

5-5-2012

The Secondary School Football Coach's Relationship with the Athletic Trainer and Their Perspectives on the Recognition and Prevention of Exertional Heat Stroke

William M. Adams

William M. Adams, william.adams@uconn.edu

Recommended Citation

Adams, William M., "The Secondary School Football Coach's Relationship with the Athletic Trainer and Their Perspectives on the Recognition and Prevention of Exertional Heat Stroke" (2012). *Master's Theses*. 284.
https://opencommons.uconn.edu/gs_theses/284

This work is brought to you for free and open access by the University of Connecticut Graduate School at OpenCommons@UConn. It has been accepted for inclusion in Master's Theses by an authorized administrator of OpenCommons@UConn. For more information, please contact opencommons@uconn.edu.

The Secondary School Football Coach's Relationship with the Athletic Trainer and Their
Perspectives on the Recognition and Prevention of Exertional Heat Stroke

William Michael Adams

B.S., University of Wisconsin-Madison, 2009

A Thesis

Submitted in Partial Fulfillment of the

Requirements for the Degree of

Masters of Science

at the

University of Connecticut

2012

APPROVAL PAGE

Masters of Science Thesis

The Secondary School Football Coach's Relationship with the Athletic Trainer and Their
Perspectives on the Recognition and Prevention of Exertional Heat Stroke

Presented By: William Michael Adams, B.S., ATC, LAT

Major Advisor _____

Douglas J. Casa, PhD, ATC, FNATA, FACSM

Associate Advisor _____

Stephanie M. Mazerolle, PhD, ATC

Associate Advisor _____

Laura J. Burton, PhD, ATC

Acknowledgements

Dr. Mazerolle: I want to thank you for being my thesis advisor. The guidance and support that you have provided me over the past two years has been very instrumental in my graduate experiences.

Dr. Casa: I want to thank you for giving me the opportunity to do my graduate studies at the University of Connecticut. The experiences that I have received at UConn are above and beyond anything that I would have received anywhere else. I also want to thank you for giving me the opportunity to continue my graduate studies at UConn for a doctorate degree.

Dr. Burton: I want to thank you for serving on my thesis committee. Your input and feedback has been very instrumental in completing this document.

Table of Contents

Abstract	vi
Chapter	
1. Review of the Literature	
a. Introduction	1
b. Sudden Cardiac Death	2
c. Cervical Spine	12
d. Exertional Heat Stroke	17
e. Exertional Sickling	24
f. Hyponatremia	28
g. Exercise Induced Anaphylaxis	33
h. Exercise Induced Asthma	36
i. Head Trauma	39
j. Traumatic Injury	45
k. Lightning	51
l. Coaching Certification	54
m. Legislation and Coaches	57
n. Coaches Knowledge and First Aid Skills	58
o. Appropriate Medical Care in the Secondary School Setting	60
p. References	61
2. Introduction	75
3. Methods	84
4. Results	84
5. Discussion	90
6. References	99

Appendix A: Invitation Letter	104
Appendix B: Background Questionnaire	105
Appendix C: Interview Guide	108

The Secondary School Football Coach's Relationship with the Athletic Trainer and Their Perspectives on the Recognition and Prevention of Exertional Heat Stroke

Context: Prior research has examined the first aid knowledge and decision making among high school coaches, but little is known about their knowledge of sudden death in sport or their relationship with an athletic trainer (AT). **Objective:** Evaluate the knowledge of the secondary school football coach regarding sudden death in sport and their relationship with an AT. **Design:** An exploratory qualitative study utilizing asynchronous online interviewing. **Setting:** Web-based management system. **Patients or Other Participants:** 38 secondary school head football coaches (37 males, 1 female) participated in this study with an average age of 47±10 years old and 12±9 years experience as a head football coach. **Data Collection and Analysis:** Participants responded to a series of online questions by journaling their thoughts and experiences. Questions were focused on knowledge of sudden death, prevention strategies, and professional relationships with ATs. Multiple analyst triangulation and peer review were included as steps to establish data credibility. The data was analyzed borrowing from the principles of a general inductive approach. **Results:** Two dominant themes emerged from the data: *the influence of the AT* and *self-efficacy of the secondary school coach*. The first theme illustrated the secondary school coach's positive professional relationships with ATs regarding patient care and emergency procedures. Thirty-four of the 38 coaches who participated, indicated positive interactions with their AT. The second theme highlights the confidence, due to basic emergency care training, of the coach regarding management of an emergency situation, despite a lack of knowledge. **Conclusions:** The secondary school football coach values and understands the role of the AT regarding patient and emergency care. The secondary coach, however, is unaware of the potential causes of sudden death in sport, symptoms associated with some conditions of sudden death, and holds a higher self-confidence in management abilities than indicated by their knowledge level. **Word Count:** 299

Literature Review

Sudden death in sport has been an issue of concern for decades. Maron et al¹ looked at 1866 deaths in sport over a 26 year period. Of the 1866 deaths, 1049 (56%) were due to cardiovascular disease, 416 (22%) to blunt trauma, 65 (3%) to commotio cordis, and 46 (2%) to heat stroke. Causes of sudden death such as exertional heat stroke have seen an increasing number of deaths each year. From 2005-2009 there were a reported 18 deaths resulting in exertional heat stroke, the most in any five-year reporting period since 1975.³ In addition to sudden cardiac death and exertional heat stroke, other causes of sudden death are exertional sickling, cervical spine injuries, exertional hyponatremia, exercise-induced anaphylaxis, exercise-induced asthma, head trauma, lightning injuries, and traumatic injury.

The 2009-2010 school years reported over 7.5 million student

athletes participating in sports at the high school level.⁴ of the 25,000 high schools in the United States, only about 42% employ an Athletic Trainer to care for sport related injuries, leaving the high school coaches responsible for handling sport-related injuries.⁵ The purpose of this literature review is to provide an overview of the top ten causes of sudden death in sport. Also, this literature review will discuss evidence showing the

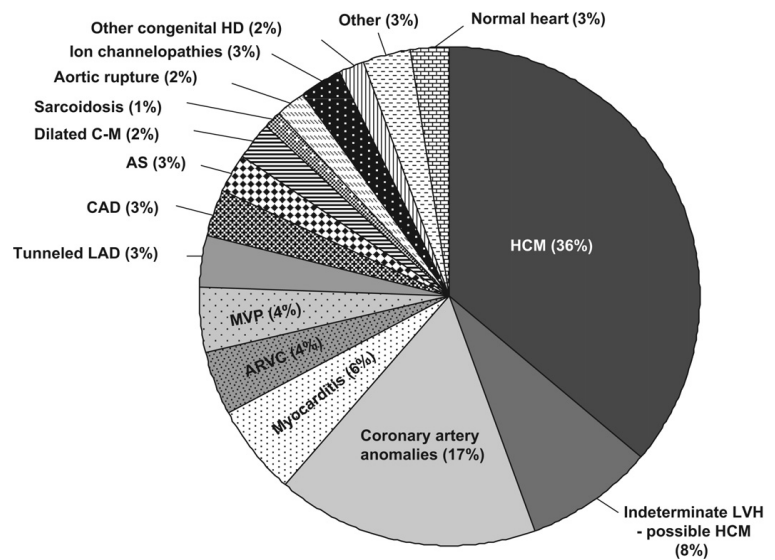


Figure 1. Distribution of cardiovascular causes of sudden death in 1,435 young competitive athletes. ARVC, arrhythmogenic right ventricular cardiomyopathy; AS, aortic stenosis; CAD, coronary artery disease; C-M, cardiomyopathy; HCM, hypertrophic cardiomyopathy; HD, heart disease; LAD, left anterior descending;

secondary school coaches' knowledge and preparedness of handling emergency situations in sport and appropriate medical coverage in the secondary school setting.

SUDDEN DEATH IN SPORT

Sudden Cardiac Death

Epidemiology. Sudden cardiac death (SCD) represents a small but significant number of deaths per year and accounts for 78% of sudden deaths in athletes. Congenital or acquired cardiac malformations, such as Hypertrophic Cardiomyopathy (HCM) and congenital coronary artery anomalies, account for 85% of sudden deaths in athletes below the age of 35.^{6,7} Figure 1 shows that roughly one third (36% in the United States) of sudden deaths arise from HCM while 17% arise from coronary artery anomalies.^{2,8}

Along with

congenital

malformations

causing sudden

cardiac death,

commotio

cordis, and

myocarditis,

	No. of Athlete-Years	No. of Deaths	Death Rate (per year)
NCAA athletes	1 969 663	45	1:43 770
Sex			
Male	1 126 557	34	1:33 134
Female	843 106	11	1:76 646
Ethnicity			
Black	300 835	17	1:17 696
White	1 583 635	27	1:58 653
By division			
Division I	788 023	27	1:29 186
Division II	424 572	10	1:42 457
Division III	760 258	8	1:95 032

Table 1. Incidence of SCD in NCAA athletes according to sex, ethnicity, and division, 2004-2008.¹²

need to be considered as separate concepts leading to SCD. The Minnesota Heart Institute Foundation has listed commotio cordis and myocarditis as the second and fourth leading cause of sudden cardiac death in athletes accounting for roughly 20% and 5-6% of the deaths respectively.^{9,10}

Studies,^{1,8,11} have shown that the occurrence of sudden cardiac death among high school and college athletes (based on 1.4 million athletes in 27 sports over 12 years) was 1/200,000. Of those deaths, males accounted for 1/133,000 deaths and females accounted for 1/796,000 deaths. Another study¹² looked at deaths at the NCAA level from 2004-2008. During the 5-year reporting period there were 273 deaths among collegiate athletes. Medical causes contributed to 29% (n=80) of the number of deaths with 56% (45/80) of the deaths being contributed to cardiac death.¹² Table 1 shows the incidence of SCD in NCAA athletes according to sex, ethnicity, and division from 2004-2008. Unfortunately, the initial symptom in 73% of the population with a cardiac anomaly is death.^{1,8,11}

Maron et al⁸ looked at clinical, demographic, and pathological profiles in young competitive athletes suffering from sudden death.

From the years 1985 through 1995 there were a recorded number of 158 sudden deaths in a population with a mean age of 17. One hundred thirty four of the 158 (85%) deaths were caused by cardiovascular diseases. Forty-eight (36%) of the athletes that died from cardiac causes

suffered from HCM, compared to the 13% that suffered from anomalous coronary artery malformations. Ninety percent of the sudden

deaths arising from cardiac diseases were of males.⁸ Dividing the cases of sudden death into race (Figure 2), 70 cases (52%) were white, 59 (44%) were African American, and the remainder were Asian, Mexican, or Native American.⁸ Football and basketball were

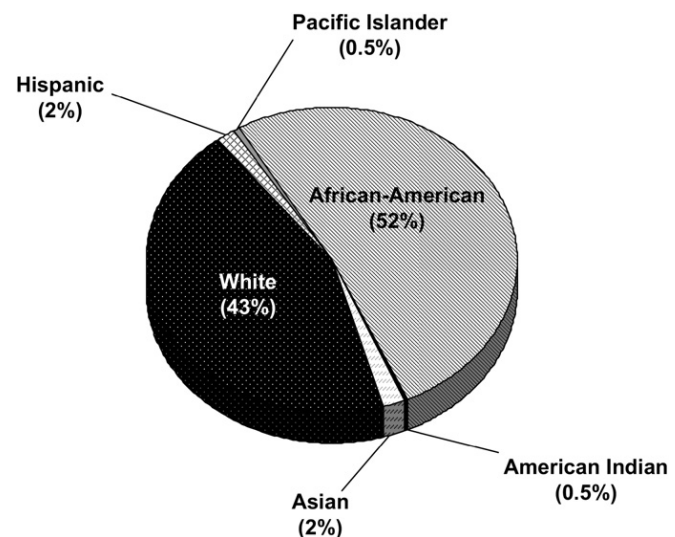


Figure 2. Sudden death in young athletes due to HCM, with respect to race. Although HCM is very uncommonly diagnosed clinically in African-Americans, the majority of sudden deaths on the athletic field due to HCM are in young

the two sports that had the most sudden death cases with 92. Of the 134 sudden deaths arising from cardiac origin, 121 (90%) athletes collapsed during or immediately after training (78 cases) or athletic competition (43 cases).⁸ To summarize the results of this study, the majority of the sudden deaths in competitive young athletes occurred in young males competing in football and/or basketball and was the result of cardiac malformations.

Etiology. Hypertrophic Cardiomyopathy, the leading cause of cardiac related sudden death, is an inherited genetic disorder.¹³ HCM is defined as an asymmetrical hypertrophy of the heart's left ventricle usually exceeding a thickness of 15mm along with a nondilated left ventricle. It is an autosomal dominant disorder and has variant penetrance in the individuals it affects. Sarcomeric protein mutations are most often attributed to the disease and histologically presents with myocyte hypertrophy, myofibril disarray, and interstitial fibrosis.^{2,10,14,15} Of the eleven identified mutations to Sarcomeric proteins, mutations to beta-myosin heavy chain and myosin-binding protein C have been shown to be the most common among people with HCM.² The structural and histological changes in a person with HCM create an arrhythmogenic environment that can lead to a fatal arrhythmia. The mechanisms that cause the fatal arrhythmia are myocardial ischemia, abnormal wall thickness of intramural arterioles, rapid atrioventricular conduction and atrial arrhythmias.¹⁶ This genetic disorder is found in American populations at a ratio of 1-2/1000^{1,8,11} and can be seen in as many as 1/500 people.^{2,13,15} The ratio of HCM in males to females is 5:1 and the prevalence among males is 1:500.^{1,8,11} With the rate of sudden death in males (1/133,000) and the prevalence of HCM in males (1/500), it is suggested that 1/266 males with HCM die each year.^{1,8,11}

Another congenital condition that can cause sudden death and can present with an unexplained cause of exercised induced syncope is an anomalous coronary artery. This is a condition, which one of the coronary arteries arises from the wrong side of the sinus; most commonly it is the left main coronary artery arising from the right sinus. An anomaly in the coronary arteries is the second leading cause of sudden cardiac death, comprising 17% of deaths.^{8,9} Patients can be asymptomatic at rest but may develop ischemia with intense exercise, which can lead to syncope, ventricular tachyarrhythmias, and death. Diagnosis is very important because it can be easily corrected with surgery, thus preventing a possible fatality in the affected athlete from going undetected.¹³ Guidelines, according to the 36th Bethesda conference, state that a patient with a diagnosed case of an anomalous coronary artery needs a six month recovery period after operative repair and needs to pass a maximal exercise stress test before resuming competition.¹³

Myocarditis is defined as inflammation of the myocardium of the heart and is responsible for roughly 5% of sudden cardiac deaths. Myocarditis is caused by an acute viral infection and is most often caused by the Cocksackie B virus. Additional causes of myocarditis, although not sport related, are drug related reactions (reaction to cocaine), immunologic responses (sarcoidosis), and physical agents (electric shock). This inflammatory process increases the risk of life-threatening ventricular arrhythmias because it causes a contractile dysfunction in the heart. Athletes with myocarditis are usually asymptomatic and the suspicion of myocarditis should increase if the athlete develops fatigue, exercise intolerance, and heart palpitations close to a systemic viral illness.^{15,17}

In addition to congenital cardiac diseases and inflammatory conditions affecting the heart, there are external factors that can also cause sudden cardiac death. Commotio cordis causes cardiac arrest from a nonpenetrating blow to the chest, specifically the myocardium, resulting in ventricular fibrillation.^{2,9,10,14,17,18} Ventricular fibrillation from commotion cordis occurs from a blow directly over the heart within the 15 to 30 milliseconds before the T-wave peak, which is the vulnerable phase of the heart's repolarization.^{2,9,10,17,18} The electrophysiological mechanisms that cause ventricular fibrillation are relatively unresolved but literature has shown that mechanical activation of ion channels appears to play a role.^{2,10,18} Commotio cordis typically affects children and adolescents because children and adolescents have pliable chest walls that aid in the transmission of energy from the chest wall to the myocardium.^{9,17,18} In addition, an impact velocity of 40 miles per hour causes ventricular fibrillation most often.^{10,18} Even with immediate management of an athlete with commotion cordis, the survival rate has only been shown to be 15%.^{2,17}

Management. Timely management in handling an athlete with sudden cardiac arrest is paramount to their survival. Athlete collapse followed by unresponsiveness should be treated as cardiac arrest until breathing and pulse are reported. Management of an athlete in sudden cardiac arrest should follow; activation of EMS, cardiopulmonary resuscitation (CPR), defibrillation using an AED, and rapid transition to advanced cardiac life support (ACLS). EMS activation and CPR should begin immediately if the athlete is found to be not breathing and has no pulse. An AED should be applied to an unresponsive athlete as soon as possible to analyze the heart's rhythm and apply an electric shock if advised. If CPR has begun before the AED is available, CPR should be

interrupted so the AED can be applied. CPR should resume after the AED analyzes the heart's rhythm and continue until EMS personnel arrive.¹⁹ Figure 3 represents a flow-chart of the proper way to manage an athlete suspected of sudden cardiac arrest.

There have been many approaches to the management of Hypertrophic Cardiomyopathy discussed in the literature and is mainly dependent upon the type of pathology identified. Abstinence from moderate to high intensity activity is warranted in patients with HCM due to the large number of sudden deaths that have been triggered by physical activity.⁶ Another approach to treatment of a patient with HCM is administering a beta-blocker.⁶ Application of a beta-blocker has shown promising results in preventing exercise induced syncope and sudden cardiac death by controlling HCM in patients.⁶ Another method for treating HCM is the prophylactic use of an implantable intracardiac defibrillator. This approach is usually only sought if the patient is at risk for life threatening ventricular tachyarrhythmias seen in HCM.⁶

Management of myocarditis and commotion cordis follow the same protocols as previously mentioned; activation of EMS, CPR if athlete is unresponsive, not breathing, and without a pulse, defibrillation and transition to ACLS. Any athlete who collapses immediately after being struck in the chest during activity should be suspected of having sudden cardiac arrest from commotion cordis and defibrillation should be initiated as soon as it is available. Any athlete suspected of having myocarditis should have an endomyocardial biopsy performed to get an accurate diagnosis of the disorder and removed from participation for a minimum of 6 months post diagnosis.^{17,18}

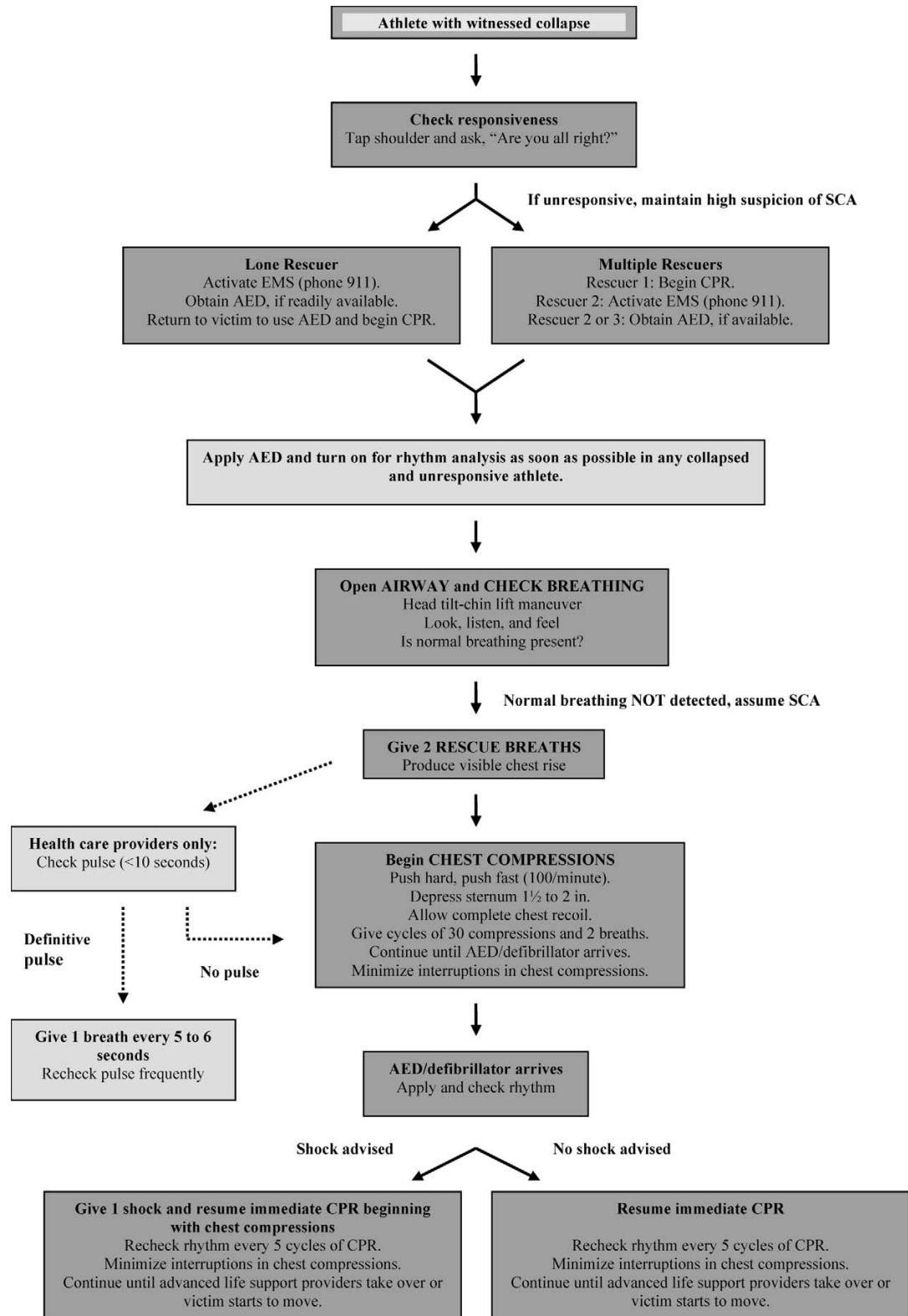


Figure 3. Management of sudden cardiac arrest. SCA indicates sudden cardiac arrest; EMS, emergency medical services; AED, automated external defibrillator; CPR, cardiopulmonary resuscitation.

Prevention. Due to the figures above, with the incidences of sudden death and their cardiac origins, it is essential to find ways to prevent sudden death from occurring. Pre-participation cardiovascular screening is a way of providing a systematic, medical approach to evaluating a large population of athletes before they participate in sports. The purpose of providing these cardiovascular screenings is to identify possible abnormalities that could provoke disease progression and sudden death.

Medical history ^a
Personal History
1. Exertional chest pain or discomfort
2. Unexplained syncope or near-syncope ^b
3. Excessive exertional and unexplained dyspnea or fatigue, associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure
Family History
6. Premature death (sudden and unexpected, or otherwise) before age 50 due to heart disease, in one or more relatives
7. Disability from heart disease in a close relative less than 50 years old
8. Specific knowledge of certain cardiac conditions in family members: hypertrophic or dilated cardiomyopathy, long QT syndrome or other ion channelopathies, Marfan syndrome, or clinically important arrhythmias
Physical Examination
9. Heart murmur ^c
10. Femoral pulses to exclude aortic coarctation
11. Physical stigmata of Marfan syndrome
12. Brachial artery blood pressure (sitting position) ^d
^a Parental verification is recommended for high
^b Judged not to be neurocardiogenic (vasovagal); of particular concern when related to exertion.
^c Auscultation should be performed in both supine and standing positions (or with Valsalva maneuver), in particular to identify murmurs of dynamic left ventricular outflow tract obstruction.
^d Preferably, taken initially in both arms.

Table 2. The 12-element AHA recommendations for preparticipation cardiovascular screening of competitive athletes.²¹

^{2,8,20,21} The purpose of the pre-participation examinations, which are mandated by high schools and colleges across the United States, is to provide the participants in sports with a form of medical clearance to participate in sports after they have been evaluated and any relevant pre-existing abnormalities have been evaluated.^{2,8,20,21}

The Pre-Participation Examination (PPE) consists of a history and physical exam. Taking a thorough history is of utmost importance because most athletes do not recognize

that any symptoms they may have experienced can be of significant importance.¹³ In addition, the history portion of the PPE (both personal and family history) detects 75% of problems.¹¹ By reporting a history of symptoms such as syncope (especially exercise induced), chest pain, and fatigue, as well as a family history of cardiac problems can help the physician in determining if more diagnostic tests are needed to find a possible underlying problem.¹³ In addition to the history, if the athlete presents with resting tachycardia or hypertension, concern should be taken. If, during the PPE, the patient complains of chest pain or syncope with exercise ordering an ECG should be warranted. However, since the patient's symptoms are exercise induced, it is rare that the ECG will show anomalies at rest. Getting a 12-lead ECG after exercise and during an episode of chest pain might reveal an abnormality. In addition to the ECG, ECHO, MRI, and Computed axial tomography can also be used as diagnostic tools because they produce a visual image of the heart and its structure.¹³ In the United States, electrocardiograms (ECGs) are not warranted unless the athlete describes symptoms of a possible cardiac problem (syncope, etc.). Similar to the ECG, an Echocardiogram (ECHO) is not ordered unless there is reason to do so.¹³

In 2007 the American Heart Association (AHA) developed a list of 12 recommendations for physicians to follow as an effective strategy to raise the suspicion of cardiovascular disease and is shown in Table 2.²¹ Of the 12 recommendations, eight of them come directly from the personal and family history, and the remaining four come from the physical examination. The recommendations for personal history include: exertional chest pain, unexplained syncope/near-syncope, excessive exertional and unexplained dyspnea/fatigue associated with exercise, prior recognition of a heart

murmur, and increased blood pressure. Recommendations for family history are: premature death before the age of 50 due to heart disease, disability from heart disease in a close relative less than 50 years of age, and specific knowledge of cardiovascular disorders. Recommendations for the physical examination are: heart murmur, femoral pulses to exclude aortic coarctation, physical stigmata of Marfan's Syndrome, and brachial artery blood pressure (while sitting).²¹

In addition to pre-participation exams, preventing sudden cardiac arrest requires that any venue that

sponsors an athletic event have a written and structured emergency action plan (EAP). The EAP should be specific for	Year	Head		Cervical Spine	
		Frequency	Percent	Frequency	Percent
each location and	1945-1954	87	17.1	32	27.3
	1955-1964	115	22.5	23	19.7
	1965-1974	162	31.8	42	35.9
	1975-1984	69	13.5	14	12
	1985-1994	33	6.5	5	4.3
include the following; emergency communication, personnel, equipment, and transport to the appropriate medical facility. Emergency communication involves having a detailed plan of who would activate EMS if an emergency situation would arise during an athletic event. It also describes the location of telephones, walkie talkies, alarms, or intercom systems that can be used to activate the emergency response system. Having a detailed plan of communication prevents delays in time that may be critical for the injured athlete. The EAP should also describe the personnel that should manage emergency situations along with the equipment available to the responders. The first person that responds to an	1995-2004	44	8.6	1	0.8
	Totals	510	100	117	100

Table 3. Head and cervical spine fatalities in 5-year increments from 1945-2004.³

include the following; emergency communication, personnel, equipment, and transport to the appropriate medical facility. Emergency communication involves having a detailed plan of who would activate EMS if an emergency situation would arise during an athletic event. It also describes the location of telephones, walkie talkies, alarms, or intercom systems that can be used to activate the emergency response system. Having a detailed plan of communication prevents delays in time that may be critical for the injured athlete. The EAP should also describe the personnel that should manage emergency situations along with the equipment available to the responders. The first person that responds to an

emergency situation should be familiar with the location's EAP and be quick to initiate the emergency action plan if required. All emergency equipment should be easily accessible at each site and should include a pocket mask (or other barrier) for rescue breathing, and an AED for early defibrillation. Lastly, the EAP should spell out the plan to transport an athlete with sudden cardiac arrest to the proper receiving medical facility to get the most appropriate care. The emergency action plan is essential in managing a situation of sudden cardiac arrest to help in preventing death of the athlete. The EAP should be practiced annually with all persons involved in emergency care at athletic venues to ensure timely care to injured athletes.¹⁹

Cervical Spine

Epidemiology. Fatalities in sport due to cervical spine injuries have accounted for 117 deaths from 1945-2004.³ The number of fatalities increased dramatically over the first three decades (1945-1974) and have since dramatically decreased. Table 3 shows the number of deaths per decade as it relates to cervical spine injuries. The dramatic decrease in fatalities since 1974 is attributed to the rule change in 1976 that eliminated players from using the head and face as the initial point of contact during activity. Data²² has shown that from 1977-2009 there were a total of 307 that had incomplete neurological recovery from spinal cord injuries and 253 of those injured were high school players. It is estimated that there are 11,000 new cases of spinal cord injuries in the United States each year with participation in sport being the fourth leading cause of spinal cord injury. In addition, during the first 3 decades of life, sport related spinal cord injuries account for second most out of all spine related injuries.^{23,24 25} The sport of football alone accounts

for the greatest number of catastrophic spinal injuries when compared to all sports played in the United States.^{24 26,27 3 3}

In football alone, cervical spine injuries occur in 10-15% of the players and most frequently among offensive and defensive lineman and linebackers. Also, cervical spine injuries occurred most often in games over practices and scrimmages. Those playing defense accounted for 71% of the injuries and of those 71%, 69% of those happened while making a tackle.²³ Defensive players are also 4 times more likely to have a fatal cervical spine injury.²⁸ Twenty-five percent of the injuries occurred when the athletes used improper tackling techniques and the result of 79% of the injuries was a fracture/dislocation of the cervical spine vertebrae.²³

Etiology. The primary mechanism for injury for a cervical spine injury is axial loading on the cervical spine.²⁸⁻³⁰ Studies by Torg et al^{31,32} helped dispel the common theories of possible mechanisms leading to cervical spine injuries and identifying that axial loading with the head down as the lone mechanism of injury. When a football player sustains a hit to the head the forces are dissipated by the paravertebral muscles, eccentric contractions, and intervertebral discs. Although these structures provide a good way of dissipating forces along the cervical spine, they can be injured when contact occurs on the top of the head resulting in a distribution of forces along the vertical axis of the cervical spine.²⁸ When the head is in a neutral position the cervical spine is extended due to the natural lordotic curve that is present. When force is applied to the head in a neutral position the forces are dissipated throughout the paravertebral muscles. When the head is flexed to 30 degrees the lordotic curve disappears so any force applied to the head while in this position causes a longitudinal force to be applied greatly increasing the risk

of injury.²⁸ When an axial load is applied the cervical spine becomes compressed between the head (which is stopped by the surface it hits) and the decelerating trunk. If the compressive force exceeds the critical level in which the cervical spine can withstand the cervical spine fails in flexion. The result is a fracture, subluxation, or dislocation of the cervical vertebrae.²⁸ The factors that must be considered when dealing with a cervical spine injury is the velocity of the resultant force, the point of contact on the head relative to the axis of the cervical spine, cervical spine buckling, and the surface in which the head came into contact with. The biggest factor to consider when determining the magnitude of injury is how much of the spinal cord is involved in the injury.²⁴

At the physiologic level, destruction of the spinal cord can occur, even if complete transection of the cord does not occur. Even if the cord is not completely transected, full motor and sensory losses can still occur and this is greatly dependent upon the degree and duration of the sustained trauma. Both acute vasospasm of the capillary network within the spinal cord and edema-causing hemorrhagic necrosis can lead to the destruction of the spinal cord. Disruption of spinal cord perfusion leads to hemorrhage of gray matter in addition to disrupting the sodium potassium pumps, which are vital for repolarization of the cord. Breakdown of the cellular membranes allow calcium to enter the cells, which is cytotoxic to the spinal cord, and breakdown of gray matter soon ensues. This cascade of events takes place within the first 2 hours of initial injury so immediate recognition and treatment is of absolute necessity.²⁴

Management. An athlete with a suspected cervical spine injury should be assessed immediately to determine the severity of the injury. Any or all of the following requires immediate cervical spine injury management: unconsciousness or altered level of

consciousness, bilateral neurologic findings or complaints, significant cervical spine pain with or without palpation, and obvious spinal column deformity.²⁴ Also, the Glasgow-coma scale can be used as a determinant of cervical spine injury in that a Glasgow-coma score of 8 or less is a good predictor of cervical spine injury with a lower score relating to a greater degree of injury.³³

If, after the initial assessment, the athlete is suspected of having a cervical spine injury proper management of the injury is necessary. The medical professional evaluating the athlete must manually stabilize the head and neck. Holding the head and neck in a neutral position minimizes additional movement, thus preventing further injury. If the athlete's head and neck is not in a neutral position upon arrival to the injured athlete, the head and neck must be moved into a neutral position allowing access to the airway and to prevent compression of the spinal cord. In addition to stabilizing the head and neck, the airway must be maintained in case that the athlete goes into respiratory distress. To ensure that movement is minimized during transport to a medical facility full-body immobilization is used via the use of a spine board. In sports where the athlete is required to wear protective equipment, like in American football for example, it can hinder the medical professional from getting access to the airway because of the helmet and facemasks all players are required to wear. Removing the helmet is not recommended in football because removing the helmet will cause extension of the neck due to the thickness of the shoulder pads the players wear. Since the helmet helps keep the head and neck in a neutral position the facemask must be removed to get access to the airway. Removal of the facemask must be done in a way that minimizes movement but can be quick and efficient. Battery powered drills are a quick and easy tool to use but medical

professionals should have more than one tool at their disposal in case the first tool fails. In addition to using a battery-powered drill many medical professionals use cutting tools capable of cutting through the plastic facemask clamps.²⁹

Prevention. The 1976 rule change in American football making it illegal to make initial contact with an opponent with the head or face has dramatically decreased the number of catastrophic cervical spine injuries.^{3,28,29} In

Year	Deaths
1975-1979	8
1980-1984	9
1985-1989	5
1990-1994	2
1995-1999	13
2000-2004	11
2005-2009	18
Five Year Average	9

Table 4. Deaths due to exertional heat stroke from 1975-2009.³

addition to rule changes education and officiating are the best prevention strategies to reduce the number of cervical spine injuries in sport. In sports like football, both players and coaches must be taught the correct techniques in blocking and tackling to reduce the

incidence of spinal injury. Leading with the shoulders and keeping the head and face up is the correct technique to use and must continue to be taught. In addition, educating players and coaches about the risks of using improper techniques can also be used as a prevention technique. Lastly, officiating can help aid in preventing cervical spine injuries. Having officials penalize teams for every illegal

Signs and Symptoms
High body-core temperature (>40C [104F])
Central Nervous System Changes: Dizziness, Drowsiness, Irrational behavior, Confusion, Irritability, Hysteria, Apathy, Aggressiveness, Delirium, Disorientation, Staggering, Seizures, Loss of consciousness, Coma
Dehydration
Weakness
Hot and wet or dry skin
Tachycardia (100-200 beats per minute)
Hypotension
Hyperventilation
Vomiting
Diarrhea

Table 5. Signs and symptoms of exertional heat stroke.³⁶

head contact made during a game will further force players and coaches to use proper techniques that have been shown to prevent cervical spine injuries.^{24,28,30}

Exertional Heat Stroke

Epidemiology. According to research published by the National Center for Catastrophic Sports Injury, since 1995 there have been 46 deaths due to exertional heat stroke (EHS) in football players with 35 being at the high school level, 8 at the collegiate level, 2 at the professional level, and one at the sandlot level. In 2010 alone, there were 4 heat stroke deaths that were reported.³ Table 4 breaks down the number of deaths due to exertional heat stroke from 1975-2009 in 5 year blocks to show the increasing number of heat stroke deaths in recent blocks of time. Death from exertional heat stroke is 100% preventable, but yet it is the leading cause of death and disability amount U.S. high school athletes.³⁴

Exertional heat stroke is defined as a body core temperature above 40 degrees Celsius in addition to central nervous system dysfunction and organ system failure.^{35,36} Additional signs and symptoms of exertional heat stroke are tachycardia, hypotension, sweating (the skin may be wet or dry at the time of collapse), hyperventilation, altered mental status, vomiting, diarrhea, seizures, and coma.^{36 36} Table 5 shows a full list of signs and symptoms of exertional heat stroke along with the possible CNS changes. Exertional heat stroke occurs when the thermoregulatory system fails to dissipate heat at the rate of which heat is produced by the working muscles of the body.^{35,36}

Etiology. The thermoregulation center is located in the hypothalamus and is in charge of regulating heat production and dissipation in the body. Core temperature is operated though an open-ended feedback loop in the body by the hypothalamus receiving

signals from peripheral heat sensors and circulating blood. Heat production is then regulated by the body via cutaneous vasodilation, increased sweat rate, increased heart rate, and increased respiratory rate. The heat production and heat storage equation of $S = M (+/-) R (+/-) K (+/-) C_v (-) E$ is used to determine core body temperature. S is the amount of heat stored in the body, M is the metabolic heat production of the body, R is the amount of heat gained or lost via radiation, K is the amount of heat gained or lost via conduction, C_v is the amount of heat gained or lost via convection, and E is evaporative heat loss.³⁶

There are both environmental and non-environmental factors that can put an athlete at risk for exertional heat stroke. Environmentally, ambient heat temperature, relative humidity, air motion, and the amount of radiant heat from the sun or other sources are factors that can put athletes at risk for exertional heat stroke. Non-environmental factors that

must be considered are described in Table 6 and include factors such as dehydration, lack of acclimatization to heat, history of heat illness, and poor physical conditioning.

Risk Factor	
	Dehydration
	Barriers to Evaporation
	Illness
	History of Heat Illness
	Increased Body Mass Index
	Wet-Bulb Globe Temperature on Previous Day and Night
	Poor Physical Conditioning
	Excessive or Dark Colored Clothing or Equipment
	Overzealousness
	Lack of Acclimatization to Heat
	Medications and Drugs
	Electrolyte Imbalance

Predisposing medical conditions such as

Table 6. Non-environmental risk factors that put athletes at risk for exertional heat stroke.³⁶

Malignant Hyperthermia, Neuroleptic Malignant Syndrome, Atherosclerotic Vascular

Disease, Scleroderma, Cystic Fibrosis, and Sickle Cell Trait are also risk factors that need to be considered in an athlete participating in the heat.^{35,36}

As core body temperature rises about critical levels (40 degrees Celsius), both cell membranes and energy systems are damaged. This damage causes a series of events at the cellular level that disrupts cell volume, metabolism, acid-base balance and membrane permeability. The result of this chain of events leads to organ dysfunction and eventual death if no intervention is implemented. At the whole body level, this series of events explains the CNS, cardiac, renal, gastrointestinal, hematologic, and muscle dysfunction associated with an exertional heat stroke case. The extent of tissue injury and damage is directly related to the amount of time the tissues are above threshold temperature. The longer the body's tissues are above threshold core temperature the greater the risk of morbidity and mortality of the tissues and person as a whole.³⁵

Management. Recognition of exertional heat stroke is of utmost importance and immediate recognition is vital in survival of the athlete.³⁵ Signs and symptoms of EHS are often non-specific and are dependent upon the degree and duration of EHS.

Subjectively, signs and symptoms may include disorientation, confusion, dizziness, irrational or unusual behavior, irritability, as well as others described in Table 5.^{35,36}

Diagnosis of exertional heat stroke is primarily based on a measurement of core body temperature (above 40 degrees Celsius). Any athlete who exhibits signs and symptoms of EHS should have their core body temperature assessed via a rectal thermometer. Research³⁷⁻³⁹ have showed that rectal thermometry is the most valid way to assess core body temperature and should be used as the gold standard for diagnosing an exertional heat stroke.

Immediate treatment via aggressive whole body cooling is pertinent in the survival of the athlete suffering from exertional heat stroke.^{35,36,40-42} Research^{35,41,42} has shown that the quickest way to lower core body temperature is via cold or ice water immersion and is considered the gold standard to whole body cooling. With cold and ice water immersion therapy, cooling rates of .15-.24 degrees Celsius per minute have been observed providing further evidence that this whole body cooling technique is the most efficient way of lowering an athlete's core body temperature.³⁵ If cold or ice water immersion is not possible at the event location, cooling the athlete's body should be done by any means possible. McDermott et al⁴³ presents a systematic review for acute whole-body cooling for exertional heat stroke. Figure 1 shows the cooling rates for different techniques showing which techniques are most effective in cooling the body if immersion therapy is not possible. Two common misconceptions regarding cold and ice water immersion are peripheral vasoconstriction that slows the cooling rate and the increased risk of cardiogenic shock. Although peripheral vasoconstriction does occur, it is overpowered by the cooling potential that immersion provides. Cardiogenic shock has not been substantiated in literature as it relates to cooling of patients with heat stroke.³⁶ Table 7 provides other common myths surrounding exertional heat stroke in addition to the aforementioned reasons. Survival of EHS is almost guaranteed if rapid cooling is initiated within 10 minutes of collapse.⁴¹

Figure 4. Mean cooling rates from case reports and critically reviewed articles. Mean cooling rates defined as unacceptable are $<0.0786^{\circ}\text{C}/\text{min}$, acceptable are $0.0786^{\circ}\text{C}/\text{min}$ to $0.1546^{\circ}\text{C}/\text{min}$, and ideal are $\geq 0.1556^{\circ}\text{C}/\text{min}$. IV indicates intravenous.⁴³

- a Ice-water immersion, 26°C (n = 7): $0.356^{\circ}\text{C}/\text{min}$
- b Ice-water immersion, $1-36^{\circ}\text{C}$ (n = 14): $0.26^{\circ}\text{C}/\text{min}$
- c Cold-water immersion, 20°C (n = 7): $0.196^{\circ}\text{C}/\text{min}$
- d Cold-water immersion, 8°C (n = 7): $0.196^{\circ}\text{C}/\text{min}$
- e Fine spray (temperature not reported) (n = 2): $0.1756^{\circ}\text{C}/\text{min}$
- f Cold-water immersion, 14.03°C (n = 17): $0.166^{\circ}\text{C}/\text{min}$
- g Ice-water immersion, 5.15°C (n = 17): $0.166^{\circ}\text{C}/\text{min}$
- h Dousing with water while fanning (n = 52): $0.156^{\circ}\text{C}/\text{min}$
- i Cold-water immersion (temperature not reported) (n = 39): $0.156^{\circ}\text{C}/\text{min}$
- j Cold-water immersion, 14°C (n = 7): $0.156^{\circ}\text{C}/\text{min}$
- k Continual dousing with ice-bag massage (n = 5): $0.146^{\circ}\text{C}/\text{min}$
- l Cold-water immersion, 7°C (n = 7): $0.1296^{\circ}\text{C}/\text{min}$
- m Ice-wet towels (n = 7): $0.116^{\circ}\text{C}/\text{min}$
- n IV fluids and ice packs at major arteries (n = 1): $0.1076^{\circ}\text{C}/\text{min}$
- o Helicopter downdraft with spraying (n = 2): $0.1026^{\circ}\text{C}/\text{min}$
- p IV fluids and ice-wet towels (n = 1): $0.0976^{\circ}\text{C}/\text{min}$
- q IV fluids (n = 1): $0.0766^{\circ}\text{C}/\text{min}$
- r Fine spray, compressed air, and fanning (n = 6): $0.0766^{\circ}\text{C}/\text{min}$
- s Fine spray with fanning (n = 6): $0.0736^{\circ}\text{C}/\text{min}$
- t Sitting on stool, 21.16°C (n = 6): $0.0666^{\circ}\text{C}/\text{min}$
- u Fine spray for 3 minutes with fanning (n = 6): $0.056^{\circ}\text{C}/\text{min}$
- v Cold IV and dousing with water (n = 1): $0.056^{\circ}\text{C}/\text{min}$
- w Dousing with water (n = 1): $0.0446^{\circ}\text{C}/\text{min}$
- x Cold-water immersion, 14.46°C (n = 6): $0.0446^{\circ}\text{C}/\text{min}$
- y IV fluid with haloperidol (n = 1): $0.0416^{\circ}\text{C}/\text{min}$
- z Fanning and compressed air (n = 6): $0.046^{\circ}\text{C}/\text{min}$
- aa Ice packs at major arteries and dousing with fanning (n = 5): $0.0366^{\circ}\text{C}/\text{min}$
- bb Dousing with water while fanning (n = 5): $0.0356^{\circ}\text{C}/\text{min}$
- cc Ice packs covering body (n = 5): $0.0346^{\circ}\text{C}/\text{min}$
- dd Ice packs at major arteries (n = 5): $0.0286^{\circ}\text{C}/\text{min}$
- ee Lying on stretcher (n = 5): $0.0276^{\circ}\text{C}/\text{min}$
- ff Fanning only (n = 6): $0.026^{\circ}\text{C}/\text{min}$
- gg Repeated gastric lavage (n = 1): $0.0186^{\circ}\text{C}/\text{min}$
- hh IV fluid with paracetamol (n = 1): $0.0156^{\circ}\text{C}/\text{min}$
- ii Ice cubes on chest (n = 1): $0.0086^{\circ}\text{C}/\text{min}$
- jj Cooling blankets (n = 1): $0.00766^{\circ}\text{C}/\text{min}$
- kk Cooling blankets (n = 1): $0.06^{\circ}\text{C}/\text{min}$

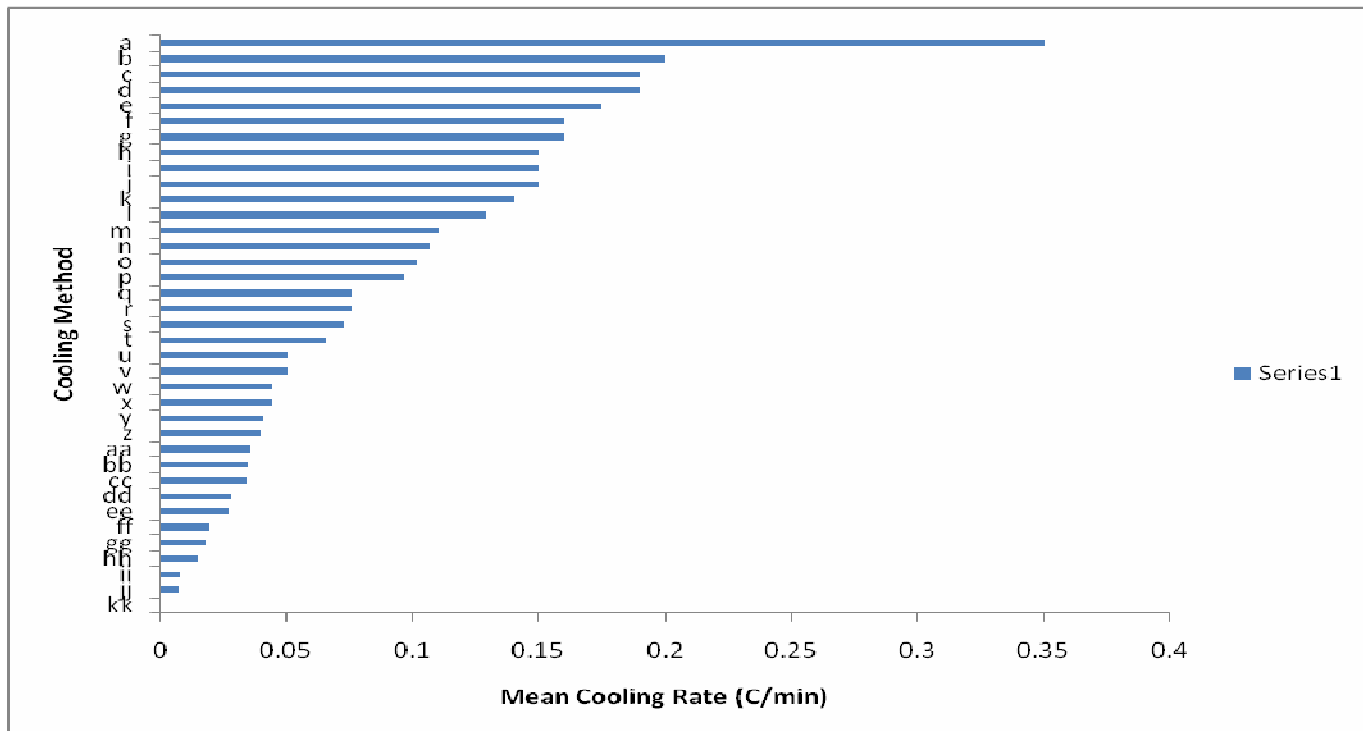


Table 7. Common misconceptions of exertional heat stroke with accompanying explanations.

Misconception	Explanation to Misconception
The onset of exertional heat stroke (EHS) is random and unpredictable.	Numerous predisposing factors of EHS have been documented. Some of these include: environmental conditions (high temperature and/or high humidity), high intensity exercise, lack of acclimatization, low physical fitness level, equipment that prevents heat loss, sleep deprivation, dehydration, fever, etc. With knowledge and recognition of these factors, high-risk individuals can be identified.
It's possible to assess body temperature by external means.	No external temperature assessment devices currently available have ever been proven valid under the conditions of intense exercise in the heat and a significant degree of hyperthermia. External temperature devices, including oral, tympanic, temporal, forehead sticker, axillary devices, should never be used to diagnose exertional heat stroke. The only device that is valid under these conditions for the immediate detection of body temperature is the rectal thermistor. The ingestible thermistor is a second viable measure. However, ingestible thermistor devices must be taken at least four hours before activity begins, therefore it cannot be used when an athlete is already suffering from an EHS.
The appearance of a lucid mental status means everything is okay.	One factor that may obscure or delay the diagnosis of EHS is the lucid interval that presents initially for many EHS patients. This lucid interval often coincides with only minimal Central Nervous System (CNS) dysfunction and misleads the caregiver regarding the severity of the condition (i.e. EHS vs. heat exhaustion). However, in the case of EHS, this lucid interval will be followed by severe CNS signs and symptoms.
Peripheral Vasoconstriction (PVC) delays cooling	While PVC may occur during cold-water immersion (CWI), it is greatly overshadowed by the great conductive and convective thermal transfer, which cools the body rapidly. Furthermore, PVC certainly occurs when a normothermic individual is placed in a cold-water bath. Although PVC may minutely increase core body temperature initially, even in a EHS victim, a rapid decrease in body temperature will immediately follow.
Shivering delays cooling	Similar to PVC, shivering will certainly occur when a normothermic individual is placed in a cold-water bath. However, this is seldom the case with a hyperthermic individual. Research shows that powerful rapid cooling will still occur in hyperthermic individuals with few occurrences of a shivering response.
Cold water immersion is uncomfortable for patient/staff It is difficult to apply supplemental treatments (AED, IV fluid, oxygen, etc.)	The physical comfort of the patient or staff should not be a primary concern during the acute treatment of EHS. Comfort, although a consideration, should be secondary to delivery of optimal treatment.
There is a risk of drowning with cold water immersion	While certainly valid, this is an emergency condition in which the risks of EHS greatly outweigh the risks or inconveniences of foregoing supplemental treatments.
Cold water immersion may be unsanitary	While unsanitary conditions may be present due to vomiting or diarrhea, an unsanitary tub is an acceptable tradeoff to a permanently disabled athlete. In the case of unsanitary conditions, the tub should be properly cleaned after use to prevent subsequent problems.
Hypothermic afterdrop (continued cooling post-immersion) can occur	Hypothermic afterdrop may be a concern if the athlete is cooled too long. However, with the use of a proper core body temperature assessment device -- rectal thermometer -- body temperature can be continuously monitored throughout cooling to prevent this afterdrop.
An athlete stops sweating during a case of EHS	Since EHS occurs during intense exercise in the heat, the athlete is almost always profusely sweating upon collapse of EHS. This is perhaps the most widely misunderstood sign of EHS and may lead to mistreatment.
An athlete must be severely dehydrated for EHS to occur	While dehydration may predispose an athlete to exertional heat illness and/or exacerbate an EHS, dehydration does not always have to be present. EHS can occur in as little as 20 minutes after the beginning of exercise before severe fluid loss is prominent. Exercise intensity and environmental conditions are the primary factors associated with EHS.

Prevention. The National Athletic Trainers' Association (NATA)³⁶ and the American College of Sports Medicine (ACSM)³⁵ have published prevention strategies as it relates to heat illness. The NATA and ACSM recommend that all athletic events should be scheduled at times to avoid extremely hot and humid conditions. During the summer months, events should

	Physiologic Variable	After Acclimatization (10-14 days)
be scheduled during	Heart rate	Decreases
times of day where it is	Stroke Volume	Increases
the coolest (early	Body-core temperature	Decreases
morning, evening, etc).	Skin temperature	Decreases
	Sweat output/rate	Increases
Also, during hot and	Onset of sweat	Earlier in training
humid conditions,	Evaporation of sweat	Increases
	Salt in sweat	Decreases
activity modifications	Work output	Increases
	Subjective discomfort (RPE)	Decreases
should be implemented.	Fatigue	Decreases
	Capacity for work	Increases
Modifications such as	Mental disturbance	Decreases
	Syncopal response	Decreases
unlimited water breaks,	Extracellular fluid volume	Increases
increased work:rest	Plasma volume	Increases

Table 8. Physiologic Responses After Heat Acclimatization Relative to Nonacclimatized State.³⁶

ratios, decreased intensity and duration of activity, and minimizing clothing/equipment requirements during extreme conditions. Maintaining proper hydration should be encouraged as dehydration places greater thermal strain on the body. Heat acclimatization is an essential prevention strategy that should be used to decrease the risk of exertional heat illness in athletes.^{35,36,45} Heat acclimatization is defined as the gradual increase in duration and intensity of exercise during the athlete's initial exposure to heat. This process occurs over a period of 10-14 days and proper heat acclimatization leads to

enhancements in physiologic function, exercise heat tolerance, and exercise performance. Table 8 shows the physiologic changes that are incurred after a period of heat acclimatization to help heat tolerance in athletes.

Exertional Sickling

Epidemiology. Sickle Cell Trait (SCT) affects 300 million individuals worldwide.⁴⁶ In the United States, it has been shown that 6-9% of African Americans and .01%-.05% of the remaining population carry the sickle cell trait.^{46-48,48-52} Sickle cell trait, a heterozygous condition, has a glutamic acid-to-valine substitution in the beta-globin gene on chromosome 11 and is denoted as HbAS.⁴⁶ Unlike the homozygous Sickle cell anemia, HbSS, HbAS is only present in 30-40% of the red blood cells of an individual and has been considered to be a benign, asymptomatic condition.⁵¹ Although considered benign and asymptomatic, SCT has been shown to have both nonlife-threatening and fatal complications.⁵¹

Sudden death by exertional sickling was first seen in 1970 in four military recruits. The recruits collapsed during basic training; three of the fourth collapses and subsequent deaths occurred during the first day of basic training and it was ruled that exertional rhabdomyolysis, a result of sickle cell trait caused the collapses and one death.⁴⁷ A study by Kark et al⁵³ showed a direct link between sickle cell trait and exertion-related deaths. Kark et al also looked into SCT related deaths in African American recruits and found that African Americans with SCT had a relative risk of sudden death 28 times more than African Americans without SCT and 40 times more than that of all recruits.⁵³

Etiology. Normally, the physiological response to exercise produces a decrease in tissue pH, an increase in tissue temperature, and an increase in cardiac output.⁵⁰ During vigorous exercise, there is an increased demand for oxygen in the working muscle leading to a hypoxic state that triggers sickling in those with SCT. Acidosis, high tissue temperature, red blood cell dehydration, and increased osmolality at the tissue level also promote a sickling event by dissociating the hemoglobin oxygen saturation curve to the right.^{47,52} Figure 5 below shows the pathogenesis of sickle cell trait associated collapse and sudden death. A hypoxic tissue state after a bout of vigorous exercise produces intravascular sickling and microvascular occlusion, which leads to rhabdomyolysis and can eventually lead to myocardial ischemia, arrhythmias, renal failure, and death.^{47,49,52}

Many people with SCT experience no problems when they are exposed to the same physical and environmental stresses. There are some risk factors that increase the chance for a sickling episode resulting in rhabdomyolysis and potentially sudden death. High environmental temperatures, high altitude, fatigue, poor physical conditioning, inadequate hydration, inadequate acclimatization to heat or altitude, and preceding or concurrent viral illness are risk factors that should be taken into an account when dealing with an athlete with SCT.^{47,49,52 54} Athletes affected by a sickling episode complain of diffuse muscle tenderness along with swelling and/or weakness after vigorous exercise. This pain is ischemic rather than due to lactic acidosis and muscle tone is normal compared to an athlete experiencing heat cramps. The most apparent symptom in an athlete suffering a sickling episode is increasing pain and weakness of the working muscles.⁴⁷ Hyperthermia, tachycardia, and hypotension may present as normal or elevated as well as the athlete presenting with a confused and agitated or lethargic state of

mind.⁵² Diagnosis of a sickling episode is primarily made by laboratory results. Rhabdomyolysis associated with sickling is shown by myoglobinuria, granular casts in the urine, and elevated serum creatine kinase levels.⁵² In addition to the above-mentioned test results, elevated uric acid levels, hyperphosphatemia, hyperkalemia, and hypocalcemia may also be the result of a sickling episode. The patient may also develop an arrhythmia and peaked t-waves from a sickling episode and is shown through electrocardiographic testing.⁵²

Management and Prevention. Immediate treatment is of utmost importance when dealing with an athlete suffering a sickling episode. Initial treatment should consist of oxygenation and fluid replacement. Forced diuresis and alkalization of the urine using an IV solution of sodium chloride and dextrose should be performed if rhabdomyolysis is diagnosed in the athlete. This treatment should be continued until myoglobinuria and hyperkalemia resolves.⁵² Prevention is the best way to prevent exertional sickling and subsequent sudden death due to a sickling episode. Conditioning regimens should progress gradually and slowly to allow for acclimatization and dehydration should be avoided. Athletes have shown the best results when allowed to work at their own pace. It is also encouraged that athletes with sickle cell trait participate in year round strength and conditioning programs in preparation for sport-specific performance testing. In addition, exercise requiring maximal exertion should be limited especially during hot and humid conditions. Lastly, and most importantly, educating the athletes with SCT, coaches, Athletic Trainers, and fellow healthcare professionals of exertional sickling is the best prevention strategy.^{50-52,55,56}

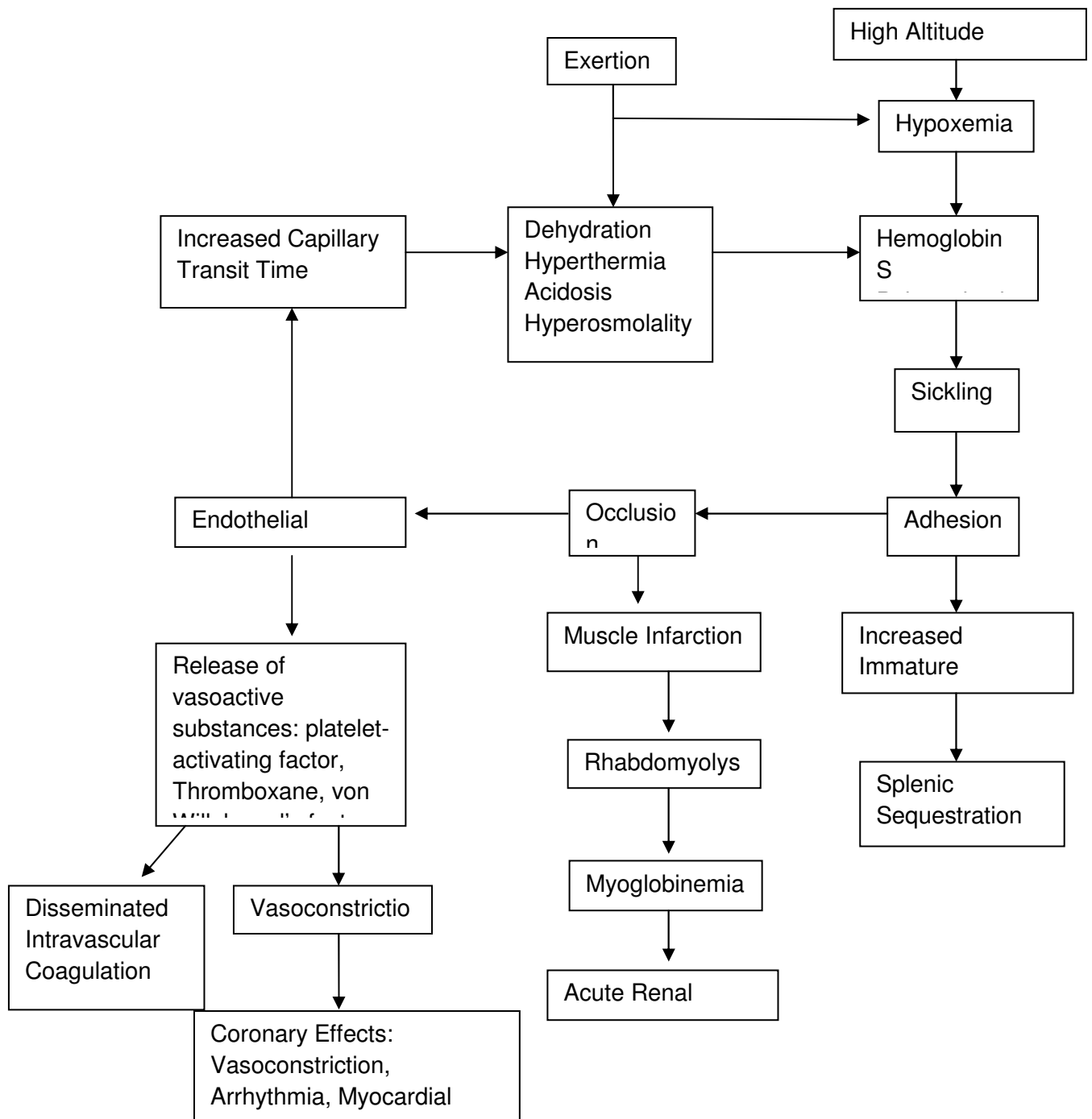


Figure 5. Pathogenesis of sickle cell trait as it relates to collapse and sudden death.^{48,52,52}

Hyponatremia

Epidemiology. Exercise-associated hyponatremia is defined as a decrease in serum sodium levels in the plasma ($<135\text{mEq/L}$) and has been reported to occur in as many as 30% of ultra-endurance participants.⁵⁷ This condition is potentially life threatening and has become the most common electrolyte disorder among athletes.⁵⁸ It was reported that from 1993-1997, 4 marathon runners died of hyponatremia.⁵⁹ A study⁶⁰ looking at runners in the Boston Marathon showed that of the 488 participants in the study, 13% had hyponatremia as defined by the serum sodium level below 135mEq/L . Of the 13% having hyponatremia, 6 of the runners had serum sodium levels below 120mEq/L and were considered to be severely hyponatremic.⁶⁰ Noakes et al⁶¹ looked at 2,135 endurance athletes and the findings of this study showed that 6% of the runners had a serum sodium concentration between $129\text{-}135\text{mEq/L}$ and 1% had a serum sodium concentration below 129mEq/L .

Etiology. It has been shown that overhydration is the primary factor leading to hyponatremia, which causes a dilutional decrease in serum sodium levels.⁶² Almond et al⁶⁰ showed a

direct	Risk Factors
relationship	Exercise duration greater than 4 hours or slow running/exercise pace
between amount	Female sex
of fluid intake	Low body weight
and the	Excessive drinking ($>1.5\text{ L/hour}$) during the event
incidence of	Pre-exercise overhydration
	Abundant availability of drinking fluids at the event
	Nonsteroidal anti-inflammatory drugs
	Other drugs associated with SIADH (SSRI's)
	Extreme hot or cold environment
	Table 9. Risk factors involved in the development of exercise-associated hyponatremia.⁵⁸

hyponatremia. The predictor measures that were used in this relationship were fluid intake greater than 3L during the race, a post-race weight that was greater than pre-race weight, as well as self-reported water loading and water intake during the race. Another study⁶³ showed that 73% of runners with severe hyponatremia had either gained weight or maintained weight throughout the race. In addition to overhydration, other risk factors associated with the development of exercise-associated hyponatremia can be seen in Table 9.

Although overhydration is a major risk factor in developing hyponatremia, hormonal and renal dysfunctions are said to be the promoting factors for the actual development and maintaining of hyponatremia.^{58,62} Defects in renal-diluting mechanisms, hormonal control of water excretion, excessive sodium losses and water intake all contribute to exercise-associated hyponatremia. Intake of hypotonic fluid in excess of sweat, urine, and insensible losses can lead to the dilution of serum sodium in the plasma and increase in total body weight as mentioned previously. Noakes et al⁶¹ showed that in 70% of the runners he looked at that had increase in body weight from excessive intake of fluids. Increased excretion of Arginine Vasopressin (AVP) has been determined as the main causative factor in the development of exercise-associated hyponatremia.⁶⁴ It has been shown that athletes presenting with hyponatremia have elevated levels of AVP.^{63,64} Elevated AVP levels in the body places the urinary diluting capability at a high osmolality level, which in turn limits the ability of the kidney to excrete water. Since the increase in urine concentration limits the ability to excrete water, the water is retained in the kidney creating a dilutional plasma level.^{58,62} Solute loss is also a contributing factor to the development of hyponatremia. Sodium losses from

sweating are variable depending of the physical fitness of the athlete and the sweat rate of the athlete. Highly fit athletes have a lower sweat sodium concentration than less fit athletes in addition to having a lower sweat volume. Serum sodium levels are increased as a direct effect of the loss of hypotonic sweat. Sweat losses are replaced with fluids which are more hypotonic than the fluid lost in sweat and if the replacing fluid is taken in excess of the sweat loss a decrease in serum sodium results.⁶²

Management. Proper recognition and diagnosis of hyponatremia is essential in order to prevent life-threatening complications. Signs and symptoms of exercise-associated hyponatremia vary depending on severity and are related to cerebral edema caused by the osmotic flow of water into the brain cells.⁵⁸ Most patients with hyponatremia are either asymptomatic or mildly symptomatic and can present with symptoms such as weakness, dizziness, headache, nausea, and/or vomiting and the resulting serum sodium levels range from 129-134mEq/L. More severe hyponatremia, serum sodium levels less than 129mEq/L, presents with signs and symptoms of seizures, coma and death.⁵⁸

Treatment of hyponatremia will vary depending on the plasma sodium level, if available, and the presented clinical status. Patients presenting with mild symptoms of hyponatremia should be treated with fluid restriction and observed until either serum sodium levels return to within normal limits or there is a resolution of symptoms and spontaneous diuresis. Administration of hypertonic saline is an option but should not be warranted unless serum sodium levels can be measured. In cases of more severe hyponatremia, administration of 3% hypertonic saline should be administered immediately due to the risk of cerebral edema that can ensue if treatment is delayed. It is

also recommended that patients presenting with hyponatremia receive supplemental in case cerebral edema leads to hypoxia.^{58,62} Figure 6 shows a flow chart of the treatment protocols that should be followed with a patient with hyponatremia and when it is necessary to transport to the hospital.

Prevention. Prevention of exercise-associated hyponatremia is focused upon educating athletes about the potential complications from overhydrating. Universal guidelines for preventing hyponatremia are not realistic because of the variation in sweat rate, sweat sodium concentration, and environmental conditions that all affect the incidence of hyponatremia.⁵⁸ The Second International Exercise-Associated Hyponatremia Consensus Development Conference recommends that athletes only drink according to thirst sensation and limit fluid intake to 400-800 ml/hr during activity.⁶⁵ In addition, The American College of Sports Medicine recommends ingestion of 500-700mg of sodium per liter of water to account for the sodium loss in sweat during endurance events.^{59 66}

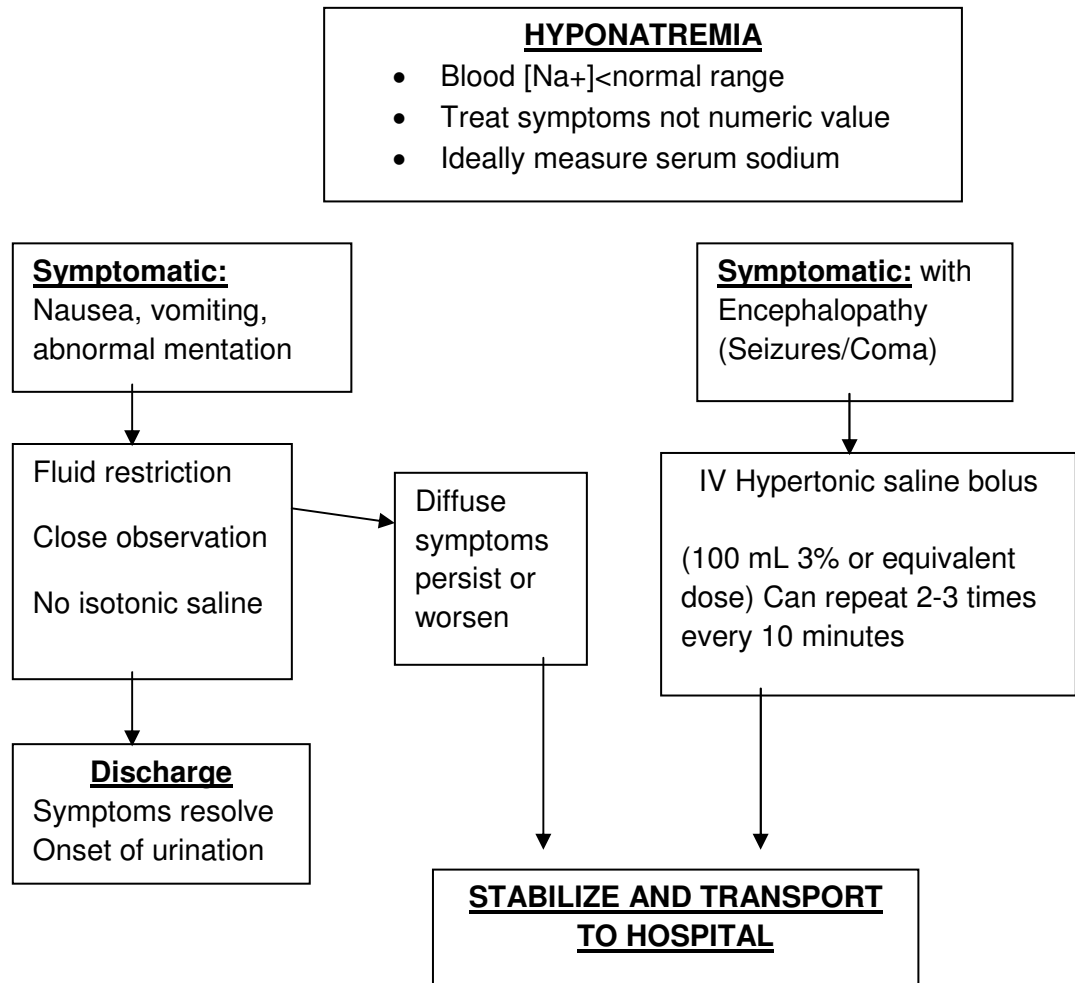


Figure 6. Treatment protocols in the treatment of a patient with exercise-associated hyponatremia.⁶²

Exercise Induced Anaphylaxis

Epidemiology. Exercise-induced Anaphylaxis (EIA) is defined as an allergic reaction that is triggered by exertion. This is a life threatening condition that creates upper respiratory distress and hypotension and can lead to death if not managed properly.⁶⁷ Shadick et al⁶⁸ found that in 78% of the subjects of the study, jogging was reported to be the trigger for an anaphylactic episode. In addition, activities that require a greater cardiovascular demand, such as aerobics, walking, dancing, tennis, bicycling, racquetball, swimming, and skiing, are more likely to trigger an attack than less intense exertional activity.^{67 69} Certain foods have been found to be predisposing factors in EIA and these are most commonly wheat, vegetables, and crustacean.⁷⁰ 54% of individuals report having an anaphylactic episode during exercise after eating certain types of foods.⁶⁷

Etiology. The pathophysiology of anaphylaxis begins when antigens react with immunoglobulin IgE which is attached to mast cells and basophils. Reaction of the antigens with IgE causes mast cells and basophils to release histamine, slow-reacting substance of anaphylaxis (SRS-

Signs and Symptoms	Frequency (%)
Pruritis	92
Urticaria	83
Angioedema	78
Excessive Cutaneous Flushing	75
Respiratory Symptoms	59
Profuse Sweating	43
Syncope	32
GI symptoms	30

Table 10. Incidence of the signs and symptoms associated with exercise-induced anaphylaxis.⁶⁷

A) and bradykinin in the body. Bronchospasm, vasodilation, and increased capillary permeability result when histamine is released in large amounts throughout the body. Like histamine, SRS-A results in bronchoconstriction as where bradykinin results in vasodilation and increased capillary permeability. It has been shown that severe adverse

effects can occur when bradykinin and histamine are released in large amounts together and have been shown to result in vascular collapse as fluid leaks into the interstitial space from the intravascular space.^{67 71} If an allergic reaction remains local, only the mast cells and basophils in the affected area are involved and can cause symptoms such as redness, swelling, and itching. If an anaphylactic reaction occurs mast cells and basophils throughout the body are involved and the extent of the symptoms are magnified and may include the patient's skin feeling hot and the patient may itch all over the body. As anaphylaxis progresses stridor, dysphagia, and dyspnea occur. The reaction then progresses to respiratory arrest brought on by bronchoconstriction and laryngeal edema. Vasodilation and increased capillary permeability causes vascular collapse will result in a large volume loss in the vasculature causing severe hypotension, decreased consciousness, and tachycardia. Death will ensue if the allergic reaction continues to progress without treatment.⁷¹

Patients suffering with EIA will present with any of the following signs and symptoms: pruritis, urticaria, angioedema, excessive cutaneous flushing, respiratory symptoms (dyspnea, wheezing, chest tightness, or hoarseness), profuse sweating, syncope, and GI symptoms. Table 10 shows the incidence (in percent) of symptoms that occur in patients with EIA. Anaphylactic attacks usually occur within the first 5 minutes of exercise but have been known to occur after the cessation of activity. Women were twice as likely as men to suffer an anaphylactic reaction and there is an increased risk of a reaction in hot and humid conditions.⁶⁷ Recognition and diagnosis of EIA can usually be made based on the patient's history. Previous history of cutaneous flushing, erythema,

pruritis is indicative of EIA. The presence of the above symptoms in addition to the patient's history further leads to a correct diagnosis of EIA.⁶⁷

Management and Prevention. Treatment of EIA is a twofold approach: both acute and prophylactic treatment. Since anaphylaxis is a medical emergency, any and all patients showing signs and symptoms of EIA should be transported to the nearest medical facility. Acutely, epinephrine has been shown to be the most consistent medication to reverse the progression of an anaphylactic reaction. In addition to administering epinephrine, maintaining an open airway and managing hypotension with fluids and medication is necessary in treatment until the patient is transported. Preventive treatment strategies are also used

to treat EIA. Exercise avoidance, discontinuation of exercise at the onset of symptoms, avoidance of corecipitating factors (ie. food) and drug therapy are most often used. Antihistamines, Cromolyn, and

Signs and Symptoms

Chest tightness (or chest pain with children)
 Coughing (especially at night)
 Prolonged shortness of breath (dyspnea)
 Difficulty sleeping
 Wheezing (especially after exercise)
 Inability to catch one's breath
 Physical activities affected by breathing difficulty
 use of accessory muscles to breathe
 Breathing difficulty upon awakening in the morning
 Breathing difficulty when exposed to certain allergens or irritants
 Exercise-induced symptoms, such as coughing or wheezing
 An athlete who is well conditioned but cannot perform at the same level as other athletes without asthma
 Family history of asthma
 Personal history of atopy, including atopic dermatitis/eczema or hay fever (allergic rhinitis)

Table 11. Signs and symptoms of exercise-induced asthma.⁷³

Ketotifen have been shown to

be effective medications that can help in preventing an anaphylactic reaction. Exercise modification has also been shown to be an effective preventive strategy in that reducing

the intensity of exercise and avoiding exercising in hot and humid conditions can further aid in the prevention of EIA.⁶⁷

Exercise Induced Asthma

Epidemiology. In a recent review,^{72,73} roughly 4,500 individuals suffer an asthma related death annually in the United States. The prevalence of asthma has increased in recent decades and studies show that those who suffer from mild forms of asthma are still at risk for a fatal asthma attack.⁷² Although asthma affects people of all ages, gender and race, including athletes, the actual rate of fatalities among athletes has been difficult to determine.⁷² Becker et al.⁷⁴ reported that during a seven year period, 61 asthma related deaths occurred during participation in sport and of those 61 deaths, 80% of the deaths occurred in individuals under the age of 21.^{72,74,75}

Asthma is a chronic inflammatory disease that causes airway obstruction, and presents with symptoms such as chest tightness, coughing (especially at night), prolonged shortness of breath (dyspnea), wheezing, and the inability to catch one's breath to name a few.^{73,75} Table 11 shows all of the signs and symptoms associated with asthma. Asthma causes a buildup of mucus in addition to the constriction of bronchial smooth muscle. Constriction of the bronchial smooth muscle results in the reduction of maximal expiratory flow rate and an increase in lung volume. The effects of an asthma attack can be deleterious to one's health. To compensate for the obstruction of the airway and the lung's inability to recoil, ventilation rate must increase causing the individual to expend more energy to breath. As the attack continues, diaphragmatic and intercostal muscle action must increase leading to further fatigue. If left untreated, eventual fatigue leads to

physical distress and can lead to death.⁷³ It has been estimated that between 10-50% of competitive athletes suffer from asthma^{75 75-77} and out of that number, 12-15% suffer from Exercise-induced asthma (EIA).^{73,78,79} Unlike those suffering from chronic asthma, those suffering from EIA only experience asthma symptoms when they exercise.

Etiology. At this time,⁷³ there are two major theories that are thought to explain the cause of EIA: the cooling/warming hypothesis and the drying hypothesis. These theories have been supported when looking at exercise in cold-weather environments and are grounded on the thought that as ventilation increases, as the case during exercise, the airway is cooled and dehydrated which leads to bronchoconstriction.^{73,77} Cool air holds less moisture and as cool air is inspired it must be humidified, thus dehydrating the airway. In addition to the aforementioned theories, environmental factors such as the presence of allergens, sulfur dioxide, nitrogen dioxide, ozone, and chlorine can induce an asthma attack in an athlete during exercise.^{73,78,80}

Diagnosis of EIA requires both presence of symptoms and evidence of airway obstruction.^{73,79} The patient

must exhibit any number of symptoms that are associated with asthma (Table 7) after the start of intense exercise; symptoms usually occur 5-8

Type of Injury	Number of Deaths	Percent
Subdural Hematoma	366	74.5
Brain Injury	32	6.5
Fracture	18	3.7
Aneurism	7	1.4
Unknown	68	13.8
Total	491	99.9

Table 12. Incidence of head-related fatalities in football from 1945-1999.⁸²

minutes after the start of intense exercise.⁷³ Secondly, the patient must show objective evidence of airway obstruction during and after exercise to be diagnosed with EIA. An athlete with EIA will experience a 10-15% drop in Forced Expiratory Volume in 1 second

(FEV₁) after a bout of intense exercise in which the athlete's heart rate is raised above 80% of their maximum value.⁷³

Management. Treatment of EIA is a twofold process: pharmacologic, nonpharmacologic, and athlete education.⁷³ The use of both long acting and short acting beta-2 agonists have been shown to be successful in the treatment of EIA. Long-term treatment, such as the use of a long acting beta-2 agonist or corticosteroid, is used to prophylactically manage asthma symptoms. These long-term interventions can manage symptoms for up to 12 hrs in duration, but due to the length of time for the medication to become active in the affected individual, long-acting treatments should not be used to treat an acute asthma attack.⁷³ Short-acting medications, such as rapid acting inhaled beta-2 agonists, act quickly to decrease bronchoconstriction and associated symptoms. To be used as a prophylactic, short-acting beta-2 agonists should be used 10-15 minutes prior to the start of exercise.⁷³ Nonpharmacologic treatment is centered on controlling asthma using various methods including nose breathing and limiting the athlete's exposure to allergens and pollutants. Also, implementing of a proper warm-up period prior to exercise can result in a refractory period up to two hours in athletes which decreases the need to use medication in some cases.⁷³

Prevention. Proper management and education is the best prevention strategy to combat asthma and the problems associated with the disease if gone undiagnosed. Educating the athlete to be aware of the signs/symptoms of asthma as well as methods for limiting exposure to possible asthma triggers will help decrease the number of attacks in an athlete.⁷³ Also, educating athletes about the proper use of medication and proper technique to administer medication is essential in the event an asthma attack does occur.

⁷³ Education among healthcare professionals about asthma is also necessary to limit the number of cases of asthma that go undiagnosed.^{73,75,78,81} Proper management and education about asthma, especially those with EIA, should reduce the number of asthma complications and lead to a higher quality of life.⁷³

Head Trauma

Epidemiology. According to the National Center for Catastrophic Sport Injury Research, there have been a total of 510 football related fatalities associated with head trauma from 1945-2004.{NCCSI, 2010}. Since 1931 there has been at least one fatality related to head trauma in football except for the year 1990 which produced zero fatalities in all of football.^{3,82} Mueller^{82 82} reported that of the 712 football fatalities between 1945-1999, 69% (n=491) were the product of head trauma. It is estimated that there are a total of 1,500,000 participants in high school football with an additional 75,000 participating at the collegiate level. Adding in an additional estimated 225,000 at the sandlot level, there were about 1,800,000 people that played football during the 2010 season. In 2010, there were 5 fatalities directly related to participation in football (2 high school, 2 collegiate, and 1 sandlot). Of the 5 direct fatalities, 4 were the result of head trauma. One fatality was due to a rebleed of a subdural hematoma, another from second impact syndrome and the last two were the result of a direct blow to the head.³

When comparing the total number of head trauma fatalities by decade from 1945, the highest number of deaths occurred during the decade of 1965-1974 and then continually decreased over the next 3 decades. The reason for the high rate of fatalities during 1965-1974 can be attributed to the tactics such as spearing, but blocking, face to

the numbers, and face to the chest hits.^{3,82} Table 3 provides a visual of total number of deaths and associated percentage of the overall number of head trauma deaths from 1945-2004. Along with the total number of head related fatalities, the incidence of head related football fatalities has decreased in both the high school and college setting. The reason for the continued decrease in the number of head related fatalities in football can be attributed to the rules and regulations that were set in place by both the NCAA and the NFHS. In 1976 rules were implemented prohibiting initial contact with the head or face.^{3,82}

Etiology. Conditions that account for fatal head injuries include subdural hematomas, brain injury (second impact syndrome), fractures, and aneurisms. Table 12 shows the type of injury and number of fatalities of each that occurred in football from 1945-1999. Most of the fatal head injuries in football occurred during the tackling process, either tackling an opponent or being tackled by an opponent. From 1945-1999 75% of the head fatalities were from subdural hematomas. If the subdural hematoma fatalities were added to the other 32 brain injury fatalities, it would result in 80% of head fatalities being due to brain injuries. Probably the most concerning issue dealing with head trauma is that the mortality and morbidity rate is approaching 50% and 100% respectively.⁸²

Traumatic brain injuries account for an estimated 225,000 concussions each year and if a second concussion is incurred before the first resolves the result can be fatal. A study⁸³ looking at the epidemiology of concussions in high school and collegiate football players found that players who sustained at least one concussion during a season was 3 times more likely to suffer a second concussion. Second impact syndrome, is a condition

that occurs when a person sustains a second brain injury before the symptoms of the first brain injury has fully cleared. This condition produces rapid brain swelling and/or herniation after the second injury is sustained and can rapidly lead to death.^{82,84} Second impact syndrome is thought to involve the loss of autoregulation of the brain's blood flow, edema, and uncontrolled intracranial hypertension. Loss of autoregulation increases intracranial pressure resulting in uncal herniation, cerebellar herniation, or both. This has been shown to occur within 2-5 minutes post second blow to the head. If a player returns to play before symptoms of the first head injury subside and receives a second blow to the head, even if the second blow is minor, the athlete may seem okay at first but then in the next few moments may collapse. Once the athlete collapses, he becomes comatose, loss of eye movement and dilation of pupils, and the appearance of respiratory failure.⁸⁴

As mentioned before, subdural hematomas account for 75% of the head related death in sport.⁸² Subdural hematomas occur after veins in the subdural space are ruptured, leading to a slow venous bleeding between the dura and parenchyma of the brain. Another complication occurs if the parenchyma is rupture in addition to the veins because ruptures of the parenchyma results in an arterial bleed.⁸⁵ The mechanism behind a subdural hematoma is an acceleration-deceleration injury and it is by this mechanism that the subdural veins are ruptured. An athlete suffering from a subdural hematoma will present with a myriad of neurological findings. The athlete may present with an altered level of consciousness, which is dependent on the degree of injury to the brain and brain stem. Athletes may also show a Cushing response (bradycardia, hypertension, and widening of pupils). Objective findings such as asymmetric pupils or motor functions are

also a good indicator of a subdural hematoma and these objective findings could be used to determine the location of the hematoma.⁸⁶

Management. Any athlete suffering from possible head trauma must undergo a thorough evaluation followed by a full neurological examination. If an athlete is downed on the field during participation due to a suspected head injury, an abbreviated but definite evaluation must be used to determine whether or not a life-threatening injury has occurred. The first step that must be done is to determine the athlete's level of consciousness. An unconscious athlete must be assumed to have a life-threatening injury and the head and neck must be stabilized along with maintaining an open airway. During this time, the most appropriate methods for transport must be decided upon that will not cause more harm to the athlete. Although loss of consciousness can be related to the seriousness of head injury, not all athletes suffering a life-threatening head injury will lose consciousness so utmost precaution should be taken when evaluating an athlete with a possible head injury. A thorough neurological evaluation can assist in determining the extent and seriousness of a suspected head injury, even in those that are unconscious or semi-conscious. As mentioned before, level of consciousness is the first step of the neurological examination. After level of consciousness is determined, assuming the athlete is conscious, an examination of cranial nerves, as well as examination of motor, sensory, and reflex functioning should be performed. The athlete's cognitive function should be evaluated next to rule out a possible concussion. If at any point during the evaluation the athlete's condition deteriorates, they present with the signs and symptoms of second impact syndrome or a subdural hematoma as mentioned above, or there is an

initial loss of consciousness, the athlete must be transported immediately to a healthcare facility to undergo additional testing.⁸⁴

If transport is indicated for a serious head injury a CT scan should be performed in addition to the neurological exam mentioned above. The CT scan is the diagnostic tool of choice because it is helpful in ruling out structural abnormalities in the brain.^{84,86} In the case of an athlete with a subdural hematoma, athletes must undergo emergency surgery if the athlete presents with neurological deficits and a positive CT scan. Emergency surgery requires a craniotomy to get rid of the hematoma. Unfortunately, those athletes with large lesions or those that arrive comatose with very little brainstem activity will not benefit from surgery. Although surgical intervention is available, the mortality rate in patients is still between 36-90%.⁸⁶

Prevention. Prevention strategies to limit the number of fatal head injuries in sport are of greatest importance. Although we may never be able to totally eliminate fatal head injuries, implementing and enforcing prevention strategies is the best way to reduce the incidence of these fatalities.⁸² Probably one of the most beneficial prevention strategies that has been implemented is the passing of a rule in 1976 prohibiting the head and face as the initial point of contact during blocking and tackling drills. The original rule stated that spearing was the “intentional use of the helmet to punish the opponent.” In 2005, the rule was rewritten and the word intentional was taken out, thus leaving the rule to read “spearing is the use of the helmet to punish the opponent.” Another rule that was implemented for the purpose of a prevention strategy was in the high school setting during the 2006-2007 season. The rule stated that it was mandatory that all helmets be fitted with a 4-point chinstrap to secure the helmet and all mouth guards must be a non-

white color. Lastly, in 2007, butt blocking, face tackling, and spearing were placed under the heading “Helmet Contact—Illegal” and was done so to place more emphasis on injury risk concerns.³ In addition to rule changes, education and proper teaching of fundamentals are important aspects in the prevention of fatal head injuries. Football coaches should teach the student athletes the proper fundamentals when teaching blocking and tackling skills. Teaching the athletes that contact should be made with the head up and never leading with the head should be done from an early age on. Educating coaches on making sure that all equipment fits properly and securely and educating them about the importance to teaching proper fundamentals can also aid in the prevention of head injuries. Also, educating athletes, parents, and coaches about the signs and symptoms of head injuries along with the dangers of recurrent head injuries should be done so all are aware of the possible dangers that can occur if left undiagnosed and untreated.⁸² If a player experiences any signs of head trauma, he or she must be removed from participation until they receive medical clearance from a physician. The National Federation and State High School Associations (NFSHSA), in 2008, stated in a concussion management recommendation, that any player sustaining a concussion should not be allowed to return to play on the same day. Coaches should never be the ones to make return-to-play decisions, especially when dealing with an athlete with a suspected head injury. Lastly, officials should be more diligent and call all illegal helmet hits. This would further push coaches to teach proper fundamentals and could potentially reduce the number of head injuries even further.³

Traumatic Injury

Epidemiology. Traumatic injury in sport, although rare, can be life-threatening if not treated immediately. Traumatic abdominal injuries are increasing in frequency and

now account for 7-10% of all sport-related injuries.⁸⁷⁻⁸⁹

While injuries to the spleen, kidney, and liver are most common in traumatic abdominal injuries, traumatic injury has also been reported to occur to the stomach, intestines, gall bladder, and diaphragm.^{90 91,92}

Etiology. Sports

related abdominal trauma

can occur from two different mechanisms; a direct blow to the abdomen or a high speed deceleration mechanism. Participants of contact sports such as

American football, soccer, rugby, and wrestling are at increased risk of abdominal trauma due to the lack of protective equipment in the abdominal region of the athletes.

Noncontact sports such as downhill skiing, water skiing, horseback riding are also at risk for severe abdominal trauma. Although initial evaluation may present as a minor injury to the abdomen, internal bleeding that can occur from abdominal trauma can be life threatening.^{90,91}

Grade	Injury	Description
I	Hematoma	Subcapsular, <10% surface area
	Laceration	Capsular tear, <1cm parenchymal depth
II	Hematoma	Subcapsular, 10-50% of surface area Intraparenchymal, <10cm in diameter
	Laceration	Laceration, 1-3cm parenchymal depth, <10cm length
III	Hematoma	Subcapsular, >50% surface area Ruptured subcapsular or parenchymal hematoma
	Laceration	Intraparenchymal hematoma more than 10cm or expanding >3cm parenchymal depth
IV	Laceration	Parenchymal disruption involving 25-75% of hepatic lobe or 1-3 Couinaud segments within a single lobe
V	Laceration	Parenchymal disruption involving >75% of hepatic lobe or >3 Couinaud segments within a single lobe
VI	Vascular	Juxtahepatic venous injuries (ie, retrohepatic cava/central major hepatic veins)
	Vascular	Hepatic avulsion

Table 13. Grading of liver injuries.⁹³

Injury to the spleen, the most commonly injured organ in sport, is the most common cause of death due to abdominal injury in sport.^{91,93} Mechanism of injury to the spleen most often occurs from a direct blow to the upper left quadrant of the abdomen, the left back or the left lower portion of the chest wall.^{90,91,93} The resulting injury to the spleen can either be a subcapsular hematoma, contusion to the spleen, or splenic rupture.⁹⁰ There is often an initial sharp pain in the upper left quadrant of the abdomen and then progresses to a dull ache centralized on the left side of the abdomen. If bleeding continues the abdominal pain will become diffuse and the patient may also complain of pain in the left shoulder (Kehr's sign). Pain in the left shoulder is referred pain from intraperitoneal blood irritating the diaphragm. Bleeding of the spleen often occurs slowly and acute distress from the injury will not present itself until weeks after the original injury. Mononucleosis is also a risk factor for splenic rupture because the infection causes distension of the organ making it more susceptible for injury from a direct blow.^{90,93}

The liver can be injured by both a deceleration mechanism and direct blow to the upper right quadrant of the abdomen.^{90,93} Laceration of the liver's thin capsule and underlying parenchyma result from a deceleration mechanism of injury to the liver. A direct blow will cause a crush injury to the liver and result in subcapsular or intraparenchymal hematoma.⁹³ The right lobe of the liver is the location of 80% of all lacerations and is thought to be because of the increased size of the right lobe of the liver and its location to the ribs.⁹⁰ The Hepatic Injury Scale is used to grade the extent of injury to the liver and are graded from I-VI. Grades I-II are considered minor, grades III-V are considered severe, and grade VI is most often fatal.⁹³ Table 13 describes each grade of hepatic injury. Original concern was that a laceration to the liver cause massive

bleeding, but in about 70% of the cases that were explored surgically the bleeding had stopped by the time surgeons entered the abdominal cavity.⁹⁰

Abdominal trauma to the kidneys, stomach, intestines, and pancreas also occurs during athletic competition. Injury to the kidneys occurs from a direct blow to the athlete's flank and injury to the stomach, intestines, and pancreas occurs via a direct blow to the abdomen.^{90,93} Injury to the kidney will cause bleeding into the renal collection system resulting in hematuria. Athletes may also experience pain in the flank from the injury sustained.⁹¹ Stomach, intestine, and pancreas trauma can result in chemical or bacterial peritonitis and also cause fever, nausea, and vomiting in the athlete.⁹³

Management. An athlete presenting with abdominal pain must undergo a thorough evaluation to rule out a possible life-threatening condition. Injury to the abdominal wall will present with immediate focal pain. An athlete suffering from an injury to the abdominal organs will present with local pain that becomes diffuse and spread to the entire abdomen as time progresses because the internal bleeding causes intraperitoneal irritation. Intraperitoneal irritation from internal bleeding will also cause abdominal guarding, rigidity, rebound tenderness, pain with laughing, coughing, and jumping or bouncing. There may also be referred pain to the left shoulder (Kehr's sign) which is indicative of diaphragm irritation or Cullen sign which is a bluish periumbilical discoloration.⁹³ Athletes with diffuse abdominal pain should be kept in a recumbent position with legs elevated (Trendelenberg sign) which assists in returning blood to the heart until transport to a medical facility becomes available.⁹¹

Splenic injury will present with tenderness over ribs 10-12. As mentioned before, initial pain will be present and then become diffuse and time progresses. With the diffusion of pain that athlete will also present with signs of shock (rapid pulse, low blood pressure, thirst, and sweating). Rebound tenderness and abdominal guarding may also be an objective finding. Suspected injury to the spleen requires immediate transport to a medical facility for further evaluation. Once admitted to the medical facility, a full blood count and CT scan must be obtained. Hemoglobin and hemocrit levels will provide an accurate measure of the amount of blood lost and an elevated leukocyte level will be present if a subcapsular hematoma has developed. The CT scan is the gold standard to diagnostic imaging of the abdomen and provides the physicians with an accurate image of the initial injury. Athletes who are hemodynamically stable are managed nonoperatively. About 60-80% of all patients with a splenic injury is managed nonoperatively and has shown a 95% success rate.⁹⁴ Athletes that are hemodynamically unstable or suffer a high-grade splenic injury will undergo an exploratory laparotomy and a splenectomy will be performed if hemorrhage is uncontrollable.^{93,94}

Athletes with a liver injury will complain of pain in the upper right quadrant. Athletes may also complain of pain over the overlying ribs and in the right shoulder. Pain may also be accompanied by nausea, vomiting, and abdominal guarding. Suspected hepatic injury requires transport to a medical facility and management of the injury depends on the athlete's level of consciousness and hemodynamic stability.⁹³ Nonoperative management of hepatic injuries has also been shown to be effective with success rates ranging from 85-98%. If the patient is hemodynamically stable, the first five grades of hepatic injury can be managed nonoperatively, but grades III-V have been

shown to have a high failure rate however. Unlike nonoperative management of splenic injuries, hepatic injuries have a high morbidity rate when managed nonoperatively because of the risk of delayed hemorrhage, hepatic abscess, biloma, and hemobilia. Athletes requiring surgical intervention will undergo a laparotomy to repair or resect the injured liver.⁹⁴

Trauma to the stomach, intestines, and pancreas also requires a thorough evaluation. As mentioned previously, an athlete should be suspected of abdominal trauma to the stomach, intestines, or pancreas if they present with persistent abdominal pain and signs of chemical or bacterial peritonitis (fever, nausea, vomiting, and referred shoulder pain). Also on evaluation, pain may be localized and the athlete may present with abdominal guarding, rebound tenderness, rigid abdomen, and loss of normal bowel sounds. Like splenic and hepatic injuries, other suspected abdominal trauma warrants immediate referral to a medical facility for further evaluation. A CT scan will be performed to better diagnose the extent of the injuries and to rule out multiple organ involvement. Unfortunately with injuries to the stomach, intestines, and pancreas, a laparotomy is required, especially if the athlete is hemodynamically unstable.⁹³

Prevention. Immediate recognition of a possible severe abdominal injury is paramount in preventing a catastrophic outcome. Due to the rarity of catastrophic abdominal trauma, medical professionals should always have an elevated suspicion when evaluating an athlete with abdominal trauma.⁹¹ Table 14 lists some of the top causes of sudden death in sport with proper treatment for each condition as well as some common mistakes made that can prove fatal in worst-case scenario. In recent years there has been advancement in protective equipment in some sports. American football for example,

gives players the opportunity to wear extra padding surround the ribs, which can assist in protecting the abdominal region of the athlete. There is also an extension of football shoulder pads that can be added that will cover the athlete's lower back where the kidneys are oriented. Unfortunately, for sports such as soccer, rugby, and wrestling protective equipment is not worn by athletes protecting the abdominal regions, so in this case proper recognition of abdominal trauma by the overseeing medical professional is the best way to prevent a catastrophic outcome.

Top Causes of Sudden Death in Sport			
Pathology	Common Scenario	Treatment	Common Mistakes
Cardiac	Basketball player suddenly collapses with no apparent contact	Activate EMS; Check ABCs; Begin CPR; use AED as soon as it becomes available	No AED; Unprompt EMS activation; No initiation of immediate CPR
Exertional Heat Stroke	Football player practicing in full pads on a hot humid summer afternoon in August	Assess core body temperature (rectal temperature); Immediate rapid cooling (cold water immersion); Monitor vitals; Cool first transport second	Not assessing core temperature; not using cold water immersion; Not initiating treatment quickly; not continuously circulating water
Asthma	Soccer player having difficulty breathing during a match	Administer rescue inhaler; Monitor vitals	No rescue inhaler; not knowing athlete has asthma
Anaphylaxis	Athlete is stung by a bee	Aid patient in administration of epi-pen; activate EMS; maintain airway	No epi-pen; No EMS activation
Head Injury	Baseball pitcher hit in head by a ball	Remove from activity; Cognitive and physical assessment; Monitor vitals; Monitor symptoms; Gradual return to play when asymptomatic	Return to play too soon; Not recognizing symptoms; Treating symptoms too lightly
Sickling	African-American player collapses while running conditioning sprints	Remove from activity; administer oxygen; hydrate; cool if necessary	Misdiagnosed with heat or cardiovascular collapse; No intervention
Diabetes	Diabetic athlete shows signs of cognitive distress, and decreased responsiveness	Assess vitals; determine hyper/hypoglycemia (blood glucose level); Monitor vitals; administer glucose;	No diabetes kit; Unaware athlete has diabetes; Confusing hyper and hypo - glycemia
Hyponatremia	Marathon runner collapses 25 yd. from finish line	Assess vitals; Check Na levels; Check core temperature; restrict fluids; administer hypertonic saline if hyponatremia is severe (Na levels must be measured)	Administer fluids;
Lightning	Lacrosse player struck by lightning	Activate EMS; Move victim to safe place; Monitor vitals; Initiate CPR; treat in a "reverse triage" strategy	Continue to play; Not utilizing flash-to-bang; Not using 30/30 rule
C-Spine	Football player tackling with head down	Stabilize and maintain neck in neutral position; Activate EMS; Monitor vitals (Remove facemask in sports requiring the use of helmet); Secure to spine board (if appropriate personal available)	Improper stabilization; excessive motion; not monitoring vitals; removal of helmet
Traumatic Injury	Soccer player getting kicked in the abdomen	Thorough abdominal evaluation (location of pain, referred pain, local or diffuse pain); EMS activation; keep player in recumbent position until EMS arrives	Not suspecting abdominal trauma (internal bleeding)

Lightning

Epidemiology. Lightning has been one of the top three causes of weather-related death over the past century accounting for an average of 100 deaths per year.⁹⁵⁻⁹⁸ The highest frequencies of lightning deaths occur in months May-September (92% of all lightning deaths) and between the hours of 10:00am and 7:00pm; these months and times coincide closely with the times of outdoor athletic activity.^{95,96} The National Oceanographic and Atmospheric Administration published data showing that 52% of the deaths associated with lightning occurred in those participating in outdoor recreation and sports activities.⁹⁵ Furthermore, data shows that fields, ballparks, and playgrounds account for 27% of the deaths whereas deaths caused by under-tree, water, golf accounted for 14%, 8%, and 5% of the deaths respectively.⁹⁵

Etiology. There are five mechanisms by which injury by lightning occur; direct strike, contact, sideflash, step voltage or ground current, and blunt injury.^{95,96} A direct strike usually strikes the person on the head (the highest point of entry) and from there the current enters the body through an orifice. A contact injury occurs when a person is in direct contact with an object that is struck by lightning. In this case, the current travels from the object to the person in contact. A side flash injury occurs when the lightning strikes an object such as a tree or building and then jumps to a person nearby. The electric current travels through a path of least resistance, so if lightning strikes a tree, for example, and a person is underneath the tree seeking shelter, the electricity will jump to travel through the person since they have the least resisted path. The fourth mechanism of

injury is a step voltage or ground current mechanism. During this mechanism, the electricity traveling through the ground radiates away from the strike point. Since humans contain a large amount of water in addition to sodium chloride, electricity flows easily through a person, so if one foot of a person is located closer to the strike point than the other a step voltage is created causing the electricity to travel through the person. Lastly, blunt injury is the last mechanism of lightning injury. A lightning strike can cause implosive and explosive forces as well as fierce muscle contractions resulting in the possibility of the person being thrown away from the strike point. ^{95,96}

Management. The current from a lightning strike is transmitted internally causing a multitude of complications. The current can cause a disruption of the electrical systems of the body such as the heart, respiratory center, and autonomic nervous system. The current from lightning causes the heart to go into asystole resulting in cardiac arrest, and although the heart's automaticity might reestablish a normal rhythm, the length of time the person is in respiratory arrest can cause the rhythm to go into refractory ventricular fibrillation or asystole. ⁹⁶ Due to the severity of complications that can arise from a lightning strike treatment should follow a "reverse triage" strategy, especially when more than one victim is present. ⁹⁵⁻⁹⁷ The thought behind this strategy is that those that appear to be conscious and breathing are already on the road to recovery and those that are unconscious with no pulse or respiration can benefit from immediate action. ⁹⁶ Initiating Cardiopulmonary Resuscitation (CPR) as soon and safely as possible is the most effective way in treating cardiac and respiratory arrest in a lightning strike victim. ^{95,96} Proper and immediate resuscitation in a person who appears to be dead might lead to recovery if acted upon immediately. ⁹⁶

Prevention. The best treatment in regards to lightning strike is prevention.⁹⁵⁻⁹⁷

The National Athletic Trainers' Association (NATA) released a position statement on lightning safety and outlined that the best prevention strategy is the implementation of a proactive lightning safety policy in institutions that have outdoor sports and recreation.⁹⁵ The NATA outlined that this policy should include the following 6 recommendations: an established chain of command to remove athletes from the field, a designated weather watcher, a means of monitoring weather forecasts and warnings, a listing of specific safe locations from the lightning hazard, the use of specific criteria for suspension and resumption of activity, and the use of recommended lightning safety strategies.⁹⁵ Having a safe location to evacuate persons is extremely important in lightning safety. Ideally, having an enclosed structure with plumbing, electricity, and telephone is the best option for a safe location. The plumbing and electrical wiring are effective in electrically grounding the structure, which diverts the current of the lightning strike. If a structure like this is not available, or no structure is present, residing in a closed car is the next best option for a safe location. The metal shell of the car guides the electric current around the passengers of the car, not through them, making the inside of a car a safe place to be during a thunderstorm. Specific criteria should be in place for the postponement and resumption of activity. The NATA recommends using the flash-bang method for determining how close the lightning strike is to the current location. In addition, using the 30-30 rule is a safe way for determining when play should resume. The thought is to wait 30 minutes after the last flash of lightning or bang of lightning is heard.⁹⁵

COACHES AND MEDICAL CARE IN THE SECONDARY SCHOOL LEVEL

Coaching Certification

The American Alliance for Health, Physical Education, Recreation and Dance (AAHPERD) recommended the implementation of coaching certification in 1968. In 1995 the National Association for Sport and Physical Education (NASPE) created standards for coaching certification and among the listed competencies is sport safety. At the state level, the requirements for coaches as it relates to sport safety range from no requirement at all to certification in CPR and First Aid.⁵ Table 15 represents each of the state's coaching education requirements and shows which states require first aid and CPR training. There are 22 states that require coaches to obtain certification in at least sports first aid and 13 states require coaches to be certified in both CPR and first aid. Nine states recommend coaches have certification in sports first aid and 4 states have no requirements for coaches dealing with sports injuries.⁹⁹ Since roughly 42% of secondary schools employ a Certified Athletic Trainer, a lot of the medical decisions fall on the shoulders of the coach.⁵

State	Content of Coaching Education Requirement
Alabama	This state stipulates coaches must complete a fundamentals of coaching course, sport first aid course, CPR training, and rules training.
Alaska	This state stipulates coaches must complete a fundamentals of coaching course, sport first aid course, and rules training.
Arizona	This state requires coaches complete a fundamentals of coaching course.
Arkansas	This state stipulates coaches must complete: 1. NFHS Fundamentals of Coaching 2. NFHS First Aid for Coaches 3. NFHS Sport-Specific course(s) for the sport(s) coached 4. AAA State Rules Test 5. CPR and AED certification
California	This state stipulates coaches must complete coaching education with the following components (a) coaching philosophy consistent with school/district/board goals, (b) sport psychology, (c) sport pedagogy, (d) sport physiology, (e) sport management, and (f) sound planning and goal setting. Coaches must complete CPR training, first aid training, and rules training.
Colorado	This state stipulates coaches must complete a fundamentals of coaching course, sport first aid or sport medicine course, and rules training.
Connecticut	This state stipulates coaches must complete coaching education with the following components: (a) legal and safety aspects of coaching children and adolescents, (b) medical aspects of coaching children and adolescents, (c) principles and practices of coaching children and adolescents, and (d) child and adolescent psychology (3 credit hours/45 semester hours). Coaches must complete CPR training and first aid training.
Delaware	This state stipulates coaches must complete CPR training. Head coaches must complete a rules training. The state recommends coaches complete a fundamentals of coaching course and sport first aid course.

Florida	This state stipulates coaches must complete a rules presentation. This state recommends coaches complete a fundamentals of coaching course and sport first aid course.
Georgia	This state stipulates coaches must complete a fundamentals of coaching course, sport first aid course, and rules training.
Hawaii	This state stipulates coaches must complete a fundamentals of coaching course, and a sport first aid course is not required, but highly recommended.
Idaho	This state stipulates head varsity coaches must complete a fundamentals of coaching course, sport first aid course, and sport skills and tactics course.
Illinois	This state stipulates coaches must complete a fundamentals of coaching course, a first aid for sport course and an online rules meeting.
Indiana	This state stipulates coaches must complete a fundamentals of coaching course.
Iowa	This stipulates coaches must complete coaching education with the following components: (a) knowledge and understand of the structure and function of the human body in relation to physical activity (1 credit hour or 10 contact hours), (b) knowledge and understanding of human growth and development of children and youth in relation to physical activity (1 credit hour or 10 contact hours), (c) knowledge and understanding of the prevention and care of athletic injuries and medical safety problems related to physical activity (2 credit hour or 20 contact hours), and (d) theory of coaching which must include knowledge and understanding of professional ethics and legal responsibilities of coaches (1 credit hour or 15 contact hours; required for initial authorization). This state recommends coaches complete CPR training and rules training.
Kansas	This state stipulates coaches must complete a fundamentals of coaching course and sport first aid course.
Kentucky	This state stipulates that head coaches must complete the Fundamentals of Coaching course, KMA/KHSAA Sport Safety Course, attend a medical symposium, CPR/AED training, and rules training. This state requires assistant coaches to complete the Fundamentals of Coaching course, KMA/KHSAA Sport Safety Course, and CPR/AED training.
Louisiana	This state stipulates coaches must complete a state Activities Association determined coaching education curriculum.
Maine	This state stipulates coaches must complete a coaching eligibility course, sport first aid course, and CPR/AED training.
Maryland	This state stipulates coaches must complete (or be enrolled in) a prevention and care of athletic injuries course (1 credit hour). This state recommends coaches complete a fundamentals of coaching course and sport first aid course.
Massachusetts	This state stipulates coaches must complete a fundamentals of coaching course, sport first aid course, and sport specific skills and technical skills course.
Michigan	This state recommends coaches complete a state Activities Association determine coaching education curriculum (i.e., 18 unit, 6 level Coaches Advancement Program).
Minnesota	This state stipulates head coaches must complete a fundamentals of coaching course, sport first aid course, and care and prevention of athletic injuries course (6 quarter hours or 60 hours).
Mississippi	This state stipulates cheerleading coaches/sponsors must attend annual state Activities Association clinics (reviews rules & safety knowledge and awareness). This state recommends coaches complete a fundamentals of coaching course, sport first aid course, CPR training, and rules training.
Missouri	This state stipulates non-faculty coaches must complete the NFHS Fundamentals of Coaching Course, Sport First Aid Course, and the MSHSAA By-Laws exam.
Montana	This state stipulates coaches must complete a state Activities Association determined coaching education curriculum.
Nebraska	This state recommends coaches complete a fundamentals of coaching course and sport first aid course.
Nevada	This state stipulates coaches must complete a fundamentals of coaching course, sport first aid course, and CPR training.
New Hampshire	This state stipulates coaches must complete a fundamentals of coaching course, sport first aid course, and CPR training. All varsity head coaches, or athletic director's designee, must attend rules training. Cheerleading coaches/sponsors are required to attend a safety clinic (includes a rules review).

New Jersey	This state stipulates coaches must complete a fundamentals of coaching course, sport first aid course, and CPR training.
New Mexico	The NMAA Coaches Training Program – Level 1 must include the NASPE National Standards for Sport Coaches, 2006.
New York	This state stipulates coaches must completed coaching education with the following components: (a) philosophy, principles, and organization of athletics, (b) health sciences applied to coaching, and (c) theory and techniques of coaching (sport specific). Coaches must complete a skills requirement, sport first aid course, and CPR training.
North Carolina	This state recommends coaches complete a fundamentals of coaching course, sport first aid course, and rules training.
North Dakota	This state recommends coaches to complete a fundamentals of coaching course and sport first aid course.
Ohio	This state stipulates coaches must have (a) the ability to work effectively with athletes, (b) knowledge of activity, and (c) knowledge of health and safety aspects of the activity. Coaches must complete a sport first aid course and CPR training.
Oklahoma	This state stipulates coaches must complete a care and prevention of athletic injuries training and first aid training. This state recommends coaches complete a rules training.
Oregon	This state stipulates coaches must complete a fundamentals of coaching course and a sport first aid course.
Pennsylvania	This state recommends coaches complete a fundamentals of coaching course and a sport first aid course.
Rhode Island	The RI Interscholastic League stipulates coaches must complete a fundamentals of coaching course, sport first aid course, and rules training.
South Carolina	This state recommends coaches complete a fundamentals of coaching course and sport first aid course.
South Dakota	This state stipulates coaches must complete a fundamentals of coaching course and sport first aid course.
Tennessee	This state stipulates coaches must complete a fundamentals of coaching course, sport first aid course, and rules training
Texas	This state stipulates coaches must complete a fundamentals of coaching course and sport first aid course.
Utah	This state stipulates coaches must complete a fundamentals of coaching course, sport first aid course, and CPR training.
Vermont	This state stipulates coaches must complete a fundamentals of coaching course and sport first aid course. Cheerleading coaches/sponsors are required to attend a safety seminar.
Virginia	The VHSL requires coaching education content consistent with meeting the 40 standards established by the National Standards for Sport Coaches, NASPE, 2006.
Washington	This state stipulates coaches must complete coaching education with the following components: (a) medical aspects, (b) legal aspects, (c) psychological/social foundations, (d) coaching technique, and (e) philosophy/sport management/pedagogy). Coaches must complete CPR training and first aid training. High school head coaches are required (middle school head coaches are recommended) to attend a rules training or pass the state Activities Association Officials' test.
West Virginia	This state stipulates head varsity and freshman team coaches must attend a rules training. This state recommends coaches attend a fundamental of coaching course and a sport first aid course.
Wisconsin	This state stipulates coaches must complete a fundamentals of coaching course and a sport first aid course.
Wyoming	This state requires coaches complete a fundamentals of coaching course and sport first aid course.

Table 15. State requirements for coach education.

Legislation and Coaches

Recently, legislation has been enacted in many states regarding traumatic brain injuries. In 2009 and 2010 seventeen states passed legislation regarding traumatic brain injuries with 11 states including Connecticut, New Jersey, and Washington implementing laws that target youth sport-related head injuries. At least 25 states have introduced legislation in 2011 regarding traumatic brain injuries and a total of 37 states have introduced legislation aimed specifically at youth sport participants. Figure 7 shows the states that have enacted legislation and the states that have filed legislation in 2011. The legislation for each state varies but the main focus is the development of guidelines to prevent and manage head injuries in sport. The law passed in Connecticut in 2010 mandates that all coaches undergo annual training and review regarding concussions and head injuries before the state will issue a coaching permit. The coach must then undergo a refresher course every five years to maintain the coaches' permit. In addition to the coaches' training, any coach that suspects an athlete has a head injury must remove the athlete from participation immediately and cannot return until they are medically cleared by a licensed medical professional (physician, physician assistant, nurse practitioner, or athletic

trainer).¹⁰⁰

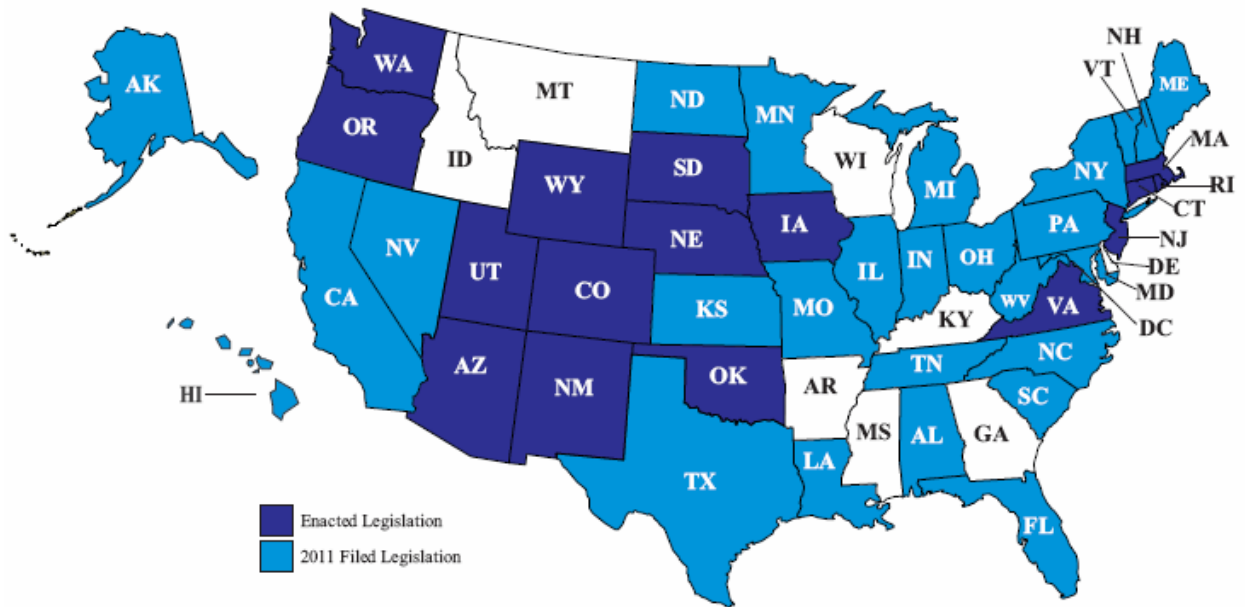


Figure 7. States with Enacted and 2011 Filed Legislation Targeting Youth Sports-Related Concussions.¹⁰⁰

Coaches Knowledge of First Aid Skills

Various studies^{5,101-106} have looked at coaches and their basic knowledge of first aid. The percentage of coaches that passed a basic first aid examination ranged from 5-36%. Ransone et al¹⁰² reported that only 36% of the respondents of their study achieved a passing score on the first aid examination. In addition, they also looked at a game situational scenario in which they had to make a decision about returning a player back to activity when losing a close game. 75% of the respondents indicated that they would return the athlete back to competition even though it was deemed an inappropriate decision. Also, the coaches who had passed the first aid test tended to return the athlete back to competition over coaches who did not pass the first aid examination.¹⁰² In addition to the study by Ransone et al, Rowe and Robertson performed a study looking at high school coaches in Alabama. They administered the Athletic Injury portion of the

Modified Inventory of Recent Knowledge in Physical Education to the coaches and only 27% of the respondents achieved a passing score of 70%. Rowe and Miller administered the same test to high school coaches in Georgia resulting in similar scores to the coaches in Alabama; 38% of the coaches in Georgia achieved a passing score of 70% or better.⁵ These studies show the lack of first aid knowledge that coaches possess and their inability to make appropriate medical decisions when faced with an injury in a game situation.

In addition to the lack of first aid knowledge in coaches, research shows that they are not confident in handling emergency situations when confronted with one. Studies by Redfearn, Stapleton, Tomlinson, Shepard, and Coon looked at coaches and their capability of them handling an emergency situation. The study by Redfearn required coaches to fill out a questionnaire and the results showed that 2.7% had Emergency Medical Training (EMT), 30% had CPR training, and 55% had first aid training. Of those responding, 56% responded that they were incapable of dealing with an emergency situation if one were to arise because they lacked the appropriate knowledge in handling an emergency situation. In the other aforementioned studies, 37% of the coaches surveyed were CPR certified and 50% were currently certified in first aid. Of those respondents, 57% reported that they were uncomfortable in administering first aid for reasons of lack of knowledge and training.⁵ This evidence further proves that coaches are incapable of handling emergency situations in sport because they lack the appropriate knowledge to care for the injured athlete.

Appropriate Medical Care in the Secondary School Setting

The American Medical Association (AMA) Council of Scientific Affairs presented a report to the AMA House of Delegates in regards to supporting efforts to place Certified Athletic Trainers in all High Schools. Evidence shows that roughly 1.3 million high school athletes are injured each year with 70-75% of these injuries being considered minor and 60% of the injuries occurring during practices.^{107,108} The severity of injuries is broken down into amount of time lost and of the yearly injuries, roughly 52% result in less than 1 week lost, 30.3% in 1-3 weeks lost, 6.8% in 3 or more weeks lost, and 10.4% result in the end of the athlete's season or career.¹⁰⁸ Proper medical supervision to allow for prompt recognition, evaluation, immediate care, and rehabilitation of athletic injuries in high schools can reduce long-term effects and speed up recovery time from injury. The AMA recommends that the Board of Education and Department of Public Health in each state establish an Athletic Medicine Unit at every school that sponsors a sports program. It is also recommended that in the case of a high school not having the resources to support an Athletic Medicine Unit, school administrators and athletic directors should require all coaches to be appropriately trained in emergency first aid and basic life support (CPR).¹⁰⁷

Proper medical support in the high school setting is essential in reducing the number and severity of injuries. Schools that do not implement Athletic Medicine Units in their school force the coaches to assume the responsibility of handling and caring for athletic injuries. In doing so, the school puts itself at a liability in the case of a student athlete receiving improper care of an athletic injury from a coach who has not been appropriately trained in emergency handling procedures. Schools can reduce the risk of liability by establishing a comprehensive risk management plan which focuses on

equipment maintenance, emergency action plans that are updated and practiced regularly, facility construction, and qualified personnel available to treat athletic injuries. Athletic Trainers are a vital part of the comprehensive risk management plan because they are trained to recognize, treat, and prevent athletic injuries in sport.¹⁰⁷

1. Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: Analysis of 1866 deaths in the united states, 1980-2006.

Circulation. 2009;119(8):1085-1092.

2. Maron BJ. Hypertrophic cardiomyopathy and other causes of sudden cardiac death in young competitive athletes, with considerations for preparticipation screening and criteria for disqualification. *Cardiol Clin*. 2007;25(3):399-414, vi.

3. Mueller FO. Annual survey of football injury research 1931 - 2010. Web site.

<http://www.unc.edu/depts/nccsi/2010FBAnnual.pdf>. Updated 2011. Accessed 3/15/2011, 2011.

4. National Federation of State High School Associations. 2009-10 high school athletics participation survey. <http://www.nfhs.org/content.aspx?id=3282>. Updated 2011.

Accessed 4/15/2011, 2011.

5. Kujawa R, Coker CA. An examination of the influence of coaching certification and the presence of an athletic trainer on the extent of sport safety knowledge of coaches. . 2000;15:14.

6. Basavarajaiah S, Shah A, Sharma S. Sudden cardiac death in young athletes. *Heart*. 2007;93(3):287-289.
7. Basavarajaiah S, Wilson M, Whyte G, Shah A, McKenna W, Sharma S. Prevalence of hypertrophic cardiomyopathy in highly trained athletes: Relevance to pre-participation screening. *J Am Coll Cardiol*. 2008;51(10):1033-1039.
8. Maron BJ, Shirani J, Poliac LC, Mathenge R, Roberts WC, Mueller FO. Sudden death in young competitive athletes. clinical, demographic, and pathological profiles. *JAMA*. 1996;276(3):199-204.
9. Maron BJ. Sudden death in young athletes. *N Engl J Med*. 2003;349(11):1064-1075.
10. Link MS, Mark Estes NA, 3rd. Sudden cardiac death in athletes. *Prog Cardiovasc Dis*. 2008;51(1):44-57.
11. Maron BJ. The young competitive athlete with cardiovascular abnormalities: Causes of sudden death, detection by preparticipation screening, and standards for disqualification. *Card Electrophysiol Rev*. 2002;6(1-2):100-103.
12. Harmon KG, Asif IM, Klossner D, Drezner JA. Incidence of sudden cardiac death in national collegiate athletic association athletes. *Circulation*. 2011.
13. Mason PK, Mounsey JP. Common issues in sports cardiology. *Clin Sports Med*. 2005;24(3):463-76, vii.

14. Ng B, Maginot KR. Sudden cardiac death in young athletes: Trying to find the needle in the haystack. *WMJ*. 2007;106(6):335-342.
15. Borjesson M, Pelliccia A. Incidence and aetiology of sudden cardiac death in young athletes: An international perspective. *Br J Sports Med*. 2009;43(9):644-648.
16. Seggewiss H, Blank C, Pfeiffer B, Rigopoulos A. Hypertrophic cardiomyopathy as a cause of sudden death. *Herz*. 2009;34(4):305-314.
17. Pigozzi F, Rizzo M. Sudden death in competitive athletes. *Clin Sports Med*. 2008;27(1):153-81, ix.
18. Montagnana M, Lippi G, Franchini M, Banfi G, Guidi GC. Sudden cardiac death in young athletes. *Intern Med*. 2008;47(15):1373-1378.
19. Drezner JA, Courson RW, Roberts WO, Mosesso VN, Link MS, Maron BJ. Inter-association task force recommendations on emergency preparedness and management of sudden cardiac arrest in high school and college athletic programs: A consensus statement. *J Athl Train*. 2007;42(1):143-158.
20. Maron BJ. Hypertrophic cardiomyopathy: A systematic review. *JAMA*. 2002;287(10):1308-1320.
21. Maron BJ, Thompson PD, Ackerman MJ, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: A scientific statement from the american heart association council

on nutrition, physical activity, and metabolism: Endorsed by the american college of cardiology foundation. *Circulation*. 2007;115(12):1643-1455.

22. Mueller FO, Cantu RC. Catastrophic sports injury research twenty-seventh annual report fall 1982 - SPRING 2009. Web site.

<http://www.unc.edu/depts/nccsi/2009ALLSPORT.pdf>. Updated 2009. Accessed 3/15/2011, 2011.

23. Gill SS, Boden BP. The epidemiology of catastrophic spine injuries in high school and college football. *Sports Med Arthrosc*. 2008;16(1):2-6.

24. Swartz EE, Boden BP, Courson RW, et al. National athletic trainers' association position statement: Acute management of the cervical spine-injured athlete. *J Athl Train*. 2009;44(3):306-331.

25. Boden BP, Prior C. Catastrophic spine injuries in sports. *Curr Sports Med Rep*. 2005;4(1):45-49.

26. Boden BP. Direct catastrophic injury in sports. *J Am Acad Orthop Surg*. 2005;13(7):445-454.

27. Boden BP, Tacchetti RL, Cantu RC, Knowles SB, Mueller FO. Catastrophic cervical spine injuries in high school and college football players. *Am J Sports Med*. 2006;34(8):1223-1232.

28. Heck JF, Clarke KS, Peterson TR, Torg JS, Weis MP. National athletic trainers' association position statement: Head-down contact and spearing in tackle football. *J Athl Train*. 2004;39(1):101-111.
29. Swartz EE, Boden BP, Courson RW, et al. National athletic trainers' association position statement: Acute management of the cervical spine-injured athlete. *J Athl Train*. 2009;44(3):306-331.
30. Lawrence DW, Stewart GW, Christy DM, Gibbs LI, Ouellette M. High school football-related cervical spinal cord injuries in louisiana: The athlete's perspective. *J La State Med Soc*. 1997;149(1):27-31.
31. Torg JS, Quedenfeld TC, Burstein A, Spealman A, Nichols C,3rd. National football head and neck injury registry: Report on cervical quadriplegia, 1971 to 1975. *Am J Sports Med*. 1979;7(2):127-132.
32. Torg JS, Quedenfeld TC, Moyer RA, Truex R,Jr, Spealman AD, Nichols CE,3rd. Severe and catastrophic neck injuries resulting from tackle football. *Del Med J*. 1977;49(5):267-8,271-3,275.
33. Holly LT, Kelly DF, Counelis GJ, Blinman T, McArthur DL, Cryer HG. Cervical spine trauma associated with moderate and severe head injury: Incidence, risk factors, and injury characteristics. *J Neurosurg*. 2002;96(3 Suppl):285-291.
34. Yard EE, Gilchrist J, Haileyesus T, et al. Heat illness among high school athletes--united states, 2005-2009. *J Safety Res*. 2010;41(6):471-474.

35. American College of Sports Medicine, Armstrong LE, Casa DJ, et al. American college of sports medicine position stand. exertional heat illness during training and competition. *Med Sci Sports Exerc.* 2007;39(3):556-572.
36. Binkley HM, Beckett J, Casa DJ, Kleiner DM, Plummer PE. National athletic trainers' association position statement: Exertional heat illnesses. *J Athl Train.* 2002;37(3):329-343.
37. Casa DJ, Becker SM, Ganio MS, et al. Validity of devices that assess body temperature during outdoor exercise in the heat. *J Athl Train.* 2007;42(3):333-342.
38. Ganio MS, Brown CM, Casa DJ, et al. Validity and reliability of devices that assess body temperature during indoor exercise in the heat. *J Athl Train.* 2009;44(2):124-135.
39. Gagnon D, Lemire BB, Jay O, Kenny GP. Aural canal, esophageal, and rectal temperatures during exertional heat stress and the subsequent recovery period. *J Athl Train.* 2010;45(2):157-163.
40. Casa DJ, Anderson JM, Armstrong LE, Maresh CM. Survival strategy: Acute treatment of exertional heat stroke. *J Strength Cond Res.* 2006;20(3):462.
41. Casa DJ, Armstrong LE, Ganio MS, Yeargin SW. Exertional heat stroke in competitive athletes. *Curr Sports Med Rep.* 2005;4(6):309-317.
42. Casa DJ, McDermott BP, Lee EC, Yeargin SW, Armstrong LE, Maresh CM. Cold water immersion: The gold standard for exertional heatstroke treatment. *Exerc Sport Sci Rev.* 2007;35(3):141-149.

43. McDermott BP, Casa DJ, Ganio MS, et al. Acute whole-body cooling for exercise-induced hyperthermia: A systematic review. *J Athl Train*. 2009;44(1):84-93.
44. Korey Stringer Institute. Myths about exertional heat stroke.
<http://ksi.uconn.edu/info/myths.html>. Updated 2011. Accessed 4/15/2011, 2011.
45. Casa DJ, Csillan D, Inter-Association Task Force for Preseason Secondary School Athletics Participants, et al. Preseason heat-acclimatization guidelines for secondary school athletics. *J Athl Train*. 2009;44(3):332-333.
46. Key NS, Derebail VK. Sickle-cell trait: Novel clinical significance. *Hematology Am Soc Hematol Educ Program*. 2010;2010:418-422.
47. Scheinin L, Wetli CV. Sudden death and sickle cell trait: Medicolegal considerations and implications. *Am J Forensic Med Pathol*. 2009;30(2):204-208.
48. Kerle KK, Runkle GP. Sickle cell trait and sudden death in athletes. *JAMA*. 1996;276(18):1472.
49. Browne RJ. Sickle cell trait and sudden death. *Sports Med*. 1994;18(6):373-374.
50. Mitchell BL. Sickle cell trait and sudden death--bringing it home. *J Natl Med Assoc*. 2007;99(3):300-305.
51. Pearson HA. Sickle cell trait and competitive athletics: Is there a risk? *Pediatrics*. 1989;83(4):613-614.

52. Kerle KK, Nishimura KD. Exertional collapse and sudden death associated with sickle cell trait. *Mil Med.* 1996;161(12):766-767.
53. Kark JA, Posey DM, Schumacher HR, Ruehle CJ. Sickle-cell trait as a risk factor for sudden death in physical training. *N Engl J Med.* 1987;317(13):781-787.
54. Makaryus JN, Catanzaro JN, Katona KC. Exertional rhabdomyolysis and renal failure in patients with sickle cell trait: Is it time to change our approach? *Hematology.* 2007;12(4):349-352.
55. Eichner ER. Pearls and pitfalls: Exertional sickling. *Curr Sports Med Rep.* 2010;9(1):3-4.
56. Anderson s, Eichner ER. Consensus statement: Sickle cell trait and the athlete. . 2010.
57. Toy BJ. The incidence of hyponatremia in prolonged exercise activity. *J Athl Train.* 1992;27(2):116-118.
58. Rosner MH. Exercise-associated hyponatremia. *Semin Nephrol.* 2009;29(3):271-281.
59. Rosner MH, Kirven J. Exercise-associated hyponatremia. *Clin J Am Soc Nephrol.* 2007;2(1):151-161.
60. Almond CS, Shin AY, Fortescue EB, et al. Hyponatremia among runners in the boston marathon. *N Engl J Med.* 2005;352(15):1550-1556.

61. Noakes TD, Sharwood K, Speedy D, et al. Three independent biological mechanisms cause exercise-associated hyponatremia: Evidence from 2,135 weighed competitive athletic performances. *Proc Natl Acad Sci U S A*. 2005;102(51):18550-18555.
62. Rosner MH. Exercise-associated hyponatremia. *Phys Sportsmed*. 2008;36(1):55-61.
63. Speedy DB, Noakes TD, Rogers IR, et al. Hyponatremia in ultradistance triathletes. *Med Sci Sports Exerc*. 1999;31(6):809-815.
64. Siegel AJ, Verbalis JG, Clement S, et al. Hyponatremia in marathon runners due to inappropriate arginine vasopressin secretion. *Am J Med*. 2007;120(5):461.e11-461.e17.
65. Hew-Butler T, Ayus JC, Kipps C, et al. Statement of the second international exercise-associated hyponatremia consensus development conference, new zealand, 2007. *Clin J Sport Med*. 2008;18(2):111-121.
66. Convertino VA, Armstrong LE, Coyle EF, et al. American college of sports medicine position stand. exercise and fluid replacement. *Med Sci Sports Exerc*. 1996;28(1):i-vii.
67. Nichols AW. Exercise-induced anaphylaxis and urticaria. *Clin Sports Med*. 1992;11(2):303-312.
68. Shadick NA, Liang MH, Partridge AJ, et al. The natural history of exercise-induced anaphylaxis: Survey results from a 10-year follow-up study. *J Allergy Clin Immunol*. 1999;104(1):123-127.

69. John M. Exercise induced anaphylaxis: One more cause for syncope. *W V Med J*. 1994;90(12):518.
70. Gani F, Selvaggi L, Roagna D. Exercise-induced anaphylaxis]. *Recenti Prog Med*. 2008;99(7-8):395-400.
71. Harmon AL, Harmon DC. Anaphylaxis sudden death anytime. *Nursing*. 1980;10(10):40-43.
72. Lang DM. Asthma deaths and the athlete. *Clin Rev Allergy Immunol*. 2005;29(2):125-129.
73. Miller MG, Weiler JM, Baker R, Collins J, D'Alonzo G. National athletic trainers' association position statement: Management of asthma in athletes. *J Athl Train*. 2005;40(3):224-245.
74. Becker JM, Rogers J, Rossini G, Mirchandani H, D'Alonzo GE, Jr. Asthma deaths during sports: Report of a 7-year experience. *J Allergy Clin Immunol*. 2004;113(2):264-267.
75. LaBella CR, Sanders DB, Sullivan C. Athletic trainers' experience and comfort with evaluation and management of asthma: A pilot study. *J Asthma*. 2009;46(1):16-20.
76. Rundell KW, Im J, Mayers LB, Wilber RL, Szmedra L, Schmitz HR. Self-reported symptoms and exercise-induced asthma in the elite athlete. *Med Sci Sports Exerc*. 2001;33(2):208-213.

77. Rundell KW, Jenkinson DM. Exercise-induced bronchospasm in the elite athlete. *Sports Med.* 2002;32(9):583-600.
78. Mellion MB, Kobayashi RH. Exercise-induced asthma. *Am Fam Physician.* 1992;45(6):2671-2677.
79. Weiler JM. Exercise-induced asthma: A practical guide to definitions, diagnosis, prevalence, and treatment. *Allergy Asthma Proc.* 1996;17(6):315-325.
80. Hough DO, Dec KL. Exercise-induced asthma and anaphylaxis. *Sports Med.* 1994;18(3):162-172.
81. Kobayashi RH, Mellion MB, Kobayashi AL. What is the current status of management of the patient with exercise-induced asthma? *Nebr Med J.* 1994;79(7):189-194.
82. Mueller FO. Catastrophic head injuries in high school and collegiate sports. *J Athl Train.* 2001;36(3):312-315.
83. Guskiewicz KM, Weaver NL, Padua DA, Garrett WE, Jr. Epidemiology of concussion in collegiate and high school football players. *Am J Sports Med.* 2000;28(5):643-650.
84. Bailes JE, Cantu RC. Head injury in athletes. *Neurosurgery.* 2001;48(1):26-45; discussion 45-6.

85. Logan SM, Bell GW, Leonard JC. Acute subdural hematoma in a high school football player after 2 unreported episodes of head trauma: A case report. *J Athl Train*. 2001;36(4):433-436.
86. Stieg PE, Kase CS. Intracranial hemorrhage: Diagnosis and emergency management. *Neurol Clin*. 1998;16(2):373-390.
87. Bergqvist D, Hedelin H, Karlsson G, Lindblad B, Matzsch T. Patients with abdominal trauma and fatal outcome: Analysis of a 30-year series. *J Trauma*. 1983;23(6):499-502.
88. Bergqvist D, Hedelin H, Karlsson G, Lindblad B, Matzsch T. Abdominal injury from sporting activities. *Br J Sports Med*. 1982;16(2):76-79.
89. Bergqvist D, Hedelin H, Karlsson G, Lindblad B, Matzsch T. Abdominal trauma during thirty years: Analysis of a large case series. *Injury*. 1981;13(2):93-99.
90. Ray R, Lemire JE. Liver laceration in an intercollegiate football player. *J Athl Train*. 1995;30(4):324-326.
91. Diamond DL. Sports-related abdominal trauma. *Clin Sports Med*. 1989;8(1):91-99.
92. WILSON DH. Incidence, aetiology, diagnosis, and prognosis of closed abdominal injuries. A study of 265 consecutive cases. *Br J Surg*. 1963;50:381-389.
93. Rifat SF, Gilvydis RP. Blunt abdominal trauma in sports. *Curr Sports Med Rep*. 2003;2(2):93-97.

94. Schroepfel TJ, Croce MA. Diagnosis and management of blunt abdominal solid organ injury. *Curr Opin Crit Care*. 2007;13(4):399-404.
95. Walsh KM, Bennett B, Cooper MA, Holle RL, Kithil R, Lopez RE. National athletic trainers' association position statement: Lightning safety for athletics and recreation. *J Athl Train*. 2000;35(4):471-477.
96. O'Keefe Gatewood M, Zane RD. Lightning injuries. *Emerg Med Clin North Am*. 2004;22(2):369-403.
97. Edlich RF, Farinholt HM, Winters KL, Britt LD, Long WB, 3rd. Modern concepts of treatment and prevention of lightning injuries. *J Long Term Eff Med Implants*. 2005;15(2):185-196.
98. Cooper MA. Emergent care of lightning and electrical injuries. *Semin Neurol*. 1995;15(3):268-278.
99. National Federation of State High School Associations. State requirements. <http://www.nfhslearn.com/StatePricingRegs.aspx>. Updated 2010. Accessed 4/15/2011, 2011.
100. National Conference of State Legislatures. Traumatic brain injury legislation. <http://www.ncsl.org/?tabid=18687>. Updated 2011. Accessed April 15, 2011, 2011.
101. Dunn, L.R. Assessment of first aid knowledge and decision-making of high school coaches. . 1996.

102. Ransone J, Dunn-Bennett LR. Assessment of first-aid knowledge and decision making of high school athletic coaches. *J Athl Train*. 1999;34(3):267-271.
103. Barron M,J. The assessment of first aid and injury prevention knowledge and the decision making of youth basketball, soccer, and football coaches. . 2004.
104. Barron MJ1emme, Powell JW2, Ewing ME2, Nogle SE2, Branta CF2. First aid and injury prevention knowledge of youth basketball, football, and soccer coaches. . 2009;3(1):55.
105. Rowe PJ, Miller LK. Treating high school sports injuries: Are coaches/trainers competent? . 1991;62(1):49.
106. Valovich-McLeod TC1, McGaugh JW2, Boquiren ML3, Bay RC4. Youth sports coaches do not have adequate knowledge regarding first- aid and injury prevention. . 2008;23:130.
107. Lyznicki JM, Riggs JA, Champion HC. Certified athletic trainers in secondary schools: Report of the council on scientific affairs, american medical association. *J Athl Train*. 1999;34(3):272-276.
108. Rechel JA, Yard EE, Comstock RD. An epidemiologic comparison of high school sports injuries sustained in practice and competition. *J Athl Train*. 2008;43(2):197-204.

Introduction:

Sudden death in sport continues to be a concern for the secondary school athlete and athletic trainer (AT), as evident by the number of deaths reported this past pre-season at the secondary school level.¹ Cardiac conditions, exertional sickling, and exertional heat stroke (EHS) were most commonly reported as the cause of death. Data regarding sudden death indicates the most common causes, in order, include cardiac death, traumatic head injuries, EHS, exertional sickling, and hyponatremia.^{2,3} Advancements have been made to help reduce and prevent sudden death in sport including screening instruments, rule changes, and guidelines for participation and activity modifications.

Sudden cardiac death accounts for 78% of the sudden deaths in sport per year.⁴⁻⁷ Rule changes in the mid 1970's have helped decrease the number of deaths related to head and cervical spine injuries by making it illegal to use the helmet as the first point of contact (spearing) in sports such as football.³ Death from EHS, exertional sickling, and hyponatremia, are completely preventable as long as proper precautions are taken during training and conditioning. From July 21-August 15, 2011, 17 deaths occurred during participation in sport and physical activity. Of those 17, 7 have been either confirmed or speculated to be the result of EHS.¹ There have been 13 deaths from EHS in the past two years alone, which is on pace to surpass the number of EHS deaths from the 5 year block from 2005-2009 that recorded 18 EHS deaths.³ These conditions can occur regardless of the time of year, but often spike during the pre-season conditioning months. Proper precautions include appropriate management and treatment of an athlete that is suspected of having one of the above-mentioned conditions, which most certainly involves having trained medical personnel, such as an AT available onsite for example. Colleges and

universities provide medical services to their athletes, however the secondary school-aged athlete does not necessarily benefit from consistency in medical care, especially with an on-site AT. When looking at appropriate medical coverage at the secondary school level, less than 45% of high schools in the United States employ an AT,⁸ potentially leaving the care in the hands of the coach, parent, or bystander.

Lack of proper medical coverage has played a role in some of the recent causes of sudden death in sport. In August 2010, Tyler Davenport, a junior football player from Arkansas collapsed during practice after suffering an EHS. He later died due to complications resulting from the condition. Unfortunately, like other secondary school aged athletes who have died from EHS, Tyler was not cooled immediately following the onset of symptoms. This was in part because an AT was not present and immediate cooling via cold-water immersion was not initiated by the coaching staff prior to the arrival of emergency response personnel.⁹ Another case highlighting the role of the coach in preventing sudden death in sport involved Max Gilpin, who like Tyler, died of an EHS during football practice. Medical reports state that, on the day of Max's death, the head football coach, had the team run condition drills in full gear without water breaks for 45 minutes. At the time of Max's EHS, there was no medical staff present to monitor practice, diagnosis his condition, or implement appropriate treatment. Max's case was the first in United States history to see a coach be prosecuted for his role in a player's death.¹⁰

Despite the recommendations of the National Athletic Trainer Association (NATA) regarding appropriate medical coverage for the secondary school¹¹ many schools fail to provide medical coverage. In lieu of having an AT employed at every

secondary school, some states have opted to implement policies placing the care in the hands of the coach;¹² as is the case in the state of Kentucky. This policy change was due to the outcome of the Max Gilpin case, which requires coaches to receive advanced sports medicine training to help prevent the occurrence of sudden death.¹² Currently, there are no universal regulations regarding coaching certifications nationally, and many states have adopted their own regulations regarding requirements for initial certification as well as maintenance of the coaching credential. Some states require that all coaches receive training in cardiopulmonary resuscitation (CPR)/automated external defibrillation (AED) and first aid as a means to address emergency care procedures, whereas other states have no mandate regarding basic emergency care training. According to the National Federation of State High Schools, only 37 states require that coaches obtain basic first aid certification in order to be eligible to coach in that respective state. Only 14 of the 37 states require coaches to obtain CPR/AED training in addition to basic first aid.¹³ Even though 37 states require some form of medical training for coaches, the curriculum often centers on more basic concepts rather than on causes, signs and symptoms, and treatment of emergency situations, like EHS or concussions for example. This most likely leaves the coach unprepared to handle an emergency situation because they do not have the proper knowledge or training. Many states have begun to take initiative in passing legislation as it relates to concussions in sports, such as Connecticut and New Jersey.

As many secondary schools continue to rely on their coaches to protect the safety of their players and many states are initiating legislation which requires more advanced training regarding sudden death in sport, it is important to gain an appreciation for the

secondary school coaches' level of understanding of sudden death in sport. This information can be helpful on multiple levels including advancing the knowledge of the coach by identifying areas of deficiency as well as providing baseline information for the AT regarding a coach's knowledge of sudden death in order to better educate him/her on ways to help prevent its occurrence. The purpose of this investigation, therefore, was to gain an understanding of the secondary coaches' perspectives on the recognition and prevention of EHS. Particular attention was paid to the coaches' relationship with an AT and their knowledge of the role of the AT.

Methods

In this investigative research study, a mixed methods design was used to evaluate the study's purpose. An online survey asking open-ended questions, a popular new tool used to gather data, was used to enhance the quality of the data. The secondary school coach has been implicated as an important catalyst to the prevention of sudden death in sport, yet limited data exists regarding their perceptions of their role in prevention, as well as their knowledge regarding the topic. The primary focus of the study is to gain an appreciation for the secondary coaches' outlook related to sudden death; therefore, an exploratory study using open-ended survey questions will allow the researchers to gain a more holistic understanding of the coaches' perspectives on this topic.^{14,15}

Participant Sampling

We purposefully recruited participants with professional experience as a head football coach in the secondary school setting using a criterion sampling procedure.¹⁶ For this study, the primary inclusion criteria included: filling the position of head football

coach at the secondary school level. In addition, a conscious effort was made to recruit participants based upon: 1) *Region*: northern and southern regions of the country; and 2) *Years of Experience*: novice coach (0 to 10 years of experience) or experienced coach (more than 10 years of experience). We actively recruited a near equal distribution between region (21 coaches from the north and 17 coaches from the south) and years of experience (22 novice coaches, and 16 experienced coaches), and relied on data redundancy,¹⁵ to guide participant recruitment, which concluded at 38 total participants.

Participant Recruitment

Potential participants were initially recruited at the 2011 American Football Coaches Association annual meeting held in Dallas TX using an informational sheet that contained the study's purpose and collection procedures. Contact information was collected at the meeting and emails were sent to those who indicated interest to initiate data collection proceedings. In addition, secondary schools were randomly selected from each state's interscholastic athletic association website and contact information for the head football coach was retrieved. Potential participants were recruited via email; contained in the email was the invitation letter, the same used at the conference, and a link to complete the online open-ended survey questions. All participants who received invitation email were sent reminder emails at one and two weeks post initial invitation email.

Participants

Thirty-eight secondary school head football coaches completed the open-ended survey questions (37 males, and 1 female). The average age of the participants was $47 \pm$

10 years of age. The average number of years that the participants have been a head football coach was 12 ± 9 years. Table 1 provides a summary of demographic data.

Participants		
	Male	37
	Female	1
Education		
	Associates	1
	Bachelors	8
	Masters	28
	PhD	0
	EdD	0
	Other	1
States Represented		
	OH	2
	CT	3
	WI	7
	MO	1
	KY	2
	GA	1
	MI	1
	SC	1
	IL	3
	NC	1
	AL	1
	LA	1
	MN	3
	FL	1
Medical Coverage		
	DE	1
	TX	5
	Full-time	5
	ATC	17
	NH	3

Data Collection Instrument

	Part-time ATC	17
Participants were directed to	EMT/EMS	10
the interview questions via email and	Nurse	0
submitted questionnaires were stored	Non-medical	0
on the website Zoomerang™. In total,	Other	4

the participants responded to 39 questions with the first 12 questions asking about the coach's experience dealing with emergency situations and their relationship with an AT (Appendix A). Examples of the questions that were asked in regards to the coach's experiences were: "Do you feel qualified and prepared to handle an emergency situation (for example: exertional heat stroke)?" "Do you feel you should gain training in emergency care procedures?" "Have you had any experience with a case of sudden death or an emergency situation (spineboarding, concussion, etc.), while coaching (describe)?" "Are you in support of having an Athletic Trainer during practices and games at your high school? Why or why not?" "What do you believe are the AT qualifications or responsibilities?"

Table 1: Demographic data of secondary school head football coaches.

The remainder of the questions were demographic in nature and asked about the coaches age, number of years coaching football at the secondary school setting, number of years as head coach at the secondary school setting, the state in which they coach, the level of education that they have received, the medical coverage they have at practices and games, the signs and symptoms that the coach believes is indicative of EHS, if they have an AT, whether the AT is full-time or part-time, and the coaches' professional relationship with an AT. When asked about the signs and symptoms of EHS, the coaches were given a list of signs and

symptoms of EHS and were instructed to indicate any and all of the signs and symptoms that would lead them to a diagnosis of EHS in an athlete.

A likert scale was used to quantitatively assess the professional relationship the coach had with an AT. A ten-point scale (1-“not” or lacking to 10-“very”) was used to assess seven attributes associated with a positive relationship. The attributes that were used included: cooperativeness, professionalism, helpfulness, honesty, respectfulness, informative, and communicative.

Data Collection Procedures

All participants responded to a series of open-ended questions asking their knowledge and level of preparedness to handle an emergency situation and the role of the AT in the secondary setting followed by a series of background questions including age, years of experience, among others. The background information also asked the participants their knowledge with EHS and their professional relationship with an AT. The online methodology was purposefully selected to allow the researchers to gain access to a regionally diverse group of participants as well as to provide a more favorable data collection procedure for the secondary coach. Although phone interviews would have added to the richness of the data and afforded flexibility with the interviewing through dialogue and follow-up, the more serious nature of the study lends itself to the anonymity of an online interview.¹⁷ The online interview offered the participant time to reflect and process the question posed before answering the question. Anonymity of participant responses was assured in that the researchers were unaware of the names of the participants that took part in the study.

The online survey was developed by a research team, which included 5 members. Those members included two athletic training educators, two graduate assistant athletic trainers, and one qualitative researcher. Prior to data collection, the online interview guide, was reviewed by an expert qualitative researcher not involved with the data collection procedures, and was piloted with a small cohort of coaches (n=2). Updates and changes were made including grammatical edits, order of questions, and rephrasing for clarity. The data gathered from the pilot was not used for data analysis, but rather ensuring credibility to the data collection procedures.

Credibility

Credibility of the research procedures and trustworthiness of the data analysis and interpretation was established using 3 strategies: 1) peer review,¹⁵ 2) multiple analyst triangulations¹⁵ and 3) data source triangulation¹⁸ As described previously, an independent researcher examined the data collection procedures prior to data collection. They also evaluated the themes as determined by the research team. Three researchers including one graduate student and two experienced qualitative researchers were involved with data analysis autonomously. Once completed, the researchers discussed their findings, and were in agreement regarding the final presentation of the data.

Data Analysis

The data was analyzed borrowing from both a modified ground theory approach^{19,20} and a general inductive analysis.²¹ All data were reviewed in its entirety prior to analysis to gain a sense of the dominant themes. The researchers utilized the study's overall research agenda to guide the next steps in analysis, which included coding

the data with labels to represent its meaning based on the frequency of common responses. Common labels were grouped together, and eventually assigned a category to represent its overall meaning as agreed upon by the research team. The Likert scale data was evaluated by finding the overall mean and standard deviation of each attribute based on the participant responses and the resulting number was used to give the overall perception of the professional relationship between the coaches and AT. The overall mean and standard deviation of each attribute was plotted along the Likert scale to give a numerical interpretation of the relationship on a scale of 1-10.

Results

Within this research study, two dominant themes emerged from the data set: *the influence of the athletic trainer and the perceived self-confidence of the secondary school football coach*. The first theme is supported by the positive professional relationship between the head football coach and the athletic trainer as well as the coach's perspectives on the role of the athletic trainer. The second theme is supported by the coach's positive self-efficacy as it relates to handling emergency situations, despite their lack of knowledge on the issue. Each theme is defined and discussed next with supporting quotes and data from the coaches.

The influence of the athletic trainer

Qualitative

The theme, *influence of the athletic trainer*, reflects a positive professional relationship that existed between the secondary school football coach and the athletic trainer. All but four of the coaches were currently working directly with an athletic

trainer, and all had previous professional relationships with an athletic trainer. One coach shared, “having a [athletic] trainer on staff has always been a positive [experience].”

Another coach, shared, “an athletic trainer is invaluable [to our athletics program].”

Another coach, also felt the athletic trainer was a valuable asset to a secondary schools athletic program. He said,

A good athletic trainer is more valuable to a high school staff than any of the coaches. If I were at a high school that did not have an [athletic] trainer, I would go with one less coach on my staff to make sure I had the money to hire an athletic trainer for my athletic program.

The coaches’ positive evaluation of the athletic trainer was partially due to their basic understanding of the role of the athletic trainer within the healthcare team, which included injury assessment and return to play decisions. One coach mentioned, “they [athletic trainers] are very important to treating injuries and getting kids back to playing.” Another coach responded comparably, “helping to diagnose injuries is valuable, more importantly the valuable input and therapy to bring that athlete back from an injury.” One response when asked about athletic trainer’s qualifications was, “for diagnosis, immediate care and prevention and also for treatment and clearance to resume play.” A second coach stated an athletic trainer is qualified to, “diagnose, treat and prevent injury (further injury).” “I believe they are qualified to identify injuries and determine the course of action following,” as was the response of yet another coach. To sum up what the coaches felt the athletic trainer was qualified to do, a coach responded, “Without an athletic trainer present, our programs and the coaches will be at a disadvantage and the health of the student-athletes would be at a higher risk level.”

Although they demonstrated a fundamental knowledge of the role of the athletic trainer, they did not

	Attribute	Mean	Standard Deviation
mention emergency or	Cooperative	9.5	0.8
acute care, until	Professional	9.5	0.9
prompted by the	Helpful	9.7	0.6
specific question	Honest	9.7	0.7
addressing emergency	Respectful	9.8	0.5
care of athletes. The	Informative	9.6	0.7
sequencing of questions	Communicating	9.3	1.1
related to the role of the			

AT in the secondary

school had the

Table 2: Professional relationship between secondary school head football coach and athletic trainer.

following order: the role of the AT in the healthcare of secondary school athletics, what the AT is qualified to do for athletes, and if the coaches believed the AT was qualified to handle emergency situations. Not until the coaches were specifically asked about the AT's qualifications in regards to emergency situations did they address this. All of the coaches were in agreement that an athletic trainer is qualified to handle life-threatening conditions, if and when necessary. One coach said, "fully capable of handling emergency situations." Another coach speaking directly about his athletic trainer, shared, "he is extremely more qualified [than I am] and probably has [experience] dealing with more serious issues than the coaches." Another coach spoke in relative terms, stating, "they are highly trained in dealing with emergency situations." One coach was very detailed, providing this; "[an athletic trainer can do] CPR, AED, first aid, oxygen administration,

mouthpiece removal, spine boarding, helmet removal, suction, all type helmet removal, current knowledge of EAP and local EMS resources.”

Quantitative

The theme was further supported by data collected from the background questionnaire in which the coach using a Likert scale evaluated their professional relationship with the athletic trainer. Seven attributes were assessed including: cooperativeness, professionalism, helpfulness, honesty, respectfulness, informative, and communicative. The Likert scale was scored on a ten-point scale (1- “not” or lacking to 10-“very”). The coach, based upon the responses, would summarize their relationships as honest ($9.7 \pm .7$), respectful ($9.8 \pm .5$), and helpful ($9.7 \pm .6$). Table 2 summarizes the data.

Perceived self-confidence of the secondary school football coach

Qualitative

The head football coaches were asked if they felt prepared to handle an emergency situation such as exertional heat stroke or cardiac arrest. Of the 38 participants, 24 (63%) felt that they were prepared to handle an emergency situation. One coach responded, “I feel qualified and prepared to handle and emergency situation. I am on our school’s emergency response team.” Another responded, “Feel prepared to deal with the initial response and identification of such injuries. We are required to have CPR training to coach.” A third coach stated, “I feel prepared because I have received training in emergency care.”

The coach's perceived positive self-efficacy, however, does not match their knowledge in relation to emergency situations. Coaches were given a list of signs and symptoms and were asked which ones are the signs and symptoms of EHS, as we were particularly interested in the coach's knowledge base on EHS. The coaches were asked to state why they chose the symptoms in which they did. Varying responses were given: "Those are what I've been told are signs to look for," "Because in my experience they are easy to see and quick to identify," "Those things happen in the heat." Other coaches responded to this question by stating: "cold and clammy no sweat is a bad sign," "dizziness is one of the 1st signs to stop everything," "I believe they would help lead me to a problem." Based on this set of responses, coaches have limited knowledge as it relates to the signs and symptoms of EHS.

In addition to their lack of knowledge as it relates to the signs and symptoms of EHS, the head football coaches also lacked knowledge as it related to the prevention of EHS. Coaches went in depth in regards to what prevention strategies they use as it relates to EHS. One coach stated, "1. We cancel activities when conditions are too severe. 2. We keep plenty of water and ice on hand. 3. We schedule many hydrations breaks 4. We are vigilant to observe athletes who might exhibit any signs." Another coach mentioned,

As a staff we monitor the heat and heat index. We try to practice during the cooler temps of the day. Also, if we must practice during higher temps, we frequent water breaks and reduce the equipment worn by our players. Also, we will lessen the high activity and try to do more of a teaching walk thru type practice.

A third coach goes on to say, “We monitor temps. At 105 practice is off. Water breaks are allowed at any time. We remove helmets and shoulder pads when it gets close to but not quite 105.”

Although most of the coaches in this study stated that they felt prepared to handle an emergency situation, it is clear that their knowledge base does not match their perceptions on the matter. The coach’s inability to recognize all the signs and symptoms of EHS along with appropriate prevention strategies provides a clear example of how secondary school coaches are unprepared to handle an emergency situation that could result in the death of an athlete.

Quantitative

As mentioned above, coaches were given a list of signs and symptoms of EHS and asked to mark which ones they would look for in a possible case of EHS. Table 3 shows the list of signs and symptoms along with the number of coaches indicating which signs and symptoms were indicative to the recognition of EHS. Central nervous system dysfunction is the most prevalent sign and symptom of EHS aside from a core temperature above 105 degrees Fahrenheit and the results showed that 92% of the coaches listed dizziness as most important in the recognition of EHS whereas only 32% listed central nervous dysfunction as most important when recognizing EHS. Coaches were also asked what prevention strategies they use during practice to prevent EHS. 25/38 (66%) mentioned frequent water breaks as a prevention strategy. Of those, 18/25 (72%) mentions that frequent water breaks are the only prevention strategy they use to prevent EHS.

Discussion

The impetus for this study stems from the realization that the secondary school coach plays a significant role in the prevention of sudden death in sport. Within the last five years several coaches have been prosecuted in both criminal and civil law for their negligent role in failing to follow safety guidelines or precautions related to preventing sudden death in sport. Many of the cases of sudden death in sport have involved EHS or exertional sickling during conditioning sessions or pre-season practices when an athletic trainer may not be present on-site to help provide medical care. This may indicate a limited understanding of the coach regarding the causes of sudden death, signs and symptoms of those conditions, and effective prevention measures. Unfortunately, there is limited data regarding the knowledge of and implementation of prevention practices of the secondary school coach. The purpose of this investigation therefore was to gain an understanding of the secondary school coaches' perspectives on sudden death and sport.

Particular attention was paid to the coaches' relationship with an AT and their knowledge of prevention practices as it relates to sudden death in sport. Since less than 45% of all secondary schools in the United States employs an AT, the coach often times is the responsible party charged with caring for the athlete.²² Previous research²³⁻²⁹ has looked at the knowledge and decision making procedures of coaches in relation to first aid, but not dealing with prevention of sudden death in sport or their relationship with an AT. Similar to the previous literature, we found that the coach is deficient in their knowledge of emergency procedures and conditions leading to sudden death in sport. Our results also demonstrate that the professional relationship between the AT and the coach is professional, honest, and important in navigating the management and treatment of an

emergency situation. Moreover, our results illustrate that the secondary school coach reported positive self-efficacy regarding their abilities to handle an emergency situation, but lack the actual knowledge regarding all aspects of sudden death in sport. A discussion ensues regarding the main findings of the study.

Influence of the athletic trainer

The coaches who participated in this study reported having a positive professional relationship with their AT and listed the relationship as honest, respectful, and helpful. Consistent with the work of Mensch and colleagues,³⁰ the secondary coaches trust the judgments of their athletic trainer and have an overall good working relationship. The positive relationship, found in our study, was partially mediated by the coaches' basic understanding of the role of the AT as it relates to injury assessment and return to play of injured athletes. Our findings refute previous literature, which had indicated a limited understanding of the roles and qualifications of the athletic trainer.³⁰ A plausible explanation for this discrepancy could be the purpose of the research study, where Mensch and colleagues³⁰ were concerned with the coaches understanding of the credentials of the AT and services provided; whereas our study was geared towards the skills and role of the AT as it pertains to sudden death in sport. The coaches in our study were aware of the AT's abilities to evaluate and diagnosis as well as treat a variety of athletic injuries. They also valued the role they played in the healthcare and well-being of their student-athletes.

Previous research, comparable to our findings, has demonstrated the importance of good communication between the coach and AT, as it is critical to the development of

a positive relationship between the two professionals.³⁰ The communication between the coach and the athletic trainer is necessary to provide appropriate continuity of care to the student-athlete especially at the secondary school level, where coverage can be limited, but also as demonstrated by our findings to promote a strong professional relationship. Moreover, our results help to exemplify the growth that has occurred related to the coaches understanding of the role of the AT, which is mostly likely attributed to increased exposure due to the increased employment in that clinical setting.

Effective communication is essential in order to develop trusting and respectful relationships with coaches as highlighted by our findings as well as those of Mensch and peers³⁰ in an earlier study. Communication must be cooperative, whereby the coach should and must respect the medical decisions made by the athletic trainer and the AT needs to be considerate of the non-medical decisions of the coach, as long as they are in the best interest of the student-athlete.³¹ Furthermore, efficient communication can better facilitate the care provided to an athlete, as a primary role of the AT is to educate the student-athlete and the coach regarding prevention and training strategies.³² Therefore, the AT can serve as a powerful tool in preventing sudden death in sport as the relationships they develop with their coach appears to positively impact the coach's awareness of these conditions as well as their understanding of their skills related to management of these conditions.

Our findings also indicate that the coach can be a strong ally for the AT at the secondary school level, as the coaches in this study were very supportive of having one on staff and could be used to help secure additional resources for the AT. The support of

the AT was most definitely facilitated by the professional relationship developed between the two parties, which was founded on respect and effective communication.

Positive Self-Confidence and Emergency Care

The coaches in this study had positive perceptions regarding their abilities to manage an emergency situation, if presented during a practice or competition. This confidence was directly attributed to annual emergency care training via CPR and first aid training offered through the American Red Cross or comparable provider. This is an interesting finding, as it appears that many coaches fail to meet the standards established by emergency response organizations for first aid and CPR.²³ Ransone *et al.* found that 36% of coaches working in metropolitan school districts achieved a passing score on an adapted first aid assessment exam, despite having the basic CPR/First Aid Certification.²³ The results generated by Ransone and colleagues are not shocking, as consistently the data regarding coaches and their first aid knowledge and decision-making has yielded unfavorable results. Barron *et al.*, reported only 5% of coaches surveyed passed a revised first aid assessment, which consisted of questions adapted from the American Red Cross first aid and CPR examinations.²⁷ Although our study did not ask the coach whether or not they were certified in CPR and first aid, many indicated that they have received CPR and first aid training. Our study did showed the coach's inability to properly recognize a possible EHS based on signs and symptoms as well as their lack of knowledge on EHS prevention. The results generated by Ransone *et al.*, Barron *et al.*, and our present study, highlight the deficiency in the coaches' knowledge regarding first aid, injury prevention, and emergency care. A concerning finding as many secondary schools do not employ the

full-time services of an AT leaving the care of the student-athlete in the hands of the coach.

Our results in combination with Ransone *et al.* also indicate that a coach is not prepared to make return to play decisions or make an initial impression of an injury or emergency situation. This information is important to share, especially with policy makers or school boards, as in some cases such as Kentucky, they have considered providing additional medical training to the coach, in lieu of an AT to help prevent sudden death in sport.^{12,33} Clearly, this is not a feasible option, as the coaches despite receiving training, do not appear to retain the information or utilize it properly.²³ This is mostly notably due to a lack of an appreciation for the seriousness of these conditions. In our study, 63% of the coaches indicated that they felt prepared to handle an emergency situation and most based this response on the fact that they held both CPR and first aid certification. Even though that is a significant number of our participants, many were unable to identify all of the signs and symptoms of EHS as well as unable to list proper preventive strategies for EHS. They were more focused on routine conditions, such as cardiac and head injuries, information that is taught during basic emergency care courses.

In addition to the coaches' lack of knowledge in regards to first aid, Ransone *et al.* showed that coaches portrayed poor decision making skills dealing with return to play of athletes. That is, many coaches, especially those with demonstration of competence in emergency care, were more likely to return an injured player despite continued symptoms or pain.²³ Although this was not evaluated in the present study, the deficiency in knowledge demonstrated regarding key signs and symptoms of EHS or plausible conditions, indicates that the coach is ill prepared to make return to play or medical

decisions. Furthermore, ATs working with secondary school coaches need to be aware that a coach's knowledge does not match their level of preparedness, which is at best over-confident. Continued education and communication between the two parties can help increase the coach's knowledge of these conditions, means to prevent a condition from happening, and an appreciation for the seriousness of sudden death in sport and the role training can have on its occurrence.

Limitations

Our study is not without limitations. We utilized asynchronous, online open-ended survey questions as a means to collect data qualitative data, and although a popular new method that produces rich data, not knowing the names and contact information of the participating coaches limits the ability for the researchers to follow-up on questions posed or responses provided. The structured interview guide was reviewed for content as well as piloted with two coaches prior to data collection, helping to reduce misinterpretation, but the researchers recognize the limitation in online interviewing. Due to the small sample size, the results cannot be generalized to all secondary school head football coaches, especially coaches without an AT at their school. All but 4 of the coaches who participated in this study had an AT employed at their secondary school therefore it is likely that the coaches responded to the survey based on their positive professional working relationship with a AT. In addition, we only looked at the perspective of the head coach in the secondary setting, so it is unclear as to whether or not an AT working in the secondary school would perceive the same thing as the head football coach. As previously indicated, communication with an AT can enhance the coaches perceptions of their role as well as increase their knowledge and comfort level

regarding sudden death in sport. Finally, the mean age for this cohort of coaches was 47 with an average of 12 years head coach coaching experience. The results may not completely reflect the secondary school coach with less experience in the role of the head coach.

Recommendations

To assist in increasing the knowledge base of the secondary school football coach as it relates to the prevention of sudden death in sport, a few recommendations can be made. First, for schools who employ an AT, the AT can teach an emergency care course for all of the coaches at the school in which he/she works. The emergency care course can discuss prevention, recognition, and treatment of the various causes of sudden death in sport such as EHS. The course could also include hands on training to familiarize the coaches with the correct way to handle emergencies. The AT should also stress the importance of effective communication between the AT and the coach to facilitate proper care of the student-athlete. In addition, states could mandate that all coaches take a course on emergency care with an annual or bi-annual recertification requirement since less than 45% of secondary schools employ an AT. By requiring coaches to take an emergency care course, it will help prepare coaches to handle an emergency situation, especially coaches that do not have the benefit of having an AT at their school.

Future Research

Our sample was small and although our results help to expand the current understanding of sudden death in sport as well as the relationship between the coach and AT, a larger study using the current model will help to justify the findings as well as

expand our understanding of the relationship between ATs, coaches, and sudden death in sport. Although our study answered questions in regards to the coaches' perspectives on the prevention of sudden death in sport and the role of the athletic trainer, questions arose from our study that could be used for further research. As an extension to this study, are the coaches' perspectives of the prevention of sudden death in sport and the role of the AT similar across other sports as they are in football? A possible study looking at head coaches in sports such as soccer, basketball, and lacrosse could be done to see if there is a comparison in answers from these coaches to the football coaches studied.

Since all but four of the participants in this study had a working relationship with an AT, a study looking at a larger sample of coaches who did not have a working relationship with an AT would provide a better understanding of the coaches' perspectives on sudden death in sport without the influence of an AT. In addition, this study only examined the coaches' perspectives on sudden death in sport and the relationship between the coach and the AT. Looking at the AT's or athletic director's perspectives in relation to sudden death in sport and the relationship between the AT and the coach might provide a different insight that should be explored.

Coaches in our study indicated a positive self-efficacy despite their lack of knowledge as it relates to emergency situations. In addition to asking if the coach feel prepared to handle emergency situations, further research looking at the coaches' decision making into the proper treatment of potential emergency situations should be explored. A study giving coaches real life situations of potential cases of sudden death in sport and asking them the steps they would take to treat the situation could provide

investigators information on not only the coaches knowledge base but what the coach would do if faced with an emergency.

References

1. Korey Stringer Institute. Real time registry of sudden death in sport and physical activity. Korey Stringer Institute Web site.
<http://ksi.uconn.edu/personalstories/RTRegistrySDinSport.html>. Updated 2011. Accessed November 16, 2011.
2. Casa DJ, ed. *Preventing sudden death in sport and physical activity*. Sudbury, MA: Jones & Bartlett; 2011.
3. Mueller FO. Annual survey of catastrophic football injuries 1977 - 2010. Web site.
<http://www.unc.edu/depts/nccsi/2010FBCatReport.pdf>. Updated 2010. Accessed 3/15/2011, 2011.
4. Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: Analysis of 1866 deaths in the united states, 1980-2006. *Circulation*. 2009;119(8):1085-1092.
5. Maron BJ. Hypertrophic cardiomyopathy and other causes of sudden cardiac death in young competitive athletes, with considerations for preparticipation screening and criteria for disqualification. *Cardiol Clin*. 2007;25(3):399-414, vi.
6. Basavarajaiah S, Shah A, Sharma S. Sudden cardiac death in young athletes. *Heart*. 2007;93(3):287-289.

7. Basavarajaiah S, Wilson M, Whyte G, Shah A, McKenna W, Sharma S. Prevalence of hypertrophic cardiomyopathy in highly trained athletes: Relevance to pre-participation screening. *J Am Coll Cardiol*. 2008;51(10):1033-1039.
8. Lyznicki JM, Riggs JA, Champion HC. Certified athletic trainers in secondary schools: Report of the council on scientific affairs, american medical association. *J Athl Train*. 1999;34(3):272-276.
9. Casa DJ. Exertional heat stroke of tyler davenport. .
10. Casa DJ. Exertional heat stroke death of max gilpin. .
11. National Athletic Trainers' Association. Appropriate medical care for secondary school-age athletes: Consensus statement.
<http://www.nata.org/sites/default/files/AppropriateMedicalCare4SecondarySchoolAgeAthletes.pdf>. Published February 2003. Updated 2003. Accessed November 16, 2011.
12. Kentucky High School Athletic Association. Bylaw 27: Requirement for coaches. . 2011.
13. National Federation of State High School Associations. 2009-10 high school athletics participation survey. <http://www.nfhs.org/content.aspx?id=3282>. Updated 2011. Accessed 4/15/2011, 2011.
14. Meriam SB. *Case study research in education: A qualitative approach*. San Francisco, CA: Jossey-Bass; 1988.

15. Pitney WA, Parker J. *Qualitative research in physical activity and the health professions*. Champaign, IL: Human Kinetics; 2009.
16. Creswell JW. *Qualitative inquiry and research design: Choosing among five traditions*. Thousand Oaks, CA: Sage Publications; 1998.
17. Meho L. E-mail interviewing in qualitative research: A methodological discussion. . 2006;57(10):1284-1295.
18. Pitney WA, Parker J. Qualitative research applications in athletic training. *J Athl Train*. 2002;37(4 Suppl):S168-S173.
19. Glaser BG SA. *Discovery of grounded theory: Strategies for qualitative research*. Chicago IL: Aldine; 1967.
20. Strauss AC, JM. *Basics of qualitative research: Grounded theory procedures and techniques*. Newbury Park, CA: Sage Publications; 1990.
21. Creswell JW. *Educational research: Planning, conducting, and evaluating quantitative and qualitative approaches to research*. Upper Saddle River, NJ: Merrill/Pearson Education; 2002.
22. Lyznicki JM, Riggs JA, Champion HC. Certified athletic trainers in secondary schools: Report of the council on scientific affairs, american medical association. *J Athl Train*. 1999;34(3):272-276.

23. Ransone J, Dunn-Bennett LR. Assessment of first-aid knowledge and decision making of high school athletic coaches. *J Athl Train*. 1999;34(3):267-271.
24. Kujawa R, Coker CA. An examination of the influence of coaching certification and the presence of an athletic trainer on the extent of sport safety knowledge of coaches. . 2000;15:14.
25. Rowe PJ, Miller LK. Treating high school sports injuries: Are coaches/trainers competent? . 1991;62(1):49.
26. Barron M,J. The assessment of first aid and injury prevention knowledge and the decision making of youth basketball, soccer, and football coaches. . 2004.
27. Barron MJ¹emme, Powell JW², Ewing ME², Nogle SE², Branta CF². First aid and injury prevention knowledge of youth basketball, football, and soccer coaches. . 2009;3(1):55.
28. Valovich-McLeod TC¹, McGaugh JW², Boquiren ML³, Bay RC⁴. Youth sports coaches do not have adequate knowledge regarding first- aid and injury prevention. . 2008;23:130.
29. Vergeer I. Coaches' decision policies about the participation of injured athletes in competition. . 1999;13(1):42-56.
30. Mensch J, Crews C, Mitchell M. Competing perspectives during organizational socialization on the role of certified athletic trainers in high school settings. *J Athl Train*. 2005;40(4):333-340.

31. Martens R. *Successful coaching*. 3rd ed. Champaign, IL: Human Kinetics; 2004.
32. National Athletic Trainers' Association. Code of ethics.
<http://www.nata.org/codeofethics>. Updated 2005. Accessed November 16, 2011.
33. Casa DJ. Should coaches be in charge of care for medical emergencies in high school sports? *Athletic Training & sports healthcare*. 2009;1(4):144-145, 146.

Appendix A: Invitation Letter

Hello

Hello, you are invited to participate in a research study looking at sudden death in the secondary school setting. This study is being conducted by William Adams and Benjamin McGrath as part of their thesis requirement at the University of Connecticut. Stephanie Mazerolle and Douglas Casa serve as academic and research advisors for this study. Your participation in this study is completely voluntary and involves electronic interviewing through a secured website. Your total participation should take approximately 30 minutes. Completing the survey will indicate consent.

The purpose of this study is to investigate the Secondary School coach's perspectives on sudden death in sport and the role of the athletic trainer as it pertains to the prevention of sudden death in sport.

This study involves an online survey via the host site Zoomerang. The survey will include both open-ended questions and demographic questions. The open-ended questions will require a typed response and will vary in length depending upon your experience to the question asked. Your responses are completely anonymous. The risks are minimal for this study and include the potential for the participant to be identified once the results of the study are published. To minimize this risk, all participants who participate in the online interviews will be identified by a pseudonym. The demographic questions located after the open-ended questions so please be sure to complete all questions before terminating the survey. You will only have a one-time access to the link, once you've initiated the online link. Reminder emails for completion will be sent at one and two-weeks.

If interested please click on the following link to begin the survey:
<http://www.zoomerang.com/Survey/WEB22C3AMHVU5S/>.

Participation is confidential and optional. Pseudonyms will be used in place of names, and there will be no link to your current/former institution(s). If you would like to participate, or if you have questions about this study, you may contact me at william.adams@uconn.edu or benjamin.mcgrath@uconn.edu or stephanie.mazerolle@uconn.edu, 860-486-4536. This research study was approved by the UCONN IRB, Protocol #H10-310.

Thank you for your consideration. Please contact me if you have any questions regarding this study.

Sincerely,

William Adams, BS, ATC
Benjamin McGrath, BS, ATC
Stephanie Mazerolle, PhD, ATC

Appendix B: Background Questionnaire

1. Gender ☐Male ☐Female
2. Age_____
3. How long have you been coaching football?_____
4. How long have you been a head football coach?_____
5. What state are you currently working in?_____
6. Indicate your highest level of education

☐Associate's Degree
Degree ☐PhD

☐Bachelor's Degree
_____EdD

☐Master's
_____Other,
specify_____
7. How long have you been employed at your current school?_____
8. Who provides medical coverage for your practices and games? If you have an Athletic Trainer, please respond to questions #9 and #10 as well

☐Full-time Athletic Trainer

☐Part-time Athletic Trainer

☐Emergency Personnel Service (EMS/EMT)

☐Nurse

☐No medical coverage is provided
9. Does the Athletic Trainer attend all practices and competitions?
 ☐Yes ☐No
10. Is the Athletic Trainer involved in pre-season practice scheduling?
 ☐Yes ☐No
11. Are you aware of the National Athletic Trainers' Association (NATA) statement on "Preseason Heat Acclimatization Guidelines for Secondary School Athletes?"
If yes, please answer #12
 ☐Yes ☐No
12. Have you read NATA's statement on "Preseason Heat Acclimatization Guidelines for Secondary School Athletes?"
 ☐Yes ☐No
13. Are you aware of the NATA's position on medical coverage and the Secondary School?

____Yes ____No

14. Have you read the NATA's position statement on medical coverage in the Secondary School?

____Yes ____No

15. How would you describe your professional relationship with your Athletic Trainer? If you do not have an athletic trainer, please skip to the next question

Not Cooperative 1 2 3 4 5 6 7 8 9 10 Very Cooperative

Not Professional 1 2 3 4 5 6 7 8 9 10 Very Professional

Not Helpful 1 2 3 4 5 6 7 8 9 10 Very Helpful

Not Honest 1 2 3 4 5 6 7 8 9 10 Very Honest

Not Respectful 1 2 3 4 5 6 7 8 9 10 Very Respectful

Not Informative 1 2 3 4 5 6 7 8 9 10 Very Informative

No Communication 1 2 3 4 5 6 7 8 9 10 No Problems Communicating

16. What do you feel is the most important aspect of recognizing an exertional heat stroke? Please circle the symptoms listed below that you believe are most important when recognizing exertional heat stroke

Sweating, redness, cramping, dizziness, nausea, vomiting, weakness, central nervous system dysfunction, fever, not sweating, hot skin

Why did you choose those symptoms?

15. Have you ever had an athlete suffer from exertional heat stroke?

____Yes ____No

a. If yes, how did you recognize it and what did you do for it?

16. Is heat stroke discussed in your school's athletic department policy and procedures manual?

___ Yes ___ No

a. If so, what is your school's policy on heat stroke?

17. Have you been given hands-on practice to utilize the skills in treating/assessing an exertional heat stroke victim?

___ Yes ___ No

18. Rank the top 5, reasons for sudden death and provide common signs and symptoms of each

1.

2.

3.

4.

5.

Please feel free to add any comments below:

Thank you for participating in this study.

Appendix C: Interview Guide

Part I: The coach and emergency situations

1. Do you feel qualified and prepared to handle an emergency situation such as exertional heat stroke or cardiac arrest?
 - a. Probe: Do you feel you should gain training in emergency care procedures?
2. What strategies, if any, do you implement into your practices to prevent exertional heat stroke?
 - a. Probe: Does your school have an emergency action plan spelling out what to do in the case of an athlete suffering from exertional heat stroke or other emergency situations?
 - b. Probe: Have you reviewed it? Do you know who developed it? Do you review it annually?
3. There has been a lot of attention placed upon the role of the coach, especially following the case of Max Gilpin (KY). Do you believe the coach has a role in preventing sudden death in sport? (what is your opinion of the case, if you are aware of it?)
 - a. Probe: How realistic do you feel a case of sudden death is for your team?
4. What environmental conditions would lead you to alter practice and/or games?
 - a. Probe: Please explain how you would alter your schedule in accordance with the environmental conditions you just listed
 - b. Probe: Are there any environmental conditions that are cause for concern in the state in which you coach? If so, please explain how you prepare for the possible issues that can arise because of the conditions
5. Have you ever had a case of an athlete suffering from exertional heatstroke? If so, please describe the situation.
 - a. Probe: What signs did you notice? Did you have an Athletic Trainer present?
 - b. Probe: What was the course of treatment?
6. Have you had any experience with a case of sudden death or an emergency situation (spineboarding, concussion, etc.)?
 - a. Probe: Please describe the situation and the course of treatment

Part II: Relationship between coach and Athletic Trainer

7. Please describe your previous experiences with having an Athletic Trainer on staff
 - a. Probe: What role do you believe that an Athletic Trainer plays in healthcare and high school athletics?
8. Are you in support of having an Athletic Trainer available during practices and games at your high school? Please explain
 - a. Probe: What is your rationale for having or not having an Athletic Trainer onsite?
 - b. Probe: What do you believe the Athletic Trainer is qualified to do for athletes?
9. What do you believe is the extent of knowledge that an Athletic Trainer has and the extent to their practical skills in dealing with an emergency situation?
 - a. Probe: What is the difference between an AT and an EMT?
10. What barriers do you perceive as the reason for a lack of Athletic Trainers employed at the secondary school level? What would you think is the biggest reason why secondary schools do not hire an Athletic Trainer?
 - a. If you have an Athletic Trainer at your school: Why do you think other schools are not following along with hiring Athletic Trainers to provide medical care for the school's athletes?
 - b. If you do NOT have an Athletic Trainer at your school: What reasons exist for not hiring an Athletic Trainer at your school? Who makes the ultimate decision as to whether or not an Athletic Trainer is hired at the school?

