

# YOUTH SPORTS SAFETY ALLIANCE™



67 STATE SAFETY  
BILLS INTRODUCED:  
ONLY 15 PASSED

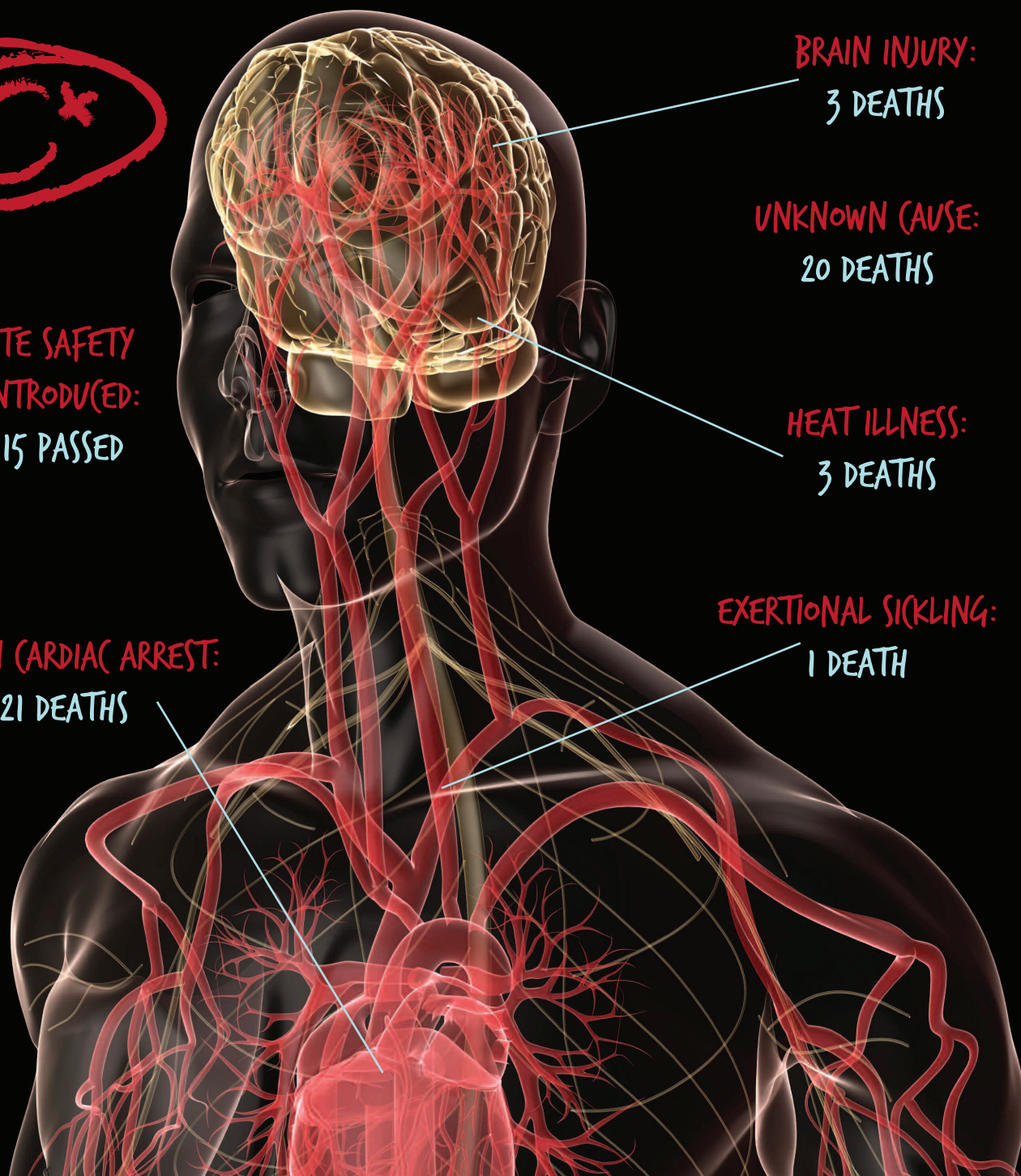
BRAIN INJURY:  
3 DEATHS

UNKNOWN CAUSE:  
20 DEATHS

HEAT ILLNESS:  
3 DEATHS

SUDDEN CARDIAC ARREST:  
21 DEATHS

EXERTIONAL SICKLING:  
1 DEATH



## ONE YEAR LATER:

*A Report Card on the Youth Sports Safety Crisis*

# IN MEMORIAM

## Youth athletes who died in 2010

<u>Month</u>	<u>Athlete</u>	<u>Age</u>	<u>Cause of Death</u>	<u>State</u>
JANUARY	LG	16	Sudden Cardiac Arrest	California
	NL	8	Sudden Cardiac Arrest	Indiana
	MC	15	Sudden Cardiac Arrest	Arkansas
	KM	15	Sudden Cardiac Arrest	Texas
	JM	14	Unknown/Inconclusive	West Virginia
	JE	17	Sudden Cardiac Arrest	California
FEBRUARY	DM	14	Sudden Cardiac Arrest	California
	WD	14	Sudden Cardiac Arrest	Georgia
	NW	17	Sudden Cardiac Arrest	Washington
	JN	16	Sudden Cardiac Arrest	Maryland
	JG	14	Unknown/Inconclusive	Tennessee
MARCH	CK	15	Unknown/Inconclusive	Kentucky
	EV	14	Sudden Cardiac Arrest	California
	AS	15	Sudden Cardiac Arrest	Texas
	JC	14	Sudden Cardiac Arrest	California
APRIL	JP	16	Sudden Cardiac Arrest	North Carolina
	AD	18	Unknown/Inconclusive	Massachusetts
	*	19	Unknown/Inconclusive	New York
	AN	12	Sudden Cardiac Arrest	Illinois
MAY	BF	13	Traumatic Brain Injury	New York
	*	17	Unknown/Inconclusive	North Dakota
	AC	15	Sudden Cardiac Arrest	Florida
	JS	18	Unknown/Inconclusive	North Carolina
	TS	15	Sudden Cardiac Arrest	California
JUNE	JG	16	Unknown/Inconclusive	Georgia
AUGUST	AP	11	Sudden Cardiac Arrest	California
	AA	9	Unknown/Inconclusive	Oklahoma
	TP	16	Heat Stroke	Oklahoma
	JW	16	Unknown/Inconclusive	Washington
	AF	16	Sudden Cardiac Arrest	Pennsylvania
	WD	18	Unknown/Inconclusive	Alabama
	BC	18	Sudden Cardiac Arrest	South Carolina
	QW	15	Traumatic Brain Injury	Pennsylvania
SEPTEMBER	KM	15	Sudden Cardiac Arrest	Louisiana
	EC	*	Traumatic Brain Injury	Wisconsin
	OL	15	Exertional Sickling	Florida
	*	*	Unknown/Inconclusive	Arizona
	RG	17	Unknown/Inconclusive	Texas
	AR	16	Unknown/Inconclusive	Florida
	KT	16	Heat Stroke	Oklahoma
	MR	17	Unknown/Inconclusive	Michigan
	MF	15	Unknown/Inconclusive	New Jersey
	LF	13	Unknown/Inconclusive	California
OCTOBER	MH	17	Unknown/Inconclusive	Georgia
	VK	17	Unknown/Inconclusive	Georgia
	TD	16	Heat Stroke	Arkansas
	DC	16	Sudden Cardiac Arrest	Louisiana
	WG	15	Unknown/Inconclusive	Illinois

\* Information not released



# YOUTH SPORTS SAFETY ALLIANCE

## ONE YEAR LATER: *A Report Card on the Youth Sports Safety Crisis*

Summit underwritten by the National Athletic Trainers' Association and its 32,000 members.

NATA would like to thank Russell T. Baker, MS, ATC, for gathering information about catastrophic injuries in high school sports.



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December 7, 2010

The National Athletic Trainers' Association spearheaded the Alliance to Address the Youth Sport Safety Crisis in America and created a call to action to raise awareness, advance legislation and improve medical care for young athletes across the country. We were gratified that 30 eminent organizations showed their support by joining the Alliance and that the January 2010 Summit was such a success.

What's happened since January 12? Fortunately, a great deal.

Millions – perhaps tens of millions – of Americans read newspaper and online stories about youth athlete safety, and watched national news programs which highlighted this important issue. State legislatures and the U. S. Congress introduced legislation, holding hearings to learn more about how to protect young athletes. Newspapers editorialized on the need to address safety issues.

National athletic organizations changed their rules to make play safer. Health care organizations representing professionals initiated safety awareness campaigns aimed at an array of injuries from the repetitive to the acute.

Many more children were provided with pre-participation physicals. Unknown numbers of parents started paying closer attention to the safety of the equipment and practices of schools hosting athletic activities, and they talked to their kids about the importance of reporting injuries.

Can we do more? Without a doubt.

Since January 1 of this year, at least 48 athletes, high school students and younger, died from brain injury, heat illness, exertional sickling, sudden cardiac arrest or as yet unknown causes. Sixty-seven pieces of safety legislation were introduced in 21 state legislatures; only 15 have passed at the time of this writing – and many of those could have been much stronger.

It is the aim of the Alliance at this early stage to keep the national focus on sports safety. Without public attention and support, other issues will eclipse this one until tragically predictable deaths of young athletes again grab the spotlight. As individuals and organizations, we each have a role to play in making sure we make significant advances in safety.

Toward that goal, we have scheduled a second Summit to be held on December 7 in the nation's Capital. This Summit, titled *ONE YEAR LATER: A Report Card on the Youth Sports Safety Crisis*, will offer two opportunities:

- Grow awareness of the potential for danger in sports
- Form partnerships to work on specific projects and issues

We offer today a revised Call to Action and some of the latest information from experts in the areas of exertional sickling, heat illness, brain injury, and sudden cardiac arrest. As we said last January, we want Americans to know there are solutions to this crisis. Thank you for helping us to continue the dialogue.

On behalf of the members and board of directors of the National Athletic Trainers' Association,

Marjorie J. Albohm, MS, ATC

President

# ONE YEAR LATER:

## *A Report Card on the Youth Sports Safety Crisis*

December 7, 2010

### Agenda

#### **Moderator:**

Marjorie J. Albohm, MS, ATC, president, National Athletic Trainers' Association

**9-10:15 a.m.**

#### **REPORT CARD**

- **Kelci Stringer**, founder and CEO, Korey Stringer Institute
- **Julie Gilchrist**, MD, pediatrician and medical epidemiologist, National Center for Injury Prevention and Control, Centers for Disease Control and Prevention
- **Dawn Comstock**, PhD, associate professor at The Ohio State University College of Medicine, Department of Pediatrics and College of Public Health, Division of Epidemiology, and a research faculty member at the Center for Injury Research and Policy (CIRP), the Research Institute at Nationwide Children's Hospital
- **Jeff Miller**, vice president for Government Relations and Public Policy, National Football League
- **Patti James**, mother of Will James

**10:15-10:30 a.m.**

#### **BREAK**

**10:30 a.m.-12:30 p.m.**

#### **MEDICAL ISSUES**

- *Brain Injury/Concussion*

**Gerard A. Gioia**, PhD, chief, Division of Pediatric Neuropsychology; director, Safe Concussion Outcome, Recovery and Education Program, Children's National Medical Center; associate professor, Departments of Pediatrics and Psychiatry, George Washington University School of Medicine

# ONE YEAR LATER:

## *A Report Card on the Youth Sports Safety Crisis*

### Agenda (continued)

- *Heat Illness*

Douglas J. Casa, PhD, ATC, FACSM, FNATA, director, Athletic Training Education; professor, Department of Kinesiology; chief operating officer, Korey Stringer Institute, University of Connecticut

- *Sudden Cardiac Arrest*

Francis G. O'Connor, MD, MPH, associate professor, Uniformed Services University; medical director, Consortium on Health and Military Performance

- *Sickle Cell Trait*

Scott Galloway, ATC, LAT, head athletic trainer, DeSoto High School, DeSoto, Texas

**12:30 p.m.-1:30 p.m.**

#### **LUNCH AND RESOURCE FAIR**

Alliance members may use this time to share information about their programs and services.

**1:30-3 p.m.**

#### **FACILITATED NETWORKING**

Participants will have the opportunity to partner with others on issues and projects of mutual interest.

**3-3:30 p.m.**

#### **WRAP-UP**

# YOUTH SPORTS SAFETY ALLIANCE

## *Members as of October 2010*

Advocates for Injured Athletes  
[www.injuredathletes.org](http://www.injuredathletes.org)

American Academy of Orthopaedic Surgeons  
[www.aaos.org](http://www.aaos.org)

American Academy of Pediatrics  
[www.aap.org](http://www.aap.org)

American Academy of Podiatric Sports Medicine  
[www.aapsm.org](http://www.aapsm.org)

American Association of Cheerleading Coaches & Administrators  
[www.aacca.org](http://www.aacca.org)

American Chiropractic Association Sports Council  
[www.acasc.org](http://www.acasc.org)

American Football Coaches Association  
[www.afca.com](http://www.afca.com)

American Medical Society for Sports Medicine  
[www.amssm.org](http://www.amssm.org)

American Orthopaedic Society for Sports Medicine  
[www.sportsmed.org](http://www.sportsmed.org)

American Osteopathic Academy of Sports Medicine  
[www.aoasm.org](http://www.aoasm.org)

California Brain Injury Association  
[www.calbia.org](http://www.calbia.org)

ImPACT  
[www.impacttest.com](http://www.impacttest.com)

KEN Heart Foundation  
[www.kenheart.org](http://www.kenheart.org)

Korey Stringer Institute  
[www.ksi@uconn.edu](mailto:www.ksi@uconn.edu)

National Academy of Neuropsychology  
[www.nanonline.org](http://www.nanonline.org)

National Association of School Nurses  
[www.nasn.org](http://www.nasn.org)

National Association of Secondary School Principals  
[www.nassp.org](http://www.nassp.org)

National Athletic Trainers' Association  
[www.nata.org](http://www.nata.org)

National Basketball Athletic Trainers Association  
[www.nbata.com](http://www.nbata.com)

National Center for Catastrophic Sports Injury Research  
[www.unc.edu/depts/nccsi](http://www.unc.edu/depts/nccsi)

National Center for Sports Safety  
[www.sportssafety.org](http://www.sportssafety.org)

National Cheer Safety Foundation  
[www.nationalcheersafety.com](http://www.nationalcheersafety.com)

National Coalition for Promoting Physical Activity  
[www.ncppa.org](http://www.ncppa.org)

National Council of Youth Sports  
[www.ncys.org](http://www.ncys.org)

National Interscholastic Athletic Administrators Association  
[www.niaaa.org](http://www.niaaa.org)

National Sports Safety Organization  
[www.nssousa.org](http://www.nssousa.org)

North American Booster Club Association  
[www.boosterclubs.org](http://www.boosterclubs.org)

North American Society for Pediatric Exercise Medicine  
[www.naspem.org](http://www.naspem.org)

Parent Heart Watch  
[www.parentheartwatch.org](http://www.parentheartwatch.org)

Pop Warner Little Scholars  
[www.popwarner.com](http://www.popwarner.com)

Professional Baseball Athletic Trainers Society  
[www.pbats.com](http://www.pbats.com)

Professional Football Athletic Trainers Society  
[www.pfats.com](http://www.pfats.com)

Safe Kids USA  
[www.usa.safekids.org](http://www.usa.safekids.org)

SportsConcussions.org  
[www.sportsconcussions.org](http://www.sportsconcussions.org)

Sudden Arrhythmia Death Syndromes Foundation  
[www.stopsads.org](http://www.stopsads.org)

Taylor Hooton Foundation  
[www.taylorhooton.org](http://www.taylorhooton.org)

US Lacrosse  
[www.uslacrosse.org](http://www.uslacrosse.org)

USA Football  
[www.usafootball.com](http://www.usafootball.com)





## CALL TO ACTION

### **The Youth Sports Safety Alliance Commits to**

- Ensure that youth athletes have access to health care professionals who are qualified to make assessments and decisions.
- Educate parents, athletes, coaches, teachers and others about the signs and symptoms of sports injuries and conditions (e.g., brain injury, heat illness and exertional sickling).
- Ensure pre-participation exams before play begins and, where appropriate, conduct baseline testing.
- Ensure that sports equipment, uniforms, playing surfaces and environmental conditions are checked for safety and best conditions.
- Write to state legislators and members of Congress, expressing concerns.
- Insist that research into youth sports injuries and their effects be undertaken immediately and be supported by tax dollars.
- Support a national registry of sport-related catastrophic injuries and fatalities to improve safety and participation.
- Demand that appropriate emergency action plans and safety and medical protocols and procedures are in place at every sporting event and facility.
- Educate players and others that there's a difference between pain and injury, and work to eliminate the culture of "playing through pain" without assessment.
- Ensure that both general and sport-specific safety education be a priority for every administrator, coach, parent and player.

For more information please visit [www.youthsportssafetyalliance.org](http://www.youthsportssafetyalliance.org)



## PATTI JAMES

Mother of Will James

Will's story: 16-year-old Will James, son of Patti and Bill James, passed out from heat stroke on Friday, August 13, 2010, after football practice at school in Little Rock, Ark. The school's athletic trainer, who is at all practices, tended to Will immediately and began the process of bringing down his core temperature while waiting for paramedics to arrive. Will was transferred to the closest hospital, intubated and stabilized. After stabilization, he was transferred to Arkansas Childrens Hospital.

Will's parents were in Canada at the time of his injury and were able to return to Arkansas the next morning. They were surprised to find their son in a medically induced coma that he stayed in for a week. Each day brought new surprises, including liver damage and kidney failure. However, Will was lucky. He spent three weeks in the hospital and his liver recovered, and after another three weeks of out-patient dialysis, his kidneys recovered.

His parents credit his recovery to the fast response of his athletic trainer and the excellent care he received in the hospital. Will has returned to school and is working out with his team to recover his strength. His parents are working with their state legislative committee and local athletic trainers' association to bring about changes to help avoid these types of injuries in the future.

ONE YEAR LATER:  
*A Report Card on the Youth Sports Safety Crisis*

Speakers



## MARJORIE J. ALBOHM, MS, ATC

President, National Athletic Trainers' Association;  
Director of Clinical Research and Fellowships, Ossur Americas

Marjorie Albohm travels internationally as director of Clinical Research and Fellowships for Ossur Americas and as president of the National Athletic Trainers' Association. She also serves on the board of directors for the Datalys Sports Injury Surveillance Center in Indianapolis. She is a highly regarded speaker, lecturing on a range of athletic training and sports medicine-related topics.

Albohm has received many honors, including the NATA Most Distinguished Athletic Trainer Award and the Tim Kerin Award for Excellence in Athletic Training. She was inducted into the NATA Hall of Fame in 1999 and is a former president of the NATA Research and Education Foundation. Guest appearances include the NBC "Today" show, CNN "House Call with Dr. Sanjay Gupta" and ESPN "Real Sports with Bryant Gumbel." As an expert in the field, she is quoted frequently in trade journals and consumer periodicals. She authored the book "Health Care and the Female Athlete" and co-authored "Your Injury – A Common Sense Guide to Sports Injuries" and "Reimbursement for Athletic Trainers."





## KELCI STRINGER

Founder and CEO, Korey Stringer Foundation

Kelci Stringer is a firm believer that your life's purpose will be revealed to you, even if it's in the midst of tragic circumstances. Her journey towards advocacy, education and prevention of sudden death in sports began on August 1, 2001, when her husband, NFL all-pro lineman Korey Stringer, died from complications of an exertional heat stroke at the age of 27, while practicing with the Minnesota Vikings.

Stringer met her husband when she was in college. The two were married for four years, and during that time, she gave birth to their son Kodie Drew. As a young widow and mother, she struggled to decide on the best way to honor her husband's memory, and ultimately established the Korey Stringer Foundation, a 501(c)3 organization. Through her foundation, Stringer was instrumental in developing a partnership with the National Football League, Gatorade and the University of Connecticut, Neag School of Education to form the Korey Stringer Institute. The university's Neag School of Education was chosen because of its reputation as a leader in the study of heat and hydration issues related to athletes and the physically active.



## **JULIE GILCHRIST, MD**

**Pediatrician and Medical Epidemiologist,  
National Center for Injury Prevention and Control,  
Centers for Disease Control and Prevention**

**D**r. Julie Gilchrist is a pediatrician and medical epidemiologist with the National Center for Injury Prevention and Control (NCIPC) at the Centers for Disease Control and Prevention. She has been at the CDC since 1997.

In her current work at the National Center for Injury Prevention and Control, Gilchrist is responsible for research and programs in drowning prevention and water safety promotion, and sports and recreation-related injury prevention, as well as other issues primarily affecting children: choking, suffocation, ingestions, dog bites, playground injuries, etc. She facilitated the development of the CDC's research agenda for prevention of injuries in sports, recreation and exercise and has been recognized for her efforts to establish a sports injury prevention program at the Centers for Disease Control and Prevention. Gilchrist has authored/co-authored more than 40 journal articles and five book chapters, and is an invited speaker both nationally and internationally. She has earned numerous awards for her efforts and accomplishments in research, communication and disaster response.



## R. DAWN COMSTOCK, PHD

Associate Professor,  
The Ohio State University College of Medicine;  
Research Faculty Member,  
Center for Injury Research and Policy,  
the Research Institute at Nationwide Children's Hospital

**D**r. Dawn Comstock is an associate professor at The Ohio State University College of Medicine, Department of Pediatrics and College of Public Health, Division of Epidemiology and a research faculty member at the Center for Injury Research and Policy (CIRP), the Research Institute at Nationwide Children's Hospital.

Comstock's research focus is the epidemiology of injury among the physically active, specifically the study of sports, recreation and leisure activity-related injuries among children and adolescents as well as the life-long health benefits associated with an active childhood. Comstock believes that to combat the epidemic of obesity in our country children must be encouraged to get up off the couch and participate in physically active sports, recreation and leisure activities. However, a certain endemic level of injury can be expected in any physical activity. The challenge is to monitor injury trends through surveillance; to investigate the etiology of preventable injuries; to develop, implement and evaluate protective interventions; and to responsibly report epidemiologic findings of injury research while promoting a physically active lifestyle for children and adolescents.



## JEFF MILLER

Vice President for Government Relations and Public Policy,  
National Football League

Jeff Miller has served as the head of the Washington office for the National Football League since 2008. As the vice president for Government Relations and Public Policy, his responsibilities include all federal and state legislative and regulatory initiatives.

Before opening the NFL's Washington office, Miller worked as the staff director and chief counsel for the Antitrust and Business Competition Subcommittee of the Senate Judiciary Committee and Sen. Herb Kohl from 2003 to 2008. He previously served as counsel on the same subcommittee. As staff director, Miller was responsible for legislation on all issues before the committee as well as investigations and hearings concerning a range of antitrust issues including mergers and anti-competitive business practices. He led investigations into mergers in the telecommunications, media, airline and pharmaceutical industries, among others.





## GERARD A. GIOIA, PHD

Chief of the Division of Pediatric Neuropsychology,  
Director, Safe Concussion Outcome, Recovery & Education  
(SCORE) Program, Children's National Medical Center;  
Associate Professor, Departments of Pediatrics and Psychiatry,  
George Washington University School of Medicine

**D**r. Gerard Gioia is a pediatric neuropsychologist and chief of the Division of Pediatric Neuropsychology at Children's National Medical Center, where he directs the Safe Concussion Outcome, Recovery & Education (SCORE) Program. He is an associate professor of pediatrics and psychiatry at the George Washington University School of Medicine. He has been the principal investigator of several multi-site CDC-funded research studies of mild TBI in children and adolescents. The focus of his research is the development and implementation of more effective methods and tools for early and ongoing evaluation of post-concussion neuropsychological functioning and symptoms.

Gioia was a panel member of the 2008 International Concussion in Sport Group Consensus meeting in Zurich, and is currently on the American Academy of Neurology Sports Concussion Guideline author panel. He is the team neuropsychologist for the NHL's Washington Capitals and a neuropsychology consultant for the Baltimore Ravens, the Howard County Public School System, Fairfax County Public Schools, multiple independent schools and numerous youth sports organizations in the Baltimore-Washington region.

### Gioia will speak about brain injury:

1. New rules are being implemented
2. Improve recognition, removal and protection
3. New tools becoming available
4. Careful individualized clinical assessment and tracking from time of injury
5. Neuropsychological testing, symptom assessment
6. Cognitive & physical exertional effects
7. Implement active treatment in home & school
8. ACE Care Plan as treatment guide
9. Symptom monitoring and activity management



## DOUGLAS J. CASA, PHD, ATC, FACSM, FNATA

Director, Athletic Training Education,  
Professor, Department of Kinesiology,  
Chief Operating Officer, Korey Stringer Institute,  
University of Connecticut

For the past 11 years, Dr. Douglas Casa has worked toward his goal of preventing sudden death in sport at the University of Connecticut, Neag School of Education, Department of Kinesiology. During this time he has published more than 100 peer-reviewed publications and presented more than 300 times on subjects related to exertional heat stroke, heat-related illnesses, preventing sudden death in sport and hydration.

Casa is the 2008 recipient of the medal for distinguished athletic training research from the National Athletic Trainers' Association. He was named a fellow of the National Athletic Trainers' Association in 2008 and received the Sayers "Bud" Miller Distinguished Educator Award from NATA in 2007. He has been a fellow of the American College of Sports Medicine since 2001. He has been a lead or co-author on numerous sports medicine position statements related to heat illness and hydration. He is also the editor of the forthcoming book, "Preventing Sudden Death During Sport and Physical Activity." Casa has worked with numerous media outlets across the country in discussing his research including the NBC "Today" show, ESPN, CNN, Sports Illustrated, USA Today, Wall Street Journal and the New York Times.

Casa, who was once a victim of heat illness, will be speaking about exertional heat stroke:

### Epidemiology of Exertional Heat Stroke

1. Increase in heat stroke deaths—five year block from 2005-2009 (worst five year period over last 35 years)
2. Reasons for increased causes

### Prevention of Exertional Heat Stroke

1. Intrinsic Factors (fitness, heat acclimatization, hydration status, prior history of heat illness, other medical problems, acute illness, etc.)
2. Extrinsic factors (state policies, hydration plan, athletic trainers present, environmental conditions, rest breaks, work demands, etc.)

### Recognition of Exertional Heat Stroke

1. Body temperature
2. CNS dysfunction
3. Others

### Treatment of Exertional Heat Stroke

1. Why rapid cooling
2. Cooling modalities
3. Cool first, transport second

### Return to Play Following Heat Stroke

1. What caused condition
2. Residual deficits
3. Phase back (athlete/teammates considerations)



## FRANCIS G. O'CONNOR, MD, MPH

Associate Professor, Uniformed Services University;  
Medical Director, Consortium on Health and Military Performance

### O'Connor will speak about sudden cardiac arrest:

#### Epidemiology of Sudden Cardiac Arrest in Athletes

1. Incidence
2. Common Etiologies

#### Strategies for Prevention

1. Preparticipation Screening
2. Electrocardiography
3. Automated Electronic Defibrillators
4. Emergency Action Plans
5. Athletic Trainers

#### Where We Go From Here

1. National Standards
2. Funded Research

**D**r. Francis O'Connor is associate professor at the Uniformed Services University and medical director for the Consortium on Health and Military Performance. He has been a leader in sports medicine education and research for the military for over 15 years and has authored over 50 articles in scientific journals and numerous book chapters/technical reports/health promotion resources for the military.

In addition, O'Connor is the editor of four texts on sports medicine including the "Textbook of Running Medicine" and "Sports Medicine for the Primary Care Physician 3rd Edition." He has been on the board of several leading organizations in sports medicine including the American College of Sports Medicine and the American Medical Athletic Association and is currently president of the American Medical Society of Sports Medicine. O'Connor is a colonel in the U.S. Army, and prior to his recent posting at Uniformed Services University in the Department of Military Medicine, he served one year as a command surgeon with Special Operations in the Middle East.



## SCOTT GALLOWAY, ATC, LAT

Head Athletic Trainer, DeSoto High School

### Galloway will speak about exertional sickling:

*In August 2002, tragedy struck Galloway's school and community when a 14-year old female basketball player fell victim to an exertional sickling death.*

1. Sick cell trait is an inherited condition of the oxygen-carrying protein, hemoglobin, in red blood cells. This genetic trait is generally benign, but during maximal exercise oxygen levels in muscles can decrease sufficiently to cause some of the red cells to change from the normal disk shape to a crescent or sickle shape.
2. In the past 11 years, exertional sickling has killed 16 athletes – 10 were NCAA division I football players, making SCT the leading cause of non-traumatic death in division I football in the past 10 years, more than double the cardiac incidents. Conditioning drills – not practice and not games – are the setting for exertional sickling deaths.
3. An SCT collapse can “mimic” other common distress symptoms, making it critical to identify at-risk students and educate and train staff and school personnel to respond to symptomatic students appropriately.
4. Students and parents have been overwhelmingly receptive to efforts to educate and encourage testing. For students in crisis situations related to SCT, education and interventions have proven to be life-changing.
5. Athletic activities at the secondary level are often a social measuring stick. Many students are not prepared for the physical and mental demands as they enter the athletic arena. Students fear social or personal repercussions of “not making the team” – so at this level they tend to under-report symptoms.
6. Screening as a solution?

Scott Galloway has served DeSoto High School in DeSoto, Texas, as head athletic trainer since 1999. In 2007, Galloway was selected to participate in the National Athletic Trainers' Association Task Force on Sick Cell Trait with the objective to encourage sickle trait testing of athletes at every level to ensure safe participation in sports. He has presented to the Sick Cell Foundation of Palm Beach, Florida, and the National Sick Cell Disease Association of America.

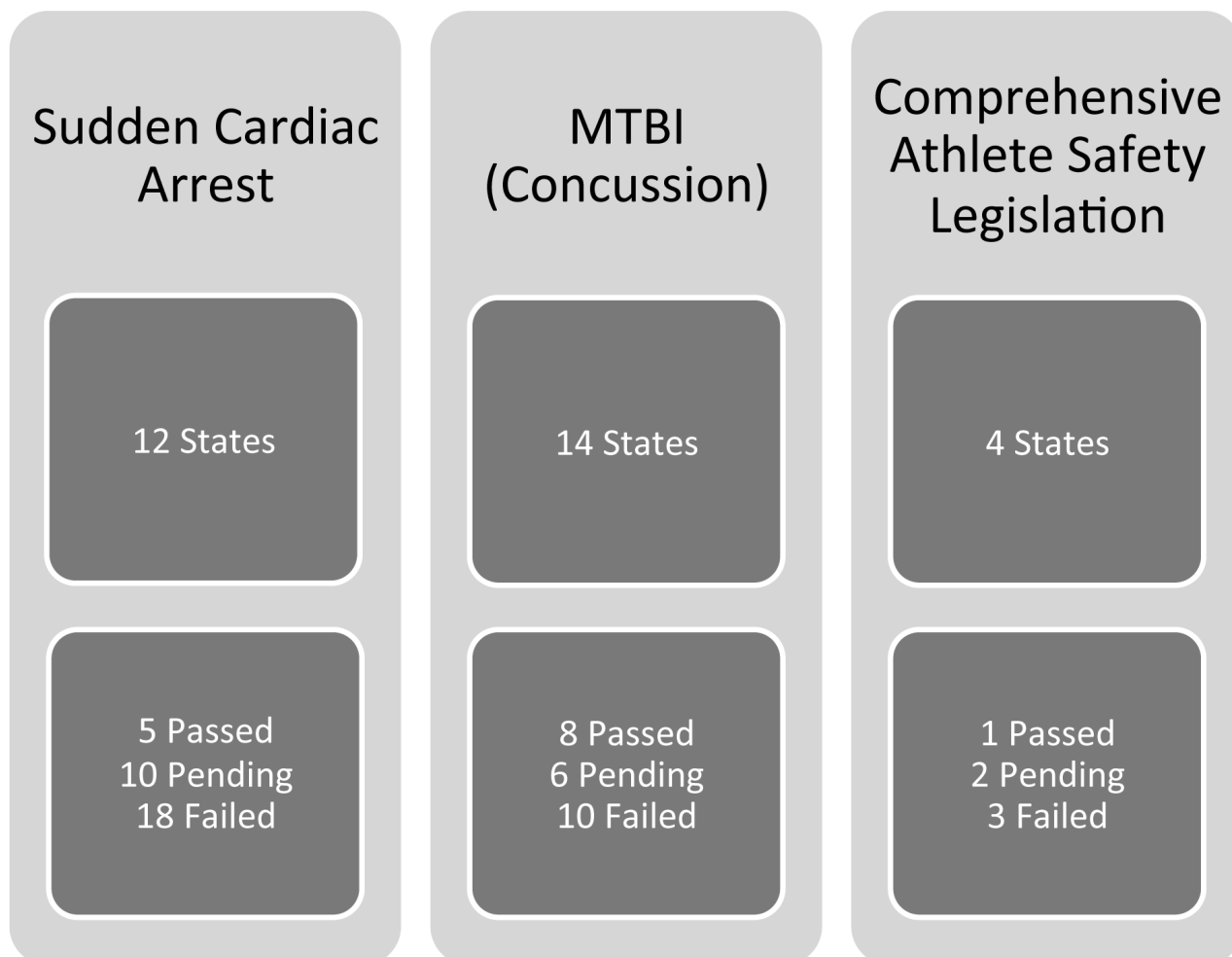
In 2008, Galloway was awarded the Southwest Athletic Trainers' Association John W. Harvey Humanitarian Award for positively impacting ethnic minority athletic trainers and/or physically active ethnic minority individuals. He currently serves as the vice chair and Region 4 director for the Texas State Athletic Trainers' Association.

# ONE YEAR LATER:

## *A Report Card on the Youth Sports Safety Crisis*

### Report Cards

The first Summit on Youth Sports Safety was held on January 12, 2010. Since that time, a lot has happened. Alliance members were invited to submit a “report card” on their organizational activities during 2010.



### FEDERAL LEGISLATIVE ACTIVITIES

H.R. 6172 “Protecting Student Athletes from Concussions Act”

H.R. 1347 “Concussion Treatment and Care Tools (ConTACT) Act” (Passed House by voice vote on September 30, 2010)

- House Education and Labor Committee hearing on H. R. 6172 -- September 23, 2010
- House Energy and Commerce Committee Subcommittee on Health field hearing on H.R. 6172 -- September 8, 2010
- House Education and Labor Committee, Healthy Families and Communities Subcommittee field hearing on “The Impact of Concussions on High School Athletes: The Local Perspective” on September 13, 2010
- House Judiciary Committee field hearing on “Legal Issues Relating to Football Head Injuries, Part II” on January 4, 2010
- House Judiciary Committee field forum on “Head Injuries and Other Sports Injuries in Youth, High School, College and Professional Football” on February 1, 2010
- GAO Report for the House Committee on Education and Labor on “Concussions in High School Sports” released on May 20, 2010
- House Energy and Commerce Committee hearing on “The Impact of Concussions on High School Sports” on May 20, 2010



# ADVOCATES *for* INJURED ATHLETES

October 8, 2010

Dear Ms. Becker-Doyle,

As a co-founder of Advocates for Injured Athletes (AIA), I am writing to inform you about the great strides AIA has made this past year relating to youth sports safety. Just over a year ago, my son Tommy, co-founder of AIA, was hit and fractured his neck in the remaining few minutes of his last high school lacrosse game. Tommy's life has since changed from that moment. Rather than starting college and playing lacrosse, Tommy spent an entire year rehabilitating and building AIA. The athletic trainer on the field that day saved Tommy's life by correctly treating his potentially catastrophic injury. This remarkable incident propelled Tommy and me to help protect other athletes as Tommy was on May 19, 2009.

In the last year Tommy and Riki Kirkhoff, the athletic trainer credited with saving his life have told their story countless times. Their story has been featured in the local San Diego press and nationally through the Scripps Howard News Network. Also, "Tommy's Story" has been featured in NATA (national athletic trainers association) magazine and featured on NBC in a special sports safety segment.

In addition, Tommy has made two trips to the California state legislature in Sacramento to call attention to the need for sports safety legislation. Specifically, to address the critical need for athletic trainers to be on every high school campus in America to protect athletes from catastrophic injuries. Tommy has partnered with Scripps Memorial Hospital La Jolla, CA to emphasize the need for sports safety education particularly, head injury or concussion. At the time of Tommy's career ending injury, he suffered his third major concussion. As a result Tommy can relate to athletes first hand about the dangers of traumatic brain injury. He has already spoken to hundreds of athletes as part of the concussion awareness campaign.

Advocates for Injured Athletes is a non-profit organization dedicated to keeping student athletes safe. Our primary goal is to make Certified Athletic Trainers available to all high school aged athletes to ensure that they receive quality, ongoing, preemptive advisement and care, ultimately to prevent catastrophic events and minimize the severity of sports related injuries. In addition, AIA provides sports safety education clinics and helps coaches develop emergency plans in the event of an injured athlete. AIA has developed a program, "Athletes Saving Athletes," designed to teach athletes the basics of concussion, head and neck injuries, sudden cardiac arrest, and heat illness. The program is designed to educate the athlete; hoping athletes can begin to save each other through education.

The last year has been a long difficult rehabilitation for Tommy. However, his injury has served as a very poignant and important lesson – that without the proper evaluation and treatment at the time of his injury, Tommy could have died and just become another sad statistic. Instead he is a valuable lesson in proper care and education.

Sincerely,

Beth Mallon  
Co-founder AIA



## **American Academy of Podiatric Sports Medicine**

The American Academy of Podiatric Sports Medicine recognizes the need to prevent and treat injuries, and the long-term impact these injuries have on people. With the initiative to encourage activity for diabetics and obese children, education and preventative measures are imperative to reach the objective of sports injury. Pain is an indication that there has been an injury and further participation could lead to long-term damage. The American Academy of Podiatric Medicine is committed to youth sports safety, and to provide foot/ankle care to injured athletes throughout the United States at the local, state, and national level. We strive to be a resource for parents, coaches and athletic trainers.

Statistics support the fact that musculoskeletal and neurological injuries are more prevalent than ever. Although our scope is foot and ankle, podiatric specialists should be cognizant of the manifestations of other injuries so the athlete can be referred to the appropriate specialists. Often, the first treating physician is the podiatric sports medicine specialist. The Academy has worked hard to ensure that athletes from every level of sport have access to a professional who will be able to diagnose and treat podiatric sports injuries.

The Academy has over one thousand members that are available to provide care to injured athletes. Many podiatrists participate in pre-participation physicals, in coordination with other specialists, to help identify any potential musculoskeletal or biomechanical abnormalities. Podiatrists are the most knowledgeable in athletic shoes, and actively participate in education of the weekend warrior or high-level athlete in appropriate shoe gear.

The American Academy of Podiatric Sports Medicine encourages research by our podiatry schools and fellowships that we support. Research is critical in identifying any factors that may contribute to, and /or reduce the risk of injury. Our members need to actively engage in dialogue with our state representatives so they are aware and support the call to action.





While there has been some movement and positive reports in cheerleading safety, there are still frustrations about how cheerleading is regulated across the country and improvements to be made.

This year the Center for Injury Research at Nationwide Children's Hospital<sup>1</sup> included cheerleading in its high school sports injury study<sup>2</sup>. The results show the overall injury rate in cheerleading is eighteenth out of the twenty sports and is twelfth in terms of head injury rate. It is important to note this is the first year of the study and more data is needed to verify these results. However, these figures are in line with older studies that also look at injuries per exposure<sup>3</sup>. More importantly, the detailed injury data collected by the CIR will help spot trends and determine the effectiveness of future educational programs and rule changes.

Recent catastrophic injury studies<sup>4</sup> demonstrate a downward trend in catastrophic injuries. We feel strongly that this is a result of our work with the NCAA and NFHS in removing certain skills from the basketball court surface as well as an increased awareness of the importance of risk management courses for cheerleading coaches. Twelve states athletic associations currently require the AACCA cheerleading safety course for their coaches and over twenty others strongly recommend it. The AACCA course is approved as a required course for NCAA cheerleading coaches, where catastrophic injury claims have been reduced from being 25% of all claims prior to initiating this program in 2006 to having no catastrophic claims since.

Beginning with the 2010-11 school year AACCA prohibited all basket tosses and double twisting dismounts for elementary, middle and junior high school teams. The goal of this change is to create more repetitions of lead-up skills at the lower grade levels and remove the pressure to perform these skills in competition.

Finally, the AACCA developed a Return to Play Protocol for concussions<sup>5</sup> in cheerleading including a specific stepwise return to participation program for cheerleading skill progressions.

However, more can be done to improve cheerleading safety. More state athletic associations need to recognize that cheerleading - while primarily a support group - is an athletic activity that requires a safe training environment, knowledgeable coaching, and access to athletic trainers like other sports. It is imperative that middle school administrators recognize the need for the additional restrictions and follow them to minimize the risk of serious injury at both the pre-high school and the high school level.

We have a positive outlook on the direction of cheerleading safety. We will continue to work with organizations across the spectrum of sports safety to improve cheerleading safety and we see only positive changes ahead.

Best Regards,

Jim Lord  
Executive Director, AACCA

1,2,3,4,5 – For citations, please visit our website at <http://www.aacca.org/sportssafetyalliance>



AMERICAN CHIROPRACTIC ASSOCIATION'S COUNCIL ON SPORTS INJURIES AND PHYSICAL FITNESS  
1701 Clarendon Blvd. Arlington, VA 22209 Phone: 800-261-1495 [www.acasc.org](http://www.acasc.org)

Dear Ms. Satlof,

The membership of the American Chiropractic Association Council on Sports Injuries and Physical Fitness is proud to be an alliance member of the Youth Sports Safety Alliance. We recognize and support your efforts in this public initiative to bring awareness to the public as well as to providers in proper, evidence based pre- participation examination and concussion management. Recognizing the need, the ACA Council on Sports Injuries and Physical Fitness trained our sports physicians at our Annual Sports Sciences Symposium and General Membership Meeting on pre-participation evaluations and the assessment, recognition, and management of concussive injuries. The training was well received and attended by over 180 doctors and chiropractic students.

The ACA Sports Council on Sports Injuries and Physical Fitness is committed to continuing the effort to educate, train and support the mission of the Youth Sports Safety Alliance.

Sincerely,

Guillermo Bermudez, D.C., .CCSP  
President ACASC.



# The American Orthopaedic Society for Sports Medicine

*A world leader in sports medicine education, research, communication, and fellowship.*

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*American Orthopaedic Society for Sports Medicine*

Report on

## **Sports Trauma and Overuse Prevention (STOP Sports Injuries) Campaign**

The American Orthopaedic Society for Sports Medicine launched the Sports Trauma and Overuse Prevention (STOP) program in the spring of 2010 to provide a source of authoritative, accessible and free information so health care providers can educate parents, coaches and athletes about youth sports safety. The National Athletic Trainers' Association (NATA) and others in the health care community have embraced the program, with 13 national health organizations, 17 medical institutions and 50 sports medicine practices, as well as a growing number of sports leagues and recreation program, joining the campaign.

Thousands of parents and coaches visit the Campaign website ([www.STOPSportsInjuries.org](http://www.STOPSportsInjuries.org)) and Facebook page on a regular basis to get sport specific information on 19 different sports, including:

Baseball	Rowing
Basketball	Running
Cheerleading	Skiing
Dancing	Soccer
Football	Softball
Golf	Swimming
Gymnastics	Tennis
Hockey	Volleyball
Lacrosse	Weightlifting
Martial Arts	

STOP Sports Injuries also is a source of information for a range of general sports topics that transcend many athletic activities, including:

Concussion	Overuse
Female Athlete Issues	Psychological Issues Related to Injury
Heat Illness	Return to Play after Injury

Public awareness about youth sports injuries has significantly improved through public education efforts that have reached millions of Americans since the Campaign was launch in April 2010. 900 television stations throughout the country have carried PSAs about the STOP Sports Injuries program, and 32 prominent athletes and business leaders have agreed to lend their support for the program through its Council of Champions. Thousands of articles have appeared in a variety of print media, from major publications such as the New York Times and Sports Illustrated to local community newspapers.

In spite of the remarkable success, the challenge remains for educating the public and making youth sports safety a part of our collective consciousness. Educating every young athlete, their parents and their coaches - in all sporting activities and in all seasons – will require a collective, ongoing effort. We applaud the NATA for helping lead the charge.

Robert A. Stanton, MD  
AOSSM President



# AOASM

AMERICAN OSTEOPATHIC ACADEMY OF SPORTS MEDICINE

2424 American Lane • Madison, WI 53704-3102 USA  
1-608-443-2477 • Fax: 1-608-443-2474 • E-Mail: [info@aoasm.org](mailto:info@aoasm.org)

Ellen Satlof  
PR Manager  
National Athletic Trainers' Association  
2952 Stemmons Freeway  
Dallas, Texas 75247

October 8, 2010

***Monitoring the head impact profile of the Virginia Tech football players has been a full-time project for Gunnar Brolinson DO, FAOASM, the head doctor for the Virginia Tech football team, since 2003. The Hokies were the first college team to put in place the Head Impact Telemetry (HIT) system.***

***Games and practices have doubled as a brain laboratory for Dr. Brolinson and his research team. The technology consists of 6 small sensors (accelerometers) placed inside players' helmets. A device on the sideline, the size of a small footlocker, receives data from the sensors and then displays information on a laptop computer screen. Dr. Brolinson monitors a screen, which depicts a computer-generated head, and colored arrows on different parts of the head that show the location and magnitude of the impact. He and his team are working on understanding the relationship between concussion and the head impact profile.***

***The HIT system, as it's called, is expensive. Dr. Brolinson says the cost is about \$60,000 for a college football team. An individual helmet is \$1,000. The price is one of the reasons the system's use is limited at this point to a handful of colleges, universities and high schools. A less expensive system is currently being developed. Its use has established peak and average hit counts for the players. The statistics show lineman and linebackers can log anywhere from 1,000 to 1,500 head hits per season. However Dr. Brolinson is cautious about drawing conclusions from the numbers alone, as the research is still very young.***

***"We have no how many hits is a bad number," he says. "That's the importance of doing this research to further characterize the head impact profile and carry it forward. We would like to be able to further quantify the impact biomechanics related to mild traumatic brain injury."***

***Dr. Brolinson says he hopes in three to five years, if his project continues, he will have a better idea of what numbers constitute a danger zone for football players.***

***Yours in Good Health,***

***Phillip Zinni III, DO, FAOASM, ATC  
President;***

***AMERICAN OSTEOPATHIC ACADEMY OF SPORTS MEDICINE  
The Oldest Primary Care Based Sports Medicine Specialty  
[JockOccDoc@Hotmail.com](mailto:JockOccDoc@Hotmail.com) T: 209-324-2255***



The **Best** Approach to  
**Concussion**  
**Management**

The issue of concussion has recently been rightfully elevated to the level of national scrutiny that it deserved. This attention is, in our opinion, both well deserved and long overdue. As is now recognized, sports related brain injury is a very frequent event that deserves the attention it is finally receiving.

The management of concussion in athletes is finally being recognized as an issue that is deserving of all of our attention and should include the implementation of a multi-disciplinary approach to diagnosis and eventual return to play following injury. The recent passage of state-wide standards for return to play is a particularly important step in the evolution of concussion management standards, ImPACT is proud to have played a role in the advancement of the standard of care in the diagnosis and treatment of concussion in athletes. As is now well-recognized, neurocognitive assessment represents an important element of the concussion management process.

We at ImPACT are completely supportive of the national efforts to improve concussion education, diagnosis and treatment. Furthermore, we look forward to developing reliable and valid tools for the identification and measurement of post concussive cognitive difficulties.

A handwritten signature in black ink, appearing to read "Mark R. Lovell".

Mark R. Lovell, Ph.D.  
Chairman, ImPACT Applications  
Professor of Orthopaedic and Neurological Surgery  
Director, UPMC Sports Concussion Program



## **Education for the Prevention of Sudden Cardiac Arrest and Death in Youth and Athletes**

**Introduction:** The KEN Heart Foundation has had a vital role helping local communities become prepared for cardiac emergencies for the past ten years. It was founded by family and friends of 17 year old Ken Derminer after he suffered sudden cardiac death from an undiagnosed heart condition at a local high school football minicamp.

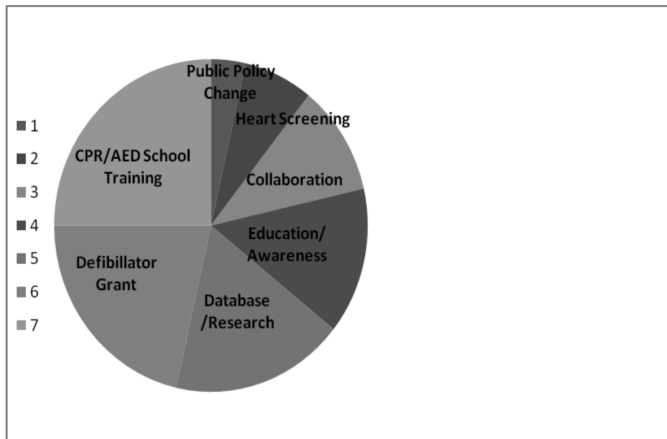
**Cause:** Sudden Cardiac Arrest can strike anyone, anywhere—and when it does; a victim's chance of survival depends on the people around them. Each year, hundreds, if not thousands of those suffering Sudden Cardiac Arrest and death are children and young adults. It is often referred to as a rare occurrence when in fact it is the leading cause of medical fatalities on school property and for student athletes. It is preventable.

**Obstacles:** Medical debate surrounding heart screenings and the occurrence of incidents.

**Campaign:** We can do more, striving to do better!

### **2010 Projects:**

- 1 None in 2010
- 2 Launching 2011
- 3 5 National Memberships
- 4 3 Exhibit Events
- 5 Ongoing-150-200 National Cases Annually
- 6 5 Schools and Youth Organizations
- 7 1333 Students-17 Sessions-91.4 Pass rate



**We are proud to be a part of the Youth Sports Safety Alliance!**



P. O. Box 237 Geneva, Ohio 44041 866-317-4034 [mail@kenheart.org](mailto:mail@kenheart.org) [www.kenheart.org](http://www.kenheart.org)



## Korey Stringer Institute

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Neag School of Education  
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Email: [ksi@uconn.edu](mailto:ksi@uconn.edu)  
[www.ksi.uconn.edu](http://www.ksi.uconn.edu)

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KELCI STRINGER  
**Chief Operating Officer**  
DOUGLAS CASA, PhD, ATC

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**Heat Stroke Consultants**  
*University of Connecticut:*  
REBECCA STEARNS, MA, ATC-*LEAD*  
JULIE DEMARTINI, MA, ATC  
KELLY PAGNOTTA, MA, ATC



The Korey Stringer Institute had its official kick-off at NFL Draft in the Radio City Hall on April 23, 2010. This organization began through the work of a widowed wife whose goal was to carry on the legacy of her late husband. In August 2001 Korey Stringer, a Minnesota Vikings offensive lineman, died from exertional heat stroke. Since the time of Korey's death, Korey's wife, Kelci Stringer, has worked tirelessly to develop a heat stroke prevention institute to honor her husband's legacy. In 2010, the Korey Stringer Institute (KSI) partnered with the NFL and Gatorade to officially open on the University of Connecticut's campus within the Neag School of Education (Storrs, CT).

Following our opening there has been a considerable amount of media attention and promotion. KSI was present at the 2010 Trade Show of the National Athletic Trainer's Association in Philadelphia, PA. Examples of articles about KSI have appeared in University of Connecticut Summer Alumni Magazine, NY Daily News, The Washington Post, ABC News, NY Post, NY Times, ESPN and USA Today.

KSI has also designed a comprehensive website that is available to the public. Highlights include links to over 100 heat and hydration references. These references are divided into 16 categories to include all areas of heat and hydration information. Included are also links to all 50 state guidelines on heat acclimatization protocols, and information for specific groups ([ksi.uconn.edu](http://ksi.uconn.edu)). The website also contains information focused towards different audiences (parents, coaches, athletes, athletic trainers, athletic directors, heat stroke victims etc).

KSI has begun its educational endeavors as well. This summer, KSI presented a webinar to 150 certified athletic trainers through efforts made by the National Athletic Trainer's Association (August 2010). KSI was featured in prominent presentations at the American College of Sports Medicine annual meeting (June 2010), Johnson & Johnson Symposium at the National Athletic Trainers Association annual meeting (June 2010), and Wilderness Medical Associates (September 2010). The KSI offices at the University of Connecticut are now also fully capable of holding distance education seminars with full video conferencing capabilities.

KSI has provided consultations to several organizations for improvements in heat illness policies. These include: the 2010-2011 NCAA sports medicine handbook, NASA physicians and their heat policies for training programs, the CDC, the United States Army (August 2010) and has reviewed other University's heat illness policies. In addition, KSI worked with the National Athletic Trainers' Association to help update their heat illness educational competencies.

Lastly, while KSI has had a great start thus far, sadly September alone had three exertional heat stroke deaths in athletics within the United States. We are continuing to work hard to prevent sudden death in sport however our work is far from over. With continued support from our partners and donors and efforts from our team and those assisting us, we *will* lower the deaths and catastrophic injuries in sport.

Sincerely,

Douglas J. Casa, PhD, ATC, FACSM, FNATA  
Chief Operating Officer  
Korey Stringer Institute



Gifts to support The Korey Stringer Institute may be made through the UConn Foundation, Inc. a tax-exempt 501(c)(3) corporation dedicated exclusively to benefit the University, and are deductible for federal income tax purposes to the extent allowable by law. All gifts will be used to benefit The Korey Stringer Institute and its mission.



The National Athletic Trainers' Association (NATA) views the athletic training profession as the leading advocate and expert in the fight to protect America's young athletes from catastrophic injury and illnesses or death on the playing field. As medical professionals who work with these players season after season, athletic trainers have a 60+ year tradition of proactively seeking newer and better ways to keep them safe and address problems when they arise.

Many health care, parent, coach, and sports organizations are diligently working on a wide spectrum of solutions to various aspects of the athlete safety crisis. We believe it is our duty to serve as a catalyst and organizer to bring these groups together to effect rules and laws, increase awareness and even affect cultural attitudes toward injuries.

In 2010, we:

- Initiated the Youth Sports Safety Alliance ([www.youthsportssafetyalliance.org](http://www.youthsportssafetyalliance.org))
- Hosted the Summit on the Youth Sports Safety Crisis (Sacramento, Calif., January 12)
- Created NATA NATION™, a national movement to improve the health and safety of athletes through research projects and fundraising to support those projects. One of the first initiatives is the Secondary School Injury Surveillance & Outcomes System.
- Garnered national media attention, resulting in extensive coverage reaching millions of Americans to raise awareness of the potential for catastrophic sports injuries.
- Lobbied 21 state legislatures for enactment of sports safety laws.
- Testified in Congressional hearings on concussion in secondary school sports. Helped to draft language for H.R. 6172 "Protecting Student Athletes from Concussions Act of 2010." Supported H.R. 1347 "Concussion Treatment and Care Tools (ConTACT) Act"
- Released new Position Statement on Skin Diseases in Athletics. Supported H.R. 2400 "Strategies to Address Antimicrobial Resistance (STAAR) Act"—to address the increasing numbers of serious skin infections.
- Reminded all state athletic associations of the importance of appropriate medical care and supervision of secondary school athletes, and asked them to include athletic trainers as approved health care providers in making return-to-play-after-concussion decisions.
- Developed an online and in-person sports safety course for youth coaches.
- Joined and supported the Pre-participation Evaluation Coalition and encouraged the use of electronic PPEs.
- Continued to educate more than 33,000 athletic trainers and other sports medicine professionals on the prevention, assessment, diagnosis and rehabilitation of sport injuries and illnesses.
- Participated and supported the USOC Working Group for Safe Training Environments.
- Joined as a founding member and supporter of the STOP Sports Injuries Campaign.
- Supported critical research through the NATA Research and Education Foundation.

Frustrations:

Many state legislatures failed to act to protect young athletes. Only 21 states considered safety measures, and relatively few meaningful laws were enacted.



# Overview: Cheerleading Safety in 2010

**The skill sets of cheerleading: acrobatics and tumbling has evolved from just a sideline activity to a sport of its own. It is the fastest growing sport in the US and as a new sport is born so is the opportunity to put safety first.**

In 2009, cheerleading is a gymnastic activity, and why it is still called cheerleading is not quite clear.

-Dr. Frederick Mueller. Journal Athletic Training  
2009 Nov-Dec; 44(6): 565-566.

Cheerleading is part entertainment, part leadership and part sport.

-Kimberly Archie, Founder

Injuries alone do not make cheerleading a sport, but cheerleading not being a sport causes more injuries.

-- Michael Minix M.D. Founder of CAPPAA and Athlete Abuse Prevention Expert

I was never a cheerleader but luckily my mom was, and she's been giving me some great tips. It's hard but I'm really enjoying it and it's a great way to keep fit. In fact I've never been so fit!

-Ashley Tisdale, cast member of HellCats about competitive college cheerleading premiering on the CW Sept. 8th

A sport governs the sport. The activity does not govern the sport.

--Herb Appenzeller, editor of From Gym to the Jury, member of 8 sports hall of fame, author 24 books on risk management in sport

Being a feminist should mean not limiting women and putting them into any slot. What am I old fashioned if I happen to like staying at home raising my children? I want my daughter to be able to be a CEO or a stay at home mom, the idea is that she is able to choose what makes her happy. That's what I think a feminist should be. She is a competitive cheerleader and she works just as hard on that court as my son does on the football field.

- Carol Crossland, Texas Native & Cheer Mom

Did you know... 71% of catastrophically injured female college athletes are cheerleaders?

--Mueller F. O., Cantu R. C. Twenty-Sixth Annual Report for Catastrophic Sports Injury Research. Fall 1982-Spring 2008. Chapel Hill, NC: National Center for Catastrophic Sports Injury Research; 2008.

## Mission

The National Cheer Safety Foundation is a cheerleading safety organization dedicated to raising the bar in sport safety to reduce injury, disability, and death from the sport of cheerleading; and assist all who are affected by catastrophic cheerleading injuries.

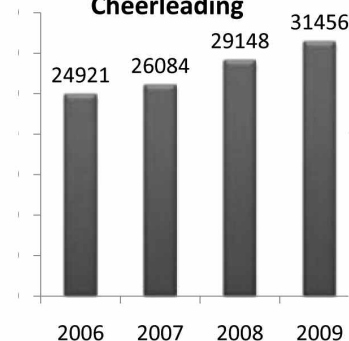
The National Cheer Safety Foundation's philosophy is to bridge the gap between the sport of cheerleading, the sports sciences, and the legal risk management arena. The National Cheer Safety Foundation's mission is to provide the sport of cheerleading with the research and educational services needed to help maximize the enjoyment and safety of all who participate within the sport.

The National Cheer Safety Foundation's Panel of Experts support the foundation by prudently disseminating proven research information and providing proper resources to help educate all who are responsible for implementing safe cheerleading programs. This information is essential to all who are responsible for training, coaching and administrating the sport of cheerleading.

## Where Do We Go From Here?

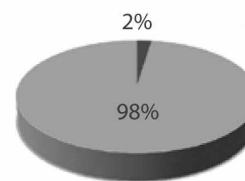
- Change the name to "Acrobatics andTumbling"
- Recognize the acrobatics and tumbling skill sets
- Support & endorse NCATA
- Educate the media, policy makers, and the general public on the differences between sideline and acrobatics and tumbling
- Promote professional sport coaching and education
- Contact the Office of Civil Rights to support "Acrobatics and Tumbling" as a sport worthy of Title IX protection. They may be contacted by email at [OCRMail@hhs.gov](mailto:OCRMail@hhs.gov).

**# of ER Visits for Cheerleading**



**26% Increase in 3 years!**

U.S. Consumer Product Safety Commission. NEISS All Injury Program. Washington, DC: 2009.



Only 2% of injuries happen at home. 98% happen at school, as part of a league or at a gym.

U.S. Consumer Product Safety Commission. NEISS All Injury Program. Washington, DC: 2009.

*Cheering for your life.*

**NATIONAL  
CHEERSAFETY  
FOUNDATION**



**Sally S. Johnson, CSA**  
Executive Director  
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Website: [www.ncys.org](http://www.ncys.org)

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National Amateur Baseball Federation

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**Jon Butler**  
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**Secretary**  
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Amateur Athletic Union

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Locker 81/SmartGivingCards

**Director**  
**Mike Millay**  
Disney Sports Attractions

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## MISSION STATEMENT

The National Council of Youth Sports represents the youth sports industry by advancing the values of participation, and educating and developing leaders.

*"A Unified Voice for Youth Sports."*

## VISION

To enhance the youth sports experience in America.

Dear Youth Sports Safety Alliance colleagues:

The National Council of Youth Sports (NCYS) commends the National Athletic Trainers Association (NATA) for its leadership in addressing youth sports-related musculoskeletal and neurological injuries (concussion, heat illness, and ACL injuries). NCYS is proud to join the Alliance supporting the NATA's initiative to take action in raising the awareness of the causes, the symptoms, the treatment, and most importantly the prevention of sports injuries.

Established in 1979, the National Council of Youth Sports membership represents more than 60,000,000 registered participants/ 44,000,000 actual boys and girls in organized youth sports programs. The NCYS members are the gatekeepers, the key decision-makers, the power of influence and behavior, and the advocates for valued amateur youth sports participation.

Overuse injuries from excessive training is unacceptable and ruins the exercise experience—children need to grow into their sport or activity preventing unnecessary injuries. Children are pushing or being pushed too hard, too soon, too much, too quick and being hurt physically and emotionally. Sports and exercise should be fun for everyone involved.

As an industry we need to partner together to create appropriate educational training materials and messaging for the administrators, coaches, parents, and athletes. We need to better understand sport skill development and how it matures to the next level while encouraging attainable goals and realistic accomplishment. And we need to be certain sports equipment; fields and facilities are all meeting the approved safety standards.

The reason the NCYS exists is to enhance the youth sports experience in America for today and for generations to come. The NCYS is a portal to and a united voice for the youth sports/youth serving industry. Clearly the safety of America's young athletes must be at the core of what we do in our youth sports programs. We have a responsibility to promote lifetime physical activity with proper training and age-appropriate competition in a safe environment.

The NCYS is committed to working together with the NATA as well as our members, the media, the government, corporate America and the leaders in and around our industry to achieve and maintain the highest standards of quality and integrity for a safe and positive youth sports experience.

Yours truly for the kids,

*Sally S. Johnson*

Sally S. Johnson, CSA  
Executive Director  
National Council of Youth Sports



# Parent Heart Watch®

PROTECTING YOUTH FROM SUDDEN CARDIAC ARREST

## One Year Later: A Report Card on the Youth Sports Safety Crisis

### ACCOMPLISHMENTS: (23% of membership reporting)

- Number of People Trained CPR/AED 860
- Number of Youth Screened with ECG and/or Echo 2,413
- Number of AEDs placed\* 124
- Approximate Population served by AEDs 167,600

*\*includes schools, athletic facilities, health clubs, and other youth serving organizations*

**Collaborations:** PHW has established working relationships with NATA and the Sudden Cardiac Arrest Coalition. In addition, PHW is working closely with the National Center for Child Death Review, National Institutes of Health, Centers for Disease Control and leading physicians throughout the country on establishing a systematic and mandatory registry for Sudden Cardiac Death (SCD) in youth.

**You Make the Difference Poster! Campaign:** Over 410,000 people were exposed to the “Warning Signs and Symptoms of a Heart Condition” and the “Cardiac Chain of Survival” posters distributed by PHW members in 24 states.

### FRUSTRATIONS:

Lack of availability of AEDs at all school sporting events and practices: While we’ve made progress, there are still many schools throughout the country that don’t have AEDs, trained CPR/AED personnel, and an Emergency Action Plan should a sudden cardiac arrest occur.

Lack of funding to support AED availability in elementary and secondary schools: While multiple states have AED mandates, few are funded.

Lack of a systematic and mandatory registry for SCD in all youth, athletes and non-athletes alike.

**ABOUT PARENT HEART WATCH:** Parent Heart Watch (PHW) is the national voice solely dedicated to protecting youth from Sudden Cardiac Arrest and preventable Sudden Cardiac Death. PHW leads and empowers others by sharing information, educating and advocating for change.

Be **Informed!**  
Be **Connected!**  
Be **Empowered!**

[www.ParentHeartWatch.org](http://www.ParentHeartWatch.org)



### **Pop Warner Little Scholars, Inc.**

586 Middletown Blvd. Suite C-100 ▪ Langhorne ▪ PA ▪ 19047

Phone: 215-752-2691 ▪ Fax: 215-752-2879

[www.popwarner.com](http://www.popwarner.com)



With the recent increase in media attention, governmental Hearings, new information and research on concussions, Pop Warner Little Scholars, Inc. has revised its rules regarding removal from play, and return to play, for athletes with possible and/or suspected concussions.

Below is an overview of the changes, which were issued on September 28, 2010 to be enforced immediately.

#### **Pop Warner Little Scholars Inc. 2010 In Season Safety Rule Amendment Re: Concussions**

- A participant who is suspected of sustaining a concussion or a head injury in a practice, game or competition shall be removed from practice, play or competition at that time based on evaluation and determination by the Head Coach.
- Any Pop Warner participant who has been removed from practice, play or competition due to a head injury or suspected concussion may not return to Pop Warner activities until the participant has been evaluated by a currently licensed medical professional trained in the evaluation and management of concussions and receives written clearance to return to play from that licensed practitioner.
- Pop Warner recommends that all decisions be made in the best interest of the children and that when any doubt exists as to the health of the participants, they sit out.
- When a participant is rendered unconscious or apparently unconscious, that participant shall not be permitted to resume participation until that participant has been evaluated by a licensed medical professional trained in the evaluation and management of concussions and receives written clearance to return to play from that licensed practitioner.





## ***Successes, Challenges and Opportunities in Youth Sports Safety***

**More** than 3.5 million children and adolescents receive medical aid due to sports-related injuries and illnesses each year. Given that, Safe Kids USA set out to reach youth coaches, parents and athletes in order to provide need-to-know information and practical tools that would reduce injuries, emergencies and illnesses in America's youth athletes. In response, the spring and summer of 2010, Safe Kids USA, along with its coalition network and partners, planned and executed more than 63 sports injury prevention clinics throughout the country, and reached more than 6,400 parents, youth coaches, athletes and major stakeholders in the youth sports arena. What is equally encouraging is that the Safe Kids coalitions who were recipients of the sports grants, indicated plans for conducting more sports safety clinics to meet the ensuing demand. The success of this program could not have been accomplished without incredible partners such as The National Athletic Trainers' Association. Valuable lessons learned from the sports clinics also illuminated the challenges we face in youth sports today:

"Coaches are happy to listen to the information, but this is a population that is still somewhat resistant to this information. They see a concussion as an injury that will keep a player out indefinitely. That said, in terms of their responsibility for other's children, they were happy to listen to the information provided." – Safe Kids Coordinator

This observation, made by a recipient of the Sports Safety Program grant, underscores both the successes and challenges in youth sports today. A fair number of coaches (and parents) want their athletes and teams to win—first and foremost. However, coaches appear to be developing a greater appreciation for their *role* and *responsibility* in reducing athletes' risk of sports-related injuries and illness. This is a key opportunity for improving youth coaches' and parents' knowledge base and practical skills in the way of injury prevention.

Furthermore, concerted efforts need to be made that help coaches and parents realize that their athlete cannot meet his/her full potential in sports, if preventative knowledge and action plans are not a critical part of their game plan. This means a paradigm shift is needed, which requires activating the youth sports community through various strategies. For example, increased participation in effective programs, like sports injury prevention clinics, will help provide practical training for coaches and parents of young athletes. Safe Kids' findings from the sports clinics revealed substantial knowledge gaps among youth coaches and parents with regards to life-threatening injuries and illnesses in sports, mainly heat illness and concussions. In fact, program participants stated that the clinics were their first exposure to many of the concussion-related issues presented (e.g. second impact injuries; return-to-play guidelines). Nonetheless, many indicated a desire to have a deeper understanding and obtain more training to prevent these types of sports-related injuries and emergencies.

Developing and conducting sports injury prevention clinics, with comprehensive evaluations in place, can help bridge the knowledge gap, heighten risk awareness, change coaching practices and encourage more parents and coaches to serve as role models for their young athletes. As Safe Kids USA learns more about the youth sporting culture and gains more experience in working with key partners, it becomes clear that this challenge is worth meeting. Success will not come without dedicated partners and a commitment towards innovative strategies that reach those who are responsible for nurturing not only skilled athletes, but healthy and injury-free ones as well.





Oct. 8, 2010

A proud member of the Youth Sports Safety Alliance, SportsConcussions.org addresses the need within our communities for increased concussion awareness. According to the CDC, approximately 3.8 million sports-and recreation-related concussions occur annually in those under the age of 18, of which 150,000 seek immediate treatment in the ER. The rest go home.

It is now readily apparent that the long and short-term prognosis for recovery from concussion can be greatly influenced by proper management of the injury. Due to the lack of education on the part of parents, coaches, athletes, and others, many athletes are returning to play before their brains are fully healed. In rare cases this can be fatal, in many more it leads to prolonged symptoms and a fundamental change in the day-to-day lives of countless children and young adults. Concussion-related fatigue, headaches, and cognitive impairment affect not only grades, but entire futures.

On January 7, 2010, the [www.SportsConcussions.org](http://www.SportsConcussions.org) website was launched to provide education, tools, ideas, and other resources for those interested in the topic. Our initial goal was to reach 100,000 people our first year, and we are on track to accomplish that. We bring information to our visitors from the national stage as well as links to state and local resources. The site also offers guidance for coaches seeking concussion training, templates for school districts to use in the development of their own management programs, and checklists for parents who may have a concussed child at home.

We are honored to have assembled an Advisory Board which includes Dr. Stanley Herring, Co-Medical Director of the Seattle Sports Concussion Program and part of the NFL's concussion committee, Dr. Gerard Gioia, Chief, Div. of Pediatric Neuropsychology, Children's National Medical Center, Richard Adler, BIAWA Board Chairman, Chris Nowinski, President of Sports Legacy Institute, involved with NFL/autopsy research, and Mike Ryan, head athletic trainer for the Jacksonville Jaguars, among others.

Our goal next year is to reach 250,000 people via the website, and open offices in several regions around the country, helping communities, particularly those in rural areas, to establish systems of care for concussed athletes. We also plan to launch an additional four satellite websites in our effort to increase awareness. We are dedicated, through these websites, to continue to provide a platform for the CDC, medical professionals, state officials, and others whose desire it is to help educate the parents, coaches, and athletes on the field.

Sincerely yours,

Jean Rickerson, President and Founder/ [www.SportsConcussions.org](http://www.SportsConcussions.org)



As the national governing body for lacrosse, US Lacrosse ([www.uslacrosse.org](http://www.uslacrosse.org)) has made a significant commitment to lead sport-specific safety education and research efforts since its formation in 1998. Through its Sports Science & Safety Committee, which is comprised of many prominent professionals from various medical specialties and representatives from a number of multi-sport organizations, US Lacrosse has developed strategic alliances with numerous organizations, conducted unprecedented injury research, provided constant review of issues affecting player safety, and provided growing information resources to its more than 320,000 members in 64 chapters throughout the United States. The committee has also issued positions statements on a number of issues related to lacrosse safety. Additionally, US Lacrosse has invested significantly to establish the sport's first standardized coaching and officiating education curricula, including both robust online resources and one-on-one clinic sessions, which have formally trained thousands of coaches and officials.

Lacrosse-specific research and related initiatives led or supported by US Lacrosse include:

- Descriptive epidemiology of scholastic lacrosse injuries
- Head, face and eye injuries in scholastic and collegiate lacrosse
- Support for the development of a mechanical model for commotio cordis research
- Risks and mechanisms of severe injuries among youth, secondary school, collegiate and post-collegiate lacrosse players using insurance claims data
- Trends in high school lacrosse injuries
- Epidemiology of concussion in boys' and girls' high school lacrosse players
- Trends in sports-related concussion incidence at the high school level, 1998-2007
- Evaluation of the women's lacrosse protective eyewear mandate
- Epidemiology of lacrosse injuries among youth players

US Lacrosse strategic alliances include:

- Acompora Foundation – Commotio Cordis education
- American College of Sports Medicine – Concussion Education
- Cardiac Science – AED awareness and grants
- Centers for Disease Control & Prevention – concussion education
- Fairfax County (VA) Public Schools – high school injury data collection
- MedStar Sports Health – injury data collection and research
- NATA – Youth Sports Safety Alliance
- NFHS – high school injury data collection
- OSSM – Sports Trauma & Overuse Prevention (STOP)
- USADA – Supplement Safety Now
- USOC – Working Group of Safe Training Environments

Lacrosse has become one of the fastest-growing sports in the nation thanks to the programs, resources and leadership US Lacrosse has provided. However, this dramatic growth has also created the need for greater resources and collaboration in order to maintain player safety and assure a high quality of experience for all participants. Among the biggest challenges US Lacrosse faces in this regard:

- Convincing youth leagues and state high school associations that requiring their coaches and officials to achieve national sport-specific education standards is essential to a safe playing experience.
- Reaching parents of young athletes to educate them about what they should expect from a best-practice youth sports organization and encouraging them to be more involved consumers in the sports experiences provided to their children.





ONE YEAR LATER:  
*A Report Card on the Youth Sports Safety Crisis*

Resources

# STATISTICS ON YOUTH SPORT SAFETY

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- There were 120 sports-related deaths in 2008-2009, in 33 states.<sup>1</sup>
- Approximately 8,000 children are treated in emergency rooms each day for sports-related injuries.<sup>2</sup>
- Among children, those aged 15-17 experience the highest emergency room visits for sports injuries.<sup>2</sup>
- Rates of sports injury visits to ERs were highest in remote rural settings.<sup>2</sup>
- 50 percent of “second impact syndrome” incidents – brain injury caused from a premature return to activity after suffering initial injury (concussion) – result in death.<sup>3</sup>
- The Centers for Disease Control and Prevention reports that high school athletes suffer 2 million injuries, 500,000 doctor visits and 30,000 hospitalizations each year.
- Female high school soccer athletes suffer almost 40 percent more concussions than males (29,000 annually).<sup>4</sup>
- Female high school basketball players suffer 240 percent more concussions than males (13,000).<sup>4</sup>
- Concussion symptoms such as headache and disorientation may disappear in 15 minutes, but 75 percent of those tested 36 hours later still had problems with memory and cognition.
- 400,000 brain injuries (concussions) occurred in high school athletics during the 2008-09 school year.<sup>5</sup>
- There are three times as many catastrophic football injuries among high school athletes as college athletes.<sup>6</sup>
- 15.8 percent of football players who sustain a concussion severe enough to cause loss of consciousness return to play the same day.<sup>7</sup>
- History of injury is often a risk factor for future injury, making prevention critical.
- 62 percent of organized sports-related injuries occur during practices.<sup>8</sup>
- Only 42 percent of high schools have access to athletic training services.<sup>9</sup>
- 47 percent of schools nationally fall short of the federally recommended nurse-to-student ratio. Many schools have no nurse at all.<sup>10</sup>
- Emergency department visits for concussions sustained during organized team sports doubled among 8-13 year olds between 1997 and 2007 and nearly tripled among older youth.<sup>11</sup>
- Concussion rates more than doubled among students age 8-19 participating in sports like basketball, soccer and football between 1997 and 2007, even as participation in those sports declined.<sup>12</sup>

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<sup>1</sup> National Athletic Trainers’ Association

<sup>2</sup> Wier, L. (Thomson Reuters), Miller, A. (Student), and Steiner, C. (AHRQ). *Sports Injuries in Children Requiring Hospital Emergency Care, 2006*. HCUP Statistical Brief #75. June 2009. Agency for Healthcare Research and Quality, Rockville, MD. <http://www.hcup-us.ahrq.gov/reports/statbriefs/sb75.pdf>

<sup>3</sup> Cantu RC. Second impact syndrome: immediate management. *Physician Sportsmed.* 1992;20(9):14–17; Mueller F. Catastrophic Head Injuries in High School and Collegiate Sports. 2001;36(3):312-315

<sup>4</sup> Covassin T; Swanik, C; *Sex Differences and the Incidence of Concussions Among Collegiate Athletes.* J Athl Train. 2003 July-Sep; 38(3):238-244

<sup>5</sup> Yard E., Comstock, R. *Compliance with return to play guidelines following concussion in US high school athletes, 2005-2008*, *Informa healthcare*, 2009, Vol. 23, No. 11, Pages 888-898

<sup>6</sup> Boden, B; *Catastrophic Head Injury Three Times Greater in High School vs. Collegiate Football Players*, July 2007, *American Journal of Sports Medicine*

<sup>7</sup> Center for Injury Research and Policy, The Research Institute at Nationwide Children’s Hospital, Dr. Dawn Comstock, Columbus, OH

<sup>8</sup> Reche J, Yard E. Comstock R. (2008) *An Epidemiologic Comparison of High School Sports Injuries Sustained in Practice and Competition*

<sup>9</sup> National Athletic Trainers’ Association

<sup>10</sup> National Association of School Nurses, 2008 Survey

<sup>11</sup> Bakhos, L; Lockhart, G; Myers, R (2009) *Emergency Department Visits for Concussion in Young Child Athletes* PEDIATRICS (doi:10.1542/peds.2009-3101)

<sup>12</sup> Bakhos, L; *Emergency Department Visits for Concussion in Young Child Athletes*, Sep 1, 2010 Volume 126, Issue 3 *Journal of the American Academy of Pediatrics*

# Sex Differences in Concussion Symptoms of High School Athletes

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**Context:** More than 1.6 million sport-related concussions occur every year in the United States, affecting greater than 5% of all high school athletes who participate in contact sports. As more females participate in sports, understanding possible differences in concussion symptoms between sexes becomes more important.

**Objective:** To compare symptoms, symptom resolution time, and time to return to sport between males and females with sport-related concussions.

**Design:** Descriptive epidemiology study.

**Setting:** Data were collected from 100 high schools via High School RIO (Reporting Information Online).

**Patients or Other Participants:** Athletes from participating schools who sustained concussions while involved in interscholastic sports practice or competition in 9 sports (boys' football, soccer, basketball, wrestling, and baseball and girls' soccer, volleyball, basketball, and softball) during the 2005–2006 and 2006–2007 school years. A total of 812 sport concussions were reported (610 males, 202 females).

**Main Outcome Measure(s):** Reported symptoms, symptom resolution time, and return-to-play time.

**Results:** No difference was found between the number of symptoms reported ( $P = .30$ ). However, a difference was seen in the types of symptoms reported. In year 1, males reported amnesia (exact  $P = .03$ ) and confusion/disorientation (exact  $P = .04$ ) more frequently than did females. In year 2, males reported more amnesia (exact  $P = .002$ ) and confusion/disorientation (exact  $P = .002$ ) than did females, whereas females reported more drowsiness (exact  $P = .02$ ) and sensitivity to noise (exact  $P = .002$ ) than did males. No differences were observed for symptom resolution time ( $P = .40$ ) or return-to-play time ( $P = .43$ ) between sexes.

**Conclusions:** The types of symptoms reported differed between sexes after sport-related concussion, but symptom resolution time and return-to-play timelines were similar.

**Key Words:** epidemiology, mild traumatic brain injuries, symptom resolution, return to play

## Key Points

- After a sport-related concussion, male and female high school athletes presented with different types of symptoms.
- Males reported more cognitive symptoms, whereas females reported more neurobehavioral and somatic symptoms.
- Symptom resolution time and time to return to play did not differ between sexes.

There are an estimated 7.3 million high school students participating in organized interscholastic athletics in the United States each year.<sup>1</sup> An estimated 1.6 to 3.8 million sport-related concussions are sustained every year,<sup>2</sup> with an average of 21% occurring in high school athletes.<sup>3,4</sup> More than 5% of all high school athletes who participate in contact sports such as football, lacrosse, and hockey sustain a concussion each year.<sup>5–8</sup> Although males continue to participate in sports at a higher rate than females, in 2008, 3.01 million (or 41%)<sup>1</sup> of high school athletes were female, up from 2.4 million 10 years earlier.<sup>9</sup> Injury incidence among females has increased with increased female sport participation, and now females have a higher incidence rate of sport-related concussions than do males.<sup>3,10,11</sup> However, the female response to concussion has not been well described, and similarities in male and female sport-related concussion symptoms remain largely anecdotal.

Diagnosing sport-related concussions is one of the most elusive tasks of sports medicine clinicians. No biological markers exist to detect this relatively mild injury, and

diagnosis largely depends on a patient's report.<sup>12</sup> Assessment is further complicated by the tendency of many athletes to underreport or hide symptoms from clinicians, coaches, and parents.<sup>13</sup>

Research<sup>14–16</sup> on more severe closed head injuries and traumatic brain injuries (TBIs) indicates that females may respond to concussions differently than males. Authors<sup>14</sup> of a meta-analysis of postconcussion outcome sex differences identified 8 studies that provided data by sex, citing worse overall outcomes in females. Twenty outcomes were compared, including variables such as postconcussive symptoms at 6 weeks, days of posttraumatic amnesia, length of hospitalization, inability to return to work, and presence of headache, dizziness, and insomnia. Retrospective studies<sup>16,17</sup> comparing males and females with TBI showed that females tended to fare worse than their male counterparts, with longer hospitalizations, longer residual disabilities, and higher mortality rates. In one pharmacologic trial,<sup>15</sup> female patients younger than 51 years had greater frequency of brain swelling and intracranial

hypertension than did male patients of the same age. These findings were not associated with increased injury severity on the Glasgow Coma Scale; in fact, females with higher scores had more pronounced swelling.<sup>15</sup> Thus, females with TBI may require greater monitoring and more aggressive treatment than males.

In an attempt to elucidate the mechanisms explaining sex differences in TBI, animal models have been developed.<sup>18,19</sup> When rats were supplemented with estrogen before a fluid-percussion brain injury was induced, a protective effect was seen among male rats, but female rats showed exacerbated effects and higher mortality rates.<sup>19</sup> Bramlett and Dietrich,<sup>18</sup> however, noted that estrogen had a neuroprotective effect on cerebral hematoma size in rats. Both estrogen and progesterone are associated with the inflammatory reaction and extent of tissue damage in the brain.<sup>20</sup> Therefore, different outcomes among females after TBI may reflect a biochemical basis, with hormonal factors playing a role in the response to head trauma. This relationship remains unclear, especially in adolescents, whose hormone levels vary; also unknown is whether similar effects occur in cases involving mild head injury and sport-related concussion.

Neuropsychological data have been examined to identify sex differences in sport-related concussions. However, most investigators have studied collegiate athletes, and whether high school athletes will behave similarly is uncertain. Covassin et al<sup>21</sup> reported differences in visual memory composite scores in collegiate athletes and suggested a relationship between postconcussive symptoms and sex. In a mixed sample of collegiate and high school athletes, Broshek et al<sup>22</sup> found that females tended to be more cognitively impaired after a sport-related concussion. Lovell et al<sup>23</sup> noted that high school athletes with concussions had memory and processing deficits, but they did not compare sex differences in symptoms. Sport-related concussions in male and female high school athletes have been the focus of limited research; specifically, sex differences in symptoms, symptom resolution time (SRT), and return-to-play (RTP) timelines in high school athletes have not been studied.

The purpose of our study was to investigate concussion symptoms in male and female athletes using a national sample of high schools. We compared the total number of symptoms reported by sex and the prevalence of each reported symptom among males and females, SRT, and RTP timelines. Based on the previous literature dealing with sport-related concussion and TBI, we hypothesized that females would report a greater number of symptoms, longer symptom duration, and later RTP.

## METHODS

During the 2005–2006 (year 1) and 2006–2007 (year 2) school years, injury and exposure data were prospectively collected from athletes via the High School Sport-Related Injury Surveillance System, RIO (Reporting Information Online, Columbus, OH).<sup>3,24–26</sup> An Internet-based injury surveillance system, RIO records injury data in US high school sports. Sports studied during years 1 and 2 were boys' football, soccer, basketball, wrestling, and baseball and girls' soccer, basketball, volleyball, and softball.

All US high schools with at least one Board of Certification–certified athletic trainer (AT) on staff with

a valid e-mail address (N = 4120 in 2005–2006 and N = 3378 in 2006–2007) were eligible to participate. A total of 425 and 316 (in years 1 and 2, respectively) of the ATs agreed to participate. Schools were then categorized into 8 sampling strata based on US census geographic regions (northeast, midwest, south, and west)<sup>27</sup> and school size (enrollment of <1000 or ≥1000 students). Schools were randomly selected from each of the sampling strata (n = 100). If a school dropped out of the study, another school from the same stratum was randomly selected to replace it.<sup>4</sup>

During each school year, ATs from participating high schools were required to log on to the High School RIO Web site weekly, using an individualized study identification number, to report athlete-exposures (AEs) and injury data. An *AE* was defined as 1 athlete participating in 1 practice or competition. *Injury* was defined by 3 factors: (1) it occurred during an organized high school practice or competition; (2) it required attention by a team AT or physician; and (3) it resulted in restriction of participation of the student-athlete for at least 1 day. The data-collection software used sequential drop-down menus to administer a questionnaire asking for detailed information regarding each injury, including student-athlete demographics. The ATs were able to view, edit, and update previously entered information throughout the study period. The data analysis for this study had institutional review board approval.

During year 1 (2005–2006), ATs were asked to record only the primary symptom experienced by the injured athletes. For year 2 (2006–2007), the data-collection procedures were changed such that the clinician was prompted to record all symptoms reported by the concussed athlete using a computerized drop-down menu. Subsequently, the AT recorded the time period during which symptoms resolved as well as the time period after which the athlete returned to play. The AT was also asked to indicate all clinicians who assessed the injury, all methods of injury assessment, and, via drop-down menu, whether the injury was new or recurrent (Table 1).

For the purpose of this study, we included only those injuries classified as concussions in the analysis. The main exposure (sex) was determined by the sport in which the student-athlete participated. Outcome variables of interest were (1) symptoms, including frequency of reported symptoms and total number of symptoms (year 2 only), (2) SRT, and (3) RTP time.

We assessed the following as potential confounders: regional strata, age, height, mass, by whom the concussion was assessed, whether the concussion took place during practice or competition, whether the concussion was a new or recurrent injury, and whether the athlete was wearing personal protective equipment (eg, helmet, mouthguard, shin guards).

## Statistical Analysis

Because the data-collection methods differed between years 1 and 2, symptom data for each year were analyzed separately. As expected, cell counts for some symptoms were less than or equal to 5, so the Fisher exact test was performed to assess differences in the proportions of athletes reporting each symptom by sex. Differences between the



**Table 1. Possible Responses for Select Questions Pertaining to Concussions in the Reporting Information Online (RIO) System**

- 1) Concussion symptoms: amnesia, concentration difficulty, confusion/disorientation, dizziness/unsteadiness, drowsiness, headache, hyperexcitability, irritability, loss of consciousness, nausea, tinnitus, sensitive to light/visual disturbance, sensitive to noise, other<sup>a,b</sup>
  - Symptom resolution time<sup>c</sup>
    - <15 min
    - 15–29 min
    - 30–59 min
    - 1–11 h
    - 12–23 h
    - 1–3 d
    - 4–6 d
    - 1 wk–1 mo
    - >1 mo
- 2) Injury assessed by<sup>b</sup>
  - Athletic trainer
  - Physician
  - Dentist/oral hygienist
  - Nurse practitioner
  - Physician assistant
  - Other
- 3) Assessment method<sup>b</sup>
  - Evaluation
  - X-ray
  - Magnetic resonance imaging
  - Computed tomography
  - Surgery
  - Bloodwork/laboratory tests
  - Other
- 4) This injury is ...<sup>c</sup>
  - New
  - Recurrent this academic year
  - Recurrent from previous academic year
  - Other
- 5) Outcome<sup>c</sup>
  - RTP <1 d
  - RTP 1–2 d
  - RTP 3–6 d
  - RTP 7–9 d
  - RTP 10–21 d
  - RTP >22 d
  - Season ended before RTP
  - Medical disqualification for season
  - Medical disqualification for career
  - Athlete chose not to continue
  - Athlete released from team
  - Permanent paralysis
  - Fatality
  - Other

Abbreviation: RTP, return to play.

<sup>a</sup> During year 1, athletic trainers recorded only the primary concussion symptom. In year 2, they were asked to select all concussion symptoms.

<sup>b</sup> Select all that apply.

<sup>c</sup> Select 1 option from drop-down menu.

mean numbers of reported symptoms by sex were also evaluated via *t* test for year 2. Logistic regression was used to assess for the confounding variables listed above.

To evaluate the relationships between sex and both SRT and the RTP, we fit complementary log-log models. These models were used for discrete time-to-event data for which it cannot be assumed that the exact time of the occurrence

of the event is known within the interval (eg, less than 15 minutes). Because the reported time intervals in the data set were not of equal length, this model is appropriate: it assumes a continuous time process with events not occurring at discrete time points but rather occurring at any time point during the interval. Multivariable analysis was used to assess for potential confounding by the variables listed above. All analyses were performed using SAS (version 9.1.3; SAS Institute Inc, Cary, NC), with  $\alpha = .05$  set a priori. The  $\alpha$  level was not adjusted for these preplanned comparisons.<sup>28</sup>

## RESULTS

A total of 391 concussions (283 males, 108 females) were reported in year 1 (Table 2), and 421 concussions (327 males, 94 females) were reported in year 2 (Table 3).

### Symptoms

Only the primary symptom was reported in year 1. Headache was the most commonly reported primary symptom (40% [*n* = 113] among males, 44% [*n* = 48] among females), although no difference was noted between sexes (exact *P* = .49) (Table 4). When we compared symptoms by sex, males reported amnesia and confusion/disorientation as their primary symptom more frequently than did females (exact *P* values = .03 and .04, respectively). No additional symptoms were reported by one sex more often than by the other sex.

In year 2, all reported symptoms were recorded. No difference (*P* = .30) was evident between the mean number of symptoms reported by males ( $4.2 \pm 1.9$ ) and females ( $4.0 \pm 1.9$ ) (Table 5). Headache was the most commonly reported symptom for both sexes (95% among males [*n* = 311] and 97% [*n* = 91] among females), although no difference was found between sexes (exact *P* = .59). Males reported amnesia (exact *P* = .002) and confusion/disorientation (exact *P* = .002) more often than did females (Table 6). However, females reported drowsiness (exact *P* = .02) and sensitivity to noise (exact *P* = .002) more often than did males.

### Time to Symptom Resolution

Most (*n* = 556, 70.8%) of the student-athletes reported resolution of their symptoms within 3 days of injury (Figure; *n* = 424 [72.2%] among males and *n* = 135 [66.7%] among females) (Table 7). Data were missing for 23 males and 4 females. The SRT was not different between the sexes (hazard ratio = 1.07; 95% confidence interval = 0.91, 1.27). The relationship between sex and SRT did not appear to be appreciably affected by any of the variables assessed as potential confounders.

### Time to RTP

Among all student-athletes, 64% (*n* = 503) returned to play by 9 days postinjury (63% [*n* = 133] of males and 66% [*n* = 370]) of females. The greatest percentage of males returned to play between 7 and 9 days after concussion (*n* = 174, 29.7%), whereas the greatest percentage of females returned between 3 and 6 days after concussion (*n* = 59,

**Table 2. Concussed Athletes and Height, Mass, and Age by Sport: Year 1**

Participants	Concussed Athletes, No.	Age, y (Mean $\pm$ SD)	Height, cm (Mean $\pm$ SD)	Mass, kg (Mean $\pm$ SD)
All athletes	391	15.9 $\pm$ 1.2	172.7 $\pm$ 10.2	72.6 $\pm$ 15.0
Males (72.38%)	283	16.0 $\pm$ 1.2	176.8 $\pm$ 8.1	76.7 $\pm$ 14.4
Football	199	15.9 $\pm$ 1.2	174.8 $\pm$ 7.1	78.9 $\pm$ 14.2
Soccer	33	16.3 $\pm$ 1.1	176.8 $\pm$ 7.1	72.8 $\pm$ 10.0
Basketball	15	16.3 $\pm$ 1.3	183.9 $\pm$ 9.1	75.8 $\pm$ 11.7
Wrestling	29	16.2 $\pm$ 1.3	171.2 $\pm$ 12.5	67.8 $\pm$ 18.4
Baseball	7	16.0 $\pm$ 1.0	177.1 $\pm$ 6.4	74.2 $\pm$ 11.0
Females (27.62%)	108	15.8 $\pm$ 1.2	165.4 $\pm$ 7.6	60.2 $\pm$ 8.5
Soccer	52	16.0 $\pm$ 1.2	162.8 $\pm$ 6.1	58.5 $\pm$ 7.6
Basketball	41	15.6 $\pm$ 1.3	166.9 $\pm$ 8.9	60.4 $\pm$ 7.8
Volleyball	6	15.5 $\pm$ 1.4	166.6 $\pm$ 4.8	64.1 $\pm$ 11.0
Softball	9	15.7 $\pm$ 1.1	170.9 $\pm$ 3.6	67.0 $\pm$ 13.3

**Table 3. Concussed Athletes and Height, Mass, and Age by Sport: Year 2**

Participants	Concussed Athletes, No.	Age, y (Mean $\pm$ SD)	Height, cm (Mean $\pm$ SD)	Mass, kg (Mean $\pm$ SD)
All athletes	421	15.9 $\pm$ 1.7	174.2 $\pm$ 10.2	74.4 $\pm$ 18.9
Males (77.67%)	327	15.9 $\pm$ 1.2	176.8 $\pm$ 9.1	79.0 $\pm$ 18.8
Football	244	15.9 $\pm$ 1.2	177.3 $\pm$ 8.9	82.4 $\pm$ 19.5
Soccer	36	15.9 $\pm$ 1.3	173.7 $\pm$ 8.4	65.2 $\pm$ 8.4
Basketball	16	16.1 $\pm$ 0.8	181.4 $\pm$ 10.9	71.9 $\pm$ 14.0
Wrestling	24	15.8 $\pm$ 1.1	176.0 $\pm$ 9.4	73.0 $\pm$ 15.2
Baseball	7	15.8 $\pm$ 0.8	175.8 $\pm$ 12.9	68.1 $\pm$ 8.3
Females (22.33%)	94	15.8 $\pm$ 1.1	165.4 $\pm$ 7.9	59.1 $\pm$ 8.2
Soccer	37	15.9 $\pm$ 1.2	163.6 $\pm$ 7.1	56.9 $\pm$ 4.9
Basketball	35	15.7 $\pm$ 1.0	167.9 $\pm$ 7.9	61.4 $\pm$ 9.5
Volleyball	12	16.2 $\pm$ 1.2	162.3 $\pm$ 10.4	57.5 $\pm$ 9.9
Softball	10	15.6 $\pm$ 0.8	164.6 $\pm$ 3.6	60.7 $\pm$ 9.3

29.4%) (Table 8). Data were missing for 24 males and 1 female. No difference was noted between male and female athletes with regard to RTP timelines (hazard ratio = 0.93; 95% confidence interval = 0.78, 1.11). The effect between sex and time to RTP also did not appear to be appreciably confounded by the previously mentioned variables.

## DISCUSSION

Historically, authors<sup>29–31</sup> of sport-related concussion studies have examined symptoms in males and focused on football because of the increased risk of concussion in

that sport. However, both the risk of injury and the incidence rate of sport-related concussions are higher among female athletes.<sup>3,11</sup> There is little information currently available that compares concussion symptoms between sexes, especially among adolescent athletes. The large database we used allowed us to examine the spectrum of sport-related concussions experienced by high school student-athletes over a 2-year period and allowed symptom comparison between males and females. These surveillance data represent what is actually happening in high school sports rather than what might happen in the atmosphere of a controlled study. Thus, our findings represent the injuries

**Table 4. Primary Concussion Symptom Reported: Year 1<sup>a</sup>**

Symptom	Males (n = 283), No. (%)	Females (n = 108), No. (%)	Exact P Value
<b>Amnesia</b>	<b>26 (9)</b>	<b>3 (3)</b>	<b>.03</b>
Concentration difficulty	13 (5)	5 (5)	1.00
<b>Confusion/disorientation</b>	<b>37 (13)</b>	<b>6 (6)</b>	<b>.04</b>
Dizziness/unsteadiness	44 (16)	22 (20)	.29
Drowsiness	1 (0.4)	2 (2)	.19
Headache	113 (40)	48 (44)	.49
Hyperexcitability	1 (0.4)	0 (0)	1.00
Irritability	0 (0)	1 (1)	.28
Loss of consciousness	6 (2)	5 (5)	.19
Nausea	5 (2)	2 (2)	1.00
Tinnitus	1 (0.4)	0 (0)	1.00
Sensitive to light/visual disturbance	0 (0)	0 (0)	NA
Sensitive to noise	10 (4)	2 (2)	.52

Abbreviation: NA, not applicable.

<sup>a</sup> Bold font indicates significant findings.

**Table 5. Number of Symptoms Reported by Sex: Year 2 Only**

Sex	Range	Mean $\pm$ SD	95% Confidence Interval	P Value
Males (n = 325)	1–10	4.2 $\pm$ 1.9	4.0, 4.4	.30
Females (n = 94)	1–12	4.0 $\pm$ 1.9	3.6, 4.3	

that are being diagnosed as concussions and how these injuries are being managed, given the reality of the wide variations in level of education, experience, and resources.

### Symptom Prevalence

Symptoms are used as a criterion for concussion evaluation and for making RTP decisions. In a 2005 survey,<sup>12</sup> clinical examination and a symptom checklist were the concussion-evaluation tools used most often by clinicians. Headache was the primary symptom most commonly reported by both sexes in year 1 and the most consistently reported symptom in year 2 (95% among males and 97% among females). Headache is considered a hallmark symptom of concussion and was reported in similar frequencies by other researchers.<sup>32–34</sup> However, symptom type has not been conclusively associated with the severity of sport-related concussion. Guskiewicz et al<sup>33</sup> associated the persistence of headache with more severe injury because a greater percentage of football players with grade II concussions had complaints of headache lasting longer than 7 days when compared with players with grade I concussions. In the same study, headache symptoms among 28% of participants resolved within 24 hours, and only 10% of those with grade I injuries had headaches lasting longer than 5 days.

Loss of consciousness (LOC) is also used to rate the severity of a sport-related concussion, but it was 1 of the 3 least-reported symptoms (<5%) in our study, consistent with the findings of Erlanger et al<sup>35</sup> and Barnes et al<sup>36</sup>. Some grading systems (eg, Torg,<sup>37</sup> Colorado Medical Society [Kelly and Rosenberg<sup>38</sup>], and American Academy of Neurology [Kelly<sup>39</sup>]) rely heavily on LOC to rate the severity of a concussion, whereas the more recent Evidence-Based Cantu Grading Scale<sup>40</sup> focuses on post-concussion signs and symptoms. In our study, the reports of LOC between sexes did not differ, nor was there a difference in SRT. Thus, both males and females sustained a high number of mild concussion injuries with no apparent difference in severity based on LOC data.

### Number of Reported Symptoms

The database we used relied on a system by which clinicians chose symptoms from a drop-down menu. In most cases, clinicians selected from the symptoms provided, but they were also provided a choice of *other* to capture injury symptoms not included on the list. Many used the *other* option to list symptoms that were already on the list, particularly in year 1, when only the primary symptom was captured. Listing several symptoms in this manner would not prioritize a specific symptom. For example, of the 35 clinicians who used *other*, 23 included headache among the symptoms listed rather than using the drop-down menu. The majority of symptoms listed using the text field were also on the drop-down menu. In year 2, it appeared that clinicians used the *other* category to list specific characteristics, such as “loss of peripheral vision in the right eye” or “difficulty using left hand.” In year 2, the *other* category was often used in conjunction with the list of multiple symptoms to provide more descriptive characteristics.

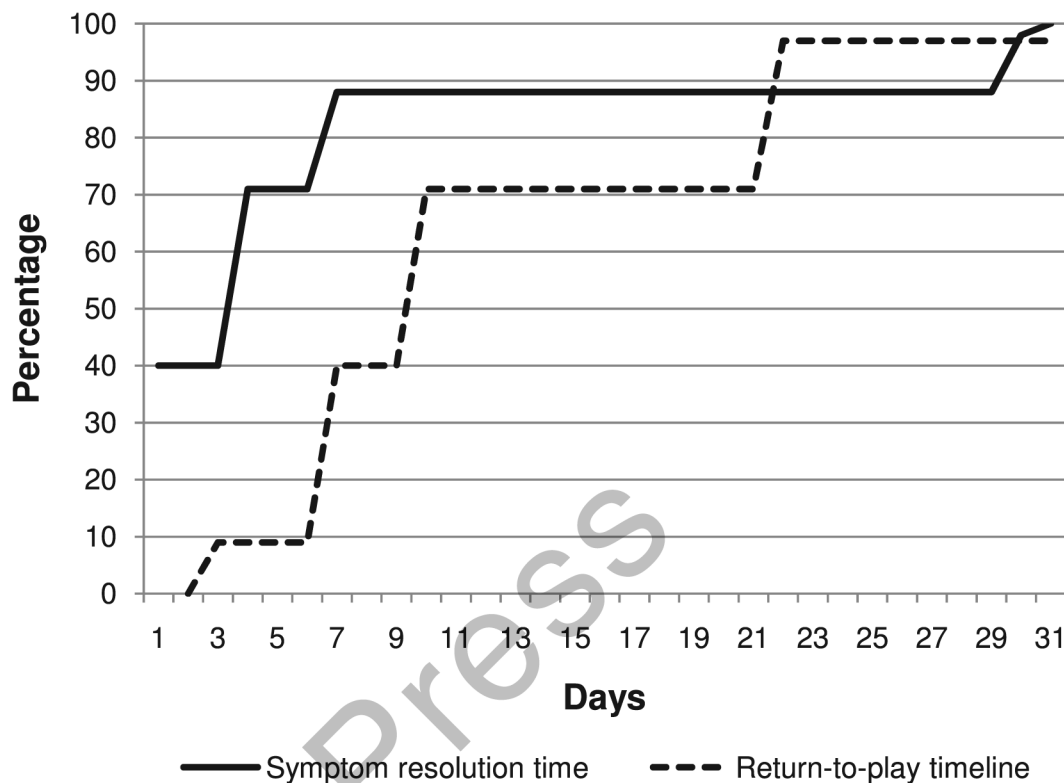
With respect to the number of symptoms reported, only the data from year 2 could be evaluated because a single, primary symptom was recorded in year 1. We found no difference in the number of symptoms reported between males and females, consistent with reports from other studies.<sup>38,41,42</sup> Some researchers,<sup>22,43</sup> however, have shown that females reported more postconcussion symptoms than did males. This result may reflect differences in the populations studied, severity of injury, and mechanism of reporting, given that Lovell et al<sup>43</sup> and Broshek et al<sup>22</sup> studied smaller samples of collegiate athletes. Those authors examined participants who had been referred by physicians for neuropsychological testing or had symptoms lasting at least 5 days. Our study included more than 800 concussions sustained by high school student-athletes that were identified by an AT and most symptoms resolved in fewer than 3 days. All concussion injuries that required disqualification from athletic participation for at least 1 day were included. Mechanisms of reporting symptoms varied from those used in our study. Lovell et

**Table 6. All Concussion Symptoms Reported: Year 2<sup>a</sup>**

Symptom	Males (n = 327), No. (%)	Females (n = 94), No. (%)	Exact P Value
<b>Amnesia</b>	<b>84 (26)</b>	<b>10 (11)</b>	<b>.0020</b>
Concentration difficulty	166 (51)	44 (47)	.50
<b>Confusion/disorientation</b>	<b>175 (54)</b>	<b>33 (35)</b>	<b>.0017</b>
Dizziness/unsteadiness	252 (77)	72 (77)	.92
<b>Drowsiness</b>	<b>64 (20)</b>	<b>29 (31)</b>	<b>.02</b>
Headache	311 (95)	91 (97)	.59
Hyperexcitability	8 (2)	2 (2)	1.00
Irritability	22 (7)	3 (3)	.20
Loss of consciousness	12 (4)	5 (5)	.55
Nausea	108 (33)	34 (36)	.57
Tinnitus	43 (13)	10 (11)	.52
Sensitive to light/visual disturbance	103 (32)	26 (28)	.48
<b>Sensitive to noise</b>	<b>15 (5)</b>	<b>13 (14)</b>	<b>.0015</b>

<sup>a</sup> Bold font indicates significant findings.





**Figure.** Time to symptom resolution and return to play.

al<sup>43</sup> and Broshek et al<sup>22</sup> used computerized neurocognitive evaluation tools that relied on self-recorded symptoms rather than reports of symptoms to a health care professional. Self-recorded symptoms are subjective and many are not reported.<sup>13,44</sup> One group<sup>13</sup> suggested that just over 50% of concussions go unreported, and others<sup>13,31,45,46</sup> suggest that underreporting may occur when an athlete is unaware of the signs and symptoms of a sport-related concussion. Thus far, studies<sup>13</sup> investigating the denial of symptoms associated with concussion have only included males.

### Symptom Classification

Previous researchers have attempted to categorize concussion symptoms based on factorial analysis. A 3-factor measurement model has been proposed by Piland et al<sup>47</sup> to evaluate the validity of the Graded Symptom Checklist. They were able to categorize self-reported symptoms into the following factors: (1) cognitive (feeling

“slowed down,” feeling like they were “in a fog,” difficulty concentrating, and difficulty remembering), (2) neurobehavioral (sleeping more than usual, drowsiness, fatigue, and nervousness), and (3) somatic (headache, nausea, sensitivity to light and noise, and balance problems).<sup>47</sup> The Post Concussion Symptom Scale (another self-reported symptom inventory) has traditionally been used to capture concussion symptoms in an uncategorized manner<sup>48</sup> and, more recently, the Acute Concussion Evaluation categorized concussion symptoms as physical, cognitive, emotional, and sleep associated.<sup>49</sup> The RIO used a pull-down menu to describe symptoms that were reported to the AT (Table 4) and incorporated a symptom list similar to the lists of the Graded Symptom Checklist, Acute Concussion Evaluation, and Post Concussion Symptom Scale.<sup>48</sup> Each symptom category was represented by RIO, although sadness, trouble falling asleep, feeling like one is “in a fog,” and feeling “slowed down” were not listed options. However, RIO did include irritability, dizziness/unsteadiness, and tinnitus. Because RIO used recorded data by a

**Table 7.** Time to Concussion Symptom Resolution by Sex

Symptom Resolution Time	Percentage	
	Males	Females
<15 min	6.1	6.6
15–29 min	9.7	8.1
30–59 min	6.8	4.1
1–11 h	9.9	7.0
12–23 h	8.5	8.6
1–3 d	31.2	32.3
4–6 d	15.6	21.2
1 wk–1 mo	10.4	11.1
>1 mo	1.8	1.0

**Table 8.** Time to Return to Play by Sex

Time to Return to Play	Percentage Returned to Play (Cumulative Percentage)	
	Males	Females
1–2 d	8.2	8.5
3–6 d	25.3 (33.5)	29.4 (37.9)
7–9 d	29.7 (63.2)	28.4 (66.3)
10–21 d	23.9 (87.1)	20.9 (87.2)
≥22 d	2.0 (89.1)	3.6 (90.8)
Medical disqualification for season	7.3 (96.4)	5.1 (95.9)
Athlete chose not to return	0.5 (96.9)	0.5 (96.4)
Other	3.1 (100)	4.1 (100)



health care professional, these data were perhaps of better quality than those collected by self-report. We do not know if ATs used the symptom checklist provided by the drop-down menus of the RIO or if they relied on the injured student-athletes for symptom reporting. Again, the potential for underreporting symptoms may be minimized by using checklists rather than relying on injured student-athletes to report their concussion symptoms.<sup>13</sup>

Our data indicate that the type of symptoms recorded for high school athletes who sustained concussions differed between males and females. In year 1, when only the primary symptom was recorded, males were more likely to report amnesia and confusion/disorientation—both cognitive symptoms—than were females. Females did not report any single primary symptom more often than males. In year 2, when all symptoms were listed, males again were more likely to report amnesia and confusion/disorientation (cognitive symptoms) than were females. Females reported drowsiness (neurobehavioral symptom) and sensitivity to noise (somatic symptom) more than did males. These data indicate that male and female high school students who suffer concussions may have different types of symptoms and, thus, may present differently to health care professionals. Without adequate symptom assessment, the neurobehavioral or somatic symptoms more commonly described by females may be more easily missed or attributed to other conditions, such as stress, depression, or anxiety.<sup>50</sup> Clearly, more information is needed to interpret subtle differences in postconcussive symptoms, but ATs need to be aware that after a concussion, the symptoms reported by females should be linked to the concussion until conflicting neuropsychological conditions can be ruled out.

From a clinical perspective, it is important to evaluate each concussion individually and to record all symptoms reported by the injured athlete. Some symptoms, especially in the neurobehavioral category, are often overlooked on a sideline or initial assessment. Because high school girls are more likely to display these symptoms, ATs should initially associate those types of symptoms with a potential concussion and should withhold an athlete from contact activity until the symptoms subside. Although no investigators have been able to accurately assess severity of the sport-related concussion based on the symptoms presented, the scales used, either self-reported or recorded, are valid assessment tools.<sup>43,47</sup> The differentiation of concussion symptoms and categorization into cognitive, neurobehavioral, and somatic groups may help to predict outcomes, RTP criteria, or likelihood of experiencing postconcussion syndrome in the future.

### Symptom Resolution Time

Research examining the SRT for high school athletes is sparse. In our study, most symptoms associated with sport-related concussion resolved in 3 days or fewer, and the SRT did not differ between males and females. This rapid resolution of symptoms is consistent with other findings in collegiate athletes. In a study of more than 1600 collegiate football players,<sup>29</sup> nearly 90% of all concussed athletes' symptoms resolved within 7 days of the initial injury. Collie et al<sup>41</sup> reported that participants' SRTs ranged between 2 and 10 days. Maddocks and Saling<sup>51</sup> noted that although

certain concussion symptoms (such as headache and nausea) resolved within 5 days, persistent neuropsychological changes were still apparent at 5 days postinjury. Furthermore, concussions are often accompanied by cognitive problems, such as increased processing time and short-term memory loss, which can easily be overlooked if they are not carefully assessed. Lovell et al<sup>52</sup> demonstrated that it takes 1 week or longer to resolve the neurocognitive deficits seen with “ding” or grade I concussions in high school athletes. Thus, symptom resolution is required before returning to play, but the evaluation of symptoms cannot be the only determining factor in making these decisions.

### Time to RTP

The median time to RTP for all participants was 3 to 6 days and was not different between males and females. These findings agree with those of another large observational cohort investigation<sup>23</sup> involving high school athletes, in which a median time to RTP of 3 days was reported. Both groups studied athletes participating in similar high school sports and used the same operational definition of concussion. Powell and Barber-Foss,<sup>10</sup> however, captured the RTP data with greater precision than RIO permitted. We were able to report only the median category of RTP, but the median time to RTP for all concussions reported by Powell and Barber-Foss<sup>10</sup> falls within our reported category. Powell and Barber-Foss,<sup>10</sup> however, did not report or conduct data analysis comparing time to RTP between sexes. Comparison of their reported data for each category indicates findings similar to ours because no difference was noted in the RTP time between sexes after concussion (Table 8).

### Strengths

We are the first to examine concussion symptoms, SRT, and RTP time in a national high school population. This study provides insight into concussion presentation, evaluation, and management at the high school level. The methods used in RIO have undergone internal validity checks with high sensitivity, specificity, and positive predictive values.<sup>53</sup> These values, combined with the very low failure-to-report and incomplete-variable rates, indicate that this is the highest-quality data available for a national sample of US high school athletes. Furthermore, no confounding variables, such as protective equipment or initial or recurrent injury, affected the results.

### Limitations

Our study had several limitations. First, no uniform definition of concussion was used by ATs reporting to RIO. As a result, disparities in the data may reflect disparities in the interpretations of concussion by the reporting ATs. Additionally, injuries were only included in the data set if the participating AT was aware of the injury and if the injury had a time-loss factor of one or more days. The software was also changed within our study period, ultimately improving the instrument. However, we were unable to combine the data for both years to analyze the number of symptoms reported. Finally, there was no operational definition for grading the severity of the

concussion, and the clinicians' grading was arbitrary. Therefore, we were not able to analyze concussion severity as a potential confounding factor.

## Conclusions

Many authors<sup>3,11,22,36</sup> have suggested that differences between sexes exist in sport-related concussion. Based on SRT and RTP time, little difference is evident in the severity or outcome of concussions sustained between sexes in high school athletes. However, male and female high school athletes appeared to present with different types of symptoms after a sport-related concussion. Males reported more cognitive symptoms, whereas females reported more symptoms in the neurobehavioral and somatic categories. Symptom evaluation is only one component of a concussion evaluation, and each symptom should be evaluated and monitored to resolution before RTP. Neurocognitive assessments, balance measurements, symptoms, and the physical examination should be used collectively to evaluate concussions.

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# The Next Practice

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

Every summer, the same news occurs again. In the blur and chaos of our crazy lives, we hardly stop to take notice that another athlete has died after performing intense exercise in the heat. When parents drop their sons and daughters off for practice, they wish their children well and establish plans for a pick-up. Yet sometimes the pick-up never happens, replaced instead by a furious rush to the hospital after a frantic call from an assistant coach: "Your child collapsed during practice and became unconscious and is currently being taken to the hospital." Parents arrive at the hospital to learn that their child had an exertional heat stroke (EHS), a sudden cardiac event, exertional sickling, asthma attack, severe head injury, or any of a myriad of other injuries and conditions that can cause an otherwise healthy child or young adult to go from extremely vibrant to death's door in a matter of minutes.

Tempting as it is, looking away will not make the problem disappear. The shock only becomes more personal when such an event occurs to your athlete. Sudden death is occurring more frequently, at a pace that we cannot completely explain.

I once had an EHS. I was 16 years old, running a 10-K race on the track at the Empire State Games in upstate New York. Looking back, as I have for the past 24 years, in a daily ritual to examine the seminal moment that shaped every subsequent action, I know that many factors leading to my EHS were preventable. The 25-lap race began in the middle of the day during a heat wave, even though events with much less risk were being run under the lights in the evening. Hydration was not allowed during the race. (Today, it's almost impossible to imagine teenagers running 6+ miles "all out" in extreme heat and not being allowed fluids.)

I first contemplated all this as I lay in my hospital room, and I have spent my career as a professor at the University of Connecticut dedicated to preventing sudden death in sport. Here is my simple message: most cases of sudden death in sport are preventable, whether through better prevention strategies or enhanced treatment plans. Exertional heat stroke is the perfect example, with 100% survival possible if immediate, on-site cooling via cold-water immersion is provided. Surprisingly, athletes continue to die from EHS. This message is not meant to depress

you but rather to inform and motivate you. When a parent drops his or her child off at the next practice, the school assumes responsibility for the child's health and well-being during that practice, but is the school ready to assume this responsibility?

As we have learned from the very high-profile case of a Kentucky high school football coach who was arrested in 2009 as a result of his alleged involvement with an EHS death, public health is at stake. The Kentucky legislature responded in an impulsive manner, mandating that coaches receive enhanced education and training regarding emergency medical care of athletic injuries. As a result, coaches, who may be largely responsible for the condition in the first place, are now in charge of providing care to help their athletes survive. This is the gut reaction of a sympathetic and caring legislature, but it does not solve the core problem. Coaches are great at coaching, and they should be encouraged to continue that pursuit with all the vim and vigor they can muster. However, athletic health care should be left to the medical professionals—athletic trainers and team physicians, who work tirelessly to create the safest environment possible. If a serious condition does occur, we are prepared to determine the exact condition that is causing the medical emergency and invoke a life-saving plan to maximize the odds of survival. I have long said that we should do whatever we can "before they die," as the gut-wrenching reality of my everyday existence is that most of the people who contact me do so after a tragedy. I lend a sympathetic ear to the traumatized parent, coach, or friend. Yet I ultimately must deliver the truth to the family and friends, that the death from EHS was completely preventable. Crushing to hear but the honest truth. It is long overdue for the knowledge we have gained from research regarding the prevention of sudden death in sport to become a greater part of our clinical practice, and the first step in that process is being sure that every high school has an athletic trainer on-site who can assume responsibility for athletic health care.

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# Electrocardiogram Testing During Athletic Preparticipation Physical Examinations

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**Context:** Sudden cardiac death (SCD) is a relatively rare yet unfortunate risk of athletic participation. To reduce the incidence of SCD, electrocardiogram (ECG) use during athletic preparticipation examinations (PPEs) has been proposed to detect underlying cardiac abnormalities.

**Objective:** To estimate the effectiveness of ECG use during athletic PPEs.

**Design:** Epidemiologic modeling.

**Populations:** Public high school athletes.

**Data Collection and Analysis:** Estimates of ECG sensitivity (70%) and specificity (84%) were drawn from the literature, as was the estimate of overall prevalence of cardiac conditions relevant to SCD (0.3%). Participation rate by sex was determined from National Federation of State High School Associations data. Participation by ethnicity was assumed to be proportionate to the public high school attendance rates for grades 9 through 12 (18.4% African American). Population-specific ECG effectiveness (positive predictive value), estimated total costs, cost per year of life saved, and cost to identify 1 additional case were computed. Total annual PPE screening

costs reflected a cardiologist's office visit, including echocardiogram for those athletes with a positive ECG screen.

**Results:** The model predicted that 16% of all athletes would be expected to have a positive ECG, but only 1.3% of athletes with a positive ECG would have a cardiac abnormality capable of causing SCD, including hypertrophic cardiomyopathy, structural defects, and various conduction abnormalities. Total annual cost estimates for ECG screening and follow-up exceeded \$126 million. Average cost per year of life saved across groups was \$2693, and the cost to identify 1 additional case averaged \$100 827. Compared with females, males had both lower cost per year of life saved and lower cost to identify 1 true case. Similarly, black males exhibited lower costs than white males. Across groups, false-positive ECG screening exams accounted for 98.8% of follow-up costs.

**Conclusions:** Large-scale, mass ECG testing would be a costly method to identify athletes with cardiac abnormalities. Targeting high-risk populations can increase the effectiveness of the ECG for athletic PPE screening.

**Key Words:** cardiac abnormalities, sudden cardiac death, prevalence

## Key Points

- Mass electrocardiographic screening during high school preparticipation physical examinations would likely be very costly, due in part to low positive predictive value and high follow-up costs resulting from excessive false-positive tests.
- Electrocardiographic screening effectiveness could be improved by testing only those athletes who are identified as being at high risk for cardiac abnormalities that could cause sudden death.

Sudden cardiac death (SCD) is a rare but tragic event associated with athletic participation. Few warning signs exist to indicate the presence of an abnormality capable of causing SCD<sup>1,2</sup>; as a result, death itself can be the first indication of an underlying cardiac condition.<sup>3</sup> Screening for cardiac conditions associated with SCD often occurs via school-based mass preparticipation physical examinations (PPEs) of hundreds of athletes in a single day. At present, however, no method exists to effectively and consistently identify those athletes at risk for SCD in a mass screening scenario. Consequently, many athletes are likely participating in organized athletics even though they harbor underlying cardiac conditions capable of triggering SCD.

The overall prevalence of cardiac abnormalities responsible for SCD has been estimated<sup>4</sup> to be 0.3% (3 in 1000) in the general athlete population. Actual incidence rates of SCD are markedly lower; relatively few cardiac abnormalities lead to a fatal event in young athletes. Both sex and ethnicity appear to play roles in the risk of SCD. Males experience SCD nearly 10 times more often than females,

and the incidence among African Americans is higher than in other ethnic groups.<sup>5</sup> Prevention of SCD in these athlete populations requires early recognition of those conditions known to cause SCD.

Athletic PPE screening standards in the United States are typically determined by state legislation, state athletic associations, or individual school districts<sup>4,6</sup> and most often consist of a medical history review and physical examination.<sup>4</sup> Despite the lack of a standardized PPE protocol, 2 widely cited monographs offer guidelines. The first publication,<sup>7</sup> a joint effort by 6 medical associations, recommends a comprehensive PPE at least every 2 years, including a cardiac-specific medical history and a dynamic cardiac evaluation. The second publication,<sup>4</sup> from the American Heart Association (AHA), recommends a thorough medical history in conjunction with a dynamic cardiac evaluation. Although these guidelines present a rational approach for reducing the incidence of SCD in athletes, a universal standard for screening high school athletes does not presently exist.<sup>4</sup> In 2005, only 3 states had PPE forms containing all 12 recommended AHA screening



items; 19 states were missing 3 or more AHA items, and 2 states had no approved questionnaire forms to use for the PPE.<sup>6</sup>

One mechanism proposed to reduce the incidence of SCD in athletes involves the integration of an electrocardiography (ECG) examination into athletic PPE screening.<sup>8–11</sup> Those in support of ECG use during athletic physical examinations often point to the success of Italy's athletic screening protocol, which includes cardiac evaluation via 12-lead ECG.<sup>9</sup> Since implementation of this program in 1982, the annual incidence of SCD in the Veneto region of Italy has decreased by nearly 90%, falling from 3.6 per 100 000 person-years in 1980 to 0.4 per 100 000 person-years in 2004.<sup>12</sup> Critics argue that mass ECG screenings create multiple problems, including practical difficulties in screening large populations, the relatively high number of athletes who will need follow-up cardiac testing, low sensitivity and specificity of resting ECGs among athletic populations,<sup>4</sup> and the lack of a standard for interpreting ECGs in athletes.<sup>13</sup>

The purpose of our study was to evaluate the population-specific effectiveness and costs of adding ECG screening to the PPE among public high school athletes in the United States using a simulation model. Effectiveness of ECG screening was investigated by determining the positive and negative predictive values of the ECG, based on published diagnostic factors in a similar population as well as the expected number of additional cardiac conditions detected via ECG screening as compared with a traditional PPE screening protocol. Costs were estimated using current (2009) Medicare reimbursement rates for Current Procedural Terminology (CPT)<sup>14</sup> for a follow-up cardiology examination for all athletes with positive ECG findings during screening. Costs related to ECG screening were evaluated by determining specific total costs per population, cost per year of life saved, and cost to identify 1 additional case.

## METHODS

We estimated high school athlete populations by using the overall public high school athletic participation rates stratified by sex and ethnicity, because these factors are associated with the rate of SCD. Population data for male and female participation in public high school athletics were drawn from the National Federation of High School Association's 2006–2007 rates.<sup>15</sup> High school athletic participation data were not available by ethnicity,<sup>16</sup> so we estimated ethnic participation rates using the 2005 US Census Bureau School Enrollment<sup>17</sup> statistics for the census groups *black* and *all students* enrolled in grades 9 through 12, and we assumed that high school athletic participation rates by ethnicity were proportional to enrollment. Data from the Census Bureau indicated that 18.4% of students in grades 9 through 12 would fall into the *black* category and 81.6% into the *nonblack* category, which we defined as *white* for the purposes of this study. We used a dichotomous ethnic categorization as a result of prior SCD estimates, which were based on a similar comparison of African American versus white athletes and the lack of published prevalence or ECG sensitivity or specificity data for other ethnicities, such as Hispanic or Asian. In our model, we assumed that the prevalence of

cardiac abnormalities and sensitivity and specificity of ECG for detecting those abnormalities among non-African American, nonwhite high school athletes were equivalent to those in white athletes. All participation estimates were then divided by 4 to represent each athlete receiving an ECG during the PPE only once during a 4-year high school career.

For purposes of this study, *cardiac abnormalities* included those potentially fatal conditions previously reported<sup>18</sup> to be detectable by ECG, including hypertrophic cardiomyopathy (HCM), anomalous coronary artery, dilated cardiomyopathy, congenital aortic stenosis, myocarditis, arrhythmogenic right ventricular dysplasia, and primary conduction abnormalities. Although HCM is the primary cause of SCD in the United States, the prevalence value of HCM alone would be somewhat lower than that for the combined prevalence of any of these abnormalities. We applied the combined prevalence in our model under the assumption that the purpose of adding ECG to the PPE process would be to detect any potential cardiac abnormality, rather than a single disease entity. Overall prevalence of cardiac abnormalities capable of causing SCD across both sex and ethnicity groups in the US population has previously been estimated at 0.3%.<sup>4</sup> In our model, we assumed that the sex distribution of cardiac abnormalities was the same as the sex distribution of SCD (9:1 male to female ratio).<sup>19,20</sup> Ethnic distribution of events capable of causing a SCD event was based on prior research<sup>5</sup> of 286 cases of SCD in predominantly young athletes, indicating that 42% of those cases had occurred in athletes of African American ethnicity, which is consistent with evidence that African Americans experience a disproportionately high rate of SCD.<sup>5,21,22</sup> Furthermore, this value is consistent with the reported<sup>5,23</sup> range (36%–55%) of SCD due to HCM in black athletes.

Prevalence estimates using the proportions listed above were generated for 4 groups: black males (BM), white males (WM), black females (BF), and white females (WF). We assumed that all athletes would undergo the 12-point AHA cardiovascular screening history and physical examination with the addition of an onsite ECG screen. Prior published sensitivity and specificity estimates were found using a PubMed search for English-language articles with a combination of relevant search terms, including *sudden cardiac death*, *high school athletes*, *ECG*, *sensitivity*, and *specificity*. Results from this search were limited to those articles reporting data specific to an athletic population. Sensitivity and specificity of the AHA screening method in high school athletes have been estimated<sup>18</sup> to be 6% and 98%, respectively. Sensitivity of ECG among elite and professional athletic populations has been estimated to range from 51% to 70%, and specificity has been reported in the range of 61% to 98%.<sup>24,25</sup> For purposes of this study, we used 70% sensitivity and 84.3% specificity, based on observations derived from testing high school athletes, the target population of the current study.<sup>25</sup>

Predictive values and costs were estimated for each sex and ethnic group (Table 1). The positive predictive value (PPV = true positive cases/total positive tests) and negative predictive value (NPV = true negative cases/total negative tests)<sup>26</sup> of using ECG during PPE were computed based on the sensitivity and specificity values. The PPV is the probability of a patient with a positive test having the

**Table 1. Estimating Sex-Specific and Ethnic-Specific Predictive Values and Cost: Numeric Example**

Step 1. Model contingency table

ECG Results	True Disease Status		Total
	Positive	Negative	
Positive	a	b	a + b
Negative	c	d	c + d
Total	a + c	b + d	a + b + c + d

Step 2. Obtain model values from literature

Total population (black males) = (a + b + c + d) = 202 012

Prevalence = (a + c)/(a + b + c + d) = 1.03%

Sensitivity = a/(a + c) = 0.700

Specificity = d/(b + d) = 0.843

Step 3. Enter derived table values

(a + c) = (a + b + c + d) × prevalence = (a + b + c + d) × 1.3% = 202 012 × 0.0103 = 2081

(b + d) = (a + b + c + d) - (a + c) = 202 012 - 2081 = 199 931

a = (a + c) × sensitivity = (a + c) × 0.70 = 2081 × 0.70 = 1457

c = (a + c) - a = 2081 - 1457 = 624

d = (b + d) × specificity = (b + d) × 0.843 = 199 931 × 0.843 = 168 542

b = (b + d) - d = 199 931 - 168 542 = 31 389

(a + b) = 1457 + 31 389 = 32 846

(c + d) = 624 + 168 542 = 169 166

Step 4. Complete estimated contingency table

ECG Results	True Disease Status		Total
	Positive	Negative	
Positive	1457	31 389	32 846
Negative	624	168 542	169 166
Total	2081	199 931	202 212

Step 5. Estimate predictive values and total cost

PPV = a/(a + b) = 1457/32 846 = 4.4%

NPV = d/(c + d) = 168 542/169 166 = 99.6%

Total cost = (total population × onsite fee) + (total positive ECGs × follow-up fee) = (202 012 × \$10) + (32 846 × \$371) = \$14 205 986

Abbreviations: ECG, electrocardiography; NPV, negative predictive value; PPV, positive predictive value.

disease, whereas the NPV is the probability of a patient with a negative test being free of the disease. We also estimated the number of additional athletes with cardiac abnormalities who would be identified if ECG was added to the PPE relative to the number identified by the PPE without ECG.

Cost of the onsite history and physical examination with ECG administration was assumed to be \$10, based on the cost of mass PPE screenings in our geographic region. We assumed that a cardiologist would not be on site at the PPE to evaluate which athletes would need further medical evaluation, so the ECG results would be evaluated by the physician conducting the PPE. We assumed that all athletes who had an ECG abnormality of any type would be referred to a cardiologist for further testing. Based on consultation with a panel of local cardiologists, follow-up cardiac testing for a positive ECG was assumed to include an office visit and echocardiogram. Costs for office-based follow-up testing were assumed to be \$371 based on the 2009 Medicare Physician Fee Schedule (adjusted using the Geographic Practice Cost Index for our region) for CPT codes 99205 (office visit) and 93303 (transthoracic echocardiogram).<sup>14</sup> These costs represent an estimate of reimbursement rather than billed amounts or actual expenses for the initial cardiology consultation after a positive ECG in the PPE. Additional office visits and

subsequent testing or treatment that may result from the initial consultation were not estimated.

Total years of life saved were determined using a previously reported method<sup>18</sup> that assumes 10% of those high school athletes with a diagnosed cardiovascular abnormality will live an additional 40 years and 90% will live an additional 20 years. Total cost per year of life saved was then calculated by dividing the total cost of ECG testing and associated follow-up costs by the total years of life saved. Total cost to identify 1 additional case was calculated by dividing the total costs by the number of additional cases identified by adding the ECG to the PPE.

## RESULTS

The model predicted that 16% of all athletes would be expected to have a positive ECG, whereas only approximately 2% would have a positive finding representing a potential cardiovascular abnormality on the standard PPE history and physical examination. Across the entire population, only 1.3% of athletes with a positive ECG would be expected to have a cardiac abnormality. The diagnostic capability of the ECG in this model, including expected number of abnormal ECGs, total years of life gained via ECG screening, and predictive capability of ECG screening per group, is presented in Table 2.



**Table 2. Predictive Values for Electrocardiography (ECG) Screening of High School Athletes**

Group	Total Population	Predicted Abnormalities	Abnormal ECGs	Total Years Gained via ECGs	PPV, %	NPV, %
Black males	202 012	2081	32 846	42 592	4.44	99.63
White males	878 264	2875	139 448	58 817	1.43	99.88
Black females	141 269	275	22 329	5634	0.86	99.93
White females	614 182	275	96 576	5634	0.20	99.98
Total	1 835 727	5506	291 199	112 677	1.31	99.89

Abbreviations: NPV, negative predictive value; PPV, positive predictive value.

Predictive values of the ECG test were highest when testing male athletes, reflecting the effect of increased disease prevalence on PPV (Figure 1). Screening of BM athletes in particular indicates that approximately 4 of every 100 positive ECGs would result in discovery of a true cardiac abnormality, whereas screening of all female athletes would detect less than 1 true cardiac abnormality per 100 positive ECG examinations. Therefore, a positive ECG would be rather costly for identifying athletes with a cardiac condition, because relatively few athletes who have a positive ECG and receive cardiologist examination, including echocardiography, would actually be expected to have a cardiac abnormality. Across groups, NPV was lowest among BM at 99.63%; all other groups exceeded 99.88%. Such high NPVs across groups indicate that nearly all athletes ( $\geq 99.6\%$ ) with a negative onsite ECG would, in fact, be free of cardiac abnormalities capable of causing SCD.

The expected outcomes from adding ECG screening to high school PPEs are listed in Table 3. The ratio of additional true-positive to additional false-positive cases found would be highest among BM (1:25), followed by WM (1:76), BF (1:126), and WF (1:550). These ratios indicate the respective inefficiencies of ECG screening among the sex-ethnicity populations. For example, for every case of a black male with a cardiac abnormality identified by implementing ECG screening, 25 BM with no abnormality will have a positive ECG that requires further medical testing. Differences in these inefficiency ratios among the groups reflect the respective group differences in the prevalence of cardiac abnormalities.

Costs associated with ECG examination during high school PPEs are listed in Table 4. Total combined annual costs for onsite ECG screening and follow-up cardiac testing for all athletes entering high school (ie, freshmen)

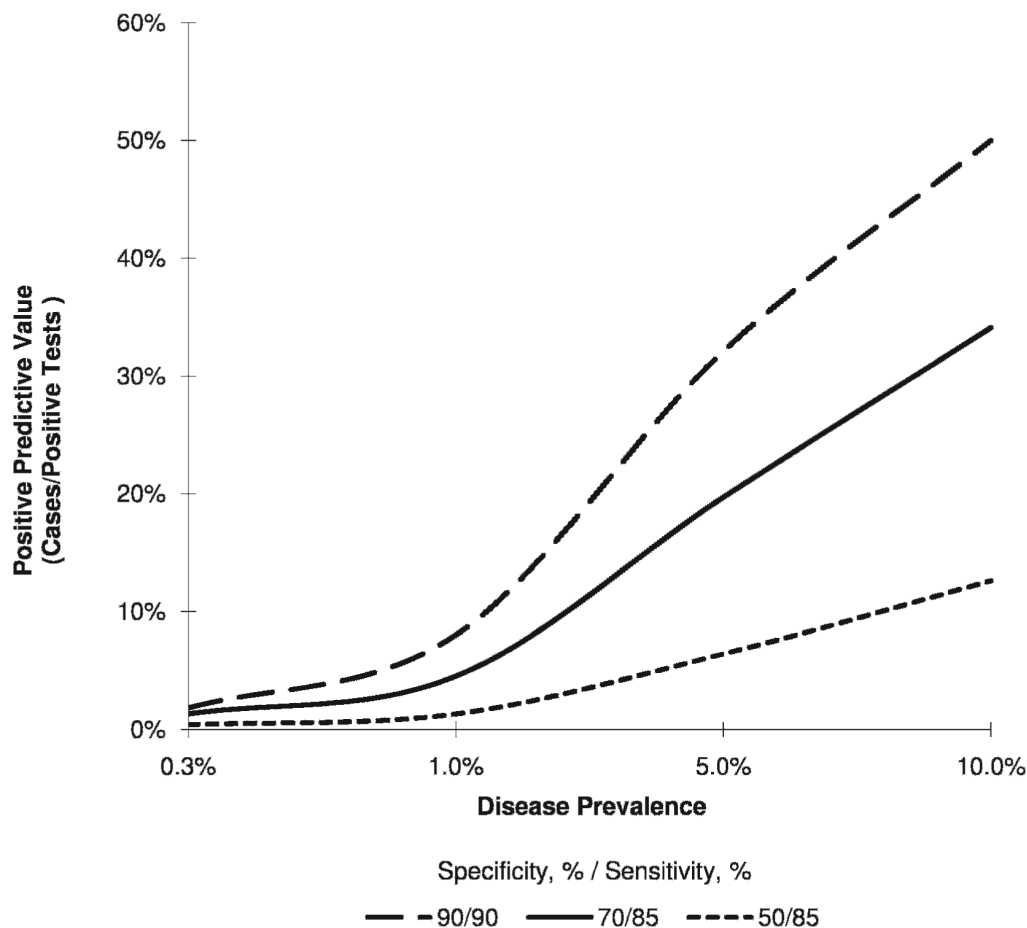


Figure 1. Positive predictive value of electrocardiography and disease prevalence.

**Table 3. Expected Results of Adding Electrocardiography (ECG) to Preparticipation Physical Examinations**

Group	Additional True-Positive Cases After Adding ECG	Additional False-Positive Cases After Adding ECG
Black males	1332	32 563
White males	1840	138 245
Black females	176	22 136
White females	176	95 742
Total	3524	288 686

exceeded \$126 million. Onsite ECG screening accounted for less than 15% of the total cost. Approximately 98.8% of the costs for follow-up evaluation resulting from a positive PPE ECG came from testing athletes who had no cardiac abnormalities.

The cost to identify 1 additional true case was more than 3 times higher for WM than for BM and more than 4 times higher for WF than for BF (Table 4). Within ethnic groups, the cost to identify 1 additional case was 5 times higher for BF than for BM and more than 7 times higher for WF than for WM.

## DISCUSSION

Our model shows that use of the resting 12-lead ECG during PPE screening for incoming high school athletes will result in high costs because of a large number of false-positive tests. This finding is consistent with prior reports<sup>4,27–30</sup> and is due in part to the relatively low cardiac disease prevalence among high school athletes. When the prevalence of a disease is low, as is the case with cardiac abnormalities associated with SCD among high school athletes, nearly all positive ECG tests occur in individuals who do not have the disease. As the false-positive rate increases, the number of healthy athletes receiving unnecessary cardiology follow-up testing increases, which consequently increases costs.

Identifying any disease within a population depends upon the accuracy (sensitivity and specificity) of the test used in diagnosis. Among well-trained (ie, professional and elite competitive) athletes, the resting 12-lead ECG has been reported<sup>24</sup> to have 51% sensitivity and 61% specificity for detecting cardiac abnormalities, indicating that the test has poor diagnostic accuracy in that population.<sup>24,31,32</sup> By contrast, high school athletes are often not highly trained; therefore, we used a sensitivity of 70% and specificity of 84.3%, based on prior research<sup>18</sup> conducted in a high school population. Using the sensitivity and specificity values observed in trained athletes would result in an increase in false-positive tests and higher costs than we estimated.

The PPV of a diagnostic test decreases as disease prevalence decreases, as observed in the results of this

study. For example, the PPV for females was predicted to be less than one-fifth that of males as a result, in large part, of the very low prevalence of cardiac disease among females. This sex discrepancy indicates that ECG screening would be relatively ineffective in screening female athletes, because very few positive ECG results (ie, 1 in 500) would indicate the presence of a true cardiac abnormality. Higher disease prevalence shows similar discrepancies. For instance, our estimates indicate that the participation rate of BM to BF in high school athletics is nearly 3:2. However, the PPV among BM is 5 times higher than that for BF as a result of the higher expected disease prevalence among BM. Therefore, our model predicts that using mass ECG screening among a high school population would result in many false-positive test results, particularly in those subpopulations with the lowest expected disease prevalence. The NPV of our model ( $\geq 99\%$ ) was somewhat higher than a prior reported<sup>13</sup> expected value of 96%. It is clear from our model and prior work that ECG is very effective at ruling out the presence of cardiac abnormalities, as few athletes (1%–4%) with a negative ECG will have an actual cardiac abnormality. These PPV and NPV results indicate that the primary limitation of the use of ECG during PPE is a high rate of false-positive tests, and most athletes ( $\geq 97\%$ ) with a positive ECG will have no actual cardiac abnormality.

The lowest total cost to identify 1 additional true case as well as the lowest cost per year of life saved were found in BM, primarily because of the higher expected disease prevalence (resulting in identifying more true cases per total expense) among the BM population. A sex-specific analysis of costs revealed that male athletes had both a lower cost per year of life saved and a lower cost to identify 1 additional case than did female athletes. In contrast, relative to WM, WF accrued more than 10 times the cost in identifying 1 additional case and cost per year of life saved.

Based on our model's prediction of improved diagnostic capability among populations with higher disease prevalence, a screening protocol to identify and provide an ECG test for only those athletes with an increased probability of cardiac potential may be warranted. For example, ECG screening of male athletes can be expected to result in a higher predictive accuracy for recognizing underlying cardiac conditions. Similarly, screening of BM would result in a PPV that is more than 3 times higher than that for all other athlete groups as well as a cost to identify 1 additional case that is one-third the cost of the next lowest group (WM). Although we are not advocating providing ECG screening for specific sex or ethnic groups, substantial improvement in the ECG effectiveness during PPE for high school athletes can be expected by using ECG only for the athletes who are at high risk.

**Table 4. Costs Associated with Electrocardiography Use in Preparticipation Physical Examinations, \$**

Group	Total Costs (Onsite + Follow-Up)	Costs Due to False-Positive Cases	Costs Due to True-Positive Cases	Cost Per Year of Life Saved	Cost per Additional Case <sup>a</sup>
Black males	14 206 051	12 080 741	494 282	286	10 663
White males	60 518 023	51 288 780	682 580	879	33 294
Black females	9 696 690	8 212 484	65 381	1470	55 023
White females	41 971 569	35 520 450	65 381	6359	241 089
Total	126 392 333	107 102 455	1 307 624	2693	100 827

<sup>a</sup> Total cost/number of additional true-positive cases after adding electrocardiography (from Table 3).

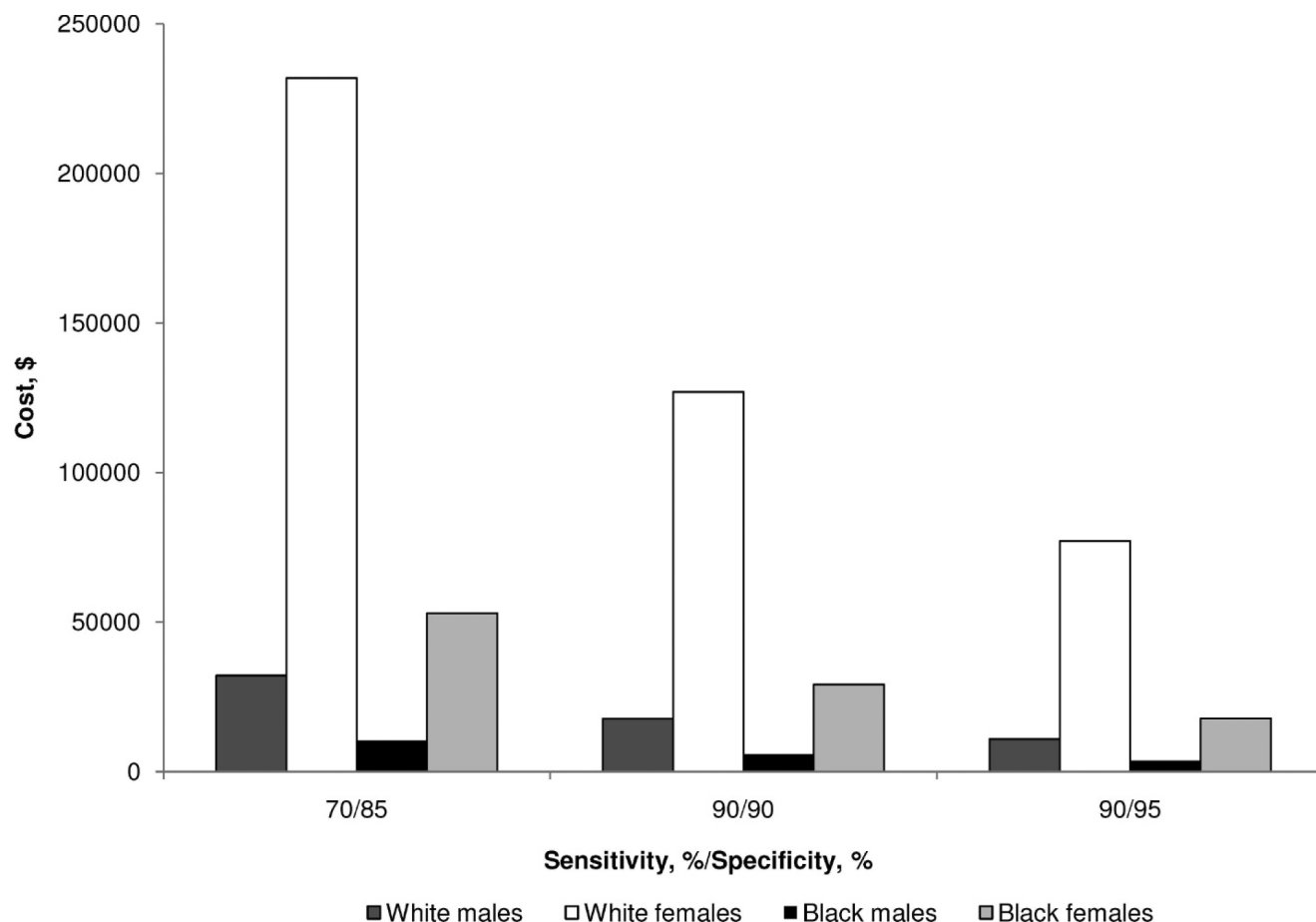


Figure 2. Cost of electrocardiography and sensitivity and specificity values.

Identifying individuals with a cardiac abnormality that is capable of causing an SCD event and withholding them from competitive athletic activity would be expected to result in a portion of those individuals being saved from a cardiac-related death. Unfortunately, the high false-positive rate associated with mass screening ECG can also be expected to introduce the psychological stress of being falsely labeled with a potentially fatal cardiac abnormality. In addition, the time frame from the positive onsite ECG examination through a full cardiology workup can be weeks, which may prevent the athlete from participating in sport. These factors should be considered when ECG screening as part of the PPE is being contemplated.

### Conflicting Protocols

Advocates for ECG implementation often point to the success of the Italian protocol in reducing the incidence of SCD<sup>12,33</sup> and identifying cardiomyopathies.<sup>10</sup> The incidence of SCD in Italy has decreased dramatically, from 3.6 to 0.4 per 100 000 individuals, since ECG screening was incorporated into athletic physical examinations.<sup>12</sup> However, 3 important issues arise when we attempt to generalize these results to American athletes. First, data from the majority of Italian studies were collected from predominantly white males, often localized to the Veneto region of the country. As a result, the athlete population considered in those studies does not represent the sexual

and ethnic diversity that is present among high school athletes in the United States. Second, the most common (25%) cause of SCD among Italian athletes is arrhythmogenic right ventricular dysplasia, also known as arrhythmogenic right ventricular cardiomyopathy,<sup>1,20,34</sup> a condition representing fewer than 5% of SCD incidents among young athletes in the United States.<sup>20,35</sup> Among the cardiovascular conditions capable of causing SCD, HCM is the most common (approximately 40%) cause of SCD in young American athletes.<sup>20</sup> Finally, the SCD incidence rate after the addition of ECG screening in Italy is strikingly similar to that reported in the United States without ECG screening<sup>3</sup>: 1 in 200 000 high school-aged participants. Therefore, based on population demographic discrepancies, varying cardiac abnormality prevalence rates, and differences in the reported rates of SCD, whether the Italian ECG screening protocol will reduce the SCD rate in the United States as effectively as in Italy is uncertain.

### Limitations

The primary limitation of this simulation study is the estimates used for prevalence, sensitivity, and specificity in our model. A range of values for each factor are reported in the literature. As a result, the data presented in this study could be expected to vary in accordance with alternative estimates for each measure (Figure 2). Despite this variation, the results would be expected to remain in



proportion to our findings, in large part as a result of the effect of disease prevalence on the PPV of a diagnostic test. Furthermore, actual costs depend upon follow-up tests selected by the athlete's individual cardiologist. These follow-up tests may differ from the physical examination and echocardiography included in our model, as may the regional reimbursement rates. Another limitation lies in the population used for this study. We evaluated the usefulness of ECG screening among public high school athletes in the United States. Absent from our modeled population are those athletes in private high schools, youth sports, recreational or club teams, and collegiate athletics. The inclusion of these additional athletic populations would result in a substantially higher total cost. Furthermore, accurate ECG interpretation is known to be challenging among the high school athlete population,<sup>13</sup> and the variability of interpretation among physicians can be an issue.<sup>13</sup> The sensitivity and specificity values used in our model represent averages of the theoretical population of physicians who would be interpreting ECG during PPE. Our model also stipulates that all cardiac conditions detected via ECG would result in cardiology follow-up; however, this may not hold true in practice, thereby potentially decreasing the costs associated with follow-up care.

## Application

Accurate detection of cardiac abnormalities associated with SCD in young athletes remains a priority for sports medicine professionals.<sup>36</sup> Despite a general perception that athletes represent the healthiest segment of society,<sup>20</sup> many remain susceptible to underlying cardiac abnormalities that, when combined with sport participation, increase their risk for SCD. One of the major challenges in preventing SCD is the relative unpredictability with which it occurs in the athletic population.<sup>20</sup> Some<sup>9,10,37</sup> have proposed that the 12-lead ECG examination be used to diagnose individuals with some types of cardiac abnormalities during preseason cardiac assessments despite ongoing debate regarding its effectiveness.<sup>4</sup> The low prevalence of cardiac conditions in a high school population can be problematic in terms of accurate detection. Therefore, in order to increase the accuracy of the onsite ECG test, either the instrumentation must be improved (ie, enhanced sensitivity and specificity), a combination of diagnostic tests should be used to identify high-risk individuals who are more likely to have the disease, or certain tests (eg, ECG) should be used only in populations with a relatively high prevalence of the disease.

Prior authors have investigated risk and prevalence of cardiac abnormalities in relatively small athlete populations. The need exists for a longitudinal study in very large populations of high school athletes to determine actual cardiac disease prevalence, SCD occurrence, and accuracy of ECG during PPE. Until such a study is conducted, the use of a model derived from prior estimates may provide reasonable expectations for adding ECG as a routine component of the PPE.

## Conclusions

Mass ECG screening during high school PPEs would be very costly and perhaps cost prohibitive, in large part because of the low PPV and high follow-up costs generated

by excessive false-positive tests. These high costs would be expected as a result of inconsistent ECG interpretation, the personnel required for administration and interpretation, and lost participation time during follow-up examinations. Effectiveness of ECG screening could be improved by testing only those athletes who are at high risk for harboring cardiac abnormalities capable of causing SCD.

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## **SICKLE CELL TRAIT AND THE ATHLETE**

**E. Randy Eichner, MD**

### **KEY POINTS**

- Sickle cell trait is an inherited condition of the oxygen-carrying protein, hemoglobin, in red blood cells. This genetic trait is generally benign, but during maximal exercise, the oxygen levels in muscles can decrease sufficiently to cause some of the red cells to change from the normal disk shape to a crescent or sickle shape. These sickled red cells can block blood vessels in muscles, kidneys, and other organs and can pose a grave risk for some athletes exercising all-out.
- In the past 6 1/2 years alone, exertional sickling has killed nine athletes, including five college football players. Death is caused by complications of a sudden and extreme breakdown of muscle tissue: cardiac arrhythmias and/or acute kidney failure.
- Sickling can begin in 2-3 minutes of sustained, maximal exertion, such as wind sprints in football or running laps in basketball. The harder and faster the athlete goes, the earlier and greater the sickling.
- The exertional sickling setting and syndrome are unique and can easily be differentiated from heatstroke or heat cramping. Sickling risk is increased by anything that increases the difficulty of the exercise, for example, hot weather, dehydration, high altitude, or asthma.
- Screening and precautions for sickle cell trait can prevent deaths from exertional sickling and enable sickle-trait athletes to thrive in their sports.

### **INTRODUCTION**

Sickle cell trait is common and generally benign. More than 3 million Americans have sickle trait and almost all live healthy, normal lives. Yet for some athletes, sickle trait can pose a grave problem – a problem that can even cause death. Understanding sickle cell trait is vital to athletes, coaches, and athletic trainers because sickling injuries are preventable with screening and proper precautions.

The sickle gene is common in people of African heritage; it produces a variant of hemoglobin, the oxygen-carrying protein of the red blood cell. Over the millennia, carrying one sickle gene—sickle cell trait—fended off death from malaria, leaving one in 12 African-Americans (versus one in 2,000 to one in 10,000 white Americans) with sickle trait. Athletes with sickle trait inherit one gene for normal hemoglobin and one gene for sickle hemoglobin (hemoglobin S). If oxygen in tissues falls to low levels, the red cells carrying the hemoglobin S can change from the usual disk shape to a crescent or sickle shape. These sickled red cells can clog blood vessels, impairing delivery of oxygen and removal of harmful metabolites, resulting in severe damage to the involved tissues.

In the past four decades, sickling has killed up to a dozen college football players during training and many more military recruits and others in boot camps (Eichner, 1993). In the past 6 1/2 years alone, sickling has killed nine athletes: five college football players, two high school athletes (including one basketball player), and two 12-year-olds training for

football. Other recent non-fatal cases exist as well. In a review of sudden nontraumatic sports deaths over a decade in high school and college athletes, sickling-associated rhabdomyolysis (accumulation in the blood of myoglobin, potassium, and other substances from damaged muscle fibers) accounted for seven (5%) of 136 well-studied deaths (Van Camp et al., 1995).

The focus of this article is on sickling in sports, but the initial reports of sickling collapse and death – and the seminal field and laboratory research on the problem – come from the U.S. military.

## RESEARCH REVIEW

### Exertional Sickling Collapse: Military Experience

Fatal sickling collapse was first described in 1970, in a report of sudden deaths in four of about 4,000 black recruits (about 0.1%) during one year of Army basic combat training at Fort Bliss, Texas, altitude 4,060 feet (Jones et al., 1970). The discussion in that report begins, "That sickle cell trait can, under certain circumstances, be fatal is generally not appreciated." From 1970 to 1974, four similar collapses (with one death) in Air Force recruits and cadets with sickle trait – two of the collapses at an altitude of only 661 feet – increased the alarm and resulted in the temporary barring of applicants with sickle trait from the Air Force Academy (Koppes et al., 1977). These four were the only recruits, among thousands of trainees seen at two military bases, hospitalized for exertional rhabdomyolysis, for kidney failure caused by the accumulation of myoglobin in the kidney blood supply, and for widespread clotting of blood throughout the body (disseminated intravascular coagulation or DIC). All needed early dialysis for kidney failure; one died of heart failure associated with excessive accumulation of potassium in the blood (hyperkalemia). The illness followed vigorous exercise: two were running 1-2 miles and two were on an obstacle course. The weather was not stressful, and no case was tied to heatstroke.

Three more military cases—virtually identical, and all fatal—were reported between 1974 and 1985. All three men collapsed after running 2-3 miles and all died from complications of acute exertional rhabdomyolysis and kidney failure.

A noted epidemiological study in 1987 strengthened the link between sickle cell trait and sudden death during physical training. Of all deaths that occurred among two million enlisted recruits during basic training in the U.S. Armed Forces over the five years from 1977 through 1981, the risk of sudden unexplained death in black recruits with sickle trait was 28 times higher than in black recruits without sickle trait and 40 times higher than in all other recruits (Kark et al., 1987).

On further analysis, the relative risk of exercise-related death in sickle trait, unexplained by pre-existing disease, was set at 30 times higher than in recruits without sickle trait. The risk of death for recruits increased with age, an 8-fold increase from age 17-18 to age 28-29. The risk plummeted, however, for career military after basic training. This observation led military researchers to speculate that the risk of exercise-related death in sickle trait is largely confined to a time of intense conditioning to unaccustomed exercise or a sustained event at a performance level for which the individual is unprepared (Kark & Ward, 1994).

Analysis also clarified the type of death. Most collapses occurred as recruits tried to run 1-3 miles. Of 40 military deaths or near-deaths from sickling collapse, some had features of exertional heat illness (but not heatstroke) and others were sudden cardiac death from arrhythmia. But most deaths were not sudden – occurred hours to a day or two after the collapse – and were from complications of extreme rhabdomyolysis, including muscle compartment syndromes (breakdown of swollen muscles confined by surrounding tough connective tissue) and myoglobinuric kidney failure. Indeed, acidosis and hyperkalemia from explosive rhabdomyolysis may have caused the arrhythmic sudden cardiac

deaths. So the main cause of death in sickling collapse is fulminant exertional rhabdomyolysis, a risk that rises 200-fold in recruits with sickle trait (Gardner & Kark, 1994).

## Physiology and Pathophysiology

Exercise physiology helps explain why extreme exertion in sickle trait can cause explosive exertional rhabdomyolysis. In sickle trait, vigorous exercise can evoke sickling that can have grave consequences. For decades, the concept was that, except in the medulla of the kidney, sickle-trait red cells "never sickle in live people" or that sickle trait "is innocuous" because no sickling occurs until blood oxygen saturation falls below 40%, a level not reached until red cells reach the venous exits of the tissue capillaries (Eaton & Hofrichter, 1987). This notion, however, fails to fathom the major metabolic changes of intense exercise – and their consequences.

When young men cycle to exhaustion in five minutes, for example, striking acidosis and low blood oxygen levels can occur: the femoral venous blood pH can fall to 7.15, the oxygen saturation to 19% (Hartley et al., 1973). With the same exercise at 4,000 m, blood oxygen saturation can fall as low as 11% (Hartley et al., 1973). When sprint-trained men run on a treadmill at a speed that exhausts them in one minute, femoral artery blood lactate levels soar and pH falls to 7.07 (Medbo & Sejersted, 1985). And when athletic men cycle or run to exhaustion in 1-2 minutes, arterialized capillary blood shows lactate levels up to 32 mM, bicarbonate levels down to 3 mEq/L, and blood pH values as low as 6.8 (Osnes & Harmansen, 1972). In addition to these extreme metabolic stresses, blood perfusing exercising muscles is exposed to excessive temperatures and to a hyperosmotic environment (partly from breakdown of large molecules of glycogen to multiple molecules of lactate) that moves water from red cells into the tissue fluids, thereby dehydrating the red cells (Eichner, 1993).

So in sickle trait, strenuous exercise evokes four forces that foster sickling. The acidosis and high tissue temperatures "shift to the right" the oxygen dissociation curve, displacing more oxygen from hemoglobin S. Dehydration of red cells increases the concentration of hemoglobin S. And a severe decline in blood oxygen because of extreme muscle demand for oxygen completes the sickling foursome.

Exactly where the change in red cell shape from disk to sickle occurs is debated. Most red cells traverse the capillary circulation in about one second. It is argued that for sickle-trait red cells the "delay time" to sickling, even at zero percent oxygen saturation, is slightly longer than one second, so even with extremely low blood oxygen levels, the red cells would escape the microcirculation before they sickle. In other words, any sickling that occurs must be in the veins, where it is "harmless" because any sickled cells will revert to normal shape as they take up oxygen in the lungs.

Two flaws weaken this hypothesis. First, red-cell dehydration makes normally nonadherent sickle-trait cells adhere to vascular endothelium, so some may linger in the microvasculature and sickle there (Hebbel, 1991). Second, even if most of the sickling occurs in veins leaving exercising muscles, some sickle cells fail to revert to normal in the lungs. Indeed, in an early study, when five black men with sickle trait performed vigorous leg cycling, nearly 1% of red cells in venous blood from the arm were sickle cells (Ramirez et al., 1976). So sickle cells likely accumulate in the blood during strenuous exercise and are pumped to the working muscles where they can "logjam" the microcirculation and cause rapid ischemic breakdown of muscles – acute, severe rhabdomyolysis. Exercise studies support this hypothesis.

## Exercise Laboratory Studies: Military

Exercise research on military recruits confirms sickling in blood draining exercising muscles. Fifteen sickle-trait men did two brief, maximal arm-cranking exercise tests, one at 1,270 m and one at a simulated 4,000 m (Martin et al., 1989).

Sickle cells were counted in venous blood draining the arms. At 1,270 m, exercise evoked a mean of 2.3% sickle cells; at 4,000 m, this increased to 8.5%. One recruit, exercising maximally at 4,000 m (28% oxygen saturation), had 25% sickle cells in venous blood from exercising muscles.

A second military study published in abstract form shows that sickle cells can accumulate in the arterial circulation (Weisman et al., 1988a). Recruits with sickle trait exercised to near exhaustion on a cycle ergometer, once at sea level and again at a simulated 4,000 m. Sickle cells were counted at peak exercise in venous blood from the forearm. Such cells likely sickled in the exercising lower limbs and traversed not only the lungs but also another capillary bed, that of the (resting) forearm. At sea level, sickle cells in forearm blood reached only about 1%. But at 4,000 m, sickle cells rose to a mean of 9% and a maximum in one man of 28%. This strongly suggests that as exercise stress increases and blood oxygen declines, athletes with sickle trait will steadily accumulate sickle cells that are pumped to the heart, brain, and muscles.

In a third military study, men with sickle trait, cycling hard at 4,000 m, developed more symptoms than control men (Weisman et al., 1988b). Symptoms included headache or dizziness, leg cramps, chest pain, and/or pain in the left upper quadrant of the abdomen. Symptoms developed in more men who had sickle trait (9 of 30) than in control men (2 of 28). Left upper quadrant pain, likely from sickling in the spleen, occurred only in men (3 of 30) with sickle trait. For sickle-trait athletes, the implications of these studies are obvious.

## Exercise Laboratory Studies: Civilian

Civilian studies complement the military studies. A 2004 study by Bergeron et al. suggests that dehydration can compound sickling during exercise. Two men with sickle trait walked briskly for 45 minutes in the heat, once while drinking fluids (1 L, to offset sweat losses) and once without fluids. Without fluids to offset dehydration, sickling (in forearm venous blood) increased steadily to peaks of 3.5% and 5.5%, respectively. In this relatively mild exercise bout, no sickling was evident when fluids were consumed (Bergeron et al., 2004).

Several studies keyed on small groups of sickle-trait subjects versus controls performing various anaerobic and/or aerobic exercises (Bile et al., 1996, 1998; Freund et al., 1995; Gozal et al., 1992; Sara et al., 2003). In general, performance differences between groups were not detected or were small and mixed. In other words, most of these studies suggest that subjects with sickle trait have normal exercise ability. Small differences observed in blood lactate accumulation or removal—lactate either higher or, more commonly, lower in sickletrait subjects – may be from imperfect matching of groups for fitness.

Very recent laboratory exercise studies suggest microcirculatory problems in sickle trait. For example, sickle-trait subjects have more rigid red blood cells at rest and a longer-lasting rise in certain vascular cell-adhesion molecules with exercise (Connes et al., 2006c; Monchanin et al., 2006). In constant strenuous cycling exercise for nine minutes, sickle-trait subjects, compared to controls, are not limited initially but are prone to exercise intolerance and lower aerobic capacity thereafter (Connes et al., 2006a). Also, when sickle-trait subjects do five 6-second maximal anaerobic cycling sprints, performance is normal on the first sprint but falls off faster (than in controls) during repeated sprinting (Connes et al., 2006b). These results tend to jibe with field studies in Africa that suggest sickle-trait runners gravitate to shorter distances (Marlin et al., 2005).

## Field Studies in Africa

Observational studies in Africa find that sickle-trait runners fare better in sprints than in longer races - and struggle at



altitude. For example, among 129 champion runners in Ivory Coast, 13 (10%) had sickle trait. These 13 runners won 33 titles, but only one was in a race of 800 m or more. The other 32 wins were all in races of 400 m or less (Le Gallais et al., 1991). A similar field study found sickle-trait runners underrepresented among top finishers in an Abidjan semi-marathon (Le Gallais et al., 1994). In a rugged distance race, the Mount Cameroon Ascent, among runners of the Bakoueri tribe, where winning confers social status, sickle-trait runners were underrepresented in general and underperformed in the high-altitude stretches (Thiriet et al., 1994). These observations, along with the laboratory studies above, suggest that sickle trait can limit performance not only at altitude but also in any all-out running that approaches or exceeds 800 m. This is borne out by observations in American college football players.

## Exertional Sickling Collapse: College Football

The first sickling death in football was in 1974, a black defensive back from Florida who collapsed two years in a row on the first day of practice at altitude (5,118 feet) at the University of Colorado (Eichner, 1993). The first year, he survived. The second year, aiming to finish the first conditioning sprint

(880 yards), he fell behind his group after 660 yards, staggered forward for a while, then fell at the edge of the track. He complained of severe leg pain. The next day, he died in the hospital, with "severe acidosis" and "severe sickling in the kidneys."

Up to 13 college football players have died after sickling in training, and although in some the proximate cause of death has been debated (and litigated), the setting and syndrome in most are similar (Table 1).

TABLE 1. Deaths after sickling collapse in college football.

Year	State	Activity to Collapse
1974	Colorado	Ran 700 meters
1985	Arkansas	Ran ¾ mile
1986	Mississippi	Ran 1 mile
1987	Indiana	Ran 1200 meters
1989	Utah	Ran ¾ mile
1990	New Mexico	Ran 800 meters
1992	Georgia	Ran 1000 meters
1995	Arizona	Ran 900 meters
2000	Tennessee	Ran 800 meters
2001	Florida	Intense drill, 1 hour
2004	Ohio	Ran for 10 minutes
2005	Missouri	Field drill, 1 hour
2006	Texas	Ran 1600 yards

The features of the sickling collapse syndrome are unique, but are not yet widely recognized (Rosenthal & Parker, 1992). Take the above Tennessee case (2000), for example. On the first day of summer practice, this second-year defensive back passed a physical examination. At 5-feet 11-inches and 190 pounds, he was the picture of health. He had kept fit all summer. One hour after passing the physical, he took the field for the conditioning test. He wore shorts and T-shirt. The field temperature was in the mid-80s, the humidity low by local standards. He breezed through the first two-thirds of the test, two series of five 60-yard sprints, 600 yards total. He collapsed late in the last third of the test – he ran less than 900



yards total.

The player was taken to a nearby hospital, where he soon died. Lacking a core temperature, the coroner attributed the death to heatstroke. The evidence points instead to exertional sickling and acute rhabdomyolysis. The coroner was unaware the victim had sickle cell trait (Eichner, 2000).

## Sickling Situations

Sickling collapse is not limited to college football training. It has killed or nearly killed several college or high school basketball players (two were female athletes) in training, typically when "running for time," such as repeated sprints up and down the basketball court ("suicide runs") or laps on a track or a 3-mile run (Eichner, 1993, 2000). A sickle-trait runner collapsed two years in a row at the end of cross-country races. He survived severe rhabdomyolysis and renal failure (Helzlsouer et al., 1983). A medical student in Chicago barely survived a sickling collapse after jogging a vigorous mile for the first time in years; he was lucky that he collapsed very near the hospital (Eichner, 1993). Sickling has killed firefighters in training (Wirthwein et al., 2001) and men captured by police after chase and struggle (Mercy et al., 1990; Thogmartin, 1998).

The harder and faster athletes go, the earlier and greater the sickling. That is why sickle collapse occurs sooner (at a shorter distance) in top college football players sprinting all out (their "drop zone" is often 800-1,200 m, see Table 1) than in military recruits trying to run 1-3 miles. Sickling can begin in only 2-3 minutes of sustained sprinting – or in any other sustained all-out exertion – and sickling can soon increase to grave levels if the symptomatic athlete tries to exceed his limits or is urged by the coach to push beyond pain and weakness. Any cramping, struggling, or collapse in a sickle-trait athlete must be considered sickling—a medical emergency—until proved otherwise.

The degree of exertional sickling also seems to vary with the percentage of hemoglobin S in each red cell. Typically in sickle trait, each red cell has about 40% S and 60% A (normal) hemoglobin. But if the athlete co-inherits a trait for alpha thalassemia, for instance, this reduces the amount of hemoglobin S in each red cell to less than 30%, and the tendency to sickle is milder (Kark, 1994; Monchanin et al., 2005). Conversely, if the athlete co-inherits hemoglobin E trait, as in the recent sickling death of a Florida boy in a youth football league (Graham, 2006), the amount of hemoglobin S may exceed 60% and the tendency to sickle is greater. Surely other unknown conditions—some inborn, some not—shape the chance and severity of sickling in individual athletes. We need more research here.

Heat and dehydration increase sickling, mainly because they make the drill more difficult and drive the blood oxygen lower. Exercise-induced asthma and the thin air of altitude also increase sickling because of lower blood oxygen.

## Differential Diagnosis

Sickling collapse has been confused with heat cramping, heat exhaustion, and heatstroke. The most telling symptom of sickling collapse is increasing pain and weakness in the working muscles, especially the legs, buttocks, and/or low back, usually during sprinting. The athlete may call the pain "cramping," but it is unique. Sickling pain is unlike that of heat cramping or "burning" muscles from middle-distance racing (Eichner, 2000). Sickling pain is from insufficient blood flow to working muscles – ischemic pain. Quickly, the blood-starved muscles fail to support the athlete. Knowing which athletes have sickle trait avoids possible confusion over "cramping" – any cramping should be considered sickling until proved otherwise.

Unlike in heatstroke, sickling athletes may be on-field only briefly, sprinting as little as 800-1,200 meters, early in training,

often on the first or second day of preseason workouts. Sickling can occur during repeated running up hills, ramps, or stairs, or even during extreme weight-lifting. Sickling collapse can occur if the tempo increases late in one-hour football drills, including "mat drills" in the winter. At the end of a long, hot football practice, if the players run sprints or "gassers" with insufficient breathers, sickling can begin in 800 meters or less. Sickling can even occur rarely during the play of the game, for example, when a running back is in almost constant action during a long, frantic drive downfield. Unlike in many cardiac collapses, especially those from ventricular fibrillation, sickling athletes can still talk when they hit the ground, even though they are gravely ill.

In general, players who have had both heat cramping and sickling can tell the difference between the two syndromes.

Five differences:

- 1) Heat cramping often has an early warning sign. Hours or minutes before the athlete suffers heat cramping, he may see or feel twitching or twinges in tired muscles, those destined to cramp. The athlete who knows heat cramps will tell you, "They are about to come on." In contrast, sickling usually hits suddenly, with no early warning.
- 2) The pain is different. Heat cramping pain is an excruciating pain of sustained, full contraction of muscles, a "lockup." Sickling pain is milder, an ischemic pain from working muscles robbed of blood supply, like the pain of intermittent claudication when leg arteries are narrowed by atherosclerosis.
- 3) What stops the athlete is different. With heat cramping, athletes "hobble to a halt" – the fully contracted muscles no longer work. With sickling, athletes "slump to a stop" - the legs become "weak and wobbly" and no longer hold them up.
- 4) The physical findings are different. In heat cramping, one can see and feel large, rock-hard muscles in full contraction – and the athlete often is yelling in pain. It can take several people to stretch out the legs of a huge football player with major heat cramps in the thighs or hamstrings. With sickling, the exhausted player lies fairly still and complains little - except to say that his legs hurt and won't hold him up - and the muscles look and feel normal.
- 5) The response is different. After 10 to 15 minutes sitting in a cold tub, drinking fluids and getting supplemental oxygen by face mask, the athlete with mild sickling "feels fine." This is likely because many sickle cells have reverted to normal as they regained oxygen. In contrast, major heat cramping often takes an hour or two to resolve, even in a player resting in the training room, being treated with stretching, massage, and intravenous fluids.

In short, although athletes and coaches tend to lump heat cramping and sickling as "cramping," the two syndromes are starkly different.

## PRACTICAL MANAGEMENT POINTS FOR COACHES AND SPORTS HEALTH PROFESSIONALS

Screen all athletes -- It is vital to know which athletes have sickle trait. Although most states test at birth for hemoglobinopathies, most college athletes do not know their sickle-trait status. All it takes is inexpensive blood testing. Informed athletes, coaches, and athletic trainers can prevent sickling deaths.

Acclimation -- Build up slowly in conditioning or lifting regimens. The harder and faster sickle-trait athletes work, the more likely they are to sickle. Slow the tempo of their training. Have supplemental oxygen ready for games at altitude.

Encourage regular sleep. Control any asthma. Do not allow ill athletes to work out.

Modify drills -- No timed sprints or miles. No repeat sprints beyond 500 m total without a "breather." No all-out exertion of any type sustained 2-3 minutes without a breather. If repeat sprints ("gassers" or "trippers") are done at the end of practice, double or triple the rest time between sprints for sickle-trait athletes. If they can set their own pace, they usually do well. They will tell you, "If I can just catch my breath, I'll do fine." During rest, sickle cells tend to revert to normal shape as they regain oxygen traversing the lungs.

Hydrate -- Dehydration fosters sickling (Bergeron et al., 2004). Make sure sickle-trait athletes stay hydrated. Modify work/rest cycles for the heat. After they have had a night's sleep (without fluids) test to see if their urine is concentrated. Because of sickling in the medulla of the kidney, some sickle trait athletes -- especially older athletes - lose the ability to concentrate urine so they excrete too much water (Kark, 1994). This can increase the risk of dehydration.

Set the tone -- The sickle-trait athlete in particular should feel comfortable reporting unusual symptoms immediately. The coach should consider any struggling, cramping, or collapse as likely sickling and seek help fast.

Act fast -- A sickling collapse is a medical emergency. Check vital signs. Give oxygen by face mask. Cool the athlete, if necessary. If there is no improvement very quickly or if vital signs or alertness decline, call 911, attach an automatic emergency defibrillator, start an intravenous drip of normal saline, and get the athlete to the hospital quickly.

## SUMMARY

Sickle cell trait can pose a grave risk for some athletes. In the past six years alone, exertional sickling has killed eight athletes, including four college football players. Exercise physiology research, both military and civilian, shows how and why sickle red cells accumulate in the bloodstream of sickle-trait athletes in a variety of exercise bouts. Sickle cells can "logjam" blood vessels and cause collapse from ischemic rhabdomyolysis. Death can ensue from complications of explosive rhabdomyolysis: cardiac arrhythmias and/or acute myoglobinuric kidney failure. Sickling can begin in 2-3 minutes of any all-out exertion. Heat, dehydration, altitude, and asthma can worsen exertional sickling. This sickling syndrome is unique and can easily be distinguished from heatstroke or heat cramping. Sickling collapse is a medical emergency. Screening and precautions for sickle trait can prevent sickling collapse and enable sickle-trait athletes to thrive in their sports.

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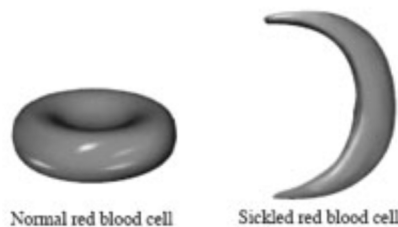
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## S U P P L E M E N T

### DANGER LURKS FOR ATHLETES WITH SICKLE CELL TRAIT

In sickle trait, each red blood cell has about 60% normal hemoglobin and 40% sickle hemoglobin. In everyday life, sickle trait causes few or no problems. However, problems arise if oxygen in the bloodstream drops too low – as it can during sustained sprinting. During such activity, some red cells change from a normal disk shape to a quarter-moon shape – the "sickle" shape. This shape change is termed "sickling."



### Exertional Sickling: Clinical Consequences

If many red cells sickle, they clump in a "log-jam" that clogs the blood vessel and cuts off blood flow to the muscles. This results in rhabdomyolysis – the breakdown of muscles. Acute, severe rhabdomyolysis can release enough lactic acid and potassium to sap the pumping power of the normal heart – even enough to cause fatal arrhythmia. In addition, it can release enough myoglobin, the oxygen-binding protein in muscle, to plug the kidneys and cause acute renal failure. So exertional sickling collapse is a medical emergency.

## SICKLING SETTINGS IN SPORT: EXAMPLES

### Football training

- Wind sprints early in practice
- Repeated runs up hills, ramps, or stairs
- "Gassers" at the end of practice
- Extreme weight-lifting bouts
- Intense drills and other spurts of exercise after 40-50 minutes of conditioning
- Rarely in the play of the game
- Normal red blood cell Sickled red blood cell

### Basketball Training

- Running laps on a track
- Running timed distances
- "Suicide runs" on-court

### Track athletes

- Running repeat hills
- Cross-country racing

## WHAT TO DO ABOUT EXERTIONAL SICKLING

- Screen athletes during pre-conditioning medical exams -- It is vital to know which athletes have sickle trait.
- Acclimation -- Build up slowly in training. Slow tempo. Longer breathers. Control asthma. Supplemental oxygen for altitude. No workout if ill.
- Modify drills -- No timed sprints or miles. No sprints > 500 m. No all-out exertion of any type for 2-3 minutes without a breather. Adjust work/rest cycles for heat.
- Hydrate -- Ensure good hydration. Check overnight urine to ensure that kidneys can concentrate urine.
- Set the tone -- Athletes should report any unusual symptoms immediately. Coach should consider any struggling, cramping, or collapse as sickling.
- Act fast -- A sickling collapse is a medical emergency. Check vital signs. Give oxygen by face mask. Cool the athlete, if necessary. If there is no improvement very quickly or if vital signs or alertness decline, call 911, attach an automatic emergency defibrillator, start an IV, and get the athlete to the hospital quickly.

## SUGGESTED ADDITIONAL RESOURCES

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# National Athletic Trainers' Association Position Statement: Management of Sport- Related Concussion

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

Research in the area of sport-related concussion has provided the athletic training and medical professions with valuable new knowledge in recent years. Certified athletic trainers, who on average care for 7 concussive injuries per year,<sup>2</sup> have been forced to rethink how they manage sport-related concussion. Recurrent concussions to several high-profile athletes, some of whom were forced into retirement as a result, have increased awareness among sports medicine personnel and the general public. Bridging the gap between research and clinical practice

is the key to reducing the incidence and severity of sport-related concussion and improving return-to-play decisions. This position statement should provide valuable information and recommendations for certified athletic trainers (ATCs), physicians, and other medical professionals caring for athletes at the youth, high school, collegiate, and elite levels. The following recommendations are derived from the most recent scientific and clinic-based literature on sport-related concussion. The justification for these recommendations is presented in the summary statement following the recommendations. The summary statement is organized into the following sections: "Defining and Recognizing Concussion," "Evaluating and Making the Return-to-Play Decision," "Concussion Assessment Tools," "When to Refer an Athlete to a Physician After Concussion," "When to Disqualify an Athlete," "Special Considerations for the Young Athlete," "Home Care," and "Equipment Issues."

## RECOMMENDATIONS

### Defining and Recognizing Concussion

1. The ATC should develop a high sensitivity for the various mechanisms and presentations of traumatic brain injury (TBI), including mild, moderate, and severe cerebral concussion, as well as the more severe, but less common, head injuries that can cause damage to the brain stem and other vital centers of the brain.
2. The colloquial term "ding" should not be used to describe a sport-related concussion. This stunned confusional state is a concussion most often reflected by the athlete's initial confusion, which may disappear within minutes, leaving

no outwardly observable signs and symptoms. Use of the term “ding” generally carries a connotation that diminishes the seriousness of the injury. If an athlete shows concussion-like signs and reports symptoms after a contact to the head, the athlete has, *at the very least*, sustained a mild concussion and should be treated for a concussion.

3. To detect deteriorating signs and symptoms that may indicate a more serious head injury, the ATC should be able to recognize both the obvious signs (eg, fluctuating levels of consciousness, balance problems, and memory and concentration difficulties) and the more common, self-reported symptoms (eg, headache, ringing in the ears, and nausea).
4. The ATC should play an active role in educating athletes, coaches, and parents about the signs and symptoms associated with concussion, as well as the potential risks of playing while still symptomatic.
5. The ATC should document all pertinent information surrounding the concussive injury, including but not limited to (1) mechanism of injury; (2) initial signs and symptoms; (3) state of consciousness; (4) findings on serial testing of symptoms and neuropsychological function and postural-stability tests (noting any deficits compared with baseline); (5) instructions given to the athlete and/or parent; (6) recommendations provided by the physician; (7) date and time of the athlete’s return to participation; and (8) relevant information on the player’s history of prior concussion and associated recovery pattern(s).<sup>3</sup>

### Evaluating and Making the Return-to-Play Decision

6. Working together, ATCs and team physicians should agree on a philosophy for managing sport-related concussion before the start of the athletic season. Currently 3 approaches are commonly used: (1) grading the concussion at the time of the injury, (2) deferring final grading until all symptoms have resolved, or (3) not using a grading scale but rather focusing attention on the athlete’s recovery via symptoms, neurocognitive testing, and postural-stability testing. After deciding on an approach, the ATC-physician team should be consistent in its use regardless of the athlete, sport, or circumstances surrounding the injury.
7. For athletes playing sports with a high risk of concussion, baseline cognitive and postural-stability testing should be considered. In addition to the concussion injury assessment, the evaluation should also include an assessment of the cervical spine and cranial nerves to identify any cervical spine or vascular intracerebral injuries.
8. The ATC should record the time of the initial injury and document serial assessments of the injured athlete, noting the presence or absence of signs and symptoms of injury. The ATC should monitor vital signs and level of consciousness every 5 minutes after a concussion until the athlete’s condition improves. The athlete should also be monitored over the next few days after the injury for the presence of delayed signs and symptoms and to assess recovery.
9. Concussion severity should be determined by paying close attention to the severity and persistence of *all* signs and symptoms, including the presence of amnesia (retrograde and anterograde) and loss of consciousness (LOC), as well as headache, concentration problems, dizziness, blurred

vision, and so on. It is recommended that ATCs and physicians consistently use a symptom checklist similar to the one provided in Appendix A.

10. In addition to a thorough clinical evaluation, formal cognitive and postural-stability testing is recommended to assist in objectively determining injury severity and readiness to return to play (RTP). No one test should be used solely to determine recovery or RTP, as concussion presents in many different ways.
11. Once symptom free, the athlete should be reassessed to establish that cognition and postural stability have returned to normal for that player, preferably by comparison with preinjury baseline test results. The RTP decision should be made after an incremental increase in activity with an initial cardiovascular challenge, followed by sport-specific activities that do not place the athlete at risk for concussion. The athlete can be released to full participation as long as no recurrent signs or symptoms are present.

### Concussion Assessment Tools

12. Baseline testing on concussion assessment measures is recommended to establish the individual athlete’s “normal” preinjury performance and to provide the most reliable benchmark against which to measure postinjury recovery. Baseline testing also controls for extraneous variables (eg, attention deficit disorder, learning disabilities, age, and education) and for the effects of earlier concussion while also evaluating the possible cumulative effects of recurrent concussions.
13. The use of objective concussion assessment tools will help ATCs more accurately identify deficits caused by injury and postinjury recovery and protect players from the potential risks associated with prematurely returning to competition and sustaining a repeat concussion. The concussion assessment battery should include a combination of tests for cognition, postural stability, and self-reported symptoms known to be affected by concussion.
14. A combination of brief screening tools appropriate for use on the sideline (eg, Standardized Assessment of Concussion [SAC], Balance Error Scoring System [BESS], symptom checklist) and more extensive measures (eg, neuropsychological testing, computerized balance testing) to more precisely evaluate recovery later after injury is recommended.
15. Before instituting a concussion neuropsychological testing battery, the ATC should understand the test’s user requirements, copyright restrictions, and standardized instructions for administration and scoring. All evaluators should be appropriately trained in the standardized instructions for test administration and scoring before embarking on testing or adopting an instrument for clinical use. Ideally, the sports medicine team should include a neuropsychologist, but in reality, many ATCs may not have access to a neuropsychologist for interpretation and consultation, nor the financial resources to support a neuropsychological testing program. In this case, it is recommended that the ATC use screening instruments (eg, SAC, BESS, symptom checklist) that have been developed specifically for use by sports medicine clinicians without extensive



training in psychometric or standardized testing and that do not require a special license to administer or interpret.

16. Athletic trainers should adopt for clinical use only those neuropsychological and postural stability measures with population-specific normative data, test-retest reliability, clinical validity, and sufficient sensitivity and specificity established in the peer-reviewed literature. These standards provide the basis for how well the test can distinguish between those with and without cerebral dysfunction in order to reduce the possibility of false-positive and false-negative errors, which could lead to clinical decision-making errors.
17. As is the case with all clinical instruments, results from assessment measures to evaluate concussion should be integrated with all aspects of the injury evaluation (eg, physical examination, neurologic evaluation, neuroimaging, and player's history) for the most effective approach to injury management and RTP decision making. Decisions about an athlete's RTP should never be based solely on the use of any one test.

### **When to Refer an Athlete to a Physician After Concussion**

18. The ATC or team physician should monitor an athlete with a concussion at 5-minute intervals from the time of the injury until the athlete's condition completely clears or the athlete is referred for further care. Coaches should be informed that in situations when a concussion is suspected but an ATC or physician is not available, their primary role is to ensure that the athlete is immediately seen by an ATC or physician.
19. An athlete with a concussion should be referred to a physician on the day of injury if he or she lost consciousness, experienced amnesia lasting longer than 15 minutes, or meets any of the criteria outlined in Appendix B.
20. A team approach to the assessment of concussion should be taken and include a variety of medical specialists. In addition to family practice or general medicine physician referrals, the ATC should secure other specialist referral sources within the community. For example, neurologists are trained to assist in the management of patients experiencing persistent signs and symptoms, including sleep disturbances. Similarly, a neuropsychologist should be identified as part of the sports medicine team for assisting athletes who require more extensive neuropsychological testing and for interpreting the results of neuropsychological tests.
21. A team approach should be used in making RTP decisions after concussion. This approach should involve input from the ATC, physician, athlete, and any referral sources. The assessment of all information, including the physical examination, imaging studies, objective tests, and exertional tests, should be considered prior to making an RTP decision.

### **When to Disqualify an Athlete**

22. Athletes who are symptomatic at rest and after exertion for at least 20 minutes should be disqualified from returning to participation on the day of the injury. Exertional

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

23. Athletes who experience LOC or amnesia should be disqualified from participating on the day of the injury.
24. The decision to disqualify from further participation on the day of a concussion should be based on a comprehensive physical examination; assessment of self-reported postconcussion signs and symptoms; functional impairments, and the athlete's past history of concussions. If assessment tools such as the SAC, BESS, neuropsychological test battery, and symptom checklist are not used, a 7-day symptom-free waiting period before returning to participation is recommended. Some circumstances, however, will warrant even more conservative treatment (see recommendation 25).
25. Athletic trainers should be more conservative with athletes who have a history of concussion. Athletes with a history of concussion are at increased risk for sustaining subsequent injuries as well as for slowed recovery of self-reported postconcussion signs and symptoms, cognitive dysfunction, and postural instability after subsequent injuries. In athletes with a history of 3 or more concussions and experiencing slowed recovery, temporary or permanent disqualification from contact sports may be indicated.

### **Special Considerations for the Young Athlete**

26. Athletic trainers working with younger (pediatric) athletes should be aware that recovery may take longer than in older athletes. Additionally, these younger athletes are maturing at a relatively fast rate and will likely require more frequent updates of baseline measures compared with older athletes.
27. Many young athletes experience sport-related concussion. Athletic trainers should play an active role in helping to educate young athletes, their parents, and coaches about the dangers of repeated concussions. Continued research into the epidemiology of sport-related concussion in young athletes and prospective investigations to determine the acute and long-term effects of recurrent concussions in younger athletes are warranted.
28. Because damage to the maturing brain of a young athlete can be catastrophic (ie, almost all reported cases of second-impact syndrome are in young athletes), athletes under age 18 years should be managed more conservatively, using stricter RTP guidelines than those used to manage concussion in the more mature athlete.

### **Home Care**

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



only at the recommendation of a physician. Additionally, the athlete should be instructed to avoid ingesting alcohol, illicit drugs, or other substances that might interfere with cognitive function and neurologic recovery.

30. Any athlete with a concussion should be instructed to rest, but complete bed rest is not recommended. The athlete should resume normal activities of daily living as tolerated while avoiding activities that potentially increase symptoms. Once he or she is symptom free, the athlete may resume a graded program of physical and mental exertion, without contact or risk of concussion, up to the point at which postconcussion signs and symptoms recur. If symptoms appear, the exertion level should be scaled back to allow maximal activity without triggering symptoms.
31. An athlete with a concussion should be instructed to eat a well-balanced diet that is nutritious in both quality and quantity.
32. An athlete should be awakened during the night to check on deteriorating signs and symptoms only if he or she experienced LOC, had prolonged periods of amnesia, or was still experiencing significant symptoms at bedtime. The purpose of the wake-ups is to check for deteriorating signs and symptoms, such as decreased levels of consciousness or increasing headache, which could indicate a more serious head injury or a late-onset complication, such as an intracranial bleed.
33. Oral and written instructions for home care should be given to the athlete and to a responsible adult (eg, parent or roommate) who will observe and supervise the athlete during the acute phase of the concussion while at home or in the dormitory. The ATC and physician should agree on a standard concussion home-instruction form similar to the one presented in Appendix C, and it should be used consistently for all concussions.

## Equipment Issues

34. The ATC should enforce the standard use of helmets for protecting against catastrophic head injuries and reducing the severity of cerebral concussions. In sports that require helmet protection (football, lacrosse, ice hockey, baseball/softball, etc), the ATC should ensure that all equipment meets either the National Operating Committee on Standards for Athletic Equipment (NOCSAE) or American Society for Testing and Materials (ASTM) standards.
35. The ATC should enforce the standard use of mouth guards for protection against dental injuries; however, there is no scientific evidence supporting their use for reducing concussive injury.
36. At this time, the ATC should neither endorse nor discourage the use of soccer headgear for protecting against concussion or the consequences of cumulative, subconcussive impacts to the head. Currently no scientific evidence supports the use of headgear in soccer for reducing concussive injury to the head.

## DEFINING AND RECOGNIZING CONCUSSION

Perhaps the most challenging aspect of managing sport-related concussion is recognizing the injury, especially in athletes with no obvious signs that a concussion has actually occurred. The immediate management of the head-injured athlete

depends on the nature and severity of the injury. Several terms are used to describe this injury, the most global being TBI, which can be classified into 2 types: focal and diffuse. Focal or posttraumatic intracranial mass lesions include subdural hematomas, epidural hematomas, cerebral contusions, and intracerebral hemorrhages and hematomas. These are considered uncommon in sport but are serious injuries; the ATC must be able to detect signs of clinical deterioration or worsening symptoms during serial assessments. Signs and symptoms of these focal vascular emergencies can include LOC, cranial nerve deficits, mental status deterioration, and worsening symptoms. Concern for a significant focal injury should also be raised if these signs or symptoms occur after an initial lucid period in which the athlete seemed normal.

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

Cerebral concussion, which is the focus of this position statement, can best be classified as a mild diffuse injury and is often referred to as mild TBI (MTBI). The injury involves an acceleration-deceleration mechanism in which a blow to the head or the head striking an object results in 1 or more of the following conditions: headache, nausea, vomiting, dizziness, balance problems, feeling "slowed down," fatigue, trouble sleeping, drowsiness, sensitivity to light or noise, LOC, blurred vision, difficulty remembering, or difficulty concentrating.<sup>6</sup> In 1966, the Congress of Neurological Surgeons proposed the following consensus definition of concussion, subsequently endorsed by a variety of medical associations: "Concussion is a clinical syndrome characterized by immediate and transient impairment of neural functions, such as alteration of consciousness, disturbance of vision, equilibrium, etc, due to mechanical forces."<sup>7</sup> Although the definition received widespread consensus in 1966, more contemporary opinion (as concluded at the First International Conference on Concussion in Sport, Vienna, 2001<sup>8</sup>) was that this definition fails to include many of the predominant clinical features of concussion, such as headache and nausea. It is often reported that there is no universal agreement on the standard definition or nature of concussion; however, agreement does exist on several features that incorporate clinical, pathologic, and biomechanical injury constructs associated with head injury:

1. Concussion may be caused by a direct blow to the head or elsewhere on the body from an “impulsive” force transmitted to the head.
2. Concussion may cause an immediate and short-lived impairment of neurologic function.
3. Concussion may cause neuropathologic changes; however, the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury.
4. Concussion may cause a gradient of clinical syndromes that may or may not involve LOC. Resolution of the clinical and cognitive symptoms typically follows a sequential course.
5. Concussion is most often associated with normal results on conventional neuroimaging studies.<sup>8</sup>

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

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

### Mechanisms of Injury

A forceful blow to the resting, movable head usually produces maximum brain injury beneath the point of cranial impact (coup injury). A moving head hitting an unyielding object usually produces maximum brain injury opposite the site of cranial impact (contrecoup injury) as the brain shifts within the cranium. When the head is accelerated before impact, the brain lags toward the trailing surface, thus squeezing away the CSF and creating maximal shearing forces at this site. This brain lag actually thickens the layer of CSF under the point of impact, which explains the lack of coup injury in the moving head. Alternatively, when the head is stationary before impact, neither brain lag nor disproportionate distribution of CSF occurs, accounting for the absence of contrecoup injury and the presence of coup injury.<sup>4,5</sup>

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

Three types of stresses can be generated by an applied force to injure the brain: compressive, tensile, and shearing. Compression involves a crushing force in which the tissue cannot absorb any additional force or load. Tension involves pulling

or stretching of tissue, whereas shearing involves a force that moves across the parallel organization of the tissue. Brief, uniform compressive stresses are fairly well tolerated by neural tissue, but tension and shearing stresses are very poorly tolerated.<sup>4,9</sup>

### Neuroimaging of Cerebral Concussion

Traditionally, computed tomography (CT) and magnetic resonance imaging (MRI) have been considered useful in identifying certain types of brain lesions; however, they have been of little value in assessing less severe head injuries, such as cerebral concussion, and contributing to the RTP decision. A CT scan is often indicated emergently if a focal injury such as an acute subdural or epidural bleed is suspected; this study easily demonstrates acute blood collection and skull fracture, but an MRI is superior at demonstrating an isodense subacute or chronic subdural hematoma that may be weeks old.<sup>10,11</sup> Newer structural MRI modalities, including gradient echo, perfusion, and diffusion-weighted imaging, are more sensitive for structural abnormalities (eg, vascular shearing) compared with other diagnostic imaging techniques.<sup>10</sup> Functional imaging technologies (eg, positron emission tomography [PET], single-photon emission computerized tomography [SPECT], and functional MRI [fMRI]) are also yielding promising early results and may help define concussion recovery.<sup>12</sup> Presently, no neuroanatomic or physiologic measurements can be used to determine the severity of a concussion or when complete recovery has occurred in an individual athlete after a concussion.

### EVALUATING AND MAKING THE RETURN-TO-PLAY DECISION

#### Clinical Evaluation

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

#### Determining Injury Severity

The definition of concussion is often expanded to include mild, moderate, and severe injuries. Several early grading scales and RTP guidelines early were proposed for classifying



and managing cerebral concussions.<sup>6,13–20</sup> None of the scales have been universally accepted or followed with much consistency by the sports medicine community. In addition, most of these classification systems denote the most severe injuries as associated with LOC, which we now know is not always predictive of recovery after a brain injury.<sup>21,22</sup> It is important for the ATC and other health care providers to recognize the importance of identifying retrograde amnesia and anterograde amnesia, LOC, and other signs and symptoms present and to manage each episode independently.

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

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

With respect to amnesia, the issue is more clouded because findings have been inconsistent. Several studies of nonathletes<sup>30–37</sup> suggest that the duration of posttraumatic amnesia correlates with the severity and outcome of severe TBI but not with mild TBI or concussion.<sup>38–40</sup> More contemporary studies of athletes with concussion are also clouded. Two unrelated, prospective studies of concussion suggest that the presence of amnesia best correlates with abnormal neuropsychological testing at 48 hours and with the duration and number of other postconcussion signs and symptoms.<sup>24,41</sup> However, more recently, investigations of high school and collegiate athletes with concussion revealed no association between (1) amnesia and duration of symptoms or (2) amnesia and neuropsychological and balance tests at 3, 24, 48, 72, and 96 hours postinjury.<sup>21,28,29</sup> Of importance in these studies is the significant association between symptom-severity score (within the initial 3 hours postinjury) and the total duration of symptoms (mea-

**Table 1. American Academy of Neurology Concussion Grading Scale<sup>6</sup>**

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

\*LOC indicates loss of consciousness.

sured until asymptomatic). Although these findings suggest that initial symptom severity is probably a better indicator than either LOC or amnesia in predicting length of recovery, amnesia was recently found to predict symptom and neurocognitive deficits at 2 days postinjury.<sup>42</sup> More research is needed in this area to help improve clinical decision making.

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

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

**Table 2. Cantu Evidence-Based Grading System for Concussion<sup>43</sup>**

Grade 1 (mild)	No LOC*, PTA† <30 min, PCSS‡ <24 h
Grade 2 (moderate)	LOC <1 min <b>or</b> PTA ≥30 min <24 h <b>or</b> PCSS ≥24 h <7 d
Grade 3 (severe)	LOC ≥1 min <b>or</b> PTA ≥24 h <b>or</b> PCSS ≥7 d

\*LOC indicates loss of consciousness.

†PTA indicates posttraumatic amnesia (anterograde/retrograde).

‡PCSS indicates postconcussion signs and symptoms other than amnesia.

tion.<sup>3,43</sup> Finally, a third approach to the grading-scale dilemma is to not use a grading scale but rather focus attention on the athlete's recovery via symptoms, neuropsychological tests, and postural-stability tests. This line of thinking is that the ATC should not place too much emphasis on the grading system or grade but should instead focus on whether the athlete is symptomatic or symptom free. Once the athlete is asymptomatic, a stepwise progression should be implemented that increases demands over several days. This progression will be different for athletes who are withheld for several weeks compared with those athletes withheld for just a few days. This multitiered approach was summarized and supported by consensus at the 2001 Vienna Conference on Concussion in Sport.<sup>8</sup>

### Making the Return-to-Play Decision

The question raised most often regarding the concussion grading and RTP systems is one of practicality in the sport setting. Many clinicians believe that the RTP guidelines are too conservative and, therefore, choose to base decisions on clinical judgment of individual cases rather than on a general recommendation. It has been reported that 30% of all high school and collegiate football players sustaining concussions return to competition on the same day of injury; the remaining 70% average 4 days of rest before returning to participation.<sup>23</sup> Many RTP guidelines call for the athlete to be symptom free for at least 7 days before returning to participation after a grade 1 or 2 concussion.<sup>6,13,15,17,43,44</sup> Although many clinicians deviate from these recommendations and are more liberal in making RTP decisions, recent studies by Guskiewicz and McCrea et al<sup>21,29</sup> suggest that perhaps the 7-day waiting period can minimize the risk of recurrent injury. On average, athletes required 7 days to fully recover after concussion. Same-season repeat injuries typically take place within a short window of time, 7 to 10 days after the first concussion,<sup>21</sup> supporting the concept that there may be increased neuronal vulnerability or blood-flow changes during that time, similar to those reported by Giza, Hovda, et al<sup>45-47</sup> in animal models.

Returning an athlete to participation should follow a progression that begins once the athlete is completely symptom free. All signs and symptoms should be evaluated using a graded symptom scale or checklist (described in "Concussion Assessment Tools") when performing follow-up assessments and should be evaluated both at rest and after exertional maneuvers such as biking, jogging, sit-ups, and push-ups. Baseline measurements of neuropsychological and postural stability are strongly recommended for comparing with postinjury measurements. If these exertional tests do not produce symptoms, either acutely or in delayed fashion, the athlete can then participate in sport-specific skills that allow return to practice but should remain out of any activities that put him or her at risk for recurrent head injury. For the basketball player, this may

include shooting baskets or participating in walk-throughs, and for the soccer player, this may include dribbling or shooting drills or other sport-specific activities. These restricted and monitored activities should be continued for the first few days after becoming symptom free. The athlete should be monitored periodically throughout and after these sessions to determine if any symptoms develop or increase in intensity. Before returning to full contact participation, the athlete should be reassessed using neuropsychological and postural-stability tests if available. If all scores have returned to baseline or better, return to full participation can be considered after further clinical evaluation. It is strongly recommended that after recurrent injury, especially within-season repeat injuries, the athlete be withheld for an extended period of time (approximately 7 days) after symptoms have resolved.

### CONCUSSION ASSESSMENT TOOLS

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

Data from objective measures of cognitive functioning, postural stability, and postconcussion signs and symptoms are most helpful in making a determination about severity of injury and postinjury recovery when preinjury baseline data for an individual athlete are available.<sup>3,8,24,29,41</sup> Baseline testing provides an indicator of what is "normal" for that particular athlete while also establishing the most accurate and reliable benchmark against which postinjury results can be compared. It is important to obtain a baseline symptom assessment in addition to baseline cognitive and other ability testing. Without baseline measures, the athlete's postinjury performance on neuropsychological testing and other concussion assessment measures must be interpreted by comparison with available population normative values, which ideally are based on a large sample of the representative population. Normative data for competitive athletes on conventional (ie, paper-and-pencil) and computerized neuropsychological tests and other concussion assessment measures are now more readily available from large-scale research studies, but baseline data on an individual athlete still provide the greatest clinical accuracy in interpreting postinjury test results. When performing baseline testing, a suitable testing environment eliminates all distractions that could alter the baseline performance and enhances the likelihood that all athletes are providing maximal effort. Most important, all evaluators should be aware of a test's user requirements and be appropriately trained in the standardized instructions for test administration and scoring before embarking on baseline testing or adopting a concussion testing paradigm for clinical use.

Several models exist for implementing baseline testing. Ide-



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

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

### Postconcussion Symptom Assessment

Self-reported symptoms are among the more obvious and recognizable ways to assess the effects of concussion. Typical self-reported symptoms after a concussion include but are not limited to headache; dizziness; nausea; vomiting; feeling "in a fog"; feeling "slowed down"; trouble falling asleep; sleeping more than usual; fatigue; drowsiness; sensitivity to light or noise; unsteadiness or loss of balance; feeling "dinged," dazed, or stunned; seeing stars or flashing lights; ringing in the ears; and double vision.<sup>8,26,48</sup> Self-reported symptoms are referenced by many of the concussion grading scales.<sup>10,43,44,49</sup> The presence of self-reported symptoms serves as a major contraindication for RTP, and, based on current recommendations, the athlete should be fully symptom free for at least 7 days at rest and during exertion before returning to play.<sup>43,44</sup>

A number of concussion symptom checklists<sup>43,50–52</sup> and scales<sup>26,41,48,53</sup> have been used in both research and clinical settings. A symptom checklist that provides a list of concussion-related symptoms allows the athlete to report whether the symptom is present by responding either "yes" (experiencing the symptom) or "no" (not experiencing the symptom). A symptom scale is a summative measure that allows the athlete to describe the extent to which he or she is experiencing the symptom. These instruments commonly incorporate a Likert-type scale that allows the player to rate the severity or frequency of postconcussion symptoms. These scores are then summed to form a composite score that yields a quantitative measure of overall injury severity and a benchmark against which to track postinjury symptom recovery. Initial evidence has been provided for the structural validity of a self-report concussion symptom scale.<sup>48</sup> Obtaining a baseline symptom score is helpful to establish any preexisting symptoms attributable to factors other than the head injury (eg, illness, fatigue, or somatization). Serial administration of the symptom checklist is the recommended method of tracking symptom resolution over time (see Appendix A).

### Mental Status Screening

Cognitive screening instruments similar to the physician's mini mental status examination objectify what is often a subjective impression of cognitive abnormalities. Various methods have been suggested for a systematic survey of mental status and cognitive function in the athlete with a concussion. The SAC was developed to provide sports medicine clinicians with a brief, objective tool for assessing the injured athlete's mental status during the acute period after concussion (eg, sport sideline, locker room, and clinic).<sup>54</sup> The SAC includes measures of orientation, immediate memory, concentration, and delayed recall that sum to 30 points.<sup>55</sup> Lower scores on the SAC indicate more severe cognitive impairment. The SAC also includes assessments of strength, sensation, and coordination and a standard neurologic examination but should not replace the clinician's thorough physical examination or referral for more extensive neuropsychological evaluation when indicated. Information about the occurrence and duration of LOC and amnesia is also recorded on the SAC. Alternate forms of the SAC are available to minimize the practice effects during retesting. The SAC takes about 5 minutes to administer and should be used only after the clinician's thorough review of the training manual and instructional video on the administration, scoring, and interpretation of the instrument.

The SAC has demonstrated reliability<sup>29,55,56</sup> and validity<sup>29,56,57</sup> in detecting mental status changes after a concussion. Recent evidence suggests that a decline of 1 point or more from baseline classified injured and uninjured players with a level of 94% sensitivity and 76% specificity.<sup>56</sup> The SAC is also sensitive to detecting more severe neurocognitive changes in injured athletes with LOC or amnesia associated with their concussions.<sup>57</sup> The SAC is most useful in the assessment of acute cognitive dysfunction resulting from concussion, with sensitivity and specificity comparable with extensive neuropsychological testing batteries during the initial 2 to 3 days after concussion.<sup>29,58,59</sup> As with neuropsychological testing, sensitivity and specificity of the SAC in concussion assessment are maximized when individual baseline test data are available.<sup>29,55,56,60</sup>



## Postural-Stability Assessment

A number of postural-stability tests have been used to assess the effects of concussion in the clinical and laboratory settings. The Romberg and stork stand were basic tests used to assess balance and coordination. Riemann et al<sup>61–62</sup> developed the Balance Error Scoring System (BESS) based on existing theories of posturography. The BESS uses 3 stance positions and tests on both a firm and a foam surface with the eyes closed (for a total of 6 trials). The administration and scoring procedures are found in several publications.<sup>61–63</sup> The BESS has established good test-retest reliability and good concurrent validity when compared with laboratory forceplate measures<sup>52,62</sup> and significant group differences, with an increased number of errors for days 1, 3, and 5 postinjury when compared with controls.<sup>52</sup> Thus, the BESS can be used as a clinical measure in identifying balance impairment that could indicate a neurologic deficit.

The NeuroCom Smart Balance Master System (NeuroCom International, Clackamas, OR) is a forceplate system that measures vertical ground reaction forces produced by the body's center of gravity moving around a fixed base of support. The Sensory Organization Test (SOT, NeuroCom International) is designed to disrupt various sensory systems, including the visual, somatosensory, and vestibular systems. The SOT consists of 6 conditions with 3 trials per condition, for a total of 18 trials, with each trial lasting 20 seconds. The complete administration has been described previously.<sup>52,64</sup> The SOT has produced significant findings related to the assessment of concussion recovery. In a sample of 36 athletes with concussion, the mean stability (composite score) and vestibular and visual ratios demonstrated deficits for up to 5 days postinjury.<sup>52</sup> The greatest deficits were seen 24 hours postinjury, and the athletes with concussion demonstrated a gradual recovery during the 5-day period to within 6% of baseline scores. These results were confirmed by Peterson et al,<sup>65</sup> who found that these deficits continued for up to 10 days after concussion. These findings reveal a sensory interaction problem from the effects of concussion with measurable changes in overall postural stability.

## Neuropsychological Testing

Neuropsychological testing has historically been used to evaluate various cognitive domains known to be preferentially susceptible to the effects of concussion and TBI. In recent years, neuropsychological testing to evaluate the effects of sport-related concussion has gained much attention in the sport concussion literature.<sup>20,21,26,29,48,52,58,59,65–69</sup> The work of Barth et al,<sup>70</sup> who studied more than 2000 collegiate football players from 10 universities, was the first project to institute baseline neuropsychological testing. Similar programs are now commonplace among many collegiate and professional teams, and interest is growing at the high school level. Several recent studies have supported the use of neuropsychological testing as a valuable tool to evaluate the cognitive effects and recovery after sport-related concussion,<sup>24,28,29,41,42,50–52,57,65,66,71–75</sup> but its feasibility for sideline use is not likely realistic. As is the case with other concussion assessment tools, baseline neuropsychological testing is recommended, when possible, to establish a normative level of neurocognitive functioning for individual athletes.<sup>24,28,29,41,50–52,57–59,66,69,73–75</sup> Baseline neuropsychological testing typically takes 20 to 30 minutes per athlete.

Before implementing a neuropsychological testing program, the ATC must consider several issues, including test-specific training requirements and methodologic issues, the practicality of baseline testing, the reliability and validity of individual tests comprising the test battery, and the protocol for interpretation of the postinjury test results. Barr<sup>76</sup> provided an excellent review on the methodologic and professional issues associated with neuropsychological testing in sport concussion assessment. Most states require advanced training and licensure to purchase and use neuropsychological tests for clinical purposes. Neuropsychological tests are also copyright protected to prevent inappropriate distribution or use by unqualified professionals. At present, these requirements necessitate that a licensed psychologist, preferably one Board certified in clinical neuropsychology or with clinical experience in the evaluation of sport-related concussion, oversee and supervise the clinical application of neuropsychological testing for sport concussion assessment. These factors likely restrict how widely neuropsychological testing can be used to assess sport-related concussion, especially at the high school level and in rural areas where neuropsychologists are not readily available for consultation.

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

Although no clear indications exist as to which are the best individual neuropsychological tests to evaluate sport concussion, the use of multiple instruments as a "test battery" offers clinicians greater potential for recognizing any cognitive deficits incurred from the injury. A number of neuropsychological tests and test batteries have been used to assess sport-related concussion. Table 3 provides a brief description of the paper-and-pencil neuropsychological tests commonly used by neuropsychologists in the assessment of sport concussion. Sport concussion batteries should include measures of cognitive abilities most susceptible to change after concussion, including attention and concentration, cognitive processing (speed and efficiency), learning and memory, working memory, executive functioning, and verbal fluency. Tests of attention and concentration<sup>50,52,74,77</sup> and memory functioning<sup>41</sup> have been reported as the most sensitive to the acute effects of concussion. The athlete's age, sex, primary language, and level of education should be considered when selecting a test battery.<sup>68</sup>

**Computerized Neuropsychological Tests.** Recently, a number of computerized neuropsychological testing programs have been designed for the assessment of athletes after concussion. The Automated Neuropsychological Assessment Metrics (ANAM), CogSport, Concussion Resolution Index, and Immediate Postconcussion Assessment and Cognitive Testing (ImPACT) are all currently available and have shown promise for reliable and valid concussion assessment (Table 4).<sup>24,41,51,53,66,71,72,75,78–84</sup> The primary advantages to computerized testing are the ease of administration, ability to baseline test a large number of athletes in a short period of time, and multiple forms used within the testing paradigm to reduce the

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

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

practice effects. Collie et al<sup>71</sup> summarized the advantage and disadvantages of computerized versus traditional paper-and-pencil testing.

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

to provide clinicians with the most effective neuropsychological assessment tools and maintain the testing standards of neuropsychology.

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

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

**Table 4. Computerized Neuropsychological Tests**

Neuropsychological Test	Developer (Contact Information)	Cognitive Domains
Automated Neuropsychological Assessment Metrics (ANAM)	National Rehabilitation Hospital Assistive Technology and Neuroscience Center, Washington, DC <sup>84</sup> (jsb2@mhg.edu)	Simple Reaction Metrics Sternberg Memory Math Processing Continuous Performance Matching to Sample Spatial Processing Code Substitution
CogSport	CogState Ltd, Victoria, Australia (www.cogsport.com)	Simple Reaction Time Complex Reaction Time One-Back Continuous Learning
Concussion Resolution Index	HeadMinder Inc, New York, NY (www.headminder.com)	Reaction Time Cued Reaction Time Visual Recognition 1 Visual Recognition 2 Animal Decoding Symbol Scanning
Immediate Postconcussion Assessment and Cognitive Testing (ImPACT)	University of Pittsburgh Medical Center, Pittsburgh, PA (www.impacttest.com)	Verbal Memory Visual Memory Information Processing Speed Reaction Time Impulse Control



**Table 5. Factors Influencing Neuropsychological Test Performance<sup>68\*</sup>**

Previous concussions
Educational background
Preinjury level of cognitive functioning
Cultural background
Age
Test anxiety
Distractions
Sleep deprivation
Medications, alcohol, or drugs
Psychiatric disorders
Learning disability
Attention deficit/hyperactivity
Certain medical conditions
Primary language other than English
Previous neuropsychological testing

\*Reprinted with permission of Grindel SH, Lovell MR, Collins MW. The assessment of sport-related concussion: the evidence behind neuropsychological testing and management. *Clin J Sport Med.* 2001; 11: 134–143.

mining postinjury recovery, above and beyond the simple conclusion that the player is “back to baseline.” The complexity of this analysis is the basis for the neuropsychologist overseeing the clinical interpretation of test data to determine injury severity and recovery. Further research is required to clarify the guidelines for determining and tracking recovery on specific measures after concussion. The clinician should also be aware that any concussion assessment tool, either brief screening instruments or more extensive neuropsychological testing, comes with some degree of risk for false negatives (eg, a player performs within what would be considered the normal range on the measure before actually reaching a complete clinical recovery after concussion). Therefore, test results should always be interpreted in the context of all clinical information, including the player’s medical history. Also, caution should be exercised in neuropsychological test interpretation when preinjury baseline data do not exist. Numerous factors apart from the direct effects of concussion can influence test performance (Table 5).

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

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

minutes after a concussion until the athlete’s condition stabilizes and improves. The athlete should also be monitored over the next few hours and days after the injury for delayed signs and symptoms and to assess recovery. Appendix B outlines scenarios that warrant physician referral or, in many cases, transport to the nearest hospital emergency department.

## WHEN TO DISQUALIFY AN ATHLETE

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

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

## Disqualifying for the Game or Practice

The decision to disqualify an individual from further participation on the day of the concussive episode is based on the sideline evaluation, the symptoms the athlete is experiencing, the severity of the apparent symptoms, and the patient’s past history.<sup>86</sup> The literature is clear: any episode involving LOC or persistent symptoms related to concussion (headache, dizziness, amnesia, and so on), regardless of how mild and transient, warrants disqualification for the remainder of that day’s activities.<sup>8,9,13,19,43,60,87</sup> More recent studies of high school and collegiate athletes underscore the importance of ensuring that the athlete is symptom free before returning to participation on the same day; even when the player is symptom free within 15 to 20 minutes after the concussive episode, he or she may still demonstrate delayed symptoms or depressed neurocognitive levels. Lovell et al<sup>88</sup> found significant memory deficits 36 hours postinjury in athletes who were symptom free within 15 minutes of a mild concussion. Guskiewicz et al<sup>21</sup> found that 33% (10/30) of the players with concussion who returned on the same day of injury experienced delayed onset of symptoms at 3 hours postinjury, as compared with only 12.6% (20/158) of those who did not return to play on the same day of injury. Although more prospective work is needed in this area, these studies raise questions as to whether the RTP criteria for grade 1 (mild) concussions are conservative enough.

## Disqualifying for the Season

Guidelines from Cantu<sup>43</sup> and the American Academy of Neurology<sup>6</sup> both recommend termination of the season after the third concussion within the same season. The decision is more difficult if one of the injuries was more severe or was a severe injury resulting from a minimal blow, suggesting that the athlete's brain may be at particular risk for recurrent injury. In addition, because many athletes participate in year-round activities, once they are disqualified for the "season," it may be difficult to determine at what point they can resume contact play. Other issues without clear-cut answers in the literature are when to disqualify an athlete who has not been rendered unconscious and whose symptoms cleared rapidly or one who suffered multiple mild to moderate concussions throughout the career and whether youth athletes should be treated differently for initial and recurrent concussive injuries.

## Disqualifying for the Career

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

Once an athlete has suffered a concussion, he or she is at increased risk for subsequent head injuries.<sup>21,43,86</sup> Guskiewicz et al<sup>21,23</sup> found that collegiate athletes had a 3-fold greater risk of suffering a concussion if they had sustained 3 or more previous concussions in a 7-year period and that players with 2 or more previous concussions required a longer time for total symptom resolution after subsequent injuries.<sup>21</sup> Players also had a 3-fold greater risk for subsequent concussions in the same season,<sup>23</sup> whereas recurrent, in-season injuries occurred within 10 days of the initial injury 92% of the time.<sup>21</sup> In a similar study of high school athletes, Collins et al<sup>82</sup> found that athletes with 3 or more prior concussions were at an increased risk of experiencing LOC (8-fold greater risk), anterograde amnesia (5.5-fold greater risk), and confusion (5.1-fold greater risk) after subsequent concussion. Despite the increasing body of literature on this topic, debate still surrounds the question of how many concussions are enough to recommend ending the player's career. Some research suggests that the magic number may be 3 concussions in a career.<sup>21,23,82</sup> Although these findings are important, they should be carefully interpreted because concussions present in varying degrees of severity, and all athletes do not respond in the same way to concussive insults. Most important is that these data provide evidence for exercising caution when managing younger athletes with concussion and athletes with a history of previous concussions.

## SPECIAL CONSIDERATIONS FOR THE YOUNG ATHLETE

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

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

Recent epidemiologic investigations of head-injury rates in high school athletes have shown that 13.3% of all reported injuries in high school football affect the head and neck, whereas the numbers in other sports range from 1.9% to 9.5% in baseball and wrestling, respectively.<sup>89</sup> Guskiewicz et al<sup>23</sup> prospectively examined concussion incidence in high school and collegiate football players and found that the greatest incidence was at the high school level (5.6%), compared with the National Collegiate Athletic Association Division I (4.4%), Division II (4.5%), and Division III (5.5%).

Authors who have tracked symptoms and neuropsychological function after concussion suggest that age-related differences exist between high school and collegiate athletes with regard to recovery. Lovell et al<sup>41</sup> reported that the duration of on-field mental status changes in high school athletes, such as retrograde amnesia and posttraumatic confusion, was related to the presence of memory impairment at 36 hours, 4 days, and 7 days postinjury as well as slower resolution of self-reported symptoms. These findings further emphasize the need to collect these on-field measures after concussion and to use the information wisely in making RTP decisions, especially when dealing with younger athletes. Field et al<sup>90</sup> found that high school athletes who sustained a concussion demonstrated prolonged memory dysfunction compared with collegiate athletes who sustained a concussion. The high school athletes performed significantly worse on select tests of memory than age-matched control subjects at 7 days postinjury when compared with collegiate athletes and their age-matched control subjects. We hope these important studies and others will eventually lead to more specific guidelines for managing concussions in high school athletes.

Very few investigators have studied sport-related injuries in the youth population, and even fewer focused specifically on sport-related concussion. One group<sup>91</sup> reported that 15% of the children (mean =  $8.34 \pm 5.31$  years) who were admitted to hospitals after MTBI suffered from a sport-related mechanism of injury. Another group<sup>92</sup> found that sport-related head injury accounted for 3% of all sport-related injuries and 24% of all serious head injuries treated in an emergency department. Additionally, sport-related concussion represented a substantial percentage of all head injuries in children under the age of 10 years (18.2%) and 10- to 14-year-old (53.4%) and 15- to 19-year-old (42.9%) populations.<sup>92</sup> Thus, sport-related head injury has a relatively high incidence rate and is a significant public health concern in youth athletes, not just participants at higher competitive levels.

Although no prospective investigations in younger athletes (younger than 15 years old) have been undertaken regarding symptom resolution and cognitive or postural-stability recovery, Valovich McLeod et al<sup>93</sup> recently determined the reliability and validity of brief concussion assessment tools in a group of healthy young athletes (9–14 years old). The SAC is valid within 48 hours of injury and reliable for testing of youths above age 5 years, but younger athletes score slightly below high school and collegiate athletes.<sup>55</sup> This issue is remedied, however, if preseason baseline testing is conducted for all players and a preinjury baseline score established for each



athlete against which changes resulting from concussion can be detected and other factors that affect test performance can be controlled. Users of standardized clinical tools should be aware of the effects of age and education on cognitive test performance and make certain to select the appropriate normative group for comparison when testing an injured athlete at a specific competitive level. Uncertainties about the effects of concussion on young children warrant further study.

## HOME CARE

Once the athlete has been thoroughly evaluated and determined to have sustained a concussion, a comprehensive medical management plan should be implemented. This plan should include frequent medical evaluations and observations, continued monitoring of postconcussion signs and symptoms, and postinjury cognitive and balance testing. If symptoms persist or worsen or the level of consciousness deteriorates at all after a concussion, neuroimaging should be performed. Although scientific evidence for the evaluation and resolution of the concussion is ample, specific management advice to be given to the athlete on leaving the athletic training room is lacking.<sup>94</sup> Athletic trainers and hospital emergency rooms have created various home instruction forms, but minimal scientific evidence supports these instructions. However, despite these limitations, a concussion instruction form (Appendix C) should be given to the athlete and a responsible adult who will have direct contact with the athlete for the initial 24 hours after the injury. This form helps the companion to know what signs and symptoms to watch for and provides useful recommendations on follow-up care.

## Medications

At this time, the clinician has no evidence-based pharmacologic treatment options for an athlete with a concussion.<sup>95</sup> Most pharmacologic studies have been performed in severely head-injured patients. It has been suggested that athletes with concussion avoid medications containing aspirin or nonsteroidal anti-inflammatories, which decrease platelet function and potentially increase intracranial bleeding, mask the severity and duration of symptoms, and possibly lead to a more severe injury. It is also recommended that acetaminophen (Tylenol, McNeil Consumer & Specialty Pharmaceuticals, Fort Washington, PA) be used sparingly in the treatment of headache-like symptoms in the athlete with a concussion. Other substances to avoid during the acute postconcussion period include those that adversely affect central nervous function, in particular alcohol and narcotics.

## Wake-Ups and Rest

Once it has been determined that a concussion has been sustained, a decision must be made as to whether the athlete can return home or should be considered for overnight observation or admission to the hospital. For more severe injuries, the athlete should be evaluated by the team physician or emergency room physician if the team physician is not available. If the athlete is allowed to return home or to the dormitory room, the ATC should counsel a friend, teammate, or parent to closely monitor the athlete. Traditionally, part of these instructions included a recommendation to wake up the athlete every 3 to 4 hours during the night to evaluate changes in

symptoms and rule out the possibility of an intracranial bleed, such as a subdural hematoma. This recommendation has raised some debate about unnecessary wake-ups that disrupt the athlete's sleep pattern and may increase symptoms the next day because of the combined effects of the injury and sleep deprivation. It is further suggested that the concussed athlete have a teammate or friend stay during the night and that the athlete not be left alone. No documented evidence suggests what severity of injury requires this treatment. However, a good rule to use is if the athlete experienced LOC, had prolonged periods of amnesia, or is still experiencing significant symptoms, he or she should be awakened during the night. Both oral and written instructions should be given to both the athlete and the caregiver regarding waking.<sup>96</sup> The use of written and oral instructions increases the compliance to 55% for purposeful waking in the middle of the night. In the treatment of concussion, complete bed rest was ineffective in decreasing postconcussion signs and symptoms.<sup>97</sup> The athlete should avoid activities that may increase symptoms (eg, staying up late studying and physical education class) and should resume normal activities of daily living, such as attending class and driving, once symptoms begin to resolve or decrease in severity. As previously discussed, a graded test of exertion should be used to determine the athlete's ability to safely return to full activity.

## Diet

Evidence is limited to support the best type of diet for aiding in the recovery process after a concussion. A cascade of neurochemical, ionic, and metabolic changes occur after brain injury.<sup>47</sup> Furthermore, some areas of the brain demonstrate glycolytic increases and go into a state of metabolic depression as a result of decreases in both glucose and oxidative metabolism with a reduction in cerebral blood flow. Severely brain-injured subjects ate larger meals and increased their daily caloric intake when compared with controls.<sup>98</sup> Although limited information is available regarding the recommended diet for the management of concussion, it is well accepted that athletes should be instructed to avoid alcohol, illicit drugs, and central nervous system medications that may interfere with cognitive function. A normal, well-balanced diet should be maintained to provide the needed nutrients to aid in the recovery process from the injury.

## EQUIPMENT ISSUES

### Helmets and Headgear

Although wearing a helmet will not prevent all head injuries, a properly fitted helmet for certain sports reduces the risk of such injuries. A poorly fitted helmet is limited in the amount of protection it can provide, and the ATC must play a role in enforcing the proper fitting and use of the helmet. Protective sport helmets are designed primarily to prevent catastrophic injuries (ie, skull fractures and intracranial hematomas) and are not designed to prevent concussions. A helmet that protects the head from a skull fracture does not adequately prevent the rotational and shearing forces that lead to many concussions.<sup>99</sup>

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

also recommended for recreational sports such as bicycling, skiing, mountain biking, roller and inline skating, and speed skating. Headgear standards are established and tested by the National Operating Committee on Standards for Athletic Equipment and the American Society for Testing and Materials.<sup>99</sup>

Efforts to establish and verify standards continue to be tested and refined, but rarely are the forces and conditions experienced on the field by the athletes duplicated. In addition to direction, speed, and amount of the forces delivered and received by the athlete, conditions not controlled in the testing process include weather conditions, changes in external temperatures and temperatures inside the helmet, humidity levels, coefficient of friction for the surfaces of the equipment and ground, and density of the equipment and ground. However, equipment that does meet the standards is effective in reducing head injuries.<sup>99</sup>

More recently, the issue of headgear for soccer players has received much attention. Although several soccer organizations and governing bodies have approved the use of protective headbands in soccer, no published, peer-reviewed studies support their use. Recommendations supporting the use and performance of headgear for soccer are limited by a critical gap in biomechanical information about head impacts in the sport of soccer. Without data linking the severity and type of impacts and the clinical sequelae of single and repeated impacts, specifications for soccer headgear cannot be established scientifically. These types of headgear may reduce the "sting" of a head impact, yet they likely do not meet other sports headgear performance standards. This type of headgear may actually increase the incidence of injury. Players wearing headgear may have the false impression that the headgear will protect them during more aggressive play and thereby subject themselves to even more severe impacts that may not be attenuated by the headgear.

## Mouth Guards

The wearing of a mouth guard is thought by some to provide additional protection for the athlete against concussion by either reducing the risk of injury or reducing the severity of the injury itself.<sup>100</sup> Mouth guards aid in the separation between the head of the condyle of the mandible and the base of the skull. It is thought that wearing an improperly fitted mouth guard or none at all increases this contact point. This theory, which is based on Newtonian laws of physics, suggests that the increased separation between 2 adjacent structures increases the time to contact, thus decreasing the amount of contact and decreasing the trauma done to the brain.<sup>100</sup> However, no biomechanical studies support the theory that the increased separation results in less force being delivered to the brain.

High school football and National Collegiate Athletic Association football rules mandate the wearing of a mouth guard, but the National Football League rulebook does not require players to wear a mouth guard. The National Collegiate Athletic Association requires mouth guards to be worn by all athletes in football, field hockey, ice hockey, and lacrosse. Researchers<sup>101,102</sup> have found no advantage in wearing a custom-made mouth guard over a boil-and-bite mouth guard to reduce the rise of cerebral concussion in athletes. However, ATCs and coaches should mandate the regular use of mouth guards because a properly fitted mouth guard, with no alterations such as cutting off the back part, is of great value in

protecting the teeth and preventing fractures and avulsions that could require many years of expensive dental care.

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<b>Graded Symptom Checklist (GSC)</b>					
<b>Symptom</b>	<b>Time of injury</b>	<b>2-3 Hours postinjury</b>	<b>24 Hours postinjury</b>	<b>48 Hours postinjury</b>	<b>72 Hours postinjury</b>
Blurred vision					
Dizziness					
Drowsiness					
Excess sleep					
Easily distracted					
Fatigue					
Feel "in a fog"					
Feel "slowed down"					
Headache					
Inappropriate emotions					
Irritability					
Loss of consciousness					
Loss or orientation					
Memory problems					
Nausea					
Nervousness					
Personality change					
Poor balance/coordination					
Poor concentration					
Ringing in ears					
Sadness					
Seeing stars					
Sensitivity to light					
Sensitivity to noise					
Sleep disturbance					
Vacant stare/glassy eyed					
Vomiting					

NOTE: The GSC should be used not only for the initial evaluation but for each subsequent follow-up assessment until all signs and symptoms have cleared at rest and during physical exertion. In lieu of simply checking each symptom present, the ATC can ask the athlete to grade or score the severity of the symptom on a scale of 0-6, where 0=not present, 1=mild, 3=moderate, and 6=most severe.

## Appendix B. Physician Referral Checklist

### Day-of-injury referral

1. Loss of consciousness on the field
2. Amnesia lasting longer than 15 min
3. Deterioration of neurologic function\*
4. Decreasing level of consciousness\*
5. Decrease or irregularity in respirations\*
6. Decrease or irregularity in pulse\*
7. Increase in blood pressure
8. Unequal, dilated, or unreactive pupils\*
9. Cranial nerve deficits
10. Any signs or symptoms of associated injuries, spine or skull fracture, or bleeding\*
11. Mental status changes: lethargy, difficulty maintaining arousal, confusion, or agitation\*
12. Seizure activity\*
13. Vomiting

14. Motor deficits subsequent to initial on-field assessment
15. Sensory deficits subsequent to initial on-field assessment
16. Balance deficits subsequent to initial on-field assessment
17. Cranial nerve deficits subsequent to initial on-field assessment
18. Postconcussion symptoms that worsen
19. Additional postconcussion symptoms as compared with those on the field
20. Athlete is still symptomatic at the end of the game (especially at high school level)

### Delayed referral (after the day of injury)

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

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

## Appendix C. Concussion Home Instructions

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

1. Please **remind** \_\_\_\_\_ to report to the athletic training room tomorrow at \_\_\_\_\_ for a follow-up evaluation.

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

### It is OK to:

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

### There is NO need to:

- Check eyes with flashlight
- Wake up every hour
- Test reflexes
- Stay in bed

### Do NOT:

- Drink alcohol
- Eat spicy foods

Specific recommendations:

Recommendations provided to: \_\_\_\_\_

Recommendations provided by: \_\_\_\_\_ Date: \_\_\_\_\_ Time: \_\_\_\_\_

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

Signature: \_\_\_\_\_ Date: \_\_\_\_\_

# Preseason Heat-Acclimatization Guidelines for Secondary School Athletics

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A proper heat-acclimatization plan in secondary school athletic programs is essential to minimize the risk of exertional heat illness during the preseason practice period. Gradually increasing athletes' exposure to the duration and intensity of physical activity and to the environment minimizes exertional heat-illness risk while improving athletic performance. Progressive acclimatization is especially important during the initial 3 to 5 days of summer practices. When an athlete undergoes a proper heat-acclimatization program, physiologic function, exercise heat tolerance, and exercise performance are all enhanced.<sup>1–6</sup> In contrast, athletes who are not exposed to a proper heat-acclimatization program face measurable increased risks for exertional heat illness.

For these reasons, the Inter-Association Task Force for Preseason Secondary School Athletics, in conjunction with the National Athletic Trainers' Association's Secondary School Athletic Trainers' Committee, recommends that these "Preseason Heat-Acclimatization Guidelines for Secondary School Athletics" be implemented by all secondary school athletic programs. These guidelines should be used for all preseason conditioning, training, and practice activities in a warm or hot environment, whether these activities are conducted indoors or outdoors. When athletic programs implement these guidelines, the health and safety of the athletes are primary. However, the recommendations outlined here are only minimum standards, based on the best heat-acclimatization evidence available. Following these guidelines provides all secondary school athletes an opportunity to train safely and effectively during the preseason practice period.

## DEFINITIONS

Before participating in the preseason practice period, all student-athletes should undergo a preparticipation medical

examination administered by a physician (MD or DO) or as required/approved by state law. The examination can identify predisposing factors related to a number of safety concerns, including the identification of youths at particular risk for exertional heat illness.

The *heat-acclimatization period* is defined as the initial 14 consecutive days of preseason practice for all student-athletes. The goal of the acclimatization period is to enhance exercise heat tolerance and the ability to exercise safely and effectively in warm to hot conditions. This period should begin on the first day of practice or conditioning before the regular season. Any practices or conditioning conducted before this time should not be considered a part of the heat-acclimatization period. Regardless of the conditioning program and conditioning status leading up to the first formal practice, all student-athletes (including those who arrive at preseason practice after the first day of practice) should follow the 14-day heat-acclimatization plan. During the preseason heat-acclimatization period, if practice occurs on 6 consecutive days, student-athletes should have 1 day of complete rest (no conditioning, walk-throughs, practices, etc).

Days on which athletes do not practice due to a scheduled rest day, injury, or illness do not count toward the heat-acclimatization period. For example, an athlete who sits out the third and fourth days of practice during this time (eg, Wednesday and Thursday) will resume practice as if on day 3 of the heat-acclimatization period when returning to play on Friday.

A *practice* is defined as the period of time a participant engages in a coach-supervised, school-approved, sport- or conditioning-related physical activity. Each individual practice should last no more than 3 hours. Warm-up, stretching, and cool-down activities are included as part of the 3-hour practice time. Regardless of ambient tempera-

ture conditions, all conditioning and weight-room activities should be considered part of practice.

A *walk-through* is defined as a teaching opportunity with the athletes not wearing protective equipment (eg, helmets, shoulder pads, catcher's gear, shin guards) or using other sport-related equipment (eg, footballs, lacrosse sticks, blocking sleds, pitching machines, soccer balls, marker cones). The walk-through is not part of the 3-hour practice period, can last no more than 1 hour per day, and does not include conditioning or weight-room activities.

A *recovery period* is defined as the time between the end of 1 practice or walk-through and the beginning of the next practice or walk-through. During this time, athletes should rest in a cool environment, with no sport- or conditioning-related activity permitted (eg, speed or agility drills, strength training, conditioning, or walk-through). Treatment with the athletic trainer is permissible.

## RECOMMENDATIONS FOR THE 14-DAY HEAT-ACCLIMATIZATION PERIOD

1. Days 1 through 5 of the heat-acclimatization period consist of the first 5 days of formal practice. During this time, athletes may not participate in more than 1 practice per day.
2. If a practice is interrupted by inclement weather or heat restrictions, the practice should recommence once conditions are deemed safe. Total practice time should not exceed 3 hours in any 1 day.
3. A 1-hour maximum walk-through is permitted during days 1–5 of the heat-acclimatization period. However, a 3-hour recovery period should be inserted between the practice and walk-through (or vice versa).
4. During days 1–2 of the heat-acclimatization period, in sports requiring helmets or shoulder pads, a helmet should be the only protective equipment permitted (goalies, as in the case of field hockey and related sports, should *not* wear full protective gear or perform activities that would require protective equipment). During days 3–5, only helmets and shoulder pads should be worn. Beginning on day 6, all protective equipment may be worn and full contact may begin.
  - A. Football only: On days 3–5, contact with blocking sleds and tackling dummies may be initiated.
  - B. Full-contact sports: 100% live contact drills should begin no earlier than day 6.
5. Beginning no earlier than day 6 and continuing through day 14, double-practice days must be followed by a single-practice day. On single-practice days, 1 walk-through is permitted, separated from the practice by at least 3 hours of continuous rest. When a double-practice day is followed by a rest day, another double-practice day is permitted after the rest day.
6. On a double-practice day, neither practice should exceed 3 hours in duration, and student-athletes should not participate in more than 5 total hours of practice. Warm-up, stretching, cool-down, walk-through, conditioning, and weight-room activities are included as part of the practice time. The 2 practices should be separated by at least 3 continuous hours in a cool environment.
7. Because the risk of exertional heat illnesses during the preseason heat-acclimatization period is high, we strongly recommend that an athletic trainer be on site before, during, and after all practices.

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The National Athletic Trainers' Association (NATA) and the Inter-Association Task Force for Preseason Secondary School Athletics advise individuals, schools, athletic training facilities, and institutions to carefully and independently consider each of the recommendations. The information contained in the statement is neither exhaustive nor exclusive to all circumstances or individuals. Variables such as institutional human resource guidelines, state or federal statutes, rules, or regulations, as well as regional environmental conditions, may impact the relevance and implementation of these recommendations. The NATA and the Inter-Association Task Force advise their members and others to carefully and independently consider each of the recommendations (including the applicability of same to any particular circumstance or individual). The foregoing statement should not be relied upon as an independent basis for care but rather as a resource available to NATA members or others. Moreover, no opinion is expressed herein regarding the quality of care that adheres to or differs from any of NATA's other statements. The NATA and the Inter-Association Task Force reserve the right to rescind or modify their statements at any time.



# Inter-Association Task Force on Exertional Heat Illnesses Consensus Statement

These guidelines were established to increase safety and performance for individuals engaged in physical activity, especially in warm and hot environments. The risks associated with exercise in the heat are well documented, but policies and procedures often do not reflect current state-of-the-art knowledge. Many cases of exertional heat illness are preventable and can be successfully treated if onsite personnel identify the condition and implement appropriate care in a timely manner.

Strategies to optimize proper care of dehydration, exertional heat stroke (EHS), heat exhaustion, heat cramps and exertional hyponatremia are presented here. This consensus statement was developed by medical/scientific experts experienced in the prevention, recognition and treatment of exertional heat illnesses.

## Overall Strategies for the Prevention of Exertional Heat Illnesses

Every athletic organization should have a policy, procedure or emergency plan established to address exertional heat illnesses. A thorough plan includes the key factors to prevent, identify and treat exertional heat illnesses.

Scientific evidence indicates the following factors may increase the risk associated with exercise in the heat. Although some factors can be optimized (e.g., heat acclimatization), others cannot (e.g., health problems). Regardless, these factors may help in developing a proactive approach to preventing exertional heat illnesses.

### *Intrinsic factors include:*

- History of exertional heat illnesses
- Inadequate heat acclimatization
- Lower level of fitness status
- Higher percent body fat
- Dehydration or overhydration
- Presence of a fever
- Presence of gastrointestinal illness
- Salt deficiency
- Skin condition (e.g., sunburn, skin rash, etc.)
- Ingestion of certain medications (e.g., antihistamines, diuretics, etc.) or dietary supplements (e.g., ephedra, etc.)
- Motivation to push oneself/warrior mentality
- Reluctance to report problems, issues, illness, etc.
- Pre-pubescence

### *Extrinsic factors include:*

- Intense or prolonged exercise with minimal breaks
- High temperature/humidity/sun exposure (Table 1 and Figure 1), as well as exposure to heat/humidity in preceding days
- Inappropriate work/rest ratios based on intensity, wet bulb globe temperature (WBGT), clothing, equipment, fitness

and athlete's medical condition

- Lack of education and awareness of heat illnesses among coaches, athletes and medical staff
- No emergency plan to identify and treat exertional heat illnesses
- No access to shade during exercise or during rest breaks
- Duration and number of rest breaks is limited
- Minimal access to fluids before and during practice and rest breaks
- Delay in recognition of early warning signs

## General Considerations for Risk Reduction

- Encourage proper education regarding heat illnesses (for athletes, coaches, parents, medical staff, etc.). Education about risk factors should focus on hydration needs, acclimatization, work/rest ratio, signs and symptoms of exertional heat illnesses, treatment, dietary supplements, nutritional issues and fitness status.
- Provide medical services onsite (e.g., certified athletic trainer [ATC], emergency medical technician [EMT], physician).
- Ensure pre-participation physical examination that includes specific questions regarding fluid intake, weight changes during activity, medication and supplement use and history of cramping/heat illnesses has been completed.
- Assure that onsite medical staff has authority to alter work/rest ratios, practice schedules, amount of equipment and withdrawal of individuals from participation based on environment and/or athlete's medical condition.

## DEHYDRATION

### *Factors Contributing to Onset of Condition*

When athletes do not replenish lost fluids, they become dehydrated. Mild dehydration (<2% body weight loss [BWL]) is often unavoidable because athletes cannot always replenish fluids at a rate equal to that being lost. Dehydration as minimal as 2% BWL can begin to hinder performance and thermoregulatory function.

**Table 1**  
Wet Bulb Globe Temperature Risk Chart

WBGT	Flag Color	Level of Risk	Comments
<65°F (<18°C)	Green	Low	Risk low but still exists on the basis of risk factors
65°-73°F (18°-23°C)	Yellow	Moderate	Risk level increases as event progresses through the day
73°-82°F (23°-28°C)	Red	High	Everyone should be aware of injury potential; individuals at risk should not compete
>82°F (>28°C)	Black	Extreme or hazardous	Consider rescheduling or delaying the event until safer conditions prevail; if the event must take place, be on high alert. Take steps to reduce risk factors (e.g., more and longer rest breaks, reduced practice time, reduced exercise intensity, access to shade, minimal clothing and equipment, cold tubs at practice site, etc.).

The WBGT can be measured with a WBGT meter. The calculation for the determination of WBGT is:  $WBGT = .7 (\text{Wet Bulb Temperature}) + .2 (\text{Black Globe Temperature}) + .1 (\text{Dry Bulb Temperature})$ .

This table was originally printed in Roberts WO. Medical management and administration manual for long distance road racing. In: Brown CH, Gudjonsson B, eds. *IAAF Medical Manual for Athletics and Road Racing Competitions: a Practical Guide*. Monaco: International Association of Athletics Federations;1998:39-75.

Optimal hydration is the replacement of fluids and electrolytes in accordance with individual needs. Fluid intake should nearly approximate fluid losses. Athletes must personally establish and monitor fluid requirements and modify behavior to ensure optimal hydration status. Fluid intake beyond fluid needs for many hours also can be quite harmful (see Exertional Hyponatremia).

### Recognition

Indicators include dry mouth, thirst, irritability, general discomfort, headache, apathy, weakness, dizziness, cramps, chills, vomiting, nausea, head or neck heat sensations, excessive fatigue and/or decreased performance.

### Treatment

The following procedures are recommended if dehydration is suspected:

- Dehydrated athletes should move to a cool environment and rehydrate.
- Maintaining normal hydration (as indicated by baseline body weight) is critical to avoiding heat illnesses. If an athlete's BWL is greater than 1% to 2% within a given day or on consecutive days, that athlete should return to normal hydration status before being allowed to practice. (Remember that pre-exercise/event/participation examination body weight baseline measures may not accurately assess hydration status if post-practice body weight is being compared to a baseline that is measured in a dehydrated state. Urine specific gravity or urine color can help with this assessment if an athlete is suspected to be dehydrated at the time baseline measurements are taken.)

- Athletes should begin exercise sessions properly hydrated. Any fluid deficits should be replaced within 1 to 2 hours after exercise is complete.
- Given the nature of sweat and variability and timing of nutritional intake, hydrating with a sports drink containing carbohydrates and electrolytes (i.e., sodium and potassium) before and during exercise is optimal to replace losses and provide energy. Because athletes replace only about half of the fluid lost when drinking water, a flavored sports drink may promote an increase in the quantity of fluids consumed.
- Replacing lost sodium after exercise is best achieved by consuming food in combination with a rehydration beverage.
- Athletes should have convenient access to fluids throughout practice and be allowed to hydrate in addition to prescribed breaks. These factors can minimize dehydration and may maximize performance.
- A nauseated or vomiting athlete should seek medical attention to replace fluids via an intravenous line.

### Return-to-Play Considerations

If the degree of dehydration is minor and the athlete is symptom free, continued participation is acceptable. The athlete must maintain hydration status and should receive periodic checks from onsite medical personnel.

## EXERTIONAL HEAT STROKE

### Factors Contributing to Onset of Condition

Exertional heat stroke is a severe illness characterized by central nervous system (CNS) abnormalities and potentially tissue damage resulting from elevated body temperatures induced by strenuous physical exercise and increased environmental heat stress.

## Recognition

The ability to rapidly and accurately assess core body temperature and CNS functioning is critical to the proper evaluation of EHS; axillary, oral and tympanic temperatures are not valid measures in individuals exercising in hot environments. Medical staff should be properly trained and equipped to assess core temperature via rectal thermometer when feasible.

*Most critical criteria for determination are* (1) CNS dysfunction (altered consciousness, coma, convulsions, disorientation, irrational behavior, decreased mental acuity, irritability, emotional instability, confusion, hysteria, apathy) and (2) hyperthermic (rectal temperature usually  $>104^{\circ}\text{F}/40^{\circ}\text{C}$ ) immediately post-incident.

*Other possible salient findings include* (1) nausea, vomiting, diarrhea, (2) headache, dizziness, weakness, (3) hot and wet or dry skin (important to note that skin may be wet or dry at time of incident), (4) increased heart rate, decreased blood pressure, increased respiratory rate, (5) dehydration and (6) combativeness.

## Treatment

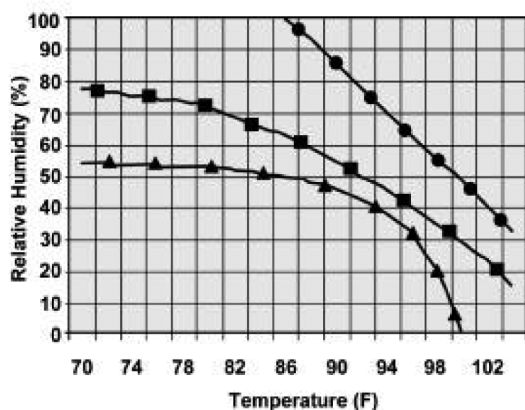
Aggressive and immediate whole-body cooling is the key to optimizing treatment.

The duration and degree of hyperthermia may determine adverse outcomes. If untreated, hyperthermia-induced physiological changes resulting in fatal consequences may occur within vital organ systems (e.g., muscle, heart, brain, liver, kidneys, etc.). Due to superior cooling rates, immediate whole-body cooling via cold water immersion is the best treatment for EHS and should be initiated within minutes post-incident.

Provided that adequate emergency medical care is available onsite (i.e., ATC, EMT or physician), it is recommended to cool first via cold water immersion, then transport second. Cooling can be successfully verified by measuring rectal temperature. If onsite rapid cooling via cold water immersion is not an option or if other complications develop that would be considered life threatening (i.e., airway, breathing, circulation), immediate transport to the nearest medical facility is essential.

The following procedures are recommended if EHS is suspected:

- Immediately immerse athlete in tub of cold water (approximately  $35^{\circ}\text{--}58^{\circ}\text{F}/1.67^{\circ}\text{--}14.5^{\circ}\text{C}$ ), onsite if possible. Remove clothing/equipment. (Immersion therapy should include constant monitoring of core temperature by rectal thermistor [or thermometer].)
- If immersion is not possible, transport immediately. Alternative cooling strategies should be implemented while waiting for and during transport. These strategies could include: spraying the body with cold water, fans, ice bags or ice over as much of the body as possible and/or cold towels (replace towels frequently).
- Monitor airway, breathing, circulation, core temperature, and CNS status (cognitive, convulsions, orientation, consciousness, etc.) at all times.
- Place an intravenous line using normal saline (if appropriate medical staff is available).
- Cease aggressive cooling when core temperature reaches approximately  $101^{\circ}\text{--}102^{\circ}\text{F}$  ( $38.3^{\circ}\text{--}38.9^{\circ}\text{C}$ ); continue to monitor.
- If rapid onsite cooling was administered and rectal temperature has reached approximately  $101^{\circ}\text{--}102^{\circ}\text{F}$  ( $38.3^{\circ}\text{--}38.9^{\circ}\text{C}$ ), transport athlete to medical facility for monitoring of possible organ system damage.



**Figure 1. Heat stress risk temperature and humidity graph.** Heat stroke risk rises with increasing heat and relative humidity. Fluid breaks should be scheduled for all practices and scheduled more frequently as the heat stress rises. Add  $5^{\circ}$  to temperature between 10 a.m. and 4 p.m. from mid-May to mid-September on bright, sunny days. Practices should be modified for the safety of the athletes to reflect the heat stress conditions. Regular practices with full practice gear can be conducted for conditions that plot to the left of the triangles. Cancel all practices when the temperature and relative humidity plot is to the right of the circles; practices may be moved into air-conditioned spaces or held as walk through sessions with no conditioning activities.

Conditions that plot between squares and circles: use work/rest ratio with 15 to 20 minutes of activity followed by 5- to 10-minute rest and fluid breaks; practice should be in shorts only (with all protective equipment removed, if worn for activity).

Conditions that plot between triangles and squares: use work/rest ratio with 20 to 25 minutes of activity followed by 5- to 10-minute rest and fluid breaks; practice should be in shorts (with helmets and shoulder pads only, not full equipment, if worn for activity).

Conditions that plot beneath triangles (through remaining range of chart): use work/rest ratio with 25 to 30 minutes of activity followed by 5- to 10-minute rest and fluid breaks.

Adapted with permission from Kulka J, Kenney WL. Heat balance limits in football uniforms: how different uniform ensembles alter the equation. *Physician Sportsmed*. 2002;30(7):29-39.



### ***Return-to-Play Considerations***

Physiological changes may occur after an episode of EHS. For example, the athlete's heat tolerance may be temporarily or permanently compromised. To ensure a safe return to full participation, a careful return-to-play strategy should be decided by the athlete's physician and implemented with the assistance of the ATC or other qualified health care professional.

The following guidelines are recommended for return-to-play after EHS:

- Physician clearance is necessary before returning to exercise. The athlete should avoid all exercise until completely asymptomatic and all laboratory tests are normal.
- Severity of the incident should dictate the length of recovery time.
- The athlete should avoid exercise for the minimum of 1 week after release from medical care.
- The athlete should cautiously begin a gradual return to physical activity to regain peak fitness and acclimatization under the supervision of an ATC or other qualified health care professional. Type and length of exercise should be determined by the athlete's physician and might follow this pattern:
  1. Easy-to-moderate exercise in a climate-controlled environment for several days, followed by strenuous exercise in a climate-controlled environment for several days.
  2. Easy-to-moderate exercise in heat for several days, followed by strenuous exercise in heat for several days.
  3. (If applicable) Easy-to-moderate exercise in heat with equipment for several days, followed by strenuous exercise in heat with equipment for several days.

## **HEAT EXHAUSTION**

### ***Factors Contributing to Onset of Condition***

Heat exhaustion is a moderate illness characterized by the inability to sustain adequate cardiac output, resulting from strenuous physical exercise and environmental heat stress. Inherent needs to maintain blood pressure and essential organ function, combined with a loss of fluid due to acute dehydration, create a challenge the body cannot meet, especially if intense exercise were to continue unabated.

### ***Recognition***

*Most critical criteria for determination* are (1) athlete has obvious difficulty continuing intense exercise in heat, (2) lack of severe hyperthermia (usually <104°F/40°C), although it would be expected to find mild hyperthermia at the time of the incident (more commonly, 100°-103°F/37.7°-39.4°C) and (3) lack of severe CNS dysfunction. If any CNS dysfunction

(see symptoms listed under EHS) is present, it will be mild and symptoms will subside quickly with treatment and as activity is discontinued.

*Other possible salient findings include* (1) physical fatigue, (2) dehydration and/or electrolyte depletion, (3) ataxia and coordination problems, syncope, dizziness, (4) profuse sweating, pallor, (5) headache, nausea, vomiting, diarrhea, (6) stomach/intestinal cramps, persistent muscle cramps and (7) rapid recovery with treatment.

### ***Treatment***

The following procedures are recommended if heat exhaustion is suspected:

- Remove athlete from play and immediately move to a shaded or air-conditioned area.
- Remove excess clothing and equipment.
- Cool athlete until rectal temperature is approximately 101°F (38.3°C).
- Have athlete lie comfortably with legs propped above heart level.
- If athlete is not nauseated, vomiting or experiencing any CNS dysfunction, rehydrate orally with chilled water or sports drink. If athlete is unable to take oral fluids, implement intravenous infusion of normal saline.
- Monitor heart rate, blood pressure, respiratory rate, rectal temperature and CNS status.
- Transport to an emergency facility if rapid improvement is not noted with prescribed treatment.

### ***Return-to-Play Considerations***

The following guidelines are recommended for return-to-play after heat exhaustion:

- Athlete should be symptom free and fully hydrated.
- Recommend physician clearance or, at minimum, a discussion with supervising physician before return.
- Rule out underlying condition or illness that predisposed athlete for continued problems.
- Avoid intense practice in heat until at least the next day to ensure recovery from fatigue and dehydration. (In severe cases, intense practice in heat should be delayed for more than 1 day.)
- If underlying cause was lack of acclimatization and/or fitness level, correct this problem before athlete returns to full-intensity training in heat (especially in sports with equipment).

## **HEAT CRAMPS**

### ***Factors Contributing to Onset of Condition***

The etiology of muscle cramps is not well understood and there may be a number of causes. Heat cramps are often present in athletes who perform strenuous exercise in the heat. Conversely, cramps also occur in the absence of warm or hot conditions (e.g., common in ice hockey players).



Whether or not heat related, cramps tend to occur later in an activity, in conjunction with muscle fatigue and after fluid and electrolyte imbalances have reached a critical level.

Dehydration, diet poor in minerals, and large losses of sodium and other electrolytes in sweat appear to increase the risk of severe, often whole-body, muscle cramps. Muscle cramps can largely be avoided with adequate conditioning, acclimatization, rehydration, electrolyte replacement and appropriate dietary practices.

### Recognition

*Most critical criteria for determination are* (1) intense pain (not associated with acute muscle strain) and (2) persistent muscle contractions in working muscles during and after prolonged exercise and most often associated with exercise in heat.

*Other possible salient findings include* (1) "salty sweaters" (those with high salt concentration in sweat), (2) high sweat rate, heavy sweating, (3) lack of heat acclimatization, (4) insufficient sodium intake (during meals and practice), (5) dehydration, thirsty, (6) irregular meals, (7) increased fatigue and (8) previous cramping history.

### Treatment

The following procedures are recommended if heat cramps are suspected:

- Re-establish normal hydration status and replace some sodium losses with a sports drink or other sodium source.
- Some additional sodium may be needed (especially in those with a history of heat cramps) earlier in the activity (pre-cramps) and is best administered by dilution into a sports drink. For example, 1/2 g of sodium (equal to the amount of sodium found in 1/4 tsp of table salt) dissolved in about 1 L (approximately 32 oz) of a sports drink early in the exercise session provides ample fluids and sodium, and the flavor (while certainly saltier) is still very palatable.
- Light stretching, relaxation and massage of the involved muscle may help acute pain of a muscle cramp.

### Return-to-Play Considerations

Athletes should be assessed to determine if they can perform at the level needed for successful participation. After an acute episode, diet, rehydration practices, electrolyte consumption, fitness status, level of acclimatization and use of dietary supplements should be reviewed and possibly modified to decrease risk of recurring heat cramps.

**Table 2**

Sample Sweat Rate Calculation\*

A	B	C	D	E	F	G	H	I	J
Body Weight									
Name	Date	Before Exercise	After Exercise	Change in BW (C-D)	Drink Volume	Urine Volume †	Sweat Loss (E+F-G)	Exercise Time	Sweat Rate (H/I)
		kg	kg	g	mL	mL	mL	min	mL/min
		(lb/2.2)	(lb/2.2)	(kg x 1000)	(oz x 30)	(oz x 30)	(oz x 30)	h	mL/h
		kg	kg	g	mL	mL	mL	min	mL/min
		(lb/2.2)	(lb/2.2)	(kg x 1000)	(oz x 30)	(oz x 30)	(oz x 30)	h	mL/h
		kg	kg	g	mL	mL	mL	min	mL/min
		(lb/2.2)	(lb/2.2)	(kg x 1000)	(oz x 30)	(oz x 30)	(oz x 30)	h	mL/h
		kg	kg	g	mL	mL	mL	min	mL/min
		(lb/2.2)	(lb/2.2)	(kg x 1000)	(oz x 30)	(oz x 30)	(oz x 30)	h	mL/h
Kelly K. ‡	9/15	61.7 kg	60.3 kg	1400 g	420 mL	90 mL	1730 mL	90 min	19 mL/min
		(lb/2.2)	(lb/2.2)	(kg x 1000)	(oz x 30)	(oz x 30)	(oz x 30)	1.5 h	1153 mL/h

\* Reprinted with permission from Murray R. Determining sweat rate. *Sports Sci Exch.* 1996; 9 (Suppl 63).

† Weight of urine should be subtracted if urine was excreted prior to post-exercise body weight.

‡ In the example, Kelly K. should drink about 1 L (32 oz.) of fluid during each hour of activity to remain well hydrated.

### Formula for Calculating Sweat Rate

When calculating an athlete's sweat rate (sweat rate = pre-exercise body weight - post-exercise body weight + fluid intake - urine volume/exercise time in hours), do so for a representative range of environmental conditions, practices and competitions.

The simplest way to get athletes to focus on their hydration needs is to teach them to compare pre-exercise and post-exercise body weights. If the athletes lost weight, they need to drink more at the next practice; if they gained weight, they should drink less. This gives the athletes immediate feedback about their drinking habits.

## EXERTIONAL HYPONATREMIA

### *Factors Contributing to Onset of Condition*

When an athlete consumes more fluids (especially water) than necessary, and/or sodium lost in sweat is not adequately replaced, sodium in the bloodstream can become diluted and cause cerebral and/or pulmonary edema. This is called hyponatremia (low blood-sodium levels) and tends to occur during warm/hot weather activities.

The risk of acquiring hyponatremia can be substantially reduced if fluid consumption during activity does not exceed fluid losses and sodium is adequately replaced. Because progressive dehydration may also compromise thermoregulatory function, it is of great value for an athlete to be aware of individual fluid needs to protect against both dehydration and overhydration.

Fluid needs can be determined by establishing an athlete's "sweat rate" (liters per hour) or the amount of fluid lost in a given length of time (usually discussed in an amount per hour) during a given intensity of activity, while wearing a given amount of clothing/equipment, for a given set of environmental conditions (Table 2). Variations can exist in sweat rates, so individual assessments can be quite helpful (especially in at-risk individuals). When establishing fluid needs, it is best to mimic the same conditions of the athletic event to establish an accurate sweat rate.

### **Recognition**

*Most critical criteria for determination are* (1) low blood-sodium levels (<130 mmol/L). Severity of condition increases as sodium levels decrease, (2) likelihood of excessive fluid consumption before, during and after exercise (weight gain during activity), (3) low sodium intake, (4) likelihood of sodium deficits before, during and after exercise and (5) if condition progresses, CNS changes (e.g., altered consciousness, confusion, coma, convulsions, altered cognitive functioning) and respiratory changes resulting from cerebral and/or pulmonary edema, respectively.

*Other possible salient findings include* (1) increasing headache, (2) nausea, vomiting (often repetitive), (3) swelling of extremities (hands and feet), (4) irregular diet (e.g., inadequate sodium intake), (5) during prolonged activity (often lasting >4 hours), (6) copious urine with low specific gravity following exercise, (7) lethargy/apathy, (8) agitation and (9) absence of severe hyperthermia (most commonly <104°F/40°C).

### **Treatment**

The following procedures are recommended if exertional hyponatremia is suspected:

- If blood sodium levels cannot be determined onsite, hold

off on rehydrating athlete (may worsen condition) and transport immediately to a medical facility.

- The delivery of sodium, certain diuretics or intravenous solutions may be necessary. All will be monitored in the emergency department to ensure no complications develop.

### **Return-to-Play Considerations**

The following guidelines are recommended for return-to-play after exertional hyponatremia:

- Physician clearance is strongly recommended in all cases.
- In mild cases, activity can resume a few days after completing an educational session on establishing an individual-specific hydration protocol. This will ensure the proper amount and type of beverages and meals are consumed before, during and after physical activity (see Table 2).

## EXPERT PANEL

The Inter-Association Task Force on Exertional Heat Illnesses  
was composed of representatives from the following organizations:

American Academy of Pediatrics Oded Bar-Or, MD	Gatorade Sports Science Institute Bob Murray, PhD North American Society for Pediatric Exercise Medicine Bareket Falk, PhD
American College of Emergency Physicians Stephen Cantrill, MD, FACEP	National Association of Sport and Physical Education/ AAHPERD Christine Bolger
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American Medical Society for Sports Medicine Kim Fagan, MD	National Strength and Conditioning Association Michael Barnes, MEd, CSCS*D, NSCA-CPT
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American Osteopathic Academy of Sports Medicine Phillip Zinni III, DO, FAOASM, ATC-L	
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CDC - Nutrition and Physical Activity C. Dexter (Bo) Kimsey, Jr, PhD, MSEH	
Department of Defense Health Affairs John W. Gardner, MD, DrPH; COL, MC, FS, USA	

*The information contained within this document does not necessarily reflect endorsement  
from the individual organizations listed above.*

# Inter-Association Task Force Recommendations on Emergency Preparedness and Management of Sudden Cardiac Arrest in High School and College Athletic Programs: A Consensus Statement

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**Objective:** To assist high school and college athletic programs prepare for and respond to a sudden cardiac arrest (SCA). This consensus statement summarizes our current understanding of SCA in young athletes, defines the necessary elements for emergency preparedness, and establishes uniform treatment protocols for the management of SCA.

**Background:** Sudden cardiac arrest is the leading cause of death in young athletes. The increasing presence of and timely access to automated external defibrillators (AEDs) at sporting events provides a means of early defibrillation and the potential for effective secondary prevention of sudden cardiac death. An Inter-Association Task Force was sponsored by the National Athletic Trainers' Association to develop consensus recommendations on emergency preparedness and management of SCA in athletes.

**Recommendations:** Comprehensive emergency planning is needed for high school and college athletic programs to ensure

an efficient and structured response to SCA. Essential elements of an emergency action plan include establishment of an effective communication system, training of anticipated responders in cardiopulmonary resuscitation and AED use, access to an AED for early defibrillation, acquisition of necessary emergency equipment, coordination and integration of on-site responder and AED programs with the local emergency medical services system, and practice and review of the response plan. Prompt recognition of SCA, early activation of the emergency medical services system, the presence of a trained rescuer to initiate cardiopulmonary resuscitation, and access to early defibrillation are critical in the management of SCA. In any collapsed and unresponsive athlete, SCA should be suspected and an AED applied as soon as possible for rhythm analysis and defibrillation if indicated.

**Key Words:** sudden cardiac death, athletes, emergency action plan, automated external defibrillators

Sudden cardiac arrest (SCA) is the leading cause of death in young athletes.<sup>1,2</sup> Athletes are considered the healthiest members of our society, and their unexpected death during training or competition is a catastrophic event that stimulates debate regarding both preparticipation screening evaluations and appropriate emergency planning for athletic events. Despite preparticipation screening, healthy-appearing competitive athletes may harbor unsuspected cardiovascular diseases with the potential to cause sudden death.<sup>3</sup> With the increasing availability of automated external defibrillators (AEDs) at athletic events, there is potential for effective secondary prevention of sudden cardiac death (SCD). The presence and timely access of AEDs at sporting venues provide a means of early defibrillation not only for athletes but also for spectators, coaches, officials, event staff, and other attendees on campus in the case of an unexpected SCA.

Many health-related organizations have guidelines for managing SCA during athletic practices and competitions. However, these guidelines have not directly linked emergency planning and SCA management in athletics. The National Athletic Trainers' Association (NATA) convened an Inter-Association

Task Force in Atlanta, Georgia, on April 24, 2006, to develop consensus recommendations on emergency preparedness and management of SCA in high school and college athletic programs. The task force included representatives from 15 national organizations with special interest in SCA in young athletes and a multidisciplinary group of health care professionals from athletic training, cardiology, electrophysiology, emergency medicine, emergency medical technicians, family medicine, orthopaedics, paramedics, pediatrics, physical therapy, and sports medicine (Appendix).

The goal of this statement is to assist those in high school and college athletic programs to prepare for and respond to an unexpected SCA by summarizing the essential elements of SCA in young athletes and outlining the necessary elements for emergency preparedness and standardized treatment protocols in the management of SCA. Management guidelines are focused on basic life support measures for SCA that can be provided by both bystanders and health care professionals before the arrival of emergency medical services (EMS) personnel. All recommendations in this statement are in agreement with the 2005 American Heart Association (AHA) guidelines



for cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC),<sup>4</sup> the AHA scientific statement on response to cardiac arrest and selected life-threatening medical emergencies and the medical emergency response plan for schools,<sup>5</sup> and the NATA position statement on emergency planning in athletics.<sup>6</sup> Recommendations are directed toward the athletic health care team, including athletic trainers, team physicians, coaches, school administrators, and other potential first responders. This statement is intended for high school and college athletic programs and institutions, although the recommendations may be applicable in other settings.

### Purposes of Consensus Statement

1. To summarize essential information regarding SCA in young athletes
2. To define appropriate emergency preparedness for SCA at athletic venues
3. To establish uniform recommendations for the management of SCA in athletes

### Organization of Consensus Statement

This statement is organized as follows:

1. Executive Summary, including key points on emergency preparedness and management of SCA (Figure)
2. Background and review of the causes of SCD in young athletes, limitations of cardiovascular screening, resuscitation pathophysiology, survival after SCA, and factors affecting survival in young athletes
3. Recommendations for emergency preparedness, including review of the "chain of survival," establishment of an emergency action plan (EAP), access to early defibrillation, emergency communication, emergency personnel, emergency equipment, emergency transportation, practice and review of the EAP, postevent catastrophic incident guidelines, current state of emergency preparedness, and obstacles to implementing AEDs
4. Recommendations for the management of SCA, including the 2005 AHA guidelines for CPR and ECC,<sup>4</sup> the collapsed athlete, recognition of SCA, and management of SCA for both witnessed and unwitnessed collapses
5. Special circumstances regarding SCA associated with cervical spine injuries; commotio cordis; exertional heat stroke; lightning; mass events; and rainy, wet, ice, and metal surfaces
6. Conclusions

## EXECUTIVE SUMMARY

### Emergency Preparedness

- Every school or institution that sponsors athletic activities should have a written and structured EAP.
- The EAP should be developed and coordinated in consultation with local EMS personnel, school public safety officials, on-site first responders, and school administrators.
- The EAP should be specific to each individual athletic venue and encompass emergency communication, personnel, equipment, and transportation to appropriate emergency facilities.
- The EAP should be reviewed and practiced at least annually with certified athletic trainers, team and consulting physi-

cians, athletic training students, school and institutional safety personnel, administrators, and coaches.<sup>6</sup>

- Targeted first responders should receive certified training in CPR and AED use.
- Access to early defibrillation is essential, and a target goal of less than 3 to 5 minutes from the time of collapse to the first shock is strongly recommended.<sup>5,7</sup>
- Review of equipment readiness and the EAP by on-site event personnel for each athletic event is desirable.

### Management of Sudden Cardiac Arrest

- The initial components of SCA management are early activation of EMS, early CPR, early defibrillation, and rapid transition to advanced cardiac life support (ACLS).
- Sudden cardiac arrest should be suspected in any collapsed and unresponsive athlete.
- An AED should be applied as soon as possible on any collapsed and unresponsive athlete for rhythm analysis and defibrillation if indicated.
- Cardiopulmonary resuscitation should be provided while waiting for an AED.
- Interruptions in chest compressions should be minimized and CPR stopped only for rhythm analysis and shock.
- Cardiopulmonary resuscitation should be resumed immediately after the first shock, beginning with chest compressions, with repeat rhythm analysis after every 2 minutes or 5 cycles of CPR, and continued until advanced life support providers take over or the victim starts to move.<sup>7,8</sup>
- Sudden cardiac arrest in athletes can be mistaken for other causes of collapse, and rescuers should be trained to recognize SCA in athletes with special focus on potential barriers to recognizing SCA, including inaccurate rescuer assessment of pulse or respirations, occasional or agonal gasping, and myoclonic jerking or seizure-like activity.
- Young athletes who collapse shortly after being struck in the chest by a firm projectile or by player contact should be suspected of having SCA from commotio cordis.
- Rapid access to the SCA victim should be facilitated for EMS personnel.

## BACKGROUND

### Causes of Sudden Cardiac Death in Young Athletes

The underlying cardiac anomaly in young athletes with SCD is usually a structural cardiac abnormality. Hypertrophic cardiomyopathy and coronary artery anomalies represent approximately 25% and 14% of cases, respectively, in the United States.<sup>1</sup> Commotio cordis is caused by a blunt, nonpenetrating blow to the chest that induces a ventricular arrhythmia in an otherwise structurally normal heart and accounts for approximately 20% of SCD in young athletes.<sup>1</sup> A variety of other structural cardiac anomalies account for most of the remaining causes of SCD in athletes. These include conditions such as myocarditis, arrhythmogenic right ventricular dysplasia, Marfan syndrome, valvular heart disease, dilated cardiomyopathy, and atherosclerotic coronary artery disease. In about 2% of sudden deaths in young athletes, postmortem examination fails to identify a structural cardiac cause of death.<sup>1,9,10</sup> These deaths may be due to inherited arrhythmic syndromes and ion channel disorders such as long QT or short QT syndrome, Brugada syndrome, or familial catecholaminergic polymorphic ventricular tachycardia (VT).<sup>1</sup>

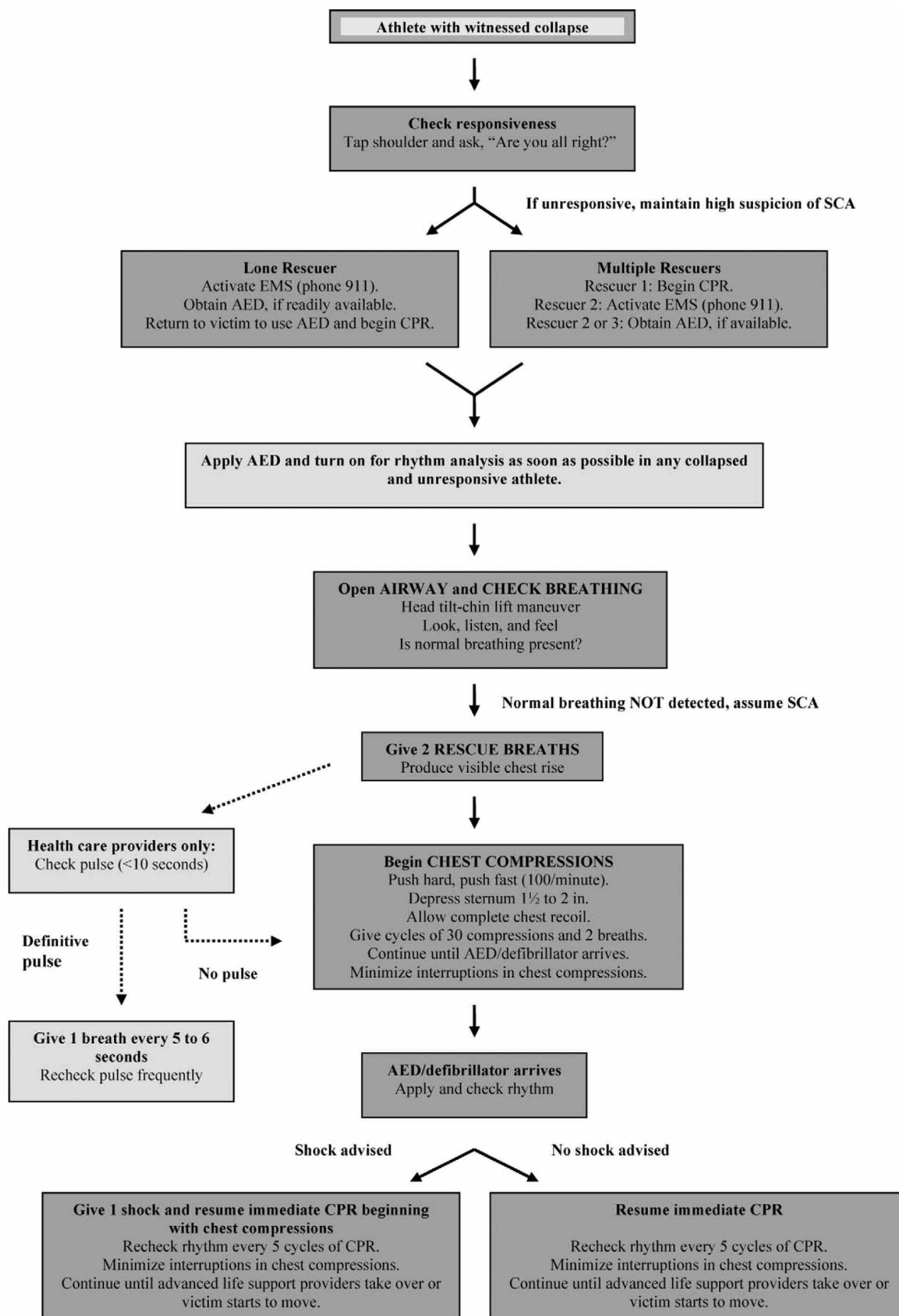


Figure 1. Management of sudden cardiac arrest. SCA indicates sudden cardiac arrest; EMS, emergency medical services; AED, automated external defibrillator; CPR, cardiopulmonary resuscitation.

Vigorous exercise appears to be a trigger for lethal arrhythmias in athletes with occult heart disease.<sup>1</sup> Authors of the best available studies estimate the incidence of SCD in high school athletes to be 1:100 000 to 1:200 000.<sup>2,3</sup> The estimated incidence of SCD in college-aged athletes is slightly higher, ranging from 1:65 000 to 1:69 000.<sup>2,11</sup> However, with no mandatory national reporting or surveillance system, the true incidence of SCA/SCD in athletes is unknown, and prior reports may have underestimated the actual occurrence of SCA/SCD in young athletes. More recently, Maron et al<sup>12</sup> reported on the frequency of sudden death in young competitive athletes and documented approximately 110 deaths per year, or about 1 death every 3 days in the United States.

## Limitations of Cardiovascular Screening

A comprehensive discussion of preparticipation cardiovascular screening in athletes is beyond the scope of this task force. However, recognizing the limitations of current preparticipation screening strategies in detecting potentially lethal cardiac abnormalities in young athletes is critical to understanding the need for emergency preparedness and management protocols to prevent SCD. Healthy-appearing athletes may harbor unsuspected cardiovascular disease with the potential to cause SCD.<sup>3</sup> In approximately 55% to 80% of cases of SCD, the athlete is asymptomatic until the cardiac arrest, with death representing the sentinel event of otherwise silent cardiovascular disease.<sup>3,13</sup> The task force supports the AHA recommendations for cardiovascular screening in athletes,<sup>14</sup> as well as the use of a standardized questionnaire to guide examiners, such as the widely accepted monograph *Preparticipation Physical Evaluation*, 3rd edition.<sup>15</sup> At this time, cardiovascular screening of asymptomatic athletes with electrocardiography or echocardiography is not recommended by the AHA in the United States because of the poor sensitivity, high false-positive rate, poor cost-effectiveness, and total cost of implementation.<sup>14,16</sup> Detection of premonitory cardiovascular symptoms such as a history of exertional syncope or chest pain should be improved by the use of standardized history forms and requires a careful and thorough cardiovascular evaluation to exclude underlying heart disease. Improved education for athletes, coaches, and health care professionals is needed regarding symptoms that may precede SCA.

## Resuscitation Pathophysiology

About 40% of out-of-hospital cardiac arrest victims demonstrate ventricular fibrillation (VF) on first rhythm analysis.<sup>17</sup> Ventricular fibrillation is characterized by chaotic rapid depolarizations and repolarizations, which cause the heart muscle to quiver and lose its ability to pump blood effectively. It is likely that a larger percentage of victims has VF or rapid VT at the time of collapse, but the rhythm has already deteriorated to asystole before the first rhythm analysis. The probability of successful defibrillation for VF SCA diminishes rapidly over time, with survival rates declining 7% to 10% per minute for every minute that defibrillation is delayed.<sup>18,19</sup> Defibrillation through deployment of electric energy terminates VF and allows the normal cardiac pacemakers to resume firing and produce an effective rhythm if the heart tissue is still viable. Survival after SCA is unlikely once VF has deteriorated to asystole.

Cardiopulmonary resuscitation is important both before and after defibrillation; it provides a small but critical amount of blood flow to the heart and brain and increases the likelihood that defibrillation will restore a normal rhythm in time to prevent neurologic damage. Chest compressions create blood flow by increasing intrathoracic pressure and directly compressing the heart. When bystander CPR is initiated, survival declines only 3% to 4% per minute for every minute defibrillation is delayed.<sup>18,20</sup> Thus, CPR can greatly improve survival from witnessed SCA for any given time interval to defibrillation. Resuming CPR immediately after shock delivery is also critical. Many victims are in pulseless electric activity or asystole for several minutes after defibrillation, and CPR is needed to provide perfusion.<sup>21–23</sup> Unfortunately, bystander CPR is initiated in less than one third of cases of witnessed SCA,<sup>24,25</sup> and, if initiated, more than 40% of chest compressions are of insufficient quality.<sup>26</sup> These deficiencies illustrate the tremendous need for increased public education and training in CPR.

Automated external defibrillators are computerized devices that analyze a victim's rhythm, determine if a shock is needed, charge to an appropriate shock dose, and use audio and visual instructions to guide the rescuer. These devices are easy to use and extremely accurate in recommending a shock only when VF or rapid VT is present.<sup>27</sup> In one study, AEDs were safely and successfully operated by untrained sixth graders almost as quickly as trained paramedics in a simulated resuscitation, with only a 23-second difference in mean time to defibrillation.<sup>28</sup>

Weisfeldt and Becker<sup>29</sup> described a 3-phase model of resuscitation to account for the changes in cardiac arrest pathophysiology that occur with time. The model emphasizes phase-specific treatments based on the time interval from collapse and includes the following: (1) the electric phase, which extends from the time of cardiac arrest to approximately 4 minutes after cardiac arrest; (2) the circulatory phase, from approximately 4 minutes to 10 minutes after cardiac arrest; and (3) the metabolic phase, extending beyond 10 minutes from cardiac arrest.<sup>29</sup> The most critical intervention during the electric phase is early defibrillation, and CPR should be provided until an AED or a manual defibrillator is available.

After 4 to 5 minutes of untreated VF (circulatory phase), some authors suggest that outcomes may be better if shock delivery is preceded by a brief period of CPR to deliver blood to the heart and brain. In animals, outcomes improved for prolonged untreated VF when CPR was initiated before defibrillation compared with immediate defibrillation.<sup>30,31</sup> In some clinical studies,<sup>32,33</sup> survival was improved when CPR was initiated before defibrillation in cases of untreated VF lasting longer than 3 to 5 minutes. Thus, as the duration of cardiac arrest increases, initial chest compressions and oxygen delivery to vital tissues may take priority over defibrillation in some patients, with delay of shock delivery until 1 to 2 minutes of CPR has been completed.<sup>29,32,33</sup>

After approximately 10 minutes of cardiac arrest (metabolic phase), additional tissue injury can occur from global ischemia and with resumption of blood flow from reperfusion injury. Cellular protection from reperfusion injury may be augmented by hypothermia-mediated therapies to attenuate the rapid oxidant burst caused by restoring oxygen and substrates to ischemic tissues, although these therapies are not widely available.<sup>34</sup>



## Survival after Sudden Cardiac Arrest

The single greatest factor affecting survival after out-of-hospital cardiac arrest is the time interval from arrest to defibrillation.<sup>19</sup> Survival after out-of-hospital cardiac arrest has been greatly improved by lay rescuer and public access defibrillation programs designed to shorten the time interval from SCA to shock delivery. These programs train lay rescuers and first responders in CPR and AED use and place AEDs in high-risk public locations for SCA. Studies of rapid defibrillation using AEDs with nontraditional first responders and trained or untrained laypersons in high-risk locations such as casinos, airlines, and airports have demonstrated survival rates from 41% to 74% if bystander CPR is provided and defibrillation occurs within 3 to 5 minutes of collapse.<sup>11,35–43</sup> Key elements to the success of these programs include training of motivated responders in CPR and AED use, a structured and practiced response, and short response times.

Although SCA is a rare but catastrophic event in young athletes, it is more common in an older population, with an estimated annual frequency of 1 in 1000 persons aged 35 years or older in the United States.<sup>19</sup> The presence of AEDs in schools and institutions provides a means of early defibrillation, not only for young athletes but also for other individuals on campus who may experience an unexpected cardiac arrest. Jones et al<sup>44</sup> found a 2.1% annual probability of SCA on high school campuses, mainly due to SCA among older school employees, spectators, and visitors on campus. At National Collegiate Athletic Association Division I universities, Drezner et al<sup>11</sup> found that older nonstudents, such as spectators, coaches, and officials, accounted for 77% of SCA cases at collegiate sporting venues and that placement of AEDs at these venues provided a significant survival benefit for older nonstudents, with a 54% overall immediate resuscitation rate.

Limited research is available regarding the survival rate in young athletes after SCA. Initially, authors<sup>11,45</sup> investigating AED use in the college athletic setting did not identify a survival benefit in a small number of collegiate athletes with SCA. Drezner and Rogers<sup>46</sup> later investigated the timing and details of resuscitation in 9 collegiate athletes with SCA. All 9 athletes had a witnessed arrest, and most received immediate assessment by an athletic trainer skilled in basic life support and CPR. Seven athletes received defibrillation, with an average time from cardiac arrest to defibrillation of 3.1 minutes. Despite a witnessed collapse, timely CPR, and prompt defibrillation in most cases, only 1 of 9 (11%) athletes in this cohort survived—an unexpected finding given the young age, otherwise good health and physical conditioning of the athletes, and early reported defibrillation.<sup>46</sup> Other groups<sup>47,48</sup> have also found the survival rate after SCA in young athletes to be lower than expected. Maron et al<sup>47</sup> analyzed 128 cases from the United States Commotio Cordis Registry and found an overall survival rate of 16%. Cardiopulmonary resuscitation was performed in 106 cases and defibrillation in 41 cases, with 19 of 41 (46%) of the individuals who received defibrillation surviving.<sup>47</sup> Successful resuscitations using AEDs have been reported in the public media and in case reports<sup>49,50</sup> and demonstrate the lifesaving potential of public access defibrillation on the athletic field. Overall, the available studies on SCA in young athletes raise concern regarding the low survival rate and highlight the need for improved and more uniform resuscitation strategies for SCA in young athletes.

## Factors Affecting Survival in Young Athletes

Several factors may contribute to a lower resuscitation rate in young athletes. Structural heart disease is consistently found in most cases of SCD in young athletes. Ventricular arrhythmias in the presence of structural heart disease, especially cardiomyopathies, may be more resistant to even short delays in defibrillation than SCA in the setting of a structurally normal heart. Berger et al<sup>51</sup> documented 18 episodes of unexpected SCA in previously asymptomatic children and adolescents aged 12 to 25 years in Wisconsin from 1999 to 2003. Survival was poor in cases of structural heart disease: 1 of 9 survived with hypertrophic cardiomyopathy, 1 of 3 survived with an anomalous origin of the left coronary artery, and 0 of 1 survived with arrhythmogenic right ventricular dysplasia. In contrast, 5 of 6 patients with long QT syndrome and no structural heart disease survived.<sup>51</sup>

In patients with hypertrophic cardiomyopathy, immediate defibrillation within 10 seconds using implantable cardioverter defibrillators is almost always effective in terminating potentially lethal ventricular arrhythmias.<sup>52,53</sup> However, in athletes with hypertrophic cardiomyopathy, even a brief delay in defibrillation may cause a steep decline in survival.<sup>46</sup> In commotio cordis or primary electric disturbances, survival rates more closely follow the traditional decline of 7% to 10% per minute for every minute defibrillation is delayed.<sup>54</sup> In an animal model using juvenile swine, Link et al<sup>54</sup> demonstrated that successful resuscitation after commotio cordis was possible and highly dependent on the time interval to defibrillation. After induction of VF via blunt chest impact using a baseball, defibrillation was performed after 1, 2, 4, and 6 minutes, with survival rates of 100%, 92%, 46%, and 25%, respectively.<sup>54</sup>

Other factors may also contribute to the apparently lower survival rate in young athletes after SCA. The overall incidence of SCA in athletes is relatively rare, and delayed recognition of cardiac arrest by first responders may lead to delay in initiating CPR and defibrillation. Rescuers may mistake agonal or occasional gasping for normal breathing or falsely identify the presence of a pulse. Sudden cardiac arrest may also be misdiagnosed as a seizure because of the presence of myoclonic activity after collapse. Thus, a high suspicion of SCA must be maintained for any collapsed and unresponsive athlete. Other potential factors affecting survival in young athletes include the duration and intensity of exercise before arrest, higher catecholamine levels produced during exercise, potential for oxygen debt and ischemia from exercise, metabolic and physiologic adaptations during exercise, and vascular changes such as decreased systemic vascular resistance.

Key changes from the 2005 AHA guidelines for CPR and ECC may positively affect survival in young athletes.<sup>4</sup> The new guidelines recommend that one attempted defibrillation be followed by immediate CPR, beginning with chest compressions.<sup>7</sup> The initiation of immediate CPR after defibrillation creates blood flow until the heart can generate adequate contractions for perfusion. This is particularly important when defibrillation is followed by pulseless electric activity or recurrent VF requiring multiple shocks. Of 9 cases reported in collegiate athletes, pulseless electric activity followed defibrillation in 2 patients and multiple shocks were deployed in 4.<sup>46</sup> Thus, incorporating changes in CPR protocol and assisting perfusion in the early moments after defibrillation may have a



significant effect on survival by limiting interruptions in blood flow and the need for repeat defibrillation.

## EMERGENCY PREPAREDNESS

### The “Chain of Survival”

Public access to defibrillators and first-responder AED programs improve survival from SCA by increasing the likelihood that SCA victims will receive bystander CPR and early defibrillation. These programs require an organized and practiced response with rescuers trained and equipped to recognize SCA, activate the EMS system, provide CPR, and use an AED.<sup>36</sup> The AHA describes 4 links in a “chain of survival” to emphasize the time-sensitive interventions for victims of SCA<sup>19</sup>:

- Early recognition of the emergency and activation of the EMS or local emergency response system: “phone 911”
- Early bystander CPR: immediate CPR can double or triple the victim’s chance of survival from VF SCA<sup>18,20</sup>
- Early delivery of a shock with a defibrillator: CPR plus defibrillation within 3 to 5 minutes of collapse can produce survival rates as high as 49% to 75%<sup>35,37–39,42,55</sup>
- Early advanced life support followed by postresuscitation care delivered by health care providers

### Establishing an Emergency Action Plan

Every institution or organization that sponsors athletic activities should have a written EAP.<sup>6</sup> The EAP should be specific to each athletic venue and encompass emergency communication, personnel, equipment, and transportation. Core elements to an effective EAP include the following: (1) establishing an efficient communication system, (2) training of likely first responders in CPR and AED use, (3) acquiring the necessary emergency equipment, (4) providing a coordinated and practiced response plan, and (5) ensuring access to early defibrillation (see Table for an EAP checklist). The plan should identify the person and/or group responsible for documentation of personnel training, equipment maintenance, actions taken during the emergency, and evaluation of the emergency response.<sup>6</sup>

The EAP should be developed by school or institutional personnel in consultation with local EMS personnel, school public safety officials, on-site first responders, and school administrators. It is important to designate an EAP coordinator, usually an athletic trainer, team physician, nurse, or sports administrator. The EAP should be reviewed at least annually with athletic trainers, team and consulting physicians, athletic training students, school and institutional safety personnel, administrators, and coaches.<sup>6</sup> The EAP should be coordinated with the local EMS agency and integrated into the local EMS system. The local EMS agency is encouraged to conduct a “preincident” survey to identify any problems or poorly accessible areas for EMS personnel.<sup>5</sup>

The National Collegiate Athletic Association recommends that all institution-sponsored collegiate practices or competitions, as well as out-of-season practices and skills sessions, have an EAP. These plans should include the presence of a person qualified to deliver emergency care; planned access to early defibrillation; and planned access, communication, and transport to a medical facility.<sup>56</sup>

## Access to Early Defibrillation

Access to early defibrillation is critical in the management of SCA. In developing an EAP, several time-sensitive intervals must be considered to increase the probability of a successful resuscitation for an SCA victim: the time from collapse to EMS activation, the time from collapse to initiation of CPR, the time from collapse to delivery of the first shock, and the time from collapse to arrival of EMS personnel at the victim’s side. The EAP should target a collapse-to-EMS call time and CPR initiation of less than 1 minute.<sup>5,19</sup> A second target goal of less than 3 to 5 minutes from time of collapse to first shock is strongly recommended.

The AHA recommends implementation of an AED program in any school that meets one of the following criteria: (1) the frequency of cardiac arrest is such that there is a reasonable probability of AED use within 5 years of rescuer training and AED placement; (2) there are children attending school or adults working at the school who are thought to be at high risk for SCA (eg, children with congenital heart disease); or (3) an EMS call-to-shock interval of less than 5 minutes cannot be reliably achieved with a conventional EMS system, and a collapse-to-shock interval of less than 5 minutes can be reliably achieved (in more than 90% of cases) by training and equipping laypersons to function as first responders by recognizing SCA, activating the EMS system, starting CPR, and using an AED.<sup>5,19</sup>

Schools and institutions sponsoring athletic programs must determine if this target time interval of less than 5 minutes from collapse to defibrillation can be reliably achieved with the conventional EMS system or if an AED program is required to achieve early defibrillation. Studies suggest that, for most EMS systems, the time interval between activating the EMS and arrival of EMS personnel at the victim’s side is usually more than 5 minutes (mean = 6.1 minutes).<sup>57</sup> In some communities, the time interval from EMS call to EMS arrival may be 7 to 8 minutes or longer.<sup>43,58</sup> Thus, achieving early defibrillation after SCA in athletes is largely dependent on the prompt availability of AEDs for responders. In high school and college athletic programs, coaches, officials, athletic trainers, and other sports medicine professionals are in a unique position to act as first responders to SCA during organized training and competition. Training in CPR and on-site AED programs are likely to be the only means of achieving early defibrillation and improving survival from SCA in athletes.

## Emergency Communication

A rapid system for communication must be in place linking all athletic facilities, practice fields, and other parts of the campus to the EMS system. When bystanders recognize an emergency and activate the EMS system, they ensure that basic and advanced life support providers are dispatched to the site of the emergency. The time required for EMS response to each sporting venue should be estimated, and a plan must be in place to efficiently direct EMS personnel to the location.<sup>5</sup> The communication network can be developed through existing telephones, cellular telephones, walkie-talkies, alarms, or an intercom system that links a rescuer directly to the EMS or to a central location responsible for contacting the EMS and activating on-site responders. Establishing an accessible communication system will prevent critical delays caused by a rescuer running from a distant athletic facility or practice field

## Emergency Action Plan Checklist\*

The following elements are recommended in the development of a comprehensive emergency action plan (EAP) for sudden cardiac arrest (SCA) in athletics. Actual requirements and implementation may vary depending on the location, school, or institution.

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### I. Development of an Emergency Action Plan

- ☐ Establish a written EAP for each individual athletic venue.
- ☐ Coordinate the EAP with the local EMS agency, campus public safety officials, on-site first responders, administrators, athletic trainers, school nurses, and team and consulting physicians.
- ☐ Integrate the EAP into the local EMS response.
- ☐ Determine the venue-specific access to early defibrillation (<3 to 5 minutes from collapse to first shock recommended).

### II. Emergency Communication

- ☐ Establish an efficient communication system to activate EMS at each athletic venue.
- ☐ Establish a communication system to alert on-site responders to the emergency and its location.
- ☐ Post the EAP at every venue and near telephones, including the role of the first responder, a listing of emergency numbers, and street address and directions to guide the EMS personnel.

### III. Emergency Personnel

- ☐ Designate an EAP coordinator.
- ☐ Identify who will be responsible and trained to respond to a SCA (likely first responders include athletic trainers, coaches, school nurses, and team physicians).
- ☐ Train targeted responders in CPR and AED use.
- ☐ Determine who is responsible for personnel training and establish a means of documentation.
- ☐ Identify the medical coordinator for on-site AED programs.

### IV. Emergency Equipment

- ☐ Use on-site or centrally located AED(s) if the collapse-to-shock time interval for conventional EMS is estimated to be >5 minutes.
- ☐ Notify EMS dispatch centers and agencies of the specific type of AED and the exact location of the AED on school grounds.
- ☐ Acquire pocket mask or barrier-shield device for rescue breathing.
- ☐ Acquire AED supplies (scissors, razor, and towel), and consider an extra set of AED pads.
- ☐ Consider bag-valve masks, oxygen delivery systems, oral and nasopharyngeal airways, and advanced airways (eg, endotracheal tube, Combitube, or laryngeal mask airway).
- ☐ Consider emergency cardiac medications (eg, aspirin, nitroglycerin).
- ☐ Determine who is responsible for checking equipment readiness and how often and establish a means of documentation.

### V. Emergency Transportation

- ☐ Determine transportation route for ambulances to enter and exit each venue.
- ☐ Facilitate access to SCA victim for arriving EMS personnel.
- ☐ Consider on-site ambulance coverage for high-risk events.
- ☐ Identify the receiving medical facility equipped in advanced cardiac care.
- ☐ Ensure that medical coverage is still provided at the athletic event if on-site medical staff accompany the athlete to the hospital.

### VI. Practice and Review of Emergency Action Plan

- ☐ Rehearse the EAP at least annually with athletic trainers, athletic training students, team and consulting physicians, school nurses, coaches, campus public safety officials, and other targeted responders.
- ☐ Consider mock SCA scenarios.
- ☐ Establish an evaluation system for the EAP rehearsal, and modify the EAP if needed.

### VII. Postevent Catastrophic Incident Guidelines

- ☐ Establish a contact list of individuals to be notified in case of a catastrophic event.
- ☐ Determine the procedures for release of information, aftercare services, and the postevent evaluation process.
- ☐ Identify local crisis services and counselors.
- ☐ Consider pre-established incident report forms to be completed by all responders and the method for system improvement.

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\*EMS indicates emergency medical services; CPR, cardiopulmonary resuscitation; and AED, automated external defibrillator.

to activate the EMS system. The communications system should be checked before each practice or competition to ensure proper working order, and a back-up communication plan should be in effect in case the primary communication system fails.

The EAP should be posted at every sporting venue and near appropriate telephones, with the role of the first responder clearly demarcated. A listing of emergency numbers should be available, as well as the street address of the venue and specific directions (eg, cross streets, landmarks) to guide EMS personnel. When activating the EMS system (calling 911), the caller should alert the EMS to the number and condition of persons injured, if an SCA is suspected, and the first aid treatment rendered. Involving representatives of the EMS system in the initial communications planning before any incidents will improve the onsite transfer of care once EMS personnel arrive on the scene.

## Emergency Personnel

The first person to respond to a medical emergency on the field of play will vary widely and may be a coach, official, student, teammate, teacher, school nurse, athletic trainer, physician, or emergency medical technician. All potential rescuers should be familiar and, ideally, trained with the EAP to ensure an effective and coordinated response to an emergency situation. Each institution or organization with a formal athletic program needs to identify who will be responsible and trained to respond to an SCA. The National Collegiate Athletic Association recommends that all athletics personnel associated with practices, competitions, skills instruction, and strength and conditioning be certified in CPR, first aid, and the prevention of disease transmission.<sup>56</sup> For secondary schools, the AHA recommends training of the school nurse and physician, athletic trainer, and several faculty members in the provision of first aid and CPR and that a sufficient number of faculty, staff, and/or students be trained to ensure that a trained rescuer can respond to an SCA within 90 seconds.<sup>5</sup> Because an athletic trainer, physician, or school nurse is not universally present at all extracurricular sporting activities, coaches for every team should receive certified training in CPR and AED use to ensure the presence of a trained rescuer.

## Emergency Equipment

All necessary emergency equipment should be at the site or quickly accessible, and personnel must be trained in advance to use it properly. Resuscitation equipment should be placed in a central location that is highly visible and near a telephone or other means of activating the EMS system. All school staff should be instructed on the location of emergency equipment. For large schools or those with distant or multiple athletic facilities, duplicate equipment may be needed. Emergency equipment should not be placed in a locked box, cabinet, or room, which could delay emergency care. Mounted cabinets with audible alarms that sound when the cabinet door is opened may decrease the theft or vandalism risk.<sup>5</sup>

Basic resuscitation equipment for management of SCA should include a pocket mask or barrier-shield device for rescue breathing, an AED for early defibrillation, and AED application supplies (heavy-duty scissors to remove clothing and expose the chest, a towel to dry the chest, and a razor to shave chest hair). Aluminum chlorhydrate (antiperspirant) spray may

help the AED leads stick to sweaty skin, and an extra set of AED pads should be considered in case of misapplication or inadvertent damage.

For high schools and colleges that have physicians and ACLS-certified responders on-site, the acquisition of advanced resuscitation equipment for the management of SCA should be considered based on the skills of the designated responders. Advanced resuscitation equipment may include bag-valve masks, oxygen delivery systems, oral and nasopharyngeal airways, advanced airways (eg, endotracheal tube, Combitube [Tyco Healthcare Nellcor, Pleasanton, CA], or laryngeal mask airway), and emergency cardiac medications. The ACLS-certified personnel should have nitroglycerin and aspirin available on-site to use for chest pain without cardiac arrest. The equipment and medications should be assembled in a code bag and stored in an easily accessible central location or at each athletic venue.

If an AED program is implemented, it should be part of the written EAP. The EMS centers should be notified of the specific type of AED and the exact location of the AED on school grounds. If a rescuer is unfamiliar with the school or where an AED is located, he or she can receive instructions from the EMS dispatcher to find and use the AED.<sup>5</sup> All AED programs should include medical or health care provider oversight, appropriate training of anticipated rescuers in CPR and AED use, coordination with the EMS system, appropriate device maintenance, and an ongoing quality improvement program.<sup>5</sup>

If possible, emergency information about the student-athletes, including relevant medical history and contact information, should be accessible to medical personnel at home sporting events and while traveling in case of an emergency.

## Emergency Transportation

The EAP should delineate the life support transportation an athlete with SCA will access. In life-threatening emergencies, an athlete should be transported by the EMS personnel to the most appropriate receiving facility that is staffed and equipped to deliver optimal emergency care. Emphasis should be placed on having an ambulance on-site at high-risk events. The EMS response time should be factored in when determining on-site ambulance coverage. Consideration should be given to the level of transportation service available (eg, basic life support, advanced life support) and the equipment and training level of the personnel who staff the ambulance.<sup>6</sup> If an ambulance is on-site, a location should be designated that allows rapid access to enter and exit the venue. A dedicated staff person should be assigned to each event and be familiar with the directions and access points for arriving EMS personnel to specific athletic facilities on campus. Each site should post written directions to read to the EMS response dispatcher. If air-medical transport may be needed, the global positioning satellite coordinates should also be listed.

## Practice and Review of the Emergency Action Plan

The EAP should be reviewed and practiced at least annually with athletic trainers, team and consulting physicians, athletic training students, school and institutional safety personnel, administrators, coaches, and other designated first responders.<sup>6</sup> More frequent practice sessions will improve the effectiveness, efficiency, and organization of the response team, and any modifications to the EAP based on practice trials should be



documented.<sup>59</sup> A mock SCA scenario can be organized using actors or manikins to simulate SCA victims. Evaluation of each emergency response rehearsal should document the time from collapse to EMS activation, the time from collapse to initiation of CPR, the time from collapse to delivery of first shock if an AED is available, and the time from collapse to arrival of EMS personnel at the victim's side.<sup>5</sup>

### Postevent Catastrophic Incident Guidelines

The EAP should include a postevent plan outlining the procedures for release of information; aftercare services for responders, teammates, coaches, and families; and the postevent evaluation process. A list of administrative and legal personnel from the school or institution to be contacted after a catastrophic event should be readily accessible in the EAP, and the methods for data collection, reporting, and incident assessment and review should be defined in the plan. Local crisis services and counselors to assist students, teammates, families, and rescuers after a catastrophic event should be defined and available. The postevent evaluation process is critical both to document the details of the event and to allow system improvement. Pre-established incident report forms to be completed by all responders should be considered to facilitate a summary report with recommendations for site management and modifications to the existing EAP if needed. Feedback, particularly of a positive nature, should also be provided to responders.

### Emergency Preparedness: Where Are We Now?

Studies demonstrating that AEDs placed at public locations can substantially improve survival from SCA have accelerated a growing national trend to broadly implement AED programs at public sporting venues and selected athletic facilities. In 2003, 91% of National Collegiate Athletic Association Division I institutions already had AEDs, with a median of 4 (range = 1 to 30) at each of these institutions.<sup>11</sup> The most common location for the AEDs was the athletic training room (82%), followed by the basketball arena (43%), campus police station (40%), football stadium (27%), baseball or softball field (21%), and recreation or fitness facility (21%).<sup>11</sup> A range of 25% to 54% of high schools had at least one AED on school grounds.<sup>44,60,61</sup> Common locations for AEDs in high schools include the school athletic training room, basketball facility or gymnasium, nurse's office, and main lobby.<sup>61,62</sup>

In the university setting, resources to purchase AEDs have largely come from the athletic department budget. In contrast, financial resources at high schools are more limited, and AED acquisition has been primarily funded through donations. In Washington State, 60% of high schools with AEDs acquired them through donated funds, and only 38% were purchased by the school, school district, or athletic department.<sup>61</sup> In the greater Boston area, after a single AED was donated to 35 schools, 25 schools purchased additional AEDs using a combination of donated funds (21), grants (11), and school budget funds (8).<sup>62</sup>

School nurses, athletic trainers, teachers, and coaches may be called on to provide emergency care during school hours and extracurricular sporting activities. However, emergency training for many of these potential first responders varies widely. A survey of school nurses in New Mexico documented that few school nurses and staff had any emergency training.<sup>63</sup>

In 3 midwestern states, one third of teachers surveyed had no first aid training, and 40% had never completed a course in CPR.<sup>64</sup> In Washington State high schools with AEDs, 78% of coaches, 72% of administrators, 70% of school nurses, and 48% of teachers received formal AED training.<sup>61</sup>

Although more schools are placing AEDs on campus grounds, significant deficiencies in emergency planning and coordination still exist. In Washington State, only 25% of high schools coordinated the implementation of AEDs with any outside medical agency, and only 6% of schools coordinated with the local EMS system.<sup>61</sup> In contrast, studies involving AED implementation as part of a more comprehensive emergency plan have demonstrated more success. In the greater Boston area, 35 high schools were given a single donated AED and educated to develop a training protocol for appropriate staff and to assess the need for purchase of additional AEDs.<sup>62</sup> In this study, 90% of schools trained their faculty, 76% trained their staff and custodial workers, 71% trained their athletic trainers, and 48% trained some or all of their student body in AED use.<sup>62</sup> Over a 2-year study period, an AED was successfully used twice for SCA in a football referee and a teacher. Every school participating in the study considered participation in the AED program to be worthwhile.<sup>62</sup> Similarly, in Wisconsin, Project ADAM has assisted 143 of 400 public high schools in implementing an AED program as part of a comprehensive EAP. The goals of Project ADAM include educating faculty, staff, parents, students, and health care professionals about SCA in children and adolescents and advocating for teaching CPR with AED instruction to all high school students before graduation.<sup>51</sup>

Legislation to require AEDs in schools is also growing but is not uniformly funded. New York and Illinois have already passed legislative mandates for AEDs in public schools, and California, Delaware, Florida, Georgia, Maine, Massachusetts, New Jersey, Nevada, Pennsylvania, Rhode Island, and Virginia have pending legislation.<sup>65</sup> However, unfunded mandates to establish AED programs in schools often lack the necessary emergency planning, coordination, and training that are essential to a successful program.

### Obstacles to Implementing Automated External Defibrillators

Although the prevalence of AEDs in schools and at public athletic venues is increasing, budgetary constraints remain an obstacle to initiating public access defibrillation programs. Policymakers must determine which sites warrant an AED and which must go without. Although uncertainty on where to place the AED (57%) and medical-legal concerns (48%) were also reported as common obstacles, National Collegiate Athletic Association Division I head athletic trainers at institutions without AEDs cited financial resources (70%) as the primary obstacle to acquiring AEDs.<sup>11</sup> In the high school setting, financial resources are the critical barrier to implementing an AED program. In Washington State, 65% of high schools without AEDs identified monetary resources as the main obstacle to acquiring AEDs.<sup>61</sup> In the greater Boston area, 93% of schools given a single AED reported lack of funding as the reason for not purchasing additional AEDs.<sup>62</sup>

The task force recognizes that budgets for many schools are already challenged, and more research is needed to explore the development of funding programs to assist schools with limited resources. Until the cost of AEDs further declines or gov-



ernment funding is allocated toward emergency preparedness in schools, administrators and those responsible for emergency planning must work within their school districts to elicit financial support from the local community, parenting and fundraising groups, and applicable state and federal grants to fund AED programs. However, the goal is not just to acquire the AED but to do so as part of a comprehensive educational and emergency action plan. As demonstrated by Project ADAM and the greater Boston area program, donations can fund both the equipment and educational materials for school administrators and lead to the development of a successful program in participating schools.<sup>51,62</sup>

The current cost (in 2006) of purchasing a single AED is approximately \$1500, and in many communities, local EMS personnel will provide emergency planning consultation at no cost. The additional cost of CPR and AED training for targeted responders must also be considered. However, with the commitment and dedication of school administrators, parents, local businesses, and health care leaders, implementation of an AED program in schools is an obtainable goal.

## MANAGEMENT OF SUDDEN CARDIAC ARREST

### 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

In December 2005, the AHA released updated guidelines on CPR and ECC.<sup>4</sup> The guidelines are based on the evidence evaluation from the 2005 International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations.<sup>66</sup> The AHA adult CPR guidelines apply to any child older than 8 years.<sup>67</sup> Thus, for the purposes of this statement, the protocols discussed are intended for youth and adult athletes greater than 8 years old. The most significant change in these guidelines is a stronger emphasis on chest compressions, increasing the number of chest compressions per minute and reducing the interruptions in chest compressions during CPR. Key changes to the guidelines are listed in the following:

- Elimination of lay rescuer assessment of circulation
- Recommendation of a universal compression-to-ventilation ratio of 30:2 for single rescuers and for all SCA victims in this age group
- Chest compressions (“push hard, push fast”) should be at a rate of 100 compressions per minute, allowing complete chest recoil and minimizing interruptions in chest compressions
- CPR should resume immediately after initial shock delivery, beginning with chest compressions
- Rescuers should not check the rhythm or pulse after shock delivery until 5 cycles (or about 2 minutes) of CPR have been performed
- Recommendations that EMS providers consider 5 cycles (or about 2 minutes) of CPR before defibrillation for unwitnessed arrest, particularly if the suspected time from collapse to arrival at the scene is more than 4 to 5 minutes

The new recommendations call for only one shock, followed immediately by chest compressions, and represent a major change from traditional treatment protocols involving a sequence of 3 stacked shocks in the treatment of VF and rapid VT. This change is based on the high success rate of a single

defibrillation, with first-shock efficacy for VF by current biphasic defibrillators reportedly higher than 90%.<sup>8</sup> In addition, if the first shock fails, CPR may improve oxygen and substrate delivery to the myocardium, making subsequent shocks more likely to be successful. Interruptions in chest compressions for rhythm analysis are associated with lower survival rates and a decreased probability of conversion of VF to another rhythm.<sup>68,69</sup>

Rhythm analysis for a 3-shock sequence in older AEDs results in delays up to 37 seconds between delivery of the first shock and delivery of the first postshock chest compression.<sup>70</sup> The new AHA guidelines necessitate reprogramming of all previously purchased AEDs, as they are programmed with older “stacked shocks” protocols. Institutions that have AEDs with older protocols should contact their vendors to reprogram the AED. If possible, AED units should also comply with the new AHA recommendations for first shock energy levels, either 360 J for monophasic waveforms or the individual manufacturer’s recommendations for biphasic waveforms.<sup>8</sup>

## The Collapsed Athlete

Fortunately, most athletes who collapse during or after exercise will not be in SCA. The differential diagnosis of non-traumatic exercise-related syncope includes but is not limited to SCA, exertional heat stroke, heat exhaustion, hyponatremia, hypoglycemia, exercise-associated collapse, neurocardiogenic syncope, seizures, pulmonary embolus, cardiac arrhythmias, valvular disorders, coronary artery disease, cardiomyopathies, ion channel disorders, and other structural cardiac diseases. A collapsed athlete who is also unresponsive should be treated as a potential cardiac arrest until either spontaneous breathing and a pulse are documented or the cardiac rhythm is analyzed. If SCA is ruled out but the athlete remains unresponsive, further immediate evaluation to determine the cause of collapse is needed, as other emergent medical interventions may still be required.

Exercise-related syncope involves a transient loss of consciousness and postural tone during or immediately after exercise. Exercise-associated collapse describes athletes who are unable to stand or walk unaided as a result of lightheadedness, dizziness, or syncope immediately after exercise.<sup>71</sup> Exertional syncope without SCA in young adults is usually benign but always requires investigation because it can be an indication of a more serious cardiac disorder and the only symptom that precedes SCD.<sup>3,13</sup> Syncope that occurs during exercise tends to be more ominous than syncope occurring after exercise.<sup>72</sup> Young and otherwise healthy adults who collapse while exercising have a greater probability of an organic cardiac abnormality, such as hypertrophic cardiomyopathy or anomalous coronary artery origin, than do athletes with postexertional or nonexertional syncope.<sup>3,13</sup> The investigation of exercise-related syncope should specifically exclude known pathologic diagnoses before a complete return to activity is permitted.

## Recognition of Sudden Cardiac Arrest

Prompt identification of SCA is critical in the management of this life-threatening emergency. Any collapsed athlete who is unresponsive requires an immediate assessment for SCA. Although SCA is relatively uncommon in the athletic setting, on-site responders must maintain a high index of suspicion, as unrecognized SCA in a collapsed athlete causes critical delays

in CPR and defibrillation. Resuscitation is often delayed because the victim is reported to have signs of life.<sup>73</sup> Sudden cardiac arrest can be misdiagnosed as a seizure in the form of involuntary myoclonic jerks; seizure-like activity is present in approximately 20% of patients with cardiogenic collapse.<sup>74</sup> Seizure-like activity has also been reported in 3 of 10 athletes with SCA.<sup>75</sup> To avoid life-threatening delays in resuscitation, brief seizure-like activity should be assumed to be due to SCA and initial management steps for SCA taken immediately until a noncardiac cause of the collapse is clearly determined.

Other barriers to recognizing SCA in athletes include inaccurate rescuer assessment of pulse or respirations. Occasional or agonal gasping can occur in the first minutes after SCA and is often misinterpreted as normal breathing, especially by lay responders.<sup>76</sup> Occasional gasping does not represent adequate breathing and, if present, should not prevent rescuers from initiating CPR. Assessment of signs of circulation and the presence of a pulse by lay rescuers and health care professionals can also be inaccurate. Lay rescuers fail to recognize the absence of a pulse in 10% of pulseless victims and fail to detect a pulse in 40% of victims with a pulse.<sup>7,77</sup> The updated AHA CPR guidelines eliminate lay rescuer assessment of pulse and recommend that cardiac arrest be assumed if the unresponsive victim does not demonstrate normal breathing.<sup>4</sup> Health care providers may also have difficulty accurately determining if a pulse is present or take too long in their assessment. Health care providers should take no longer than 10 seconds to check for a pulse and should proceed with chest compressions if a pulse is not definitively detected.<sup>7</sup> Sports medicine professionals and other potential first responders to an SCA in athletes must understand these potential obstacles to recognizing SCA, as inaccurate initial assessment of SCA results in critical delays or even failure to activate the EMS system, initiate CPR, and provide early defibrillation.

### Management of Sudden Cardiac Arrest: Witnessed Collapse

Sudden cardiac arrest in the athletic setting is likely to be witnessed by a bystander, coach, official, teammate, or athletic trainer. When more than one person is present at the scene, several management steps can be taken simultaneously (Figure). Management of a collapsed athlete begins with an initial assessment of responsiveness. The rescuer should check the victim for a response by tapping the victim on the shoulder and asking, “Are you all right?”<sup>7</sup> If the athlete is unresponsive, one or more trained rescuers should begin CPR while another bystander activates the EMS system by calling 911 or the local emergency number and retrieves the AED if available. When contacting the EMS system, the rescuer should be prepared to provide the exact location of the emergency and a brief account of what happened and the initial care given. In the case of a lone rescuer and a witnessed arrest, the rescuer should first activate the EMS system by calling 911 or the local emergency number, obtain an AED if readily available, and return to the victim to initiate CPR and AED use. If an on-site response system is activated, the central communication center is responsible for contacting the EMS and activating on-site responders to facilitate transport of an AED to the victim.

An AED should be applied to the victim as soon as possible and turned on for rhythm analysis and defibrillation if indicated. If an AED is not immediately available, the rescuer

should open the airway by using the head tilt–chin lift maneuver and then “look, listen, and feel” for breathing. If a cervical spine injury is suspected, the modified jaw thrust maneuver is recommended to open the airway. The presence of normal breathing should distinguish the victim who has collapsed but does not require CPR. Agonal or occasional gasping should not be mistaken for normal breathing and should be recognized as a sign of SCA.<sup>67</sup> If a collapsed athlete is determined not to be in SCA and does not become normally responsive, the athlete should be continually reassessed for signs of, or progression to, SCA. The athlete may require other medical interventions depending on the specific cause of collapse.

If normal breathing is not detected within 10 seconds, 2 rescue breaths should be given, followed by chest compressions.<sup>7</sup> Lay rescuers (coaches, officials, and other bystanders) should not assess the athlete for a pulse or signs of circulation. Health care providers (athletic trainers, school nurses, emergency medical technicians, and physicians) should deliver 2 rescue breaths and may consider checking for a pulse. If the health care provider does not definitively feel a pulse within 10 seconds, CPR should be initiated. Rescue breaths are provided by pinching the victim’s nose, creating an airtight mouth-to-mouth seal, and giving one breath over 1 second that produces a visible chest rise. If the victim’s chest does not rise with the first rescue breath, the head tilt–chin lift maneuver should be repeated before the second breath in an attempt to open the airway. If the rescuer is unable or unwilling to give rescue breaths, then chest compressions should be initiated.

Effective chest compressions in adults are performed by compressing the lower half of the sternum, using the heels of the hands, and should depress the chest a depth of 3.8 to 5.1 cm; complete chest recoil permits venous return between compressions. Rescuers should “push hard and push fast.”<sup>67</sup> The CPR should be performed using the universal compression-to-ventilation ratio of 30:2 at a rate of 100 compressions per minute. When multiple rescuers are present, they should rotate the compressor role about every 2 minutes or earlier if fatigue develops.

An AED or a manual defibrillator should be applied for rhythm analysis as soon as it arrives. All interruptions in chest compressions for analyzing the rhythm or delivering a shock should be minimized. With 2 or more rescuers, CPR can be continued while attaching the AED leads. Defibrillation is more likely to be successful with a shorter time between chest compressions and delivery of a shock.<sup>78,79</sup> If VF or rapid VT is detected, the AED will instruct the rescuer to deploy a shock. The rescuer should deliver 1 shock and then immediately resume CPR, beginning with chest compressions. When 2 rescuers are present, the rescuer operating the AED should be prepared to deliver a shock as soon as the compressor removes his or her hands from the chest and all rescuers are clear of contact with the victim. The rescuer should not delay resuming chest compressions to allow for repeat rhythm analysis, and CPR should be continued for 5 cycles or about 2 minutes before rechecking the rhythm or until the victim becomes responsive. If no shock is advised after rhythm analysis, CPR should be immediately resumed and continued for 5 cycles before rechecking the rhythm or until advanced life support measures are available.

Some advanced airway devices such as an endotracheal tube, esophageal-tracheal Combitube, or laryngeal mask airway can be used by health care professionals with sufficient training and experience. Once an advanced airway is in place,



pauses or interruptions in chest compressions for ventilation are no longer necessary. Rescuers should deliver 100 compressions per minute and 8 to 10 breaths per minute continuously.<sup>67</sup> If available, bag-mask ventilation can also be performed by health care providers with the use of supplementary oxygen at a minimum flow rate of 10 to 12 L/min.<sup>7</sup>

### **Management of Sudden Cardiac Arrest: Unwitnessed Collapse**

If an athlete is found collapsed and unresponsive and the time lapse from onset of SCA is unknown, rescuers may consider 5 cycles or 2 minutes of CPR before checking the rhythm and attempting defibrillation.<sup>7,8</sup> After a prolonged cardiac arrest, a brief period of CPR can deliver oxygen and energy substrates, perhaps increasing the likelihood that a perfusing rhythm will return if defibrillation is possible.

## **SPECIAL CIRCUMSTANCES**

### **Cervical Spine Injury**

Any athlete suspected of having a cervical spine injury should not be moved, and the cervical spine should be immobilized. Unconscious athletes in collision sports are presumed to have unstable spine injuries until proved otherwise. High cervical spine injuries can cause apnea, ineffective breathing patterns, and paralysis of the phrenic nerve. Although rare, prolonged hypoxemia can lead to cardiac arrest. Protective equipment used in collision sports such as football and hockey makes the management of SCA in a spine-injured athlete difficult. The facemask should be removed (leaving the helmet in place) as soon as possible before transportation, regardless of respiratory status and even if the athlete is conscious.<sup>80</sup> Shoulder pads should also be opened (but not removed) before transportation to provide access to the chest if CPR or defibrillation is required. A designated rescuer must be responsible for manually stabilizing the head and neck during CPR and any transfer of the victim. The rescuer responsible for neck stabilization should disengage if defibrillation is necessary. For more information on the care of the spine-injured athlete, refer to "Prehospital Care of the Spine-Injured Athlete," available at <http://www.nata.org/statements/consensus/NATAPreHospital.pdf>.<sup>81</sup>

### **Commotio Cordis**

Commotio cordis, also called cardiac concussion, involves a blunt, nonpenetrating blow to the chest during a vulnerable phase of ventricular repolarization, leading to a ventricular arrhythmia with no structural damage or cardiac contusion present. Commotio cordis occurs most commonly in young male adolescents (mean age = 13.6 years) with compliant chest walls.<sup>47</sup> Approximately 80% of cases involve blunt chest impact by a firm projectile, such as a baseball, softball, hockey puck, or lacrosse ball, and 20% of cases are due to chest contact with another person.<sup>47</sup> To date, commercially available chest protectors have not been shown to prevent commotio cordis.<sup>82</sup> Survival after commotio cordis closely depends on the time to defibrillation.<sup>54</sup> Overall survival as reported from the United States Commotio Cordis Registry was only 16%, but for those victims still in VF who were reached in time to receive defibrillation, the survival rate was 46%.<sup>47</sup> Young ath-

letes who collapse shortly after being struck in the chest should be suspected of having commotio cordis until the athlete is clearly responsive. Rescuers can improve survival by promptly recognizing SCA due to commotio cordis, activating the EMS system, immediately initiating CPR, and using an AED as soon as possible.

### **Exertional Heat Stroke**

Heat stroke is a life-threatening emergency, and treatment of concurrent cardiac arrest requires simultaneously cooling the athlete and performing CPR. The presence of exertional heat stroke in a collapsed athlete must be suspected in hot, humid environments, especially if the athlete is wearing athletic clothing and equipment that limits heat loss, and the EMS system must be activated. Both heat exhaustion and heat stroke can cause syncope in the athlete. Heat stroke is differentiated by the presence of mental status changes and a core temperature of greater than 40°C (104°F). Untreated exertional heat stroke can progress to end-organ damage, adult respiratory distress syndrome, disseminated intravascular coagulation, neurologic injury, cardiac arrest, and death. The diagnosis of heat stroke can be confirmed on-site with a rectal temperature measurement. If the athlete is unresponsive but has normal breathing and circulation, rapid cooling by ice water bath immersion is recommended.<sup>83</sup> If an ice water tub is not available or if concurrent SCA is suspected, rotating ice water towels applied to the head, trunk, and extremities and ice packs applied to the neck, axilla, and groin represent an alternative method for cooling while performing CPR and using an AED. Because prompt temperature reduction is critical, transport to the emergency department for heat stroke victims without SCA should be delayed if sideline cooling measures, such as ice water bath immersion, are available.<sup>83</sup> Cooling should also be continued during transport if needed.

### **Lightning**

Lightning presents an environmental hazard with the potential for multiple victims. If the lightning storm is ongoing, rescuers must ensure their personal safety by moving an SCA victim indoors if possible. Spine immobilization should be considered. Cardiac arrest from lightning strike is associated with significant mortality and requires modification of standard ACLS measures to achieve successful resuscitation. Most lightning strike victims have associated multisystem involvement, including neurologic complications, cutaneous burns, soft tissue injury such as rhabdomyolysis, and associated blunt trauma.<sup>84</sup> When managing several lightning strike victims, the normal multiple casualty triage priorities are reversed. Casualties who appear unresponsive require prompt, aggressive resuscitation using standard CPR and ACLS protocols, including defibrillation and cardiac pharmacotherapy.<sup>84</sup> The chance for a successful outcome is greater for lightning-related cardiac arrest, even with initial rhythms that are traditionally unresponsive to therapy.

### **Mass Events**

Emergency preparation and management of SCA at mass athletic events require additional planning. Schools and institutions may have their own athletic staffs with them, and advanced communication with the host organization is helpful to

ensure that visiting athletic staffs are familiar with the EAP and central medical area or equipment. Distance events such as cross-country meets, triathlons, and marathons present an additional challenge because running or biking courses are often spread out over long distances, sometimes in remote areas. Among marathoners, SCA occurs in approximately 1 in 40 000 runners across the age spectrum.<sup>85</sup> Distributing medical staff and AEDs along the course or field of play and using bicycle or “golf-cart” rescue teams will improve response times should an emergency arise.

### Rainy, Wet, Ice, and Metal Surfaces

Defibrillators used in a wet environment or on an ice playing surface are considered safe and do not pose a shock hazard for rescuers or bystanders. If a collapsed victim with suspected SCA is lying on a wet surface or in a puddle, the patient should not be moved to avoid delays in initiating CPR. Simulation of a patient and a rescuer in a wet environment does not show a significant risk of electric shock.<sup>86</sup> Responders to an SCA on an ice playing surface should consider foot traction devices and helmets for their own safety.<sup>87,88</sup> In contrast, SCA victims found immersed in a pool or contained body of water should be removed from the water before defibrillation. Any SCA victims lying on metal conducting surfaces (eg, bleachers) should be moved to a nonmetal surfaces or placed onto spine boards before defibrillation if that can be done quickly and without significant delays.

### CONCLUSIONS

The most important factor in SCA survival is the presence of a trained rescuer who can initiate CPR and has access to early defibrillation. The athletic community is in a unique position to have trained coaches, officials, and other targeted responders, and, in some circumstances, on-site athletic trainers, school nurses, and team physicians respond immediately to SCA at organized athletic events and practices. Comprehensive emergency planning is needed for high school and college athletic programs to ensure an efficient and structured response to SCA. Essential elements to an EAP include establishing an effective communication system, training of anticipated responders in CPR and AED use, access to an AED for early defibrillation, acquisition of necessary emergency equipment, coordination and integration of on-site responder and AED programs with the local EMS system, and practice and review of the response plan. High suspicion of SCA should be maintained in any collapsed and unresponsive athlete, with application of an AED as soon as possible for rhythm analysis and defibrillation if indicated. Interruptions in chest compressions for rhythm analysis and shock delivery should be minimized, and rescuers should be prepared to resume CPR, beginning with chest compressions, as soon as a shock is delivered. Improved education in the recognition of SCA, enhanced emergency preparedness, training in current CPR protocols, and increased access to AEDs for early defibrillation are needed to improve survival from SCA in athletics.

### DISCLAIMER

The National Athletic Trainers’ Association and the Inter-Association Task Force advise individuals, schools, and institutions to carefully and independently consider each of the

recommendations. The information contained in the statement is neither exhaustive nor exclusive to all circumstances or individuals. Variables such as institutional human resource guidelines, state or federal statutes, rules, or regulations, as well as regional environmental conditions, may impact the relevance and implementation of these recommendations. The NATA and the Inter-Association Task Force advise their members and others to carefully and independently consider each of the recommendations (including the applicability of same to any particular circumstance or individual). The foregoing statement should not be relied on as an independent basis for care but rather as a resource available to NATA members or others. Moreover, no opinion is expressed herein regarding the quality of care that adheres to or differs from any of NATA’s position statements. The NATA and the Inter-Association Task Force reserve the right to rescind or modify their statements at any time.

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American Heart Association  
American Medical Society for Sports Medicine  
American Orthopaedic Society for Sports Medicine  
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American Physical Therapy Association Sports Physical Therapy Section  
National Association of Emergency Medical Service Physicians  
National Association of Emergency Medical Technicians  
National Athletic Trainers' Association  
National Collegiate Athletic Association  
National Federation of State High School Associations  
Sudden Cardiac Arrest Association

#### Endorsing national societies

For an updated list of endorsing national societies, go to [http://www.nata.org/statements/consensus/sca\\_endorsements.htm](http://www.nata.org/statements/consensus/sca_endorsements.htm)





## **Consensus Statement: Sickle Cell Trait and the Athlete**

### **Purpose**

In a recent review of non-traumatic sports deaths in high school and college athletes (1), the top four killers, in order of occurrence, were: cardiovascular conditions, hyperthermia (heatstroke), acute rhabdomyolysis tied to sickle cell trait, and asthma. Acute exertional rhabdomyolysis (explosive muscle breakdown) from sickle cell trait is the least understood of these conditions. The purpose of this Task Force is to raise awareness of this condition and provide measures to reduce the risk of exertional collapse related to sickle cell trait.

### **Introduction**

Sickle cell trait is the inheritance of one gene for sickle hemoglobin and one for normal hemoglobin. During intense or extensive exertion, the sickle hemoglobin can change the shape of red cells from round to quarter-moon, or "sickle." This change, exertional sickling, can pose a grave risk for some athletes. In the past seven years, exertional sickling has killed nine athletes, ages 12 through 19.

Research shows how and why sickle red cells can accumulate in the bloodstream during intense exercise. Sickle cells can "logjam" blood vessels and lead to collapse from ischemic rhabdomyolysis, the rapid breakdown of muscles starved of blood. Major metabolic problems from explosive rhabdomyolysis can threaten life. Sickling can begin in 2-3 minutes of any all-out exertion – and can reach grave levels soon thereafter if the athlete continues to struggle. Heat, dehydration, altitude, and asthma can increase the risk for and worsen sickling, even when exercise is not all-out. Despite telltale features, collapse from exertional sickling in athletes is under-recognized and often misdiagnosed. Sickling collapse is a medical emergency.

We recommend confirming sickle cell trait status in all athletes' preparticipation physical examinations. As all 50 states screen at birth, this marker is a base element of personal health information that should be made readily available to the athlete, the athlete's parents, and the athlete's healthcare provider, including those providers responsible for determination of medical eligibility for participation in sports.

Knowledge of sickle cell trait status can be a gateway to education and simple precautions that may prevent sickling collapse and enable athletes with sickle cell trait to thrive in sport. Nearly all of the 13 deaths in college football have been at institutions that did not screen for sickle cell trait or had a lapse in precautions for it. Small numbers preclude cogent evidence to support screening. All considered, however, we believe that each institution should carefully weigh the decision to screen based on the potential to provide key clinical information and targeted education that can save lives. Irrespective of screening, the institution should educate staff, coaches, and athletes on the potentially lethal nature of this condition.

### **Background**

A condition of inheritance versus race, the sickle gene is common in people whose origin is from areas where malaria is widespread. Over the millennia, carrying one sickle gene fended off death from malaria, leaving one in 12 African-Americans (versus one in 2,000 to one in 10,000 white Americans) with sickle cell trait. The sickle gene is also present in those of Mediterranean, Middle Eastern, Indian, Caribbean and South and Central American ancestry; hence, the required screening of all newborns in the United States.

In the past four decades, exertional sickling has killed at least 15 football players. In the past seven years alone, sickling has killed nine athletes: five college football players in training, two high school athletes (one a 14-year-old female basketball player), and two 12-year-old boys training for football. Of 136 sudden, non-traumatic sports deaths in high school and college athletes over a decade, seven (5%) were from exertional sickling (1).



The U. S. military tied sickle cell trait to sudden death during recruit basic training. The relative risk of exercise-related death in sickle cell trait was about 30 (2). In other words, recruits with sickle cell trait were 30 times more likely to die during basic training. The main cause of death was rhabdomyolysis – and the risk of exertional rhabdomyolysis was about 200 times greater for those with sickle cell trait (3).

In sickle cell trait, strenuous exercise evokes four forces that in concert foster sickling, 1) severe hypoxemia, 2) metabolic acidosis; 3) hyperthermia in muscles, and 4) red-cell dehydration.

Evidence supports this syndrome. Military research shows that, during intense exertion and hypoxemia, sickle cells can accumulate in the blood (4). Recent research also shows that systemic dehydration worsens exertional sickling (5). Field studies in Africa suggest that sickle-trait runners are limited not in single sprints but in middle distance or altitude running (6). The pattern in American athletes is similar.

### **Sickling Collapse: Football and Other Sports**

The first known sickling death in college football was in 1974. A defensive back from Florida ran a conditioning test on the first day of practice at altitude in Colorado. He had collapsed on the first day of practice the year before. This time, near the end of the first long sprint, at about 700 meters, he collapsed again – and died the next day. The most recent sickling death, a freshman defensive back at Rice University in the fall of 2006, is similar. He collapsed after running 16 sprints of 100 yards each – and died the next morning. The cause of death for both athletes was acute exertional rhabdomyolysis associated with sickle cell trait.

Up to 13 college football players have died after a sickling collapse. The setting and syndrome in most are similar:

- Sickling players may be on-field only briefly, sprinting only 800-1,600 meters, often early in the season.
- Sickling can also occur during repetitive running of hills or stadium steps, during intense sustained strength training, if the tempo increases late in intense one-hour drills, or at the end of practice when players run “gassers.”
- Sickling can even occur rarely in the game, as when a running back is in constant action during a long, frantic drive downfield (7).

Sickling collapse is not limited to football. It has occurred in distance racing and has killed or nearly killed several college or high school basketball players (two were females) in training, typically during “suicide sprints” on the court, laps on a track, or a long training run.

The harder and faster athletes go, the earlier and greater the sickling, which likely explains why exertional collapse occurs “sooner” in college football players sprinting than in military recruits running longer distances. Sickling can begin in only 2-3 minutes of sprinting – or in any other all-out exertion – and sickling can quickly increase to grave levels if the stricken athlete struggles on or is urged on by the coach.

### **Sickling Collapse: Telltale Features**

Sickling collapse has been mistaken for cardiac collapse or heat collapse. But unlike sickling collapse, cardiac collapse tends to be “instantaneous,” has no “cramping” with it, and the athlete (with ventricular fibrillation) who hits the ground no longer talks. Unlike heat collapse, sickling collapse often occurs within the first half hour on-field, as during initial windsprints. Core temperature is not greatly elevated.

Sickling is often confused with heat cramping; but, athletes who have had both syndromes know the difference, as indicated by the following distinctions:

- 1) Heat cramping often has a prodrome of muscle twinges; whereas, sickling has none;
- 2) The pain is different – heat-cramping pain is more excruciating;
- 3) What stops the athlete is different – heat crampers hobble to a halt with “locked-up” muscles, while sickling players slump to the ground with weak muscles;
- 4) Physical findings are different – heat crampers writhe and yell in pain, with muscles visibly contracted and rock-hard; whereas, sicklers lie fairly still, not yelling in pain, with muscles that look and feel normal;
- 5) The response is different – sickling players caught early and treated right recover faster than players with major heat cramping (7).

This is not to say that all athletes who sickle present exactly the same way. How they react differs, including some stoic players who just stop, saying “I can’t go on.” As the player rests, sickle red cells regain oxygen in the lungs and most then revert to normal shape, and the athlete soon feels good again and ready to continue. This self-limiting feature surely saves lives.

### **Precautions and Treatment**

No sickle-trait athlete is ever disqualified, because simple precautions seem to suffice. For the athlete with sickle cell trait, the following guidelines should be adhered to:

- 1) Build up slowly in training with paced progressions, allowing longer periods of rest and recovery between repetitions.
- 2) Encourage participation in preseason strength and conditioning programs to enhance the preparedness of athletes for performance testing which should be sports-specific. Athletes with sickle cell trait should be excluded from participation in performance tests such as mile runs, serial sprints, etc., as several deaths have occurred from participation in this setting.
- 3) Cessation of activity with onset of symptoms [muscle ‘cramping’, pain, swelling, weakness, tenderness; inability to “catch breath”, fatigue].
- 4) If sickle-trait athletes can set their own pace, they seem to do fine.
- 5) All athletes should participate in a year-round, periodized strength and conditioning program that is consistent with individual needs, goals, abilities and sport-specific demands. Athletes with sickle cell trait who perform repetitive high speed sprints and/or interval training that induces high levels of lactic acid should be allowed extended recovery between repetitions since this type of conditioning poses special risk to these athletes.
- 6) Ambient heat stress, dehydration, asthma, illness, and altitude predispose the athlete with sickle trait to an onset of crisis in physical exertion.
  - a. Adjust work/rest cycles for environmental heat stress
  - b. Emphasize hydration
  - c. Control asthma
  - d. No workout if an athlete with sickle trait is ill
  - e. Watch closely the athlete with sickle cell trait who is new to altitude. Modify training and have supplemental oxygen available for competitions
- 7) Educate to create an environment that encourages athletes with sickle cell trait to report any symptoms immediately; any signs or symptoms such as fatigue, difficulty breathing, leg or low back pain, or leg or low back cramping in an athlete with sickle cell trait should be assumed to be sickling (7).

In the event of a sickling collapse, treat it as a medical emergency by doing the following:

- 1) Check vital signs.
- 2) Administer high-flow oxygen, 15 lpm (if available), with a non-rebreather face mask.
- 3) Cool the athlete, if necessary.
- 4) If the athlete is obtunded or as vital signs decline, call 911, attach an AED, start an IV, and get the athlete to the hospital fast.
- 5) Tell the doctors to expect explosive rhabdomyolysis and grave metabolic complications.
- 6) Proactively prepare by having an Emergency Action Plan and appropriate emergency equipment for all practices and competitions.

## **IMMEDIATE ACTION CAN SAVE LIVES**

### **What We Can Do**

Though screening is done at birth; many athletes do not know their sickle-trait status, rendering self-report in a questionnaire unreliable. Many institutions have employed screening strategies to rectify this. A recent survey of NCAA Division I-A schools found that 64% (of respondents) screen (8). The NFL Scouting Combine screens for sickle cell trait. All considered, despite no evidence-based proof yet that screening saves lives, each institution should carefully weigh the decision to screen in the absence of documented newborn screen results.

### **The Consensus of this Task Force is:**

- 1) There is no contraindication to participation in sport for the athlete with sickle cell trait.
- 2) Red blood cells can sickle during intense exertion, blocking blood vessels and posing a grave risk for athletes with sickle cell trait.
- 3) Screening and simple precautions may prevent deaths and help athletes with sickle cell trait thrive in their sport.
- 4) Efforts to document newborn screening results should be made during the PPE.
- 5) In the absence of newborn screening results, institutions should carefully weigh the decision to screen based on the potential to provide key clinical information and targeted education that may save lives.
- 6) Irrespective of screening, institutions should educate staff, coaches, and athletes on the potentially lethal nature of this condition.
- 7) Education and precautions work best when targeted at those athletes who need it most; therefore, institutions should carefully weigh this factor in deciding whether to screen. All told, the case for screening is strong.

### **Glossary**

**Acute Ischemic rhabdomyolysis:** the rapid breakdown of muscle tissue starved of blood

**Acute Rhabdomyolysis:** a serious and potentially fatal condition involving the breakdown of skeletal muscle fibers resulting in the release of muscle fiber contents into the circulation

**Contraindication:** circumstance or condition that makes participation unsafe or inappropriate

**Exertional rhabdomyolysis:** muscle breakdown triggered by physical activity

**Exertional sickling:** hemoglobin [red blood cell] sickling due to intense or sustained physical exertion

**Hyperthermia:** body temperature elevated above the normal range

**Hypoxemia:** decreased oxygen content of arterial blood

**Ischemia:** a deficiency of blood flow to tissue

**Metabolic acidosis:** a condition in which the pH of the blood is too acidic because of the production of certain types of acids

**Nontraumatic:** not related to a physical injury caused by an external force

**Obtunded:** having diminished arousal and awareness; mentally dull

**Sickling collapse:** the collapse of an athlete who shows features consistent with exertional sickling

**Ventricular Fibrillation:** a condition in which there is uncoordinated contraction of the cardiac muscle of the ventricles in the heart

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The following individuals and associations were members of the Inter-Association Task Force on Sickle Cell Trait and the Athlete. Their participation is not an endorsement of this document. For a complete list of supporting associations, please visit [http://www.nata.org/statements/consensus/sct\\_endorsements.htm](http://www.nata.org/statements/consensus/sct_endorsements.htm).

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Athletic trainers are health care professionals who specialize in the prevention, diagnosis, treatment and rehabilitation of injuries and sport-related illnesses. They prevent and treat chronic musculoskeletal injuries from sports, physical and occupational activity, and provide immediate care for acute injuries. Athletic trainers offer a continuum of care that is unparalleled in health care. NATA represents and supports 32,000 members of the athletic training profession.

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