Top ten research questions related to preventing sudden death in sport and physical activity

By: Rachel K. Katch, Samantha E. Scarneo, William N. Adams, Lawrence E. Armstrong, Luke N. Belval, Julie M. Stamm, and Douglas J. Casa

Katch RK, Scarneo SE, Adams WM, Armstrong LE, Belval LN, Stamm JM, Casa DJ. Top ten research questions related to preventing sudden death in sport and physical activity. *Research Quarterly for Exercise and Sport.* 2017;88(3):251-268.
https://doi.org/10.1080/02701367.2017.1342201

This is an Accepted Manuscript of an article published by Taylor & Francis in *Research Quarterly for Exercise and Sport* on 14 August 2017, available online: http://www.tandfonline.com/10.1080/02701367.2017.1342201.

Abstract:

Participation in organized sport and recreational activities presents an innate risk for serious morbidity and mortality. Although death during sport or physical activity has many causes, advancements in sports medicine and evidence-based standards of care have allowed clinicians to prevent, recognize, and treat potentially fatal injuries more effectively. With the continual progress of research and technology, current standards of care are evolving to enhance patient outcomes. In this article, we provided 10 key questions related to the leading causes and treatment of sudden death in sport and physical activity, where future research will support safer participation for athletes and recreational enthusiasts. The current evidence indicates that most deaths can be avoided when proper strategies are in place to prevent occurrence or provide optimal care.

Keywords: Education | exertional heat stroke | preparticipation screening | sudden cardiac death

Article:

Participation in sport and physical activity is on the rise, with an estimated 7.8 million and 482,533 student-athletes participating in sport at the secondary school and collegiate levels, respectively (Kucera, Yau, Thomas, Wolff, & Cantu, 2016). These numbers, stemming from the 2014 to 2015 academic year in the United States, have nearly doubled in the last 40 years (National Federation of State High School Associations, 2015a). Additionally, according to the 2017 U.S. labor force statics (U.S. Department of Labor, 2017) and National Defense Authorization Act (2016), there are an estimated 1.2 million labor workers and approximately 2.9 million U.S. military personnel (i.e., active, guard, reserve, and civilian). Combined, that number is approximately 12.4 million individuals who are involved in athletics, warfighting, and

physical labor and are exposed to strenuous workloads and intensities that could potentially lead to suffering sudden death.

At the collegiate and secondary school levels during 2013 to 2014, there were a total of 92 catastrophic injuries/illnesses, with the most common areas of the body affected being the heart (46%), neck/cervical spine (14%), and the head/brain (13%; Kucera et al., 2016). Investigating the sport of football alone at the same levels of participation, Boden, Breit, Beachler, Williams, and Mueller (2013) broke down the causes of 243 catastrophic deaths from 1990 to 2010 (Figure 1) and found the leading causes of death to be cardiac-related (41%), head injuries (25%), and heat illness (16%). In examining the warfighter, Eckhart et al. (2004) investigated all nontraumatic deaths in the U.S. military during a 25-year period and found out of 126 deaths, 108 (86%) were related to exercise, while identifiable cardiac abnormalities (51%) were among the top causes of death. Unfortunately, as physical activity participation rates increase, so do the number of catastrophic injuries; however, many of these cases of sudden death could have been prevented and/or survivable if current evidence-based best practices and guidelines were followed (Andersen, Courson, Kleiner, & McLoda, 2002; Armstrong et al., 2007; Casa et al., 2000, 2012, 2013, 2015; Heck, Clarke, Peterson, Torg, & Weis, 2004; Maron & Zipes, 2005).

FIGURE 1 IS OMITTED FROM THIS FORMATTED DOCUMENT
Figure 1. Number and percentage of football fatalities by diagnosis at the secondary school and collegiate level from July 1990 to June 2010. From Boden et al. (2013). © SAGE Publications.

With the enhancement of technology naturally comes the enhancement of strategies to prevent sudden death, leading to best-practice guidelines requiring regular review and sometimes revision. Thus, this manuscript will provide insight into some of the field's most pressing research questions regarding a multitude of different topics and may potentially question the norm on what truly are the best methods for preventing sudden death in sport and physical activity. Furthermore, answering these research questions will allow for critical review of current evidence-based literature and will lead the discussion on improving the care and well-being of athletes, warfighters, laborers, and any other individual participating in physical activity; because for an individual suffering from a life-threatening injury and/or illness, prompt and proper care can be the difference between life and death.

Top 10 research questions

1. Does preparticipation cardiac screening effectively identify disqualifying cardiac abnormalities?

Sudden cardiac death (SCD) is the leading cause of death during sport and physical activity and continues to be a concern as participation rates increase (Grazioli, Sanz, Montserrat, Vidal, & Sitges, 2015). According to the National Center for Catastrophic Sport Injury Research, from July 1, 2014, to June 30, 2015, there were a total of 92 catastrophic injuries/illnesses in the secondary school and collegiate levels of sport, with 46% of these incidents being caused by cardiac-related issues (Kucera et al., 2016). The most common types of events that occurred were sudden cardiac arrest (18%) and other cardiac conditions (24%), followed by fractures

(14%), brain trauma (10%), and heat-related illness (6%; Kucera et al., 2016). Additionally, Maron, Doerer, Haas, Tierney, and Mueller (2009) examined 1,866 deaths of young (M_{age} = 19 ± 6 years) competitive athletes in a multitude of sports in the United States during 1980 to 2006 and found that 56% were due to cardiovascular disease, while another 3% were due to commotio cordis (Figure 2). With SCD being the most common cause of sudden death in physical activity, research started to focus on preparticipation cardiac screening.

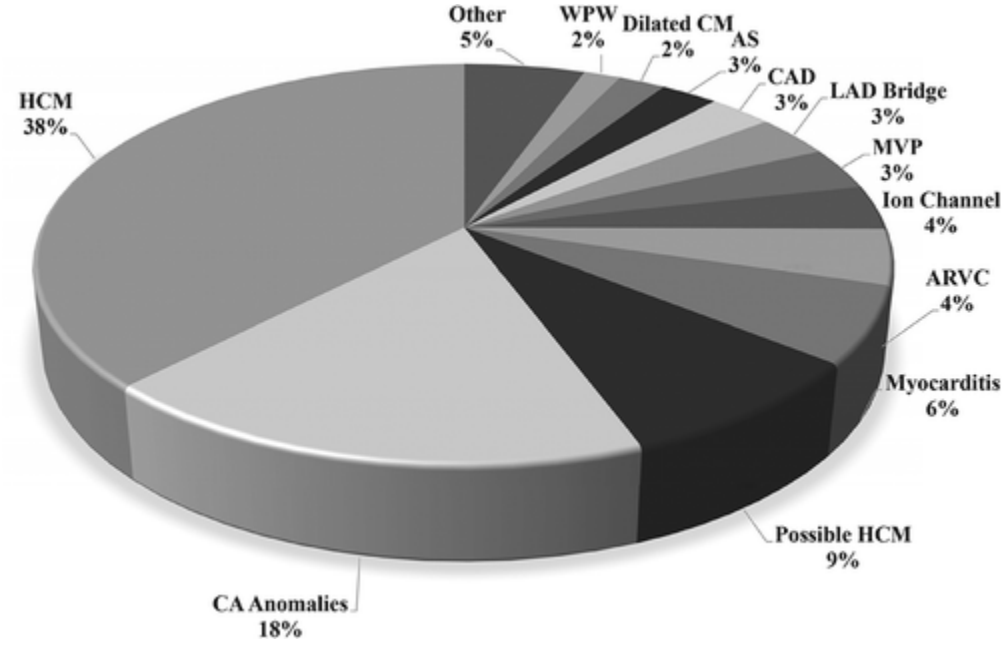

Figure 2. Confirmed cases of cardiovascular disease among young (M_{age} = 19 ± 6 years) competitive athletes in numerous U.S. sports during 1980 to 2006 (n = 690). *Note.* ARVC = arrhythmogenic right ventricular cardiomyopathy; AS = aortic stenosis; CA = coronary artery; CAD = coronary artery disease; CM = cardiomyopathy; HCM = hypertrophic cardiomyopathy; LAD = left anterior descending coronary artery; MVP = mitral valve prolapse; WPW, = Wolff-Parkinson-White. Data from Maron et al. (2009).

The three primary forms of cardiac prescreening include: (a) family and personal history, (b) physical examination, and (c) 12-lead electrocardiogram (ECG; Grazioli et al., 2015). Traditionally, conducting a comprehensive screening of personal and family history alongside a physical examination was considered the gold standard; however, data supporting the efficacy of such a screening strategy alone are very limited (Maron et al., 2014). From the available evidence, studies have shown that personal and family history accompanied by a physical examination was relatively insensitive in identifying, or even raising suspicion of, cardiovascular abnormalities including many of the diseases responsible for SCD in competitive athletes (Drezner et al., 2012, 2013). Furthermore, in a retrospective study conducted by Maron, Roberts, and Epstein (1982), of 71 patients who died from hypertrophic cardiomyopathy, only 15.5% of patients had definitive symptoms prior to death, with 33 patients (46.5%) having sudden death as their first definitive manifestation of an underlying cardiac disease (Maron et al., 1982).

Although many researchers agree that family history and physical examinations can be beneficial due to many young adults at risk for SCD showing signs, symptoms, and potentially a family

history of cardiovascular disease (Drezner et al., 2012, 2013; Maron et al., 2014), it has also been documented that many medical providers either will misinterpret or disregard these warning signs (Drezner et al., 2012; Maron et al., 2014; Wisten & Messner, 2009). Drezner et al. (2012) found that of 87 individuals who suffered from SCD, 72% had at least one cardiovascular symptom, with these symptoms presenting 30 months before suffering from SCD. Additionally, 40% of the study population identified as having at least one significant family history component that was present prior to the SCD (Drezner et al., 2012). Due to so many individuals potentially being asymptomatic prior to suffering SCD or symptoms and family history being misinterpreted/disregarded, a more definitive means for identifying those at risk for SCD needed to be investigated.

Global prescreening, including ECG, for cardiac abnormalities as a form of primary prevention of SCD has been highly debated due to the cost, manpower, and updated medical systems needed to support such a program (Grazioli et al., 2015; Maron et al., 2014; Piper & Stainsby, 2013). Currently, one of the advocators of incorporating global ECG screening is the European Society of Cardiology, which stands by its 25 years of Italian-based research that has shown ECG testing is effective in preventing SCD (Corrado et al., 2005, 2006). According to Grazioli et al. (2015), ECG has demonstrated a 70% sensitivity to detect the most frequent causes of SCD in young athletes; however, about a third of these athletes with an anomalous origin of coronary arteries, aortic diseases, and incipient forms of cardiomyopathies presented with a normal ECG. Furthermore, the study of the effectiveness of cardiovascular prescreening revealed that the sensitivity and specificity of ECG was 94% and 93%, respectively, compared with history (20% sensitivity and 94% specificity) and physical examination (9% sensitivity and 97% specificity; Harmon, Zigman, & Drezner, 2015). It was concluded was that the most effective strategy for screening cardiovascular disease in athletes is the inclusion of ECG as a prescreening tool, with a false-positive rate of 6% compared with history and physical examination alone (8% and 10% false-positive rates, respectively; Harmon et al., 2015). While evidence supports a low false-positive rate with ECG, the use of this tool as a method of cardiovascular screening is still controversial, especially in younger populations (12–25 years of age) due to the wide variety of predominantly genetic/congenital cardiovascular diseases that are becoming of increasing concern in this age group (Drezner et al., 2013, 2016).

Currently, the American Heart Association (AHA) and the American College of Cardiology (ACC) do not advocate for universal screening using ECGs in asymptomatic young people for cardiovascular disease (Drezner et al., 2016; Maron et al., 2014). Pelliccia et al. (2000) found 14% of athletes (> 1,000 elite athletes) had abnormal ECG readings that were highly suggestive of pathological conditions; however, these individuals received a false-positive reading leading to undue medical costs and potential psychological distress due to the additional cardiac screening. Thus, for mass screening, the AHA and ACC recommend a 14-point screening of cardiovascular history, as well as a physical examination to determine if ECG testing is needed (Maron et al., 2014).

Based on the literature, it is still up to debate on what screening method should be utilized to identify preparticipation cardiac abnormalities. Further, large-scale research needs to be conducted on the efficacy of all three methods of cardiac prescreening individually, as well as

the efficacy of the prescreening methods in combination with detecting cardiac abnormalities prior to SCD occurring in sport and physical activity.

2. Can individuals with implantable cardioverter defibrillators return to full participation in sport and physical activity?

The use of implantable cardioverter defibrillators (ICDs) as a primary and secondary prevention of SCD in individuals with preexisting cardiac conditions has increased in the last two decades due to their efficacy in treating life-threatening cardiac arrhythmias (e.g., ventricular tachycardia, ventricular fibrillation; Ponamgi, DeSimone, & Ackerman, 2015). In a meta-analysis, Theuns, Smith, Hunink, Bardy, and Jordaens (2010) found that ICDs significantly reduced arrhythmic mortality (relative risk [RR] = .40, 95% confidence interval [CI] [0.27, 0.67]) and all-cause mortality (RR = .73, 95% CI [0.64, 0.82]) in 5,343 sedentary patients; however, athletes with ICDs represent a diverse group of individuals who are at increased risk for suffering SCD when engaging in vigorous physical activity (Ponamgi et al., 2015). A lack of substantial data on the natural history of cardiac diseases and the unknown efficacy of implanted ICDs in terminating life-threatening arrhythmias occurring during intense exercise have resulted in the very restrictive nature of now decades-old guidelines (Ponamgi et al., 2015).

Table 1. Classification of sports.

Class IA	**Class IIA**	**Class IIIA**
Billiards, Bowling, Cricket, Curling, Golf, Riflery	Archery, Auto Racing, Diving, Equestrian, Motorcycling	Bobsledding, Luge, Field Events (throwing), Gymnastics, Martial Arts, Sailing, Sport Climbing, Water Skiing, Weight Lifting, Windsurfing
Class IB	**Class IIB**	**Class IIIB**
Baseball, Softball, Fencing, Table Tennis, Volleyball	American Football, Field Events (jumping), Figure Skating, Rodeoing, Rugby, Sprint Running, Surfing, Synchronized Swimming	Body Building, Downhill Skiing, Skateboarding, Snowboarding, Wrestling
Class IC	**Class IIC**	**Class IIIC**
Badminton, Cross-Country Skiing (classic), Field Hockey, Orienteering, Race Walking, Racquetball, Squash, Long-Distance Running, Soccer, Tennis	Basketball, Ice Hockey, Cross-Country Skiing (skating), Lacrosse, Middle-Distance Running, Swimming, Team Handball	Boxing, Canoeing, Kayaking, Cycling, Decathlon, Rowing, Speed Skating, Triathlon

Note. Data from Baman, Gupta, and Day (2010).

According to the 36th Bethesda Conference on eligibility recommendations for competitive athletes with cardiovascular abnormalities, athletes with ICDs should not participate in competitive sports with the exception of low-intensity Class IA sports (Table 1) or any sport that could potentially involve bodily trauma (Maron & Zipes, 2005). It was concluded that the presence of an ICD in competitive athletes with cardiovascular disease should not be regarded as protective therapy (Maron & Zipes, 2005). Additionally, the uncertainties associated with ICDs during intense competitive sports, including the possibility that the device will (a) not perform effectively at peak exercise, (b) produce a sinus tachycardia-triggered inappropriate shock or an appropriate discharge, and (c) increases the risk for physical injury to the athlete or other competitors as the result of an ICD shock, disqualifies an athlete from competitive sport (Maron

& Zipes, 2005). Recent updates to the Bethesda guidelines concurred that the recommendations for allowing athletes with ICDs to participate in competitive sports need to be reevaluated, and the updates further state that participation in sports with higher peak static and dynamic components than Class IA may be considered if the athlete is free of episodes of ventricular flutter or ventricular fibrillation requiring device therapy for 3 months (Maron, Zipes, & Kovacs, 2015). However, these guidelines have still been under scrutiny recently in the literature mainly due to the restrictive nature for athletes wanting to compete in competitive sports (Backhuijs et al., 2016; Heidbuchel & Carre, 2014; Lampert et al., 2013).

In contrast to current recommendations (Maron & Zipes, 2005), it has been shown that cardiologists tend to allow participation in competitive sports in particular cases (Heidbuchel & Carre, 2014). Previous literature (Lampert et al., 2013) revealed no instances of death or errant shock-related injuries, although there were shocks administered in 372 athletes with ICDs during a range of 21 months to 46 months of competitive sport participation. Additionally, there were 49 shocks in 37 participants (10% of the study population) during competition/practice, 39 shocks in 29 participants (8%) during other physical activity, and 33 shocks in 24 participants (6%) at rest (Lampert et al., 2013). In eight ventricular arrhythmia episodes (device defined), multiple shocks were received: one at rest, four during competition/practice, and three during other physical activity (Lampert et al., 2013). Of these incidences of shock, the ICD terminated all cardiac episodes. The most common sports in which these athletes were involved were running, basketball, and soccer—sports that are currently banned under the current guidelines (Lampert et al., 2013). Another study (Backhuijs et al., 2016) that investigated 71 young (younger than 40 years of age) patients with ICDs revealed no evidence that participation in sport (mainly endurance sports and fitness activities) contributed to the risk for life-threatening arrhythmias and inappropriate or appropriate ICD shocks.

Based on this information, it appears that many athletes with ICDs can engage in vigorous and competitive sports (e.g., basketball, soccer, running, etc.) without physical injury or failure to terminate a cardiac arrhythmia (Backhuijs et al., 2016; Lampert et al., 2013), which contradicts current recommendations (Maron & Zipes, 2005). It is feared that the current guidelines regarding sport participation and ICDs are too restrictive for some patients; nevertheless, further research is needed to determine the degree to which athletes with ICDs can participate in competitive contact sports.

3. Does adoption of a comprehensive emergency action plan reduce catastrophic outcomes or sudden death during sport and physical activity?

Efforts to reduce or prevent catastrophic sport-related injury include health and safety policy implementation based on best practices. Numerous medical organizations have advocated for this implementation, and it has been well documented in previous position statements and interassociation task-force documents demonstrating the need for comprehensive emergency action plans (EAPs; Andersen et al., 2002; Casa et al., 2013). EAPs are specific policies that are vital to mitigate the risk for a potentially fatal or long-term disability outcome for a patient in distress.

EAPs provide a step-by-step plan of action in the event of a sport-related emergency and allow for a thorough and specific plan to be in place to prevent critical delays in care from occurring. These plans include information for all potential personnel involved with activating the plan and should incorporate properly trained medical and health care professionals. When responding to an emergency, preventing critical delays in care is imperative to optimize patient outcomes. For example, the greatest factor in determining survival from a cardiac arrest is time from collapse to defibrillation. Automated external defibrillator (AED) application within 1 min to 3 min of collapse can lead to survival rates as high as 90%, and for every minute defibrillation is delayed, survival rates decrease by 7% to 10% (Drezner et al., 2007, 2013; Valenzuela, Roe, Cretin, Spaite, & Larsen, 1997). With sudden cardiac arrest as the leading cause of death in sport (Toresdahl, Rao, Harmon, & Drezner, 2014), prompt recognition and care are vital to improving outcomes.

Despite the robust literature to support these claims, only 2% of states require an on-site AED at every school-sanctioned athletics event and 14% of states do not require coaches to be trained in cardiopulmonary resuscitation (CPR) and AED administration (Korey Stringer Institute, unpublished data). Implementing and activating a written EAP can help facilitate quicker access to emergency care needed to prevent death in many scenarios. In addition to sudden cardiac arrest, other conditions such as exertional heat stroke (EHS), traumatic brain injuries, sickling, asthma, and others also require prompt recognition and care for optimal outcomes. For organizations to be properly prepared to respond to potentially serious or life-threatening injuries, emergency equipment, training, and response plans should be incorporated into the athletics organization. However, despite this knowledge, studies have identified that 13% to 70% of secondary school athletics programs adopt an EAP (Harer & Yaeger, 2014; S. T. Johnson et al., 2017; Lear, Hoang, & Zyzanski, 2015; Monroe, Rosenbaum, & Davis, 2009; K. Schneider, Meeteer, Nolan, & Campbell, 2017; Wasilko & Lisle, 2013). A recent study revealed only 11% of high schools in Oregon adopt EAP venue-specific policies, ensure coaches are CPR- and AED-trained, and ensure access to an AED for early defibrillation, and staggeringly, nearly 30% of schools report not implementing any of these recommendations (S. T. Johnson et al., 2017).

While these studies provide preliminary data depicting a lack of EAP adoption in the secondary school setting, there is paucity in the literature as to the adoption of comprehensive EAPs nationwide in all levels of sport and physical activity. Although EAPs have been demonstrated to reduce delays in critical care, further research is needed to determine the efficacy of an EAP when less than standard information is included versus including all components outlined in best-practice documents.

4. What should be included in a comprehensive coaching education program to decrease the risk for severe injuries and/or illnesses?

It is unfortunate that there are instances when athletes do not have access to an appropriate medical provider such as an athletic trainer. Without access to appropriate health care providers, providing educational information to coaches who interact with or have influence on the safety of athletes on the importance of proper policies and procedures to reduce the risk for severe injuries and/or illnesses is vital to reduce catastrophic outcomes. Despite the need for comprehensive educational strategies, very few states require athletics coaches to hold

educational certificates in CPR, first aid, AED training, concussion training, and heat training (National Federation of State High School Associations, 2015b). Ransone and Dunn-Bennett (1999) observed that only 36% of coaches achieved a passing score on an adapted first-aid assessment despite possessing CPR and first-aid certifications. Barron, Powell, Ewing, Nogle, and Branta (2009) reported only 5% of coaches passed a revised first-aid assessment. More recently, Adams, Mazerolle, Casa, Huggins, and Burton (2014) demonstrated that coaches lacked the fundamental knowledge on prevention and recognition of EHS. The findings from these investigations indicate that coaches are ill-prepared for assessment and treatment in emergency situations. Improved educational strategies are needed to better inform these key stakeholders on the proper prevention, recognition, and treatment of catastrophic injuries.

Comprehensive education on brain and spine injuries, environmental-based activity modifications and heat-related injuries, and appropriate hydration strategies should be conducted for all coaches and has been shown to be one of the most beneficial prevention strategies for several injuries in athletics (Barron et al., 2009; Broglio et al., 2014; Kerr, Dalton, et al., 2016). Education focused on the mechanism of injury, signs and symptoms, return to play and return to learn, protective equipment, and proper tackling should be conducted annually for coaches prior to the start of a sports season (Broglio et al., 2014; Kerr et al., 2017; Schneider et al., 2016). One educational program developed by USA Football is the Heads-Up Football Program, an educational program to inform coaches on the fundamental aspects of football, injury prevention, and injury recognition. Through the comprehensive educational strategies included in this relatively novel program, coaches are educated on components of football tackling; recent research has revealed that implementing this program has been related to lower injury rates compared with athletics programs that have not implemented Heads-Up Football (Kerr, Simon, et al., 2016).

Though there is promising evidence to support the overall reduction of injuries through the Heads-Up Program, there is paucity in the literature as to the efficacy of other comprehensive programs such as those required by state high school athletics associations. Additional research is needed to identify the retention of programs that incorporate education on all aspects of sudden death in sport and their effect on coach knowledge and perceptions. It is not satisfactory to simply identify coach educational knowledge with simple pen-and-paper assessments. Literature for nurses has evaluated memory recall, which involves searching memory storage within our brains. The research in this area has demonstrated that topics are recalled earlier if they are constantly practiced compared with retrieval of information without constant rehearsal (Broomfield, 1996; Gross, 2015). For example, CPR retraining literature has suggested there is decay in knowledge as soon as 2 weeks after training up to 18 months. Due to these findings, CPR retraining must be conducted every 2 years (Hamilton, 2005; Kaye & Mancini, 1986; Sullivan, 2015). Evaluating hands-on skills during mock and real-life scenarios is necessary to increase the efficacy of these programs to decrease both severity and time loss from injuries. Educational programs should include information related to head injuries, heat injuries, cardiac arrest, and other methods for preventing sudden death in sport. These topic areas should include prevention, recognition, and treatment best practices to improve the knowledge base of these topics. Further, prospective studies investigating the epidemiology of injury rates following various comprehensive programs are warranted to identify the most effective program.

5. What are the serious short-term complications of repeated mild traumatic brain injury?

Concussive and subconcussive brain trauma are induced by biomechanical forces on the brain as a result of either a direct blow to the head or forces distributed to the head from a blow to the body (Baugh et al., 2012; McCrory et al., 2013). A concussion is a pathophysiological process affecting the brain that results in altered neurological function, with symptoms that may include confusion, dizziness, irritability, headache, and balance difficulties (McCrory et al., 2013). Though loss of consciousness may occur, it is not necessary for the diagnosis of a concussion (McCrory et al., 2013). With the majority of concussions, symptoms resolve within 7 to 10 days, although they can persist for months or even years (McCrory et al., 2013). Though concussive and subconcussive brain trauma occurs by similar mechanisms, subconcussive brain trauma does not result in concussion symptoms (Baugh et al., 2012). Incurring repeated concussive and subconcussive brain trauma can lead to both serious acute and long-term consequences (Baugh et al., 2012; McKee et al., 2013; Stern et al., 2013).

The most concerning potential acute consequence of incurring repeated concussive impacts is second-impact syndrome (SIS). Though rare, SIS can occur if a concussed athlete returns to play prematurely and incurs additional brain trauma before symptoms associated with the initial concussion have resolved (Cantu, 1998; Cantu & Gean, 2010; McLendon, Kralik, Grayson, & Golomb, 2016). This second impact can seem relatively mild and may not involve a direct blow to the head, such as with a routine tackle observed during American football. In reported cases, the second hit has occurred within a range from 1 hr to several weeks following the initial injury (Cantu & Gean, 2010; McLendon et al., 2016). Immediately following the second impact, the athlete tends to have a lucid period during which they appear dazed but are able to walk under their own power. After approximately 15 s to 1 min, the athlete generally collapses and their condition rapidly decompensates (Cantu & Gean, 2010). It is thought that following the initial injury, the brain's ability to autoregulate its vasculature is impaired (Jünger et al., 1997; Strebel, Lam, Matta, & Newell, 1997). Then, following the second hit, this dysautoregulation leads to cerebral edema and rapidly increased intracranial pressure (Cantu, 1998; Cantu & Gean, 2010). Although it is the most widely accepted underlying mechanism for SIS, it has also been suggested that metabolic disturbances following the initial injury may make the brain more susceptible to further injury (McLendon et al., 2016).

SIS most commonly affects youth athletes younger than the age of 18 years; however, it has been reported in collegiate athletes as well, suggesting that young age may be a risk factor for SIS. Therefore, it is particularly important to ensure that concussions are recognized and properly managed in youth athletes. Poor outcomes following SIS occur in approximately 70% of cases and may include persistent motor, sensory, and cognitive deficits or death (Cantu & Gean, 2010; McLendon et al., 2016). Twelve of the 17 individuals with SIS in a case review by McLendon et al. (2016) had poor outcomes, including death or permanent disability. Similarly, 7 of 10 patients with SIS in a review by Cantu and Gean (2010) had poor outcomes, while the 3 patients with "good outcomes" still had persistent cognitive, sensory, and/or motor deficits. As current concussion diagnostic evaluation methods are largely subjective in nature, it is critical that athletes are honest in reporting concussion symptoms and clinicians recognize and properly manage these injuries to prevent SIS.

6. How does exertional hyponatremia develop, and what can athletes, warfighters, and laborers do to prevent this illness?

Symptomatic exertional hyponatremia (EH) occurs most often among healthy endurance athletes and military personnel during prolonged exercise and labor (e.g., > 4 hr, continuous exercise), while consuming a large volume of diluted fluid. Retention of a large water volume results in dilution of the intracellular and extracellular fluids, pulmonary edema, cerebral edema, coma, or sudden death. The term hyponatremia refers to a low sodium (Na^+) concentration in the blood. The normal range of serum sodium Na^+ concentration is 135 m Eq L^{-1} to 145 m Eq L^{-1}, whereas the symptoms of EH appear when serum Na^+ falls below 130 m Eq L^{-1}. When serum concentrations fall below 125 m Eq L^{-1}, immediate medical treatment is required as the approximate threshold for coma is 120 m Eq L^{-1} (Armstrong, McDermott, & Hosokawa, 2017). During exercise, athletes may recognize warning symptoms such as headache, dizziness, muscular twitching, tingling or swelling in extremities, and physical exhaustion; advanced cases involve nausea, vomiting, light headedness, or coma (Armstrong, McDermott, et al., 2017; Hew, Chorley, Cianca, & Divine, 2003). The most severe symptomatic EH cases (i.e., serum Na^+ concentration < 125 m Eq L^{-1}) present with cerebral edema and potentially fatal encephalopathy.

Some EH cases involve dehydration and body mass loss with large unreplaced Na^+ loss (i.e., high sweat Na^+ concentration plus high sweat rate) and partially replaced water loss (Hiller, 1989; Hoffman, Hew-Butler, & Stuempfle, 2013; Hoffman & Myers, 2015). Other EH cases involve hypervolemic hyponatremia with body mass gain (i.e., overhydration and net water retention exceeding sweat, urine, and respiratory water losses; Frizzell, Lang, Lowance, & Lathan, 1986; Noakes, Goodwin, Rayner, Branken, & Taylor, 1985).

Although athletes are often advised to avoid body weight gain (i.e., representing retention of excess water) during endurance exercise, research has indicated that body weight is not a valid indicator of EH when used as the sole diagnostic factor. For example, the data of Noakes and colleagues (2005) involving 2,135 competitive athletes (i.e., triathletes, cyclists, runners) showed that change in body mass accounted for only 10% of the statistical variance in postrace serum Na^+ concentration. These data support the EH treatment guidelines of the Wilderness Medical Society (Bennett, Hew-Butler, Hoffman, Rogers, & Rosner, 2013), which declare that body mass alone is not a reliable method to diagnose EH.

Because hyponatremia refers to a low serum Na^+, a few authorities have proposed that laborers and athletes consume Na^+ during prolonged work or exercise (Montain, 2008). Also, because some athletes have a high sweat rate (> 2.5 L h^{-1}), a high sweat Na^+ concentration (e.g., 60–80 mmol $Na+{\cdot}L^{-1}$), and observe salt stains on clothing after exercise (Meyer, Zbigniew, & Boguslaw, 2015), they believe that salt replacement is necessary and they consume salt tablets or salty foods during exercise (Armstrong, Lee, et al., 2017). However, controlled laboratory studies (Barr, Costill, & Flink, 1991; Vrijens & Rehrer, 1985) and field studies (Armstrong, Lee, et al., 2017; Hoffman et al., 2013; Hoffman & Myers, 2015) have shown that Na^+ consumption influences final serum Na^+ minimally. In fact, an athlete's beliefs may influence the etiology of EH more than dietary Na^+ (Winger, Dugas, & Dugas, 2011). One published case report (Armstrong et al., 1993) described a healthy young man who consumed 10.3 L of water, retained

2.77 L of water in 7 hr, and developed symptomatic EH during low-intensity exercise in a hot environment (41°C). He consumed this large volume of fluid because he believed that it would protect him from heat illness (Armstrong et al., 1993).

Because retention of excess water is a hallmark of serious EH, a few authors have proposed that athletes drink only when thirsty (Dugas & Noakes, 2005). However, this method is presently debated (Armstrong, Johnson, & Bergeron, 2016), and no evidence has demonstrated that drinking to thirst prevents EH. Indeed, the opposite is true. Published observations (Armstrong, Johnson, McKenzie, Ellis, & Williamson, 2015) of endurance cyclists (i.e., who rode 164 km in a sunny, 35.5°C environment and consumed fluids *ad libitum*) indicated that thirst was not statistically correlated with total fluid intake, body water balance ($L \cdot 164\ km^{-1}$), percent change in body mass, body mass index, height, or cycling ground speed. Further, the sensation of thirst, the desire to seek water, and the volume consumed are complex entities (Almiron-Roig & Drewnowski, 2003; Armstrong et al., 2014) that are influenced by physiologic responses, sensations, preferences, cultural influences, learned behaviors, fluid characteristics, and gender differences (Armstrong, Johnson, McKenzie, Ellis, & Williamson, 2016; Greenleaf, 1992; A. K. Johnson, 2007). Further, the vague concept of "drinking to thirst" easily can be misinterpreted by athletes (e.g., "Should I drink only when I perceive thirst? Should I drink so that thirst is always absent?") and may overemphasize the importance of drinking.

Statistical models (Armstrong, McDermott, et al., 2017; Montain, Sawka, & Wenger, 2001) have estimated the volume of water that an athlete must consume and retain (i.e., in excess of sweat + urine losses) to experience severe, symptomatic EH. These calculations have indicated that a serum Na^+ concentration of 120 m Eq L^{-1} can be reached by consuming an *excess* of only 2.2 L of water during a 9-hr ultra-endurance competition (rate = 200 mL h^{-1}) in a small runner who weighs 50 kg (111 lb). Table 2 illustrates this principle and suggests that drinking should not be a random, unplanned process.

Table 2. Calculated fluid excess required to develop severe, symptomatic exertional hyponatremia in endurance runners with different body mass.

		Runner A			**Runner B**			**Runner C**		
Body mass (kg, lb)		50, 111			70, 155			90, 199		
Total body water (L)		31.5			44.0			56.6		
Initial plasma Na^+ concentration ($mEq \cdot L^{-1}$)		140.0			140.0			140.0		
Volume of pure water consumed ($L \cdot 9h^{-1}$)		6.1			8.6			11.1		
Sweat loss ($L \cdot 9h^{-1}$)[a]		6.1			8.6			11.1		
Inherent sweat Na^+ concentrations ($mEq \cdot L^{-1}$)		20	40	60	20	40	60	20	40	60
Excess fluid retained[b]	(L)	4.2	3.2	2.2	5.9	4.5	3.0	7.6	5.8	3.9
	($L \cdot h^{-1}$)	0.5	0.4	0.2	0.7	0.5	0.3	0.8	0.6	0.4

Note. Data from Armstrong, McDermott, et al. (2017) and Montain et al. (2001) In this example, pure water intake equals sweat loss.

[a] Running at a pace of 10 km h^{-1} for 9 h.

[b] To dilute plasma Na^+ to 120 m Eq L^{-1}, which is the approximate threshold of severe symptoms and coma; the three values for each body mass represent three different sweat Na^+ concentrations.

L = liters; L h^{-1} = liters per hour; mEq = milliequivalents; Na^+ = sodium; TBW = total body water.

The safest and most effective plan for fluid intake during prolonged exercise involves an individualized drinking schedule that is developed during training, simulates competitive conditions, and is based on individual fluid needs (Armstrong, Lee, et al., 2017; Casa et al., 2000). The goal is to consume an optimal volume of fluid by determining sweat rate (Casa et al., 2000). This means drinking enough to avoid body weight loss of more than 2% to 3% (i.e., which compromises endurance exercise performance), while drinking a safe volume that avoids consumption of excess fluid.

7. Are current preparticipation prevention and prehospital treatment procedures effective at preventing exertional sickling-related death?

Sickle-cell trait (SCT), the inheritance of one gene for beta-S hemoglobin, is comparatively common in African Americans and affects approximately 7% of the population compared with only 1 in 625 (< 1%) in Caucasians (Harmon, Drezner, Klossner, & Asif, 2012). Exertional sickling or exercise collapse associated with sickle trait (ECAST) is an emergent condition that can affect individuals with SCT (Quattrone, Eichner, Beutler, Adams, & O'Connor, 2015). During exercise, the red blood cells of an individual with SCT can sickle and lead to fulminant rhabdomyolysis and potentially death (O'Connor et al., 2012). In particular, ECAST typically occurs during intense or novel exertion, especially when it occurs at altitude or in the heat (Drezner et al., 2007; National Athletic Trainers' Association, 2007; O'Connor et al., 2012).

From 2004 to 2008, the risk for exertional death was 1 in 827 in athletes with SCT, which was 37 times higher than in athletes without the condition (Harmon et al., 2012). Furthermore, from 2002 to 2011, 11 National Collegiate Athletic Association (NCAA) athletes died from ECAST, primarily during American football conditioning sessions and oftentimes performing novel activities (Anderson, 2017; Maron et al., 2014). ECAST was the greatest cause of death in NCAA football training for more than a decade (Adams, Casa, & Drezner, 2016).

With regards to the prevention of ECAST, there are generally two schools of thought. In the U.S. Army, a key study in the 1970s led to universal precautions (Brodine & Uddin, 1977). Recruits are not screened specifically for SCT, but rather, global training modifications were made to attempt to minimize the situations in which ECAST could occur. While ECAST deaths still occur, a recent study has indicated that soldiers with SCT have similar risks for death compared with individuals without the condition (Nelson et al., 2016). This finding contrasts the findings of Kark, Posey, Schumacher, and Ruehle (1987) in the 1980s, who found a higher risk for death in individuals with SCT.

Meanwhile, in response to several high-profile NCAA athlete deaths from ECAST, the NCAA has adopted a screening and education policy for SCT (Eichner, 2013). Athletes are provided screening for SCT to allow for medical staff to educate and intervene to minimize the risks for ECAST (Table 3; Parsons, 2014). Remarkably, in the first 5 years the policy was instituted, only one athlete died from ECAST (Adams et al., 2016).

One of the greatest challenges associated with ECAST is the lack of a definitive prehospital intervention during a sickling crisis. Current recommendations are to rapidly transport the patient

to an emergency department for the management of a fulminant metabolic crisis (O'Connor et al., 2012); however, the patient with ECAST relies on rapid recognition above all else.

Table 3. National Collegiate Athletic Association recommendations for student athletes with sickle-cell trait (Parsons, 2014).

- Set their own pace.
- Engage in a slow and gradual preseason conditioning regimen to be prepared for sports-specific performance testing and the rigors of competitive intercollegiate athletics.
- Build up slowly while training (e.g., paced progressions).
- Use adequate rest and recovery between repetitions, especially during "gassers" and intense station or "mat" drills.
- Not be urged to perform all-out exertion of any kind beyond 2 min to 3 min without a breather.
- Be excused from performance tests such as serial sprints or timed mile runs, especially if they are not normal sport activities.
- Stop activity immediately upon struggling or experiencing symptoms such as muscle pain, abnormal weakness, undue fatigue, or breathlessness.
- Stay well hydrated at all times, especially in hot and humid conditions.
- Maintain proper asthma management.
- Refrain from extreme exercise during acute illness, if feeling ill, or while experiencing a fever.
- Access supplemental oxygen at altitude as needed.
- Seek prompt medical care when experiencing unusual distress.

Despite ECAST being one of the most common causes of sudden death in the physically active, best practices currently rely on prevention to minimize morbidity and mortality. With all infants in the United States now being tested at birth (O'Connor et al., 2012), it is vital that SCT status is communicated appropriately to the health care professionals responsible for our athletes. Future research should focus on treatments for ECAST to minimize the metabolic crisis that can end playing careers and lives.

8. Does a noninvasive, real-time method exist to assess internal body temperature during exercise and prevent or diagnose exertional heat stroke?

The increased metabolic demands of exercise result in an acute rise in body temperature and cause a state of hyperthermia. Correspondingly, mechanisms of conduction, convection, evaporation, and radiation act on the body to achieve equilibrium where the rate of heat loss matches the rate of heat gain, thus preventing an uncompensable rise in body temperature (Galaski, 1985; Morrison, 2016). Maintenance of thermoregulatory equilibrium becomes strained during exercise in hot environmental conditions as the evaporation of sweat becomes the primary method for dissipating stored body heat (Kenney, 1996). In situations of high ambient temperature and high relative humidity, the ability of the body to dissipate body heat is further reduced, which may cause an uncompensable rise in body temperature and greatly increase the risk for EHS (Brewster, O'Connor, & Lilegard, 1995).

EHS, defined as a body temperature greater than 40.5°C with concurrent neuropsychiatric impairment (Casa et al., 2015), is a medical emergency, and without prompt recognition and treatment, it can lead to death or long-term sequelae. When EHS is suspected, an accurate body temperature is an essential component of the clinical decision algorithm to provide the appropriate treatment to the ailing individual. Current best practices (Armstrong et al., 2007;

Casa et al., 2015) dictate that when EHS is suspected, the utilization of rectal thermometry is the criterion standard-of-care measure for obtaining an accurate body temperature measure. Laboratory (Ganio et al., 2009; Miller, Hughes, Long, Adams, & Casa, 2017; Robinson, Seal, Spady, & Joffres, 1998) and field (Casa et al., 2007) assessment of rectal temperature has shown rectal temperature to be the gold-standard method for accurately measuring internal body temperature in exercising scenarios. Correspondingly, esophageal and gastrointestinal temperature have also been found to be valid measures of internal body temperature during exercise similar to that of rectal temperature (Byrne & Lim, 2007; O'Brien, Hoyt, Buller, Castellani, & Young, 1998; Robinson et al., 1998; Teunissen, de Haan, de Koning, & Daanen, 2012). However, due to the prohibitive and impractical applications of esophageal and gastrointestinal temperature assessment with cases of suspected EHS, rectal temperature is the diagnostic tool of choice for the clinical diagnosis of the condition.

The use of gastrointestinal temperature is commonly used in field settings to provide a continuous measure of internal body temperature; however, the utilization of the telemetric pill is fraught with potential flaws. Ingesting the pill 6 hr to 8 hr prior to activity is required to ensure that the pill has entered the lower intestines at the time of exercise to limit errant readings that could be observed if the pill is located within or in proximity to the stomach (Savoie et al., 2015). The risk of passing the pill from defecation as well as device failure (i.e., faulty battery or radio frequency signal) also potentiates its usage as a diagnostic tool and preventive measure as unreliable (Byrne & Lim, 2007).

While the aforementioned modes of temperature assessment most closely reflect that of pulmonary artery temperature and are deemed accurate and valid tools of obtaining a measure of internal body temperature during exercise, noninvasive methods have largely failed to do so. Previous literature assessing methods of noninvasive body temperature assessment such as oral temperature, tympanic temperature, temporal temperature, and axillary temperature (Casa et al., 2007; Ganio et al., 2009; Huggins, Glaviano, Negishi, Casa, & Hertel, 2012; Kistemaker, Den Hartog, & Daanen, 2009; Low et al., 2007; Mazerolle, Ganio, Casa, Vingren, & Klau, 2011; Nagano et al., 2010; O'Brien et al., 1998; Pryor et al., 2012; Ronneberg, Roberts, Mcbean, & Center, 2008) has shown that these methods are invalid in tracking body temperature during exercise and should not be used to obtain an accurate assessment of internal body temperature, especially when EHS is suspected.

Current research (Buller, Castellani, Roberts, Hoyt, & Jenkins, 2011; Buller et al., 2013; Buller, Tharion, Duhamel, & Yokota, 2015; Gribok, Buller, Hoyt, & Reifman, 2010; Laxminarayan, Buller, Tharion, & Reifman, 2014; Richmond, Davey, Griggs, & Havenith, 2015; Xu, Karis, Buller, & Santee, 2013; Yokota et al., 2012) has sought to identify other noninvasive physiologic measures and parameters to garner a real-time assessment of body temperature during activity. The ability to utilize noninvasive physiologic measures during exercise to estimate internal body temperature enhances the capabilities of using real-time temperature assessment as an injury prevention tool. Furthermore, the development of statistical algorithms to predict changes in body temperature during exercise while accounting for environmental conditions, clothing worn, and exercise intensity, among other factors, allows clinicians to identify persons at risk for exertional heat illness before the heat injury occurs to make the appropriate modifications or clinical decisions. Although this work has shown promise in accurately estimating true internal

body temperature, further work should identify the most vital physiologic parameters needed to make an accurate assessment of internal body temperature and develop methods of acquiring these measures in all applications of sport, within military operations, and within occupational settings.

9. What field interventions reduce the pathophysiological injuries of exertional heat stroke or enhance treatment and recovery?

During exercise in hot environmental conditions, the coordinated increase in skin blood flow and decrease in visceral blood flow create a potential hypoxic environment for the internal organs (Epstein & Roberts, 2011; Leon & Helwig, 2010). This heat stress-mediated hypoxic response within the visceral organs increases the permeability within the gastrointestinal tract allowing endotoxins such as lipopolysaccharide to be released into systemic circulation. The resulting inflammatory response contributes to EHS (Bouchama & Knochel, 2002; Lambert, 2004, 2008; Leon, 2007). It is through this pathophysiological pathway that the risk for multisystem organ failure and increased risk for death occur during EHS if not treated promptly (Bouchama & Knochel, 2002; Epstein & Roberts, 2011).

The key to survival of EHS is aggressive whole-body cooling aimed at reducing body temperature to below the critical threshold for cell damage as quickly as possible with the goal of reducing body temperature within 30 min of collapse (Adams, Hosokawa, & Casa, 2015). Evidence-based best-practice recommendations (Armstrong et al., 2007; Casa et al., 2012, 2015) have identified cold-water immersion (CWI) as the gold-standard method of treatment based on water's greater capacity to cool the body over that of air (Casa et al., 2007). With cooling rates of ~0.22°C (range = 0.13–0.35°C•min^{-1}; McDermott et al., 2009), CWI expedites the timing of cooling a patient with EHS and thus minimizes time above the critical threshold for cell damage. DeMartini et al. (2015) further supported the effectiveness of CWI in the treatment of EHS from the documented 274 cases of EHS that were successfully treated at the Falmouth Road Race with prompt recognition and care.

In many situations, the application of CWI to treat EHS may not be feasible. Situations such as remote military training/operations, wilderness firefighters, or occupational settings without the ability to store and maintain a tub for cooling, require other effective cooling modalities to expedite the cooling of a patient with EHS. Tarp-assisted cooling (Hosokawa, Adams, Belval, Vandermark, & Casa, 2017; Luhring et al., 2016), rotating ice water towels (McDermott et al., 2009), and cold-water dousing (McDermott et al., 2009) have been shown to produce effective cooling rates to successfully treat EHS in an adequate amount of time.

Although evidence has shown 100% survival from EHS with prompt recognition and care, the long-term risks associated with suffering EHS from a cellular level must not be overshadowed, especially when treatment is delayed. Specifically, the metabolic cascade of events that accompany EHS may cause long-term issues to one's health. This is evident in a recent case study by Stearns, Casa, O'Connor, and Lopez (2016), in which two patients with EHS were highlighted; one patient received appropriate care while the other's care was delayed. The ensuing physiologic responses from the latter case following his EHS exemplified the need for appropriate on-site care. Furthermore, the growing evidence of a potential link between EHS and

genetic disorders such as malignant hyperthermia (Bourdon & Canini, 1995; Hosokawa et al., 2017; Sagui, 2016) raises a question as to whether on-site treatment of EHS via cooling should be supplemented with another form of field-based treatment to reduce the potential detrimental effects of the metabolic cascade that occurs. To date, it is unclear as to what the true long-term effects of EHS are on one succumbing to this condition as there are no longitudinal studies that have examined the health and well-being of those suffering EHS 10 years, 20 years, or 30 years after the event. Furthermore, it is unclear as to what may be most beneficial as an adjunct treatment in addition to CWI to help minimize the pathophysiological response from EHS.

10. Does employing an athletic trainer improve patient outcomes during severe injuries and/or illnesses?

Athletic trainers are health care professionals trained in the prevention, treatment, recognition, and care of athletic-related injuries. As described in other sections of this article, injuries are an unfortunate, yet inherent risk of participation in sport and physical activity, and athletic trainers are properly trained to treat these injuries. Athletic trainers are licensed or otherwise regulated in 49 states and the District of Columbia and undergo academic curriculum and clinical training following the medical model. The medical model for athletic training is when the athletic trainer is employed under a direct medical services department (i.e., medical department, student health service, clinic, etc.). In this model, the athletic trainer directly reports to the director of the department (athletic trainer or other allied health care provider) to allow for the supervisor to understand the roles and responsibilities better than a non-medically trained individual. Under this model, the athletic trainer is evaluated on their medical skills as a professional, in addition to their day-to-day responsibilities. Specifically, athletic trainers are trained in emergency care of life-threatening injuries, which are common in sports.

Table 4. Percentage of full-time athletic training services by school student enrollment numbers.

	Pike et al. (2016)*	**Pryor et al. (2015)****
Student Enrollment Number	**% Full-Time**	**% Full-Time**
0–99	2	5
100–199	41	12
200–299	52	21
300–399	61	36
400–499	69	43
≥ 500	72	44†
3,300–3,399	—	93
3,400–3,499	—	93
3,500–3,599	—	92
> 3,600	—	88

Note. Data from Pike et al. (2016) and Pryor et al. (2015).
*U.S. private secondary schools.
**U.S. public secondary schools.
†Number based off 500 to 599 student enrollment.

Access to an appropriate medical professional ensures that a properly trained individual is caring for an injured athlete. Although athletic trainers are vital to ensuring the safety of athletes, a

benchmark study published in 2015 revealed that only 70% of public secondary schools and only 58% of private secondary schools in the nation have access to an athletic trainer (Pike, Pryor, Mazerolle, Stearns, & Casa, 2016; Pryor et al., 2015). Access to a full-time athletic trainer ensures that the athletes at the school have access to appropriate medical care for all practices and games; however, only 37% of public secondary schools have access to a full-time athletic trainer, a percentage about 9% higher than the private school setting (Pike et al., 2016; Pryor et al., 2015). Additionally, both studies (Pike et al., 2016; Pryor et al., 2015) revealed that as enrollment increased, so did access to athletic training services, with 72% of private and 93% of public secondary schools with an enrollment of more than 500 students and 3,300 students, respectively, having full-time athletic trainer access (Table 4). Access to an athletic trainer at other levels of sport, including road races, youth sport organizations, and other physical activity events, however, is currently unknown.

Without fully understanding the extent of athletic training care across various levels of athletics, researchers are unable to fully understand the benefits of athletic training services. One of the potential benefits of having athletic trainers provide care to athletes includes improving catastrophic outcomes of injuries or illnesses sustained during sport participation. One study (Olivadoti, 2016) showed that of 49 catastrophic deaths in the secondary setting, 71% of the fatalities occurred when the athletic trainer was not present to provide medical coverage. Because athletic training students' academic curriculum requires in-depth instruction in the management of the injuries and illnesses discussed in this manuscript, athletic trainers are uniquely qualified to manage these emergency and life-threatening situations compared with other health care providers who may have received minimum instruction on these conditions. Future research should investigate if there is a correlation between the lack of an athletic trainer present and an increased incidence of sudden death during physical activity at all levels of sports participation.

Conclusion

Preventing sudden death in sport and physical activity is a multifaceted process, with evidence-based best-practice guidelines being developed from cutting-edge research and investigations. The questions presented in this manuscript provide a foundation as to where future work is needed to further enhance evidence-based practices for preventing sudden death in sport and physical activity. As research continues to evolve, the health care provided to athletes and the physically active will continue to be enhanced as advancements in science and health care will equip health care providers with the most up-to-date and evidence-based guidelines to increase positive patient outcomes.

Acknowledgments

The authors would like to show their gratitude and thanks to the following individuals for sharing their insights, knowledge, and experience in their respective domains, leading to a well-rounded and informative manuscript: Craig R. Denegar, Ph.D., PT, ATC, FNATA; Yuri Hosokawa, Ph.D., ATC; Rebecca L. Stearns, Ph.D., ATC; and Robert A. Huggins, Ph.D., ATC.

References

Adams, W. M., Casa, D. J., & Drezner, J. A. (2016). Sport safety policy changes: Saving lives and protecting athletes. *Journal of Athletic Training*, 51, 358–360. doi:10.4085/1062-6050-51.4.14

Adams, W. M., Hosokawa, Y., & Casa, D. J. (2015). The timing of exertional heat stroke survival starts prior to collapse. *Current Sports Medicine Reports*, 14, 273–274. doi:10.1249/JSR.0000000000000166

Adams, W. M., Mazerolle, S. M., Casa, D. J., Huggins, R. A., & Burton, L. (2014). The secondary school football coach's relationship with the athletic trainer and perspectives on exertional heat stroke. *Journal of Athletic Training*, 49, 469–477. doi:10.4085/1062-6050-49.3.01

Almiron-Roig, E., & Drewnowski, A. (2003). Hunger, thirst, and energy intakes following consumption of caloric beverages. *Physiology Behavior*, 79, 767–773. doi:10.1016/S0031-9384(03)00212-9

Andersen, J. C., Courson, R. W., Kleiner, D. M., & McLoda, T. A. (2002). National Athletic Trainers' Association position statement: Emergency planning in athletics. *Journal of Athletic Training*, 37, 99–104.

Anderson, S. (2017). NCAA football off-season training: Unanswered prayers … a prayer answered. *Journal of Athletic Training*, 52, 145–148. doi:10.4085/1062-6050-52.3.02

Armstrong, L. E., Casa, D. J., Millard-Stafford, M., Moran, D. S., Pyne, S. W., & Roberts, W. O. (2007). American College of Sports Medicine position stand. Exertional heat illness during training and competition. *Medicine & Science in Sports & Exercise*, 39, 556–572. doi:10.1249/MSS.0b013e31802fa199

Armstrong, L. E., Curtis, W. C., Hubbard, R. W., Francesconi, R. P., Moore, R., & Askew, E. W. (1993). Symptomatic hyponatremia during prolonged exercise in heat. *Medicine & Science in Sports & Exercise*, 25, 543–549. doi:10.1249/00005768-199305000-00003

Armstrong, L. E., Johnson, E. C., & Bergeron, M. F. (2016). Counterview: Is drinking to thirst adequate to appropriately maintain hydration status during prolonged endurance exercise? No. *Wilderness & Environmental Medicine*, 27, 195–198. doi:10.1016/j.wem.2016.03.002

Armstrong, L. E., Johnson, E. C., Kunces, L. J., Ganio, M. S., Judelson, D. A., Kupchak, B. R., … Williamson, K. H. (2014). Drinking to thirst versus drinking ad libitum during road cycling. *Journal of Athletic Training*, 49, 624–631. doi:10.4085/1062-6050-49.3.85

Armstrong, L. E., Johnson, E. C., McKenzie, A. L., Ellis, L. A., & Williamson, K. H. (2015). Ultraendurance cycling in a hot environment. *Journal of Strength and Conditioning Research*, 29, 869–876. doi:10.1519/JSC.0000000000000822

Armstrong, L. E., Johnson, E. C., McKenzie, A. L., Ellis, L. A., & Williamson, K. H. (2016). Endurance cyclist fluid intake, hydration status, thirst, and thermal sensations: Gender differences. *International Journal of Sport Nutrition and Exercise Metabolism*, 26, 161–167. doi:10.1123/ijsnem.2015-0188

Armstrong, L. E., Lee, E. C., Casa, D. J., Johnson, E. C., Ganio, M. S., McDermott, B. P., … Williamson, K. H. (2017). Exertional hyponatremia and serum sodium change during ultraendurance cycling. *International Journal of Sport Nutrition and Exercise Metabolism*, 27, 139–147. doi:10.1123/ijsnem.2016-0135

Armstrong, L. E., McDermott, B. P., & Hosokawa, Y. (2017). Exertional hyponatremia. In D. J. Casa & R. I. Stearns (Eds.), *Preventing sudden death in sport & physical activity* (2nd ed., pp. 219–238). Burlington, MA: Jones & Bartlett Learning.

Backhuijs, T. A., Joosten, H., Zanen, P., Nathoe, H. M., Meine, M., Doevendans, P. A., … Rienks, R. (2016). Are the current guidelines for performing sports with an ICD too restrictive? *British Journal of Cardiology*, 23, 16–20. doi:10.5837/bjc.2016.008

Baman, T. S., Gupta, S., & Day, S. M. (2010). Cardiovascular health, part 2: Sports participation in athletes with cardiovascular conditions. *Sports Health*, 2, 19–28. doi:10.1177/1941738109356941

Barr, S. I., Costill, D. L., & Flink, W. J. (1991). Fluid replacement during prolonged exercise: Effects of water, saline, or no fluid. *Medicine & Science in Sports & Exercise*, 23, 811–817. doi:10.1249/00005768-199107000-00007

Barron, M. J., Powell, J. W., Ewing, M. E., Nogle, S. E., & Branta, C. F. (2009). First aid and injury prevention knowledge of youth basketball, football, and soccer coaches. *International Journal of Coaching Science*, 3, 55–67.

Baugh, C. M., Stamm, J. M., Riley, D. O., Gavett, B. E., Shenton, M. E., Lin, A., … Stern, R. A. (2012). Chronic traumatic encephalopathy: Neurodegeneration following repetitive concussive and subconcussive brain trauma. *Brain Imaging and Behavior*, 6, 244–254. doi:10.1007/s11682-012-9164-5

Bennett, B. L., Hew-Butler, T., Hoffman, M. D., Rogers, I. R., & Rosner, M. H. (2013). Wilderness Medical Society practice guidelines for treatment of exercise-associated hyponatremia. *Wilderness & Environmental Medicine*, 24, 228–240. doi:10.1016/j.wem.2013.01.011

Boden, B. P., Breit, I., Beachler, J. A., Williams, A., & Mueller, F. O. (2013). Fatalities in high school and college football players. *American Journal of Sports Medicine*, 41, 1108–1116. doi:10.1177/0363546513478572

Bouchama, A., & Knochel, J. P. (2002). Heat stroke. *New England Journal of Medicine*, 346, 1978–1988. doi:10.1056/NEJMra011089

Bourdon, L., & Canini, F. (1995). On the nature of the link between malignant hyperthermia and exertional heatstroke. *Medical Hypotheses*, 45, 268–270. doi:10.1016/0306-9877(95)90116-7

Brewster, S. J., O'Connor, F. G., & Lilegard, W. A. (1995). Exercise-induced heat injury: Diagnosis and management. *Sports Medicine and Arthroscopy Review*, 3, 260–266. doi:10.1097/00132585-199500340-00002

Brodine, C. E., & Uddin, D. E. (1977). Medical aspects of sickle hemoglobin in military personnel. *Journal of the National Medical Association*, 69, 29–32.

Broglio, S. P., Cantu, R. C., Gioia, G. A., Guskiewicz, K. M., Kutcher, J., Palm, M., & Valovich-McLeod, T. C. (2014). National Athletic Trainers' Association position statement: Management of sport concussion. *Journal of Athletic Training*, 49, 245–265. doi:10.4085/1062-6050-49.1.07

Broomfield, R. (1996). A quasi-experimental research to investigate the retention of basic cardiopulmonary resuscitation skills and knowledge by qualified nurses following a course in professional development. *Journal of Advanced Nursing*, 23, 1016–1023. doi:10.1111/j.1365-2648.1996.tb00084.x

Buller, M. J., Castellani, J. W., Roberts, W. S., Hoyt, R. W., & Jenkins, O. C. (2011). Human thermoregulatory system state estimation using non-invasive physiological sensors. *Conference Proceedings IEEE Engineering in Medicine and Biology Society*, 2011, 3290–3293.

Buller, M. J., Tharion, W. J., Cheuvront, S. N., Montain, S. J., Kenefick, R. W., Castellani, J. W., … Hoyt, R. W. (2013). Estimation of human core temperature from sequential heart rate observations. *Physiological Measurement*, 34, 781–798. doi:10.1088/0967-3334/34/7/781

Buller, M. J., Tharion, W. J., Duhamel, C. M., & Yokota, M. (2015). Real-time core body temperature estimation from heart rate for first responders wearing different levels of personal protective equipment. *Ergonomics*, 58, 1830–1841. doi:10.1080/00140139.2015.1036792

Byrne, C., & Lim, C. L. (2007). The ingestible telemetric body core temperature sensor: A review of validity and exercise applications. *British Journal of Sports Medicine*, 41, 126–133. doi:10.1136/bjsm.2006.026344

Cantu, R. C. (1998). Second-impact syndrome. *Clinics in Sports Medicine*, 17, 37–44. doi:10.1016/S0278-5919(05)70059-4

Cantu, R. C., & Gean, A. D. (2010). Second-impact syndrome and a small subdural hematoma: An uncommon catastrophic result of repetitive head injury with a characteristic imaging appearance. *Journal of Neurotrauma*, 27, 1557–1564. doi:10.1089/neu.2010.1334

Casa, D. J., Almquist, J., Anderson, S. A., Baker, L., Bergeron, M. F., Biagioli, B., … Valentine, V. (2013). The Inter-Association Task Force for Preventing Sudden Death in Secondary School Athletics Programs: Best-practices recommendations. *Journal of Athletic Training*, 48, 546–553. doi:10.4085/1062-6050-48.4.12

Casa, D. J., Armstrong, L. E., Hillman, S. K., Montain, S. J., Reiff, R. V., Rich, B. S., … Stone, J. A. (2000). National Athletic Trainers' Association position statement: Fluid replacement for athletes. *Journal of Athletic Training*, 35, 212–224.

Casa, D. J., Becker, S. M., Ganio, M. S., Brown, C. M., Yeargin, S. W., Roti, M. W., … Maresh, C. M. (2007). Validity of devices that assess body temperature during outdoor exercise in the heat. *Journal of Athletic Training*, 42, 333–342.

Casa, D. J., DeMartini, J. K., Bergeron, M. F., Csillan, D., Eichner, E. R., Lopez, R. M., … Yeargin, S. W. (2015). National Athletic Trainers' Association position statement: Exertional heat illnesses. *Journal of Athletic Training*, 50, 986–1000. doi:10.4085/1062-6050-50.9.07

Casa, D. J., Guskiewicz, K. M., Anderson, S. A., Courson, R. W., Heck, J. F., Jimenez, C. C., … Walsh, K. M. (2012). National Athletic Trainers' Association position statement: Preventing

sudden death in sports. *Journal of Athletic Training*, 47, 96–118. doi:10.4085/1062-6050-47.1.96

Casa, D. J., McDermott, B. P., Lee, E. C., Yeargin, S. W., Armstrong, L. E., & Maresh, C. M. (2007). Cold water immersion: The gold standard for exertional heatstroke treatment. *Exercise and Sport Sciences Reviews*, 35, 141–149. doi:10.1097/jes.0b013e3180a02bec

Corrado, D., Basso, C., Pavei, A., Michieli, P., Schiavon, M., & Thiene, G. (2006). Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. *Journal of the American Medical Association*, 296, 1592–1601. doi:10.1001/jama.296.13.1593

Corrado, D., Pelliccia, A., Bjornstad, H. H., Vanhees, L., Biffi, A., & Borjesson, M.; Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. (2005). Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: Proposal for a common European protocol: Consensus statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *European Heart Journal*, 26, 516–524.

DeMartini, J. K., Casa, D. J., Stearns, R., Belval, L., Crago, A., Davis, R., & Jardine, J. (2015). Effectiveness of cold water immersion in the treatment of exertional heat stroke at the Falmouth Road Race. *Medicine & Science in Sports & Exercise*, 47, 240–245. doi:10.1249/MSS.0000000000000409

Drezner, J. A., Ackerman, J. J., Anderson, J., Ashley, E., Asplund, C. A., Baggish, A. L., … Wilson, M. G. (2013). Electrocardiographic interpretation in athletes: The 'Seattle Criteria.' *British Journal of Sports Medicine*, 47, 122–124. doi:10.1136/bjsports-2012-092067

Drezner, J. A., Courson, R. W., Roberts, W. O., Mosesso, V. N., Link, M. S., & Maron, B. J. (2007). Inter-Association Task Force recommendations on emergency preparedness and management of sudden cardiac arrest in high school and college athletic programs: A consensus statement. *Journal of Athletic Training*, 42, 143–158.

Drezner, J. A., Fudge, J., Harmon, K. G., Berger, S., Campbell, R. M., & Vetter, V. L. (2012). Warning symptoms and family history in children and young adults with sudden cardiac arrest. *Journal of the American Board of Family Medicine*, 25, 408–415. doi:10.3122/jabfm.2012.04.110225

Drezner, J. A., O'Connor, F. G., Harmon, K. G., Fields, K. B., Asplund, C. A., Asif, I. M., … Roberts, W. O. (2016). AMSSM position statement on cardiovascular preparticipation screening in athletes. *Clinical Journal of Sport Medicine*, 26, 347–361. doi:10.1097/JSM.0000000000000382

Dugas, J. P., & Noakes, T. D. (2005). Hyponatraemic encephalopathy despite a modest rate of fluid intake during a 109 km cycle race. *British Journal of Sports Medicine*, 39, e38. doi:10.1136/bjsm.2005.018820

Eckhart, R. E., Scoville, S. L., Campbell, C. L., Shry, E. A., Stajduhar, K. C., Potter, R. N., … Virmani, R. (2004). Sudden death in young adults: A 25-year review of autopsies in military recruits. *Annals of Internal Medicine*, 141, 829–834. doi:10.7326/0003-4819-141-11-200412070-00005

Eichner, E. R. (2013). Preventing exertional sickling deaths: The right way, the wrong way, and the Army way. *Current Sports Medicine Reports*, 12, 352–353. doi:10.1249/JSR.0000000000000011

Epstein, Y., & Roberts, W. O. (2011). The pathophysiology of heat stroke: An integrative view of the final common pathway. *Scandinavian Journal of Medicine & Science in Sports*, 21, 742–748. doi:10.1111/j.1600-0838.2011.01333.x

Frizzell, R. T., Lang, G. H., Lowance, D. C., & Lathan, S. R. (1986). Hyponatremia and ultramarathon running. *Journal of the American Medical Association*, 255, 772–774. doi:10.1001/jama.1986.03370060086025

Galaski, M. (1985). Hyperthermia. *Journal of Canadian Athletic Therapy*, 12, 23–26.

Ganio, M. S., Brown, C. M., Casa, D. J., Becker, S. M., Yeargin, S. W., McDermott, B. P., … Maresh, C. M. (2009). Validity and reliability of devices that assess body temperature during indoor exercise in the heat. *Journal of Athletic Training*, 44, 124–135. doi:10.4085/1062-6050-44.2.124

Grazioli, G., Sanz, M., Montserrat, S., Vidal, B., & Sitges, M. (2015). Echocardiography in the evaluation of athletes. *F1000Research*, 4, 151. doi:10.12688/f1000research.6595.1

Greenleaf, J. E. (1992). Problem: Thirst, drinking behavior, and involuntary dehydration. *Medicine & Science in Sports & Exercise*, 24, 645–656. doi:10.1249/00005768-199206000-00007

Gribok, A. V., Buller, M. J., Hoyt, R. W., & Reifman, J. (2010). A real-time algorithm for predicting core temperature in humans. *IEEE Transactions on Information Technology in Biomedicine*, 14, 1039–1045. doi:10.1109/TITB.2010.2043956

Gross, R. (2015). *Psychology: The science of mind and behaviour* (7th ed.). London, UK: Hodder Education.

Hamilton, R. (2005). Nurses' knowledge and skill retention following cardiopulmonary resuscitation training: A review of the literature. *Journal of Advanced Nursing*, 51, 288–297. doi:10.1111/j.1365-2648.2005.03491.x

Harer, M. W., & Yaeger, J. P. (2014). A survey of certification for cardiopulmonary resuscitation in high school athletic coaches. *Wisconsin Medical Journal*, 113, 144–148.

Harmon, K. G., Drezner, J. A., Klossner, D., & Asif, I. M. (2012). Sickle cell trait associated with a RR of death of 37 times in National Collegiate Athletic Association football athletes: A database with 2 million athlete-years as the denominator. *British Journal of Sports Medicine*, 46, 325–330. doi:10.1136/bjsports-2011-090896

Harmon, K. G., Zigman, M., & Drezner, J. A. (2015). The effectiveness of screening history, physical exam, and ECG to detect potentially lethal cardiac disorders in athletes: A systematic

review/meta-analysis. *Journal of Electrocardiology*, 48, 329–338. doi:10.1016/j.jelectrocard.2015.02.001

Heck, J. F., Clarke, K. S., Peterson, T. R., Torg, J. S., & Weis, M. P. (2004). National Athletic Trainers' Association position statement: Head-down contact and spearing in tackle football. *Journal of Athletic Training*, 39, 101–111.

Heidbuchel, H., & Carre, F. (2014). Exercise and competitive sports in patients with an implantable cardioverter-defibrillator. *European Heart Journal*, 35, 3097–3102. doi:10.1093/eurheartj/ehu130

Hew, T. D., Chorley, J. N., Cianca, J. C., & Divine, J. G. (2003). The incidence, risk factors, and clinical manifestations of hyponatremia in marathon runners. *Clinical Journal of Sports Medicine*, 13, 41–47. doi:10.1097/00042752-200301000-00008

Hiller, W. D. (1989). Dehydration and hyponatremia during triathlons. *Medicine & Science in Sports & Exercise*, 21(Suppl. 5), S219–S221.

Hoffman, M. D., Hew-Butler, T., & Stuempfle, K. J. (2013). Exercise-associated hyponatremia and hydration status in 161-km ultramarathoners. *Medicine & Science in Sports & Exercise*, 45, 784–791. doi:10.1249/MSS.0b013e31827985a8

Hoffman, M. D., & Myers, T. M. (2015). Case study: Symptomatic exercise-associated hyponatremia in an endurance runner despite sodium supplementation. *International Journal of Sport Nutrition and Exercise Metabolism*, 25, 603–606. doi:10.1123/ijsnem.2014-0241

Hosokawa, Y., Adams, W. M., Belval, L. N., Vandermark, L. W., & Casa, D. J. (2017). Tarp-assisted cooling as a method of whole-body cooling in hyperthermic individuals. *Annals of Emergency Medicine*, 69, 347–352. doi:10.1016/j.annemergmed.2016.08.428

Hosokawa, Y., Casa, D. J., Rosenberg, H., Capacchione, J. F., Sagui, E., Riazi, S., … Vandermark, L. W. (2017). Round table on malignant hyperthermia in physically active populations: Meeting proceedings. *Journal of Athletic Training*, 52, 377–383. doi:10.4085/1062-6050-52.2.06

Huggins, R., Glaviano, N., Negishi, N., Casa, D. J., & Hertel, J. (2012). Comparison of rectal and aural core body temperature thermometry in hyperthermic, exercising individuals: A meta-analysis. *Journal of Athletic Training*, 47, 329–338. doi:10.4085/1062-6050-47.3.09

Johnson, A. K. (2007). The sensory psychobiology of thirst and salt appetite. *Medicine & Science in Sports & Exercise*, 39, 1388–1400. doi:10.1249/mss.0b013e3180686de8

Johnson, S. T., Norcross, M. F., Bovbjerg, V. E., Hoffman, M. A., Chang, E., & Koester, M. C. (2017). Sports-related emergency preparedness in Oregon high schools. *Sports Health*, 9, 181–184. doi:10.1177/1941738116686782

Jünger, E. C., Newell, D. W., Grant, G. A., Avellino, A. M., Ghatan, S., Douville, C. M., … Winn, H. R. (1997). Cerebral autoregulation following minor head injury. *Journal of Neurosurgery*, 86, 425–432. doi:10.3171/jns.1997.86.3.0425

Kark, J. A., Posey, D. M., Schumacher, H. R., & Ruehle, C. J. (1987). Sickle-cell trait as a risk factor for sudden death in physical training. *New England Journal of Medicine*, 317, 781–787. doi:10.1056/NEJM198709243171301

Kaye, W., & Mancini, M. E. (1986). Retention of cardiopulmonary resuscitation skills by physicians, registered nurses, and the general public. *Critical Care Medicine*, 14, 620–622. doi:10.1097/00003246-198607000-00007

Kenney, L. W. (1996). Thermoregulation during exercise in the heat. *Human Kinetics Journal*, 1(4), 13–16. doi:10.1123/att.1.4.13

Kerr, Z. Y., Dalton, S. L., Roos, K. G., Aristarque, D., Phelps, J., & Dompier, T. P. (2016). Comparison of Indiana high school football injury rates by inclusion of the USA football 'Heads Up Football' player safety coach. *Orthopaedic Journal of Sports Medicine*, 4, 5. doi:10.1177/2325967116648441#

Kerr, Z. Y., Roos, K. G., Djoko, A., Dalton, S. L., Broglio, S. P., Marshall, S. W., & Dompier, T. P. (2017). Epidemiologic measures for quantifying the incidence of concussion in National Collegiate Athletic Association sports. *Journal of Athletic Training*, 52, 167–174. doi:10.4085/1062-6050-51.6.05

Kerr, Z. Y., Simon, J. E., Grooms, D. R., Roos, K. G., Cohen, R. P., & Dompier, T. P. (2016). Epidemiology of football injuries in the National Collegiate Athletic Association, 2004–2005 to 2008–2009. *Orthopaedic Journal of Sports Medicine*, 4(9), 1–8. doi:10.1177/2325967116664500

Kistemaker, J. A., Den Hartog, E. A., & Daanen, H. A. M. (2009). Reliability of an infrared forehead skin thermometer for core temperature measurements. *Journal of Medical Engineering & Technology*, 30, 252–261. doi:10.1080/03091900600711381

Kucera, K. L., Yau, R., Thomas, L. C., Wolff, C., & Cantu, R. C. (2016). *Catastrophic sports injury research: Thirty-third annual report: Fall 1982–spring 2015*. Retrieved from https://nccsir.unc.edu/files/2013/10/NCCSIR-33rd-Annual-All-Sport-Report-1982_2015.pdf

Lambert, G. P. (2004). Role of gastrointestinal permeability in exertional heatstroke. *Exercise and Sport Sciences Reviews*, 32, 185–190. doi:10.1097/00003677-200410000-00011

Lambert, G. P. (2008). Intestinal barrier dysfunction, endotoxemia, and gastrointestinal symptoms: The 'canary in the coal mine' during exercise-heat stress? *Medicine and Sport Science*, 53, 61–73.

Lampert, R., Olshansky, B., Heidbuchel, H., Lawless, C., Saarel, E., Ackerman, M., … Cannom, D. (2013). Safety of sports for athletes with implantable cardioverter-defibrillators: Results of a prospective, multinational registry. *Circulation*, 127, 2021–2030. doi:10.1161/CIRCULATIONAHA.112.000447

Laxminarayan, S., Buller, M., Tharion, W., & Reifman, J. (2014). Human core temperature prediction for heat-injury prevention. *IEEE Journal of Biomedical and Health Informatics*, 19, 883–891. doi:10.1109/JBHI.2014.2332294

Lear, A., Hoang, M., & Zyzanski, S. J. (2015). Preventing sudden cardiac death: Automated external defibrillators in Ohio high schools. *Journal of Athletic Training*, 50, 1054–1058. doi:10.4085/1062-6050-50.8.01

Leon, L. R. (2007). Heat stroke and cytokines. *Progress in Brain Research*, 162, 481–524. doi:10.1016/S0079-6123(06)62024-4

Leon, L. R., & Helwig, B. G. (2010). Heat stroke: Role of the systemic inflammatory response. *Journal of Applied Physiology*, 109, 1980–1988. doi:10.1152/japplphysiol.00301.2010

Low, D. A., Vu, A., Brown, M., Davis, S. L., Keller, D. M., Levine, B. D., & Crandall, C. G. (2007). Temporal thermometry fails to track body core temperature during heat stress. *Medicine & Science in Sports & Exercise*, 39, 1029–1035. doi:10.1249/mss.0b013e318050ca3e

Luhring, K. E., Butts, C. L., Smith, C. R., Bonacci, J. A., Ylanan, R. C., Ganio, M. S., & McDermott, B. P. (2016). Cooling effectiveness of a modified cold-water immersion method after exercise-induced hyperthermia. *Journal of Athletic Training*, 51, 946–951. doi:10.4085/1062-6050-51.12.07

Maron, B. J., Doerer, J. J., Haas, T. S., Tierney, D. M., & Mueller, F. O. (2009). Sudden deaths in young competitive athletes: Analysis of 1866 deaths in the United States, 1980–2006. *Circulation*, 119, 1085–1092. doi:10.1161/CIRCULATIONAHA.108.804617

Maron, B. J., Friedman, R. A., Kligfield, P., Levine, B. D., Viskin, S., Chaitman, B. R., … Thompson, P. D.; American College of Cardiology. (2014). Assessment of the 12-lead ECG as a screening test for detection of cardiovascular disease in healthy general populations of young people (12–25 years of age). *Circulation*, 130, 1303–1334.

Maron, B. J., Roberts, W. C., & Epstein, S. E. (1982). Sudden death in hypertrophic cardiomyopathy: A profile of 78 patients. *Circulation*, 65, 1388–1394. doi:10.1161/01.CIR.65.7.1388

Maron, B. J., & Zipes, D. P. (2005). Introduction: Eligibility recommendations for competitive athletes with cardiovascular abnormalities: General considerations. *Journal of the American College of Cardiology*, 45, 1318–1321. doi:10.1016/j.jacc.2005.02.002

Maron, B. J., Zipes, D. P., & Kovacs, R. J. (2015). Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: Preamble, principles, and general considerations. *Journal of the American College of Cardiology*, 66, 2343–2349. doi:10.1016/j.jacc.2015.09.032

Mazerolle, S. M., Ganio, M. S., Casa, D. J., Vingren, J. L., & Klau, J. (2011). Is oral temperature an accurate measurement of deep body temperature? A systematic review. *Journal of Athletic Training*, 46, 566–573. doi:10.4085/1062-6050-46.5.566

McCrory, P., Meeuwisse, W. H., Aubry, M., Cantu, R., Dvořák, J., Echemendia, R. J., … Turner, M. (2013). Consensus statement on concussion in sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012. *British Journal of Sports Medicine*, 47, 250–258. doi:10.1136/bjsports-2013-092313

McDermott, B. P., Casa, D. J., Ganio, M. S., Lopez, R. M., Yeargin, S. W., Armstrong, L. E., & Maresh, C. M. (2009). Acute whole-body cooling for exercise-induced hyperthermia: A systematic review. *Journal of Athletic Training*, 44, 84–93. doi:10.4085/1062-6050-44.1.84

McKee, A. C., Stein, T. D., Nowinski, C. J., Stern, R. A., Daneshvar, D. H., Alvarez, V. E., … Cantu, R. C. (2013). The spectrum of disease in chronic traumatic encephalopathy. *Brain*, 136, 43–64. doi:10.1093/brain/aws307

McLendon, L. A., Kralik, S. F., Grayson, P. A., & Golomb, M. R. (2016). The controversial second impact syndrome: A review of the literature. *Pediatric Neurology*, 62, 9–17. doi:10.1016/j.pediatrneurol.2016.03.009

Meyer, F., Zbigniew, S., & Boguslaw, W. (2015). *Fluid balance, hydration, and athletic performance*. Boca Raton, FL: CRC Press.

Miller, K. C., Hughes, L. E., Long, B. C., Adams, W. M., & Casa, D. J. (2017). Validity of core temperature measurements at 3 rectal depths during rest, exercise, cold-water immersion, and recovery. *Journal of Athletic Training*, 52, 332–338. doi:10.4085/1062-6050-52.2.10

Monroe, A., Rosenbaum, D. A., & Davis, S. (2009). Emergency planning for sudden cardiac events in North Carolina high schools. *North Carolina Medical Journal*, 70, 198–204.

Montain, S. J. (2008). Strategies to prevent hyponatremia during prolonged exercise. *Current Sports Medicine Reports*, 7(4), S28–S35. doi:10.1249/JSR.0b013e31817f3a64

Montain, S. J., Sawka, M. N., & Wenger, C. B. (2001). Hyponatremia associated with exercise: Risk factors and pathogenesis. *Exercise and Sport Sciences Reviews*, 29, 113–117. doi:10.1097/00003677-200107000-00005

Morrison, S. F. (2016). Central control of body temperature. *F1000Research*, 5, 816–880. doi:10.12688/f1000research.7958.1

Nagano, C., Tsutsui, T., Monji, K., Sogabe, Y., Idota, N., & Horie, S. (2010). Technique for continuously monitoring core body temperatures to prevent heat stress disorders in workers engaged in physical labor. *Journal of Occupational Health*, 52, 167–175. doi:10.1539/joh.L9160

National Athletic Trainers' Association. (2007). *Fact sheet: 'Sickle cell trait and the athlete' consensus statement*. Retrieved from https://www.nata.org/sites/default/files/SickleCellFactSheet.pdf

National Defense Authorization Act for Fiscal Year 2017, 114th, S. 2943. (2016). Retrieved from http://docs.house.gov/billsthisweek/20161128/CRPT-114HRPT-S2943.pdf

National Federation of State High School Associations. (2015a). *2014–15 High School Athletics Participation Survey*. Retrieved from http://www.nfhs.org/ParticipationStatics/PDF/2014-15_Participation_Survey_Results.pdf

National Federation of State High School Associations. (2015b). *Tools for Coaches Association members*. Retrieved from https://nfhslearn.com/courses

Nelson, A. D., Deuster, P. A., Carter, R., III, Hill, O. T., Wolcott, V. L., & Kurina, L. M. (2016). Sickle cell trait, rhabdomyolysis, and mortality among U.S. Army soldiers. *New England Journal of Medicine*, 375, 435–442. doi:10.1056/NEJMoa1516257

Noakes, T. D., Goodwin, N., Rayner, B. L., Branken, T., & Taylor, R. K. (1985). Water intoxication: A possible complication during endurance exercise. *Medicine & Science in Sports & Exercise*, 17, 370–375. doi:10.1249/00005768-198506000-00012

Noakes, T. D., Sharwood, K., Speedy, D., Hew, T., Reid, S., Dugas, J., … Weschler, L. (2005). Three independent biological mechanisms cause exercise-associated hyponatremia: Evidence from 2,135 weighed competitive athletic performances. *Proceedings of the National Academy of Sciences*, 102, 18550–18555. doi:10.1073/pnas.0509096102

O'Brien, C., Hoyt, R. W., Buller, M. J., Castellani, J. W., & Young, A. J. (1998). Telemetry pill measurement of core temperature in humans during active heating and cooling. *Medicine & Science in Sports & Exercise*, 30, 468–472. doi:10.1097/00005768-199803000-00020

O'Connor, F. G., Bergeron, M. F., Cantrell, J., Connes, P., Harmon, K. G., Ivy, E., … Deuster, P. A. (2012). ACSM and CHAMP Summit on Sickle Cell Trait: Mitigating risks for warfighters and athletes. *Medicine & Science in Sports & Exercise*, 44, 2045–2056. doi:10.1249/MSS.0b013e31826851c2

Olivadoti, J. M. (2016). *The examination of medical coverage, emergency policies and procedures, and the implementation of prevention strategies before and after sudden death in the secondary school setting* (Master's thesis). Retrieved from DigitalCommons@UConn. (891)

Parsons, J. T. (2014). *2014–15 NCAA sports medicine handbook*. Indianapolis, IN: National Collegiate Athletic Association. Retrieved from http://www.ncaapublications.com/productdownloads/MD15.pdf

Pelliccia, A., Maron, B. J., Culasso, F., Di Paolo, F. M., Spataro, A., Biffi, A., … Piovano, P. (2000). Clinical significance of abnormal electrocardiographic patterns in trained athletes. *Circulation*, 102, 278–284. doi:10.1161/01.CIR.102.3.278

Pike, A., Pryor, R. R., Mazerolle, S. M., Stearns, R. L., & Casa, D. J. (2016). Athletic trainer services in US private secondary schools. *Journal of Athletic Training*, 51, 717–726. doi:10.4085/1062-6050-51.11.04

Piper, S., & Stainsby, B. (2013). Addressing the risk factors and prevention of sudden cardiac death in young athletes: A case report. *Journal of the Canadian Chiropractic Association*, 57, 350–355.

Ponamgi, S. P., DeSimone, C. V., & Ackerman, M. J. (2015). Athletes with implantable cardioverter defibrillators. *Clinics in Sports Medicine*, 34, 473–487. doi:10.1016/j.csm.2015.03.010

Pryor, R. R., Casa, D. J., Vandermark, L. W., Stearns, R. L., Attanasio, S. M., Fontaine, G. J., & Wafer, A. M. (2015). Athletic training services in public secondary schools: A benchmark study. *Journal of Athletic Training*, 50, 156–162. doi:10.4085/1062-6050-50.2.03

Pryor, R. R., Seitz, J. R., Morley, J., Suyama, J., Guyette, F. X., Reis, S. E., & Hostler, D. (2012). Estimating core temperature with external devices after exertional heat stress in thermal

protective clothing. *Prehospital Emergency Care*, 16, 136–141. doi:10.3109/10903127.2011.614047

Quattrone, R. D., Eichner, R. E., Beutler, A., Adams, B. W., & O'Connor, F. G. (2015). Exercise collapse associated with sickle cell trait (ECAST). *Current Sports Medicine Reports*, 14, 110–116. doi:10.1249/JSR.0000000000000137

Ransone, J., & Dunn-Bennett, L. R. (1999). Assessment of first-aid knowledge and decision making of high school athletic coaches. *Journal of Athletic Training*, 34, 267–271.

Richmond, V. L., Davey, S., Griggs, K., & Havenith, G. (2015). Prediction of core body temperature from multiple variables. *Annals of Occupational Hygiene*, 59, 1168–1178. doi:10.1093/annhyg/mev054

Robinson, J. L., Seal, R. F., Spady, D. W., & Joffres, M. R. (1998). Comparison of esophageal, rectal, axillary, bladder, tympanic, and pulmonary artery temperatures in children. *Journal of Pediatrics*, 133, 553–556. doi:10.1016/S0022-3476(98)70067-8

Ronneberg, K., Roberts, W. O., Mcbean, A. D., & Center, B. A. (2008). Temporal artery temperature measurements do not detect hyperthermic marathon runners. *Medicine & Science in Sports & Exercise*, 40, 1373–1375. doi:10.1249/MSS.0b013e31816d65bb

Sagui, E. (2016). Malignant hyperthermia, exertional heat illness, and RYR1 variants. *Anesthesiology*, 124, 510–511. doi:10.1097/ALN.0000000000000979

Savoie, F. A., Dion, T., Asselin, A., Gariepy, C., Boucher, P. M., Berrigan, F., & Goulet, E. D. B. (2015). Intestinal temperature does not reflect rectal temperature during prolonged, intense running with cold fluid ingestion. *Physiological Measurement*, 36, 259–272. doi:10.1088/0967-3334/36/2/259

Schneider, D. K., Grandhi, R. K., Bansal, P., Kuntz, IV, G. E., Webster, K. E., Logan, K., … Myer G. D., (2016). Current state of concussion prevention strategies: A systematic review and meta-analysis of prospective, controlled studies. *British Journal of Sports Medicine*. Advance online publication. doi:10.1136/bjsports-2015-095645

Schneider, K., Meeteer, W., Nolan, J. A., & Campbell, H. D. (2017). Health care in high school athletics in West Virginia. *Rural and Remote Health*, 17, 3879. doi:10.22605/RRH3879

Stearns, R. L., Casa, D. J., O'Connor, F. G., & Lopez, R. M. (2016). A tale of two heat strokes. *Current Sports Medicine Reports*, 15, 215–218. doi:10.1249/JSR.0000000000000265

Stern, R. A., Daneshvar, D. H., Baugh, C. M., Seichepine, D. R., Montenigro, P. H., Riley, D. O., … McKee, A. C. (2013). Clinical presentation of chronic traumatic encephalopathy. *Neurology*, 81, 1122–1129. doi:10.1212/WNL.0b013e3182a55f7f

Strebel, S., Lam, A. M., Matta, B. F., & Newell, D. W. (1997). Impaired cerebral autoregulation after mild brain injury. *Surgical Neurology*, 47, 128–131. doi:10.1016/S0090-3019(96)00459-4

Sullivan, N. (2015). An integrative review: Instructional strategies to improve nurses' retention of cardiopulmonary resuscitation priorities. *International Journal of Nursing Education Scholarship*, 12, 12. doi:10.1515/ijnes-2014-0012

Teunissen, L. P. J., de Haan, A., de Koning, J. J., & Daanen, H. A. M. (2012). Telemetry pill versus rectal and esophageal temperature during extreme rates of exercise-induced core temperature change. *Physiological Measurement*, 33, 915–924. doi:10.1088/0967-3334/33/6/915

Theuns, D. A. M. J., Smith, T., Hunink, M. G. M., Bardy, G. H., & Jordaens, L. (2010). Effectiveness of prophylactic implantation of cardioverter-defibrillators without cardiac resynchronization therapy in patients with ischaemic or non-ischaemic heart disease: A systematic review and meta-analysis. *Europace*, 12, 1564–1570. doi:10.1093/europace/euq329

Toresdahl, B. G., Rao, A. L., Harmon, K. G., & Drezner, J. A. (2014). Incidence of sudden cardiac arrest in high school student athletes on school campus. *Heart Rhythm*, 11, 1190–1194. doi:10.1016/j.hrthm.2014.04.017

U.S. Department of Labor. (2017). *Labor force statistics from the current population survey*. Retrieved from https://www.bls.gov/cps/data.htm

Valenzuela, T. D., Roe, D. J., Cretin, S., Spaite, D. W., & Larsen, M. P. (1997). Estimating effectiveness of cardiac arrest interventions: A logistic regression survival model. *Circulation*, 96, 3308–3313. doi:10.1161/01.CIR.96.10.3308

Vrijens, D. M., & Rehrer, N. J. (1985). Sodium-free fluid ingestion decreases plasma sodium during exercise in the heat. *Journal of Applied Physiology*, 86, 1847–1851.

Wasilko, S. M., & Lisle, D. K. (2013). Automated external defibrillators and emergency planning for sudden cardiac arrest in Vermont high schools. *Sports Health*, 5, 548–552. doi:10.1177/1941738113484250

Winger, J. M., Dugas, J. P., & Dugas, L. R. (2011). Beliefs about hydration and physiology drive drinking behaviours in runners. *British Journal of Sports Medicine*, 45, 646–649. doi:10.1136/bjsm.2010.075275

Wisten, A., & Messner, T. (2009). Symptoms preceding sudden cardiac death in the young are common but often misinterpreted. *Scandinavian Cardiovascular Journal*, 39, 143–149. doi:10.1080/14017430510009168

Xu, X., Karis, A. J., Buller, M. J., & Santee, W. R. (2013). Relationship between core temperature, skin temperature, and heat flux during exercise in heat. *European Journal of Applied Physiology*, 113, 2381–2389. doi:10.1007/s00421-013-2674-z

Yokota, M., Berglund, L. G., Santee, W. R., Buller, M. J., Karis, A. J., Roberts, W. S., … Hoyt, R. W. (2012). Applications of real-time thermoregulatory models to occupational heat stress. *Journal of Strength and Conditioning Research*, 26(Suppl. 2), S37–S44. doi:10.1519/JSC.0b013e31825ceba4