training intensity, load, time, and distance should only increase by 10% each week to allow for adequate adaptation and to avoid overload.5,31 Evidence Category: C

6. After injury or delayed time without throwing, pediatric throwing athletes should pursue a gradual return-to-throwing program over several weeks before beginning or resuming full throwing activities.7,32 Evidence Category: C

Delayed Specialization

1. Pediatric athletes should be encouraged to participate in multiple sports and recreational activities throughout the year to enhance general fitness and aid in motor development.5,13 Evidence Category: C

2. Pediatric athletes should take time off between sport seasons and 2 to 3 nonconsecutive months away from a specific sport if they participate in that sport year-round.31 Evidence Category: C

3. Pediatric athletes who participate in simultaneous (eg, involvement in high school and club sports at the same time) or consecutive seasons of the same sport should follow the recommended guidelines with respect to the cumulative amount of time or pitches over the year.31 Evidence Category: C

BACKGROUND AND LITERATURE REVIEW

Repetitive stress on the musculoskeletal system without adequate and appropriate preparation and rest can result in chronic or overuse injuries in athletes of any age. In children and adolescents, this fact is complicated by the growth process, which can result in a unique set of injuries among pediatric athletes. Growth-related injuries most frequently affect the epiphyseal plates, where long bone growth occurs, and the apophyses, which serve as the bony attachments for musculotendinous units.6 Compression is usually responsible for epiphyseal injuries, whereas repeated tension or traction forces injure the apophyses.39 Differences in growth rates between the epiphyses and apophyses and between bone and muscle tissue are factors in apophyseal injury risk. These different growth rates may lead to relative myotendinous inflexibility and increased traction forces on the apophyses, contributing to traction apophyseal injuries.40,41 In throwers, repetitive microtrauma can lead to further bony insult, resulting in capitellar osteochondritis dissecans, a localized lesion of uncertain cause that involves the separation of articular cartilage and subchondral bone.42,43 Although most cases of osteochondritis dissecans resolve without consequence, lesions that do not heal after surgical intervention or a period of reduced repetitive impact loading may be responsible for future sequelae, including degenerative changes.44

Growth-center injuries may have long-term physical consequences and affect normal growth and development.16,40 However, little high-quality evidence supports or refutes this suggestion. In a systematic review45 of repetitive loading in gymnasts, females were at risk for stress-related injuries of the distal radius, including distal radial physeal arrest, but the lower-quality evidence of most of the included studies limited the strength of conclusions regarding whether physeal injury can inhibit radial growth. In a more recent systematic review,16 12
studies of baseball pitchers (3 case series, 9 case studies) with acute or chronic physeal injuries related to organized sport were analyzed. Stress-related changes were reported in all studies, including physeal widening in 8 reports, osteochondritis and radiographic widening of the proximal humeral growth plate in 2 reports, and humeral physeis in 1 report. Most of these patients improved with rest and were able to return to baseball, although some did not continue to pitch.

Data from lower extremity physeal injury studies were also extracted for review. Ten studies of lower extremity physeal injury revealed that these injuries occurred mainly in runners, but soccer, tennis, baseball (catcher), and gymnastics athletes also showed radiographic changes of physeal widening. Among the 17 studies (11 case reports, 6 case series) of physeal injury in gymnasts, traumatic physeal arrest was described in 1, stress changes or fractures in 6, physeal widening in 5, and premature growth-plate closure in 5. In the 8 cross-sectional gymnastics studies reviewed, a distal physeal stress reaction was noted on radiographs from 10% to 85% of the athletes. Although the authors concluded that stress-related physeal injuries in pediatric athletes often resolve without growth complication after adequate rest and rehabilitation, prospective, randomized studies must be performed to provide stronger evidence before clinicians should relax their vigilance concerning the potential for growth disturbance.

An estimated 50% of overuse injuries in physically active children and adolescents may be preventable. The prevention of pediatric overuse injuries requires a comprehensive, multidimensional approach that includes (1) improved injury surveillance (ie, improved understanding of epidemiology), (2) identification of risk factors for injury, (3) thorough PPEs, (4) proper supervision and education (both coaching and medical), (5) sport alterations, (6) improved training and conditioning programs, and (7) delayed specialization. This preventive approach has been advocated by several prominent sports and health care organizations, including the American College of Sports Medicine, the World Health Organization and International Federation of Sports Medicine, the American Academy of Pediatrics, and the International Olympic Committee.

**Injury Surveillance**

Before implementing any new prevention strategies or aiming to improve injury management, we must have adequate studies of epidemiology and a good understanding of the risk factors for pediatric overuse injuries. The literature regarding the epidemiology of overuse injuries in pediatric athletes is scarce at best, particularly the literature concerning American children.

However, the epidemiology of chronic injuries has been investigated in several international studies. In a 2003 Japanese study, the authors reviewed 196 stress fractures (125 males, 71 females) among 10,726 patients over a 10-year period. The average age of those with stress fractures was 20.1 years (range, 10–46 years), with 42.6% of patients between the ages of 15 and 19 years. The location of the stress fracture was somewhat specific to sport: the olecranon in baseball players, ribs in rowers, and tibial shaft stress fractures in ballet dancers, runners, and tennis, basketball, and volleyball players. Basketball players also sustained stress fractures to the metatarsals, whereas track athletes and soccer players incurred stress fractures to the tibial shaft and pubic bone. The authors concluded that stress fractures were common in high-functioning adolescent athletes, with similar proportions among male and female athletes. In another Japanese study of stress fractures in 208 athletes under the age of 20 years, the researchers found that the peak age of occurrence was 16 years, the most frequent site was the tibial shaft, and basketball was the sport most commonly associated with stress fractures. A 2006 retrospective study of stress fractures among 25 juveniles demonstrated that the age of onset was 12.9 ± 4.3 years (range, 3–17 years) and the tibia was most often affected (48%, n = 13), followed by the metatarsals (18.5%, n = 5). Using data from the High School Report Injuries Online (RIO) injury surveillance system, Fernandez et al reported that 4,350 athletic injuries occurred among athletes participating in 9 high school sports during 1 academic year. Although these authors did not focus solely on overuse injuries, they noted that 53% (n = 2,298) of these injuries were to the lower extremity; 2% of these injuries were classified as tendinitis and 1.3% as stress fractures. Specific investigations of the epidemiology of overuse injuries are warranted in the high school population.

Although studies on the general prevalence of pediatric overuse injuries are lacking, investigators have addressed the sport-specific prevalence of overuse injuries. Du-bravic-Simunjak et al retrospectively surveyed 469 elite junior figure skaters in Croatia, with 42% of female skaters and 45% of male skaters self-reporting an overuse injury at some point in their skating careers. In female singles ice skaters, the most frequent injury was a stress fracture (approximately 20%), followed by patellar tendinitis (14.9%). Male singles ice skaters were more likely to experience jumper’s knee (16%), followed by Osgood-Schlatter disease (14.2%). Maffulli et al reported on overuse injuries of the elbow among elite gymnasts in the United Kingdom and found that 12 elbows of 8 patients (aged 11–15 years) displayed a spectrum of radiologic abnormalities, including olecranon physeal widening and epiphyseal fragmentation.

In a recent investigation of Norwegian soccer players, the rates of overuse injuries were 0.2 (95% confidence interval [CI] = 0.1, 0.4) and 0.4 (95% CI = 0.0, 0.8) per 1,000 player-hours in 6- to 12-year-old boys and girls, respectively. An increase in the incidence of overuse injuries was noted in an older cohort (13–16 years old) of boys (0.7, 95% CI = 0.4, 1.0) and girls (0.7, 95% CI = 0.3, 1.1) per 1,000 player-hours, with the relative risk (RR) of overuse injury calculated as 2.9 (95% CI = 1.3, 6.4) and 1.7 (95% CI = 0.6, 5.5) in older boys and girls, respectively. In addition, 87% of the reported overuse injuries resulted in time loss from soccer that ranged from 1 to more than 21 days. Similarly, LeGall et al investigated the incidence of soccer-related injuries in elite French youth players and found that those younger than age 14 had more injuries during training sessions (ie, practices) and more growth-related overuse injuries, whereas older athletes more often sustained injuries during games. Overuse injuries accounted for 17.2% of all injuries and were mainly classified as tendinopathies (n = 108, 9.4%), osteochondroses (n = 72, n = 72,
6.3%), or other overuse (n = 19, 1.6%). In a follow-up study of adolescent female soccer athletes over 8 seasons, overuse injuries accounted for 13.4% of all injuries.52

We need to better understand the prevalence, incidence, and economic impact of overuse injuries among pediatric athletes in the United States. Although few data are currently available about overuse injuries, the more than 7.5 million young people who participate in interscholastic sports53 and millions of others who participate in youth sports programs across the country represent a very large at-risk population worthy of the expenditure of time, effort, money, and improved surveillance by clinicians and researchers alike.

Preparticipation Physical Examination

A consensus has emerged that the PPE, as defined by several leading medical and allied health specialty societies, is the primary means of identifying at-risk athletes and initiating preventive measures.5,6,14,15,17 The main objectives of the PPE are to detect life-threatening or disabling medical or musculoskeletal conditions and to screen athletes for medical or musculoskeletal conditions that may predispose them to injury or illness.17 In the context of this position statement, the history and musculoskeletal examination are important in detecting and possibly preventing overuse injuries. Additional information on other aspects of the PPE can be obtained from the Preparticipation Physical Evaluation monograph.17

The history portion of the PPE should be used to recognize previous injuries and other possible signs of overtraining. Many overuse injuries can be identified from the answers provided in the history component.17 Furthermore, a history of stress fractures or chronic or recurring musculoskeletal injuries may be associated with nutritional insufficiencies, which should be investigated as needed.17

The physical examination of the musculoskeletal system should include evaluation of the athlete’s physical stature and maturity (Tanner stage) and any deficits in strength and flexibility.5,17 In addition, the stability, symmetry, and range of motion of all joints and the relative symmetry, strength, and flexibility of all major muscle groups should be evaluated. These musculoskeletal assessments include range-of-motion tests, manual muscle tests, joint stress tests, flexibility tests, and balance tests, compared bilaterally. The Preparticipation Physical Evaluation17 describes the general 14-point musculoskeletal screening examination as an acceptable approach to evaluate the musculoskeletal system in athletes who are asymptomatic and without a history of previous injury. If the athlete describes a previous injury in the history portion or pain or positive findings are noted on the general screening examination, a more thorough joint-specific examination should be conducted.17 Ideally, a biomechanical assessment or functional screening test(s) should be incorporated to evaluate overall posture, gait mechanics, core stability, and functional strength.54 This may include either a single functional screening test, such as the overhead squat test,55 or a series of tasks56 to identify abnormal movement patterns. However, these tasks have yet to be validated in the pediatric population. Any deficits or concerns should be discussed with the athlete and parent, along with recommendations for correcting these deficits, including referral to a physician specialist, athletic trainer, or physical therapist if needed.5,14,15 Future researchers should focus on improving aspects of the musculoskeletal history, validating the general 14-point musculoskeletal screening examination and incorporating additional screening tests as possible predictors of overuse injuries.

A well-designed PPE can serve as a screening process from which prevention mechanisms can be developed.57 This process should be simple to administer and reliable and should use a combination of anthropometric and biomechanical measures to identify risk factors. Unfortunately, the PPE is often incompletely and inconsistently delivered. In most states, the PPE is mandated for high school athletes, but it is often not a requirement for those participating in club-based or youth sports.Required elements in the history and physical examination vary widely and are often not consistent with published national guidelines.58 The Preparticipation Physical Evaluation17 suggests that the ideal location for the PPE is within the athlete’s primary care physician’s office; however, it acknowledges other approaches, including the coordinated medical team examination and, although not recommended, the locker room-based examination. As a consequence of the lack of mandates, many adolescents seek their PPEs with a variety of providers and may be diverted from a medical “home,” where they can receive ongoing health immunizations and screening for the common psychosocial morbidities of adolescence.59 The lack of continuity also precludes connection with the rehabilitative follow-up deemed essential to injury prevention.60

Finally, the PPE may not be able to meet criteria for an appropriate screening process, even if implemented perfectly, because it is neither sensitive nor specific enough to adequately detect the life-threatening medical conditions that are so exceedingly rare60 or, in its current state, predict the potential for overuse injuries. Furthermore, as noted throughout this position statement, the evidence base is lacking as to which historical, anthropometric, and biomechanical findings confer increased musculoskeletal injury risk and which may be amenable to preventive interventions.

Identification of Risk Factors

Little rigorous research has been conducted to investigate potential risk factors for overuse injuries in pediatric athletes. Table 2 lists a number of suspected growth-related intrinsic and extrinsic risk factors for overuse injuries, although these classifications and listings of risk factors are primarily based on anecdotal evidence.5,61

One group10 attempted to identify accident-prone and overuse-prone profiles in young adults by prospectively investigating the effects of numerous physical and psychosocial characteristics on the rate of acute and overuse injuries. They developed an overuse-injury–prone profile for both males and females, which included physical factors such as a lack of stability (eg, decreased static strength coupled with laxity), muscle tightness, malalignment, more explosive strength, and large body size (ie, height and mass), and psychological traits including degree of carefulness, dedication, vitality, and sociability (Table 3).10 Many of these characteristics (eg, anatomical
alignment, flexibility, strength, speed) can be measured during a PPE or baseline fitness test, allowing clinicians to identify athletes potentially at risk for overuse injuries and to develop preventive measures.

Not surprisingly, baseball has been the most widely studied youth sport in the United States. Lyman et al\textsuperscript{18} prospectively evaluated 9- to 12-year-old baseball pitchers for pain or soreness in the shoulder or elbow during or after a pitching outing. Over 2 seasons, shoulder or elbow pain was noted in 47\% (n = 141) of the pitchers, with most of the pain complaints considered mild (ie, without loss of time in games or practices). The authors also provided some associated pain-related factors that may be important in identifying those potentially at risk for subsequent overuse injuries. Elbow pain was related to increased age, decreased height, increased mass, increased cumulative pitch counts, arm fatigue, decreased self-perceived performance, participation in a concurrent weightlifting program, and participation in additional baseball leagues.\textsuperscript{18} The presence of shoulder pain was associated with an increased number of pitches thrown in games, increased cumulative pitch counts, participating with arm fatigue, and decreased self-perceived performance. Both arm fatigue and self-perceived performance were risk factors for both elbow and shoulder pain. Therefore, pain should not be ignored, because it is often the first indicator of an overuse problem.\textsuperscript{20} Rest should be incorporated in all programs; athletes who participated with arm fatigue were almost 6 times more likely to suffer from elbow pain and 4 times more likely to have shoulder pain that those who did not have arm fatigue.\textsuperscript{18}

A subsequent investigation\textsuperscript{20} of 3 suspected risk factors (pitch type, pitch count, and pitching mechanics) found that the use of breaking pitches and high pitch counts increased the risk of both elbow and shoulder pain among youth pitchers. Specifically, the risk of elbow pain among pitchers using the slider increased 86\% and the risk of shoulder pain in those throwing curveballs increased 52\%. In addition, higher single-game pitch counts and higher cumulative (season-long) game pitch counts were associated with an increased risk of shoulder pain. This association between game pitch count and shoulder injury was strongest among 9- to 10-year-old and 13- to 14-year-old pitchers. Interestingly, pitching mechanics were not significantly associated with either elbow or shoulder pain in any of the age groups studied.\textsuperscript{20} The authors\textsuperscript{20} concluded that changeups remain the safest pitch for 9- to 14-year-olds to throw and that pitch limits, rather than inning limits, may be a better indicator of when pitchers should be removed from pitching to allow adequate rest.

More recently, Olsen et al\textsuperscript{19} investigated risk factors for shoulder and elbow injuries in adolescent pitchers. Group analyses between pitchers with or without elbow or shoulder injury revealed that a greater percentage of injured pitchers started at another position before pitching, pitched with arm fatigue, and continued to pitch even with arm pain.\textsuperscript{19} In addition, those who suffered an injury had a greater fastball speed and participated in a greater number of showcases (multiday, high-level events in which athletes may participate in numerous games within a short time span). A subsequent factor analysis revealed the following risk factors: participating in more than 8 months of competitive pitching (odds ratio [OR] = 5.05, 95\% CI = 1.39, 18.32), throwing more than 80 pitches per appearance (OR = 3.83, 95\% CI = 1.36, 10.77), having a fastball speed greater than 85 mph (136.8 kph) (OR = 2.58, 95\% CI = 0.94, 7.02), and pitching either infrequently (OR = 4.04, 95\% CI = 0.97, 16.74) or regularly (OR = 36.18, 95\% CI = 5.92, 221.22) with arm fatigue.\textsuperscript{19}

With respect to lower extremity injuries, few authors have attempted to identify specific overuse-injury risk factors in pediatric athletes, and their findings are

### Table 2. Potential Risk Factors Predisposing Pediatric Athletes to Overuse Injuries\textsuperscript{a}

<table>
<thead>
<tr>
<th>Growth-Related Factors</th>
<th>Intrinsic Factors</th>
<th>Extrinsic Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cartilage at growth plate is more susceptible to injury</td>
<td>Previous injury</td>
<td>Training and recovery</td>
</tr>
<tr>
<td>Period of adolescent growth increases the risk of injury (eg, osteochondritis dissecans, apophysitis, physisal injuries)</td>
<td>Malalignment</td>
<td>Equipment</td>
</tr>
<tr>
<td></td>
<td>Menstrual cycle</td>
<td>Poor technique</td>
</tr>
<tr>
<td></td>
<td>Psychological issues</td>
<td>Psychological issues</td>
</tr>
<tr>
<td></td>
<td>Muscle imbalances</td>
<td>Training errors</td>
</tr>
<tr>
<td></td>
<td>Inflexibility</td>
<td>Environment</td>
</tr>
<tr>
<td></td>
<td>Muscle weakness</td>
<td>Sport-acquired deficiencies</td>
</tr>
<tr>
<td></td>
<td>Instability</td>
<td>Conditioning</td>
</tr>
<tr>
<td></td>
<td>Level of play</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Height</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sex</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tanner stage</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Laxity</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Experience</td>
<td></td>
</tr>
</tbody>
</table>

\textsuperscript{a} From DiFiori\textsuperscript{9} and O’Connor et al.\textsuperscript{61}

### Table 3. Profiles of Overuse-Injury–Prone Male and Female Young Athletes\textsuperscript{a}

<table>
<thead>
<tr>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tall stature</td>
<td>Tall stature</td>
</tr>
<tr>
<td>Endomorph body structure</td>
<td>Decreased upper extremity strength</td>
</tr>
<tr>
<td>Less static strength</td>
<td>Less static strength</td>
</tr>
<tr>
<td>More explosive strength</td>
<td>More explosive strength</td>
</tr>
<tr>
<td>Decreased muscle flexibility</td>
<td>Increased muscle tightness</td>
</tr>
<tr>
<td>High degree of ligamentous laxity</td>
<td>Increased ligamentous laxity</td>
</tr>
<tr>
<td>Large Q angle</td>
<td>Greater leg-length discrepancy</td>
</tr>
<tr>
<td>Pronation</td>
<td>Large Q angle</td>
</tr>
</tbody>
</table>

\textsuperscript{a} From Lysens et al.\textsuperscript{10}
inconclusive. These studies are limited to research on medial tibial stress syndrome (MTSS) and stress fractures.\textsuperscript{50,62,63} In 2 investigations of risk factors for MTSS in high school cross-country runners, predictive variables differed.\textsuperscript{62,63} One group\textsuperscript{62} found that athletes with MTSS had a greater navicular drop (6.6 mm) than those who were asymptomatic (3.6 mm) and that the combination of navicular drop and sex accurately predicted 78\% of MTSS cases. However, resting calcaneal position, tibiofibural varus, and gastrocnemius length (ie, tightness) were not predictive. A subsequent investigation\textsuperscript{63} revealed that sex and body mass index were predictors of MTSS, with the latter being the only predictor when controlling for orthotic use. Additionally, compared with those without an injury history, high school cross-country runners with a history of previous injury were 2 times more likely to report MTSS (OR = 2.18, 95\% CI = 0.07, 6.4) and 3 times more likely to use orthotics (OR = 3.0, 95\% CI = 0.09, 9.4).\textsuperscript{63} Correlates of stress fractures in a general population of female adolescents have also been researched and, although age had the strongest association with a stress-fracture history (27\% to 29\% increased odds for each year beyond age 11), participation in more than 16 hours per week of vigorous activity (OR = 1.88, 95\% CI = 1.18, 3.03) and in high-impact physical activity, such as basketball, soccer, volleyball, running, tennis, or cheerleading (OR = 1.06, 95\% CI = 1.03, 1.10), was also related to stress-fracture history.\textsuperscript{30} They reported a slight (but nonsignificant) increased risk for stress fracture in the most sedentary girls, a reminder that participation in impact-loading physical activity is important in this population because of its positive effects on bone mineral density.\textsuperscript{30}

In a subsequent clinic-based study of adolescent female athletes, only family history of osteoporosis or osteopenia was associated with stress fracture (OR = 2.96, 95\% CI = 1.36, 6.45).\textsuperscript{64} However, in neither adolescent athlete investigation was stress fracture associated with irregular menstrual periods, as has been demonstrated in adult women athletes and military recruits.\textsuperscript{65} In combination, these investigations may begin to identify safe thresholds for participation in vigorous physical activity (16–20 h/wk). They also suggest that risk stratification must incorporate both intrinsic (eg, inherited skeletal quality) and extrinsic (eg, training volume) factors. Another area of focus concerning risk factors has been generalized joint hypermobility, which is characterized by mobility of multiple joints beyond the normal range of motion. Community prevalence of generalized joint hypermobility appears to depend on age, sex, and race, with reports ranging from 2\% (adult Caucasian males) to 57\% (African females of mixed ages).\textsuperscript{66} A considerable number of studies of rheumatology and pediatric clinic-based populations appear to demonstrate relationships between generalized joint hypermobility and insidious-onset arthralgia and fibromyalgia.\textsuperscript{67–75} Yet prospective studies of nonclinic populations are at best inconclusive as to whether joint hypermobility increases injury risk.

In a prospective study\textsuperscript{23} of 17-year-old military recruits, those with hypermobility had more injuries during boot camp than those who were not hypermobile. Another prospective study\textsuperscript{24} of youngsters 6 to 14 years old demonstrated that children with hypermobile joints had more complaints of joint pain than nonhypermobile children. A retrospective study\textsuperscript{76} of pediatric (aged 6–16 years) netball players in Australia showed that hypermobility was associated with an increased prevalence of self-reported injuries. In another small retrospective study\textsuperscript{77} including children and adults, more injuries were reported by hypermobile ballet dancers than by their nonhypermobile counterparts. In an attempt to describe overuse-prone profiles of young adults, Lysens et al\textsuperscript{10} reported that males and females with weak muscles, poor flexibility, and hypermobility may be at increased risk for overuse injuries.

Alternatively, several prospective studies of mixed (child and adult) or adult athletic populations do not support the conclusion that joint hypermobility is related to injury risk. A prospective study\textsuperscript{78} of netball players aged 15 to 36 years demonstrated no differences in injuries based on hypermobility status. Studies of National Collegiate Athletic Association lacrosse players\textsuperscript{79} and professional soccer players\textsuperscript{80} also have indicated no differences in injuries based on hypermobility status. Finally, a retrospective study\textsuperscript{81} of female gymnasts aged 10 to 21 years found no relationship between hypermobility status and reported history of injuries.

Screening for generalized joint hypermobility is relatively easy using the methods first proposed by Carter and Wilkinson\textsuperscript{82} and later modified by Beighton and Horan.\textsuperscript{83} This multijoint active range-of-motion screening procedure is widely accepted (Table 4). Incorporating this screening into the PPE might add only a few minutes to each assessment, but its use should depend upon the time, cost, and level of experience of the examiner administering the PPE.

**Proper Supervision and Education**

Organizations sponsoring interscholastic or club-based athletics in which pediatric athletes participate have the responsibility to ensure adequate coaching and medical supervision.\textsuperscript{5,13,14} Proper supervision by coaches and enforcement of the rules of the sport (which includes adequate education of both coaches and officials) may serve as a means to decrease overuse injury risk in this age group.\textsuperscript{9,15,50} For example, Little League Baseball provides pitch-count regulation (Appendix A), tracking sheet (Appendix B), and pitching eligibility forms, all of which are easily accessible to youth baseball coaches. The guidelines mandating pitch-count limits are adapted from scientific evidence and are updated frequently as new research emerges.\textsuperscript{84} Moreover, proper medical supervision at competitions and practices may allow for early recognition of overuse injuries in the beginning stages to permit proper evaluation, referral, and rehabilitation before they result in time lost from participation.\textsuperscript{14,15}

Education of all athletes, parents, coaches, and officials regarding overuse injuries and preventive mechanisms is advocated. Athletes, parents, and coaches should all have knowledge of general signs and symptoms of overuse, including but not limited to a gradual onset of pain, pain presenting as an ache, no history of direct injury, stiffness or aching after or during training or competition, increasing periods of time for pain to resolve, point tenderness, visible swelling, missed training sessions as a result of the pain or injury, and a problem that persists.\textsuperscript{25}
These signs and symptoms should not be ignored as “growing pains” but should be taken seriously by the athlete, parent, and coach. Athletes involved in running-based sports should be educated regarding sensible training habits and the proper fit and selection of running shoes to reduce impact forces. Athletes involved in throwing sports should be educated as to the potential risk factors for upper extremity overuse injuries, with specific emphasis on using arm fatigue as an indicator to stop throwing. All athletes should be educated on proper exercise progression and should gradually increase time, distance, and intensity by the 10% rule (see “Training and Conditioning” section below).57

To our knowledge, no published studies have addressed the general knowledge of overuse injuries among coaches; however, reports describe the general lack of first aid, injury recognition, and management knowledge of high school26 and youth27 coaches. No mandated national coaching education program exists in the United States for youth sports, and the requirements for high school athletic coaches vary from state to state, with some requiring only first aid and cardiopulmonary resuscitation (CPR) certification. However, numerous coaching education programs provide information related to proper biomechanics of sporting skills, nutrition, physical conditioning, development of athletes, and prevention, recognition, and management of injuries (Table 5). Completion of at least 1 of these courses is recommended for all coaches working with pediatric athletes. Additionally, coaches should be encouraged to maintain their certifications and participate in continuing education opportunities to remain current with the latest sports safety information.

**Sport Alterations**

Alterations or modifications to the existing rules for adults may prevent overuse injuries in children and adolescents. These modifications may be simple, including shorter quarters or halves, bases closer together,
less frequent games or practices, or pitch-count limits; or they may be more complex, such as the recommendation to match athletes by height, maturity, or skill as opposed to age.

Some experts are now moving away from the long-held and perhaps largely anecdotal belief that throwing breaking pitches is related to arm injuries in young baseball players. The only prospective study we were able to find appears to support this belief: pitchers throwing sliders had an 86% increased risk of elbow pain, and pitchers throwing curveballs had a 52% increased risk of shoulder pain. However, biomechanical studies comparing torque and moments generated by different types of pitches in 11- to 14-year-olds and 14- to 18-year-olds showed that the fastball imposed more demand than the curveball. Based on the results of these biomechanics studies, some researchers have postulated that throwing breaking pitches is not necessarily risky for young athletes. Yet it is important to recognize that the participants in these studies were healthy, with no history of arm injury, and, in the case of the Nissen et al. study, perhaps slightly older than the players who are generally the target of the recommendation against throwing breaking balls.

Furthermore, pitching limits should be established for 9- to 14-year-olds, with full-effort (ie, competition) throwing limited to 75 pitches per game and 600 pitches per season. Young throwers should also have adequate rest after a pitching event and adjust pitch limits for those rest days accordingly (Table 6). Table 7 lists baseball-pitcher–specific modification recommendations.

In addition to Little League, other athletic governing bodies and organizations have instituted sport modifications. The US Cycling Federation has imposed gear-ratio limits for athletes between 10 and 16 years of age, limiting the maximal stress or effort in those at the lower end of that age range. Running organizations in Australia also have age-related regulations, including set distances in which younger athletes may participate. Adolescents can begin participating in 5-km (3.1-mile) races at age 12 and in 10-km (6.2-mile) races at age 14. Half-marathon (21.1-km; 13.1-mile) and marathon (42.2-km; 26.2-mile) distances can be run beginning at ages 15 to 16 and >18 years, respectively. As described in Table 8, USA Swimming has recommendations for the number of sessions per week and the length of each session for various levels of competitive age-group swimming.

### Training and Conditioning

Proper training and conditioning, both before and during the season, may prevent overuse injuries. Unfortunately, in today’s society, many youngsters are not as active as previous generations, leading to a phenomenon of cultural deconditioning. There has been a general decline in physical activity, including free play, walking to school, and regular physical-education classes, with a concurrent increase in sedentary activities, including watching television, playing video games, and, in some cases, physical activity limited to sport participation. Athletes with poorer levels of general fitness or conditioning may not be able to tolerate the demands of training required for sport participation. Therefore, all pediatric athletes should begin by establishing a good general-fitness routine that encompasses strengthening, endurance, and flexibility. Sufficient participation in general strength, endurance, and flexibility training, as well as lifestyle physical fitness (eg, taking the stairs instead of the elevator), should precede sport-specific training. Once a general foundation of fitness has been established, athletes should begin to gradually increase their training loads. Pediatric athletes are advised to follow the 10% rule, which allows for no more than a 10% increase in the amount of training time, distance, repetitions, or load each week. For example, a runner who is currently running 15 miles/wk (24 km/wk) should only be allowed to increase mileage to 16.5 miles (27 km) the following week. Similarly, athletes participating in strength training should increase only either repetitions or weight by 10% each week, not both. The goal of the 10% rule is to allow the body to adjust gradually to increased training intensity.

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Table 6. Recommendations for Pitch Counts on the First 4 Days After a Pitching Event

<table>
<thead>
<tr>
<th>Age, y</th>
<th>1 d Rest</th>
<th>2 d Rest</th>
<th>3 d Rest</th>
<th>4 d Rest</th>
</tr>
</thead>
<tbody>
<tr>
<td>9–10</td>
<td>21–33</td>
<td>34–42</td>
<td>43–50</td>
<td>51+</td>
</tr>
<tr>
<td>11–12</td>
<td>27–34</td>
<td>35–54</td>
<td>55–57</td>
<td>58+</td>
</tr>
<tr>
<td>13–14</td>
<td>30–35</td>
<td>36–55</td>
<td>56–69</td>
<td>70+</td>
</tr>
<tr>
<td>15–16</td>
<td>30–39</td>
<td>40–59</td>
<td>60–79</td>
<td>80+</td>
</tr>
<tr>
<td>17–18</td>
<td>30–39</td>
<td>40–59</td>
<td>60–89</td>
<td>90+</td>
</tr>
</tbody>
</table>

a From Andrews and Fleisig. Reprinted with permission from USA Baseball.

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Table 7. Suggested Sport-Modification Recommendations for Adolescent Pitchers

1. Avoid pitching with arm fatigue.
2. Avoid pitching with arm pain.
3. Avoid pitching too much. Future research needed, but the following general limits are
   a. Avoid pitching more than 80 pitches per game.
   b. Avoid competitive pitching more than 8 months of the year.
   c. Avoid pitching more than 2500 pitches in competition per year.
4. Pitchers with the following characteristics should be monitored closely for injury
   a. Those who regularly use anti-inflammatories to “prevent” injuries
   b. Regularly starting pitchers
   c. Pitchers who throw > 85 mph (137 kph)
   d. Taller and heavier pitchers
   e. Pitchers who warm up excessively
   f. Pitchers who participate in showcases

have also investigated whether or that multisport athletes who do not obtain 0.49, 95 CI. reported that participants had a reduction in overall 5, 35) injuries, several authors34–36 have also investigated whether overuse-injury risk could be reduced among program participants. In a prospective study of a structured warm-up program including technique training, neuromuscular control, and balance and strengthening exercises, Olsen et al36 reported that participants had a reduction in overall injuries (RR = 0.49, 95% CI = 0.36, 0.68), lower limb injuries (RR = 0.51, 95% CI = 0.36, 0.74), and overuse injuries (RR = 0.43, 95% CI = 0.25, 0.75). Similarly, a program that focused on educating and training coaches to incorporate an overall-prevention mentality (consisting of improved warm-up, cool-down, taping unstable ankles, rehabilitation, promoting fair play, and a set of 10 exercises designed to improve joint stability, flexibility, strength, coordination, reaction time, and endurance) resulted in a reduction in both total injuries (0.76 ± 0.89 versus 1.18 ± 1.04, P < .01) and overuse injuries (0.26 ± 0.48 versus 0.44 ± 0.65, P < .05) per player-year.35 Specific to physically active adolescents, a 6-month, home-based balance-training program resulted in improvements in both static and dynamic balance among program participants.34 However, because of the limited number of injuries reported, no conclusions regarding the effectiveness of the program on reducing injuries could be drawn. Still, a clinically important difference was noted in self-reported injuries: program participants reported 3 (95% CI = 5, 35) injuries per 100 adolescents, compared with 17 (95% CI = 3, 24) in the control groups. Interestingly, the program was more effective in reducing injuries among those adolescents who reported sustaining an injury in the previous year,90 thus highlighting the need to identify injury history through a thorough PPE. In general, programs that are successful in reducing the risk of overuse injuries among pediatric athletes seem to include strengthening, neuromuscular control, flexibility exercises, balance, and technique training.

**Delayed Specialization**

One of the more controversial areas with respect to pediatric overuse injuries deals with the early specialization of athletes participating in the same sport year-round from a young age. Although little evidence-based research demonstrates that this practice has negative consequences on physical growth or psychological outcomes, many clinicians and health care organizations have advocated for diversity in sport participation or delayed specialization.5,9,91,92,93 It is theorized that participation in only 1 sport can result in increased risk for repetitive microtrauma and overuse3 or that multisport athletes who do not obtain adequate rest between daily activities or between seasons and those who participate in 2 or more sports that emphasize the same body part are at higher risk for overuse injuries than those in multiple sports with different emphases.31 Young athletes who participate in a variety of sports tend to have fewer injuries and play longer, thereby maintaining a higher level of physical activity than those who specialize before puberty.92 In addition to the potential for repetitive microtrauma and overuse injury, specialization in 1 sport may be associated with nutritional and sleep inadequacies, psychological or socialization issues, and ultimately burnout. These problems might be avoided with a balanced lifestyle and a strong support system made up of parents, friends, coaches, and health care providers.12

**CONCLUSIONS**

The major objective in managing repetitive or training injuries in athletes of any age should be to determine risk factors for injury and identify steps to prevent the occurrence of these injuries. Knowledge is growing about

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**Table 8. Developmental Progression for Pediatric Swimmers**

<table>
<thead>
<tr>
<th>Level</th>
<th>Category</th>
<th>Commitment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Sport preparation: ages 6–9 y</td>
<td>2–3 sessions/wk 30–60 min/session</td>
</tr>
<tr>
<td>2</td>
<td>Basic skill development: ages 8–11 y</td>
<td>2–4 sessions/wk 30–60 min/session Encourage other activities, sports Intrasquad or low-pressure competition</td>
</tr>
<tr>
<td>3</td>
<td>Basic training development: ages 11–14 y</td>
<td>4–6 sessions/wk 60–90 min/session Year-round participation Encourage other activities, sports while understanding the need to meet attendance expectations</td>
</tr>
<tr>
<td>4</td>
<td>Progressive training: ages 13–18 y</td>
<td>6–10 sessions/wk 90–120 min/session Year-round competition Including long-course (50-m pool-length) competition Commit to swimming Shorter breaks to minimize deterioration of aerobic base</td>
</tr>
<tr>
<td>5</td>
<td>Advanced training: age 14 y and older</td>
<td>8–10 sessions/wk 90–120 min/session Year-round High commitment level Short breaks to minimize deterioration of aerobic base</td>
</tr>
</tbody>
</table>

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*Reprinted with permission from USA Swimming.*
risk factors for the occurrence of both acute traumatic injuries and repetitive microtrauma overuse injuries in adults, particularly in such activities as military training, work activities, and sports. However, too little is known about risk factors for overuse injury in pediatric athletes.

Injury surveillance in young athletes should be improved to record the occurrence of injury and the determination of associated risk factors, as well as epidemiologic data (eg, age, sex, height, mass, and, if possible, Tanner stage). Epidemiologic studies in specific environments in pediatric populations would add greatly to the understanding of the risk associated with particular sport activities, thus providing a foundation for future studies of prevention and treatment efficacy.

ACKNOWLEDGMENTS

We gratefully acknowledge the efforts of Michael C. Koester, MD, ATC; Laura Purcell, MD; Angela Smith, MD; and the Pronouncements Committee in the preparation of this document.

DISCLAIMER

The NATA publishes its position statements as a service to promote the awareness of certain issues to its members. The information contained in the position statement is neither exhaustive not exclusive to all circumstances or individuals. Variables such as institutional human resource guidelines, state or federal statutes, rules, or regulations, as well as regional environmental conditions, may impact the relevance and implementation of these recommendations. The NATA advises its members and others to carefully and independently consider each of the recommendations (including the applicability of same to any particular circumstance or individual). The position statement should not be relied upon as an independent basis for care but rather as a resource available to NATA members or others. Moreover, no opinion is expressed herein regarding the quality of care that adheres to or differs from NATA’s position statements. The NATA reserves the right to rescind or modify its position statements at any time.

REFERENCES


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**Appendix A. Little League Pitch Count Tracker. Form provided by Little League International, Williamsport, PA.**

**Little League -- Baseball Game Pitch Log**

<table>
<thead>
<tr>
<th>Team</th>
<th>Opponent</th>
<th>X</th>
<th>O</th>
<th>Cross out the number as that pitch is thrown.</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pitcher's Name</strong></td>
<td><strong>Uniform Number</strong></td>
<td><strong>League Age</strong></td>
<td><strong>X</strong></td>
<td><strong>O</strong></td>
<td><strong>Circle the number for the last pitch thrown in each half-inning.</strong></td>
</tr>
<tr>
<td>1 2 3 4 5 6 7 8 9 10</td>
<td>11 12 13 14 15</td>
<td>16 17 18 19 20</td>
<td>21 22 23 24 25</td>
<td>26 27 28 29 30</td>
<td>31 32 33 34 35</td>
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<td>36 37 38 39 40 41 42 43 44 45</td>
<td>46 47 48 49 50</td>
<td>51 52 53 54 55</td>
<td>56 57 58 59 60</td>
<td>61 62 63 64 65</td>
<td>66 67 68 69 70</td>
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<td>71 72 73 74 75 76 77 78 79 80</td>
<td>81 82 83 84 85</td>
<td>86 87 88 89 90</td>
<td>91 92 93 94 95</td>
<td>96 97 98 99 100</td>
<td>101 102 103 104 105</td>
</tr>
</tbody>
</table>

Pitching eligibility varies by the league age of the pitcher, which is the pitcher's age as of May 1 of the current year. The pitching eligibility regulation is Regulation VI (see current rule book for details). A blank electronic version of this form is available for free download at www.littleleague.org.
Appendix B. Little League Pitcher Eligibility Tracker. Form provided by Little League International, Williamsport, PA.

Little League -- Baseball Pitcher Eligibility Tracking Form

<table>
<thead>
<tr>
<th>Division</th>
<th>Team</th>
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<table>
<thead>
<tr>
<th>Date of Game</th>
<th>Pitches Thrown</th>
<th>Pitcher's Name</th>
<th>Unif. No.</th>
<th>League Age</th>
<th>Tm. Manager's Signature *</th>
<th>Opp. Manager's Signature *</th>
<th>Scorekeeper/Ump Signature *</th>
<th>Eligible to pitch again on (date)</th>
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Pitching eligibility varies by the league age of the pitcher, which is the pitcher's age as of May 1 of the current year. The pitching eligibility regulation is Regulation VI (see current rule book for details). An electronic version of this form is available for free download at www.LittleLeague.org.

* Note: These signatures may be optional as determined by the local league.
Official Statement from the National Athletic Trainers’ Association on Communicable and Infectious Diseases in Secondary School Sports

The National Athletic Trainers’ Association (NATA) recommends that health care professionals and participants in secondary school athletics take the proper precautions to prevent the spread of communicable and infectious diseases.

Due to the nature of competitive sports at the high school level, there is increased risk for the spread of infectious diseases, such as impetigo, community acquired methicillin-resistant staphylococcus infection (MRSA) and herpes gladiatorum (a form of herpes virus that causes lesions on the head, neck and shoulders). These diseases are spread by skin-to-skin contact and infected equipment shared by athletes, generally causing lesions of the skin. The following

The following are suggestions from NATA to prevent the spread of infectious and communicable diseases:

- Immediately shower after practice or competition
- Wash all athletic clothing worn during practice or competition daily
- Clean and disinfect gym bags and/or travel bags if the athlete is carrying dirty workout gear home to be washed and then bringing clean gear back to school in the same bag. This problem can also be prevented by using disposable bags for practice laundry.
- Wash athletic gear (such as knee or elbow pads) periodically and hang to dry
- Clean and disinfect protective equipment such as helmets, shoulder pads, catcher’s equipment and hockey goalie equipment on a regular basis
- Do not share towels or personal hygiene products with others
- All skin lesions should be covered before practice or competition to prevent risk of infection to the wound and transmission of illness to other participants. Only skin infections that have been properly diagnosed and treated may be covered to allow participation of any kind
- All new skin lesions occurring during practice or competition should be properly diagnosed and treated immediately.
- Playing fields should be inspected regularly for animal droppings that could cause bacterial infections of cuts or abrasions
- Athletic lockers should be sanitized between seasons
- Rather than carpeting, locker or dressing rooms should have tile floors that may be cleaned and sanitized
- Weight room equipment, including benches, bars and handles should be cleaned and sanitized daily
National Athletic Trainers’ Association Position Statement: Preventing Sudden Death in Sports

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*Korey Stringer Institute, University of Connecticut, Storrs; †Matthew Gfeller Sport-Related Traumatic Brain Injury Research Center, University of North Carolina at Chapel Hill; ‡University of Oklahoma, Norman; §University of Georgia, Athens; ||Richard Stockton College, Pomona, NJ; #West Chester University, PA; #University of Tennessee at Chattanooga; **Western Michigan University, Kalamazoo; ††University of New Hampshire, Durham; ‡‡East Carolina University, Greenville, NC

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). 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 on-site 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. Evidence Category: B
2. Athletes with asthma should participate in a structured warm-up protocol before exercise or sport activity to decrease reliance on medications and minimize asthmatic symptoms and exacerbations. 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 [FEV1] 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. Evidence Category: A

**Treatment**

7. For an acute asthmatic exacerbation, the athlete should use a short-acting β2-agonist to relieve symptoms. In a severe exacerbation, rapid sequential administrations of a β2-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. Evidence Category: A
8. Inhaled corticosteroids or leukotriene inhibitors can be used for asthma prophylaxis and control. A long-acting β2-agonist can be combined with other medications to help control asthma. Evidence Category: B
9. Supplemental oxygen should be offered to improve the athlete’s available oxygenation during asthma attacks. 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. 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. Asthma is a disease in which the airways become inflamed and airflow is restricted. Airway inflammation, which
may lead to airway hyperresponsiveness and narrowing, is associated with mast cell production and activation and increased number of eosinophils and other inflammatory cells. 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.

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, non-steroidal anti-inflammatory drugs, inhaled irritants, exposure to cold, and exercise.

**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.

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. 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.

**Recognition.** Athletes with asthma may display the following signs and symptoms: confusion, sweating, drowsiness, FEV₁ of less than 40%, low level of oxygen saturation, use of accessory muscles for breathing, wheezing, cyanosis, coughing, hypotension, bradycardia or tachycardia, mental status

![Asthma pharmacologic management](http://www.jblearning.com. Reprinted with permission.)
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 β₂-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 β₂-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.

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.6,10 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, 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, 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%) 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%, which is consistent with previous reports.

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), 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 and Zurich guidelines.

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). 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. 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**

<table>
<thead>
<tr>
<th>Exertion Step</th>
<th>Activities</th>
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<tbody>
<tr>
<td>1.</td>
<td>20-min stationary bike at 10–14 mph (16–23 kph)</td>
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<td>2.</td>
<td>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</td>
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<td>3.</td>
<td>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</td>
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<td>4.</td>
<td>Limited, controlled return to practice with monitoring for symptoms</td>
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<td>5.</td>
<td>Full sport participation in practice</td>
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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. 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. *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. *Evidence Category: A*

**Treatment and Management**

6. The cervical spine should be in a neutral position, and manual cervical spine stabilization should be applied immediately. *Evidence Category: B*

7. Traction must not be applied to the cervical spine. *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. *Evidence Category: B*

10. If the spine is not in a neutral position, rescuers should realign the cervical spine. However, the presence or development of any of the following, alone or in combination, is a contraindication to realignment: 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. Whenever possible, manual stabilization 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. *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 until the athlete has been transported to an emergency medical facility except in 3 circumstances: 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. *Evidence Category: C*

14. Full face-mask removal using established tools and techniques 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. 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. 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 column during the initial 24 to 72 hours after injury. 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 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.68

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.69 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
Mild Hypoglycemia

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).

Severe Hypoglycemia

1. Activate EMS.
2. Prepare glucagon for injection, following directions in glucagon kit.
3. Once athlete is conscious and able to swallow, provide food.


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.67,73,74

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 intensity73,74 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 a hyperglycemic period.78 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.75,76

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.77,81

Treatment and Management. Treatment guidelines for mild and severe cases of hypoglycemia are shown in Table 2.52,83 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.87

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.
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 classic 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).87–90–97 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.90,93

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.97 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, axillary 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.96

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.104 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°C) 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. 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. Although no incidence data are available from organized athletics, the condition is seen in fewer than 1% of military athletes and up to 30% of distance athletes. 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. 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. 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. 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. 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. 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.

Dietary sodium is important for normal body maintenance of fluid balance and can help prevent muscle cramping, heat exhaustion, and EH. The AT should encourage adequate dietary sodium intake, especially when athletes are training in a hot environment and as a part of daily meals. 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 ac-

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**EH onsite is the use of a handheld analyzer, which can identify the serum sodium concentration within minutes.** Athletic trainers should work with physicians and EMS to maximize access to these analyzers when EH is likely.

A collapsed, semicomatose, 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. 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.

**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. 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. Normal saline (0.9% NaCl) IV fluids should not be provided to patients without prior serum sodium assessment. 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.

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 hypovolemia. Patients with persistent hypovolemia despite normal serum sodium values should receive 0.9% NaCl IV until eu-

**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.

Return to activity should be guided by a plan to avoid future EH episodes, specifically an individualized hydration plan (documented earlier). 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. 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. 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.

The premise behind the suggested precautions is that exertional sickling can be brought about through intense, sustained activity with modifiers that increase the intensity. One precaution that can mitigate exertional sickling is a slow, paced training progression that allows longer periods of rest and recovery between repetitions. 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. 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). In general, when athletes with SCT set their own pace, they seem to do well. 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.

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. 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.

Recognition. The PPE monograph 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 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. 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).

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, 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.

Sickling collapse has been mistaken for cardiac collapse or heat illness. 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 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 during “suicide sprints” on the court, laps on a track, or a long training run.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 lives123:

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 hospitalization133 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-
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 and Tackle Progression 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 and Tackle Progression 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. 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 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 and 18% of helmet collisions in 2007. In contrast, NCAA Division I officials called 1 helmet contact penalty in every 75 games in 2007. If illegal helmet contact is not enforced, the message is that the technique is acceptable. 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 is seen, people should vacate to a previously identified safe location.

2. Establish an EAP or policy specific to lightning safety.

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. 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.

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.

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.

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.

**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. 

8. Triage first lightning victims who appear to be dead. Most deaths are due to cardiac arrest. Although those who sustain a cardiac arrest may not survive due to subsequent apnea, aggressive CPR and defibrillation (if indicated) may resuscitate these patients.

9. Apply an AED and perform CPR as warranted.

10. Treat for concussive injuries, fractures, dislocations, and shock.

**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. Lightning occurs with greater frequency in the southeastern United States, the Mississippi and Ohio river valleys, the Rocky Mountains, and the Southwest, 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. Most deaths and injuries are recorded between 10:00 AM and 7:00 PM, when many people are engaged in outdoor activities.

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. The lightning channel has an average peak current of 20000 A and is 5 times hotter than the surface of the sun.

**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. 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. 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. 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.

**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. 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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{173,181} They may never fully return to desired levels, and they need consistent and perhaps multidisciplinary medical and psychological follow-up.\textsuperscript{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,\textsuperscript{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.\textsuperscript{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.\textsuperscript{185} 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.\textsuperscript{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.\textsuperscript{183}

The incidence of SCD in high school athletes is estimated to be 1:100,000 to 1:200,000.\textsuperscript{184,185} In collegiate athletes, this incidence is slightly higher, with estimates ranging from 1:6500 to 1:6900.\textsuperscript{184,186} A recent report\textsuperscript{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.\textsuperscript{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,\textsuperscript{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

<table>
<thead>
<tr>
<th>Medical history*</th>
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<tbody>
<tr>
<td>1. Exertional chest pain/discomfort</td>
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<tr>
<td>2. Unexplained syncope/near-syncope*</td>
</tr>
<tr>
<td>3. Excessive exertional and unexplained dyspnea/fatigue, associated with exercise</td>
</tr>
<tr>
<td>4. Prior recognition of a heart murmur</td>
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<tr>
<td>5. Elevated systemic blood pressure</td>
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<tr>
<td><strong>Family history</strong></td>
</tr>
<tr>
<td>6. Premature death (sudden and unexpected, or otherwise) before age 50 years due to heart disease, in ≥1 relative</td>
</tr>
<tr>
<td>7. Disability from heart disease in a close relative &lt;50 years of age</td>
</tr>
<tr>
<td>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</td>
</tr>
</tbody>
</table>

**Physical examination**

9. Heart murmur*

10. Femoral pulses to exclude aortic coarctation

11. Physical stigmata of Marfan syndrome

12. Brachial artery blood pressure (sitting position)*

*Parental verification is recommended for high school and middle school athletes.

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

*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.


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 SCA193, 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 Survival rates have been reported at 41%–74% if bystander CPR is provided and defibrillation occurs within 3 to 5 minutes of collapse.198,199

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**

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Appendix. National Athletic Trainers’ Association Statements*

<table>
<thead>
<tr>
<th>Topic (Year)</th>
<th>Citation</th>
<th>URL</th>
</tr>
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* 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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