A G E N D A

National Collegiate Athletic Association

Committee on Competitive Safeguards and Medical Aspects of Sports

National Office – Grant Ballroom B June 12 – 14, 2012
Indianapolis

1. Welcome and introductions, new CSMAS members, review current committee roster and preview meeting agenda. [Refer to Collaboration Zone (CZ) – Committee Home Tab/Information folder]

2. Review CSMAS committee procedures. [Refer to CZ – Committee Home Tab/Information folder]

   a. Mission statement and duties.

   b. Conflict of Interest.

   c. Committee materials and open records. (Naima Stevenson)

3. Report of the December 2011 CSMAS meeting. [Supplement No. 1] **[Action Anticipated]**

4. NCAA governance actions and issues.

   a. NCAA Division III. (Dan Dutcher, Louise McCleary via teleconference)

      (1) Sickle Cell Trait. [Supplement No. 2 - To Be Distributed] (Jim Schmotter)

      (2) Management Council. [Supplement No. 3] (Lori Runksmeier)

         - Football walk-throughs. [Supplement No. 4]

      (3) Ice Hockey start date for practice and competition. [Supplement No. 5] (Runksmeier)

      (4) Student-Athlete Advisory Committee (SAAC). (Jacqui Cohen)

b. NCAA Division II.

(1) Management Council. [Supplement No. 6] (Will Prewitt)

- Drug testing manipulation-tampering penalty. [Supplement No. 7]

(2) SAAC. (David Pillen)

c. NCAA Division I.

(1) Institutional Performance Program. (Troy Arthur)

(2) Bylaw Deregulation. (Danielle Ghiloni, Charnele Kemper, Binh Nguyen) [Supplement No. 8]

(3) Championships/Sports Management Cabinet. [Supplement No. 9]

- Football walk-throughs.

(4) SAAC. (Matthew Horn)

5. Medical Issues.

a. Concussions.

(1) Research. (Kevin Guskiewicz, Tom Dompier)

(2) Ivy League. [Supplement No. 10]

(a) Football preseason modifications. (Brant Berkstresser)

(b) Lacrosse concussion prevention through rules modifications in the Men's game. (Berkstresser)

(3) Chronic traumatic encephalopathy (CTE). [Supplement No. 11]

(4) Concussion management plans.

(5) Concussion summit meeting outcomes revisited. [Supplement No. 12]

(6) Concussion perspectives. [Supplement No. 13] (Jim Morgan)

(7) Webinar.

(8) Education and resources.

b. Catastrophic sport injury. [Refer to CZ]

(1) University of North Carolina Research paradigm. [Supplement No. 14]

(2) Inter-Association Task Force outcomes on preventing sudden death in sport. [Supplement No. 15]

(3) Interaction between strength and conditioning and sports medicine staff.

(4) Certification of Division I and Division II strength and coaches legislation. [Supplement No. 16]

c. Cardiac update.

(1) Research.

(2) Coach sports safety training in Division I. [Supplement No. 17]

d. Sickle cell trait (SCT).

(1) Quest Diagnostics Agreement. [Supplement No. 18]

(2) External publications.

(a) American Society of Hematology (ASH) position statement. [Supplement No. 19]

(b) National Athletic Trainers Association (NATA) position statement. [Supplement No. 20]

(c) American College of Sports Medicine (ACSM)/NCAA Summit. [Supplement No. 21]

(d) Various study findings. [Supplement No.22]

(3) Research. [Supplement No. 23 - To Be Distributed]

e. Medical examinations.

(1) Division I Men's Basketball Tryout. [Supplement No. 24]

(2) Overview of purpose for medical examination and whether any other required components besides SCT should be mandatory. [Supplement No. 25]

f. Psychological/mental health. (Chris Carr)

(1) Research.

(2) Sports Medicine Handbook guideline. [Supplement No. 26 - To Be Distributed]

g. Intersex education plan. [Supplement No. 27] **[Action Anticipated]**

h. Sun Safety. **[Action Anticipated]**

(1) Research. [Supplement No. 28]

(2) Sports Medicine Handbook draft guideline. [Supplement No. 29]

(3) Fact sheet. [Supplement No. 30]

6. Research issues.

a. NCAA Athletics Healthcare Survey report and future steps. [Supplement No. 31]

b. NCAA Survey of Institutional reliance on Supplement Sponsorships. [Supplement No. 32] (Annie Kearns)

c. NCAA Student-Athlete Substance Use Survey 2012 - final draft. [Supplement No. 33] (Nicole Bracken)

d. NCAA Injury Surveillance Program and the Datalys Center. (Dompier)

7. Substance use issues.

a. NCAA drug-testing backgrounder. [Supplement No.34]

b. Medical Controlled Substances Transportation Act of 2011. [Supplement No. 35]

c. Attention Deficit Hyperactivity Disorder (ADHD) Reporting Form – edits. [Supplement No. 36]

d. Institutional responsibility for NCAA Drug Testing. [Supplement No. 37] **[Action Anticipated]**

e. Growth hormone testing proposed protocol. [Supplement No. 38 - To Be Distributed] **[Action Anticipated**]

f. Marijuana.

(1) Drug-testing cut-off. [Supplement No. 39 - To Be Distributed] **[Action Anticipated]**

(2) Marijuana use deterrence issues.

g. Synthetic cannabinoid testing. [Supplement No. 40 - To Be Distributed] **[Action Anticipated]**

h. Division III and Student Affairs Administrators in Higher Education (NASPA)/Alcohol and Other Drug (AOD) collaborative.

i. Division II AOD working group.

j. Tobacco prevention outreach. [Supplement No. 41]

k. Nutritional/dietary supplements.

(1) Advertising Policy update. (Scott Bearby)

(2) SAAC review of 16.5.2.g.

8. NCAA legal affairs update**.** (Stevenson)

9. Outreach.

a. Coalition of Higher Education Associations for Substance Abuse Prevention (CoHEASAP). [Supplement No. 42]

b. American College Health Association (ACHA)/NATA/NCAA collaborative presentations. [Supplement No. 43]

c. NATA.

(1) NATA College and University Athletic Trainer (CUATC) Committee. [Supplement No. 44] (Melinda Larson)

(2) 2013 NATA annual meeting affiliate session topics.

d. National Federation of State High School Associations (NFHS) - Sports Medicine Committee. [Supplement No. 45 - Refer to CZ/To Be Distributed Folder] (Bob Colgate)

e. Board of Certification. Facility and Patient Care Principles. [Supplement No. 46]

f. Sports Cardiovascular and Wellness Nutrition (SCAN) and Collegiate and Professional Sports Dietitians Association (CPSDA) update. (Amy Freel)

g. Other.

-Sports Science Safety Subcommittee -

10. Datalys Center and NCAA Injury Surveillance. [Supplement No. 47 - Refer to CZ/To Be Distributed folder] (Dompier)

a. Recruitment and school participation.

b. List of deliverables and future data projects.

c. Future system changes and updates.

d. 2009-10, 2010-11, 2010-12 fall sport data reports. [Supplement No. 48 - Refer to CZ/To Be Distributed folder]

e. Report on external researcher scientific research requests. [Refer to CZ]

f. Sport fact sheets. [Refer to CZ]

11. Health and safety graduate research grant proposals. [Refer to CZ]

a. Review Criteria. [Supplement No. 49]

b. "Too Rough For Bare Heads": The Adoption of Helmets and Masks in Intercollegiate Ice Hockey.

c. Effect of Competition on Performance and Physiological Responses.

d. Body Symmetry as a Predictor of Performance in Elite Student-Athlete Populations.

e. Perceptions of Performance-enhancing Drug Use Among College Athletes and Risk Related Correlates: A Social Cognitive Theoretical Exploration.

f. Why do some sickle cell carrier athletes suffer from heat illness?

g. The effects of body consciousness on energy balance in female collegiate athletes.

h. Institutional concussion education and within season change in concussion-related knowledge, attitudes and behaviors on male collegiate ice hockey teams.

i. Does a static stretching program reduce the amount of atrophy and the loss of strength caused by immobilization of a joint?

j. Metabolic Syndrome in the Elite Athlete.

k. Student-Athlete Attitudes toward Seeking Mental-Health Help.

12. Sports Medicine Handbook.

a. Identify guidelines for review.

b. Rhabdomyolysis.

c. Mental health. [Refer to Supplement No. 26]

d. Sun Safety. [Refer to Supplement No. 29]

13. Severe Weather and Lightning. [Supplement No. 50]

14. Out of season/preseason training best practices.

a. Football.

(1) Out of season training best practices for performance development/sudden death prevention. [Supplement No. 51] (Eric Hall, Sourav Poddar)

(2) Southern Conference Proposal. [Supplement No. 52]

b. Preseason models for fall sports other than football.

15. Equipment issues.

a. Field turf standards. [Supplement No. 53]

b. Review Shock Strips, Brain Pad mouth guards, heat alert mouth guards, and the Guardian Cap for football helmets. [Sample products - To Be Distributed]

-Drug-Education and Drug-Testing Subcommittee-

16. Report from The National Center for Drug Free Sport, Inc. [Supplement No. 54 – Refer to CZ/To Be Distributed folder] (Mark Bockelman, Andrea Wickerham)

a. Dietary Supplement Resource Exchange Center (REC) Report.

b. Drug-test results.

c. Laboratory testing issues.

d. Drug-testing basics booklet.

17. Evaluation of Drug Free Sport.

a. Crew chief evaluations. [Supplement No. 55] **[Action Anticipated]**

b. Subcommittee meeting evaluation.

18. Banned Drug Classes. [Supplement No. 56]

a. World Anti-Doping Agency (WADA) list. [Refer to website – http://www.wada-ama.org/en/]

b. Testing for synthetic cannabinoids and "bath salts" - mephedrone. [Refer to Supplement No. 40]

c. Other.

19. Review drug-testing protocol changes. [Supplement No. 57 – To Be Distributed] **[Action Anticipated]**

20. Review Appeal process.

- Revisit edits to Section 5. [Supplement No. 58] (Bockelman, Morgan)

21. Review Medical Exceptions process. [Supplement No. 59]

- Medical Marijuana update.

22. Drug-testing compliance forms.

a. Consent form. [Supplement No. 60]

b. Student-Athlete Statement. [Supplement No. 61]

-Full Committee-

23. Sport Playing Rules.

a. Lacrosse.

(1) US Lacrosse safety initiatives. [Supplement No. 62]

(2) National Operating Committee on Standards for Athletic Equipment (NOCSAE) Lacrosse ball standard and recommendations for adoption. [Supplement No. 63]

b. Field hockey eyewear. [Supplement No. 64]

c. Ice hockey face protection. [Supplement No. 65] (Berkstresser, Ty Halpin via teleconference, Ed McLaughlin)

d. Pole vault safety. [Supplement No. 66]

e. Wrestling. (Teresa Smith, Jim Thornton)

(1) Skin infection prevention video script. [Supplement No. 67]

(2) Wrestling Rules committee proposals. [Supplement No. 68]

(3) Skin infection policy. [Refer to CZ/Sports Medicine Handbook]

f. Football.

(1) Playing rules changes overview. [Supplement No. 69]

(2) Safety video and poster education. [Supplement No. 70]

(3) Bounty concerns. [Supplement No. 71] **[Action Anticipated]**

24. Subcommittee reports/final recommendations. **[Action Anticipated]**

25. NCAA Sponsored Programs.

a. APPLE Conferences report. [Supplement No. 72] **[Action Anticipated]**

b. CHOICES Grants. [Supplement No. 73] **[Action Anticipated]**

c. NCAA Student-Athlete Affairs Grant report. [Supplement No. 74 – Refer to CZ/To Be Distributed folder]

d. REC. [Refer to website - www.drugfreesport.com/rec]

e. NCAA Youth Clinics.

- Stop Sports Injuries campaign. [Refer to website – http://www.stopsportsinjuries.org/]

f. NCAA webinars. [Supplement No. 75]

g. Leadership Programming – conferences and workshops. [Refer to website - www.ncaa.org/studentathleteaffairs] (Curtis Hollomon)

h. Videos.

(1) SCT.

- Division III Presidents webinar. [Supplement No. 76]

(2) NCAA Drug Testing – draft script. [Supplement No. 77]

26. Education resources and opportunities overviews.

a. Student-Athlete Affairs Advisory Group.

b. NCAA public website at www.ncaa.org

c. NCAA health and safety link at www.ncaa.org/health-safety

d. NCAA drug-testing link at www.ncaa.org/drugtesting

e. NCAA nutrition link at www.ncaa.org/nutritionandperformance

27. Other business.

a. NCAA Violence Prevention Think Tank. (Cari Klecka)

b. Step UP! Bystander Intervention Facilitator Training. [Refer to website - http://www.stepupprogram.org/]

c. Coaches' Assist web resource. [Supplement No. 78]

d. NCAA Governance Leadership Scorecard – CSMAS evaluation. [Supplement No. 79]

e. Committee evaluation of CSMAS meeting online.

28. Future meetings. [Supplement No. 80]

a. December 2012 – Date To Be Determined – Indianapolis, Indiana.

b. June 2013 – Date To Be Determined – Indianapolis, Indiana.

29. Adjournment.

REPORT OF THE NCAA COMMITTEE ON COMPETITIVE SAFEGUARDS AND MEDICAL ASPECTS OF SPORTS DECEMBER 11-13, 2011, MEETING

ACTION ITEMS.

1. Legislative Items.

- **Football walk-throughs.**

(1) Recommendation. To amend NCAA Division I Bylaw 17.9.2.4.1 and Division III Bylaw 17.9.2.2 to align the language with that of Division II, which states that **student-athletes must be provided with at least three continuous hours of recovery time between any sessions occurring that day.**

(2) Effective Date. August 1, 2012.

(3) Rationale. The committee reviewed the current permissible walk-through legislation in the sport of football and found inconsistencies across divisions. In the interest of providing consistent safeguards for student-athletes, the committee recommended that the three divisions evaluate this legislation and align the language with that of Division II, which states that student-athletes must be provided with at least three continuous hours of recovery time between any sessions occurring that day. In Division III, removal of the current exception should also be considered: "if the walk-through session occurs first, three hours of recovery time is not required." The impact of this legislation would ensure student-athletes have recovery from the cumulative effects of on-field activities, especially in the heat.

(4) Estimated Budget Impact. None.

(5) Student-Athlete Impact. Improved health and safety of the student-athlete.

2. Nonlegislative Items.

- None.

INFORMATIONAL ITEMS.

1. Championship Drug Testing Communications. The committee discussed concerns related to the administration of NCAA championships drug-testing and communications by the host site administrators. The committee reaffirms that an emphasis should be in place at

every site to assure that the drug-testing statement is read and preparations for potential drug testing are in place in any event as outlined in the Championships manuals. The committee requested staff to ensure these policies are adopted at all venues.

2. **Marijuana positive test threshold**. The committee discussed laboratory testing thresholds for marijuana and asked Drug Free Sport to provide a report in June with information from the NCAA drug-testing lab that identifies marijuana levels in NCAA samples between 5-10 and 10-15 ng/mL. This information will be considered for a potential recommendation to amend the threshold for a positive test for marijuana. A recommendation in June would allow for notice to the membership in advance of fall championship testing.

3. **Drug-Testing Protocol.** As it does on a biannual basis, the committee reviewed the NCAA drug-testing protocol. Changes to the drug-testing protocol were approved that provide for testing with a full panel (including marijuana and stimulants) in the event a student-athlete is found in breach of protocol, including attempted manipulation or tampering with the sample and failure to show for scheduled drug testing.

4. **Drug-Testing Appeal Procedures.** As it does on a biannual basis, the committee reviewed the NCAA drug-testing appeal procedures. Amendments to the Drug-Testing Appeal Procedures were approved that provide clarifying language on what constitutes grounds for appeal. The amended appeals language will be published for 2012-13.

5. **Need to Increase Drug-Testing Numbers.** In light of increases in championship events and general growth of the membership, including approximately 60,000 additional student-athletes in the last 5 years, the committee will review in June a plan for expanding drug testing to maintain the necessary deterrence effect and to assist the committee with potential budget recommendations.

6. **Dietary Supplement Resource Exchange Center (REC).** The committee reviewed a report from the REC outlining utilization for 2010-11. In order to better inform the membership of this authoritative resource to assist with questions related to nutritional/dietary supplements and NCAA banned drugs, the committee identified additional outreach to collegiate strength coaches, the College and University Athletic Trainers and the College Physicians Health Association; recommended the REC add a positive nutrition performance message and link to the final REC disclaimer on all inquiry

responses; and recommended the REC continue to investigate a mobile application, currently under consideration by the REC staff.

7. **NCAA Study of Substance Use.** The committee reviewed and recommended to the NCAA research staff proposed changes to the 2012-13 Substance Use survey instrument, including the addition of questions related to synthetic cannabinoids, growth hormone and insulin. The paper survey will be administered by faculty athletics representatives (FAR) during that academic year; research staff continues to evaluate the potential for an electronic administration, which presents some challenges related to Institutional Review Board (IRB) approval; the preliminary report of the 2009 Substance Use Study was provided for committee final review and comment, noting the report established a new trend line beginning with the 2005 re-weighted data.

8. **Institutional Drug-Testing Programs.** The committee received a preliminary report from the NCAA research staff of the 2011 NCAA Survey of Institutional Drug Education and Testing. The committee recommended addressing concerns about relatively low response rates by exploring the following: returning to paper administration; target a different recipient who can still answer institutional policy questions, including the senior woman administrator (SWA), compliance officer, director of sport medicine; provide results of last survey administration to inform institutions of the value of participating in the survey; and discuss survey administration issues with the Faculty Representative Advisory Panel (FRAP). Also, noting that institutional testing programs vary greatly in their practices and sanctions, the committee directed staff to promote the best practices for institutional drug testing to the membership.

9. **Division II Alcohol, Other Drug (AOD) Concerns.** The committee received a verbal report from the Division II Management Council and met with Division II staff to discuss information that would assist Division II to provide enhanced drug use deterrence, including factors to consider if adding marijuana testing to year-round testing, and to identify best practices for AOD education and partnering with student affairs personnel. This information will be collected and submitted for the Division II Management Council April meeting.

10. **Growth Hormone Testing.** The committee requested that an education module be presented at the June committee meeting to address issues related to growth hormone testing, including protocol, cost, feasibility and education of the membership.

11. **Reduced Drug-Testing Penalty.** The committee reaffirmed its intent that a reduction of the sanction for a positive drug test reduced to a 50 percent penalty upon appeal should be for only withholding from the next 50 percent of competition in all sports, and not carry the loss of an additional year of remaining eligibility.

12. **Medical Exception Request for ADHD Stimulant Medicine.** The committee approved, with edits, a cover form to submit for a medical exception for a positive test for stimulant medication for Attention Deficit/Hyperactivity Disorders (ADHD). The form will be made available to the membership as an option for the remainder of the 2011-12 drug testing cycle, and will be identified as the required form to submit beginning August 1, 2012.

13. **NCAA Year-Round Drug-Testing Notice.** The committee reaffirmed its position to provide no more than 48 hour notice to member institutions in year-round testing, noting that drug testing best practices include short or no-notice testing to enhance deterrence and detection. The practice of short (no more than 24 hour notice) and no-notice testing is in place in Division I and in some Division II year-round testing events.

14. **NCAA Drug Testing for Synthetic Cannabinoids.** The committee has sponsored research with the UCLA testing laboratory to validate a test for synthetic cannabinoids. Upon the results of this study, the committee will discuss the feasibility of testing for these substances within the championship testing panel.

15. **Tobacco Use.** The committee reviewed the results of the 2009 Substance Use study and noted seemingly unyielding spit tobacco use in several men's sports (baseball, wrestling, ice hockey and lacrosse). The committee reaffirms support for maintaining the NCAA bylaws that prohibit tobacco use by student-athletes and athletics personnel during practice or competition, and recommended increased educational outreach to the membership.

16. **Nutritional/Dietary Supplements.** The committee recommended staff conduct research to investigate member institutions' perceived and real benefits related to their sponsorships with supplement products/companies. The committee recommended investigating the feasibility to support, through funding, resources and partnerships, the promotion of a campaign to assist student-athletes' understanding of performance and health benefits of good nutrition. The committee reviewed with NCAA staff the NCAA advertising standards that preclude advertising of impermissible supplement products at NCAA

sanctioned events; additional review is being sought from the Student-Athlete Advisory Committees (SAAC). Staff is currently collecting this feedback and will draft a rationale statement for committee review and position development at the June meeting.

17. Ad Hoc Committee Positions. The committee recommended inviting a dietitian and a licensed psychologist that work directly with collegiate student-athletes as non-voting consultants to subsequent meetings in an effort to provide the committee expert consultation on student-athlete health and safety.

18. Field Hockey. Based on feedback and questions about the June 2011 CSMAS recommendation to require field hockey field player to wear American Society for Testing and Materials (ASTM) certified eye protection, further investigation was conducted through independent testing of current ASTM approved eye protection for field hockey on the market. Upon committee review and further discussion, the committee recommends placing a hold on the implementation of the recommendation for field hockey eye protector until the equipment on the market can be certified to meet the demands for the collegiate sport. The next steps identified by the committee included: (a) the need to confirm a standard ball velocity for the collegiate game; (b) the need to confirm a field hockey ball standard that describes the mechanical properties and performance criteria of the ball so that it is consistent for both testing and playing purposes; (c) the need to consider a refinement in the ball velocity requirements in the ASTM standard; and (d) confirmation that the products on the market meet the demands for the collegiate sport. The committee reaffirmed its goal to minimize the risk of eye injury. It was noted that the National Federation of State High School Associations (NFHS) required field hockey players to wear ASTM -approved protective eyewear for the high school game, effective with the 2011-12 season.

19. Sports Injury Surveillance. The committee received a report by the Datalys Center for Sports Injury Prevention and Research on the NCAA Injury Surveillance program. Institution participation rates have continued to steadily increase yet are not to a level that provides for annual data review for injury analysis across the divisions. At the given rate of institution participation, data can only be aggregated over a three year period. The goal is to be able to evaluate the impact of sport rule change interventions from one year to the next and provide real time analysis of injury characteristics. Given the significance of injury data for NCAA decision making purposes, more schools are needed and the committee encourages athletics department leadership to support their staff so they can submit the required injury and exposure data. Review of the 2009-2010 and 2010-2011 datasets revealed insufficient data for comparison in most sports and thus require a third

year of data to report outcomes. The Datalys Center will conduct a retrospective study of women's rugby injury in an effort to better understand the scope of injuries in the NCAA emerging sport. The NCAA is releasing approved sport injury data sets to scientific researchers to study sport injury questions and will be publishing sports fact sheets for public access.

20. Concussions. The committee reviewed and discussed the current legislation, educational material and website resources. The committee recommended that grant funds be made available to support further research on the extent of neurological changes due to head trauma in sports. In addition, the committee suggested that examples of division-specific concussion plans be made available on the website. The NCAA is participating in an initiative with other sport governing bodies to create common data elements for research purposes that would encourage the study of mild traumatic brain injury (mTBI) over a lifespan and better understand the development of neurological conditions such as early onset chronic traumatic encephalopathy and what intrinsic and extrinsic risk factors, if any, contribute to this phenomena.

21. Sickle Cell Trait. The committee discussed the medical risks of a student-athlete with sickle cell trait competing at altitude. Due to the variability of factors involved with each individual and their medical conditions, the committee agreed in concept that the decision to arrive early to a competition site, including NCAA championships sites, should be permissible and the responsibility of the institution, the institution's medical staff and the student-athlete. The committee will work with Championships staff to outline guidelines that event management staff should consider for medical arrangements. The committee reviewed the proposals for Division II and Division III to legislate the sickle cell solubility test during the medical examination. The committee also reviewed current resources and website material. The committee reviewed the current agreement with Quest Diagnostics Laboratories to provide three options available to the membership and student-athletes for sickle cell trait screening.

22. Cardiac. The committee reviewed the educational material that was sent to the membership in August 2011 and is available online. It was noted that cardiac sudden death is the leading medical cause of death in student-athletes during exertion. The committee has funded a study to evaluate the feasibility of conducting diagnostic testing to screen for cardiac disorders. Upon the results of the study, the committee will discuss the feasibility of additional diagnostic testing during medical examinations for collegiate athletes.

23. Psychological and Mental Health. The committee discussed the continued growing need for more information about mental health in athletes and has identified a working group to consider a new guideline in the sports medicine handbook and to review the current handbook for coaches.

24. Playing Rules. The staff for playing rules administration provided an overview of the playing rules process and how the committee plays a role in consulting on athlete safety issues, primarily through injury surveillance data and review of rules committee recommended rules changes involving health and safety.

25. Pole Vault. The committee reviewed the outcomes of April 2011 meeting on pole vault safety and received a verbal update on the subsequent actions from the NCAA Track and Field Committee including permissive helmets as well as padding the perimeter and the vaulting box. The proposals on padding are pending further standards development by ASTM. A recently approved action included requiring that if a helmet is worn, it must meet the ASTM standard. Pole vault safety education continues as a priority for the NCAA, NFHS, USA Track and Field and is under development with the coaching community.

26. Ice Hockey. The committee reaffirmed its position that the current playing rules for men's and women's ice hockey requiring the full face protection provides the optimal protection from a health and safety perspective. The committee will track on continued discussions and participate in meetings involving the coaching community evaluating the option for wearing a half to three quarter face shield.

27. Football. Based on the committee's ongoing review of injuries and fatalities during NCAA sports activity, the committee recommends developing a best practice module for the reintroduction of Division I football players into conditioning and training sessions. The recommendation was in part based on these data points, relevant scientific research, experience from the 2003 football out of season and preseason model changes and input from the outcomes of the National Athletic Trainers' Association (NATA) inter-association task force meeting in January 2012. The NCAA will be a participant in the NATA inter-association task force.

28. National Operating Committee on Standards for Athletic Equipment (NOCSAE). The committee received a verbal report from Mike Oliver and Dave Halstead representing

NOCSAE on the current state of affairs in sports equipment standards including football helmets, soccer shin guards, baseball and softball fielder's helmet proposal, and ice hockey equipment. The committee also received a written response to an NCAA inquiry about the status of the current football helmet standard. The committee did not take action on these topics but will continue to monitor these standards and work with the playing rules committees on key athlete safety topics.

29. Fall Preseason Sport Models. The committee recommended further development of fall sport preseason best practices and potential regulatory options for sports other than football. Initially this process will begin with a focus on soccer through a multidisciplinary approach. The committee also recommended that a news story be published for the membership on preseason guidelines for fall sports other than football in late spring that outlines best practices. Even though football has legislated the preseason period in all three divisions, the lack of regulations surround student-athlete activity for other fall sports during the preseason have been a concern since 2003.

30. Mouth Guards. The committee reaffirmed its position that mouth guards should continue to be required for covering all upper teeth rather than allowing the option for a lower teeth only coverage in sports that mandate mouth guard use. Current sports requiring mouth guards include field hockey, football, ice hockey and lacrosse.

31. Sports Medicine Handbook. As it does on a biannual basis, the committee reviewed the guidelines in the sports medicine handbook. The committee agreed to continue to keep the sports medicine handbook guidelines as identified in the June 2011 meeting separate rather than combining them. The guidelines represent a quick topic specific reference for the membership on topics not otherwise readily accessible. The concussion guideline will continue to be reviewed and updated annually. A new guideline on psychological and mental health concerns is under development with a 2012-13 anticipated publication. The weight loss and dehydration guideline will be reviewed for consistency with current scientific findings. Guidelines with similar content will be reviewed and edited within the same year to ensure consistency.

32. 2011 Athletics Healthcare Survey. The committee received a report by Eric Hartung, NCAA research staff, on the results of the athletics healthcare survey. The purpose of this survey was to help the NCAA to better understand the state of athletics healthcare access within each NCAA division. The results of the study will help the NCAA make appropriate policy decisions in each division and identify best practices that institutions can

use to support the health and safety principle of the NCAA. More than half of NCAA member institutions responded. Ninety-six percent of institutions noted they have an emergency care plan on file yet responses varied by division as it related to who was certified in Cardio Pulmonary Resuscitation (CPR), first aid or Automated External Defibrillator (AED) trained. The variability could be attributed to the legislation that is in place or not in place across the divisions. Ninety-six percent noted they had a designated team physician or medical director authorized to oversee medical services for student-athletes while only 83 percent reported they had a physician identified to supervise certified athletic trainers. Ninety-six percent of institutions reported that their athletics department employed a certified athletic trainer (ATC); however, the number of full time equivalent ATC's varied across divisions (DI – 8.1, DII – 3.4, DIII – 2.8). Although often referred to as a benchmark, the NATA's Appropriate Medical Coverage for Intercollegiate Athletics calculated value was not reported by the institution in enough responses to be of value. Information related to access to campus health services, staff furloughs and common metrics were also recorded. No additional action was taken.

33. Championships Events. The committee discussed the use of NCAA Sports Medicine Handbook guidelines for event management purposes particularly involving heat, lightning and cold weather influences. The committee reaffirmed that due to the unique circumstances at each outdoor venue, guidelines should be applicable rather than creating set rules for modifying or canceling events. The committee suggested that during event planning, the host venue submit their emergency action plans for each site. In addition, the committee requested staff provide clarification on the current policy for medical credentials at various championships events.

34. Basketball camp certification. The committee deferred discussion surrounding health and safety topics involving certified basketball camps until the summer meeting pending additional clarification from NCAA staff.

35. Soccer headbands. The issue regarding the use of protective headgear for the prevention of concussion in sports such as soccer was also addressed by the committee. It was noted that there is still no clinical evidence that the use of such products has a protective effect regarding concussions. It was further noted that the design and recommended use of these devices fail to address the proposed mechanism of concussive injury, that being acceleration and deceleration forces acting on the brain. There is, therefore, little likelihood of these devices being shown to be beneficial. The committee could find no reason to recommend their use.

36. The committee discussed the following items but no actions were taken: soccer shin guards, baseball and softball fielders helmet NOCSAE proposed standard, ice hockey face protectors, lacrosse ball standards, and lacrosse headgear.

37. Future meeting dates and sites.

- June 12-14, 2012 - Indianapolis, Indiana.

Committee Chair: *Jeffrey Anderson, University of Connecticut*

Staff Liaison(s): *Mary Wilfert, Student-Athlete Affairs – Health and Safety*
David Klossner, Student-Athlete Affairs – Health and Safety
Latrice Sales, Student-Athlete Affairs – Health and Safety

DECEMBER 11-12, 2011	
Attendees	Absentees
Brant Berkstresser, Harvard University	Bob Colgate, National Federation of State High School Associations (NFHS)
Scot Dapp, Moravian College	Samantha Krueger, University of Wisconsin, Eau Claire
Michelle Gober, Kutztown University of Pennsylvania	Felix (Buddy) Savoie, Tulane University
Eric Hall, Elon University	Division II SAAC Representative
Matthew Horn, Winthrop University	
Melinda Larson, Whitworth University	
Kelsey Logan, The Ohio State University	
Jeff Martinez, University of Redlands	
Lois Mattice, California State University, Sacramento	
James Morgan, California State University, Chico	
Doug Padron, University of San Francisco	

Attendees	Absentees
Sourav Poddar, University of Colorado, Boulder	
Will Prewitt, Great American Conference	
Andrew Smith, Canisius College	
Christopher Ummer, Lyndon State College	
Scott Bennett, James Madison University (Ad HOC liaison)	
Connie Dillon, University of Oklahoma, (DI Sports Management Cabinet liaison)	
Other Participants	
Robyn Bailey, NCAA Staff	
Scott Bearby, NCAA Staff	
Nicole Bracken, NCAA Staff	
Jackie Campbell, NCAA Staff	
Ty Halpin, NCAA Staff	
Eric Hartung, NCAA Staff	
Brian Hendrickson, NCAA Staff	
Leah Kareti, NCAA Staff	
Dave Knopp, NCAA Staff	
Delise O'Meally, NCAA Staff	
Rachel Seewald, NCAA Staff	
Terrie Steeb, NCAA Staff	
Naima Stevenson, NCAA Staff	
Jill Corlette, Datalys	
Tom Dompier, Datalys	
Mark Bockelman, Drug Free Sport	
Frank Uryasz, Drug Free Sport	
Andrea Wickerham, Drug Free Sport	
Elliott Hopkins, NFHS	
Mike Oliver, NOCSAE	
Dave Halstead, Southern Impact Research Center	

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ACTION: Request from Division I and III for more research to back up the request for a recovery period between practices sessions relative to June December 2011 meeting recommendation:

Recommendation. To amend NCAA Division I Bylaw 17.9.2.4.1 to state that **student-athletes must be provided with at least three continuous hours of recovery time between any sessions occurring that day.**

17.9.2.3 **Five-Day Acclimatization Period.** FBS/FCS

(b) During the five-day period, participants shall not engage in more than one on-field practice per day, not to exceed three hours in length.

(1) Exception -- During the five-day acclimatization period, an institution has the option of conducting one on-field practice per day, not to exceed three hours in length, or one on-field testing session (speed, conditioning or agility tests) per day, not to exceed one hour in length and one on-field practice, not to exceed two hours in length. In addition, an institution may conduct one one-hour walk-through session per day of the acclimatization period, provided protective equipment (e.g., helmets, shoulder pads) is not worn, equipment related to football (e.g., footballs, blocking sleds) is not used and conditioning activities do not occur. Student-athletes must be provided with at least three continuous hours of recovery time between any sessions (testing, practice or walk-through) occurring that day. During this time, student-athletes may not attend any meetings or engage in other athletically related activities (e.g., weight lifting); however, time spent receiving medical treatment and eating meals may be included as part of the recovery time. If an institution conducts only a walk-through on a particular day during the acclimatization period, that day does not count toward the required five days of acclimatization, but the walk-through counts toward the limit of 29 on-field practices for the preseason practice period. *(Revised: 4/28/05 effective 8/1/05, 7/18/05, 12/13/05)*

17.9.2.4.1 **Exception -- "Walk-Throughs."** FBS/FCS

During the preseason practice period only and after the five-day acclimatization period, a walk-through is not considered to be an on-field activity under Bylaw 17.9.2.4, provided protective equipment (e.g., helmets, shoulder pads) is not worn, equipment related to football (e.g., footballs, blocking sleds) is not used and conditioning activities do not occur. If a walk-through is conducted on a day in which no other on-field activities occur, the walk-through must be included in the limit of 29 on-field practices for the preseason practice period. In championship subdivision football, on-field walk-throughs shall not exceed a total of two hours per day on the days one practice is conducted and one hour per day on the days multiple practice sessions are conducted. *(Adopted: 4/24/03 effective 5/1/03, Revised: 8/7/03, 12/13/05, 12/15/06)*

Notice the 3-hours of continuous recovery between sessions is missing in the Division I walk through legislation. Walk-through opportunities do extend beyond the 5-day acclimatization period.

Rationale for the recommendation. The committee reviewed the current permissible walk-through legislation in the sport of football and found inconsistencies across divisions. In the interest of providing consistent safeguards for student-athletes, the committee recommended that the three divisions evaluate this legislation and align the language with that of Division II, which states that student-athletes must be provided with at least three continuous hours of recovery time between any sessions occurring that day. In Division III, removal of the current exception should also be considered: "if the walk-through session occurs first, three hours of recovery time is not required." The impact of this legislation would ensure student-athletes have recovery from the cumulative effects of on-field activities, especially in the heat.

Additional Data:

- The five year period from 2005-2009 had the most deaths from heat stroke in high school/collegiate sports for any five year period over the previous 35 years.
- From 1988–2004, the National Collegiate Athletic Association (NCAA) Injury Surveillance Summary reported 1,687 EHI occurrences (resulting in time loss injuries for athletes) during fall football practices.

Recovery periods were part of the original 2003 football model. Dehydration has been shown to influence exercise performance. Participation in strenuous activities while incurring environmental stress with additional equipment and/or clothing has also been proven to increase the risk of EHI occurrence.

Recovery periods are a best practice cited in the NCAA Sports Medicine Handbook guideline "Preseason Preparation".

- An institution should ensure student-athletes have continuous recovery time (e.g., three hours) between multiple practice sessions on the same calendar day.

NCAA Funded Football Studies:

- J Strength Cond Res. 2006 Aug;20(3):463-70. Heat acclimatization and hydration status of American football players during initial summer workouts.
- Am J Sports Med. 2005 Jun;33(6):843-51. Epub 2005 Apr 12. Hydration status in college football players during consecutive days of twice-a-day preseason practices
- In Press at JAT 2012; a study of that collected 4 years of data from 2004-2007 from 60 distinct colleges and universities participating in Division I and III NCAA football:
    - "The first 14 days of the pre-season period had the highest rate of EHI with the greatest risk on practice days 1,2,3,7, and 10 with a gradual decline to day 14."
    - "high intensive practice that generally starts the football season places greater demands on the physiological system causing the higher EHI rates compared to other times in the season."

Other Reference Studies (there are 35 just in our new 2012 publication coming out):

- J Athl Train. 2006; 41(3): 332–336. Exertional Heat Illness and Environmental Conditions During a Single Football Season in the Southeast
- J Athl Train. 2010 Mar-Apr;45(2):117-27. The American football uniform: uncompensable heat stress and hyperthermic exhaustion.
    - Their results revealed that the addition of a full football uniform (helmet, shoulder pads and pants) or a partial uniform (pants only without shoulder pads or helmet) exhibited greater physiological strain which resulted in decreased exercise capacities relative to those who exercised without a uniform.
- J Athl Train. 2010 Mar-Apr;45(2):136-46. Thermoregulatory responses and hydration practices in heat-acclimatized adolescents during preseason high school football.
- Int J Sport Nutr Exerc Metab. 2005 Dec;15(6):641-52. Sweat and sodium losses in NCAA football players: a precursor to heat cramps?
- Med Sci Sports Exerc. 2003 May;35(5):832-7. Thermal insulation and evaporative resistance of football uniforms.
- National Athletic Trainers' Association Inter-Association Task Force on Exertional Heat Illnesses – Consensus Statement. http://www.nata.org/sites/default/files/inter-association-task-force-exertional-heat-illness.pdf .

DIVISION I

17.9.2.3 **Five-Day Acclimatization Period.** FBS/FCS

(b) During the five-day period, participants shall not engage in more than one on-field practice per day, not to exceed three hours in length.

(1) Exception -- During the five-day acclimatization period, an institution has the option of conducting one on-field practice per day, not to exceed three hours in length, or one on-field testing session (speed, conditioning or agility tests) per day, not to exceed one hour in length and one on-field practice, not to exceed two hours in length. In addition, an institution may conduct one one-hour walk-through session per day of the acclimatization period, provided protective equipment (e.g., helmets, shoulder pads) is not worn, equipment related to football (e.g., footballs, blocking sleds) is not used and conditioning activities do not occur. **Student-athletes must be provided with at least three continuous hours of recovery time between any sessions (testing, practice or walk-through) occurring that day.** During this time, student-athletes may not attend any meetings or engage in other athletically related activities (e.g., weight lifting); however, time spent receiving medical treatment and eating meals may be included as part of the recovery time. If an institution conducts only a walk-through on a particular day during the acclimatization period, that day does not count toward the required five days of acclimatization, but the walk-through counts toward the limit of 29 on-field practices for the preseason practice period. *(Revised: 4/28/05 effective 8/1/05, 7/18/05, 12/13/05)*

17.9.2.4.1 **Exception -- "Walk-Throughs."** FBS/FCS

During the preseason practice period only and after the five-day acclimatization period, a walk-through is not considered to be an on-field activity under Bylaw 17.9.2.4, provided protective equipment (e.g., helmets, shoulder pads) is not worn, equipment related to football (e.g., footballs, blocking sleds) is not used and conditioning activities do not occur. If a walk-through is conducted on a day in which no other on-field activities occur, the walk-through must be included in the limit of 29 on-field practices for the preseason practice period. In championship subdivision football, on-field walk-throughs shall not exceed a total of two hours per day on the days one practice is conducted and one hour per day on the days multiple practice sessions are conducted. *(Adopted: 4/24/03 effective 5/1/03, Revised: 8/7/03, 12/13/05, 12/15/06)*

DIVISION II

17.9.2.2 **Five-Day Acclimatization Period.**

(b) Participants shall not engage in more than one on-field practice per day during the five-day acclimatization period. On-field practices shall last no longer than three hours.

(1) **Exception** -- During the five-day acclimatization period, an institution has the option of conducting one on-field practice per day, not to exceed three hours in length, or one on-field testing session (speed, conditioning, or agility tests) per day, not to exceed one hour in length and one on-field practice, not to exceed two hours in length. In addition, an institution may conduct one one-hour walk-through session per day of the acclimatization period, provided protective equipment (e.g., helmets, shoulder pads) is not worn, equipment related to football (e.g., footballs, blocking sleds) is not used and conditioning activities do not occur**. Student-athletes must be provided with at least three continuous hours of recovery time between any session occurring that day.** During this time, student-athletes may not attend any meetings or engage in other athletically related activities (e.g., weight lifting); however, time spent receiving medical treatment and eating meals may be included as part of the recovery time. *(Revised: 1/9/06)*

17.9.2.3.1 **Exception -- "Walk-Through."**

During the preseason practice period, on-field walk-throughs are not considered an on-field activity under Bylaw 17.9.2.3, provided protective equipment (e.g., helmets, shoulder pads) is not worn, equipment related to football (e.g., footballs, blocking sleds) is not used and conditioning activities do not occur. The walk-through shall not last longer than one hour and may only be conducted on days in which one practice is permitted. **Further, student-athletes must be provided with at least three continuous hours of recovery time between the on-field practice and the walk-through.** During this recovery time, student-athletes may not attend any meetings or engage in other athletically related activities, including weight-lifting; however, time spent receiving medical treatment and eating meals may be included as part of the recovery time. *(Adopted: 1/12/04, Revised: 1/9/06 effective 8/1/06)*

DIVISION III

17.9.2.2 **Five-Day Acclimatization Period.**

(1) One on-field practice, not to exceed three hours in length. The institution may also conduct an one one-hour walk-through session. No protective equipment (e.g., helmet, shoulder pads) may be worn, no equipment related to football (e.g., football, blocking sled) may be used and conditioning activities may not occur during the walk-through session. **Student-athletes must be provided with at least three hours of continuous recovery time between the end of the on-field practice session and the start of the walk-through session (if the walk-through session occurs first, three hours of recovery time is not required);** or *(Revised; 8/10/11)*

(2) One on-field testing session (e.g., speed, conditioning or agility tests), not to exceed one hour in length, and one on-field practice, not to exceed two hours in length. The institution may also conduct an one one-hour walk-through session. No protective equipment (e.g., helmet, shoulder pads) may be worn, no equipment related to football (e.g., football, blocking sled) may be used and conditioning activities may not occur during the walk-through session. **Student-athletes must be provided with at least three hours of continuous recovery time between the end of each on-field session (e.g., testing session and on-field practice session) and the beginning of the next session (e.g., conditioning session, on-field practice, or walk-through session). Three hours of continuous recovery time is not required after the walk-through session.** *(Revised: 8/10/11)*

(c) During the remainder of the five-day period, participants shall not engage in more than one on-field practice, not to exceed three hours in length, and one one-hour walk-through session. No protective equipment (e.g., helmet, shoulder pads) may be worn, no equipment related to football (e.g., football, blocking sled) may be used and conditioning activities may not occur during the walk-through session. **Student-athletes must be provided with at least three hours of continuous recovery time between the end of the on-field practice session and the start of the walk-through session (if the walk-through session occurs first, three hours of recovery time is not required).** *(Adopted: 1/12/04, Revised: 1/16/10 effective 8/1/10, 7/26/11, 8/10/11)*

17.9.2.3.1 **Exception -- Walk-Through Sessions.**

During the preseason practice period only, on-field walk-through sessions are not considered an on-field activity under Bylaw 17.9.2.3, provided protective equipment (e.g., helmets, shoulder pads) is not worn, equipment related to football (e.g., footballs, blocking sleds) is not used and conditioning activities do not occur. Walk-through sessions may occur only on days during which a single on-field practice session is permitted and shall be limited to one hour in length. **Student-athletes must be provided with at least three hours of continuous recovery time between the end of the on-field practice session and the start of the walk-through session.** *(Adopted: 1/12/04, Revised: 1/16/10 effective 8/1/10)*

NCAA® GUIDELINE 1i

Preseason Preparation

July 2011

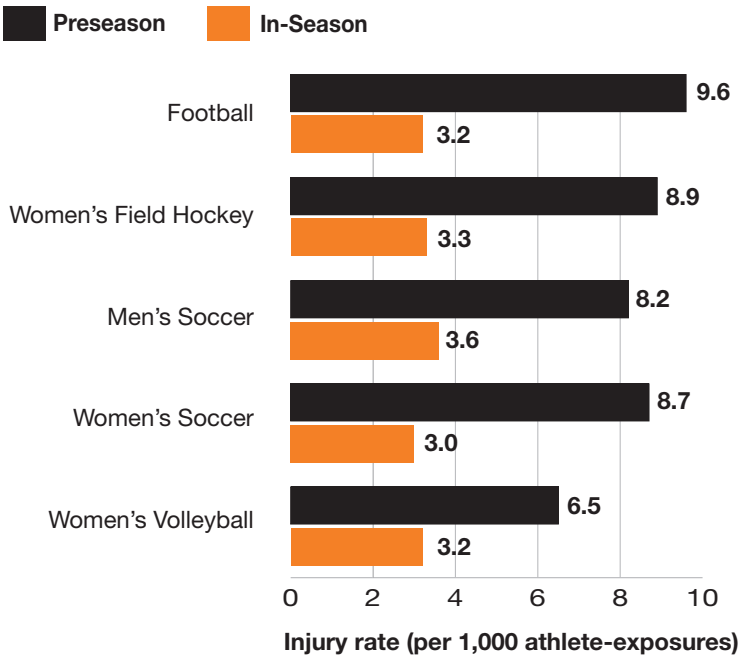
Athletic performance training is often divided into separate segments: preparation segment, competitive segment and offseason segment. The NCAA Sports Medicine Handbook Guideline 1a notes that the student-athlete should be protected from premature exposure to the full rigors of sports. Optimal readiness for the first practice and competition is often individualized to the student-athlete rather than a team as a whole. However, there is a lack of scientific evidence to set a specific number of days of sport practice that is needed for the first sport competition.

It is commonly accepted that student-athletes should participate in at least six to eight weeks of preseason conditioning. Gradual progression of type, frequency, intensity, recovery and duration of training should be the focus of the preparation segment. In addition to these areas warranted for progression, 10 to14 days are needed for heat acclimatization when applicable (see Guideline 2c). The fall sport preseason period is often challenging as August presents added heat risks for sports and there is a lack of time limits for practice activities (with the exception of football).

Changes to practice opportunities or the preseason period should be accompanied by an educational campaign for both coaches and student-athletes as to the expectations for the sport season. Specifically, student-athletes should know that the designated preseason practice period might be considered part of the competitive season and therefore a time when they may practice at contest-level intensities. A shortened preseason period based only on time spent on campus or coach expectations for contest-level intensities during the preparation period often increases the time spent practicing sport-specific skills without ample opportunity for preparatory conditioning exercises and can lead to injury and overtraining. If this is the expectation for the preparatory on-campus experience, athletes should be encouraged to improve fitness through a progressive training and conditioning program at least four weeks before starting the preseason segment.

The preparatory and preseason phases provide ample time to improve fitness and skill; however, performing novel exercise or actively doing too much too soon can result in a disparity between workload and load tolerance, thus increasing risk for injury. In addition, a student-athlete's psychological well-being can be

Practice Injury Rates for Fall Sports (2004-05 to 2008-09 NCAA Injury Surveillance)



Preseason Preparation

directly dependent on the level of fatigue driven by volume (quantity) and intensity of training. Similarly, the incidence in stress-related injuries (e.g., stress fractures, tendinitis) can be proportional to the work-rest ratio of the athlete.

Preparatory Phase. The following are general concepts to consider during the preparatory phase of training:

- Training should be periodized so that variation in the volume and intensity occurs in a scheduled manner.
- Plan recovery to allow for growth and development while avoiding acute and overtraining injuries.
- A proper heat acclimatization plan is essential to minimize the risk of exertional heat illness during the fall preseason practice period. Minimizing exertional heat illness risk requires gradually increasing athletes' exposure to the duration and intensity of physical activity and to the environment over a period of 10 to 14 days.
- Prolonged, near-maximal exertion should be avoided before acquired physical fitness and heat acclimatization are sufficient to support high-intensity, long duration exercise training or competition.

Fall Preseason Period. Institutions are encouraged to regularly review their preseason policies for fall sports and consider the following points of emphasis for protecting the health of and providing a safe environment for all student-athletes participating in preseason workout sessions.

- Before participation in any preseason-practice activities, all student-athletes should have completed the medical examination process administered by medical personnel (see Bylaw 17.1.5).
- Institutions should implement an appropriate rest and recovery plan that includes a hydration strategy.
- Preseason practice should begin with an acclimatization period for first-time participants, as well as continuing student-athletes.
- During the acclimatization period, an institution should conduct only one practice per calendar day.
- Practice sessions should have maximum time limits based on sport and individual needs, as well as environmental factors.
- An institution should ensure student-athletes have continuous recovery time (e.g., three hours) between multiple practice sessions on the same calendar day.
- Subsequent to the initial acclimatization period, an institution should consider a practice model that promotes recovery if practice sessions are to occur on consecutive days (e.g., two-one-two-one format).
- Student-athletes should be provided at least one recovery day per week on which no athletics-related activities are scheduled, similar to the regular playing season.
- Coaches are encouraged to consult with healthcare staff (e.g., athletic trainer) in the development of the conditioning sessions. All personnel should be aware of the impact of exercise intensity and duration, heat acclimatization, hydration, medications and drugs, existing medical conditions, nutritional supplements, and equipment on student-athletes' health while participating in strenuous workouts.
- Appropriate on-field personnel should review, practice and follow their venue emergency plan, as well as be trained in administering first aid, cardiopulmonary resuscitation (CPR) and AED use.

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Preseason Preparation

NCAA Football Preseason Model (see Bylaw 17).

The following concepts outline the legislation involving the NCAA football preseason period. Institutions should refer to division-specific legislation for exact requirements.

Five-Day Acclimatization Period.

In football, preseason practice begins with a five-day acclimatization period for both first-time participants (e.g., freshmen and transfers) and continuing student-athletes. All student-athletes, including walk-ons who arrive to preseason practice after the first day of practice, are required to undergo a five-day acclimatization period. The five-day acclimatization period should be conducted as follows:

(a) Before participation in any preseason practice activities, all prospects and student-athletes initially entering the intercollegiate athletics program shall be required to undergo a medical examination administered by a physician.

(b) During the five-day period, participants shall not engage in more than one on-field practice per day, not to exceed three hours in length.

(c) During the first two days of the acclimatization period, helmets shall be the only piece of protective equipment student-athletes may wear. During the third and fourth days of the acclimatization period, helmets and shoulder pads shall be the only pieces of protective equipment student-athletes may wear. During the final day of the five-day period and on any days thereafter, student-athletes may practice in full pads.

The remaining preseason practice period is conducted as follows:

(a) After the five-day period, institutions may practice in full pads. However, an institution may not conduct multiple on-field practice sessions (e.g., two-a-days or three-a-days) on consecutive days;

(b) Student-athletes shall not engage in more than three hours of on-field practice activities on those days during which one practice is permitted;

(c) Student-athletes shall not engage in more than five hours of on-field practice activities on those days during which more than one practice is permitted; and

(d) On days that institutions conduct multiple practice sessions, student-athletes must be provided with at least three continuous hours of recovery time between the end of the first practice and the start of the last practice that day. During this time, student-athletes may not attend any meetings or engage in other athletically related activities (e.g., weightlifting); however, time spent receiving medical treatment and eating meals may be included as part of the recovery time.

SUPPLEMENT NO. 5

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Division III Legislation Display - 4 Cites

17.12
Ice Hockey.

Previous Cite: 17.11.6 Out-of-Season Athletically Related Activities.

Next Cite: 17.12.1 Length of Playing Season.

Regulations for computing the ice hockey playing season are set forth in Bylaw 17.1, General Playing-Season Regulations. Institutions that conduct women's ice hockey shall comply with the same playing and practice season legislation that currently exists for men's ice hockey. (See Figure 17-1.) *(Revised: 1/9/96 effective 8/1/96)*

17.12.2
Preseason Practice.

Previous Cite: 17.12.1 Length of Playing Season.

Next Cite: 17.12.3 First Contest.

A member institution shall not commence practice sessions in ice hockey before October 15. *(Revised: 1/10/91 effective 8/1/91 and 8/1/92, 1/10/95 effective 8/1/95, 9/2/08, 1/17/09 effective 8/1/09)*

Change History

Date	Reason for change
01/17/2009	Proposal No. 2009-4

17.12.3
First Contest. Effective Date: August 1, 2012

Previous Cite: 17.12.3 First Contest.

Next Cite: 17.12.4 End of Playing Season.

A member institution shall not play its first contest (game, exhibition or scrimmage) against outside competition in ice hockey before October 15.

(1) Competitive Safeguards and Medical Aspects of Sports—Executive Regulations—Eligibility for Championships—Ineligibility for Use of Banned Drugs—Manipulation of Drug Test Sample.

Management Council. The Management Council reviewed a recommendation from the committee to sponsor noncontroversial legislation that would specify that a student-athlete who is involved in a case of clearly observed manipulation of an NCAA drug test shall be declared ineligible for further participation in postseason and regular-season competition during the time period ending two calendar years (730) days after the student-athlete was involved in the manipulation of a drug test.

The Management Council agreed to send the recommendation back to the committee for clarification of the term 'manipulation' versus 'masking.' The issue will be on the agenda at the July management Council/SAAC Summit as part of the legislative review.

The Management Council also agreed to establish a project team to study the issue of education and testing related to alcohol and drug usage in Division II. This project team will be comprised of members of the Management Council and NCAA affiliated associations and will undertake a comprehensive study of the relevant issues to determine where Division II should focus its future efforts.

Presidents Council. The Presidents Council received information on the formation of a project team without taking any formal action.

Title: EXECUTIVE REGULATIONS -- ELIGIBILITY FOR CHAMPIONSHIPS -- INELIGIBILITY FOR USE OF BANNED DRUGS -- MANIPULATION OF DRUG TEST SAMPLE

Convention Year: 2013

Effective Date: August 1, 2013

Official Notice Number: NC-2013-2

Source: NCAA Division II Management Council (Committee on Competitive Safeguards and Medical Aspects of Sports).

Category: Noncontroversial

Topical Area: Executive Regulations

Status: Ready for Consideration by Management Council

Intent: To specify that a student-athlete who is involved in a case of clearly observed manipulation of an NCAA drug test shall be declared ineligible for further participation in postseason and regular-season competition during the time period ending two calendar years (730 days) after the student-athlete was involved in the manipulation of a drug test.

A. Bylaws: Amend 18.4.1.5.1.1, as follows:

18.4.1.5.1.1 Breach of NCAA Drug-Testing Program Protocol. A student-athlete who is in breach of the NCAA drug-testing program protocol (e.g., no show~~*, tampering with sample*~~) shall be considered to have tested positive for the use of any drug other than a "street" drug.

18.4.1.5.1.1.1 Manipulation of Drug Test Sample. A student-athlete who is involved in a case of clearly observed manipulation of an NCAA drug test, as documented per NCAA drug-testing protocol by a drug-testing crew member, shall be declared ineligible for further participation in postseason and regular-season competition during the time period ending two calendar years (i.e., 730 days) after the student-athlete was involved in the manipulation of a drug test.

B. Administrative: Amend 31.2.3.1, as follows:

31.2.3.1 Breach of NCAA Drug-Testing Program Protocol. A student-athlete who is in breach of the NCAA drug-testing program protocol (e.g., no show~~*, tampering with sample*~~) shall be considered to have tested positive for the use of any drug other than a "street" drug.

31.2.3.1.1 Manipulation of Drug Test Sample. A student-athlete who is involved in a case of clearly observed manipulation of an NCAA drug test, as documented per NCAA drug-testing protocol by a drug-testing crew member, shall be declared ineligible for further participation in postseason and regular-season competition

during the time period ending two calendar years (i.e., 730 days) after the student-athlete was involved in the manipulation of a drug test.

Additional Information: The penalty for testing positive for use of a banned substance is a serious penalty, supported by the membership as a strong deterrent to banned drug use to protect the integrity of the game and student-athlete health and safety. The current penalty for breach of protocol (e.g., tampering with a sample) is the same as that for a positive test. A deliberate attempt by a student-athlete to alter his or her urine specimen (documented per NCAA drug-testing protocol by a drug-testing crew member), through the use of added substances or by substituting another's urine, demonstrates an egregious violation of ethical conduct. This behavior warrants an even more serious automatic penalty as a deterrent to such behavior and as a sanction when it occurs. As with all drug-testing penalties, there is an appeal process available to the institution and student-athlete.

Review History:

June 13, 2011: Recommends Approval - Committee on Competitive Safeguards and Medical Aspects of Sports

October 17, 2011: Approved in Concept - Management Council

Division I Bylaw Deregulation

Working groups were created after the August 2011 Presidential Retreat to affect needed change in the areas of student-athlete well-being; NCAA bylaws; the enforcement and penalty process; and fiscal sustainability in Division I.

The following concepts by the Collegiate Model – Rules working group are out for membership comment to deregulate current rules involving medical expenses, meals and snacks.

Concept No. 4: Modify the medical expenses legislation to permit the NCAA, institution or conference to provide medical expenses and services that are determined to be reasonable and appropriate for the health, safety and physical and mental well-being of the student-athlete.

Concept No. 7: Modify the meals legislation to permit an institution or conference to provide, as a benefit incidental to participation, food to student-athletes at any time or during specified time periods.

Bylaw 16 deregulation concepts will likely not include deregulation of 16.5.2.g, which is the restriction on what nutritional supplement products a school may provide to student-athletes.

REPORT OF THE
NCAA DIVISION I CHAMPIONSHIPS/SPORTS MANAGEMENT CABINET
FEBRUARY 15, 2012, MEETING

The NCAA Division I Championships/Sports Management Cabinet submits this report from its February 15, 2012, meeting.

***KEY ITEMS*.**

1. **Strategic Direction.** The NCAA Division I Championships/Sports Management Cabinet Administrative Committee discussed the need for the cabinet to operate on a more strategic level. It was agreed that the Administrative Committee would lead a discussion with the cabinet in June 2012 focusing on enhanced communication, strategic oversight of the groups reporting to the cabinet, best practices for operating in a more fluid governance structure, and strategies for the work of the cabinet and NCAA staff to be as transparent as possible. A discussion document will be shared with the cabinet in advance of the June 2012 meeting.

2. **Championships Financial Reporting.** Greg Shaheen, interim executive vice president for championships and alliances, presented an overview of financial data and other key measures of the championships that are being developed. When completed, this enhanced financial information will be shared with the cabinet, sports committees, the membership at-large and the general public to provide greater understanding, transparency and guidance on decision-making relative to the championships. The Administrative Committee reviewed examples of what this data might look like and provided feedback on the reports and data being developed. The information will be shared in the future to assist the cabinet with a deeper understanding of all aspects of the championships.

***ACTION ITEMS*.**

1. **Legislative Items**.

   - **None.**

2. **Nonlegislative Items.**

   - **None.**

INFORMATIONAL ITEMS.

1. **Approval of the Championships/Sports Management Cabinet Report from the September 13-14, 2011, meeting.** The cabinet report from the September 13-14, 2011, meeting was approved as previously distributed.

2. **Report from recent meetings of the Division I Leadership Council, Division I Board of Directors, Executive Committee and the championships and alliances staff.** The cabinet received the informational reports without taking action.

3. **Report of the NCAA Division I Championships/Sports Management Cabinet Administrative Committee.**

   a. **Update on revisions to the bowl certification process.** The cabinet received a summary of the current recommendations from the NCAA Bowl Certification Task Force. It was noted that the task force's initial recommendations are intended to strengthen bowl certification criteria in the areas of bowl governance, sponsorship/advertising, and student-athlete well-being. Additional work will be completed in the areas of postseason bowl eligibility, the "window" during which bowl games would occur, and auditing procedures. The task force will report to the NCAA Division I Board of Directors in April 2012 and an additional update will be provided to the cabinet in June 2012.

   b. **Division I budget process.** Mr. Shaheen and Kathleen McNeely, vice president for administrative services and chief financial officer provided an update on the status of budget requests and prioritization from cabinet. It was noted that the Association is moving to a three-year budget cycle from a two-year budget cycle. In addition, it was reported that the budget requests from the cabinet were advanced for consideration and that any final decisions would likely not occur until June 2012. Staff shared with the Administrative Committee work continues to develop more detailed financial information regarding each championship for more effective planning and resource allocation in the future. Finally, the cabinet reviewed the ongoing Presidential-level discussions regarding the NCAA Football Championship Subdivision (FCS). The cabinet emphasized the importance that prospective budget impacts include any costs associated with assuring equitable opportunities as a result of any adjustment. The cabinet will monitor the outcome of the ongoing discussions and stands ready to provide assistance as appropriate.

   c. **Update on work of presidential working groups impacting the cabinet.** The cabinet received a report on the work of the presidential working groups and

discussed the topics that will be of particular interest to the cabinet. It was noted that the cabinet would have an opportunity to provide significant feedback on the work related to playing and practice seasons and the cabinet expressed its desire to begin work in this area as soon as possible. Members of the cabinet were encouraged to provide feedback on all areas currently under review, including enforcement, through the NCAA's website and by participating in focus groups whenever possible.

(1) NCAA Working Group on Collegiate Model - Resource Allocation. The cabinet noted that in January 2012 the Board of Directors, in response to a recommendation from the Resource Allocation Working Group, adopted a resolution to institute an immediate moratorium to cap the number of contests/dates of competition at the levels that currently exist in all sports; to commission a study to determine the maximum number of contests/dates of competition that is essential to the success of each NCAA sport and examine how basketball contests are counted; and, once the study is completed and the appropriate contest/dates of competition limits are in place for each sport, to keep those limits in place for 10 years.

(2) NCAA Working Group on Collegiate Model - Enforcement. The cabinet received an update on the Enforcement Working Group's progress with creating a multi-level NCAA rules violation structure, an enhanced penalty structure for NCAA rules infractions and re-establishing a sense of shared responsibility among the interested individuals and entities in intercollegiate athletics. The cabinet also was informed of the Enforcement Working Group's desired outcomes, and timeline for developing and presenting its recommendations to the Board of Directors. Cabinet members were encouraged to provide feedback from their institutions and conferences and were informed of a new website, accessible to the membership via ncaa.org, that contains information about the work of the Enforcement Working Group.

d. **Administrative Policies.** The staff provided the Administrative Committee with an overview of championship administrative policies that govern deadlines for submission of regular-season results and championships entries, as well as, the corresponding fines structure by sport that is in place for breach of these policies. The Administrative Committee directed the staff to continue its work in refining these policies with the goal of achieving greater consistency across all championships and maintaining the Administrative Committee's preference of holding student-athletes harmless for the negligence of institutions. Furthermore, the Administrative Committee requested that recommendations for adjustment be provided to the full cabinet for review during the June 2012 meeting.

4. **Report of the NCAA Committee on Competitive Safeguards and Medical Aspects of Sport (CSMAS).** The cabinet reviewed the CSMAS's request to support, as noncontroversial legislation, an amendment to NCAA Division I Bylaw 17.9.2.4.1 (exception – "walk-throughs") to align the language with that of NCAA Division II, which states that student-athletes must be provided with at least three continuous hours of recovery time between any sessions occurring that day. The cabinet noted there could be circumstances (e.g., weather, facility scheduling, etc.) that warrant allowing fewer than three continuous hours of recovery time between any sessions occurring that day, and referred the issue back to the CSMAS. The cabinet asked the CSMAS to provide statistical or other information demonstrating a need for requiring three continuous hours of recovery time between any sessions occurring that day. The cabinet will review the information during its June 2012 meeting.

5. **Men's soccer officiating selection/assigning.** Staff provided the cabinet with an update on the review that is being conducted related to in-season and post-season men's soccer officiating selections and assignments. It was noted that the group selected to assist the NCAA with conducting this review was comprised of conference administrators who oversee the sport of men's soccer. It was noted that the group's discussions have focused on the following areas: increasing conference office involvement in the overall process; determining an established minimum number of Division I men's games required for an official to be eligible to work in the NCAA tournament; determing that experience as a referee and as an assistant referee should be considered separately; consideration of a waiver provision in emergency or extenuating circumstances such as geographic location of teams; and increasing communication between NCAA regional assignors and conference assignors. Final recommendations will be provided to the cabinet during its June 2012 meeting.

6. **Championships reimbursement and per diem.** The cabinet was provided a financial analysis of the costs associated with reimbursement for qualifying/preliminary rounds of individual/team sports that currently are not covered by the Association's travel guarantee. Further discussion will occur at the cabinet's June 2012 meeting in conjunction with a comprehensive financial review of all NCAA Division I championships.

7. **Modification to misconduct appeals.** Staff provided the cabinet with an update on current internal work being conducted to develop a more consistent approach along with a revised process for handling misconduct cases and appeals of those cases, and the review of failure to adhere to policies and procedures. The internal group anticipates forwarding their recommendations to the Administrative Committee and ultimately the full cabinet for review during the June 2012 meeting.

8. Eligibility for Postseason Competition. The cabinet received an informational report from staff related to changes to the NCAA Division I Academic Performance Program. No action was taken by the cabinet.

9. Reports of Sports and Sport Issues Committees.

a. NCAA Division I Baseball Committee. The cabinet received an informational report from the NCAA Division I Baseball Committee without taking action.

b. NCAA Division I Softball Committee. The cabinet reviewed a request from the NCAA Division I Softball Committee to no longer consider the Sotball Committee's previously approved super-regional budget increase ($45,000) as one of the cabinet's top priorities but rather to substitute an increase in the travel party size for the Women's College World Series ($57,000). The cabinet agreed to defer action until its June 2012 meeting.

c. NCAA Division I Volleyball Committee. The cabinet received an informational report related to postseason officiating requirements from the NCAA Division I Volleyball Committee without taking action.

d. NCAA Division I Women's Basketball Issues Committee. The cabinet received an informational report from the NCAA Division I Women's Basketball Issues Committee without taking action.

e. NCAA Men's and Women's Swimming and Diving Committee. The cabinet approved a modification to the tie-breaking procedure effective with the 2012 championship as follows: "When two swimmers are tied for the final selection spot, the NCAA Men's and Women's Swimming and Diving Committee will use the swimmers' times in the next event in which each swimmer is highest on the list and therefore closest to being selected in that event. The Men's and Women's Swimming and Diving Committee will compute the percentage of each respective swimmer's time in his or her next-best event to the automatic qualification time, or A standard for the event. The competitor who is closest in percentage to the A standard time for their next-best event will fill the final slot."

10. Playing Rules Oversight Panel. The cabinet received an informational report from the NCAA Playing Rules Oversight Panel without taking action.

11. Olympic Sport Liaison Committee. The cabinet received an informational report from the NCAA Olympic Sports Liaison Committee without taking action.

12. NCAA Division I Student-Athlete Advisory Committee. The cabinet received an informational report from student-athlete Logan Roberts, Baylor University.

13. Committee Chair Appointments. The cabinet took the following action related to committee chairs:

- **NCAA Division I Women's Basketball Committee.** The cabinet appointed Carolayne Henry, associate commissioner of the Mountain West Conference, chair effective September 1, 2012.

14. Future meeting dates and sites.

a. June 20, 2012; Hyatt Regency, Indianapolis.

b. September 11-12, 2012; national office, Indianapolis.

c. February 12-13, 2013; national office, Indianapolis.

d. June 11-12, 2013; national office, Indianapolis.

e. September 10-11, 2013; national office, Indianapolis.

Committee Chair: *Judy Rose, University of North Carolina, Charlotte*
Staff Liaisons: *Joni Comstock, Championships*
David W. Schnase, Academic and Membership Affairs

Attendees	Absentees
Shelley Appelbaum, Michigan State University	Raynoid Dedeaux, Mid-Eastern Athletic Conference
Barbara Burke, Eastern Illinois University	
Greg Burke, Northwestern State University	
Doze Y. Butler, Southern University, Baton Rouge	
Kenneth Casavant, Washington State University	
Carrie Coll, Mountain West Conference	
Connie Dillon, University of Oklahoma	
Tom Douple, The Summit League	
Rich Ensor, Metro Atlantic Athletic Conference	
Jim Fallis, Northern Arizona University	
James D. Fiore, Stony Brook University	
Richard Greenspan, Rice University	
Rick Hart, University of Tennessee at Chattanooga	
Tom Hickman, Winthrop University	
Ken Kavanagh, Florida Gulf Coast University	
Robert S. Krimmel, Saint Francis University (Pennsylvania)	
Scott Leykam, West Coast Conference	
Jeffrey Long, University of Arkansas, Fayetteville	
Warde Manuel, University at Buffalo, the State University of New York	
Erin McDermott, Princeton University	
Jane Miller, University of Virginia	
Mario Moccia, Southern Illinois University at Carbondale	
Marilyn Moniz-Kaho'ohanohano, University of Hawaii, Mona	
Lee Reed, Georgetown University	
Logan Roberts, student-athlete from the University of North Carolina, Chapel Hill	
Judy Rose, University of North Carolina, Charlotte	
Jim Schmidt, University of Illinois, Chicago	
Camden Wood Selig, Old Dominion University	
Rosemary A. Shea, College of Holy Cross	
Paula Smith, University of California, Irvine	
Rick Villarreal, University of North Texas	
NCAA Participants Jackie Campbell, Kelsey Cermak, Diane Dickman, D'Ann Keller, Kathleen McNeely, Roberta Page, Carol Reep, Kris Richardson, Julie Roe, Greg Shaheen, Juanita Sheely	

Ivy League

Practice limitations adopted July 2011 with respect to the sport of football to reduce the amount of "helmet or head hit" exposures

1. Pre-Season: Limiting to one the number of days that pads can be worn during both sessions of two-a-day practices
2. In-Season: Limit of two full contact days per week
    1. NCAA allows a maximum of 5
3. Spring: Reduction by one in the number of allowable full contact practices
    1. 4 out of possible 12 practices must be non-contact
    2. NCAA 3 out of a possible 15 practices must be non-contact

NFL Collective Bargaining Agreement:

1. Offseason Program
    a. Nine week program over a twelve week period for teams with a new head coach and 10 weeks with a returning head coach
        i. Required to be 8 consecutive weeks
        ii. 4 days per week only
        iii. No weekends
        iv. 3 phases to offseason program-may not exceed 90 minutes on the field for all phases
    b. Phase 1
        i. First two weeks of offseason program
        ii. Limited to strength and conditioning coaches only
        iii. No footballs with exception of QB may throw to WR
        iv. No helmets
    c. Phase 2
        i. Weeks 3-5 of offseason program
        ii. Coaches allowed on field
        iii. Drills may include individual instruction as well as "perfect play" drills
        iv. No offensive vs. defensive team drills or special team drills
        v. No "one on one" OL vs. DL drills, WR vs. DB drills
        vi. No helmets
    d. Phase 3
        i. Weeks 6-9 of offseason program
        ii. 10 days of "Organized Team Activities" (OTA)permitted
        iii. Maximum of 3 days of OTA's during 1st two weeks of Phase 3
        iv. Maximum of 4 days of OTA's either 3rd or 4th week of Phase 3 with Mandatory Veterans Minicamp to be held during the other week

v. During weeks of 3 OTA's a 4th day of activity is permitted but must follow rules of Phase 2
vi. No live contact is permitted
vii. No "one on one" offensive vs. defensive drills are permitted-no OL vs. DL drills, WR vs. DB drills
viii. Special team drills are permitted without contact
ix. Helmets are allowed-No shoulder pads

e. Mini-Camp
i. Maximum of one mandatory mini-camp for veteran players with the exception of new head coach may have an additional week
ii. Must be 3 consecutive days (T-TH)
iii. Must be conducted week 3 or 4 of Phase 3 offseason program
iv. Two-a-Day practices are permitted at 2 of the 3 practice days
1. Players cannot exceed 3 ½ hours total on field
2. 1st practice cannot exceed 2 ½ hours
3. 2nd practice must be walk through

2. Pre-Season Training Camp
a. Practice Days one and two must be no pads with one practice only
b. May begin two-a-day practices on practice day three
i. No more than 4 hours per day total practice time
ii. One padded practice permitted no longer than 3 hours
iii. Second practice-no helmet or pads, walk through only
iv. Minimum 3 hours between practices
c. One day off every seven days

3. Regular Season Practices
a. Limited to 14 total padded practices
i. Eleven must take place during 1st eleven weeks of season
ii. 3 must be held during final 6 weeks of season
iii. Club may hold two padded practices during same week provided such week falls within 1st 11 weeks of season
iv. Padded practices may not exceed 3 hours

4. Post Season Practices
a. May conduct only one padded practice per week

Report regarding the Ivy League Review of Concussions In Football

(Released July 2011)

I. Introduction

The Ivy League Council of Presidents in December 2010 agreed to form an ad hoc Concussion Committee (see Attachment A for the Committee roster) to discuss how the Ivy League could assume a leadership role in trying to limit concussive hits, including the evaluation of the playing rules for Ivy League football games and policies regarding practices. The Council also charged the committee with considering whether a similar review should be conducted in any other sports. This report focuses exclusively on football, other than Section V, which addresses the sports that the Ivy League should consider reviewing in the future.

A "Core Group", consisting of the majority of the Committee, met for a full-day in person and by conference call to discuss and develop the recommendations contained in this report. The full Concussion Committee accepted this report of the Core Group, including all of the recommendations.

Consistent with the Council's charge, the Core Group focused on the following goals:

- To develop specific recommendations regarding football practices and games aimed at trying to limit concussive hits or repetitive subconcussive hits
- To evaluate the playing rules for Ivy League football games and policies regarding practices
- To provide guidance for developing a framework for postgame ramifications (e.g., suspensions, other actions) for illegal hits or actions
- To make recommendations regarding enhancing existing educational resources
- To assess current Ivy League medical staff policies regarding concussion and make recommendations regarding student welfare after concussive injury
- To assess other methods for reducing concussive injury (e.g., using improved recognition and detection as a preventative tool)

<u>Executive Summary of Recommendations and Conclusions</u>

The Core Group recognizes that concussion is a significant injury in football, with not only acute consequences, but also more long-term sequelae. In addition, the multiple hits that are sustained in football, distinct from those causing a concussion, may have a role in the development of Chronic Traumatic Encephalopathy (CTE) in some individuals. Education can play an essential role in encouraging the reporting of injuries, early recognition and treatment of concussion, as well as a thorough evaluation of the concussed athlete, all of which may prevent severe injury. It is also important to evaluate the culture of football and ensure the welfare of student-athletes is emphasized, including recognition that there can

be great harm caused by playing with a concussion, respect for other players' health and safety, and encouragement of safe behaviors on the field.

After reviewing and discussing data and research regarding concussions and head hits in football, and current NCAA and Ivy League rules and practices, the Group determined that while even one serious head or helmet hit can have long-term repercussions, the following context is important in the consideration of these important issues. There are over 40 games annually involving Ivy League teams, with the number of plays exceeding 6000 each year and with very few plays involving penalties for hits to the head. For example, in 2009 out of 6355 plays, five involved penalties for helmet hits or blows to the head and in 2010 out of 6665 plays, there were eight such plays. In 2010 there were also four plays involving penalties for hitting a defenseless player ("targeting"). It thus appears, based on the available data, that the number of plays involving significant helmet hits or hits to the head is approximately 0.18%, and well under one per game (less than one out of every three games would on average have such a hit).

Despite these low numbers for Ivy League football, the Group recognizes the concern about potential long-term sequelae and complications from not only recurrent and/or severe injury, but also from cumulative head trauma (sub-concussive hits). Given the lack of data regarding how many or the type of hits that may cause long-term consequences in certain individuals, it is the Group's conclusion that it is important to minimize the likelihood and severity of hits to the head. The Group thus focused its recommendations on limiting contacts and tackling in practices in all three segments of the season (preseason, in-season and spring), and on education (continued teaching of proper technique and information regarding concussions). A key component of this effort is continuing to change the mentality of student-athletes regarding the seriousness of concussive injuries.

In terms of the current medical assessments and return-to-play decision making that is occurring at Ivy League schools, the Group believes that all are compliant with and/or exceed the 2010 NCAA requirements and "best practices". Further, in accordance with NCAA rules, any athlete that is diagnosed with a concussion cannot return to activity that day and may not return in the future until cleared by a medical professional. One area that the Group, particularly the medical staff (e.g., team physicians, athletics trainers), highlighted as extremely critical to student-athlete welfare is the importance of academic accommodations that are often necessary after concussive injury. Cognitive rest, along with physical rest, is an essential aspect of the recovery process.

As this remains an evolving area, the Core Group recommends that the Ivy League continue to monitor the research, data, best practices and other information regarding concussions and cumulative head trauma (e.g., short-term and long-term effects, causes of long-term consequences, diagnosis of permanent injury) to determine if these recommendations need to be revisited in the future.

The following is an Executive Summary of the Group's recommendations:

Practice Limitations

The following recommended practice limitations are intended to reduce the overall number of opportunities for helmet or head hits to occur, beyond the current NCAA and Ivy League restrictions. Further, these new limitations, some of which codify existing Ivy practice philosophies, will be included in Ivy legislation, thereby requiring future expansion regarding full-contact during practices to be approved through the Ivy legislative process.

- In-Season – Limit of two full-contact days per week (a 60% reduction from the NCAA limit).

- Spring Practice – Reduction by one in the number of allowable full-contact practices in the spring (resulting in a 12% reduction from the current Ivy League limit and a 42% reduction from the NCAA limit).

- Preseason – New rule limiting to one the number of days that pads can be worn during both sessions of two-a-days during the preseason.

- Greater time and attention devoted to the teaching of and emphasis on proper techniques for avoiding helmet hits.

- Continued adjustment to practice plans when depth at a given position is low to reduce the number of hits experienced by a particular player.

- Adherence to recommended guidelines for in-season full-pad, contact practice sessions.

Games

- Officials will be directed to continue erring on the side of caution by calling penalties if a helmet or head hit might have occurred, which will in turn allow the Ivy League office to assess and determine any appropriate postgame ramifications.

Education

- Continue providing educational information to student-athletes summarizing the signs and symptoms of a concussion, and emphasizing the potential long-term risks of repetitive brain trauma and the need to report and not play with any symptoms of a concussion.

- Practices will continue to include and will emphasize and enhance the teaching of proper football fundamentals and technique to avoid leading with the head, including a de-emphasis of hits on defenseless players.

- Teams should view, at least annually, educational videos regarding concussion in sports (e.g., existing NFL and NCAA videos) and videos of specific examples of legal and illegal football hits provided by the NCAA.

- Each team should conduct an additional in-person, annual educational session aimed at connecting directly with the student-athletes by involving individuals personally knowledgeable about concussions (e.g., former student-athletes, researchers, athletics trainers, team physicians).

- The annual officials clinic conducted by the Ivy League Coordinator of Football Officials will include a session on concussion information.

- The Group is interested in developing educational materials or information for high school and youth coaches, but recognizes the need to determine how to realistically achieve this goal, in a practical and cost-effective manner.

Diagnosing Injury as a Preventative Tool

- Educational programs geared towards athletes and coaches are helpful in encouraging student-athletes to report symptoms to their medical staff, thereby potentially reducing the risk of a more severe injury.

- Improving the recognition of injury and the evaluation process to accurately diagnose concussions may improve outcomes. An evaluation that includes a focused neurologic exam, which incorporates a cognitive evaluation (e.g. orientation, concentration, memory), symptoms, and a balance evaluation is important (and is considered "best practices" by the NCAA). Other diagnostic tools may also prove to be effective for evaluating and diagnosing concussion. Additional study and research is needed regarding diagnostic tools.

Recovery Process

- Given the importance of avoiding cognitive work during the healing process after diagnosis of a concussion, it is recommended that the Ivy League Presidents communicate with the appropriate deans on each campus to ensure that they are aware of this serious and critical part of the recovery process and of the need to make appropriate academic accommodations for the timeframe specified by the physician for student-athletes diagnosed with concussions.

- In response to the Council's December 2010 request, the Ivy League athletics trainers and team physicians have developed specific Return-To-Play Guidelines for use after a concussion diagnosis, incorporating NCAA best practices and supported by all eight schools.

Other Sports

- The Core Group recommends that the Ivy League consider what changes or recommendations might be necessary to address the risk of concussions in the following sports (in priority order): Men's and Women's Ice Hockey, Men's and Women's Lacrosse, and Men's and Women's Soccer.

- In addition, after completion of the review for these sports, the Ivy League should consider whether other sports should be reviewed.

II. Background Information

A. Review of Research

Among the information the Core Group reviewed, and of particular note, was data from the Brown, Dartmouth, and Virginia Tech studies using Head Impact Telemetry (HIT) system technology to quantify the frequency, location and severity of head impacts collegiate football players experience in practice and game sessions, and by player position. In brief, the studies reported the following key findings:[1] [2]

- Players received up to 1444 head impacts in one season, with an average of 6.3 impacts per practice and 14.3 impacts per game.

- Impact frequency and location differed among player positions, with linemen and linebackers having the largest numbers of impacts per practice and per game.

- The offensive linemen had the largest percentage of impacts to the front of the helmet.

- Most impacts occurred to the front of the helmet for all player positions except for quarterbacks, who received the most impacts to the back of the helmet.

- Player position and impact location were the largest factors associated with differences in head impact severities. Running backs consistently sustained the greatest impact magnitudes.

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[1] Crisco JJ, Fiore R, Beckwith JG, Chu JJ, Brolinson PG, Duma S, McAllister TW, Duhaime AC, Greenwald RM. Frequency and Location of Head Impact Exposures in Individual Collegiate Football Players. *J of Athletic Training.* 2010;45(6):549-559.
[2] Crisco JJ, Fiore R, Wilcox BJ, Machan JT, McAllister TW, Duhaime AC, Duma S, Rowson S, Beckwith JG, Chu JJ, Greenwald RM. Magnitude of Head Impact Exposures in Individual Collegiate Football Players. *J Applied Biomechanics.* [Accepted for publishing.]

- Peak linear accelerations were greatest for impacts to the top of the helmet, whereas rotational accelerations were greatest for impacts to the front and back of the helmet.

- Head impact severity measures were generally not different for games compared to practices.

Interestingly, another study using accelerometers in collegiate football players' helmets found that the impacts sustained during helmet-only practices were greater than those sustained during games.[3] Another study, which used instrumented helmets in youth hockey players, suggested that head impact severity decreases when players anticipate the impending contact, particularly with head impacts of moderate severity (moderate impacts that were anticipated resulted in greater differences in rotational acceleration than high or low impact anticipated hits, thus possibly reducing the risk of injury).[4]

Although these studies are helpful in beginning to quantify the risks from helmet or head impacts in football, the data do not reveal when short-term injury or long-term consequences are likely to occur. There currently is insufficient available data regarding the threshold limits as to when long-term consequences will or are likely to occur, including the number of concussive and/or subconcussive hits, the severity of the hit(s), the location of the hits, the total trauma experienced per player (youth through college and professional), and individual predisposition to develop long-term consequences.[5] In addition, the risk of sustaining a concussive injury may depend not only on a threshold to a single impact but also on an individual's impact history, which is currently difficult, if not impossible, to measure. It is difficult to know the number, severity or extent of hits athletes sustain prior to college. Further, experts intuitively believe that rotational accelerations, when not anticipated, have greater risk of injury. (Accordingly, NCAA football playing rules incorporate and emphasize protecting defenseless players.) Further study and research is needed in these areas.

The Core Group also received information regarding the available research of actual cases of diagnosed Chronic Traumatic Encephalopathy (CTE), based on autopsies the Boston University Center for the Study of Traumatic Encephalopathy conducted

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[3] Mihalik JP, Bell DR, Marshall SW, Guskiewicz KM. Measurement of Head Impacts in Collegiate Football Players: An Investigation of Positional and Event-Type Differences. *Neurosurgery.* 2007 Dec;61(6):1229-35.
[4] Mihalik JP, Blackburn JT, Greenwald RM, Cantu RC, Marshall SW, Guskiewicz KM. Collision Type and Player Anticipation Affect Head Impact Severity Among Youth Ice Hockey Players. *Pediatrics*. 2010 May;125(6):1394-1401.
[5] There is some preliminary research through the use of impact data from instrumented helmets that indicates the probability of concussion may reach 50% at a certain linear impact.

of 30 former NCAA and NFL players (29 of the 30 former players were diagnosed with CTE). Cumulative trauma, not necessarily concussion, is felt to be the major risk factor for CTE. Accordingly, the Core Group agreed that, despite the lack of specific data and research regarding the causes of CTE, the Ivy League should carefully consider the potential long-term effects of multiple hits in football and make appropriate changes to reduce the number of hits and to enhance the welfare of football student-athletes.

B. Summary of Current NCAA Policies regarding Concussion Management in Football

In April 2010, the NCAA suggested best practices for concussion management and adopted a policy requiring all institutions to have a concussion management plan on file that mandates removing from practice or competition a student-athlete who exhibits signs, symptoms or behaviors consistent with a concussion. The NCAA policy specifies that student-athletes may not return to activity the same day as being diagnosed with a concussion. Representatives of Ivy League team physicians and head athletics trainers agree that the NCAA's requirements are reasonable and appropriate standards and that the recommended best practices go further than prior NCAA guidance and are also reasonable and appropriate. [Note: In June 2010, the Ivy League Council of Presidents agreed to accept the NCAA's actions as sufficient and to continue to monitor any new research and guidelines to determine whether there should be any changes to League policies regarding concussion management among student-athletes.]

The NCAA has also required since the 2009 football season that conference offices conduct a video review of football plays during which a foul is called against a player for initiating contact above the shoulders or targeting an opponent, including helmet-to-helmet hits, when the player was not ejected from the game. The conference is charged with considering and assessing possible sanctions before the next game. The Ivy League office, in consultation with the League's Coordinator of Football Officials, has reviewed a handful of such plays during each of the past two seasons, with a few reprimands issued, but no other action deemed necessary at that time. [Note: In December 2010, the Council of Presidents specified that the Executive Director, in consultation with the League's Coordinator of Football Officials, should take appropriate but firm action in response to future helmet hits, including suspension for intentional hits.]

In addition, the NCAA playing rules for football include penalties for initiating and targeting an opponent with the top of the helmet, and for initiating contact with and targeting defenseless players above the shoulders. Also under the NCAA playing rules, intentional helmet-to-helmet contact is never legal and flagrant offenders shall be disqualified. These areas are also a "point of emphasis". (See Attachment B for a summary of relevant NCAA playing rules in football.)

III. Ivy League Current Policies and Customs

A. League Office Review of Plays

In accordance with the NCAA policy regarding conference review of plays involving a foul against a player for initiating contact above the shoulders or targeting an opponent, including helmet-to-helmet hits, the Ivy League Executive Director, Associate Executive Director for Championships and Sports Administration, and the Coordinator of Football Officials have conducted such reviews the past two seasons. In 2009, there were a total of five penalties called for: targeting a defenseless receiver (0); butting/ramming with the helmet (3); and blow to the head (2). The Ivy League office reviewed two plays involving helmet-to-helmet or targeted hits. In 2010, there were a total of 12 such penalties called for: targeting a defenseless receiver (4); butting/ramming with the helmet (5); and blow to the head (3). The number of plays the Ivy office reviewed in 2010 increased to seven in large part due to the increased emphasis on officials erring on the side of caution by calling fouls even when it is not certain that helmet contact occurred. Upon further review and study of the video, in slow motion several times, several of the fouls in both seasons were determined to not involve head contact.

B. New Policy regarding Postgame Ramifications

During the 2009 and 2010 football seasons, helmet-to-helmet or targeted hits resulted in an email reprimand from the Executive Director to the head coach, with copies to the athletics director and the opposing Ivy League school's head coach and athletics director, with a reminder that the football coaches should continue to review and emphasize the importance of player safety, particularly regarding helmet hits.

Per the Council's December 2010 direction, beginning with the 2011 season, the Ivy League Executive Director will take appropriate but firm action in response to helmet hits, including suspension for intentional hits. The Executive Director will determine the appropriate penalty in consultation with other individuals as deemed appropriate, including the Ivy League Coordinator of Football Officials, the Ivy League Associate Executive Director for Championships and Sports Administration, the Officiating Observer in attendance at the game and the NCAA Coordinator of Football Rules, and upon consideration of relevant factors, including the following:

- Intent of the player(s)/Whether the hit was accidental;
- Severity of the hit;
- Injury likelihood/Dangerousness of the play; and
- Actions immediately following the play, if applicable.

Additional or modified policies may be developed as deemed appropriate. In addition, the League office will track incidents from year to year to identify any trends involving a particular team or player.

C. Number of Plays Involving Significant Head Hits is a Small Percentage of Total Plays

Although there are more than 40 Ivy League games each year, with the number of plays exceeding 6000 annually, very few plays involve hits to the head. For example, in 2009 out of 6355 plays, five involved penalties for helmet hits or blows to the head and in 2010 out of 6665 plays, there were eight such plays. In 2010 there were also four plays involving penalties for hitting a defenseless player ("targeting"). It thus appears, based on the available data, that the percentage of plays involving significant helmet hits or hits to the head is less than one percent (0.18%) and that such hits occur on average approximately once in every three games.

IV. Committee Recommendations

The Core Group's recommendations are divided into five areas: practice limitations, games, education, diagnosis as a preventative measure and the recovery process.

A. Practice Limitations

Existing Ivy League rules, which reduce the number of permissible practices otherwise allowed under NCAA limits, already provide for a reduction in the number of opportunities for helmet or head hits to occur as compared to other NCAA Division I teams. In addition, a reduction in hitting during practices has happened naturally over the years as coaches, at least in the Ivy League, have generally been modifying practice plans to reduce hitting to keep players healthy. Existing routines for full-pad, contact practice sessions minimize the emphasis and incidence of tackling.

The recommended practice limitations are intended to reduce the overall number of opportunities for helmet or head hits to occur, beyond the current NCAA and Ivy League restrictions already in place. Further, these new limitations, some of which codify existing Ivy practice philosophies, will be included in Ivy legislation, thereby requiring future expansion regarding full-contact during practices to be approved through the Ivy legislative process.

The Core Group recommends the following additional restrictions for football practices:

1. **In-Season – Limit of two full-contact days per week.** The number of full-contact practice days will be limited to two per week during the

season. During the remaining days there may be no full-contact practices or live tackles and no player may be "taken to the ground." There will be no full pads but helmets, uppers, girdles and thigh pads will be permissible for safety reasons. No kneepads will be allowed. This represents a 60% reduction from the NCAA limit of five days per week.

2. **Spring Practice – Reduce by one the number of allowable full-contact practices in the spring.** Four spring practices (instead of the current Ivy League limit of three) must not involve full contact or live tackles and no player may be "taken to the ground." There will be no full pads but helmets, uppers, girdles and thigh pads will be permissible for safety reasons. No kneepads will be allowed. This is a 12% reduction in practices involving contact from the current Ivy League limit (a reduction from eight to seven full-contact practices) and results in a 42% reduction in the number of contact practices allowed under NCAA rules.

3. **Preseason – Limit regarding two-a-days.** There will be a new rule limiting to one the number of days that pads can be worn during both sessions of two-a-days during the preseason.

4. **Emphasize teaching proper technique.** The head football coaches agreed to spend more time during practices emphasizing and teaching proper techniques for avoiding helmet hits. They will also annually show to their teams instructive videos of permissible and non-permissible hits.

5. **Adjust practice plans when depth is low.** Coaches will continue to be sensitive to the need for adjusting practice plans when depth at a given position is low (e.g., due to injury or other absences) to reduce the number of hits experienced by a particular player.

6. **Adherence to the recommended guidelines for in-season full-pad, contact practice sessions.** Coaches will follow the recommended guidelines for in-season full-pad, contact practice sessions specified in Attachment C.

The following chart summarizes the reduction in the number of opportunities for helmet hits during practice sessions as a result of existing Ivy League rules being more restrictive than NCAA rules, and the reduction resulting from the Core Group's recommendations:

	NCAA Rules	Existing Ivy Rules or Practices Different from NCAA Rules	Recommended Change
Preseason	• 29 practices • First practice date: allows 40 units prior to first contest • 5-day acclimatization period • No consecutive days with 2-a-days • 3-hour daily limit with 1 practice; 5-hour daily limit with 2 practices • 1st two days – helmet only • 3rd and 4th days – helmets and shoulder pads • 5th and beyond – full pads	• First practice date: allows 35 units prior to first contest	• Limit to 1 the number of days that pads can be worn during both sessions of 2-a-days
Fall – In season	• 1 day off per week • Limit of 4 hours per day and 20 hours per week	• Existing routines for full-pad, contact practice sessions minimize the emphasis and incidence of tackling. See Attachment C for a description of recommended guidelines, consistent with current custom.	• Limit full-contact practice days to two per week • During remaining days, no contact or live tackles may occur and no player may be "taken to the ground". No full pads but other helmets, uppers, girdles and thigh pads are permissible for safety reasons (no knee pads). • This represents a 60% reduction from the NCAA limit of 5 contact practices per week.
Spring	• 2 days off per week • 15 practices (including scrimmage) • 12 may be contact (8	• 12 practices (first 3 and 1 other practice no contact) • 8 allowing contact; 5	• 4 practices will be no full-contact (instead of 3), with 1 other practice remaining

	NCAA Rules	Existing Ivy Rules or Practices Different from NCAA Rules	Recommended Change
	tackling; no tackling in 4; no more than 3 devoted to 11-on-11 scrimmage) • Limit of 20 hours practice per week; 4 hours per day • 8 hours per week voluntary weight training and/or review of game film	tackling; • Only 1 of 3 scrimmages – the final practice – may be an "event" (with officials, uniforms, announcements to spectators) • 6 hours per week voluntary weight training and/or review of game film • Limit of 5 tackling practices is reduction of 37% from NCAA limit of 8.	no contact. • 7 practices allowing contact (5 tackling) • Clarified that the full-contact restrictions specified above apply. • Reduction of 42% in practices involving contact from the NCAA limit of 12 and of 12% from the current Ivy limit of 8.

B. Games

Existing NCAA rules – including those that penalize targeting, butting and ramming with the helmet, and causing blows to the head, and that require additional review by the conference office for possible postgame ramifications, as summarized in Attachment B – already provide a deterrent for helmet or head hits. The Core Group determined that it was not necessary at this time to recommend further changes to the NCAA playing rules, given the apparent deterrent effect of the existing rules, as illustrated by the fact that very few plays in Ivy League games during 2009 and 2010 (well under one per game) resulted in penalties for significant hits to the head.

Nonetheless, the Ivy League office and Coordinator of Football Officials will continue to direct officials to err on the side of caution by "throwing the flag" when in doubt as to whether a helmet or head hit occurred. This policy in turn allows the League office to assess and determine the appropriate postgame ramifications, which will be administered with a more stringent approach and may include game suspension, as discussed above. It should be noted that the Ivy League head football coaches fully support this philosophy and will use it to focus on continuing to teach players the proper technique, as discussed in the next section.

C. Education

The Core Group believes that a fundamental component of preventing future head hits and concussions is a continued and increased emphasis on education – via a variety of methods and focusing on the signs/symptoms of a concussion, the potential long-term risks of repeated brain trauma, and the proper hitting techniques.

Specifically, the Core Group recommends the following educational strategies, many of which are already being used by some or all Ivy League schools:

1. **Student-Athlete educational materials.** Student-athletes will continue to receive specific information regarding the signs and symptoms of a concussion and the importance of reporting to a medical professional any symptoms as soon as possible. In addition, education will emphasize the potential long-term risks of concussion and repetitive brain trauma, including recurrent injury when a concussion has not yet resolved (second-impact syndrome), post-concussive syndrome, and possibly depression and early onset of dementia. Athletes should understand that concussion has both short-term and long-term complications. This education should remain consistent with current research, as this continues to be an evolving area. Possible educational tools include:

   - NCAA materials and flyers/posters – signs/symptoms
   - NFL/NFLPA/CDC Poster and Youth Fact Sheet on Concussion

2. **Education regarding proper technique and fundamentals.** Practices should on a regular and ongoing basis continue to include, enhance and emphasize, the teaching of proper technique and fundamentals to avoid leading with the head, including a de-emphasis of hits on defenseless players.

3. **Annual presentation of educational videos.** The Group reviewed NCAA and NFL videos regarding concussions, which contain informative and useful information geared for players. The NCAA video is not specific to football, and the NFL included current and former players speaking to the importance of avoiding and not playing with concussions. The Group considered recommending producing an Ivy-specific video, but determined existing videos, combined with other education, were sufficient. The Group recommended that teams should present these videos (or USA Football or other videos, if available) at least annually during an educational session on concussions for players and coaches.

The Group also reviewed videos of specific examples of legal and illegal football hits provided by the NCAA and agreed that such examples should be presented to coaches and teams annually during the preseason, as well as the other educational videos (e.g., from NCAA and NFL).

4. **Additional in-person educational presentation.** The Core Group recommends that each team conduct an additional in-person, annual educational session aimed at connecting directly with the student-athletes and coaches by involving individuals personally knowledgeable about the potential risks and actual effects of concussions (e.g., former student-athletes, researchers, team physicians, athletics trainers). The session should also focus on identifying and reporting symptoms and the potential damage to brain cells caused by playing when a concussion is not fully healed.

5. **Educational session for football officials.** The annual officials clinic conducted by the Ivy League Coordinator of Football Officials will include a session on concussion information, including recognizing visible signs of a concussion and possibly involving a presentation from experts in the field.

6. **Education to high school and youth coaches.** It is hoped that with the increased awareness of and attention to concussions, an emphasis on proper injury management, improved technique, education and an overall reduction in hard hitting will trickle down to the youth levels. Nonetheless, the Group is cognizant of the role the Ivy League has in terms of interacting with, educating and serving as role models for youth players. The Group is interested in developing educational materials (e.g., videos) or information for high school and youth coaches, but recognizes the need to determine how to realistically achieve this goal, in a practical and cost-effective manner. The Ivy League should consider partnering with other groups (e.g., USA Football, the AFCA, CDC, Sports Legacy Institute) to implement this educational initiative.

D. Diagnosing Injury as a Preventative Tool

The Core Group discussed the fact that a reliable objective assessment tool is critical in identifying with certainty if a concussion has occurred. Detecting the signs of a concussion as soon as possible is a vital factor in initiating treatment, improving outcomes and preventing further injury or a repeated concussion. Educational programs geared towards athletes and coaches should be helpful in encouraging student athletes to report symptoms to the medical staff. In addition, in accordance with NCAA rules, any athlete that is diagnosed with a concussion cannot return to participation that day and

must be cleared by a medical professional prior to future participation. Thus, the increased reporting of symptoms and the potential for improving diagnosis, as well as the more conservative management of concussions, may reduce the risk of a more severe injury.

In addition, improving the recognition of injury and the evaluation process to accurately diagnose concussions may improve outcomes. Existing diagnostics techniques tend to involve an evaluation that incorporates a focused neurologic exam, a cognitive evaluation (e.g. orientation, concentration, memory), assessment of symptoms, and a balance evaluation, as they are the best indicators for concussion based on current data and research (and are considered "best practices" by the NCAA). Ivy League team physicians and athletics trainers have exchanged information on current techniques and systems used to evaluate symptoms and diagnose concussions. Ivy League institutions are all following NCAA requirements and are generally following NCAA best practices.

These diagnoses involve objective and subjective components, as they rely in large part on the professional judgment of the athletics trainer or team physician and their assessment and knowledge of the individual. Signs and symptoms of concussion are often delayed for hours and even days. Other management tools that are utilized post-injury include neuropsychological test batteries (computerized and/or paper and pencil tests), which are often compared to a preseason assessment. A majority of the Ivy League schools utilize these tools in their current management of football student athletes. The management of and return-to-play decision regarding concussive injury is multifaceted, and the Group underscored the importance of the individualized nature of these decisions (i.e., there is no "cookbook" approach to concussive injury as each injury in each individual is different).

Other diagnostic tools may also prove to be effective for evaluating and diagnosing concussion. The Core Group discussed the potential of an objective test for concussion. In particular, the Group received a presentation on the King-Devick test, a one-minute sideline assessment tool based on eye movement that measures the speed of rapid number naming. The King-Devick test has been used with mixed-martial-art athletes and appears sensitive to concussive injury. It has also been used with one football team with players already known to be concussed. Additional research regarding the King-Devick test as well as other sideline tools is needed, including prospective studies with multiple teams.

E. Recovery Process

1. **Need for academic accommodations to avoid cognitive strain on the brain.** The Core Group discussed the importance of avoiding cognitive work during the healing process after diagnosis of a

concussion. This is a critical component to allow the brain to recover. The Core Group recommended that the Ivy League Presidents communicate with the appropriate deans on each campus to ensure that they are aware of this serious and critical part of the recovery process and of the need to make appropriate academic accommodations for the timeframe specified by the physician for the student-athletes diagnosed with concussions.

2. **Return-to-Play Guidelines.** In response to the Council's December 2010 request that the Ivy League's athletics trainers and team physicians consider and recommend rules (e.g., a checklist) specifying when a player in any sport can return to play following evidence of a concussion, the team physicians and athletics trainers developed specific Return-To-Play Guidelines for use after a concussion diagnosis, incorporating NCAA best practices and supported by all eight schools.

V. Other sports

The Core Group reviewed NCAA data from 2004 to 2009, indicating that the rate of concussion during games per 1,000 athlete exposures accounted for between 4 and 16.2 percent of the injuries for these sports. This data, as reported by the NCAA Injury Surveillance Program by the Datalys Center, is as follows:

- Football: 3.1
- Men's lacrosse 2.6
- Men's ice hockey 2.4
- Women's ice hockey 2.2
- Women's soccer 2.2
- Wrestling 1.4
- Men's soccer 1.4
- Women's lacrosse 1.2
- Field hockey 1.2
- Women's basketball 1.2
- Men's basketball 0.6

The Core Group agreed that much of the epidemiology regarding concussive injury significantly under-reports injury. This reporting bias may explain some of the difference in injury incidence, especially as it relates to women versus men; however, the consensus was that the under-reporting of injuries is likely equal across sports, allowing for a comparison of incidence rates to help determine those sports at greatest risk for concussive injury.

Accordingly, in response to the Council's charge, the Core Group recommends that the Ivy League consider what changes or recommendations might be necessary to address the risks of concussions in the following sports (in priority order):

- Men's and Women's Ice Hockey
- Men's and Women's Lacrosse
- Men's and Women's Soccer

The Group acknowledged that research and data regarding concussion in these sports would be more limited than that available for football. In addition, the Group noted that men's and women's sports may need to be reviewed separately due to significant differences in how the games are played, particularly for ice hockey and lacrosse. It is anticipated that the educational recommendations for these six sports would be similar to those contained in this report and that the reviews would focus on the unique mechanisms for injury, possible practice limitations and the playing rules.

In addition, after completion of the review for these sports, the Ivy League should consider whether other sports should be reviewed.

VI. Conclusion

The recommendations set forth in this report are intended to enhance student-athlete welfare and help protect against concussive hits and the long-term effects of such hits, by focusing on reducing full-contact practices (and the opportunities for hits to occur), continuing to emphasize proper technique, and enhancing existing education regarding the need to report symptoms of a concussion – immediately and longer term – and of the potential long-term consequences.

Although these recommendations should place the Ivy League at the forefront of concussion management and prevention, research is still needed to improve our understanding of concussions, including threshold(s) for tolerance of hits, when concussions are most likely to occur and earlier diagnosis of long-term or permanent damage. Ivy League schools are already participating in some of this ongoing research.

In addition, as this remains an evolving area, the Core Group recommends that the Ivy League continue to monitor the research, data, best practices and other information regarding concussions and cumulative head trauma (e.g., short-term and long-term effects, causes of long-term consequences, diagnosis of permanent injury) to determine if these recommendations need to be revisited in the future.

ATTACHMENT A

Ivy League Ad Hoc Concussion Committee
Roster

Co-Chairs
Jim Kim, President, Dartmouth College
David Skorton, President, Cornell University

Committee Members
Al Bagnoli, Head Football Coach, University of Pennsylvania ("Core Group")
Brant Berkstresser, Head Athletic Trainer, Harvard University ("Core Group")
Phil Estes, Head Football Coach, Brown University ("Core Group")
Lindsay Huston, MD, Team Physician, Yale University ("Core Group")
Eric Laudano, Head Athletic Trainer, University of Pennsylvania ("Core Group")
Tim Murphy, Head Football Coach, Harvard University ("Core Group")
Andy Noel, Athletics Director, Cornell University (Committee on Administration and Policy Committee representative)
Margot Putukian, MD, Team Physician, Princeton University ("Core Group")
Ray Tellier, Associate Athletics Director, Columbia University ("Core Group")
Jack Turco, MD, Team Physician, Dartmouth College ("Core Group")
Jim Maconaghy, Ivy League Coordinator of Officials and former collegiate football official ("Core Group")

Experts/Consultants
Chris Nowinski, Co-Director of Boston University's Center for Study of Traumatic Encephalopthy (CTSE) and former Harvard football student-athlete ("Core Group")
Steven L. Galetta, MD and Laura Balcer, MD, Neuro-Opthomalogists at the University of Pennsylvania School of Medicine ("Core Group")
Sean Morey, Co-Chair of the NFL Players Association's Mackey-White Traumatic Brain Injury Committee, former NFL player and Super Bowl XL Champion, former Brown football student-athlete ("Core Group")
Bob Iuliano, General Counsel, Harvard University (to provide legal counsel as needed)

Ivy Office
Robin Harris, Executive Director
Celene McGowan, Associate Executive Director for Championships and Sport Administration

ATTACHMENT B

APPLICABLE NCAA FOOTBALL PLAYING RULES

I. Points of Emphasis

The NCAA Football Rules Committee has extraordinary pride in the Football Code, which was introduced in 1916 and has been updated several times. These guidelines form a harmony of agreement among coaches, players, game officials and administrators that places each contest in an environment of fairness and sportsmanship. It is noted that the Code emphasizes the following unethical practices: "Using the helmet as a weapon. The helmet is for protection of the player..." and "players and coaches should emphasize the elimination of targeting and initiating contact against a defenseless opponent and/or with the crown of the helmet." Every participant in the collegiate football scene shares a responsibility for ethical conduct that enhances the future of this American tradition.

PROTECTION OF DEFENSELESS PLAYERS—In 2008, the committee introduced a separate rule prohibiting initiating contact with and targeting a defenseless opponent (Rule 9-1-3). The following are situations in which defenseless players are susceptible to serious injury:

- The quarterback moving down the line of scrimmage who has handed or pitched the ball to a teammate, and then makes no attempt to participate further in the play;
- The kicker who is in the act of kicking the ball, or who has not had a reasonable length of time to regain his balance after the kick;
- The passer who is in the act of throwing the ball, or who has not had a reasonable length of time to participate in the play again after releasing the ball;
- The pass receiver whose concentration is on the ball;
- The pass receiver who has clearly relaxed when the pass is no longer catchable;
- The kick receiver whose attention is on the downward flight of the ball;
- The kick receiver who has just touched the ball;
- The player who has relaxed once the ball has become dead; and
- The player who is obviously out of the play.

These players are protected by rules that have been in place for many years. It is of the utmost importance that participants, coaches and game officials carefully and diligently observe safety rules. Intentional helmet-to-helmet contact is never legal, nor is any other blow directed toward an opponent's head. Flagrant offenders shall be disqualified.

II. Initiating Contact/Targeting an Opponent
ARTICLE 3. a. No player shall initiate contact and target an opponent with the crown (top) of his helmet. When in question, it is a foul.
b. No player shall initiate contact and target a defenseless opponent above the shoulders. When in question, it is a foul. (Refer to Points of Emphasis for a description of "Defenseless Player.")

PENALTY (a-b)—Personal foul, 15 yards. For dead-ball fouls, 15 yards from the succeeding spot. Also, automatic first down for Team B fouls if not in conflict with other rules. (Exception: Penalties for Team A personal fouls behind the neutral zone are enforced from the previous spot. Safety if the foul occurs behind Team A's goal line) [S7, S24, S34, S38, S39, S40, S41, S45 or S46]. Flagrant offenders shall be disqualified [S47]. For Team A fouls during free or scrimmage kick plays (field goal plays exempted): Enforcement may be at the previous spot or the spot where the subsequent dead ball belongs to Team B (Rules 6-1-8 and 6-3-13).

Note: See Rule 9-6 for mandatory conference review procedures.

SECTION 6. Flagrant Personal Fouls Player Ejection
ARTICLE 1. When a player is disqualified from the game due to a flagrant personal foul, that team's conference shall automatically initiate a video review for possible additional sanctions before the next scheduled game.

Initiating Contact/Targeting an Opponent
ARTICLE 2. When there is a foul called for initiating contact/targeting an opponent (Rule 9-1-3) that does not result in a player disqualification, there shall automatically be a video review by the conference for possible additional sanctions before the next scheduled game.

Foul Not Called
ARTICLE 3. If subsequent review of a game by a conference reveals plays involving flagrant personal fouls that game officials did not call, the conference may impose sanctions prior to the next scheduled game.

ATTACHMENT C

RECOMMENDED GUIDELINES
IN-SEASON FULL PADS CONTACT PRACTICE SESSIONS

Coaching emphasis is on the execution of proper assignments and techniques, ensuring safety and minimizing the risk of injury to all players.

Emphasis for all team (11 on 11), group or individual contact practice periods is for all participants to remain on their feet during the entire repetition or play. Players are instructed to not leave their feet to initiate a block or tackle.

The point of contact for all blocks and tackles is above the waist. Contact must be initiated from a position in front of or facing their opponent. Blocks and tackles initiated directly from the side or behind their opponent are not permitted.

Contact to the quarterback whether in the pocket or throwing on the run from behind the line of scrimmage is not permitted. Defenders are instructed to only touch the quarterback with two hands, run by the quarterback, or pull-up and stop in front of the quarterback to avoid contact.

Contact to ball carriers is permitted only from a position in front of the ball carrier, usually by one defender. Contact is taught to be above the waist with an emphasis on proper tackling technique. Ball carriers are not brought to the ground. Defenders pursuing from the side or behind the ball carrier are instructed to continue proper pursuit angles but not to initiate contact. Pursuing defenders are allowed to practice stripping the ball from the ball carrier with their hands and arms.

Contact to receivers who are reaching or jumping to catch the ball in a defenseless position is not permitted. Once a pass is thrown defenders are taught to pursue to the ball in the air at full speed and play the ball. Defenders are instructed to run by the receiver or pull-up and stop prior to initiating contact. Once a receiver catches the ball and turns up-field to run, the rules pertaining to ball carriers apply.

Men's Lacrosse

NCAA Statistics:

- 9 times more likely to sustain a concussion in a game as compared to practice
- Concussions account for 8.6 % of all game injuries, 3.6% of all practice injuries
- 78% of game concussions occur from contact with another player
- Significant increase in both game/practice concussion rates after implementation of new Lacrosse helmet in 1996/1997.
    - Decrease in attenuating forces from the front of the helmet as compared to previous helmet
        - Limitations include:
            - Increased in concussion education in mid-nineties

Ivy Statistics: 3 year retrospective data

- 40 of the 50 reported concussions resulted from player contact
- All 22 recorded competition concussions resulted from player to player contact

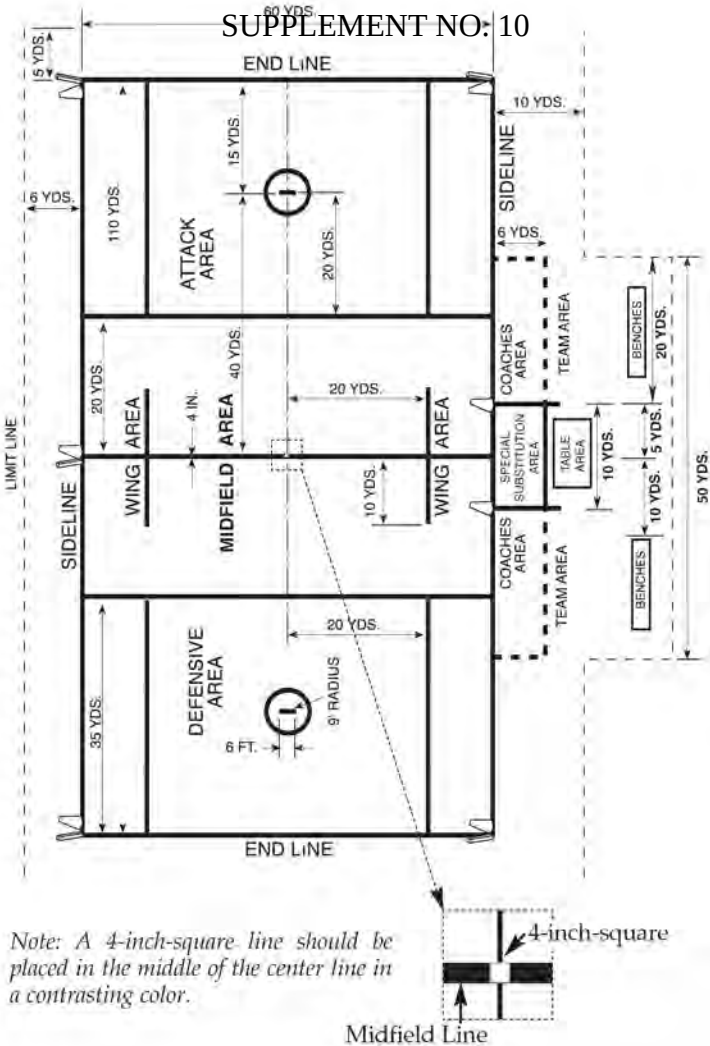
Video – Biggest Hits 2009

http://www.youtube.com/watch?v=0UApadps1xk

Discussions at Ivy League level: which have not been finalized; providing suggestions to the lacrosse rules committee regarding the following areas?

- In the rule regarding targeting the head
    - Remove intention
    - Increase penalty time
- Add a point of emphasis to penalize players who initiate a hit or check without the intention to go after the ball
    - Similar to rules in football to protect a defenseless player
    - Large number of concussions come from ground balls
- Need for formal process to hold officials accountable for missed calls in relation to player safety.
    - Men's Lacrosse officiating is handled by national lacrosse association and not conference affiliation
        - Difficult to implement officiating accountability-player suspensions etc.
- Consideration of face off rule-changes to reduce incidences of concussions
    - Face-off circle
    - Decrease winger distance

2005 NCAA Men's Lacrosse Field of Play



Note: A 4-inch-square line should be placed in the middle of the center line in a contrasting color.

Boston University School of Medicine
Center for the Study of Traumatic Encephalopathy

Medical Campus
72 East Concord Street, B-7380
Boston, Massachusetts 02118
T 617-414-1326 F 617-638-5679
www.bu.edu/cste

BOSTON UNIVERSITY

SLI SPORTS LEGACY INSTITUTE

David Klossner
Director of Health & Safety
National Collegiate Athletic Association

Dear Mr. Klossner,

Thank you for our recent discussion. I reached out because I think it is time for colleges and universities to begin to address the issue of Chronic Traumatic Encephalopathy (CTE) in sports. The NCAA has taken a leadership role on the concussion issue, and I think it is appropriate for the NCAA to take a leadership role on CTE in prevention, education, and research. The goal of this letter is to provide the latest information on CTE to NCAA thought leaders, including unpublished information, so that the NCAA can begin to take appropriate steps to address this problem. I have included steps that we feel would be appropriate, and our goal is to begin a dialog on this issue.

Chronic Traumatic Encephalopathy (also known as *dementia pugilistica*) is a progressive neurodegenerative disease that is caused, at least in part, by repeated concussive and subconcussive brain trauma. These types of hits are regularly incurred in contact sports. The Boston University Center for the Study of Traumatic Encephalopathy (CSTE) was established in 2008 as a collaboration between Boston University School of Medicine and the Sports Legacy Institute, a non-profit organization dedicated to "solving the concussion crisis" through prevention, education, and research.

CTE has been a neglected field of study, and the CSTE was the first center in the world dedicated to the study of CTE. To date, the CSTE has established the following programs examining CTE:

> **Brain Bank.** The CSTE, in conjunction with the Department of Veterans Affairs, has the largest CTE Brain Bank in the world with over 100 brains, and over 70 positive cases of CTE. These are brains of former athletes at the high school, college, and professional level as well as former military personnel. Unfortunately, <u>we have found CTE in the brains of multiple individuals who have only played high school and college level football</u>. It is imperative that we learn more. The National Football League and National Football League Players Association collaborate with the CSTE brain bank and encourage their players and families to participate in the research.
>
> **DETECT Study.** Diagnosing and Evaluating Traumatic Encephalopathy using Clinical Tests (or DETECT Study) is the first CTE-specific research study ever funded by the

National Institutes of Health. The ultimate goal of this study is to learn how to diagnose CTE during life, by learning more about the clinical presentation and possible biomarkers of the disease. This will be accomplished through a comparison of a group of high risk individuals (former professional football players) and a group of low risk individuals (former athletes from non-contact sports).

Brain Donation Registry. The CSTE Brain Donation Registry includes several hundred athletes of all sports and levels of play who have pledged to donate their brain when they pass away.

LEGEND Study. The Longitudinal Examination to Gather Evidence of Neurodegenerative Disease (or LEGEND Study) interviews athletes once per year to understand their background and history, exposure to brain trauma, and how they are thinking, feeling, and acting. This study includes cognitive testing and has the option for genetic testing. Individuals from all across the United States and Canada are welcomed to participate, with the ultimate goal of learning more about the risk factors and clinical presentation of CTE.

Moving forward, the CSTE has several research objectives:

Animal Model. The CSTE is currently collaborating in the design and study of an animal model of concussive and subconcussive injury and its long-term effects.

Diagnosis. Currently there is no way to definitively diagnose CTE during life. Finding accurate, predictive diagnostic tools is the first step toward being able to help individuals who may be suffering from CTE.

Treatment. The ultimate goal is to learn how to treat CTE. By utilizing treatments already being studied for similar diseases, such as Alzheimer's and Frontotemporal Dementia, we hope to be able to move quickly to be able to help individuals who may be suffering from this disease.

State of the Science. It is worth noting that while there are many things we have yet to learn about CTE, including its disease course, genetic, and environmental risk factors, it is true that in the world's experience, both published and unpublished, everyone with neuropathologically confirmed CTE (over 120 cases) received extraordinary brain trauma.

The CSTE has collaborated with other researchers in the field and remains open to collaboration with researchers and organizations who wish to advance the study of CTE. We would like to invite the NCAA to join other leading organizations such as the NFL in collaborating to advance the study, treatment, and education surrounding CTE.

In light of this new research, we encourage the NCAA to take the following actions:

1. **Education.** The NCAA now educates athletes on concussions, but does not warn them of CTE. After reviewing the research, in 2010 the NFL began warning their athletes about CTE. We encourage the NCAA to follow suit.
2. **Prevention.** The NCAA should consider CTE risks in its rules and regulations and aim to reduce the overall amount of brain trauma incurred by its athletes.
3. **Research.** The NCAA should include CTE, along with concussions, as a focus of the funds it awards to research. Specific to CSTE research, we would like to discuss working with the NCAA to recruit non-contact elite athletes to the DETECT study.

Attached to this letter include a number of supporting documents, including:

1. Stern RA, Riley DO, Daneshvar DH, Nowinski CJ, Cantu RC, McKee AC. Long-term Consequences of Repetitive Brain Trauma: Chronic Traumatic Encephalopathy. Phys Med Rehabil Clin N Am, 3, S460-S467, October 2011.
2. McKee AC, Cantu RC, Nowinski CJ, Hedley-Whyte ET, Gavett BE, Budson AE, Santini VE, Lee H-Y, Kubilus CA, Stern RA. Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy following Repetitive Head Injury. J Neuropath Exp Neurol, 2009; 68(7): 709-735.
3. McKee AC, Gavett BE, Stern RA, Nowinski CJ, Cantu RC, Kowall NW, Perl D, Hedley-Whyte ET, Price B, Sullivan C, Morin P, Lee HS, Kubilus CA, Daneshvar DH, Wulff M, Budson AE. TDP-43 Proteinopathy and Motor Neuron Disease in Chronic Traumatic Encephalopathy. J Neuropath Exp Neurol, 2010; 69(9): 918-929
4. Schwarz, Alan. Suicide Reveals Signs of a Disease Seen in N.F.L. *New York Times*. September 13, 2010.

I look forward to discussing this further.

Best regards,

Chris Nowinski
Co-Director, Center for the Study of Traumatic Encephalopathy
Boston University School of Medicine
Co-Founder & CEO, Sports Legacy Institute

SPORTS LEGACY INSTITUTE

Boston University Center for the Study of Traumatic Encephalopathy

Suicide Reveals Signs of a Disease Seen in N.F.L.

The New York Times ON THE WEB

By Alan Schwarz

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ALLENTOWN, Pa. — A brain autopsy of a University of Pennsylvania football player who killed himself in April has revealed the same trauma-induced disease found in more than 20 deceased National Football League players, raising questions of how young football players may be at risk for the disease.

Owen Thomas, a popular 6-foot-2, 240-pound junior lineman for Penn with no previous history of depression, hanged himself in his off-campus apartment after what friends and family have described as a sudden and uncharacteristic emotional collapse. Doctors at Boston University subsequently received permission from the family to examine Thomas's brain tissue and discovered early stages of chronic traumatic encephalopathy, a disease linked to depression and impulse control primarily among N.F.L. players, two of whom also committed suicide in the last 10 years.

Doctors in the Boston University group and outside it cautioned that Thomas's suicide should not be attributed solely or even primarily to the damage in his brain, given the prevalence of suicide among college students in general. But they said that a 21-year-old's having developed the disease so early raised the possibility that it played a role in his death, and provided arresting new evidence that the brain damage found in N.F.L. veterans can afflict younger players.

Thomas never had a diagnosis of a concussion on or off the football field or even complained of a headache, his parents said, although they acknowledged he was the kind of player who might have ignored the symptoms to stay on the field. Because of this, several doctors said, his C.T.E. — whose only known cause is repetitive brain trauma — must have developed from concussions he dismissed or from the thousands of subconcussive collisions he withstood in his dozen years of football, most of them while his brain was developing.

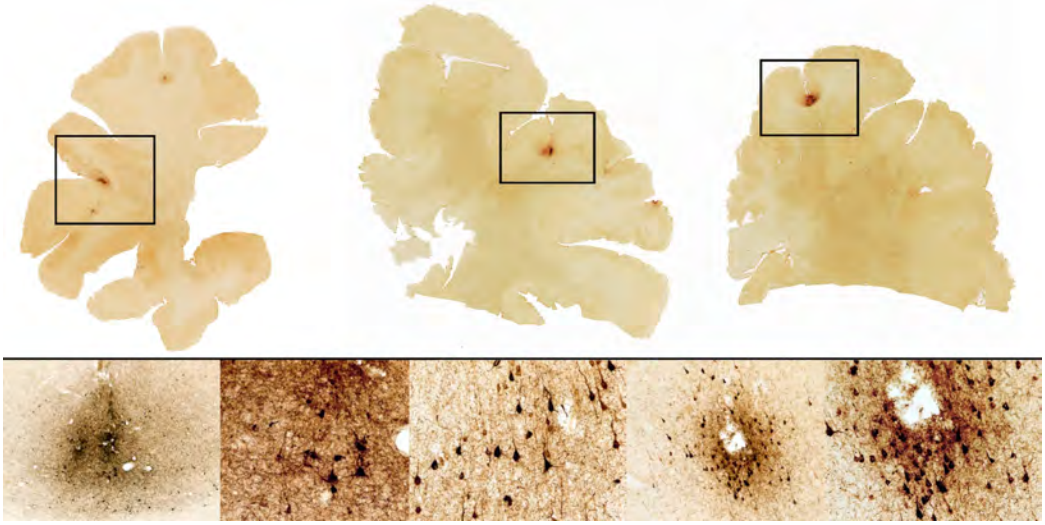
The idea that C.T.E. can stem from hits below the level of concussion — which are endemic to football and all but impossible for doctors to see or manage — is relatively new. Ever since C.T.E. in professional football players began making national headlines in early 2007, it has generally been ascribed to mistreated or at least cumulative concussions, for which awareness and education can be an antidote.

The diagnosis in Thomas's case was independently confirmed by Dr. Daniel Perl, a professor of pathology at Uniformed Services University of the Health Sciences, the medical school for the United States military.

"It's not unreasonable that aspects of his behavior were related to the underlying brain disease that was detected," said Dr. Perl, adding that he was speaking as an experienced neuropathologist and not on behalf of his organization. "This is real."

He added, "This is a call for a broader range of research into this problem that extends beyond the heavy duty N.F.L. level of athletics."

Thomas is the youngest and first amateur football player to be found with clear C.T.E., which is linked with cognitive impairment, depression and ultimately dementia. One 18-year-old former high school player who died two years ago, and whose name has been withheld by the Boston University researchers at his family's request, had only incipient traces of the disease.



Thomas's parents, the Rev. Tom Thomas and the Rev. Kathy Brearley, requested that their son's case be made public to educate other families about the possible and perhaps addressable risks of football at all levels. About 1.4 million children ages 14 to 18 play high school football every fall, and about three million others play in youth leagues at younger ages.

Thomas's parents emphasized that they did not hold responsible the University of Pennsylvania specifically or their son's youth and high school programs in South Whitehall Township, which is outside Allentown. They also said they were not considering legal action.

"This is an issue beneath the N.F.L. level," Mr. Thomas said. "I want people to take this seriously."

Sitting with her husband on the porch that overlooks the yard where Owen once played, Ms. Brearley added, "We have to think of different options that can take a hearty, meaty, great contact sport but minimize the risk to young people."

Owen Thomas was the second Penn player to commit suicide in five years; running back Kyle Ambrogi killed himself in 2005. The university will honor Thomas at a ceremony before Penn's opening home game against Lafayette on Saturday. He had been elected one of the team's captains before he died.

"Obviously this is a contact sport — could this happen? Absolutely," said Penn Coach Al Bagnoli, noting how Penn trainers never clear a player with a concussion to return until he withstands appropriate medical scrutiny. "Do people take as many precautions as we can? Absolutely."

Before Thomas, 21, the youngest player who previously received a diagnosis of C.T.E. was Chris Henry, 26, a Cincinnati Bengals receiver who died in December during a domestic dispute in which he appeared to jump from the back of a moving pickup truck. The only previous non-N.F.L. player with a clear case of C.T.E. was Mike Borich, a former Western Illinois receiver who died in February 2009 after a drug overdose at 42.

The Thomas case will almost certainly prove more arresting to those assessing the long-term risks of football at all levels, as he had developed the disease before leaving college and, for reasons that remain unknown, developed severe depression and killed himself.

“It’s pretty hard to make a jump with one case,” said Dr. James Moriarity, the University of Notre Dame’s head physician, who oversees the athletic department’s medical care. “But if it’s true that that happened, it would kill the sport,” he said, referring to an amateur player getting C.T.E. “As a parent, it’s going to be hard to justify kids going out and doing that.”

Owen Thomas was a third generation college football player. His grandfather Frank Thomas played for Millersville (Pa.) University in the 1930s, and his father played four years at the University of Virginia in the late 1960s.

Owen started playing at age 9 and relished football’s physicality.

“He loved to hit people,” his mother said. “He loved to go into practice and hit really hard. He loved to intimidate. It’s kind of sad. We all love football. We all love watching. We all love these great hits.”

Thomas played three seasons at Parkland High School, talented enough at linebacker and tight end to often play every down of every game — even blocking on punts and kickoffs, one of his favorite responsibilities. He was bright enough to be admitted to Penn’s Wharton School of Business, one of the best undergraduate business programs in the country. He played freshman football and then started the last two seasons on the varsity, earning second-team all-Ivy League honors in 2009 and helping lead the Quakers to the Ivy title.

Dr. Robert Stern, a director of the Boston University group, said the identification of factors like genetics would probably someday explain why some people develop C.T.E. while most do not. Thomas’s case, he said, proves that the disease can begin, and perhaps influence behavior, among football players below the N.F.L. level.

“We don’t know if it’s a specific age, we don’t know if it’s a cumulative number of years of exposure to head trauma, we don’t know what combination of hits to the head set this disease in motion,” Dr. Stern said. “These are critical issues that need to be answered in order to help guide any dramatic policy changes and individual decisions down the road.”

Dr. Stern and other experts in the field emphasized that C.T.E. could not be blamed solely for a person’s suicide. But some of the clues left from Thomas’s case, they said, suggested that the damage in his brain might have exacerbated his sudden depression and compromised his ability to think clearly about his actions.

Thomas left no note and still had his cellphone in his pocket — which his mother said indicated that he was acting on impulse, not forethought.

Dr. Perl said that although links were easy to make in hindsight, lack of impulse control is a consistent manifestation of how executive function can be compromised by C.T.E.'s neurofibrillary tangles and tau protein formations in the frontal lobe of the cerebral cortex. Dr. Perl added that C.T.E. typically impaired a person's short-term memory — which in a college student approaching exams would be harrowing — but that the relatively mild C.T.E. in Thomas's hippocampus did not suggest severe memory problems, though that was possible.

Ms. Brearley said she would never know the root of her son's actions, but the C.T.E. diagnosis gave her solace, if not a solution.

"It gives me some peace in my heart to think this is a missing piece of a jigsaw puzzle," she said.

Thomas's parents said they wanted to share his story to warn other families of young football players that C.T.E. and all of its still-unknown ramifications were no longer confined to the N.F.L. In this respect, Owen Thomas will probably become amateur football's counterpart to Andre Waters, the former hard-hitting Philadelphia Eagle whose suicide in 2006 catalyzed much of the awareness of head injuries in the N.F.L.

Mr. Thomas said he recalled the day he read about Waters's brain damage in a newspaper. He also remembered what he had thought ever since: "Thank goodness that's only the N.F.L. — it can't happen to Owen."

Long-term Consequences of Repetitive Brain Trauma: Chronic Traumatic Encephalopathy

Robert A. Stern, PhD, David O. Riley, BS, Daniel H. Daneshvar, MA, Christopher J. Nowinski, BA, Robert C. Cantu, MD, Ann C. McKee, MD

Abstract: Chronic traumatic encephalopathy (CTE) has been linked to participation in contact sports such as boxing and American football. CTE results in a progressive decline of memory and cognition, as well as depression, suicidal behavior, poor impulse control, aggressiveness, parkinsonism, and, eventually, dementia. In some individuals, it is associated with motor neuron disease, referred to as chronic traumatic encephalomyelopathy, which appears clinically similar to amyotrophic lateral sclerosis. Results of neuropathologic research has shown that CTE may be more common in former contact sports athletes than previously believed. It is believed that repetitive brain trauma, with or possibly without symptomatic concussion, is responsible for neurodegenerative changes highlighted by accumulations of hyperphosphorylated tau and TDP-43 proteins. Given the millions of youth, high school, collegiate, and professional athletes participating in contact sports that involve repetitive brain trauma, as well as military personnel exposed to repeated brain trauma from blast and other injuries in the military, CTE represents an important public health issue. Focused and intensive study of the risk factors and in vivo diagnosis of CTE will potentially allow for methods to prevent and treat these diseases. Research also will provide policy makers with the scientific knowledge to make appropriate guidelines regarding the prevention and treatment of brain trauma in all levels of athletic involvement as well as the military theater.

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R.A.S. Center for the Study of Traumatic Encephalopathy, Boston University School of Medicine, Boston University, 72 East Concord St, 7380, Boston, MA 02118; Department of Neurology and Neurosurgery; Alzheimer's Disease Center Clinical Core, Boston University School of Medicine, Boston, MA. Address correspondence to: R.A.S.; e-mail: bobstern@bu.edu
Disclosure: nothing to disclose

D.O.R. Center for the Study of Traumatic Encephalopathy, Department of Neurology, Boston University School of Medicine, Boston, MA
Disclosure: nothing to disclose

D.H.D. Department of Neurology, Boston University School of Medicine, Boston, MA
Disclosure: nothing to disclose

C.J.N. Center for the Study of Traumatic Encephalopathy, Boston University School of Medicine, Boston, MA, Sports Legacy Institute, Waltham, MA
Disclosure: nothing to disclose

R.C.C. Center for the Study of Traumatic Encephalopathy, and the Department of Neurosurgery, Boston University School of Medicine, Boston, MA; Sports Legacy Institute, Waltham, MA; Neurosurgery Service, and the Department of Surgery, Emerson Hospital, Concord, MA; and Sports Medicine, Emerson Hospital, Concord, MA
Disclosure: nothing to disclose

A.C.M. Center for the Study of Traumatic Encephalopathy; Neurology and Pathology, Boston University School of Medicine, Boston, MA
Disclosure: nothing to disclose

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INTRODUCTION

There has been increased attention given to the recognition, diagnosis, and management of sports-related concussion. Participants in popular sports such as American football, hockey, wrestling, rugby, soccer, and lacrosse all risk exposure to brain injury that range from asymptomatic subconcussive blows to symptomatic concussion to more moderate or severe traumatic brain injury (TBI). In addition, military service and many other activities, including, but not limited to, downhill skiing, martial arts, horse riding, parachuting, and other adventure sports have been associated with TBI [1-3]. An estimated 1.6-3.8 million sports- and recreation-related concussions occur in the United States each year, although the true figure is unknown because most concussions are not recognized and reported [4-6]. In addition to symptomatic concussions, players in collision sports such as American football may experience many more subconcussive impacts throughout a season and career. Athletes at certain positions (eg, linemen) may sustain up to 1400 impacts per season, and high school players who play both offense and defense potentially sustain closer to 2000 impacts [7-10].

A mild TBI (mTBI) or concussion is a brain injury that results from a force transmitted to the head that leads to a collision between the brain and skull or to a strain on the tissue and vasculature of the brain [11,12]. This injury can lead to a variety of physical, psychosocial, and cognitive symptoms, and, when these deficits do not resolve, can result in postconcussion syndrome (PCS) [13]. Common symptoms include fatigue, dizziness, headache, light and noise sensitivity, mental fogginess, depression, irritability, and impairments of executive functioning and concentration. Because a concussion and its symptoms result from temporary, reversible cytoskeletal and metabolic derangements that involve shifts in ion

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channels and energy imbalance, the majority of deficits associated with a concussive injury resolve within a matter of days, weeks, or months [13-16]. In some instances, PCS may persist for months or years after the initial injury. However, it is believed that no more than 15% of individuals with a history of mTBI still experience symptoms 1 year after injury [13,17,18].

To date, the resulting progressive tauopathy, known as chronic traumatic encephalopathy (CTE), has only been found in individuals with a history of repetitive brain trauma [3,7]. Despite the recent increase in attention on the long-term effects of repetitive brain trauma, including CTE, it has been known for some time that contact sports may be associated with neurodegenerative disease. In 1928, Martland [19] described a symptom spectrum in boxers, which he termed "punch drunk," that appeared to result from the repeated blows experienced in the sport, particularly in slugging boxers who took significant head punishment as part of their fighting style. In 1937, Millspaugh [20] introduced the term dementia pugilistica to describe the syndrome characterized by motor deficits and mental confusion in boxers. By 1973, a neuropathologic report, by Corsellis et al [21], of dementia pugilistica in 15 boxers concluded that, although similar to other neurodegenerative diseases, dementia pugilistica is a neuropathologically distinct disorder.

After its initial description, evidence emerged that the clinical symptoms and neuropathology associated with dementia pugilistica were not solely limited to the boxing population. As a result, the term CTE surfaced in the 1960s and is now the term used to describe the neurologic deterioration that results from repetitive brain trauma [3,22]. Recent research results have demonstrated neuropathologic evidence of CTE in participants of many sports outside of boxing, including American football, professional hockey, and professional wrestling. CTE also has been found in those with a history of repetitive brain trauma aside from athletics, including a victim of physical abuse, a person who is a self-injurer, a person with epilepsy, and a circus clown [3,7,23-28].

Although CTE is associated with a history of repetitive brain trauma, the exact relationship between the acute traumatic injury and CTE is unclear. It has been hypothesized that a neurodegenerative cascade is triggered by repetitive axonal stretching and deformation induced by trauma, particularly in individuals with previous unresolved concussive and/or subconcussive injuries [14,29]. It also is unknown whether CTE is more likely to occur after extended exposure to repetitive brain trauma or whether a single traumatic injury can initiate this neurodegenerative cascade in susceptible individuals. Given the current understanding of CTE, it seems likely that trauma type and frequency play a role in CTE development [30]. An athlete's specific sport, level of competition (eg, professional versus collegiate), position, and playing career duration may all confer different degrees of CTE risk [30]. Other factors, such as age, gender, and genetic predisposition, may contribute to CTE's development in susceptible individuals, although these variables require further investigation [3].

To date, there has been no randomized neuropathologic study of CTE, and, as a result, there is a selection bias in the reported cases. Fourteen of the 15 professional American football players examined neuropathologically at the Veterans Affairs Center for the Study of Traumatic Encephalopathy (VA CSTE) Brain Bank have been diagnosed with CTE. This is a biased sample, which overrepresents the actual incidence of CTE in professional American football players, because families are more likely to consider neuropathologic examination if they suspect that their loved one has symptoms related to CTE or another neurodegenerative disease. Future research, perhaps in vivo studies that use biologic markers of disease and new clinical diagnostic criteria for CTE, will lead to improved understanding of the incidence, prevalence, and risk factors for CTE.

In the sections below, we will provide (1) an overview of the neuropathologic findings of CTE (including gross and microscopic pathology) and a related variant, chronic traumatic encephalomyelopathy that is associated with motor neuron disease; (2) descriptions of the early and later clinical presentations and course of CTE; and (3) a description of the possible risk factors for CTE, in addition to the necessary repetitive brain trauma.

GROSS NEUROPATHOLOGY

The gross changes of CTE are typically observed in late-stage disease (Table 1). Advanced cases of CTE demonstrate generalized atrophy, most prominent in the frontal and medial temporal lobes; enlargement of the lateral and third ventricles; cavum septum pellucidum; and septal fenestrations (Figure 1). There also may be thinning of the hypothalamic

Table 1. *Gross neuropathology of chronic traumatic encephalopathy*

Overall
Decrease in brain mass
Cavum septum pellucidum
Septal fenestrations
Ventricles
Enlarged lateral ventricles
Enlarged third ventricle
Atrophy
Generalized atrophy, particularly of the frontal and temporal lobes
Atrophy of the medial temporal lobes
Mammillary body atrophy
Thalamic atrophy
Pallor
Locus coeruleus
Substantia nigra

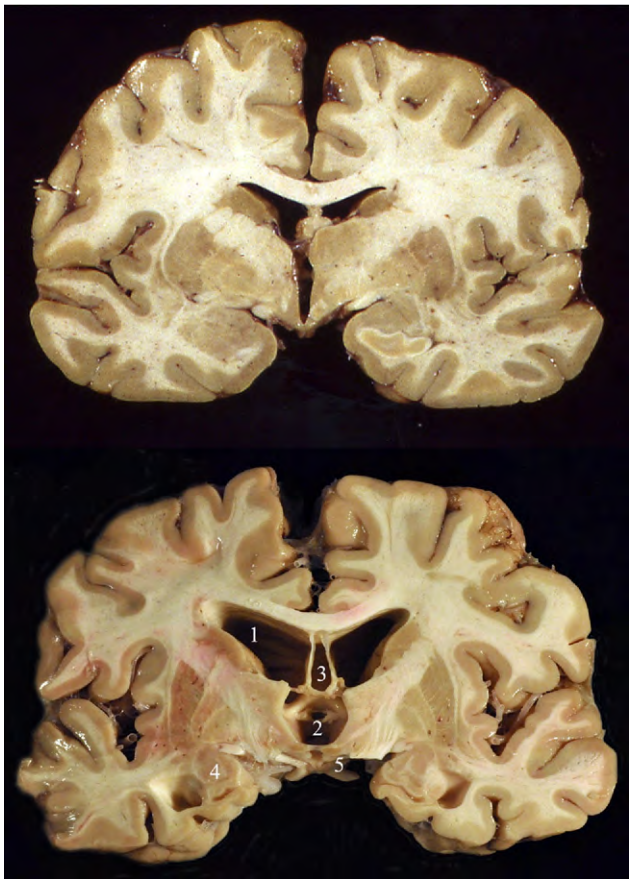

Figure 1. Gross pathology of chronic traumatic encephalopathy (CTE). The coronal section of normal brain (top), showing the expected size and relationship of the cerebral cortex and ventricles. The brain from a retired professional football player (bottom), showing the characteristic gross pathology of CTE with severe dilatation of ventricles II (1) and III (2), cavum septum pellucidum (3), marked atrophy of the medial temporal lobe structures (4), and shrinkage of the mammillary bodies (5).

floor, shrinkage of the mammillary bodies, and atrophy of the hippocampus, entorhinal cortex, and amygdala [3,7].

MICROSCOPIC NEUROPATHOLOGY: GENERAL DESCRIPTION

CTE is characterized by a unique pattern of microscopic changes (Table 2). There are extensive tau-immunoreactive neurofibrillary tangles (NFT), neuropil neurites (NT), and glial tangles (GT) in the frontal and temporal cortices (Figure 2). Unlike Alzheimer disease (AD) or many other tauopathies, the tau immunoreactive abnormalities tend to cluster at the depths of sulci, around small blood vessels, and in superficial cortical layers [3,7]. In advanced cases, there are tau-immunoreactive inclusions in the limbic and paralimbic regions, diencephalon, brainstem, and subcortical white matter. The specific soluble and insoluble tau isoforms found in CTE are indistinguishable from those found in AD, and the ratio of tau isoforms with 4 versus 3 microtubule binding repeats is approximately 1 in both diseases [31]. Importantly, these changes seen in CTE usually occur in the relative absence of beta-amyloid (Aβ) deposits [3,7].

In addition to the widespread tau immunoreactivity, the majority of CTE cases also are marked by TDP-43 proteinopathy. The TDP-43 inclusions can be widespread and are typically found in the brainstem; basal ganglia; diencephalon; medial temporal lobe; frontal, temporal, and insular cortices; and subcortical white matter. In a subset of individuals with CTE, abundant TDP-43 immunoreactive inclusions and neurites are found in the anterior horns of the spinal cord and motor cortex, combined with corticospinal-tract degeneration, loss of anterior horn cells of the spinal cord, and ventral root atrophy. These individuals develop a progressive motor neuron disease that appears very similar to amyotrophic lateral sclerosis (ALS) and is characterized by profound weakness, muscular atrophy, spasticity, and fasciculations [32]. The fact that an ALS-like condition is found in some individuals with CTE suggests that some forms of clinical ALS may be associated with TBI [33,34].

Table 2. *Microscopic neuropathology of chronic traumatic encephalopathy*

Neurofibrillary tangles, frequent
Olfactory bulb
Dorsolateral frontal cortex
Orbital frontal cortex
Subcallosal frontal cortex
Insular cortex
Superior and/or middle temporal gyri
Inferior temporal gyrus
Entorhinal cortex
Hippocampus
Amygdala
Mammillary bodies
Substantia nigra
Locus coeruleus
Neurofibrillary tangles, common
Hypothalamus
Substantia innominata
Medulla
Thalamus
Neurofibrillary tangles, rare
Cingulate gyrus
Inferior parietal cortex
Occipital lobe
β-Amyloid deposits
Diffuse plaques in 45%
Sparse neuritic plaques
White matter
Loss of myelinated fibers
Perivascular macrophages
Cribriform state

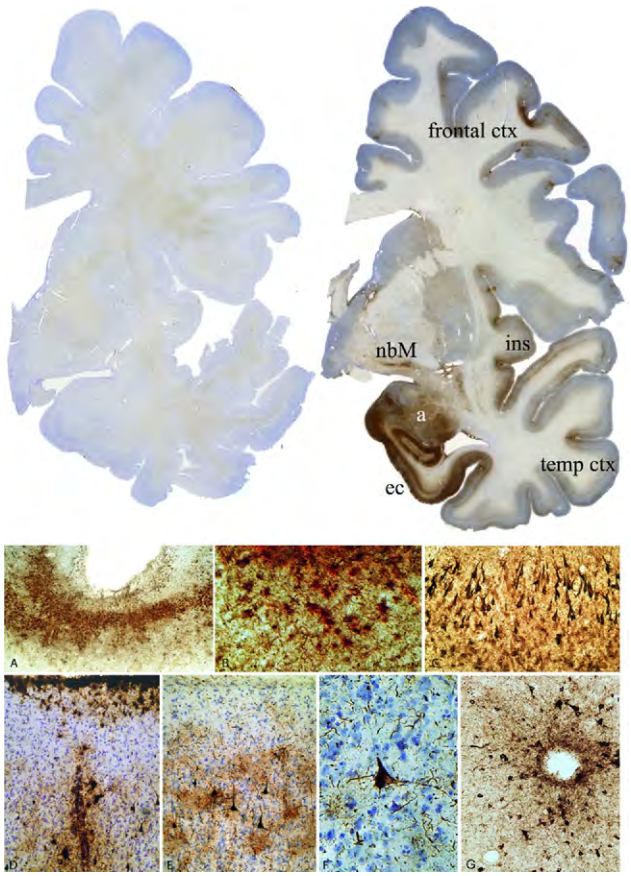

Figure 2. Microscopic pathology of chronic traumatic encephalopathy (CTE). **Top panel:** Phosphorylated tau (AT8) immunostained coronal hemisections of a normal brain (left) and a brain from a retired professional football player with CTE (right). The brain with CTE, showing severe neurofibrillary degeneration of the amygdala (a), entorhinal cortex (ec), temporal cortex, insular cortex (ins), nucleus basalis of Meynert (nbM), and frontal cortex. The cortical changes are most severe at the depths of the sulci. **Lower panels:** (A) Tau neurofibrillary tangles (NFT) are often prominent at depths of the sulci (AT8 immunostain, original magnification ×60). (B) Subpial tau immunoreactive tangles are found in both neurons and astrocytes (double immunostained section for GFAP (red) and AT8 (brown), showing colocalization of tau and GFAP; original magnification ×350). (C) Extremely dense NFTs are found in the medial temporal lobe structures, including CA1 of the hippocampus, shown here. Senile plaques are absent (AT8 immunostain, original magnification ×150). (D) NFTs and astrocytic tangles tend to be centered around small blood vessels and in subpial patches (AT8 immunostain, original magnification ×150). (E) NFTs characteristically involve cortical layers II and III (AT8 immunostain, original magnification ×150). (F) NFT in a Betz cell of primary motor cortex (AT8 immunostain, original magnification ×350). (G) Perivascular tau immunoreactive NFTs are a characteristic feature of CTE (original magnification ×150).

CLINICAL PRESENTATION

To date, we have found more than 50 neuropathologic-confirmed cases of CTE, with patients ranging in age from teens to their 80s, and occurring in individuals who have played contact sports as well as military personnel exposed to blast injuries. During diagnosis, the neuropathologist (A.C.M.) remained blinded to the patient's clinical history (eg, medical, behavioral, cognitive, brain trauma exposure) until after the neuropathologic examination was completed and the pathologic diagnosis was made. This clinical history was obtained from semistructured interviews with next of kin and by review of medical records by the neuropsychologist (R.A.S.) who remained blinded to the neuropathologic results until after all aspects of the clinical history were completed. The results of this experience, along with our previously published review of the literature of CTE and/or dementia pugilistica as of 2009, resulted in a surprisingly consistent description of the clinical course and presentation of CTE [3].

It is important to note that the clinical presentation of CTE is distinct from the long-term sequelae of a concussion or from PCS. CTE is not the accumulation of symptoms from the earlier injuries. Rather, the symptoms of CTE, like other neurodegenerative diseases, results from the progressive decline in functioning of neurons or of the progressive neuronal death. That is, when there is sufficient disruption of normal neuronal functioning, symptoms specific to the area(s) of that disruption will begin to exhibit. Based on our currently unpublished observations from our series of more than 50 cases, there may have been no earlier symptoms of concussion or PCS, and, therefore, the symptoms of CTE begin insidiously and are apparently unrelated to earlier impairment. In other cases, PCS symptoms may completely abate months or years before the onset of CTE symptoms. In still other cases, there may be overlap; that is, the PCS symptoms may begin to abate but CTE symptoms gradually worsen at the same time.

Typically, CTE symptoms present in midlife, usually years or decades after the end of exposure to repetitive brain trauma (ie, retirement from sports). Although we have seen the earliest stages of neuropathologic changes of CTE in the brains of individuals in their teens or early 20s, it is unclear if any cognitive, mood, or behavioral symptoms at that time

Table 3. *Early clinical presentation of chronic traumatic encephalopathy*

Short-term memory problems
Executive dysfunction (eg, planning, organization, multitasking)
Depression and/or apathy
Emotional instability
Impulse control problems (eg, disinhibition, having a "short fuse")
Suicidal behavior

Table 4. *Later clinical presentation of chronic traumatic encephalopathy*

Worsening memory impairment
Worsening executive dysfunction
Language difficulties
Aggressive and irritable behavior
Apathy
Motor disturbance, including parkinsonism
Dementia (ie, memory and cognitive impairment severe enough to impair social and/or occupational functioning)

were directly the result of the mild neuronal disruption caused by the disease. When symptoms begin, the onset is earlier than that of sporadic AD and usually earlier than that of frontotemporal lobar degeneration or frontotemporal dementia (FTD). Symptom progression is slow and gradual, often over several decades. Early symptoms (Table 3) are consistent with those expected from the neuropathologic changes observed at autopsy. These include impairments in cognition (eg, from medial temporal and dorsolateral frontal degeneration), mood (eg, amygdala degeneration), and behavior (eg, amygdala and orbitofrontal degeneration). Although the cognitive difficulties (including short-term memory problems and executive dysfunction) are similar to those seen in other neurodegenerative diseases, the mood and behavioral symptoms may be the most concerning, especially to family members and coworkers [35]. These include depressed mood and/or apathy, emotional instability, suicidal ideation and behavior, and problems with impulse control, especially having a "short fuse." Substance abuse (sometimes fatal) and suicide are not uncommon.

As the disease progresses, symptoms become more severe and their scope broadens. The primary clinical features of worsening CTE are listed in Table 4, including worsening memory impairment (although it may not resemble the severe rapid forgetting typical of the hippocampal episodic memory impairment of AD), worsening executive dysfunction (eg, poor planning, organization, multitasking, judgment), language difficulties (including speech output), aggressive and irritable behavior (including physical aggression), apathy (sometimes profound and in contrast to the outward aggressive behavior), and motor disturbance (most frequently parkinsonism, as well as difficulties with gait and falling). As the condition progresses, instrumental activities of daily living worsen and the symptoms are severe enough to impair social and/or occupational functioning, with eventual dementia.

RISK FACTORS

Repetitive brain trauma appears to be a necessary variable for the development of CTE but may not be sufficient. All neuropathologically confirmed cases of CTE have had a history of brain trauma exposure but not all individuals with exposure to brain trauma develop CTE. A major goal of CTE research must be epidemiologic and prospective studies to identify the specific risk factors for the development of this neurodegenerative disease.

An important potential risk factor for CTE may be genetic predisposition. There have been preliminary studies that linked the apolipoprotein E (APOE) gene, specifically the APOE $\varepsilon 4$ allele, to worse cognitive functioning in boxers and professional football players, and to prolonged recovery after a single TBI [36-38]. In our case series of 12 professional football players and boxers with neuropathologically confirmed CTE, 5 were APOE $\varepsilon 4$ carriers, and 2 of those were homozygous for the $\varepsilon 4$ allele [32]. Although these findings taken together suggest that APOE may be a susceptibility gene for CTE, much more research is required. Moreover, because of the presence of TDP-43 in CTE brains and the similar clinical presentations between CTE and FTD, additional genes associated with familial FTD, and perhaps ALS, may prove to play a role in CTE risk.

There are many other nongenetic variables to consider when evaluating an individual's risk of developing CTE (Table 5). As stated above, all confirmed cases of CTE have had a history of repetitive brain trauma. However, the specific nature of the brain trauma exposure necessary for the development of the disease is not yet known. For example, it is unknown if CTE is any more likely to manifest after a few severe TBIs versus numerous repetitive subconcussive impacts. Further complications arise when comparing impact exposure and type both between and within sports. For example, although boxing and American football both have a high incidence of head impacts, results of a study have shown that boxers are exposed to a greater amount of rotational forces, whereas American football players receive more linear blows [39]. More recently, a study that used an accelerometer-based system in the helmets of 3 college American football teams throughout a season found that head-impact exposure differed significantly based on position [9]. Linemen (both offensive and defensive) and linebackers received more impacts per practice and game than other positions. Lineman, linebackers, and defensive backs received more impacts to the front of the head than the back, whereas quarterbacks had a higher percentage of impacts to the back of the head compared with the front [9]. Future implementation of similar technology will help clarify the type and volume of impacts in American football and other sports, which can be extrapolated to estimate total impacts received over the career duration and may translate to information on the risk of developing CTE based on the sport and position.

Age at the time of head injury also may affect an individual's risk of developing CTE later in life, although the relationship is not yet understood. It has been suggested that the increased plasticity of a younger brain may allow a younger individual to better compensate and recover after brain injury [40]. However, current literature indicates that a younger brain may be more susceptible to diffuse brain injury, which leads to more pronounced and prolonged

Table 5. *Potential additional risk factors for chronic traumatic encephalopathy*

Potential General Risk Factor	Specific Examples and/or Questions
Genetics	APOE ε4; MAPT; GRN; TARDP
Family history	First- and/or second-degree relatives with history of dementia
Type of brain trauma exposure	Symptomatic concussions; asymptomatic subconcussive blows; blast wave; minimum gravitational force; degree of axonal injury and/or microhemorrhages
Age and duration of brain trauma exposure	Susceptibility period during youth; years of overall exposure
Frequency of brain trauma exposure	Minimum number of injuries (eg, can one moderate-severe TBI lead to CTE, without any additional repetitive concussions or subconcussive exposure history?); amount of "rest" (and overall time interval) between injuries
Chronic inflammation	Obesity, hypertension, diabetes, and heart disease may exacerbate neurodegeneration and NFT formation
Cognitive reserve	Greater cognitive reserve (or brain reserve capacity) may be less likely to display the clinical symptoms associated with the neurodegeneration or exhibit them later in the neuropathologic process
Gender	Are women at greater risk if they had the same exposure as men?
Race	Are there racial differences in risk?

CTE = chronic traumatic encephalopathy; NFT = neurofibrillary tangles; TBI = traumatic brain injury.

cognitive deficits [41,42]. Therefore, it is possible that exposure to repetitive brain trauma at an early age may increase the risk of CTE more than exposure later in life, although this has yet to be proven. Gender also may play a role, as girls and women appear to be at greater risk for concussion and PCS-related symptoms, although this may be due in part to girls and women being more forthcoming when reporting their symptoms [43,44]. Other health-related variables may affect the neurodegeneration and clinical symptom spectrum associated with CTE. For example, chronic inflammation associated with obesity, hypertension, diabetes, and heart disease may exacerbate neurodegeneration and NFT formation in AD [45-48]. Cognitive reserve (ie, differential resilience to the clinical presentation of underlying neuropathologic disease) also may play a role in the clinical manifestation of CTE neuropathology. That is, given identical neuropathologic severity, individuals with greater cognitive reserve may be less likely to display the clinical symptoms of the disease than individuals with less cognitive reserve [49].

ONGOING AND FUTURE RESEARCH

As stated above, CTE is a neuropathologically distinct disorder, different in many ways from AD, FTD, sporadic ALS, Parkinson disease, or other neurodegenerative diseases. Nevertheless, its clinical presentation can be similar to these diseases, especially in the later stages when subtleties in presentation are less likely to be delineated. In recent years, research to investigate other neurodegenerative diseases, for example, AD, has been moving toward the integration of clinical (eg, neurologic, neuropsychological), biologic (eg, cerebrospinal fluid [CSF] and/or functional neuroimaging measurements of proteins), anatomical (eg, structural neuroimaging), biochemical (eg, magnetic resonance spectroscopy), and genetic (eg, APOE genotype) information for the purposes of early detection, differential diagnosis, treatment, and prevention [50]. In addition, recent advances in CSF biomarkers have resulted in a "signature" biomarker for AD of low CSF Aβ and elevated CSF tau [51]. Our group has recently begun investigations aimed at developing biomarkers for CTE through funding from the National Institute of Neurological Disorders and Stroke, the National Institute on Aging, and the National Institute of Child Health and Human Development. Through this research on biomarker development for CTE, accurate diagnostic criteria will be able to be proposed and validated by using similar approaches to the recently published revised diagnostic criteria for Alzheimer disease [52-55]. This approach includes clinical symptoms and history combined with objective biomarker evidence of the disease. This method will allow for the detection and diagnosis of the CTE in the early symptomatic stages and, possibly, in the preclinical stages of the disease. Theoretically, the earlier in the disease process one can intervene, the more effective disease-modifying agents will likely be. If a disease-modifying agent (eg, a tau antagonist) can be developed, proven effective, and has an adequate risk profile, then preclinical intervention with the drug could result in such a long delay in symptom presentation that there would be de facto prevention of the clinical disorder or CTE.

DISCUSSION

There has been a tremendous growth in the awareness of CTE in both scientific and lay circles in recent years. What was believed to be a very rare disease only seen in boxers is now commonly discussed as a potential consequence of repetitive brain trauma seen in multiple different sports and at all levels of play. In contrast to the rapid increase in media coverage, new policies, and culture change, the rate of new published research on CTE has been relatively slow. Although we know much more about certain aspects of the disease now compared with just 5 years ago, especially the neuropathology

and clinical history associated with CTE, we remain in infancy in the study of CTE. Because it has been well over 100 years since AD was first described, and there remains no consensus as to the underlying causal mechanism of AD, no highly accurate method of in vivo AD diagnosis, and no available Alzheimer disease–modifying intervention, it should not be surprising that many critical questions remain regarding CTE. These questions include the following: What is the underlying mechanism of disease? What are the risk factors for disease (including susceptibility genes, brain trauma exposure variables, and others)? What is the population prevalence of CTE? What is the incidence of CTE? How common is the disease among individuals with specific athletic and/or military histories? How can CTE be detected and diagnosed accurately during life? How can CTE be prevented? What interventions can result in successful disease modification?

Only through further study (including animal modeling, basic and translational investigations, and clinical and epidemiologic research) can these questions be answered and can CTE move from a disease only diagnosed postmortem to one that can be identified in life and eventually be treated, prevented, and cured. In addition, by addressing these critical questions, new research findings will provide policy makers with urgently needed scientific knowledge to make appropriate guidelines regarding the prevention and required treatment of brain trauma in all levels of athletic involvement as well as the military theater.

CONCLUSION

CTE has been linked to participation in contact sports such as boxing, American football, and hockey, and has been seen in individuals with non–sports-related histories of repetitive head injuries. It is believed that repetitive brain trauma, with or without symptomatic concussion, sets off a cascade of events that results in neurodegenerative changes marked by a unique tauopathy and TDP-43 proteinopathy. Symptoms may begin years or decades after the cessation of brain trauma exposure although earlier than most other neurodegenerative diseases. Early symptoms include a decline of memory and executive functioning, depression, suicidal ideation and/or behavior, and poor impulse control. Disease progression is relatively slow and eventually leads to dementia. In some individuals, CTE may lead to a motor neuron disease, similar to ALS. Recent neuropathologic research suggests that CTE may be more widespread than previously believed. Given the millions of athletes participating in contact sports that involve repetitive brain trauma, as well as military troops exposed to repetitive brain trauma from blast and other injuries and others in society who experience repetitive head injuries, CTE represents an important public health issue.

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Policy and Best Practice Outcomes from the Concussion in Sports Collegiate Medical Summit Draft 04/12/2010

The NCAA hosted a Concussion in Sports Collegiate Medical Summit to review policies and best practices for the medical evaluation and management of concussions appropriate to the NCAA collegiate environment. Determination of appropriate care and treatment of student-athletes injuries and illness are best handled at the local institutional level; however, establishing a set of best practices can help provide consistency and encourage collegiate healthcare professionals to follow a medical model that has physician oversight and direction. Institutions should consider the healthcare of student-athletes a critical component of their athletics services as each member school is responsible for protecting the health and safety of its student-athletes. Documentation of these steps and actions at the institutional level is critical to creating transparency of policy, provider roles and the events that lead to an appropriate return-to-play.

Relative to concussions and head injuries, institutions should be prepared to respond to immediate emergency situations (e.g., intracranial hemorrhage) but also continued evaluation and care for non-emergency yet serious conditions (concussion). Concussion however is not the only known condition that can be anticipated to occur within athletics. Student-athlete catastrophic injuries and illnesses also include heat illness, spine injury, cardiac arrest, respiratory distress (e.g. asthma), and sickle cell trait collapses.

With this in mind, institutions should have both a written emergency action plan as well as a written concussion management plan on file such that a student-athlete who exhibits signs, symptoms or behaviors consistent with a concussion should be removed from practice or competition and evaluated by an athletics healthcare provider with experience in the evaluation and management of concussion. As recommended by the NCAA CSMAS in December 2009, based solely on signs and symptoms, athletes who are rendered unconscious or have amnesia or persistent confusion shall not be permitted to continue for the remainder of the day. A student-athlete diagnosed with a concussion should be withheld from the current competition or practice and not return to participation for the remainder of that day. Student-athletes that sustain a concussion outside of their sport should be managed in the same manner as those sustained during sport activity.

The cornerstones of proper concussion management are proper identification of the injury, rest until all symptoms resolve at rest and upon exertion, return to baseline levels of functioning (e.g. neurocognitive, balance) , and a stepwise program of exertion before return to sport. The student-athlete should be monitored for recurrence of symptoms both from physical exertion and also mental exertion, such as reading, phone texting, computer games, working on a computer, classroom work, or taking a test.

Given the dynamic and evolving nature of the injury, concussions are not always immediately apparent. Healthcare professionals should take time to evaluate the disposition of the student-athlete over hours and days following the injury. The collegiate healthcare model should support having appropriate access to providers on the sidelines as team physicians and athletic trainers are in the best position to know the subtle changes that may identify a concussion among their student-athletes.

Neuropsychological testing has proven to be a very effective tool in assessing neurocognitive changes following concussion and can serve as an important component of an institution's concussion management plan. However, neuropsychological tests should not be used as stand alone measures to diagnose the presence or absence of a concussion and should not be used in lieu of a comprehensive assessment by qualified healthcare providers.

Healthcare professionals should assume a concussion when unsure and waiting for final assessment. When in doubt, sit the athlete out. Institutions should ensure healthcare professionals attain continuing education on concussion evaluation and management annually. Structured and documented education of student-athletes and coaches is also recommended to improve the success of a consistent concussion management program.
Beyond the current concerns around concussion prevalence, evaluation and management,

Next Steps: The CSMAS will review the following and determine if the suggested policy actions, with or without modifications, should be forwarded for Executive Committee consideration. The Executive Committee would be asked to consider the platform as a policy, in part or as a whole; whether possible legislative concepts could be forwarded to each Divisional governance body for adoption consideration; and/or whether a subgroup should be formed to further evaluate specific concepts.

The following suggested policy actions outline a comprehensive approach (education, legislation, policy, and universal best practices) that the NCAA and each of its member institutions should consider when developing a plan to address concussion in sport.

The committee believed that an educational video on concussions should be developed targeting both student-athletes and coaches and used by institutions annually. Concussions presented themselves differently than other common orthopedic injuries such as an ankle sprains and muscle strains. A better understanding of why a student-athlete needs physical and mental rest, often missing team video session or meetings is important for both coaches and student-athletes to support.

Draft Best Practices for a Concussion Management Plan for all NCAA Institutions

1. Institutions should require student-athletes to sign a statement so that student-athletes accept the responsibility for reporting their injuries and illnesses to the institutional medical staff, including signs and symptoms of concussions. During the review and signing process student-athletes should be presented with educational material[1] on concussions.
2. Institutions should have on file and annually update an emergency action plan[2,3,4] for each athletics venue to respond to student-athlete catastrophic injuries and illnesses, including but not limited to concussions, heat illness, spine injury, cardiac arrest, respiratory distress (e.g. asthma), and sickle cell trait collapses. All athletics healthcare providers and coaches should review and practice the plan at least annually.
3. Institutions should have on file an appropriate healthcare plan[5] that includes equitable access to athletics healthcare providers for each NCAA sport.
4. Athletics healthcare providers should be empowered to have the unchallengeable authority to determine management and return-to-play of any ill or injured student-athlete, as he or she deems appropriate. For example, a countable coach should not serve as the primary supervisor for an athletics healthcare provider nor should they have sole hiring or firing authority over that provider.
5. Institutions should have on file a written team physician–directed concussion management plan[2,6] that specifically outlines the roles of athletics healthcare staff (e.g., physician, certified athletic trainer, nurse practitioner, physician assistant, neuropsychologist, neurosurgeon). In addition, the following components have been specifically identified for the collegiate environment:
   a. Institutions should ensure coaches have acknowledged they understand the concussion management plan, their role within the plan and that they received education[1] about concussions.
   b. Athletics healthcare providers should practice within the standards as established for their professional practice. (e.g., physician[7], certified athletic trainer[8], nurse practitioner, physician assistant, neurologist[9], neuropsychologist[10])
   c. Institutions should record a baseline assessment[6,10,11,12] for each student-athlete prior to the first practice in the sports of baseball, basketball, diving, equestrian, field hockey, football, gymnastics, ice hockey, lacrosse, pole vaulting, rugby, soccer, softball, water polo, and wrestling, at a minimum. The same baseline assessment tools should be used post-injury at appropriate time intervals. The baseline assessment should consider one or more of the following areas of assessment.
      - At a minimum, the baseline assessment should consist of the use of a symptoms checklist and standardized cognitive and balance assessments (e.g., SAC; SCAT; SCAT II[6]; Balance Error Scoring System (BESS); Neurocom; Wii Fit Concussion Balance Testing).
      - Additionally, neuropsychological testing (i.e., paper and pencil; computerized) has been shown to be effective in the evaluation and management of concussion as noted above. The development and implementation of a neuropsychological testing program should be performed in consultation with a neurology specialist (e.g., neuropsychologist , neurologist, neurosurgeon). Ideally, post injury neuropsychological test data should be interpreted by a neurology specialist.
   d. When a student-athlete shows any signs, symptoms or behaviors consistent with a concussion, the athlete should be removed from practice or competition and evaluated by an athletics healthcare provider with experience in the evaluation and management of concussion.
   e. A student-athlete diagnosed with a concussion shall be withheld from the competition or practice and not return to activity for the remainder of that day.
   f. The student-athlete shall receive serial monitoring for deterioration. Athletes shall be provided with written instructions upon discharge; preferably with a roommate, guardian, or someone that can follow the instructions.

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g. The student-athlete shall be evaluated by a team physician as outlined with within the plan. Once asymptomatic and post-exertion assessment is within normal baseline limits, return to play shall follow a medically supervised stepwise process.

h. Final authority for Return-to-Play[13] shall reside with the team physician or his/her designee.

6. Institutions shall document the incident, evaluation, continued management, and clearance of the student-athlete with a concussion.
7. Purposeful or flagrant head-to-head contact in any sport should not be permitted and should be subsequently reviewed for consideration of further sanctions.

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Core Principle 1: Education.

Action 1.1: Consider the development of materials to educate student-athletes about concussions, its signs and symptoms, their risks and importance for prompt reporting.

Action 1.2: Consider the development of a "concussion management" continuing education program for medical professionals to gain experience and seek certification of completion in knowledge attainment about prevention, diagnosis, management and return-to-play.

Action 1.3: Consider the development of a concussion awareness education program for coaches to gain certification of completion of knowledge attainment about concussions, its signs and symptoms, their risks and importance for prompt referral for medical assessment. This component could become part of first aid certification. Primary sports of focus should begin with the sports of baseball, basketball, diving, equestrian, field hockey, football, gymnastics, ice hockey, lacrosse, pole vaulting, rugby, soccer, softball, water polo, and wrestling, at a minimum.

Action 1.4: Consider ongoing evaluation to ensure progress and modify actions as indicated.

Core Principle 2: Appropriate healthcare model.

Action 2.1: Consider a requirement that ensures medical supervision and positional authority in all divisions for the evaluation and return to play decisions of student-athlete injury and illnesses incidental to a student-athlete's participation in intercollegiate athletics. For example:

2.1.a. Team Physician. "Institutions shall designate a licensed, board certified physician to serve as team physician authorized to oversee the medical services for injuries and illnesses incidental to a student-athlete's participation in intercollegiate athletics."

2.1.b. Athletics Healthcare Coordinator. "Institutions shall designate an individual with a healthcare credential and who is a current employee of the institution to serve as athletics healthcare coordinator. An individual so designated shall coordinate, monitor, and evaluate the healthcare, emergency action plans, and event coverage services for student-athletes as determined by the member institution. An individual so designated shall not hold a coaching position in the athletics department nor shall a countable coach have a sole hiring or firing role in determining this individual's employment. The individual shall be identified as "athletics healthcare coordinator" in the NCAA membership database annually."

Action 2.2: Consider a requirement that ensures equitable access to healthcare across sports and divisions for all sponsored activities. For example:

2.2.a. Emergency Action Plan. "Institutions shall have on file an emergency action plan for each athletics venue to respond to student-athlete catastrophic injuries and illnesses, including but not limited to concussions, heat illness, spine injury, cardiac arrest, respiratory distress (e.g. asthma), and sickle cell trait collapses. All athletic healthcare providers and coaches shall review and practice the plan at least annually."

2.2.b. Healthcare Plan. "Institutions shall have on file an appropriate healthcare plan that includes equitable access to athletics healthcare providers for each NCAA sport. Athletics healthcare providers shall not report to a countable coach and shall be empowered to have the unchallengeable authority to determine management and return-to-play of any ill or injured student-athlete, as he or she deems appropriate**.**"

Action 2.3: Consider a requirement that requires Division I coaches to seek Sports Safety Training. Note: Division II and Division III already have legislation in place. For example:

2.3.a. Sports-Safety Training (Division I)**.** "Each head coach and all other coaches who are employed full time at an institution shall maintain current certification in first aid, cardiopulmonary resuscitation (CPR) and automatic external defibrillator (AED) use. In sports in which the safety exception is applicable in Bylaw 17, a coach

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conducting the safety or skill instruction shall maintain current certification in first aid, cardiopulmonary resuscitation (CPR) and automatic external defibrillator (AED) use."

Action 2.4: Consider the development of an audit tool for institutions to use to help assess their care and coverage needs based around the sport risk data, NCAA Athletics Certification criteria, Self-Study requirements and NCAA Bylaws per division.

Core Principle 3: Concussion-Specific Policy and Legislation.

Action 3.1: Consider playing rules in all sports that allow for stoppage of play and medical evaluation of an injured athlete, specifically for a possible concussion. For example:

3.1.a. Playing Rule. "An athlete who exhibits signs, symptoms, or behaviors consistent with a concussion, either at rest or exertion, shall be immediately removed from practice or competition and shall not return to play until cleared by a physician or her/his designee."

Action 3.2: Consider a requirement for a concussion-specific management plan in all Divisions so that if a concussion is suspected, the athlete is removed from the contest or practice until they can be examined by a healthcare professional (ATC or MD). For example:

3.2.a. Concussion Management Plan. "Institutions shall have a concussion management plan on file such that a student-athlete who exhibits signs, symptoms or behaviors consistent with a concussion shall be removed from practice or competition and evaluated by an athletics healthcare provider with experience the evaluation and management of concussion. Student-athletes diagnosed with a concussion shall not return to participation for the remainder of that day. Medical clearance shall be determined by the team physician or their designee according to their concussion management plan."

Action 3.3: Consider the development and distribution of best practices for member institutions to consider in their development of a concussion management plan.

Action 3.4: Consider a plan to monitor and report on division and campus action on concussion policy and legislation.

Core Principle 4: Athletics Administration.

Action 4.1: Acceptance of Responsibility. Consider adding language to the NCAA Student-Athlete Statement so that student-athletes accept the responsibility for reporting their injuries and illnesses to the institutional medical staff, including signs and symptoms of concussions. During the review and signing process student-athletes shall be presented with educational material on concussions.

Action 4.2: Record keeping. Consider a requirement that requires institutions to document student-athlete athletic-related injuries and illnesses.

Core Principle 5: Communication.

Action 5.1: Develop and promote the use of consistent messaging, while allowing for sport-specific and division-specific needs.

Action 5.2: Develop a dedicated Web site to serve as a resource center for all materials.

Action 5.3: Utilize the collective platform of NCAA Conferences to educate their respective institutions on the issue and to be advocates for change in athletics.

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Sample Frequently Asked Questions

What is a concussion? Concussions are brain injuries. Concussion is a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces such as a blow to the head or body. Signs and symptoms very among individuals can present well after the incident. Brain injuries are not a part of sport and should be considered a serious health condition.

Does reporting or the presence of signs, symptoms or behavior of a concussion automatically require the student-athlete to be withheld for the remainder of the day? No. There is a medical evaluation period just as there is with any other injury or illness during which athletics healthcare providers take time to serially evaluate the athlete condition. The athlete may have a condition other than a concussion.

If an athlete is diagnosed with a concussion are they removed for the remainder of the day? Yes. In fact, symptoms often appear on subsequent days or become more pronounced. Studies on college-aged athletes have shown that they are more susceptible to a second concussion within the first 10 days. Some evidence suggests that return to play can be faster the sooner an athlete with symptoms begins to rest. Return to play can take days, weeks or even months.

Should all student-athletes undergo computerized neuropsychological testing? Not necessarily. Using a computerized tool should not replace the clinical judgment or management of an athletic healthcare provider. Institutions electing to use a computerized neuropsychological tool should do so with the consultation of a neuropsychologist, particularly upon evaluating post-concussive testing results.

Does a designated Team Physician have to be an employee of the institution? No.

What are the preferred qualifications of a Team Physician? Team physicians should be in good standing with their state licensure board and be credentialed as a medical doctor (M.D.) or doctor of osteopathic medicine (D.O.).

Can the Team Physician or Head Athletic Trainer also serve as the Athletics Healthcare Coordinator? Yes, as long as they are an employee of the institution.

Who are considered athletics healthcare providers? Primarily, certified athletic trainers provide the sideline and clinical healthcare services for student-athletes at NCAA athletics departments nationwide as part of a physician supervised medical model. In addition, the athletics healthcare team can include nutritionists, psychologists, physical therapists, nurse practitioners, physician assistants, neuropsychologists, and other health-care professionals responsible for providing access to quality sports healthcare to student-athletes. These individuals should meet current state-credentialing requirements for their profession (e.g., licensure, certification, registration).

What should be considered when evaluating "appropriate healthcare coverage for each sport"?
Healthcare coverage decisions involve more than what athletic events and training sessions should be covered; the qualifications of staff conducting those duties are important; the scope of services as required by NCAA bylaws and sport playing rules; administrative responsibilities; and prevention services.

Does every athletics event need to have an appropriate healthcare provider present or on-site? Not necessarily. The healthcare coverage plan will take into consideration the emergency action plans for venues, the qualified coaches under the sports safety training legislation trained to respond to an emergency as well as the systemic approach to determine additional healthcare needs for the event. Criteria for evaluating event risk might include the type and frequency of contact in the sport, location of the event, the emergency plan coverage, local standards of care, NCAA injury surveillance data, NCAA Sport Playing Rules, and legislation. The National Athletic Trainers' Association has an online tool to help institutions determine staffing healthcare needs based on institutional philosophies.

Do teams have to travel with an appropriate healthcare provider? Not necessarily. This will be determined by the systematic evaluation of the sport. If a sport team does not travel with a healthcare provider, that team will rely on the host institution's healthcare providers and emergency plans.

Can students in athletic training education programs be counted as an appropriate healthcare provider? No. They may still serve as first responders similar to coaches but cannot provide sole healthcare coverage for student-athletes of a team both at home or while traveling.

Why should an institution designate an individual on staff as an Athletics Healthcare Coordinator? Coordinating the medical, healthcare, and support services for today's student-athlete are critical components to an effective athletics department. Institutions should document in writing all of the components of the healthcare services. In addition, the coordinator can serve as a critical point person from the national perspective to help deliver key communications on critical issues such as policy, legislation, and best practices on concussions, drug testing, sickle cell trait, injury prevention, cardiac, H1N1, pandemic flu, skin infection and other communicable disease outbreaks. Additional coordinating activities could include a year-round assessment for events such as those traditional and non-traditional season practices and competitions, skill instruction sessions, and year-round strength and conditioning through a systematic approach to determine the appropriate level of medical coverage for each sport at an institution. Examples of relevant administrative duties that should be monitored for NCAA institutions include drug testing, insurance claims management, medical examinations, team travel, compliance, medical record keeping, drug education programs, coach meetings, medical hardship waivers processing, rehabilitations, meeting with recruits, coach and athlete education, first aid/cpr certification training, championship/tournament hosting, academic teaching, clinical supervision of students, concussion management programs, hosting physician clinics, budgeting, staff scheduling, inventory, staff performance evaluations, medications, student-athlete transport for doctor visits, and visiting team services.

Concussion Perspectives

- Legal: The status any litigation against the NCAA, membership institutions and other leagues for failure to adequately manage concussions? Future considerations.

- Medical: The latest research regarding the connection between concussions and suicide, depression, CTE, etc? Latest on grading severity: severe vs. moderate? Latest on repetitive concussions? Repetitive head hits not leading to concussions? Return to Play criteria?

- Ethics: What do professional associations say regarding the ethics relating to putting student/athletes in play after evidence of a concussion? Are all associations in alignment on this aspect? The role of medical staff / coach reporting lines and influence of hiring/firing relationships.

- Sport: Does normal (i.e., actual) treatment of concussion-like symptoms vary sport by sport? Division by division? During championships-type competition?

- Costs: With sickle cell trait, there is confusion about actual costs for assessment, care, and education. Is this also true with concussion management?

- Compliance: While member institutions may have a policy regarding concussion management, is there evidence that such policies are being followed?

- Education: Are institutions conducing education? Is the education being assessed for effectiveness? If we ask athletes, do they understand the parameters of concussion management? If we ask coaches? If we ask athletic trainers? If we ask s/c coaches?

National Center for Catastrophic Sports Injury Research (NCCSIR)

March 15, 2012 Meeting Summary

The NCCSIR hosted a meeting in Chapel Hill, NC on March 15, 2012 to discuss the potential expansion of the NCCSIR and to identify goals of an expanded Center. Meeting participants included Fred Mueller, David Klossner, Bob Colgate, Dawn Comstock, Steve Marshall, Doug Casa, Kevin Guskiewicz, Bob Cantu (by phone), and Jon Drezner (by phone).

The meeting was called following discussions over the past year among several of the participants about creating a more efficient and streamlined surveillance of sport-related catastrophic injury/illness, as well as an investigative model that could drive evidence-based injury prevention models.

Fred Mueller started the meeting with an overview of the Center's long history, dating back to 1965 when the Center was founded by Carl Blyth at UNC-Chapel Hill. Fred Mueller (Research Director) and Bob Cantu (Medical Director) have led the Center for the past 25 plus years, and have published an Annual Report annually since 1981. The Center has survived on a limited budget with annual contributions from the NCAA, American Football Coaches Association, and UNC Department of Exercise and Sport Science, yet much has been accomplished. Fred has tracked injuries through newspaper clipping services and internet based search engines, and together he and Bob have written the annual reports on catastrophic sports injuries/illnesses which have helped various sporting organizations develop health and safety policies.

Recent research has noted gaps in various registries and research databases based on unique case definitions and under reporting due to methodological variances. The development of a more robust national active surveillance program would create a more accurate and efficient sport catastrophic injury monitoring registry.

With this in mind, the NCCSIR will expand to include two other research universities creating the "Consortium for Catastrophic Injury Monitoring in Sport".

The ***mission*** of the **National Center for Catastrophic Sport Injury Research (NCCSIR)** is to conduct surveillance of catastrophic injuries and illnesses related to participation in organized sports in the United States at the collegiate, high school, and youth levels of play. In working through a consortium for catastrophic injury monitoring, the NCCSIR aims to track cases through a systematic data reporting system that allows for longitudinal investigation of athletes suffering from catastrophic injuries and illnesses. The goal of the Center is to improve the prevention, evaluation, management, and rehabilitation of catastrophic sports-related injuries.

Themes that developed from the meeting include the following:

- The target group (athletes) for injury surveillance will include professional, collegiate, high school, and organized youth sports programs.

- The Center will capture fatality, catastrophic and survivor incidents longitudinally and report in both a sentinel and aggregated formats.

- The NCAA, NFSHA (and individual state associations), state epidemiologists (child death review board?), and national association of state medical examiners should all play a role in an expanded and more comprehensive surveillance program.
    - Surveillance can add a sentinel system aspect that has yet to be used in the sports setting.

- The "Consortium for Catastrophic Injury Monitoring in Sport" will be created within the NCCSIR. Doug Casa (KSI at UCONN) will lead the division on *non-traumatic illnesses* to include: heat stroke, sickle cell trait, and diabetes. Jon Drezner (UW) will lead the division on *cardiac injury/illness* to include all traumatic and non-traumatic cardiac injuries and illnesses. Kevin Guskiewicz (Gfeller TBI Research Center at UNC) will lead the division on *traumatic injuries* to include TBI, spine, lightning, and other. Each group will work synergistically with the director of NCCSIR to capture relevant surveillance data and follow-up reporting (investigations).

- Regional representatives, such as a group of certified athletic trainers from each state AT association will assist with reporting catastrophic cases, and then later be deployed to investigate cases. These representatives will work directly with the divisional directors and NCCSIR director in this expanded program. Funding for the work conducted by the regional representatives will be provided through the NCCSIR budget.

- Existing surveillance programs such as Datalys and RIO will be instrumental in our attempt to provide a more systematic approach to data collection. The *Datalys Center for Sports Injury Research and Prevention* currently collects all injuries (including fatalities and other catastrophic injuries) investigates on-field athletic events and all cardiac, head, spine, and exertion-related. RIO's internet-based high school sports injury surveillance system includes fatalities and other catastrophic injuries. The challenge will be how to expand the surveillance into the youth sports programs.

- In addition to the *surveillance* of catastrophic injuries/illnesses, the expanded program will work diligently to develop the *investigative model* to study each case with longitudinal follow-up. The NCCSIR Annual Report will be expanded to include new cases in all areas, as well as updates on the longitudinal follow-up of prior cases.

- An oversight board will be created for the program. Potential candidates include: Barry Boden, Randy Hanzlick (medical examiner from Atlanta), Randy Eichner, Andy Gundstein

(climatologist), Julie Gilchrist, Jon Kaltman (NHLBI?), Mark Proctor (Neurosurgeon), Mike Ackerman (Mayo)

- Who should do we enlist as NCCSIR partners?
  NCAA and NFSHA (primary collaborators and funding partners)
  AFCA, CDC, NATA, ACSM, NCAA catastrophic insurance vendor, NFL (secondary collaborators and funding partners)

- Different levels of data access will be established and agreed upon:
  1. The NCCSIR consortium researchers will have direct initial access for peer-reviewed publications.

  2. Public access to summary data will be provided through Annual Reports posted on the website(s).

  3. NCCSIR partners may be given access to specific data as requested for rule changes or policy-related issues. These requests will be reviewed by the oversight board.

  4. The oversight board may also review requests for researchers who may request access to more specific data for grant development.

  5. Partners will receive an annual population specific report with comparative data.

Q How will data be made available for medical legal cases, when the Center has investigated a case and information is requested by a plaintiff or defense attorney?

Phase II
- Training for medical examiners, coroners, medical staff, etc.
- Intervention tracking following incidents from local schools, states and rules making bodies
- Serve as a sentinel system
- What role should the Center play in deploying injury/illness prevention programs? - perhaps this is phase 2 of our initiative after the expanded NCCSIR is up and running efficiently.

- Funding Model:
  1. Initially $35k of annual funding for operating expenses will be needed at each of the divisional sites (UCONN, UW, UNC). This will allow for salary support for staff to track cases, assist in investigations, and work directly with the director of NCCSIR in developing the Annual Report.

  2. Initially $45k of annual funding for operating expenses will be needed for the director of NCCSIR. This will allow for partial salary for the director (~1/3 faculty release time to

direct NCCSIR, track catastrophic cases, coordinate investigations, and provide stipends to regional representatives for investigations).

In summary, the annual cost of the expanded NCCSIR will be approximately $150,000.

Next Steps

- Establish Oversight Board.
- Definition for a reportable incident.
- Develop common data elements for a standard case report.
- Seek IRB approval.
- Create regional reporting structure.
- Provide education to MEs.
- Secure funding.
- Create and maintain secure web reporting tool.
- Rebrand considerations.

The Inter-Association Task Force for Preventing Sudden Death in Collegiate Conditioning Sessions Best Practices Recommendations

Douglas J. Casa, PhD, ATC, FNATA, FACSM[a] (Chair); Scott A. Anderson, ATC[a]; Lindsay Baker, PhD[b]; Scott Bennett, MS, MSCC, SCCC, CSCS*D[c]; Michael F. Bergeron, PhD, FACSM[d]; Declan Connolly, PhD[c]; Ron Courson, ATC, PT, NREMT-I, CSCS[a]; Jonathan A. Drezner, MD[e]; E. Randy Eichner, MD[f]; Boyd Epley, MEd, FNSCA, RSCC*E[g]; Steve Fleck, PhD, CSCS, FNSCA, FACSM[g]; Rob Franks, DO, FAOASM[h]; Julie Gilchrist, MD[i]; Kevin M. Guskiewicz, PhD, ATC, FNATA, FACSM[a]; Kimberly G. Harmon, MD[e]; Jay Hoffman, PhD, CSCS, FNSCA, FACSM[g]; Jolie Holschen, MD, FACEP[j]; Peter Indelicato, MD[k]; Jon Jost, MS, RSCC*E[g]; Alan Kinniburgh, PhD[g]; David Klossner, PhD, ATC[l]; Christine Lawless, MD, MBA, FACC, FACSM, CAQSM[m]; Rebecca M. Lopez, PhD, ATC, CSCS[a]; Gerard Martin, MS, RSCC*D[g]; Brendon P. McDermott, PhD, ATC[a]; Jason P. Mihalik, PhD, CAT, ATC[n]; Bill Moreau, DC, DACBSP, CSCS[o]; Tom Myslinski, MS, RSCC[g]; Kelly Pagnotta, MA, ATC, PES[p]; Sourav Poddar, MD[l]; Brian Robinson, MS, LAT, ATC[a]; George Rodgers, MD, PhD[c]; Alan Russell, MS, ATC, PES, CSE[q]; Latrice Sales[l]; David Sandler, RSCC*D[g]; Rebecca L. Stearns, MA, ATC[p]; Chuck Stiggins, EdD[c]; Charlie Thompson, MS, ATC[a]; Reginald Washington, MD[m]

[a]National Athletic Trainers' Association; [b]Gatorade Sports Science Institute; [c]Collegiate Strength and Conditioning Coaches association; [d]American College of Sports Medicine; [e]American Medical Society for Sports Medicine; [f]Retired, University of Oklahoma; [g]National Strength and Conditioning Association; [h]American Osteopathic Academy of Sports Medicine; [i]Centers for Disease Control and Prevention; [j]American College of Emergency Physicians; [k]American Orthopaedic Society for Sports Medicine; [l]National Collegiate Athletic Association; [m]American College of Cardiology Council on Sports and Exercise Cardiology; [n]Canadian Athletic Therapists' Association; [o]United States Olympic Committee; [p]Korey Stringer Institute; [q]National Academy of Sports Medicine

Maximizing strength and conditioning sessions has become fundamental to sport. The right combination of strength, speed, cardiorespiratory fitness, and other components of athletic capacity can complement skill and enhance performance for athletes. A sound and effective training program that relies on scientific principles of exercise physiology and biomechanics intended to produce outcomes sensitive and specific to the sport should be the goals. Unfortunately, the athlete's development, health, and safety are sometimes overshadowed by a culture that values making athletes tough, instilling discipline and success at all costs.

This ill-conceived philosophy has been a key contributor to the alarming increase in collegiate athlete deaths and serious injuries during conditioning sessions. A total of 21 National Collegiate Athletic Association (NCAA) football players have died during conditioning workouts since 2000.[1] The 3 most common causes of the fatalities were (in order) exercise-related sudden death associated with sickle cell trait (SCT), exertional heat stroke, and cardiac conditions.[1] Seventy-five percent of the fatalities (n=16) were Division I football players.

Also, the incidence of exertional rhabdomyolysis in collegiate athletes appears to be increasing. Excesses in strength training and conditioning – workouts that are novel, too much, too soon,

and/or too long – have a strong connection to exertional rhabdomyolysis. Introducing full-intensity workouts too quickly is especially high risk: 11 of the 21 deaths occurred during day-1 or day-2 workouts.

Rule changes related to heat acclimatization procedures during August football practices enacted in 2003 and since have been extremely efficacious. In NCAA Division I bowl sub-division football, no player has died from practicing between 2003 and 2011 or playing in a game. Conditioning workouts continue to be a catalyst for catastrophic outcomes, however. It is imperative that similar guidelines are implemented to improve the safety of conditioning sessions. This consensus statement provides specific conditioning recommendations with the intent of ending conditioning-related morbidity and deaths of collegiate athletes.

1) Progressive Acclimatization is the Cornerstone of Safety
Conditioning periods should be phased in gradually and progressively to encourage proper exercise acclimatization and to minimize the risk of adverse effects on health. The first 7 to 10 days (at minimum, the first 4 separate-day workouts), of any new conditioning cycle (including, but not limited to: return in January, after spring break, return in summer, or return after an injury) are referred to as transitional periods. A written, progressive program of increasing volume, intensity, mode, and duration should be instituted for all transitional periods. These conditioning programs should be approved by a credentialed Strength & Conditioning Coaches (S&CCs) (see below). The S&CC should work cooperatively with medical staff (certified athletic trainer (ATC) and/or MD) when developing transitional workout plans, particularly if the athlete is recovering from an injury or if any uncertainty exists regarding the pace of exercise progression.

Transitional periods should invoke an appropriate work-to-rest ratio for the sport. A 1:4 work-to-rest ratio (with greater rest permissible) when conducting serial activity of an intense nature, for example, is a good starting place to emphasize recovery.

A qualified S&CC is knowledgeable about and employs acclimatization principles. Participation in summer workouts on campus under the supervision of an S&CC, with an ATC nearby, is recommended over unsupervised workouts elsewhere.

Training programs should be individualized and some will require a longer acclimatization process. An athlete at a different level of preparedness from his or her teammates (due to injury or time away from training) should utilize a training program tailored to his or her level.

2) Introduce New Conditioning Activities Gradually
Any new exercise introduced into a strength and conditioning program should be added in a deliberate, gradual fashion by a qualified S&CC. This guideline is true for any aspect of the regimen but is particularly important during the transitional periods of a conditioning program.

3) Do Not Use Exercise and Conditioning Activities as Punishment
Physical activity should not be used as retribution, for coercion, or as discipline for unsatisfactory athletic or academic performance or unacceptable behavior. No additional

physical burden that would increase the risk of injury or sudden death should be placed on any athlete under any circumstance.

4) Proper Education, Experience, and Credentialing of the S&CC

A) Education S&CCs are required to obtain an undergraduate degree to sit for the exam and become credentialed. It is recommended that the degree be in exercise science or a related field of study.

The strength and conditioning profession is urged to establish an accredited educational curriculum at the collegiate level for entry-level programs. A suitable amount of coursework should be dedicated to the health and safety concerns of and challenges facing athletes, with a focus on preventing sudden death. This includes the prevention, recognition, and management of on-field emergencies.

Continuing education requirements should ensure that certified strength and conditioning coaches engage in educational opportunities that provide applicable, up-to-date information regarding important health and safety topics, emergency procedures, and preventing sudden death. Content should be reviewed annually, so that timely topics can be incorporated.

B) Experience Collegiate S&CCs should have adequate mentoring and experience in order to independently design and implement individual and team conditioning programs.

C) Credentials S&CCs should be required to pass a certification examination credentialed by an independent accreditation agency. Competency standards, ongoing assessment, and continuing education requirements should be clearly documented.

S&CCs should maintain certification in first aid, cardiopulmonary resuscitation (CPR), and use of an automated external defibrillator (AED).

5) Provide Appropriate Medical Coverage

S&CCs should be present during all strength and conditioning sessions and be prepared to provide first aid as soon as an athlete shows signs of distress. S&CCs should be able to administer CPR, apply an AED and activate the emergency action plan (EAP) if needed. High-risk collegiate strength and conditioning sessions should have medical staff (athletic trainer or physician) present during each conditioning session (e.g., sprinting, timed sessions, mat drills, stations). For lower-risk conditioning sessions and the strength portion of a conditioning program, an S&CC should be present and an athletic trainer should be on-campus and accessible to assist if a problem arises. The institution should determine the need for and level of medical coverage for conditioning sessions. Among the parameters to be considered are squad size, type of athlete, time of year (immediately post-season, off-season, in-season, summer), expected environmental conditions, and planned duration and intensity level of the workout.

6) Develop and Practice Emergency Action Plans

Strength and conditioning venues should have an EAP specific to the venue, sport, and circumstances.[1] The EAP should be developed by the sports medicine staff with the input of all

concerned parties, approved by the head team physician, and, most importantly, reviewed and rehearsed at least annually by all staff involved. A conditioning session should not take place if those supervising the session are not familiar with the EAP.

7) Be Cognizant of Key Medical Conditions

The most likely causes of sudden death during collegiate strength and conditioning sessions are atraumatic cardiac conditions, exertional collapse associated with SCT, exertional heat stroke, and asthma.[1,3–8] The designated medical supervisor must be familiar with the characteristics of exertional collapse and the differential diagnosis of the conditions listed above.

Institutional, governing, and credentialing agencies for S&CCs, sport coaches, and sports medicine professionals should require ongoing continuing education covering these major health concerns.

The National Athletic Trainers' Association position statement[9] on preventing sudden death in sport is the recommended resource for recognition and treatment of these conditions. The likelihood of preventing problems is enhanced when S&CCs, sport coaches, and the medical staff are aware of the athlete's medical history, supplement use, medications, conditioning status, and acute illnesses, as well as other predisposing factors.

Key Points:

A) Exertional sickling and SCT related issues:

- Athletes should know their SCT status. Athletes who do not should be encouraged to undergo testing and not invoke the waiver.
- The S&CCs, sport coaches, and medical staff should be aware of the sickle cell trait status of each athlete as they would any other medical condition that may predispose an athlete to exertional death.
- The supervising staff should know the common prevention, recognition, and treatment strategies for exertional collapse in those athletes with SCT.[9]
- Athletes tested for SCT should be made aware of the health implications of both positive and negative tests for SCT, be provided genetic counseling and education regarding prevention and recognition of exertional sickling.

B) Exertional heat stroke:

- An exertional heat-stroke cooling plan (using a cooling modality with proven efficacy) should be developed for each venue.
- A heat acclimatization plan should be in place for when transitional period practice sessions occur in warm or hot environments.
- Appropriate work-to-rest ratios based on intensity of activity, environmental conditions, and individual factors should be implemented. These breaks allow the body to cool and provide ample time for rehydration.
- The S&CCs, sport coaches, and medical staff should be well versed in common prevention, recognition, and treatment strategies.[9]

C) Cardiac:

- Sudden cardiac arrest should be suspected in any collapsed and unresponsive athlete, traumatic or atraumatic, and an AED applied as soon as possible.
- A collapsed athlete experiencing seizures should be treated as sudden cardiac arrest until proven otherwise.[10]

- Initial management of sudden cardiac arrest includes early activation of the emergency medical services (EMS) system, early CPR, and early defibrillation.
- Prompt resuscitation of young athletes with sudden cardiac arrest generates a high survival rate.[10]

Exertional rhabdomyolysis should also be comprehensively addressed. This condition can have serious health ramifications and is easily prevented when basic precautions are followed.

8) Administering Strength and Conditioning Programs
It is recommended that a sports medicine staff member (athletic trainer and/or team physician) be part of the institution's athletics administration to encourage institutional ownership of the sports medicine program and effectively manage health and safety related issues for the student-athlete.

Ideally, a sport coach should not serve as the primary supervisor for an athletic health care provider or for an S&CC, nor should he or she have sole hiring or firing authority over those positions. The S&CC should work closely and cooperatively with the sports medicine staff. It is critical that S&CCs and ATCs have a harmonious, synergistic working relationship with open lines of communication.

A meeting should be held between the strength and conditioning staff and the sports medicine staff at the beginning of each semester and/or training season to discuss the health and safety concerns of at-risk-athletes, including the sharing of pertinent medical information/conditions (i.e. sickle cell trait status, asthma, diabetes, history exertional heat illness, etc.), as well as applicable educational materials and any updated information regarding these sudden death topics. The emergency action plan should be reviewed and rehearsed to assure proper assignment and execution of duties and responsibilities in the event the plan is implemented.

9) Partnership of Recognized Professional Organizations
The key organizations responsible for athletes' safety during strength and conditioning sessions should formalize a partnership to periodically review these best practices. This includes relevant athletic, coaching, sports medicine and strength and conditioning organizations.

10) Adequate Continuing Education for the Entire Coaching Staff and Medical Team
The task force strongly recommends key professions – S&CCs , sport coaches, athletic trainers, and team physicians – adopt requirements for education and training, and require individuals to demonstrate proficiency in the area of preventing sudden death in sport. Each reporting cycle should require a continuing education component on managing emergencies and preventing sudden death in athletes.

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Best practices developed to address heat illness in fall sports

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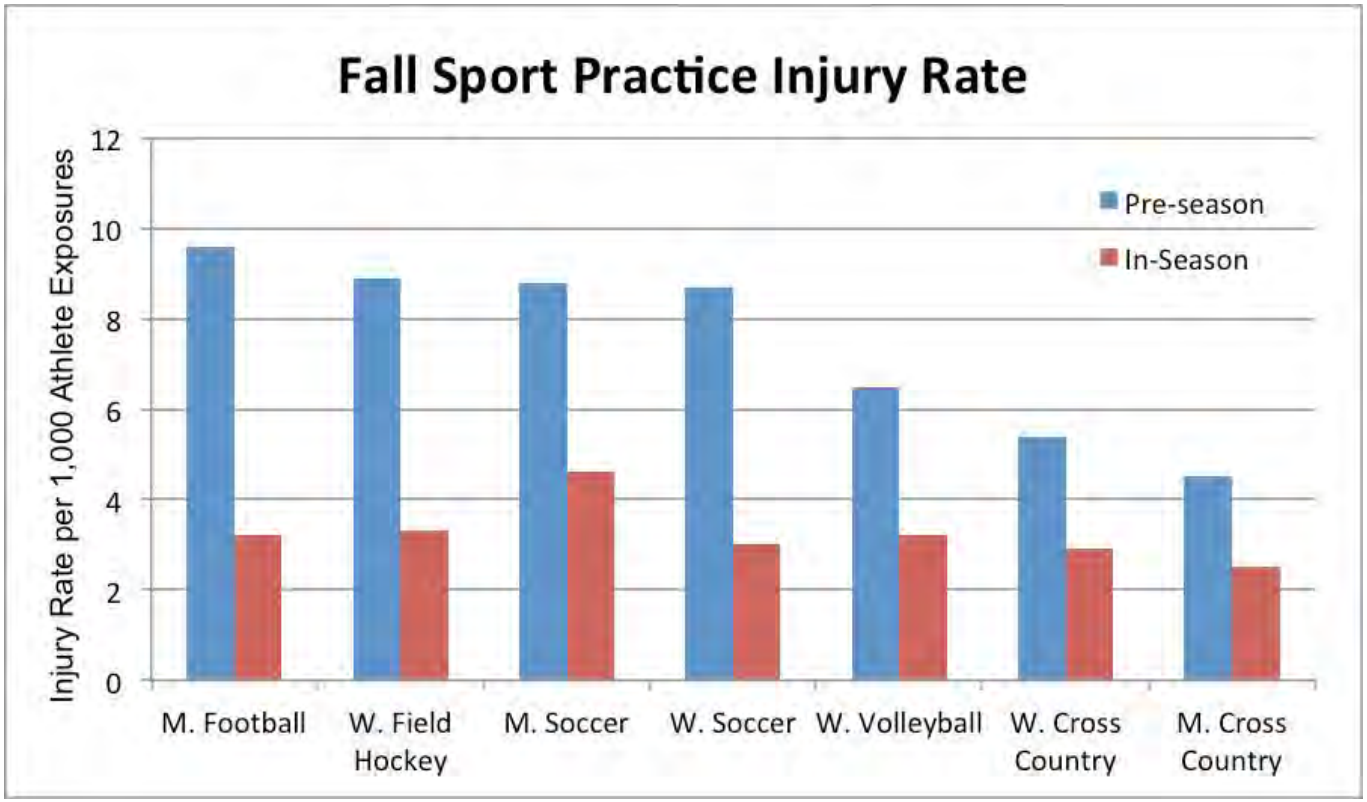
By Brian Hendrickson
NCAA.org



As the NCAA's football programs prepare for the fall season, all will follow a strict road map to guide them past the potential hazards that can accompany premature exposures to intense exercise under the hot summer sun.

NCAA policies lay down specific regulations for acclimatizing football players to the rigors of preseason conditioning. This includes regulating the number of on-field practices each day, the length of those sessions, at which points of the preseason certain pieces of equipment can be worn, and even the frequency of multiple-practice days. The policies are designed to protect the student-athlete's

health and avoid exposure to a rapid acceleration of activity before their body's physical conditioning is ready to handle the load. The risk of sudden death is highest when athletes begin any new practice period, especially within the first few days. And sport injuries can be three times higher during the preseason than in the regular-season practice periods. Conditioning sessions are also a higher risk activity than the normal sport drills and skills sessions.

Other fall sports face similar challenges when starting up following the summer period, though they do not have specific practice policies to guide them. They must design appropriate practice plans that address heat, hydration, conditioning and rest periods that balance the needs for protecting the health of their student-athletes while at the same time preparing them for competition.

As institutions search for the best approaches, the NCAA Committee on Competitive Safeguards and Medical Aspects of Sports developed these practices that should be followed to develop an effective preseason conditioning strategy:

- **Develop a preseason practice plan:** Athletes cannot be conditioned into shape over a two-week period. It takes much more time to reach competitive-level condition. Coaches and student-athletes should anticipate a period of six to eight weeks for preseason preparation, during which the type, frequency and intensity of exercises is progressively increased and ample recovery time is provided. As part of this preparation, student-athletes should be encouraged to prepare for preseason activities independently with a progressive strength and conditioning plan at least four weeks before the start of the preseason period. This will prepare their bodies for the increased activity in practices, which coaches often conduct at contest-level intensity.
- **Start slow:** During the first three days of preseason workouts, practices should be limited to one session per day that lasts no more than two hours. This will give student-athletes enough time to steadily build up their endurance without the risks of overtraining. Freshmen, transfers and walk-on student-athletes may need additional time to acclimate to the rigors of collegiate sports, so extending this period to six or seven days would be appropriate to consider. This can also apply to any athlete returning from an injury or illness and those beginning after a school break period.
- **Limit daily practice sessions:** After the initial acclimatization period, limit practice sessions to no more than two per day and avoid scheduling multiple practice sessions on consecutive days. Instead, schedule multiple practice sessions every other day, with a single workout in between, until the academic year begins. Each practice should last no more than two hours.
- **Plan for a heat acclimatization period:** Student-athletes should gradually increase exposure to hot, humid summer weather for a minimum of 10 to 14 days at the beginning of preseason activities. Similar to the preseason conditioning plan, activity during each exposure to heat should gradually increase in intensity and duration until the exercise is comparable to the exposure likely to be experienced in competition. During periods of intense heat, practice times should be reduced and scheduled during cooler times of the day.
- **Phase in exertional activity.** Coaches should consider allowing student-athletes to become accustomed to heat and exercise intensity before conducting highly exertional activities such as timed runs, max lifting sessions, repeat sprints or competitive running. An example of a progressive training plan is to start at 50 percent of total workload and intensity, and then move up incrementally over a set number of days. Workout sessions should not be used for punishment or disciplining behavior,

academic or athletic performance.

- **Take time off between practices**: On days when multiple practice sessions are held, provide at least three hours of continuous recovery time between the end of the first practice and the start of the next. During that time, student-athletes should not attend meetings or engage in physical activities related to their sport. However, time spent receiving medical treatment or eating meals can be included as part of the recovery period.
- **Walk-through after a break**: Walk-through sessions can be used as a teaching opportunity during the preseason to supplement the physical practice sessions, but just like multiple-practice days, student-athletes should also be given a rest period of at least three hours between practice and the walk-through. During the walk-through, student-athletes should not use equipment related to the sport or perform conditioning exercises.
- **Take a day off:** Student-athletes should not participate in practice sessions for more than six consecutive days. This includes any organized physical activity related to the sport, such as warm-ups, stretching, scrimmaging, weight lifting, fitness testing, conditioning, yoga, pilates, cool downs, non-medically related rehabilitation or captain's practices.
- **Communicate:** Encourage coaches to get involved with medical staff in the development of a conditioning plan. All personnel should be aware of the potential impact acclimatization, hydration, medications and drugs, existing medical conditions, nutritional supplements, clothing and equipment have on student-athletes' health while participating in strenuous workouts. When determining if a student-athlete is ready to return to competitive activities following an injury or illness, there should be a joint evaluation between the sports medicine staff and the strength and conditioning staff when making a judgment on the best return-to-play model for each individual.
- **Have an emergency plan on file:** On-field personnel should review, practice and follow their venue emergency plan and be trained in administering first aid, cardiopulmonary resuscitation and automated external defibrillator use.

http://www.ncaa.org/wps/myportal/ncaahome?WCM_GLOBAL_CONTEXT=/ncaa/ncaa/ncaa+news/ncaa+news+online/2012/association-wide/best+practices+developed+to+address+heat+illness+in+fall+sports

Division I

- Proposal No. 2010-19 was adopted last year with an effective date of 8/1/12. It limits FBS programs to five strength and conditioning coaches:

- 11.7.2.1.1 Weight or Strength Coach. A weight (strength and conditioning) coach may conduct flexibility, warm-up and physical conditioning activities prior to any game and prior to or during any practice or other organized activities without being included in the limitations on number of coaches. **Not more than five weight or strength coaches are permitted to work with a football program in any capacity, including all workouts (required and voluntary), practices and game-related activities.**

Strength and Conditioning Personnel

- Division III requires national certification
- Division I and II do not.

- Published Educational Column emphasizing importance of certification.
- Institutions should consider whether the certifying agency:

1. Is accredited by an oversight organization (e.g., NCCA-accredited);
2. Requires an undergraduate college degree;
3. Requires a continuing education component;
4. Requires current first aid, CPR and AED use certification.

Conditioning Activities

- Revised Staff Interpretation.
  - determined that required conditioning activities outside the playing season may not include contact-related drills or activities (e.g., wrestling, combative activities, blocking drills).
  - Further, determined that the use of training or resistance sleds in required conditioning activities is permissible, provided such equipment is not related to the sport (e.g., blocking sleds in football).

CSCCa

COLLEGIATE STRENGTH & CONDITIONING COACHES ASSOCIATION

P.O. Box 7100 University Station Provo, UT 84602

ph (801) 375-9400 • fax (801) 375-9401 • email: info@cscca.org • www.cscca.org

May 1, 2012

Dear Athletic Director:

This letter is being sent to you regarding NCAA legislation which limits to five the number of strength and conditioning coaches allowed to work with football Division IA programs, which is scheduled to go into effect August 1, 2012. While we understand the intent behind this legislation to level the playing field so that some schools do not have an unfair advantage by utilizing a significantly larger number of individuals working with football athletes under the guise of being strength and conditioning coaches, we at the Collegiate Strength & Conditioning Coaches association (CSCCa) have serious concerns regarding the impact of this legislation in several areas. I would like to share these concerns with you as an athletic director and ask that you help us stop this legislation from being implemented as it is currently written. We strongly encourage you to visit with your strength and conditioning staff members to hear their concerns and that you work with your NCAA compliance officer to voice your concerns and those of your strength and conditioning staff members to the NCAA.

The concerns of the CSCCa and a majority of its members primarily revolve around the impact this legislation could potentially have on the health and safety of the student athlete, although there are a variety of concerns regarding the increased work load on the football strength and conditioning staff that will undoubtedly be a result of this reduction in staff size. As I am sure you are well aware, there have been over 20 deaths of NCAA football athletes since 2000. Even one death is too many, and we must all do everything possible to protect these young athletes entrusted to our care and supervision. The strength and conditioning staff members have an extremely important role in preparing the athletes for competition by maximizing performance and making sure that the programs they design and implement are safe and effective. It is also extremely important that these athletes are closely monitored during training, especially incoming freshmen, walk-ons, and transfers. The strength and conditioning staff members have not had the opportunity to get to know the strengths and weaknesses of these individual athletes and do not yet fully know their responses to training. Even with returning veteran athletes, there are a large number of variables that can impact the athletes' training response at any given time. Severely reducing the number of strength and conditioning staff members available to work with the football athletes will obviously result in less individual interaction between the strength and conditioning coaching staff and the athlete, and there will be more athletes for each strength and conditioning coach to monitor and assist. With the recent injuries and deaths of NCAA football players, we should be doing everything possible to increase qualified supervision—not decrease it.

The strength and conditioning coaching staff is responsible for training the entire football team—not just a specific group, and they have interaction with the athletes throughout the entire year. It simply does not make sense to severely limit the number of strength and conditioning staff members allowed to work with the football athletes. There are 10 position coaches allowed to work with football athletes (not including graduate assistants), each responsible for a limited number of athletes, and this legislation restricts the number of strength and conditioning coaches working with the entire football team to just five. Again, in light of the tremendous responsibilities of the strength and conditioning coaches regarding the training of the athletes, it does not make sense to severely decrease the supervision of the football athletes during training.

As the Executive Director of the CSCCa, I am also extremely concerned regarding the impact this legislation will undoubtedly have on the practicum/internship program offered by this organization, in which individuals who are interested in becoming collegiate-level strength and conditioning coaches are provided the opportunity to work under the guidance and supervision of a qualified mentor. This practicum/internship experience is a vital part of the CSCCa's three-part certification process to ensure that individuals holding the CSCCa's certification (SCCC—Strength and Conditioning Coach Certified) possess the necessary knowledge, skills, techniques, and expertise to be a qualified and effective strength and conditioning coach on the collegiate level. Many individuals are able to pass a written certification test based upon information gleaned from a textbook or a classroom lecture, but they lack the practical skills and experience necessary to safely and effectively train a group of athletes and prepare them for an athletic game/competition. The experience these individuals gain in this internship is critical in preparing for a career in strength and conditioning coaching. The CSCCa's SCCC certification program is the gold standard in the industry and has helped to significantly raise the level of competency of individuals entering the profession. This NCAA legislation will severely impact the ability of the CSCCa to implement its internship program, which, in turn, will have a negative impact on the quantity and quality of individuals entering the profession.

Some might argue that the limiting to five individuals working with football does not apply to other sports at Division IA institutions and that it does not apply to football programs that are not Division IA. It is, however, the Division IA Football Programs that provide the majority of internships for individuals preparing to take the SCCC certification. Division IA programs will not be willing to utilize an intern as one of the five staff members eligible to work with football. These positions will obviously and necessarily be reserved for full-time strength and conditioning staff members. In addition, football is the sport in which we have seen the greatest number of injuries/deaths occurring during conditioning-related activities. Consequently, it is extremely important that these interns have the opportunity to work directly and intimately with football. Division IA provides a more rigorous and intense learning environment for these interns. It would be a tremendous loss for these interns to be denied the opportunity to work with Division IA Football Programs, and that is exactly what will happen if interns are included in the five individuals allowed to work with football. Please understand that this legislation will have a devastating effect on the CSCCa's internship program. The resultant decrease in the quality and quantity of qualified coaches entering the strength and conditioning coaching profession could potentially have a detrimental effect on the health and safety of the student athlete as well as the quality of collegiate strength and conditioning programs in the future.

The CSCCa is supportive of the NCAA's efforts to level the playing field and **to ensure that only qualified, certified individuals are working as full-time strength and conditioning coaches at NCAA institutions**, but this legislation is not the way to accomplish this goal. The recommendation of the Collegiate Strength & Conditioning Coaches association (CSCCa)—the nationally recognized governing body of the collegiate-level strength & conditioning profession— regarding the ratio of strength & conditioning staff to athletes is as follows:

Incoming freshman, Jr. College transfers, and walk-ons should not exceed a 1:10 ratio. These groups of athletes require significantly more individualized attention from the strength & conditioning staff as staff members teach the athletes the fundamentals of strength and conditioning and become familiar with their specific strengths and weaknesses. Veteran athletes should not exceed a 1:15 ratio. These ratios are necessary to optimize instruction and supervision and to maximize the health & safety of the athlete.

With an average of approximately 125 football players in a Division IA Football Program, it is clear that limiting the number of strength and conditioning coaches working with these athletes to five is not in line with this recommendation.

The CSCCa would support including a provision limiting the number of strength and conditioning interns being allowed to work with Division IA Football Programs and requiring that these individuals be preparing for certification with the Collegiate Strength and Conditioning Coaches association (CSCCa). This would ensure that these individuals are legitimately preparing to become full-time strength and conditioning coaches on the collegiate and/or professional level.

We ask for your help and support and that you work with the CSCCa and strength and conditioning coaches of Division IA Football Programs to develop fair and effective NCAA legislation regarding this matter. We strongly urge you to visit with your institution's strength and conditioning coaches, head football coach, and NCAA compliance officer to develop a letter to the NCAA voicing your concerns regarding this legislation and its potential impact as soon as possible.

If you have any questions or concerns regarding this matter or the CSCCa and its certification program, please contact me at the CSCCa National Office at 801-375-9400 or by email at cstiggins@cscca.org.

Sincerely,

Dr. Chuck Stiggins

Dr. Chuck Stiggins

CSCCa Executive Director

SUPPLEMENT NO. 16

MAY 07 2012

COLLEGE ATHLETIC TRAINERS' SOCIETY

EST. 1992

April 30, 2012

PLS PREPARE REPLY: MY SIG
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Mark A. Emmert PhD
President, National Collegiate Athletic Association
Office of the President
P.O. BOX 6222
Indianapolis, IN 46206

Dr Emmert:

For the college athletic trainer the preeminent priority is protecting life.

Commissioner Roger Goodell continues to impress and encourage with his sincere and public stance toward safety in sport...changing the culture. The NFL has 'Bountygate' with ***no*** directly discernible morbidity or mortality but has responded...directly and discernibly.

NCAA DI football ***has*** direct and discernible morbidity and mortality in our off-season football training. Recent deaths have occurred in baseball, basketball, and track. Absent direct and discernible action on behalf of our athletes in this regard...there are predictable recurrent outcomes.

As protecting life becomes an agenda item of action, please know the College Athletic Trainers Society and I stand ready to work with you!

With kindest personal regards,

Scott Anderson ATC
President

HENRY J. FREEDE, M.D.
CENTER FOR ATHLETIC MEDICINE
180 W. BROOKS
NORMAN, OK 73019

OKLAHOMA
DEPARTMENT OF INTERCOLLEGIATE ATHLETICS

SCOTT ANDERSON, ATC
HEAD ATHLETIC TRAINER

PHONE: 405.249.5907 • FAX: 405.325.8388
sanderson@ou.edu

SOONERSPORTS.COM

The Junction Boys Syndrome

Scott A. Anderson[1,2]

[1]University of Oklahoma; and [2]College Athletic Trainers' Society, Norman, Oklahoma

Abstract

Anderson, SA. The junction boys syndrome. *J Strength Cond Res* 26(5): 1179–1180, 2012—Strength and conditioning training for football has become year-round in virtually all levels of play, but none so much as contemporary NCAA DI football. Football players spend more off-season months and weeks in preparation for sport than is given to in-season practice, let alone play. A present peculiarity to NCAA Football Bowl Subdivision is conditioning and off-season football training is the sole setting for non-traumatic death in its participant players. Training regimens are too often built on tradition versus based on science and place players at-risk.

Key Words nontraumatic death, football, training

"The Junction Boys" are the so-named survivors of a renowned 10-day preseason football ordeal orchestrated by the legendary "Bear" Bryant. Although Coach Bryant achieved unqualified success, the Junction Boys did not. Those boys went 1–9. It was not until 2 seasons later, another "generation" in college football, that Texas A&M saw success with the core of the Junction survivors having departed the program because of graduation or exhausted eligibility. Nonetheless, Bryant's failed field experiment in football training, extreme activity in a punishing environment, remains a practiced pattern for "success." Just as it placed those players at peril in 1954, the "Junction Boys syndrome" is killing kids today.

In a recent Listserv thread themed at keeping living football players from converting to dead ones, James Moriarity, MD, past president of the American Medical Society for Sports Medicine stated, "I support the broad concept of having better fire trucks; but I would champion even more that we not have houses made of straw. And I truly believe that football conditioning, or what passes for football conditioning in this country, is a house of straw. Conditioning as practiced in many high schools and colleges, including elite college programs is antiquated, scientifically unstudied, and can be, obviously, dangerous" (8). Review of recent off-season football training practices reflects the described "danger" with allegation of workout guidelines dismissed, individual risk factors denied, relentless driving by coaches, deprivation of fluids, dependence upon junk science as justification for workout construct, and "discipline, accountability, and toughness" as the designed endgame vs. science-based strength and conditioning.

The Junction Boys syndrome's true trait is *intensity*—too much, too fast, too long, and often-but-not-always too soon in a workout regimen—and is the thread that ties all known recent nontraumatic deaths in NCAA football. "Just remember, gentlemen, the body is a wonderful machine. You will pass out before you die. If you pass out, the [athletic] trainers will take care of you" (9). Absent science, sport specificity, and safety, neither success nor failure in sport, is reflected in the drills.

The 4 leading causes of nontraumatic death in the working athlete are cardiac, exertional heat stroke (EHS), exertional sickling in the athlete with sickle cell trait (SCT), and asthma (10). These causes are reflected in nontraumatic NCAA football deaths, 2000–2011: 6 cardiac, 4 EHS, 1 asthma, and 10 exertional sickling.

Although asthma death is rare in NCAA football, the lone case study is telling, "... Wheeler and his teammates were running a series of gut-busting sprints—10 100-yard dashes in 15 seconds or less, then 15 seconds of rest; eight 80s in 13 seconds or less, then 13 seconds of rest; and so on. Wheeler had four 40s left when he collapsed, unable to find his breath, and died an hour later" (4). If the NCAA college football conditioning template reflects scripted and progressive daily or weekly or monthly progressions of volume, density, intensity, and periodization with work to rest ratios representative of sport ... wherein, then, does 2,160 yards of serial sprinting with a 1:1 work to rest ratio in approximately 12 minutes gain justification?

While in a relative and historical sense, EHS in football is epidemic; NCAA football players have suffered 4. One EHS death occurred in a summer workout and the remaining 3 in either day-1 or day-2 football practice with a tie to "conditioning." The evidence is clear that EHS is mainly a consequence of *exercise* rather than *environment*. The preseason model adopted by the NCAA in 2003 has, undoubtedly, improved safety for our athletes in reducing risk for EHS but cannot offset the too much, too fast, too long, and too soon inherent in initial practice days. EHS practice deaths occur at no other time.

Address correspondence to Scott A. Anderson, sanderson@ou.edu

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An aura of inevitability envelopes sudden cardiac death as it is deemed "unknowable" and thus "unpreventable." However, Maron, in his 1986 retrospective review of sudden death in young athletes noted, "... cardiovascular collapse was clearly associated with intense physical activity ..." (5). All NCAA football cardiac deaths since 2000 occurred either in day-1 conditioning or off-season workouts.

The number one cause of nontraumatic death in NCAA football is currently exertional sickling. There is no evidence that any player has ever died an exertional sickling death as SCT status is known, athlete and staff are educated, and precautions are heeded. The primary product of precaution is mitigated intensity.

The common thread of 'cause' becomes evident but the solution seems, so far, stymied by a 'culture' willingly excusing the inexcusable. Referencing again the lone NCAA asthma death, in what seemed to be contrived cover for the irrational intensity in off-season football performance enhancement, subsequent to his athlete's death, his coach said, "I don't think it was the workout that was extraordinarily difficult. It probably was the easiest day of summer workouts" (6). Another institution sought an independent investigation, as, within months, there were 2 collapses in their off-season football workouts with 1 dead and 1 survivor. The investigator abdicated the opportunity to elucidate and educate and instead offered the summary excuse, "The conditioning activities and requirements for football student-athletes are rigorous, but within the range normal to other Division I programs" (3). In training for sport, intensity–or "rigor"–is a double-edged sword in that it builds, but it also breaks. The construct of our conditioning is killing athletes, and the critical component is intensity.

"People just aren't aware. Coaches and ... Doctors and [athletic] trainers are a whole lot better ... [but] the way we're conducting conditioning at this time is putting some people at risk" (2). The answer is as evident as the cause, "Extreme conditioning drills and endurance activities that focus more on creating mental toughness than on developing proper ... techniques should be eliminated" (1). NCAA guidelines have addressed preseason practices and the athlete with SCT. However, the NCAA football population remains at risk from inappropriate conditioning sessions. "It makes much more sense to modify or eliminate these inappropriate and unnecessary drills for the safety of all NCAA football players, before there are more needless conditioning-related football deaths" (7).

One writer opined, "Only a fool would call it [collapse and death of working athletes] an aberration, the freakiest of flukes .... Human tragedy, one case after another, deserves a better and deeper explanation" (4). Of the 21 nontraumatic deaths in NCAA football, only 3 have occurred in practice; all 3 occurred, based on known evidence, in either day-1 or day-2 practices. All 16 NCAA Division I nontraumatic deaths have occurred in strength and conditioning activity. There have been, in NCAA Division I, no deaths in practice and no deaths in games. Since January 1, 2000, were one to start a clock at the conclusion of the Bowl Championship Series National Championship game and stop the clock approximately 100 hours into August preseason football practice, one will have accounted for the window of occurrence for virtually every nontraumatic death in NCAA football, save 2 September conditioning deaths. It is past time to stop the clock on these deaths.

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Question: Should the committee recommend again to Division I to visit coach sport safety training.

January 2006

Coaches Sport Safety Training.

Committee Action: The committee recommended the resubmission of the Coaches Sport Safety training legislative initiative with additional information.

1. Recommendation: To mandate that all coaches conducting practice, competition, skill instruction, and strength and conditioning sessions shall be certified in first aid and cardiopulmonary resuscitation and have training in emergency plan activation.

2. Rationale: The proposed legislation is intended for all coaches and the training should be consistent with certification from a nationally recognized organization in first aid, cardiopulmonary resuscitation (CPR) and automatic external defibrillation (AED). The goal of this initiative is to ensure there is always someone is certified in these skills to care for all student-athletes working out. CPR is more effective if multiple certified personnel are present. The number of individual skill sessions, workouts, and the logistics of practices the committee believes the only way to ensure that emergency care is to require all coaches to participate. The NCAA currently requires that all wrestling coaches including volunteers have first aid and CPR certification, that all strength and condition personnel have CPR certification, and that all cheerleading coaches certify they have completed a safety training program to be insured by the NCAA under the catastrophic plan. Member schools and even some conferences currently mandate similar minimal standards of care. The committee encouraged conferences to implement the process and institutions to police themselves by reporting violations to the conference office and setting their own sanctions. Furthermore, the committee suggests that institution compliance officers keep copies of emergency care plans for facilities and a list of personnel that have completed each of the requirements.

July 2006 CSMAS Meeting

1. NCAA Governance Actions and Issues. The committee reviewed relevant actions from the NCAA Executive Committee, the NCAA Division I Championships/Competition Cabinet, and the NCAA Divisions II and III Management Councils.

   - The committee reaffirmed its position that all coaches (including strength and conditioning staff) be certified in first aid and cardiopulmonary resuscitation (CPR). The committee also supported the concept of a common provision across divisions since the committee noted that there is a lack of evidence that would require variance between divisions. In an effort to assist the governance process and engage membership feedback, the committee did not elect an effective date so that it could collect further data on the current practice of the certification of coaches on member campuses. The committee conducted a survey examining emergency care policies on campuses in 1998. At that time, most sports required less than 50 percent of personnel to be CPR certified and even less required first aid certification. The committee also will examine membership support for the inclusion of first aid certification or whether CPR certification is most paramount for

rendering first response emergency care on our campuses. The committee will also address issues of liability, volunteer coaches, time frames for certification and cost. The committee noted that this training certification is intended to provide initial care until more skilled responders arrive as a supplement to an emergency situation and is not intended to replace appropriate medical care and coverage. Over 80 percent of student-athlete sudden deaths occur during practices and conditioning sessions. Sixty percent of those sudden deaths are attributed to heart conditions. Rapid response and immediate care is paramount in these situations. The requirement of an emergency action plan and mandatory cardiopulmonary resuscitation certification of coaches for every practice, conditioning session, and event is the key preliminary step toward activating medical care response for student-athletes. The committee emphasized that even with other skilled personnel available (ATC, MD), emergency situations require multiple rescuers and the CPR trained coach may be able to assist the other rescuers. The growth of practice opportunities that include year-round exposures and extensive out-of-season sessions has greatly increased the risk that coaches will be the first to respond to a collapsed student-athlete.

NCAA Legislation Involving Health and Safety Issues

Student-Athlete Welfare and Safety	Voluntary Summer Conditioning (Div. I only)	13.11.3.9 (Sports Other Than Football and Basketball)	In sports other than football and basketball, a prospective student-athlete may engage in voluntary summer workouts conducted by an institution's strength and conditioning coach with department-wide duties and may receive workout apparel (on an issuance and retrieval basis), provided he or she is enrolled in the institution's summer term prior to the student's initial full-time enrollment at the certifying institution. Such a prospective student-athlete may engage in such workouts only during the period of the institution's summer term or terms (opening day of classes through last day of final exams) in which he or she is enrolled.
	Voluntary Weight-Training or Conditioning Activities (Div. I only)	13.11.3.9.4	A strength and conditioning coach who conducts voluntary weight-training or conditioning activities is required to maintain certification in first aid and cardiopulmonary resuscitation. If a member of the institution's sports medicine staff (e.g., athletic trainer, physician) is present during voluntary conditioning activities conducted by a strength and conditioning coach, the sports medicine staff member must be empowered with the unchallengeable authority to cancel or modify the workout for health and safety reasons, as he or she deems appropriate.
	Sports-Safety Training	11.1.6 (Div. II)	Each head coach and all other coaches who are employed full time at an institution shall maintain current certification in first aid, cardiopulmonary resuscitation (CPR) and automatic external defibrillator (AED) use.
		11.1.6 (Div. III)	Each head coach shall maintain current certification in first aid, cardiopulmonary resuscitation (CPR) and automatic external defibrillator (AED) use.
	Discretionary Time (Div. I only)	17.02.14	Student-athletes may only participate in athletics activities at their initiative during discretionary time.
	Mandatory Medical Examinations	17.1.5	All student-athletes beginning their initial season of eligibility and students who are trying out for a team must undergo a medical exam before they are permitted to engage in any physical activity. The exam must take place within six months before the physical activity. Each subsequent year, an updated medical history must be administered by an institutional medical staff member.

NCAA partners with national laboratory to provide access to affordable screening for sickle cell trait as part of a student-athlete's medical examination.

Overview

In an effort to provide a more efficient and effective process for sickle cell trait (SCT) screening, the NCAA has signed an agreement with Quest Diagnostics to provide three different service models for member institutions and their student-athletes to access affordable screening exams to determine SCT status as part of the student-athlete's medical examination process. Effective October 1, 2011.

All student-athletes beginning their initial season of eligibility and students who are trying out for a team are required to undergo a medical examination before engaging in physical activity.

In NCAA Division I and Division II member institutions, this medical examination must include a sickle cell solubility test, unless documented results of a prior test are provided or the student-athlete declines the test and signs a written release. Although SCT screening is performed on all U.S. babies at birth, many student-athletes may not know whether they have the trait. SCT status can be accomplished with a simple blood test that is relatively inexpensive. Screening can be used as a gateway to targeted precautions and counseling on the implications of SCT, including health, athletics and family planning. The NCAA Division III membership continues to evaluate legislative concepts as part of the medical examination.

Institutions can select from the following three service models:

1. Institution On-Site Services Model.

2. Patient Service Center Model.

3. Student-Athlete Service Model.

Service Models for NCAA Member Institutions

1. Institution On-Site Services Model

The following service model is available to all NCAA member institutions that want to conduct onsite campus collections for their student-athletes as part of their medical examination through an agreement with Quest Diagnostics. Institutions are responsible for fees associated with this model and Quest Diagnostics will bill the institution on a monthly invoice. The average turnaround time for results mainly depends on the physician. The laboratory will turnaround the results in 24 hours to the ordering physician/institution designated physician either via fax or online.

Quest Diagnostics will provide on-site blood drawing (phlebotomy) services at an NCAA Institution with this service model. The institution will contact the Quest Diagnostics Client

Service Team to schedule a blood-drawing event at **1-866-226-8046**. Institutions should indicate they are calling to setup an "onsite blood drawing event for sickle cell trait testing". The event must be scheduled with a minimum notice of ten business days prior to the date the service is required.

All laboratory testing must be ordered by a licensed physician and Quest Diagnostics must be provided with the licensed physician information. An institution physician or campus health center physician could fill the role of ordering physician as long as the results reporting and follow-up are provided to the student-athlete.

Quest Diagnostics will provide an appropriate number of phlebotomists to accommodate the number of student-athletes that need their blood drawn. In the event that phlebotomy support is requested and confirmed as scheduled, then subsequently cancelled with less than 3 business days notice, a cancellation charge equal to two hours/phlebotomist staffed at the stated hourly rate will be assessed.

Quest Diagnostics will provide, as part of the set established fee, all supplies necessary for the proper collection and transport of specimens to Quest Diagnostics. This includes requisition forms, specimen transport containers, specimen bags and labels, packaging materials, courier pick-up and transportation back to a Quest Diagnostics facility. The Quest Diagnostics phlebotomists will make all of the arrangements to have the blood specimens picked up by a Quest Diagnostics courier and transported to the laboratory for testing.

A positive screening test will result in an automatic reflexive test for confirmation. All laboratory testing results will be reported back to the ordering physician as designated by the institution per state and federal regulations for sharing and transmitting medical information. If a test is positive, the student-athlete should be offered counseling on the implications of SCT, including health, athletics and family planning.

Fees
On-Site Blood-Draw at Institution Location - **$65.00** per phlebotomist, per hour
Sickle Cell Screen test code 825- **$8.50**
Confirmatory Reflex Testing test code 35489/37679 **- $30.00**

2. Patient Service Center Model

The following service model is available to all NCAA member institutions that want to pay for their student-athletes SCT screening test as part of their medical examination but do not want to collect the samples onsite. Institutions are responsible for fees associated with this model and Quest Diagnostics will bill the institution on a monthly invoice. The average turnaround time for results mainly depends on the physician. The laboratory will turnaround the results in 24 hours to the ordering physician/institution designated physician either via fax or online.

Quest Diagnostics operates a network of over 2,000 company owned and managed Patient Service Centers (PSCs). These PSCs provide member institutions' student-athletes with walk-in

phlebotomy blood-drawing services. Student-athletes of the NCAA member institutions can use this national network of PSCs for their blood draw where the SCT Screening test will be analyzed.

This process begins with the institution contacting the Quest Diagnostics Client Service Team at **1-866-226-8046** to setup an account for the forms. The institutions *name* and *contact information* will be listed on each form provided to the student-athlete. The participating NCAA institution provides the student-athlete with a Quest Diagnostics Test Requisition Form and an institution physician, campus health center physician, or family physician fills the role of ordering physician. The student-athlete will look up a Quest Diagnostics PSC location nearest their location by using a zip code (school address or home address, whichever is more convenient) at :

https://secure.questdiagnostics.com/hcp/psc/jsp/SearchLocation.do?newSearch=FindLocation.

Upon presenting the form at the PSC, the blood will be drawn and sent to a Quest Diagnostics laboratory for testing. The testing will be performed and results will be reported back to the Physician with Prescriptive Authority for ordering the testing per state and federal regulations for sharing and transmitting medical information.

A positive screen results in an automatic reflexive test for confirmation. The student-athlete or physician will then report the testing results to the institution's designee per state and federal regulations for sharing and transmitting medical information. If a test is positive, the student-athlete should be offered counseling on the implications of sickle cell trait, including health, athletics and family planning.

Fees
Blood-Draw at Quest Diagnostics PSC test code 3259 **- $9.00**
Sickle Cell Screen test code 825- **$8.50**
Confirmatory Reflex Testing test code 35489/37679 **- $30.00**

3. Student-Athlete Service Model

The following service model is available to all NCAA member institutions that want their student-athletes to pay for the SCT screening test as part of their medical examination. This is a patient-centered laboratory test ordering model and the student-athlete will pay for the fees associated with this model through an easy to use, and secure online system run by Quest Diagnostics and Medivo. The student-athlete must be 18 years of age or older to use the online service. Student-athletes can expect that their results will be ready between 24-48 hours, on average.

As instructed by their institution, the student-athlete will go directly to the Medivo website to generate the test order, pay for the SCT screening test and physician service with a single $32.50 fee and print a Quest Diagnostics test requisition form.

Web site: https://sicklecelltesting.medivo.com/order/am

The student-athlete will be able to determine the most convenient Quest PSC for their blood draw by performing a search on the Medivo website as they are generating the test order.

Medivo will provide the prescriptive authority for the SCT screening test order and confirmatory test (if necessary) as well as the payment platform for the student-athlete. The student-athlete *must* take the Quest Diagnostics test requisition form (generated after they make payment on the Medivo website) to a Quest Diagnostics PSC to have their blood drawn. There are no exceptions.

Test results will go back to Medivo for review. Medivo will post the results to their website for the student-athlete to view and print. If a test result is *positive*, the student-athlete will receive a phone call directly from a physician at Medivo as well as an email with a link to go back online to request and pay for the confirmation reflex testing ($30.00) through the Medivo online system. The student-athlete will be directed to a new web site to order the confirmation test:

https://confirmationtest.medivo.com/order/am

The student-athlete may need to have an additional blood draw for the confirmation testing. The student-athlete will then provide the results to their primary care physician or the institution's designee per state and federal regulations for sharing and transmitting medical information. If a test is positive, the student-athlete should be offered counseling on the implications of sickle cell trait, including health, athletics and family planning.

Fees
SCT Screening Test - **$32.50**
Confirmation Reflex Testing - **$30.00**

AMERICAN SOCIETY OF HEMATOLOGY
2021 L Street, NW, Suite 900, Washington, DC 20036 ph 202.776.0544 fax 202.776.0545 e-mail ASH@hematology.org

Statement on Screening for Sickle Cell Trait and Athletic Participation

Background

The American Society of Hematology (ASH) represents approximately 16,000 physicians, scientists, and medical trainees committed to the study and treatment of blood and blood-related diseases. ASH members include clinicians who specialize in treating children and adults with sickle cell disease (SCD) and researchers who investigate the causes and potential treatments of SCD manifestations.

Sickle cell disease is an inherited blood disorder that affects 80-100,000 Americans, mostly but not exclusively of African ancestry. SCD causes production of abnormal hemoglobin, resulting in severe anemia, pain, other devastating disabilities, and, in some cases, premature death. SCD requires the inheritance of two defective globin genes. Eight to 10 percent of African Americans have sickle cell trait. Individuals with sickle cell trait do not have SCD, but are carriers of one defective gene associated with SCD. The majority of their hemoglobin is normal. Millions of Americans and hundreds of millions of individuals worldwide with the sickle cell trait enjoy normal life spans without serious health consequences. Rarely, clinical issues such as exertional rhabdomyolysis (the rapid breakdown of skeletal muscle due to injury to muscle tissue) have been reported in individuals with sickle cell trait. These incidents have generally occurred under extreme conditions such as severe dehydration and high intensity physical activity.

In April 2010, the National Collegiate Athletic Association (NCAA) adopted a policy requiring Division I institutions to perform sickle cell trait testing for all incoming student athletes. This policy arose from the settlement of a lawsuit against the NCAA following the death during preseason training of a college football player, who was subsequently found to have sickle cell trait. The policy includes an opt-out provision for students who can provide results from a prior test and for those who are willing to sign a waiver of liability against the university and the NCAA. There have been no well-controlled, hypothesis-driven prospective studies on sickle cell trait and exertional collapse. Most cases reported to date are autopsy descriptions or case reports in which other potential contributors are not fully investigated. The extremely small number of deaths in a highly prevalent carrier state implies that other genetic or environmental factors play a role. Rhabdomyolysis and exertional collapse also occur in individuals without sickle cell trait.

Universal preventive measures implemented by the U.S. Army to mitigate risk of exertion-related heat illness for all soldiers include heat acclimatization, monitoring work-rest cycles, guidelines for nutrition and hydration, and maintaining staff preparedness for early and rapid detection and treatment of heat illnesses. Using these universal guidelines, the death rate has been significantly lowered for all individuals undergoing training, including those with sickle cell trait. Similarly, governmental authorities in Brazil (which has similar sickle cell carrier rates as the U.S.) have promulgated guidelines to avoid heat-related injuries and see no need for athletes or military personnel to be screened for sickle cell trait.

ASH Policy

ASH does not support testing or disclosure of sickle cell trait status as a prerequisite for participation in athletic activities. ASH believes that current scientific evidence does not justify this requirement. It is also not consistent with good medical practice or established principles of public health ethics. Screening for sickle cell trait should be voluntary and should take place in a setting that ensures privacy and is performed by a knowledgeable provider who is able to offer comprehensive counseling.

ASH recommends the implementation of universal interventions to reduce exertion-related injuries and deaths, since this approach can be effective for all athletes irrespective of their sickle cell status. Universal interventions are used successfully by organizations like the U.S. Army and the Brazilian military. Until the NCAA requires universal interventions, student athletes remain at risk for suffering from significant heat stress/exertion-related injury or death. Participation in athletics can be made safer with these measures, thereby rendering screening for and disclosure of sickle cell trait status unnecessary.

ASH believes that the NCAA Division I policy, as currently written and implemented, has the potential to harm the student athlete and the larger community of individuals with sickle cell trait. The NCAA policy attributes risk imprecisely, obscures consideration of other relevant risk factors, fails to incorporate appropriate counseling, and could lead to stigmatization and racial discrimination.

ASH strongly supports increased biomedical and population-based research on sickle cell trait as it relates to exertion-related illness, as well as other clinical conditions. New knowledge derived from research should benefit individuals with sickle cell trait and help inform public policy.

The American Society of Hematology (*www.hematology.org*) is the world's largest professional society concerned with the causes and treatment of blood disorders. Its mission is to further the understanding, diagnosis, treatment, and prevention of disorders affecting blood, bone marrow, and the immunologic, hemostatic, and vascular systems by promoting research, clinical care, education, training, and advocacy in hematology. The official journal of ASH is *Blood* (*www.bloodjournal.org*), the most cited peer-reviewed publication in the field, which is available weekly in print and online.

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Contact:
Andrea Slesinski, American Society of Hematology
aslesinski@hematology.org; 202-552-4927

ASH Policy Opposes Mandatory Sickle Cell Trait Screening for Athletic Participation, Recommends Universal Training Interventions & Additional Research

(WASHINGTON, January 26, 2012) – The American Society of Hematology (ASH), the world's largest professional society concerned with the causes and treatment of blood disorders, today issued a policy statement opposing mandatory screening of athletes for sickle cell trait as a prerequisite to athletic participation and urging athletics programs to adopt universal preventive interventions in their training programs to protect athletes from exertion-related illness and death. ASH's position is in direct contrast to a policy enacted in 2010 by the National Collegiate Athletic Association (NCAA) requiring Division I institutions to perform sickle cell testing for all incoming student athletes.

"Screening alone is an extremely limited approach intended to protect the liability of the NCAA and the athlete's university, not the student athlete," said ASH President-Elect Janis Abkowitz, MD, of the University of Washington School of Medicine in Seattle. "Furthermore, sickle cell trait is not the only condition that can lead to death from athletic over-exertion, underscoring the need for NCAA to require universal preventive interventions in its training programs that will better protect everyone."

Sickle cell trait is an inherited blood disorder that affects approximately 8 percent of African-Americans. Unlike sickle cell disease, a serious illness in which patients have two genes that cause the production of abnormal hemoglobin, individuals with sickle cell trait carry only one defective gene and typically live normal lives. Rarely, extreme conditions such as severe dehydration and high-intensity physical activity can lead to serious health issues, including sudden death, in these individuals.

The ASH policy statement on screening for sickle cell trait and athletic participation, derived from the recommendations of experts in sickle cell biology and care, concludes that current scientific evidence does not justify the screening of all athletes for sickle cell trait as a prerequisite to participation. The ASH policy calls for the implementation of universal preventive interventions that will render screening unnecessary and better protect all athletes from exertion-related illness and death.

The U.S. Army currently utilizes universal preventive interventions for all recruits undergoing training, including monitoring heat acclimatization and work-rest cycles adjusted for the environment, implementing guidelines for hydration, and maintaining staff preparedness for early and rapid detection and treatment of heat illness. The Army began implementing these interventions in 1996 after a military-sanctioned study concluded that such measures successfully prevented subsequent sudden death for all recruits, including those with sickle cell trait. Following the study, the military discontinued mandatory screening of all incoming recruits for sickle cell trait.

"The U.S. Army protects recruits from exertion-related injury and death, and the NCAA should follow their lead to protect its athletes," said Dr. Abkowitz. "Until the NCAA moves to universal preventive interventions in their conditioning programs, all student athletes remain at risk for suffering significant exertion-related injury or death."

The Society also strongly supports increased biomedical and population-based research examining how sickle cell trait, as well as other clinical conditions, relate to exertion-related illness.

"Our policy serves as a call for additional research in this area that will benefit those with sickle cell trait, help inform public policy, and save lives," said Dr. Abkowitz.

Reporters who wish to arrange an interview with Dr. Abkowitz may contact Andrea Slesinski at 202-552-4927 or aslesinski@hematology.org.

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The American Society of Hematology (*www.hematology.org*) is the world's largest professional society concerned with the causes and treatment of blood disorders. Its mission is to further the understanding, diagnosis, treatment, and prevention of disorders affecting blood, bone marrow, and the immunologic, hemostatic, and vascular systems by promoting research, clinical care, education, training, and advocacy in hematology. The official journal of ASH is *Blood* (*www.bloodjournal.org*), the most cited peer-reviewed publication in the field, which is available weekly in print and online.

National Athletic Trainers' Association

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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Task Force Participants

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.

Co-Chairs
Scott Anderson, ATC
E. Randy Eichner, MD

At-Large Members:
Mary L. Anzalone, MD
James C. Puffer MD
Brock Schnebel, MD

American Academy of Pediatrics
Jorge Gomez, MD

American College of Sports Medicine
Michael F. Bergeron, PhD, FACSM
Don Porter, MD

American Medical Society for Sports Medicine
James Moriarity, MD

American Orthopaedic Society for Sports Medicine
James C. Walter, II, MD

American Osteopathic Academy of Sports Medicine
Jeffrey Bytomski, DO
Angela Cavanna, DO, FAOASM

Association of Black Cardiologists
B. Waine Kong, PhD, JD

College of American Pathologists
Michael J. Dobersen, MD, PhD

Gatorade Sports Science Institute
Jeff Kearney
Craig Horswill, PhD
Magie Lacambra, MEd, ATC

Military Medicine
Fred Brennan, Jr., DO

National Association of Basketball Coaches
Reggie Minton

National Association of EMTs
Connie Meyer, MICT

National Association of Medical Examiners
Jeffery Barnard, MD

National Athletic Trainers' Association
Veronica Ampey, MS, ATC
Douglas Casa, PhD, ATC, FACSM
Terry Dewitt, PhD, ATC
Scott Galloway, ATC, LAT
Chris A. Gillespie, MEd, ATC, LAT
Eric Howard, EdD, MS, ATC
Bob Toth, MS, ATC
Torrance Williams, ATC, LAT

National Basketball Athletic Trainers' Association
Dionne Calhoun, ATC

National Collegiate Athletics Association
David Klossner, PhD, ATC
John W. Scott, PhD, MD
Tracy Ray, MD

National Federation of State High School Associations
Bob Colgate

National Football League
Gary W. Dorshimer, MD, FACP

National Strength and Conditioning Association
Avery Faigenbaum, EdD, CSCS

Professional Football Athletic Trainers' Society
Corey Oshikoya, ATC

The Sickle Cell Disease Association of America, Inc.
National Medical Association
Betty S. Pace, MD

The Sickle Cell Foundation of Georgia, Inc.
Rudolph Jackson, MD

Women's Basketball Coaches Association
Marsha Sharp

Sickle Cell Trait

Sickle cell trait is not a disease. It is a descriptive term for a hereditary condition in which an individual has one normal gene for hemoglobin (A) and one abnormal gene for hemoglobin (S), giving the genetic hemoglobin type (AS). Sickle cell trait is common in areas where malaria is endemic and provides a survival advantage against that disease. Having sickle cell trait (AS) is different from sickle cell disease (SS), in which two abnormal genes are present. Individuals with sickle cell trait commonly have ancestors from Africa, South or Central America, the Caribbean, Mediterranean countries, India, or Saudi Arabia. Even though sickle cell trait occurs in eight to ten percent of U.S. African-American population, it is present in athletes at all levels of competition and ethnicities, including professional and Olympic.

Sickle Cell Trait is not a barrier to exercise or participation in sport. Persons with sickle cell trait alone do not have anemia and sickle cell ***trait*** cannot turn into sickle cell ***disease***. There are more than four million people in the United States with sickle cell trait and very few experience any related complications. In general, sickle cell trait is a benign condition. No sports medicine body currently suggests exclusion from sport or exercise for the athlete with sickle cell trait.

Exercise and sport are safe and, in fact, may be somewhat protective in individuals with sickle cell trait. Regular exercise can lower blood viscosity, inflammation and oxidative stress in those with sickle cell trait and provides many positive health benefits in individuals both with and without sickle cell trait. It appears that problems can occur primarily with intense physical exertion when a coach or other individual is trying to push athletes to extreme physiologic limits or when an individual exercises intensely in the setting of illness (even relatively mild illness) or perhaps stimulant use. There is evidence in the laboratory and in the field that hydration can counteract or improve hematologic parameters in exercising individuals with sickle cell trait; therefore, paying careful attention to and ensuring adequate hydration may reduce clinical risk.

Sickle cell trait has been linked to an increased risk of exercise-associated sudden death in individuals undergoing intense physical exertion, and possibly rhabdomyolysis. Exercise-associated sudden death in individuals who have sickle cell trait most commonly occurs in those undergoing intense physical exertion such as military recruits in basic training and football athletes during conditioning workouts; although, it can occur in other sports and activities as well. The definitive contributing mechanisms for the increased risk of death are unknown; however, it is reported that at baseline individuals with sickle cell trait have increased blood viscosity, slightly reduced red blood cell deformability, increased red blood cell fragility and increased levels of inflammatory markers. The clinical implications of these distinguishing features are unknown.

Exertional rhabdomyolysis and exercise-associated death in those with sickle cell trait are hypothesized to occur because of micro-vascular occlusion in the capillary beds of the muscles leading to muscle cell breakdown and consequent release of intracellular contents potentially causing renal failure and/or cardiac arrhythmia. Clinically, when rhabdomyolysis occurs, it seems to present more quickly in athletes with sickle cell trait than those without. While the proposed underlying mechanisms seem to fit clinical observations, much more research is needed to determine the definitive cause of pathology and how or if sickle cell trait contributes. Although not well-documented in population-based studies, case reports provide evidence that individuals with sickle cell trait may also be at greater risk for non-fatal collapse.

Sickle cell trait has also been linked to an increased risk of splenic infarction, as well as an inability to concentrate urine. Splenic infarction typically occurs at high altitudes (usually greater than 5,000 feet), and symptoms may include sudden acute pain in the lower ribs, weakness and nausea. Strenuous physical exertion after a recent arrival at altitude may contribute to its development. Hyposthenuria (the inability to concentrate urine) occurs in most individuals with sickle cell trait and may contribute to dehydration.

Knowledge of one's sickle cell trait status is an important piece of personal health information with possible health and genetic implications. All infants born in the U.S. and some other countries are tested at birth; but this information is not always communicated to the parents or, later in life, to the individual. Accordingly, the following points should be considered by healthcare providers for all athletes:

- Serious medical problems associated with sickle cell trait are rare, even during intense athletic training, practice and competition. Unwarranted restrictions or limitations on physical activity and sports participation should not be placed on individuals with sickle cell trait; however, during intense exercise, athletes with sickle cell trait have experienced significant physical distress, collapsed and even died.

- Prompt access to medical care and planned emergency response are critical components to ensure adequate response to an athlete who collapses or is in distress.

- Everyone should know their sickle cell trait status. If unknown, individualized voluntary testing for sickle cell trait should be considered. If testing is performed, it must always be accompanied by education and counseling on the health implications of a positive or negative test, as well as genetic counseling. The athlete should be encouraged to share this information with medical professionals and coaching staff, as they would any other piece of medical information.

- All exercising individuals, including those with known sickle cell trait, should be counseled to:
  - Hydrate sufficiently (link to ACSM hydration policy);
  - Acclimatize gradually to heat, humidity, and altitude (link to any relevant policies);
  - Condition carefully and gradually for up to several weeks, before engaging in exhaustive exercise regimens;
  - Refrain from extreme exercise during or while recovering from an illness, especially one involving fever;
  - Immediately stop exercise and seek prompt medical care when experiencing unusual physical distress;
  - Recognize variable signs and symptoms of physical distress. These may include: muscle "cramping" pain with weakness, while notably the muscles are usually not tense or hard, especially in the back or lower extremity; hyperventilation or difficulty to catch one's breath; other muscle pain; abnormal general weakness; or undue fatigue.

- Coaches should conduct appropriate sport-specific conditioning based on sound scientific principles and be ready to promptly intervene when athletes show signs of distress. Individuals should be allowed to self-limit their intensity and discontinue exercise when experiencing unusual fatigue and/or physical distress.

- Ongoing, collaborative research in the area of exercise and sickle cell trait should be vigorously pursued with sport and sports medicine governing bodies and other appropriate medical associations and experts.

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4000 W. 114th Street, Suite 100
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FOR IMMEDIATE RELEASE (Jan. 26, 2012)

AMSSM Statement on Sickle Cell Trait

LEAWOOD, Kan. - On January 26, 2012, the American Society of Hematology (ASH) released a position statement calling for the reversal of the 2010 NCAA policy for mandatory screening of all Division I athletes for sickle cell trait (SCT).

The American Medical Society for Sports Medicine (AMSSM) is a multi-disciplinary organization of sports medicine physicians whose members are dedicated to education, research, advocacy, and the care of athletes of all ages. AMSSM members are closely involved in the medical care of collegiate athletes and many carry out the responsibilities of a team physician. AMSSM acknowledges the issues raised in the ASH statement, as well as the safety concerns that prompted the NCAA to move forward with its policy recommendation.

SCT has been associated with cases of exertional sudden death in NCAA athletes, in particular in African American football players. While rare, these tragic deaths appear to be largely preventable through education, implementation of safety guidance on heat acclimatization and hydration, adherence to appropriate strength and conditioning programs, and effective emergency action planning. Team physicians play a key role in educating athletes, coaches, and college medical staff regarding safety precautions in athletes with SCT, as well as other conditions that may affect safe participation.

The AMSSM notes that the NCAA policy provides all athletes with education on SCT as well as the option to opt out of the process of screening, without any penalty to participation. The AMSSM also agrees with ASH in supporting universal preventive measures to make participation in sports safer for all athletes. We strongly support increased laboratory and clinical research to promote evidence-based practice in this complex and controversial area.

The AMSSM looks forward to working with both the NCAA and ASH on this important issue. As a leading sports medicine organization, AMSSM is fully committed to protecting the health and safety of college athletes.

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For more information, contact:
Jim Griffith, MBA, CAE
Executive Director
American Medical Society for Sports Medicine
(913) 327-1415
jgriffith@amssm.org
www.amssm.org

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EDITOR'S CHOICE

Sickle cell trait associated with a RR of death of 37 times in national collegiate athletic association football athletes: a database with 2 million athlete-years as the denominator

Kimberly G Harmon,[1] Jonathan A Drezner,[1] David Klossner,[2] Irfan M Asif[3]

[1]Department of Family Medicine, University of Washington, Seattle, Washington, USA
[2]National Collegiate Athletic Association, Indianapolis, Indiana, USA
[3]Department of Family Medicine, University of Tennessee, Knoxville TN, USA

Correspondence to
Kimberly G Harmon, Departments of Family Medicine and Sports Medicine and Orthopaedics, 4060 East Stevens Circle, Hall Health Sports Medicine Clinic, University of Washington, Seattle, WA 98195, USA; kharmon@u.washington.edu

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ABSTRACT

Background This study examines sickle cell trait (SCT) as a cause of sudden death in National Collegiate Athletic Association (NCAA) athletes and explores the cost-effectiveness of different screening models.

Methods The authors reviewed the cause of all cases of sudden death in NCAA student-athletes from January 2004 through December 2008. The authors also explored the cost-effectiveness of screening for this condition in selected populations assuming that identifying athletes with SCT would prevent death.

Results There were 273 deaths and a total of 1 969 663 athlete-participant-years. Five (2%) deaths were associated with SCT. In football athletes, there were 72 (26%) deaths. Of these, 52 (72%) were due to trauma unrelated to sports activity and 20 (28%) were due to medical causes; nine deaths were cardiac (45%), five were associated with SCT (25%). Thirteen of the 20 deaths due to medical causes occurred during exertion; cardiac (6, 46%) SCT associated (5, 39%), and heat stroke unrelated to SCT (2, 15%). All deaths associated with SCT occurred in black Division I football athletes. The risk of exertional death in Division I football players with SCT was 1:827 which was 37 times higher than in athletes without SCT. The cost per case identified varied widely depending on the population screened and the price of the screening test.

Conclusions Exertional death in athletes with SCT occurs at a higher rate than previously appreciated. More research is needed to (1) understand the pathophysiology of death in SCT-positive athletes and (2) determine whether screening high-risk populations reduces mortality.

INTRODUCTION

Sickle cell trait (SCT) has received significant attention as a cause of death in American football athletes. It has been cited in the medical literature as a leading cause of death in military basic training and civilian organised sport,[1–4] and the lay press has characterised death in football athletes with SCT as nearly epidemic. SCT is common, with 1 in 14 (7%) of blacks having SCT and up to 1 in 625 Caucasians carrying the gene.[5] However, while SCT is common in the general population, the RR of exertional death related to SCT is unknown.

Normal haemoglobin (Hgb) is composed of two α and two β chains. Hgb S results from the substitution of a valine for glutamic acid at the sixth amino acid of the β globin chain. Hgb S produces a haemoglobin tetramer that is poorly soluble when deoxygenated which can cause the red blood cell to form a sickle shape. Sickle cell disease occurs when an individual is homozygous for Hgb S. Vaso-occlusion and haemolysis leading to sickle cell crisis are the hallmark of sickle cell disease which is generally incompatible with strenuous exercise. SCT occurs when an individual has one normal Hgb gene and one for Hgb S and is usually considered benign. Although there is an increased incidence of exertional death in those with SCT, the underlying pathophysiological mechanism is unclear.

An association of SCT with sudden death was first reported in 1970 when it was noted that a disproportionate number of deaths in military recruits training at moderate altitude were positive for SCT.[6] Kark expanded on these findings reporting that the risk of sudden death in black recruits with SCT was 28 times those without SCT in a review of medical records and autopsy data.[7] Other studies in military basic training have confirmed an association of sudden death with SCT.[8 9] Concern of a similar association in athletes developed after the 1973 death of a college football player and was first addressed by an National Collegiate Athletic Association (NCAA) guideline in 1975. Despite NCAA guidelines, there have continued to be intermittent deaths related to SCT in the NCAA. There is no published literature quantifying the risk of death in NCAA athletes with SCT; however, in 2010 legislation was enacted requiring all Division I schools to screen athletes or have them sign a waiver declining the screen, if the athletes' SCT status was not already known from neonatal birth screening. This study examines SCT associated sudden death in NCAA athletes and explores the cost-effectiveness of different screening models.

METHODS

All cases of sudden death in NCAA student-athletes from January 2004 through December 2008 were examined for cause of death using a previously defined database of death in athletes.[10] The database was developed using two different data sources:[1] the NCAA Resolutions list and[2] from the Parent Heart Watch (PHW) database. The NCAA Resolutions list is compiled annually in honorarium of NCAA student-athletes who have died of any cause and is voluntary. PHW is a not-for-profit group dedicated to the prevention and awareness of sudden cardiac arrest (SCA) in young people. They keep an ongoing database of sudden

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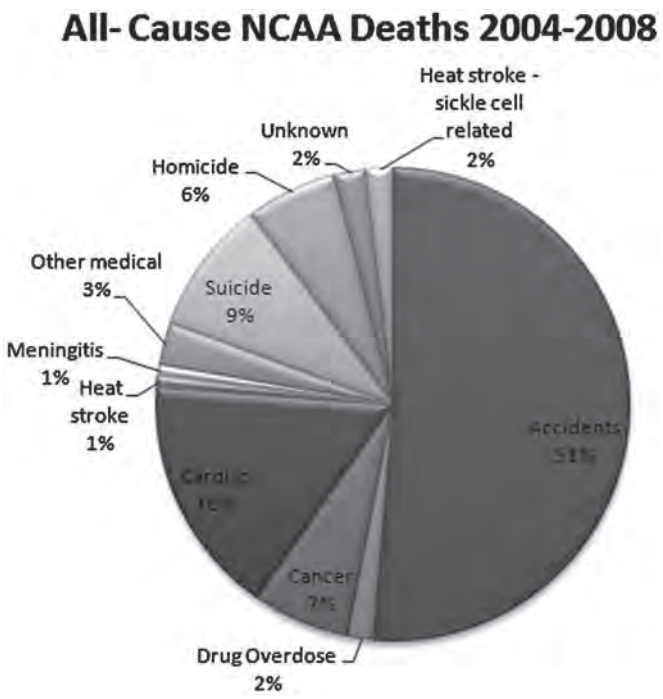

Figure 1 All-cause NCAA deaths 2004–2008.

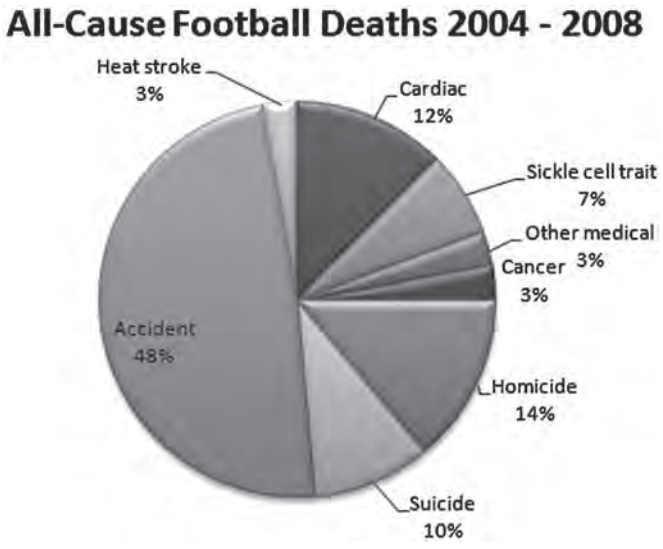

Figure 2 All-cause football deaths 2004–2008.

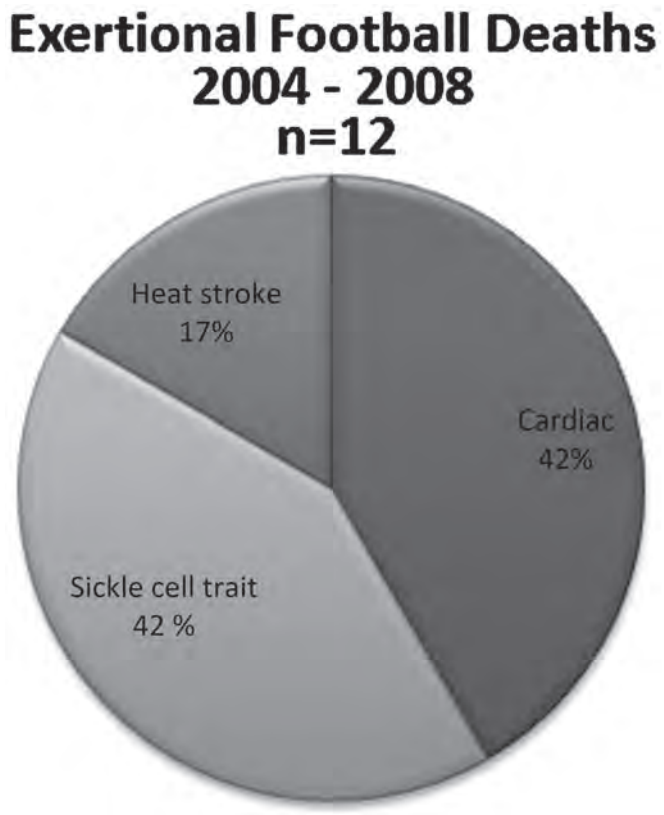

Figure 3 Exertional deaths 2004–2008; n=12.

death and SCA in young people created from weekly internet searches. The PHW Database was reviewed, and deaths in NCAA athletes were identified and cross-referenced with the NCAA Resolutions list, which was then combined into a single database.

Missing information regarding cause of death was acquired through internet searches and media reports, reports in the NCAA News as well as emails and telephone calls to sports information directors, head athletic trainers, athletic directors and coroners. When possible, the student-athlete's parents or guardians were interviewed. The study was approved by the University of Washington Division of Human Subjects.

Demographic data in NCAA athletes were obtained from the NCAA Sport Sponsorship and Participation Rates Report[11] and the NCAA Student Ethnicity Report.[12] The gender and ethnic information is provided by institutions to the NCAA annually, and these reports are compiled periodically by the NCAA Department of Research. The number of athletes participating overall and in football in each division was recorded. The numbers of black athletes and non-black athletes were also noted, and the numbers of those with SCT were estimated as being 7% of black athletes and 0.16% of non-black athletes (the vast majority of who were Caucasian) according to prevalence rates reported by the Centers for Disease Control (CDC).[5] Death in NCAA athletes was noted and death in football athletes was examined with particular attention to death in athletes with SCT.

The cost-effectiveness of screening athletes for SCT was explored to estimate the cost per life-year gained of different screening models assuming that identification of an athlete with SCT would prevent their death. The cost of screening tests used to identify SCT varies according to the type of test as well as regional variations in cost. Three test costs were examined:[1] $5,[2] $30 and[3] $150. The 'simple $5 screening test' was initially identified by the NCAA as a viable test method. Beginning in October 2011, the NCAA has contracted with a laboratory to provide screening haemoglobin solubility tests to member institutions for about $10 and haemoglobin electrophoresis for ~$30. Haemoglobin electrophoresis can cost up to $150 at some institutions. Cost-effectiveness was calculated assuming an initial start-up year when all athletes were screened. Screening in subsequent years occurred only for incoming athletes and was calculated assuming no start-up year. For simplicity, it was assumed that testing for SCT was 100% sensitive and specific and that identification of an athlete with SCT would prevent their death representing a best-case scenario. It was also assumed that prevention of death in a college-aged athlete would afford an additional 50-year life expectancy (approximating the average life expectancy).

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Table 1 Exertional death rates with and without sickle cell trait

NCAA Athletes 2004 to 2008

	Number of athletes 2004 to 2008	Estimated number of athletes with SCT	Annual risk of death for all athletes related to SCT	Risk of exertional death in athletes with SCT	Estimated number of athletes without SCT	Number of exertional deaths in athletes without SCT	Risk of exertional death in athletes without SCT	RR of exertional death in those with SCT versus those without
All ethnicities								
All divisions, all sports	1 969 663	23 529	393 933	4706	1 946 134	28	69 505	15
Division I athletes all sports	788 033	12 512	157 607	2502	775 521	16	48 470	19
All divisions, football athletes	308 042	7432	61 608	1486	300 610	7	42 944	29
Division I football athletes	126 447	4134	25 289	827	122 313	4	30 578	37
Black athletes								
All divisions, all sports	297 911	20 854	59 582	4171	277 057	13	21 312	5
Division I athletes all sports	164 490	11 514	32 898	2303	152 976	8	19 122	8
All divisions, football athletes	101 450	7102	20 290	1420	94 349	4	23 587	17
Division I football athletes	57 488	4024	11 498	805	53 464	3	17 821	22
Non-black athletes								
All divisions, all sports	1 671 752	2675	–	–	1 669 077	15	111 272	–
Division I athletes all sports	623 543	998	–	–	622 545	7	88 935	–
All divisions, football athletes	206 592	331	–	–	206 261	3	68 754	–
Division I football athletes	68 959	110	–	–	68 849	1	68 849	–

SCT, sickle cell trait;
NCAA, National Collegiate Athletic Association;

A cost per life year saved less than $50 000 was considered cost-effective.

RESULTS

During the 5-year period from 2004 to 2008, there were 273 deaths and a total of 1 969 663 athlete-participant-years. Five (2%) of all deaths occurred in athletes with SCT(figure 1). Football represented 72 (26%) of all student-athlete deaths. Of these, 52 (72%) were due to trauma unrelated to sports activity and 20 (28%) were due to medical causes (figure 2). The leading medical cause of death in football athletes was cardiac (9, 45%), followed by death associated with SCT (5, 25%), heat stroke unrelated to SCT (2, 10%), cancer (2, 10%), diabetes (1, 5%) and sepsis (1, 5%). No deaths were related to head trauma or concussion. No deaths occurred during competition.

Deaths which occurred with exertion were analysed. These included deaths due to cardiac causes, heat illness and deaths associated with SCT. In football players, there were 12 exertional deaths. Of the exertional deaths, five were related to cardiac causes (42%), five were in athletes with SCT (42%) and two were from heat stroke in athletes without SCT (15%) (figure 3).

All deaths in athletes with SCT occurred in black Division I football athletes during practice or conditioning. Thus, the risk of death associated with SCT in non-football athletes or non-black athletes in this cohort was zero. For those identified with SCT, the risk was quite high. Overall, the risk of exertional death associated with SCT was 1:4706 for all athletes (all sports, all divisions, all ethnicities) and 1:4171 for black athletes (all sports, all divisions). For Division I football players (all ethnicities) with SCT the risk of death was 1:827, and in black Division I football athletes with SCT the risk was 1:805 (table 1).

The RR of exertional death in athletes with SCT (all divisions, all sports) was 15 times higher than exertional death in athletes without SCT. In Division I football athletes (all ethnicities) with SCT, the risk of exertional death was 37 times greater than exertional death in those without SCT, and in black Division I football players with SCT the risk of exertional death was 22 times greater than exertional death in black players without SCT (table 1). The overall risk of exertional death from any cause was 5.2 times higher in black athletes with or without SCT compared with that in non-black athletes.

Cost-effectiveness of screening was dependent on the population screened, the price of the screening test and the assumption that identification of athletes with SCT prevents death. Four subgroups were examined;[1] black football athletes (all divisions),[2] football athletes (all ethnicities, all divisions),[3] black athletes (all sports, all divisions) and[4] all athletes (all ethnicities, all sports and all divisions). The cost estimate included a first year of screening all athletes at an institution followed by only screening incoming athletes over the 5 year study. The total cost of the programme ranged from $202 900 (black football athletes) to $119 539 920 (all athletes). The cost to prevent one death over 5 years ranged from $40 580 (black football athletes) to $23 907 984 (all athletes). The cost per life-year saved ranged from $812 to $478 160 (table 2). Screening only incoming athletes was cost-effective in all groups if a $30 test was used costing $2435 per year life saved (black football athletes) to $47 816 (all athletes) (table 3).

DISCUSSION

SCT is associated with exertional death in NCAA football players with a rate of 1:827 in Division I football athletes. The death rate is 37 times higher in football athletes with SCT than those without. Death in other athletes with SCT or those undergoing strenuous physical exertion has been reported although in this cohort no other sports were represented.[1 3 4 8 9 13–15] The National Center for Catastrophic Sport Injury Research has documented 12 deaths association with SCT, seven of which occurred between 2000 and 2008. Other experts have cited up to 19 deaths associated with SCT in NCAA athletes since 1973 (Eichner, personal communication). All but 2 of these 19 deaths have been in Division I football players. There has been one NCAA male basketball athlete playing pick-up basketball

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Table 2 Cost-effectiveness analysis of sickle cell screening for 2004 to 2008 (including first year start-up cost)

Cost-effectiveness analysis of sickle cell screening for 2004 to 2008 (including first year start-up costs)				
	Black football athletes	**All football athletes**	**All black athletes**	**All athletes**
Number of athletes	101 450	308 043	297 911	1 992 332
Total screens first year	20 290	61 609	59 582	398 466
Total screens every year after	5073	15 402	14 896	99 617
Total number of athletes screened in 5 years	40 580	123 217	119 164	796 933
Cost of screening programme				
'Simple $5 test'	$202 900	$616 086	$595 822	$3 984 664
$30	$1 217 400	$3 696 516	$3 574 932	$23 907 984
Hgb electrophoresis – $150	$6 087 000	$18 482 580	$17 874 660	$119 539 920
Cost to identify one athlete with SCT				
'Simple $5 test'	$71.43	$207.24	$71.43	$427.35
$30	$429	$1243	$429	$2564
Hgb electrophoresis – $150	$2143	$6217	$2143	$12 821
Cost to prevent one death over 5 years				
'Simple $5 test'	$40 580	$123 217	$119 164	$796 933
$30	$243 480	$739 303	$714 986	$4 781 597
Hgb electrophoresis – $150	$1 217 400	$3 696 516	$3 574 932	$23 907 984
Cost per life-year saved				
'Simple $5 test'	$812	$2464	$2383	$15 939
$30	$4870	$14 786	$14 300	$95 632
Hgb electrophoresis – $150	$24 348	$73 930	$71 499	$478 160

SCT, sickle cell trait.

Table 3 Cost-effectiveness analysis sickle cell screening for 2004 to 2008 (no start-up costs with $30 test)

Cost-effectiveness analysis sickle cell screening for 2004 to 2008 (no start-up costs with $30 test)				
	Black football athletes	**All football athletes**	**All black athletes**	**All athletes**
Total number of athletes screened in 5 years	20 290	61 609	59 582	398 466
Cost of screening	$30	$30	$30	$30
Cost of screening programme	$608 700	$1 848 270	$1 787 460	$11 953 980
Cost to identify one athlete with SCT	$429	$27 957	$429	$22 047
Cost to prevent one death over 5 years	$121 740	$369 654	$357 492	$2 390 796
Cost per life-year saved	$2435	$7393	$7150	$47 816

SCT, sickle cell trait.

and one male track athlete who have reportedly died from exertional collapse in athletes with SCT.

Although there is an association between exertional death and SCT, the pathophysiology of death is unknown. It has been proposed that a combination of high-intensity exercise, dehydration and thermal strain may induce sufficient hyperthermia, hyperosmolality, acidosis and red cell dehydration to lead to significant erythrocyte sickling, inflammation and microvascular occlusion causing exertional collapse in some exercisers with SCT.[2 15–18] Blood viscosity and adhesion properties are also altered in athletes with SCT.[19–26] The proposed mechanism of death is arrhythmia caused by hyperkalaemia from muscle necrosis and altered ion channel permeability.[1 2 16 18] However, others argue that this is unlikely citing the contrast between the proposed pathophysiology and the known problems seen in people with sickle cell disease who experience sickling episodes with splenic, pulmonary and bony infarcts. Environmental factors such as exercise intensity, altitude, hydration status and concurrent illness likely play a role in the development of exertional death in SCT. There may also be other genetic co-factors which make some athletes with SCT more likely to be affected than others.[18]

Strategies to prevent death in those with SCT seem warranted. Restriction from play is not recommended, but instead screening and targeted interventions are needed in those with SCT.[27 28] Determining the SCT status of athletes can be accomplished by reviewing previous medical history or repeat testing. All newborns are screened at birth for sickle cell disease where early interventions in those homozygous for Hgb S have shown to positively impact health. While SCT status is also identified, this information may not be communicated and is often not recalled by the athlete or their parents. If SCT status is not known, testing can be accomplished by several methods. A haemoglobin solubility test is recommended by the NCAA at a minimum. In this test, a reagent is added to blood. Hgb S is insoluble compared with other haemoglobins and the blood will appear cloudy instead of clear. This test does not distinguish between sickle cell disease, SCT and other haemoglobin variants and false positives and false negatives occur. A positive test should be followed up with additional testing to confirm and further define any abnormalities. The NCAA has contracted with a national laboratory to provide this testing to NCAA institutions for less than $10.

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Haemoglobin electrophoresis is a more accurate method to determine haemoglobin type. With electrophoresis, the haemoglobin migrates through a gel at different rates when an electrical charge is applied. Hgb electrophoresis can differentiate between different Hgb subtypes and is considered the most accurate method for identification of haemoglobinopathies. Isoelectric focusing has also been used to identify those with SCT. This test is similar to electrophoresis except the blood is run through a gel with a pH gradient. Electrophoresis and isoelectric focusing are considerably more expensive and more time-consuming to perform than Hgb solubility testing. Many institutions do not have the capacity to quickly perform multiple tests which can be problematic if athletic participation is restricted until SCT status is known.

From a public health perspective, screening should only be performed if there is an effective intervention. It is unclear if recommended interventions in athletes with SCT are effective. Recommended interventions include screening to identify athletes followed by education, targeted modifications of training and conditioning, an acclimatisation schedule, cessation of activity with any symptoms, and allowing SCT athletes to set their own pace. Athletes must also emphasise hydration, controlling asthma, oxygen availability at altitude and restricting workouts if he/she is ill.[27] However, deaths do occur in athletes despite knowledge of sickle cell status. During the cohort studied, the status of one athlete with SCT who died (20%) was known. In the last decade, there have been 10 exertional deaths in NCAA football players with SCT. The SCT status was known in three athletes (30%) based on publicly available records.

The armed services have different approaches to prevention of exertional death related to SCT. The United States Navy, Marine Corps and Air Force all screen recruits for SCT. The Army does not screen for SCT status; however, in 1982 in response to the increased number of basic training deaths in SCT recruits, the US Army implemented strict hydration policies including direct observation of recruit drinking and measurement of wet bulb globe temperature with modification of training if a certain threshold was reached. During the 10 years of the study period (1982 to 1991), there were no deaths related to SCT in the intervention group (2.3 million recruits) compared with an unchanged rate of deaths in non-participating centres (1.2 million recruits).[29] The conclusion of this study was that exertional heat illness is the predominant factor causing exercise-related deaths associated with SCT. It has also been demonstrated that exertional sickling can be prevented with adequate hydration.[17 30] Recruit deaths in the Army have increased since the initial intervention prompting the military to re-examine its policies.

CONCLUSIONS

SCT is associated with an increased risk of death in NCAA football athletes. While the most common medical cause of death in football athletes is sudden cardiac death, most athletes dying during exertion in football are positive for SCT. NCAA football players identified with SCT have an exceedingly high-risk of exertional death (1:827) that is 37 times higher than those without SCT. Screening for SCT status or signing a waiver is required of NCAA Division I athletes although the value of SCT athlete identification and targeted training modifications to reduce mortality has not been proven. If identification of athletes with SCT is effective in preventing mortality, then screening high-risk groups would be cost-effective.

What this study adds

- This study is the first study to examine the incidence of death associated with sickle cell trait (SCT) in college athletes.
- Sudden cardiac death is the leading cause of death in National Collegiate Athletic Association football athletes but SCT is an important and potentially preventable cause of death associated with exertion.
- The incidence of exertional death in athletes with SCT is 1:827.
- It is unknown whether screening prevents death associated with SCT.

Additional research is needed to understand the value of screening for SCT in NCAA athletes. Given all deaths in athletes with SCT in this cohort occurred in black Division I football athletes during practice or conditioning, it seems medically and ethically prudent to screen the highest risk group of athletes – at least black Division I football players, with consideration in all black athletes and/or all football players – and target educational interventions for the player, coaches and medical staff. The value of screening in other populations, for instance high school or recreational athletes, is unknown. Strict attention to heat and hydration has been demonstrated to prevent death in US military recruits and should be heavily emphasised in football practice and conditioning. Universal attention to adequate hydration, heat mitigation practices and not continuing to push a struggling athlete should be adopted in all sports.

Competing interests None.

Ethics approval Human Subjects Division at the University of Washington.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement De-identified data may be available. Requests should be made to the corresponding author.

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To screen or not to screen for sickle cell trait in American football?

Kimberly G Harmon,[1] Jonathan A Drezner,[2] Douglas J Casa[3]

Sickle cell trait (SCT) in America football athletes has been a topic of recent media attention. The deaths of college football athletes Ereck Plancher, Aaron O'Neil, Dale Lloyd and others as well as the recent decision of Pittsburgh Steelers safety Ryan Clark to forego a play-off game at altitude in Denver have contributed to the characterisation in the lay press of SCT as a dangerous condition. In fact, deaths in National Collegiate Athletic Association (NCAA) football players and concern from NCAA member institutions for the health and safety of student athletes led to legislation mandating all Division I athletes be screened or know their SCT status since August of 2010. On 26th January 2012, the American Society for Haematology (ASH) denounced the NCAA position. The disparity between these two positions is striking and deserves comment.

THE RELEVANT FACTS

SCT is common, with 1 in 14 (7%) of blacks having SCT and up to 1 in 625 Caucasians carrying the gene. SCT is generally considered benign and provides a survival advantage in areas endemic with malaria. In military recruits, the risk of exertional death is 28–30 times higher in those with SCT compared with those without SCT.[1] In Division I football players, the risk of exertional death in those with SCT is 1:827 versus 1:30 578 in those without (a 37-fold greater risk in athletes with SCT).[2] Exertional deaths have been reported in other athletes (basketball, track) but the vast majority of exertional deaths in those with SCT have occurred in African–American athletes playing Division I football. Although there is a clear association of exertional death with SCT, the precise pathophysiology of death is unknown.[3]

CONCERNS ABOUND

ASH expressed many concerns in their statement including the disclosure of protected health information and mandatory (vs voluntary) screening. However, some of these concerns may not apply to the college athletic setting. The transmission of medical information in an elite team setting is very different than informing an employer regarding personal medical information. Coaches are necessarily informed of not just SCT status, but of any medical condition a player has which may affect ability to perform or compromise their safety. Informing a coach of a player's SCT status is not much different than discussing an athlete with diabetes or asthma and precautions that should be undertaken.

ASH also recommends using universal preventive interventions for all athletes/recruits undergoing training. They recommend monitoring heat acclimatisation and work–rest cycles adjusted for the environment, implementing guidelines for hydration, and maintaining staff preparedness for early and rapid detection and treatment of heat illness. This approach has been used in the US Army although other branches of the military still opt to screen. Although these hydration and acclimation measures are advocated by the NCAA, deaths have occurred.

As the NCAA looks for additional strategies, it is unclear whether knowing an athlete's SCT status will prevent exertional death. Proponents of screening support directed education of athletes with SCT, and increased awareness and emergency planning for coaches and medical staff responsible for players with SCT. The last decade saw at least nine exertional deaths in NCAA athletes with SCT – three of these individuals were known to have SCT. Screening low-risk groups is likely to be ineffective. Perhaps the NCAA should focus screening on groups such as football athletes with demonstrated excessive risk.

On one hand, the NCAA has the duty to protect the health and safety of its athletes and a known high-risk group. NCAA policy provides all athletes with education on SCT as well as the option to opt out of screening without penalty to participation. On the other hand, ASH expresses concern that 'the NCAA policy attributes risk imprecisely, obscures consideration of other relevant risk factors, fails to incorporate appropriate counselling' and has the potential to harm the student athlete and the larger community. Caught in the middle is the sports physician.

Clearly, this debate is just maturing with major issues obscured by legal, societal and ethical concerns. In the meantime, clinical judgment and experience suggest the following:

- targeted screening of high-risk groups;
- aggressive educational intervention for athletes with SCT and those who supervise them (sports coaches, strength and conditioning coaches and medical staff);
- allowing appropriate time and access for hydration of athletes;
- gradual acclimation to novel activity and the heat;
- modification of activity in the heat and at altitude;
- appropriate strength and conditioning programmes developed by qualified strength and conditioning coaches;
- punitive exercise and conditioning sessions be prohibited;
- early recognition of athletes who are struggling, so they can immediately be allowed to rest and not pushed past their physiologic limit;
- adequate emergency planning for all individuals responsible for athletes during training and conditioning.

Competing interests None.

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[1]Department of Family Medicine and Sports Medicine and Orthopaedics, Hall Health Sports Medicine Clinic, University of Washington, Seattle, Washington, USA
[2]Department of Family Medicine, Hall Health Sports Medicine Clinic, University of Washington, Seattle, Washington, USA
[3]Department of Kinesiology, Neag School of Education, University of Connecticut, Connecticut, USA

Correspondence to Kimberly G Harmon, Departments of Family Medicine and Sports Medicine and Orthopaedics, 4060 East Stevens Circle, Hall Health Sports Medicine Clinic, University of Washington, Seattle, WA 98195, USA; kharmon@u.washington.edu

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Sudden exertional death in sickle cell trait

Mike Loosemore,[1,2] Stephen B Walsh,[3] Emma Morris,[4] Gordon Stewart,[5] John B Porter,[5] Hugh Montgomery[1]

[1]UCL Institute for Sport, Exercise & Health, London, UK
[2]English Institute of Sport, London, UK
[3]UCL Centre for Nephrology, Royal Free Hospital, London, UK
[4]Department of Haematology, UCL Cancer Institute, University College London, London, UK
[5]Division of Medicine, UCL, London, UK

Correspondence to
Dr Mike Loosemore, UCL Institute for Sport, Exercise and Health, Rosenheim Building, 25 Grafton Way, London WC1E 6DB, UK; mike.loosemore@eis2win.co.uk

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INTRODUCTION

While generally considered benign, sickle cell trait (SCT) is both common and associated with a risk of sudden exertional death. Indeed, all high-level US intercollegiate athletes now face mandatory screening, raising legal and ethical concerns that resonate given the 2012 London Olympics' role in driving universal sports' participation, and increasing public health messaging promoting physical activity. This issue is also of substantial clinical and scientific interest: the pathogenesis of such deaths is unknown and successful management thus impeded.

This article discusses both clinical presentation and relevant (patho)physiology. It also proposes that a vicious cycle of skeletal muscle microvascular occlusion and 'metabolic failure' drives potassium efflux from erythrocytes and myocytes and therefore, hyperkalaemic death. Such discussion may be of value to all dealing with subjects with SCT in the community, to emergency/intensive care physicians who treat such cases and to researchers seeking new methods of screening and modes of treatment.

STANDFIRST

The pathogenesis of exercise-related death among those with SCT is not understood. We hypothesise that potassium efflux from red cells and skeletal muscle causes hyperkalaemic death.

SICKLE CELL HAEMOGLOBIN

Each of the haemoglobin (Hb) molecule's four subunits contains an oxygen-carrying haem group and a globin molecule. Two α-subunits are bound to two γ-subunits in fetal haemoglobin (HbF, $\alpha_2\gamma_2$) or two β-subunits in adult haemoglobin (HbA, $\alpha_2\beta_2$). The haemoglobin in which two α-subunits are associated with two mutant β-subunits (in which a hydrophobic valine replaces a hydrophilic glutamic acid) is referred to as sickle cell haemoglobin (HbS).

Hypoxia and falling pH cause HbS to polymerise, distorting red blood cell (RBC) shape ('sickling'), whose extent depends upon the RBC concentration of HbS relative to HbA/HbF. This is lower in heterozygotes (HbAS with SCT) than homozygotes (HbSS with sickle cell disease (SCD)). Sickling-induced vaso-occlusion can lead to so-called 'sequestration crises', but can also damage organs including the bone, brain, lung and gut.

The mechanism of microvascular occlusion in SCD is not simply flow limitation through RBC deformation, however. Reticulocytes and permanently deformed dense cells adhere to the postcapillary venule endothelium via adhesion molecules including thrombospondin, vascular cell adhesion molecule-1 and cluster of differentiation 36. Leucocytes bind to sickled cells to form heterocellular aggregates, worsening obstruction through their inferior deformability, and through leucocyte-endothelial adhesion. Local inflammation amplifies endothelial activation and cellular adhesion. Vaso-occlusion worsens local hypoxia and thus HbS polymerisation, driving further occlusion (reviewed in reference 1).

VARIATION IN SCD PHENOTYPE

While possession of HbS is determined monogenically, variation in other genes influences SCD phenotype[2]: leucocyte concentration affects the incidence of acute chest syndrome and cerebral infarction while higher HbF concentration ameliorates SCD phenotype, and both are under strong genetic influence.[2] Nitric oxide (NO) bioavailability influences endothelial/sickle cell adhesion and variation in the endothelial nitric oxide synthase (eNOS) gene is associated with variation in the incidence of chest syndrome in SCD.[3]

Other candidate genes of influence might include those regulating RBC membrane cation transport, which regulate cell hydration state and consequently cellular HbS concentration and the propensity to polymerisation. Such cation transporters include the calcium-activated K^+ (Gardos) channel, the potassium–chloride co-transporter and the deoxygenation-induced cation-selective channel (P_{sickle}), which promotes calcium influx and Gardos activation.[4]

SCT AND SUDDEN DEATH

SCT is common (affecting <8% of the USA black population) and is associated with a modest increased risk of thromboembolism, haematuria and impaired urinary concentrating ability. However, the risk of exertional death is greatly elevated, with athletes now being screened for SCT in the USA.[5] Among 2.1 million US military recruits, rates of otherwise unexplained death were 32.2/100 000 and 1.1/100 000 for recruits with and without SCT, respectively (RR 27.6). Risk has been confirmed in US military recruits[6] and in recreational and professional athletes,[7] and rises with age (an eightfold cline from late teens to late twenties) and intensity of exertion.[8]

In such cases, intense exercise immediately precedes a rapid decline and death within minutes or hours. Such rapid unanticipated collapse (together with ignorance of HbS status and of the clinical scenario) means that the mode of death is often poorly described. While 'heat stroke' and rhabdomyolysis are often associated, they might occur

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independently and neither seems universally present (perhaps one third of cases exhibit neither). Profound hyperkalaemia is, however, frequently identified,[9] whether in isolation or in the context of rhabdomyolysis, and may prove resistant to interventions including haemodialysis.[10]

PUTATIVE MODEL

Sustained exercise evokes four pro-sickling forces: local hypoxia, acidosis, raised temperature and RBC dehydration. Even in the absence of sickling-related microvascular occlusion, however, exercise-related changes in RBC rheology may impair tissue oxygen delivery directly and indirectly by driving local inflammatory responses and adhesion molecule expression.[11]

Whether through exercise-induced microvascular sickling or rheological impairment, SCT may suddenly impede skeletal muscle oxygen delivery and worsen local acidaemia, thus worsening flow limitation in a positive feedback loop and triggering skeletal muscle 'metabolic failure'. The frequent identification of rhabdomyolysis is in keeping with this hypothesis: a fall in ATP production plays a fundamental role in the pathogenesis of exertional rhabdomyolysis through failure of the muscle sodium pump. In this context, associated potassium efflux could drive catastrophic hyperkalaemia.

Serum potassium might abruptly rise for other reasons too. RBC membrane deformation during HbS polymerisation induces potassium leak[12] that worsens cellular dehydration, favouring further HbS polymerisation. The anion exchanger 1 (AE1), a major structural membrane component that also mediates chloride–bicarbonate exchange may play a crucial role in this. Normal RBCs demonstrate a (likely AE1-mediated) cation leak when undergoing shear stress,[13] and AE1 is the proposed mediator of deoxygenation-induced cation-selective channel (P_{sickle}) cation flux.[14] When compared with normal RBCs, sickle RBCs demonstrate an exaggerated potassium leak in response to shear stress, which is further enhanced at lower pHs.[15] Furthermore, sickle RBC membrane components including AE1 are readily oxidised and even subtle peroxidation causes exaggerated cation leaks from the RBC membrane in response to mild shear stress. Furthermore, RBC deformation and oxidation interact synergistically to promote cation leak.[16] Thus, deformation, hypoxia and low pH conditions that the RBC encounters in exercising muscle promote the efflux of potassium from RBCs.

The skeletal muscle vascular bed and thus the mass of affected RBCs and the scale of potassium efflux is large. Augmenting that derived from skeletal muscle metabolic failure and in the context of the described positive feedback loops of sickling and potassium efflux, catastrophic hyperkalaemia may result (figure 1).

DEATH IN SCD

If such a mechanism exists, ought it not to be readily identified in those with SCD?

In general, SCD-related death may result from acute end-organ damage from sickle crises or sepsis related to functional hyposplenism. Chronic end-organ damage (renal failure or pulmonary hypertension) contributes to SCD deaths, rarely, exclusively explaining a sudden fatal deterioration. Transfusion-related iron overload (causing cirrhosis and cardiomyopathy) also occurs.

However, SCD death is 'frequently sudden and unexpected',[17] one detailed series documenting 'sudden unexpected hospital or home death in an otherwise relatively healthy patient, with or without vaso-occlusive crisis' as the cause in 23.4%.[18] A further 22.6% died of 'renal failure' and 4.2% of 'multiorgan failure', the authors commenting that 'most patients had more than one factor contributing to death'. The prevalence of identified hyperkalaemia or rhabdomyolysis (or a history

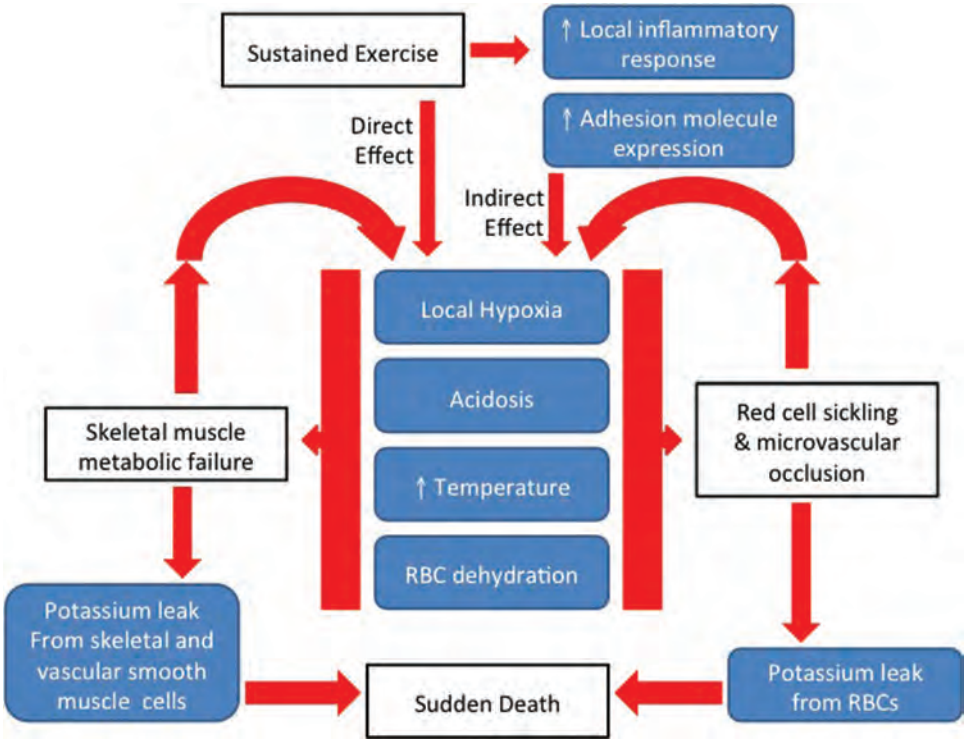

Figure 1 A proposed schematic of the pathophysiological processes which might culminate in exertional death in SCT subjects: a final common pathway of hyperkalaemia is suggested.

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of preceding exertion) in these cases is not known. It might thus be that hyperkalaemia related to muscle microvascular flow limitation does occur in SCD but goes unrecognised. Alternatively, such cases might not be prevalent in adults if a propensity to such pathology proved lethal earlier in life ('survivor bias').

A POLYGENIC PATHOGENESIS?

But why would only some individuals with SCT be affected by exertional sudden death? Environmental factors (exercise intensity, altitude, hydration status and the presence of intercurrent infection) may interact with genetic variation to influence risk.

A variety of candidate genes might be suggested. Gene variation influences cation leak via AE1[19] or the ammonium transporter rhesus-associated glycoprotein,[20] while variation in the eNOS gene may also play a role (above), especially given the male preponderance of sudden death in SCT and the reduced bioavailability of NO in males with SCD.[1] Further candidates include genes influencing HbA/HbF/HbS concentrations; nicotinamide adenine dinucleotide phosphate oxidase 1 (NOX-1) or NOX-4,which are expressed in vascular smooth muscle cells and generate vasoconstricting local superoxides; and glucose-6-phosphate dehydrogenase (G6PD), the rate-limiting step in the pentose phosphate pathway that generates nicotinamide adenine dinucleotide phosphate, eliminating vasoconstricting reactive oxygen species. G6PD gene variants would also influence RBC oxidation and thus potassium permeability, while G6PD deficiency decreases endothelial NO bioavailability.

SUMMARY

In summary, SCT is associated with exertional sudden death. We propose a fundamental role for hyperkalaemia in mediating such deaths. We suggest that local hypoxia and acidosis worsen HbS polymerisation in a vicious (and viscous) circle. Resultant rheological impairment (possibly augmented by local sickling) leads to a sudden decline in skeletal muscle oxygen availability causing metabolic failure. Resultant potassium extrusion is augmented by that resulting directly from RBC deformation. Variation in genes influencing microvascular flow and potassium efflux may modulate the risk of exertional death across a seemingly homogeneous population.

We would advocate research in this area. Once initiated, catastrophic decline may prove hard to arrest, meaning that a preventive strategy might best be deployed. While this should certainly include modification of training patterns, it might also be that individuals at risk could be identified through the use of genetic markers in the future.

Competing interest None.

Provenance and peer review Not commissioned; externally peer reviewed.

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SPECIAL ISSUE: BRIDGING THE GAP BETWEEN RESEARCH AND HEALTH POLICY–INSIGHTS FROM THE ROBERT WOOD JOHNSON FOUNDATION CLINICAL SCHOLARS PROGRAM

A Policy Impact Analysis of the Mandatory NCAA Sickle Cell Trait Screening Program

Beth A. Tarini, Margaret Alison Brooks, and David G. Bundy

Objective. To estimate the impact of the mandatory National Collegiate Athletic Association (NCAA) sickle cell trait (SCT) screening policy on the identification of sickle cell carriers and prevention of sudden death.

Data Source. We used NCAA reports, population-based SCT prevalence estimates, and published risks for exercise-related sudden death attributable to SCT.

Study Design. We estimated the number of sickle cell carriers identified and the number of potentially preventable sudden deaths with mandatory SCT screening of NCAA Division I athletes. We calculated the number of student-athletes with SCT using a conditional probability based upon SCT prevalence data and self-identified race/ethnicity status. We estimated sudden deaths over 10 years based on published attributable risk of exercise-related sudden death due to SCT.

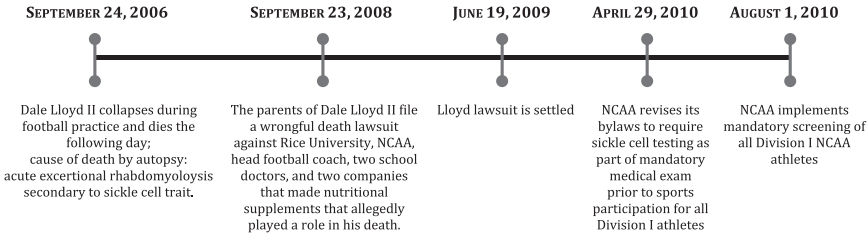
Principal Findings. We estimate that over 2,000 NCAA Division I student-athletes with SCT will be identified under this screening policy and that, without intervention, about seven NCAA Division I student-athletes would die suddenly as a complication of SCT over a 10-year period.

Conclusion. Universal sickle cell screening of NCAA Division I student-athletes will identify a substantial number of sickle cell carriers. A successful intervention could prevent about seven deaths over a decade.

Key Words. Sickle cell trait, National Collegiate Athletic Association, screening, sudden death, athletes

On August 1, 2010, the National Collegiate Athletic Association (NCAA) implemented one of the largest mandated genetic screening programs in the United States: universal sickle cell trait (SCT) screening of all Division I student-athletes (NCAA 2010b). The policy resulted from a legal settlement with the family of Dale Lloyd II, a Rice University football player, who collapsed during football practice and later died from acute exertional rhabdomyolysis attributed to SCT (Figure 1).

Figure 1: Timeline of Significant Events in the Implementation of NCAA Division I Sickle Cell Screening Program



The morbidity associated with SCT has long been debated (Sears 1978). Complications of SCT are rare and include infarction associated with hypoxia from high altitudes and renal medullary carcinoma (Tsaras et al. 2009). However, there is also concern that athletes with SCT have an elevated risk of exercise-related sudden death secondary to exertional heat illness or acute exertional rhabdomyolysis (Eichner 2010).

The NCAA's mandatory, universal SCT screening program (NCAA Proposal No. 2009-75-B) has sparked significant debate. Endorsement of this policy from professional organizations has been mixed (College of American Pathologists 2007; National Athletic Trainers' Association 2007; Secretary's Advisory Committee on Heritable Disorders and Genetic Diseases in Newborns and Children's 2010). This is due to fear that screening may lead to discrimination and confusion (Bonham, Dover, and Brody 2010), as occurred with previous community and public health sickle cell screening programs (Whitten 1973; Rutkow and Lipton 1974), as well as a paucity of evidence that screening improves health outcomes for student-athletes.

Given the scope and controversy of the screening program, we conducted a policy impact analysis (Porell and Adams 1995) to estimate the number of athletes with SCT identified and the anticipated number of exercise-related sudden deaths attributable to SCT among NCAA student-athletes.

Address correspondence to Beth A. Tarini, M.D., M.S., Assistant Professor of Pediatrics, Child Health Evaluation and Research Unit, Division of General Pediatrics, University of Michigan, 300 N. Ingalls Street, Room 6C11, Ann Arbor, MI 48109-5446; e-mail: btarini@umich.edu. Margaret Alison Brooks, M.D., M.P.H., is with the Department of Orthopedics Surgery & Pediatrics, University of Wisconsin, Madison, WI. David G. Bundy, M.D., M.P.H., is with the Department of Pediatrics, Johns Hopkins University School of Medicine, Baltimore, MD.

METHODS

Study Population: Number of Student-Athletes

We used the most recently published, publicly available NCAA participation rates (academic year 2007–2008) to estimate the number of Division I student-athletes in a 4-year cohort (e.g., all participating student-athletes in an academic year) (NCAA 2009). These data are self-reported by the 335 NCAA Division I member institutions (National Collegiate Athletic Association 2011).

The NCAA defines student-athletes as individuals who meet at least one of the following criteria by the first scheduled competition: (1) are listed as a team member, (2) practice with the varsity team and receive coaching from one or more varsity coaches, or (3) receive athletically related student aid. If a student participates in more than one sport, the NCAA counts him/her as a student-athlete participant for each sport. However, we are unaware of any publicly available NCAA statistics on the number of multisport student-athletes. To decrease the likelihood of double-counting athletes, we assumed as follows: (1) athletes who competed in cross-country also competed in track and (2) athletes who competed in indoor track also competed in outdoor track and so only included participants in outdoor track (i.e., did not include participants listed for cross country and indoor track) in our analyses.

The NCAA assigns sports to one of three categories: championship (men and women, e.g., basketball), nonchampionship (men only, e.g., rowing), and emerging (women only, e.g., rugby). The NCAA policy does not explicitly exempt student-athletes in nonchampionship and emerging sports from screening, so we included these athletes in our overall calculation of "screen-able" student-athletes. Moreover, the NCAA requires that "the [medical] examination or evaluation [which includes a sickle cell solubility test] must have been administered within six months prior to participation in any weight-training or conditioning activity" (NCAA 2010b). As all athletic activities involve weight-training or conditioning activity, student-athletes in all three NCAA sport categories meet criteria for SCT screening.

Study Population: Race/Ethnicity of Student-Athletes

Because the prevalence of SCT is increased in Black and Hispanic/Latino individuals compared to Whites (Blacks: 1/14, Hispanic/Latino: 1/183, Whites: 1/625) (Lorey, Arnopp, and Cunningham 1996), we gathered data

on the race/ethnicity distribution among all NCAA Division I student-athletes for the years 2007–2008 (NCAA 2010a). These data are self-reported by athletes. The NCAA categorizes race/ethnicity as follows: American Indian/Alaskan Native, Asian, Black/African American, Hispanic/Latino, Native Hawaiian/Pacific Islander, Other, Two or More Races, White/Non-Hispanic. "Black, Non-Hispanic" was defined as "A person having origins in any of the black racial groups of Africa (except those of Hispanic origin)"; "Hispanic/Latino" was defined as "a person of Mexican, Puerto Rican, Cuban, Central or South American or other Spanish culture or origin, regardless of race" (NCAA 2010a). We collapsed these categories as follows (Lorey, Arnopp, and Cunningham 1996): Black, Non-Hispanic; Hispanic; All Other.

Prevalence of SCT

Literature-derived estimates for the prevalence of SCT vary by ethnicity. Classically, discussion of the prevalence of SCT has focused exclusively on individuals of African ancestry. However, studies report an increased prevalence in Hispanics/Latinos compared to individuals of non-Hispanic/non-African ancestry. We used the following published point estimates for prevalence: 7 percent of blacks, 0.5 percent of Hispanics/Latinos, and 0.2 percent of whites (Lorey, Arnopp, and Cunningham 1996). We applied the 0.2 percent prevalence for whites to our "Other" race/ethnicity category.

Rates of Death for Exercise-Related Sudden Death from SCT

We identified only one study that systematically quantifies the risk of exercise-related sudden death associated with SCT. It was conducted in U.S. military recruits and categorized reasons for death into three categories: sudden unexplained death, sudden nonunexplained death (e.g., silent structural heart disease, asthma), nonsudden death (Kark et al. 1987). "Sudden death" was defined as "death due to an illness producing an irreversible critical condition within 1 hour of onset." The authors categorized exertional heat stress, heat stroke, rhabdomyolysis, and unknown mechanism as "sudden unexplained death." We used the death rate for "sudden unexplained death" in our analyses because it included hypothesized mechanisms (e.g., exertional heat stress, rhabdomyolysis) of exercise-related sudden death among individuals with SCT (Eichner 2010). For the risk of sudden death due to SCT in our analysis, we used the attributable death rates for sudden death among black recruits (31 deaths per 100,000 individuals) with SCT,

calculated by subtracting the death rate among black recruits with SCT (32.2 per 100,000) from the death rate among black recruits without SCT (1.2 per 100,000).

Analysis

To estimate the number of student-athletes who would suffer exercise-related sudden death due to SCT, we calculated the conditional probability of an exercise-related sudden death for student-athletes based upon the likelihood of an athlete having SCT. We assumed full compliance of all Division I member institutions with this mandatory screening policy.

Number of Athletes with SCT. First, we calculated the conditional probability of having SCT based upon ethnic group status, stratified by sport and by gender:

$$P(\textit{Having SCT}) = P(\textit{Race/Ethnicity}) \times P(\textit{Sickle cell trait given race/ethnicity})$$

We multiplied this probability by the number of Division I student-athletes in a 4-year cohort to estimate the number of individuals who have SCT among all Division I student-athletes. We multiplied this estimate by the sensitivity of the sickle cell solubility test (98.9 percent) to determine the number of athletes with SCT identified by testing (Hicks and Hughes 1975). We then divided our total student body estimate by 4 to determine the number of athletes with SCT identified in each academic class (i.e., those identified annually).

Number of Sudden Deaths Attributable to SCT. We estimated the probability of exercise-related death from SCT conditioned upon the likelihood of having SCT, using published risk estimates (Kark et al. 1987):

$$P(\textit{Death/SCT}) = P(\textit{Death among individuals with SCT}) \times P(\textit{Having SCT})$$

To estimate the number of expected sudden deaths in an academic year, we multiplied this probability by the number of Division I athletes participating in each sport during an academic year (i.e., 4-year cohort). We used this statistic because each year every participating athlete (newly identified SCT or already known) is at risk of sudden death from SCT. We multiplied the

number of deaths by 10 to estimate deaths over a 10-year time period, which allowed us to benchmark our model against estimates of sudden death attributable to SCT based on case reports.

Although female athletes anecdotally constitute a lower proportion of exercise-related death cases related to SCT than male athletes, we are unaware of any published data confirming this and so did not apply a gender-based differential probability of death.

Number Needed to Screen and Cost of Testing. Finally, we calculated the number needed to screen (NNS) to prevent one student-athlete death from SCT (assuming a 100 percent effective intervention). Because our data do not provide the athlete's eligibility year, we calculated the NNS over a 4-year period by assuming that ¼ of participants represented a freshman class that would only be screened once during this 4-year period.

The NCAA policy recommends that athletes be screened with a sickle cell solubility test (National Collegiate Athletic Association 2010). Positive sickle cell solubility tests must be confirmed with hemoglobin (Hb) electrophoresis. We calculated the number of student-athletes with positive sickle cell solubility tests that would undergo Hb electrophoresis by multiplying the published sensitivity estimate of 98.9 percent for sickle cell solubility testing by the number of athletes with SCT in the total athletic student body (Table 1). We did not examine the costs for false-positive results since the published specificity estimate for sickle cell solubility testing is 100 percent (Hicks and Hughes 1975). To calculate the cost of screening and confirmatory testing alone (excluding education and counseling costs), we used a range of costs for the sickle cell solubility testing ($10–$20) provided by different collegiate institutions (Secretary's Advisory Committee on Heritable Disorders in Newborns Meeting 2010; University of California-Berkeley 2010; Wesleyan University 2010). We used $50 for the cost of hemoglobin electrophoresis (Chasen, Loeb-Zeitlin, and Landsberger 1999; January 22, 2010).

RESULTS

In the 2007–2008 season, 81,073 male and 63,108 female student-athletes participated in NCAA Division I sports. Football (n = 25,658) had the most participants for men's sports and track for women's sports (n = 11,230) (Appendix SA2, Supporting Information). Overall, the majority of Division I

Table 1: Estimated Number of Division I NCAA Student-Athletes with Sickle Cell Trait

	Black	*Hispanic*	*Other*	*Total*
Archery	0	0	0	0
Badminton	0	0	0	0
Baseball	43	3	18	64
Basketball	384	1	9	393
Bowling	9	0	0	9
Equestrian	0	0	1	2
Fencing	3	0	1	4
Field hockey	2	0	3	6
Football	833	3	26	863
Golf	13	1	9	23
Gymnastics	5	0	3	8
Ice hockey	1	0	5	6
Lacrosse	6	0	9	16
Rifle	0	0	1	1
Rowing	11	1	12	24
Rugby	0	0	0	0
Sailing	0	0	0	0
Skiing	0	0	1	1
Soccer	68	4	23	95
Softball	28	2	9	39
Squash	0	0	1	1
Swimming/diving	8	1	17	26
Sync. swimming	0	0	0	0
Team handball	0	0	0	0
Tennis	22	2	10	34
Track, outdoor	430	4	29	463
Volleyball	40	1	9	50
Water polo	1	0	2	4
Wrestling	11	1	5	16
All sports	1,918	25	204	2,147*

*This number is multiplied by 0.989 to calculate the number of athletes identified by sickle cell solubility testing = 2,123 athletes.

student-athletes self-reported a non-Black, non-Hispanic/Latino race/ethnicity (male 71.1 percent, female 80.4 percent, Appendix SA3).

Among the estimated 2,147 athletes with SCT, we estimate that 2,123 NCAA Division I student-athletes in one 4-year cohort of athletes (i.e., total athletic student body) would be identified as having SCT under this new screening policy (about 530 new students annually) (Table 1). Of these, 89 percent will self-report as Black race/ethnicity. Most student-athletes with SCT will compete in football (n = 863), track (n = 463), and basketball (n = 393).

In a 4-year cohort of screened NCAA student-athletes (e.g., a 4-year period with each athlete screened only once), there would be one death. Based on our model, we estimated that without intervention, about seven student-athletes with SCT would suffer exercise-related sudden death by year 10 of the screening program (Table 2).

Assuming a 100 percent effective intervention, this screening program requires that 144,181 student-athletes (e.g., a 4-year cohort of athletes) be screened to prevent one death. For each death prevented, we estimate that the cost of testing alone (i.e., without education or counseling) would range from $1,441,810 to $2,883,620 for sickle cell solubility testing and be about $106,150 for hemoglobin electrophoresis confirmatory testing for those with positive sickle cell solubility tests.

Discussion

The NCAA recently implemented mandatory SCT screening of all Division I student-athletes. Using publicly available data, we estimated that this policy would identify over 2,000 athletes with SCT among a 4-year cohort of student-athletes. Alongside a 100 percent effective intervention, screening could prevent the deaths of seven student athletes over a 10-year period. Our estimate approximates the number of case reports of collegiate student-athletes that have suffered exercise-related death attributed to SCT over a similar historical time interval (Eichner 2010, 2011).

A powerful motivating force behind the mandatory NCAA SCT screening policy was a wrongful death suit. As part of the settlement, the NCAA agreed to require that all Division I athletes be screened for SCT before sports participation (Lanier Law Firm 2009). Previously, the NCAA had recommended, but not required, that member institutions screen athletes for SCT (NCAA 2008b); compliance from member institutions had been variable (Clarke et al. 2006).

The NCAA SCT screening policy yields interesting comparisons to the controversy surrounding cardiovascular screening of college athletes to prevent sudden death. The NCAA does not currently recommend universal ECG screening for student athletes despite the fact that cardiovascular conditions underlie more than half of medically related sudden deaths among college athletes (Harmon et al. 2011) and data exist to support the notion that ECG screening may reduce the incidence of cardiovascular sudden death (Corrado et al. 1998). Admittedly, issues remain to be resolved (e.g., nontrivial

Table 2: Estimated Exercise-Related Deaths from Sickle Cell Trait in NCAA Division I Student-Athletes That Would Be Prevented over 10 Years

	Deaths
Archery	0.0
Badminton	0.0
Baseball	0.2
Basketball	1.2
Bowling	0.0
Equestrian	0.0
Fencing	0.0
Field hockey	0.0
Football	2.7
Golf	0.1
Gymnastics	0.0
Ice hockey	0.0
Lacrosse	0.0
Rifle	0.0
Rowing	0.1
Rugby	0.0
Sailing	0.0
Skiing	0.0
Soccer	0.2
Softball	0.1
Squash	0.0
Swimming/diving	0.1
Sync. swimming	0.0
Team handball	0.0
Tennis	0.1
Track, outdoor	1.4
Volleyball	0.2
Water polo	0.0
Wrestling	0.1
All sports	6.5

incidence and consequence of false-positive test results, uncertainty that screening would lead to a reduction in the number of deaths from cardiovascular causes). However, a reasonable argument could be made that the evidence base supporting an NCAA recommendation for universal ECG screening appears more robust than that for universal SCT screening.

The relationship between exercise-related death and SCT has long been debated. While ample evidence suggests that SCT has not limited the competitiveness of professional football players (Murphy 1973), national track champions (Pearson 1989; Bile et al. 1998), or elite sprinters (Marlin et al. 2005), case reports of exercise-related sudden death in athletes with SCT at all levels

of sports (e.g., high school through professional) suggest a compelling pattern. Exercise-related sudden deaths attributed to SCT tend to occur in male athletes, early in training (often on the first day), and during conditioning workouts. They tend to be associated with high levels of exertion, such as short, maximum-effort drills/testing, such as those frequently used in football, a sport in which numerous sudden deaths have been attributed to SCT (Eichner 2010, 2011). Environmental conditions, such as the heat and hydration status of the player, also play an important role. These data suggest that a gene–environment interaction promotes the initiation of acidosis and rhabdomyolysis that triggers sickling and creates a rapid downward spiral from which recovery is difficult (Eichner 2010).

The "treatment" for the athlete with SCT is proactive prevention that focuses on appropriate preseason and in-season conditioning. To this end, the NCAA recommends a number of precautionary strategies for athletes with SCT (Figure 2). There is evidence that preventive hydration and temperature-monitoring strategies are effective. A 1970s study of U.S. military recruits found an increase in unexplained sudden death among individuals with SCT, compared to those without (Kark et al. 1987). However, unlike the NCAA, the U.S. military implemented universal *intervention* rather than *screening*. In an experimental study of universal precautionary measures (e.g., aggressive hydration, temperature monitoring) *without* a priori knowledge of recruits' SCT status, the military was able to prevent all subsequent sudden death in recruits with SCT (Kark et al. 1999). This universal intervention program may yield positive spillover effects to other life-threatening conditions unrelated to SCT (e.g., exertional heat stroke) that benefit *all* athletes, not just those with SCT.

Of course, successful screening programs depend upon effective and accessible interventions. It is unclear how the NCAA enforces compliance with its "safe conditioning" recommendation and there does not seem to be a strong mandate for intervention. The NCAA informs athletes and member institutions that "Student-athletes with SCT should be knowledgeable of these precautions and institutions should provide an environment in which these precautions may be activated" (2008a). However, the ongoing challenge of compliance with sports-related concussion guidelines highlights the power of the "pressure to play" after an individual is identified as being at risk for sports-related injury. Such pressure to perform and dismiss early symptoms is likely no different for student-athletes with SCT. For example, in 2008 a collegiate football player died during a football-conditioning workout despite the fact that he and the athletic staff were aware that he had SCT. He had reportedly

Figure 2: NCAA Recommended Precautions for Student-Athletes with Sickle Cell Trait (NCAA 2010b)

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

tested positive for SCT *twice* in 2007 (Fainaru-Wada 2008)—highlighting that screening can lead to a false sense of security if an effective intervention is not enforced.

Although the NCAA policy may be based more litigation based, rather than evidence based, it has real-life implications for the athletes and schools forced to comply with it. First, there are economic costs to the member institutions that must carry out the education and testing. Second, there is the potential for discrimination toward athletes both during and after their collegiate

athletic careers. While precautions should be taken, athletic programs should not exclude these athletes or discourage them from being competitive, but rather, according to the NCAA "enable them to thrive in their sport" (2008a). Similarly, when some of these student-athletes transition to professional athletics, coaches and athletic directors should ensure that athletes with SCT do not suffer employment discrimination due to their genetic status. The history of screening for SCT is littered with stories of misinformation, stigma, and unjustified exclusion from activities and professions (Levin 1958; Whitten 1973).

Our policy analysis has limitations. First, NCAA Division II and III athletes are also at risk for exercise-related sudden death due to SCT. While they comprise a significant proportion (61 percent) of collegiate athletes, we did not include them in our formal analyses because the current NCAA SCT screening policy is limited to Division I athletes. Using identical methods, we calculated that 1,210 Division II and 675 Division III athletes would be identified as having SCT (Appendices SA4 and SA5). Assuming an effective intervention, it will take about 3 years to prevent a death in Division II and over 4 years in Division III (data not shown). Second, student-athletes that participate in multiple Division I sports could have been double-counted, leading to an overestimate of individuals with SCT. We have attempted to account for this by only including athletes who participated in outdoor track (i.e., not indoor track or cross country). Although some multisport athletes probably remain in our model, their effect on our results should be small and lead to only a slight overestimation of sudden deaths. Third, testing costs are based on publicly available costs provided to student-athletes by institutions and probably vary across institutions. However, we feel that the testing cost estimates used in our analysis are reasonable and illustrate that while the costs of individual tests are inexpensive, the cost of testing across all student-athletes is not trivial. Finally, we assumed that Division I college student-athletes do not have a greater or lesser proportion of individuals with SCT compared to the general population. Based on previous studies of elite athletes, we have no reason to believe that we have overestimated the prevalence of SCT in this population (Murphy 1973; Bile et al. 1998). In fact, a study of elite sprinters found an overrepresentation of individuals with SCT among title winners (Marlin et al. 2005).

Despite these limitations, we feel that our analysis has important implications for policy makers. First, it provides data about the scope and impact of a policy whose implementation was driven by litigation rather than evidence. Additional studies could explore the scope of screening-related harms, the costs of screening, and the effectiveness of and compliance with interventions. A successful screening program consists of both identification and action—in

this case, rigorous compliance with conditioning precautions. Evidence of this intervention's effectiveness is sorely needed, because *interventions*, not screening tests, save lives. For this screening policy to be successful, testing must be only the first step in the NCAA's overall strategy. However, the question still remains whether SCT screening is a necessary step to prevent exercise-related sudden death in student-athletes.

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Supporting Information

Additional supporting information may be found in the online version of this article:

Appendix SA1: Author Matrix.

Appendix SA2: Number of NCAA Division I Athletes in 2007–2008, by Sport and Gender.

Appendix SA3: Race/Ethnicity Distribution for NCAA Division I Athletes, 2007–2008, by Sport and Gender (%).

Appendix SA4: Estimated Number of Division II NCAA Student-Athletes Who Have Sickle Cell Trait.

Appendix SA5: Estimated Number of Division III NCAA Student-Athletes Who Have Sickle Cell Trait.

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Final Report

Perceptions of the NCAA Sickle Cell Trait Policy among Division I Student-Athletes

Shawn M. Bediako, Ph.D.
Department of Psychology
University of Maryland, Baltimore County

Abstract and Specific Aims

On April 13, 2010, the NCAA Division I Legislative Council decided that all incoming Division I student-athletes must be tested for sickle cell trait, show proof of a prior test, or sign a waiver releasing an institution from liability if they decline to be tested. However, very little is known about how student-athletes perceive the policy – or the extent to which their prior knowledge and attitudes about sickle cell trait influence how they choose to comply with it.

The specific aims of the study were to: (a) evaluate perceptions and attitudes toward the policy among NCAA Division I student-athletes; and (b) explore relations between student-athlete attitudes and the manner in which they decide to comply.

Summary of Results

We contacted a diverse range of personnel associated with both regional and national Division I athletic programs. These personnel included sports medicine directors, coaches, associate athletic directors, conference and national Student Athlete Advisory Committees, and Faculty Athletic Representatives. There was nominal interest and a *general lack of enthusiasm* for participating in the study from these personnel. Even after modifying the study protocol so that individual institutions were not identified (i.e., respondents would only indicate the conference and primary sport in which they competed), very few institutions responded to requests to inform their student-athletes about the study.

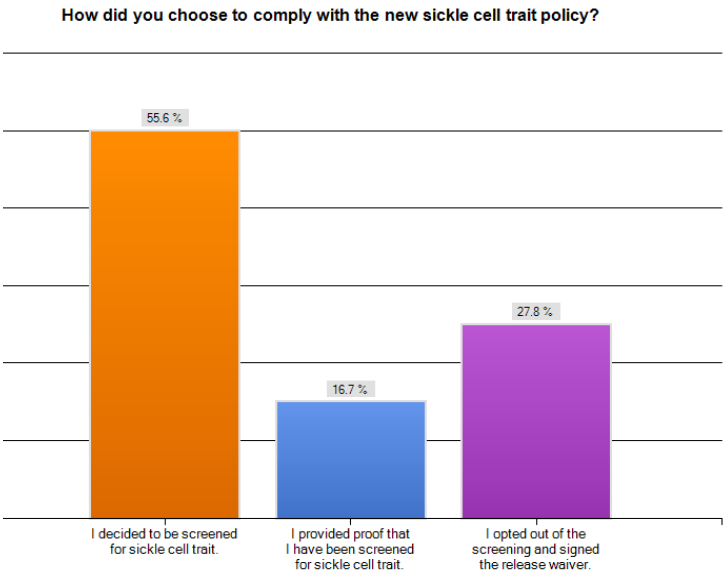
Of the 198 Division I programs that we contacted, only two agreed to distribute the secure web-based survey to their student-athletes. Thus, we summarize the results of our survey based on the responses of 81 student-athletes from the Atlantic Coast (n = 57) and America East (n = 24) conferences. Of these, 67 student-athletes completed all measures in the survey.

Females made up 53.7% of the sample. Approximately 19.5% of respondents failed to report their sex. Respondents were between the ages of 18 and 22, with an average age of 19.61 years. Of the 67 respondents who indicated their ethnicity, 63.4% were White, 11% were Black, and 3.7% were Asian. One respondent was Hispanic/Latino and 2 self-identified as American Indian/Alaska Native.

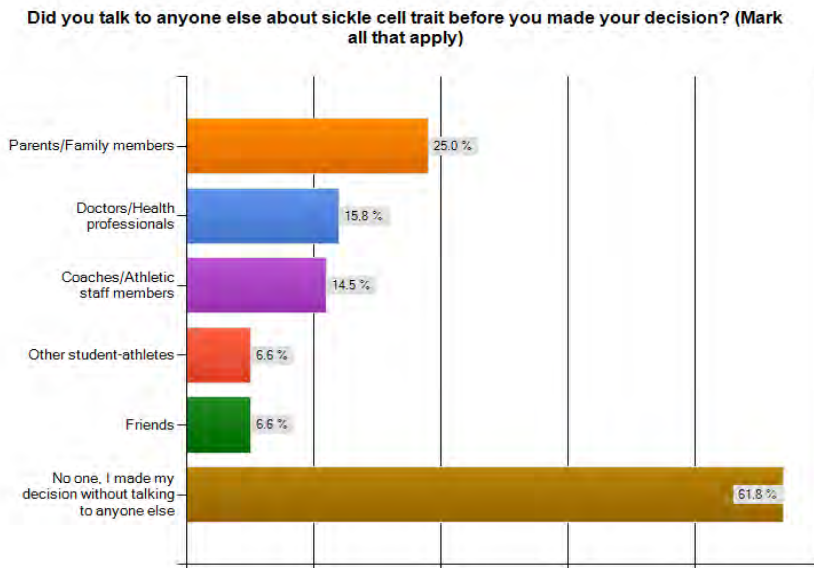
There was a significant underrepresentation of respondents who competed in major revenue-producing sports. None of the respondents indicated “men’s basketball” or “football” as their primary sport. Fencing, rowing, soccer, track and field, and volleyball were among the most commonly reported sports identified by respondents.

With respect to the primary study aims, 53.4% of respondents indicated that they *did not* know

their sickle cell trait status prior to August 1, 2010. As shown below, the majority of respondents (more than half) decided to be screened for sickle cell trait, while nearly one-third opted out of screening.



Despite the majority of respondents deciding to be screened for sickle cell trait, we observed that the decision was often made without consultation, education, or counseling. For example, as shown below, almost two-thirds of respondents *did not* talk to anyone about sickle cell trait prior to making their decision about complying with the policy.



Most importantly, 89% of respondents were unaware that the NCAA's website houses informational material about sickle cell trait. Furthermore, none of the respondents who decided to be screened indicated that they were offered an opportunity to have the results of their screening explained to them.

Collectively, respondents had rather poor knowledge of sickle cell trait: on average, they correctly answered only 4 out of 10 questions about trait incidence, etiology, and heritability. One of the most common misunderstandings about sickle cell trait was the idea that it would *lead to* sickle cell disease. Nearly three-quarters of respondents said that this statement was "true" when, in fact, it is completely false.

Sixty-seven participants completed the Sickle Cell Trait Stigma Scale, a 30-item measure comprised of four factors: *Personalized Stigma* (perceptions of negative social interactions with others if one were to have sickle cell trait); *Disclosure Concerns* (fear of repercussions if one's trait status was to be known); *Negative Self Image* (internalized stigma that results in low self-esteem); and *Concern with Public Attitudes* (perceptions of being generally discriminated against because of one's trait status). The table below shows the mean values and range of scores. Our analyses indicated that respondents tended to report moderately low levels of stigma about sickle cell trait, and that there appeared to be slightly greater concerns about disclosure relative to other factors.

Descriptive Statistics

	N	Minimum	Maximum	Mean
Personalized Stigma	67	.86	4.00	1.39
Disclosure Concerns	67	1.00	4.00	1.57
Negative Self Image	67	1.00	4.00	1.48
Concern with Public Attitudes	67	1.00	4.00	1.43

A multinomial logistic regression was utilized in which the method of compliance was the dependent variable and predictor variables were: (1) overall concern about the negative impact of sickle cell trait on the student-athlete's playing career; (2) sickle cell trait knowledge; (3) sickle cell trait stigma; and (4) gender. The results of the analysis indicate that none of the predictor variables were statistically significant. Our findings suggest that – compared to individuals who decided to be screened for sickle cell trait – neither gender nor negative concerns, knowledge, or stigma about sickle cell trait are associated with decisions to show evidence of prior screening or opt out of screening altogether (all p values > .95).

Recommendations for Further Study and/or Action

- Given the small sample size, these results should be interpreted with caution. A larger study should be conducted with a larger sample in order to increase the generalizability of these findings.
- The general lack of enthusiasm and participation from Division I programs reported here may be due to a general hesitancy on the part of athletic programs to collaborate with researchers who are not affiliated with their institution. The challenges to establishing collegial rapport may be reduced or eliminated if the NCAA were to conduct or explicitly endorse a similar project. If concerns exist about the appearance of coercion, then perhaps using the Student-Athlete Advisory Committee would help. The SAAC's stated

aims are to "*provide insight on the student-athlete experience*" and offer "*input on the rules, regulations and polices that affect student-athletes' lives*". A study on the impact of the sickle cell trait policy certainly falls within this purview and could easily be conducted on an anonymous and voluntary basis through the SAAC social networks and email list serves.

- While student-athlete *perceptions* were the primary focus of this study, the results also suggest that greater emphasis should be placed on *education*. There was a significant lack of knowledge among respondents in our study about basic aspects of sickle cell trait, and this knowledge gap may contribute to negative attitudes about the policy.
- Public health and bioethical concerns also need to be addressed. Most respondents did not consult with *anyone* prior to making their compliance decision, and our data also indicate that there was very little post-screening counseling about the meaning of the results. A basic tenet of public health education and bioethics is for individuals to be fully informed about risk. If the results from this project are indicative of the general population, it does not appear that this objective is being met. This is perhaps a paramount area of immediate intervention.

Publications/Presentations

Findings from this project will be prepared for publication to a peer-reviewed scientific journal according to the stipulations provided in the award letter dated March 22, 2011. In addition, it is anticipated that the findings will be presented in a poster or oral presentation format at the 6th Annual Sickle Cell Disease Research and Educational Symposium & Annual National SCD Scientific Meeting February 29-March 2, 2012 in Hollywood, Florida.

Research Materials/Data Sharing

All research material (e.g., surveys and de-identified data) will be made available upon written request to the investigator.

The investigator gratefully acknowledges and thanks the NCAA Committee on Competitive Safeguards and Medical Aspects of Sports for their support of this study. It is hoped that their commitment to student-athlete health and safety will be reflected in continued and expanded support of this very important topic.

Shawn M. Bediako, Ph.D.
Associate Professor
Director, Center for Transformative Community Health Psychology
Department of Psychology
University of Maryland, Baltimore County
1000 Hilltop Circle
Baltimore, MD 21250-0001

410-455-2349 (office)
410-455-1055 (fax)
bediako@umbc.edu

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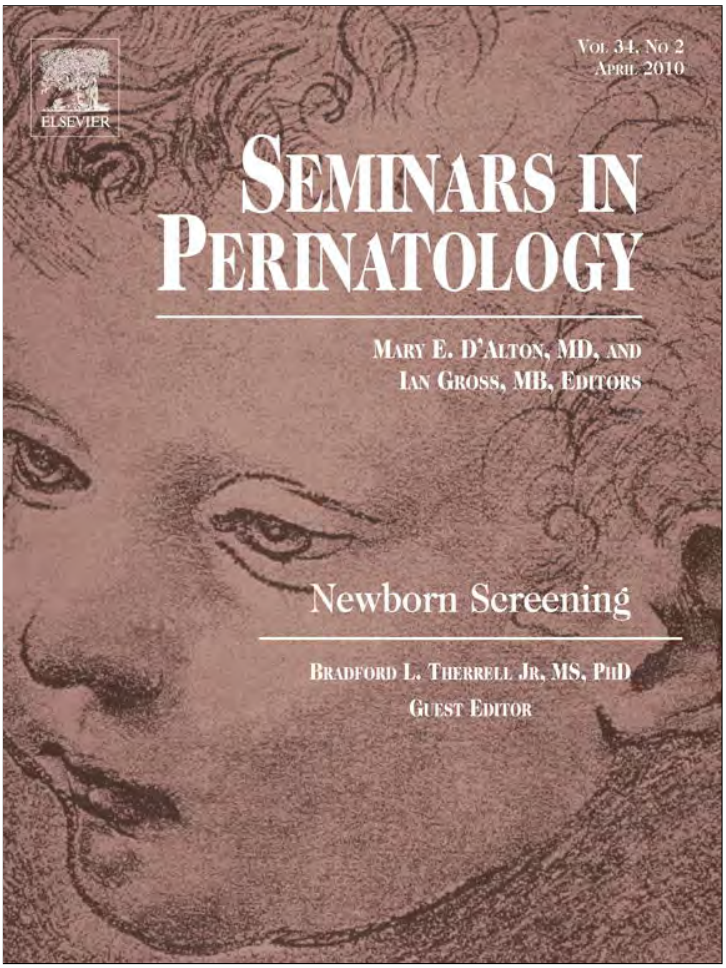

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SUPPLEMENT NO. 22

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SEMINARS IN PERINATOLOGY

History and Current Status of Newborn Screening for Hemoglobinopathies

Jane M. Benson, BA,* and Bradford L. Therrell Jr, MS, PhD†

The impact of hemoglobinopathies on healthcare in the United States, particularly sickle cell disease (SCD), has been significant. Enactment of the Sickle Cell Anemia Control Act in 1972 significantly increased the federal interest in the SCDs and other hemoglobinopathies. Only since May 1, 2006, have all states required and provided universal newborn screening for SCD despite a national recommendation to this effect in 1987. In this article, we review the history of screening for SCD and other hemoglobinopathies, along with federal and state activities that have contributed to improved health outcomes for patients with SCD, as well as current newborn screening practices. We also chronicle the federal activities that have helped to shape and to refine laboratory screening and diagnostic proficiency. Finally, we review molecular testing strategies that have evolved and outline their possible future impacts on disease detection and outcome improvement.
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KEYWORDS newborn screening, hemoglobinopathies, sickle cell disease, history

The term "hemoglobinopathies" refers to a group of genetic conditions that result in the production of abnormal hemoglobin(s) or in the lack of production of normal hemoglobins. The effect of hemoglobinopathies on healthcare in the United States over time, particularly sickle cell disease (SCD), can be measured by the availability of various federal and state public health programs and funding streams that have encouraged the study of these important conditions. As a partial result, universal newborn screening (NBS) for hemoglobinopathies is now required in all 50 states and the District of Columbia (hereafter referred to as 51 state programs). However, NBS expansion to include hemoglobin disorders did not progress quickly. Despite a National Institutes of Health (NIH) consensus statement recommending universal NBS in 1987, almost 20 years elapsed before all 51 state programs included universal (as opposed to targeted) hemoglobin screening as part of their required screening panels (Table 1).

In this report, we review the history of screening for SCD and other hemoglobinopathies from the perspective of federal and state activities that have contributed to NBS policies and program improvements. We also chronicle the federal activities, particularly those of the Centers for Disease Control and Prevention (CDC), the Health Resources and Services Administration (HRSA), and the National Heart, Lung and Blood Institute (NHLBI), that helped shape and refine laboratory screening and diagnostic proficiency. Finally, we review molecular testing strategies that have evolved and outline possible future effects of current laboratory testing procedures.

Nomenclature

Conventional hemoglobin nomenclature evolved from test results that described protein changes rather than molecular gene variations.[1] Descriptive names and shorthand abbreviations for various conditions loosely combined phenotypic and genetic information. For example, a sickle hemoglobin carrier (person with normal adult hemoglobin [Hb A] who is heterozygous for sickle hemoglobin [Hb S]) was denoted as Hb AS (with hemoglobin in greatest concentration listed first). Nomenclature has developed with advances in technol-

*Division of Blood Disorders, National Center on Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention, Atlanta, GA.

†Department of Pediatrics, University of Texas Health Science Center at San Antonio, National Newborn Screening and Genetics Resource Center, Austin, TX.

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The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention, the Health Resources and Services Administration, the US Department of Health and Human Services, or the University of Texas Health Science Center at San Antonio.

Address reprint requests to Bradford L. Therrell Jr, MS, PhD, National Newborn Screening and Genetics Resource Center, 1912 W. Anderson Lane 210, Austin, TX 78757. E-mail: therrell@uthscsa.edu

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Table 1 Date of Universal Screening for Sickle Hemoglobinopathies was Required and Available in US Newborn Screening Programs. (Dates validated through email and telephone survey of state programs conducted by the National Newborn Screening and Genetics Resource Center; completed October 20, 2009.)

State/ Territory	Universal Screening Required/Available	State/Territory	Universal Screening Required/Available	State/Territory	Universal Screening Required/Available
Alabama	Jan 1, 1987	Kentucky	Jan 1, 1995	North Dakota	Apr 1, 2003
Alaska	Oct 1, 2003	Louisiana	Jan 1, 1992	Ohio	Jul 1, 1989
Arizona	Jan 1, 1988	Maine	Jul 1, 2001	Oklahoma	May 1, 1991
Arkansas	Oct 1, 1988	Maryland	Jul 1, 1985	Oregon	Feb 1, 1995
California	Feb 7, 1990	Massachusetts	Mar 26, 1990	Pennsylvania	Sep 28, 1992
Colorado	Jan 1, 1979	Michigan	Jul 1, 1987	Rhode Island	May 1, 1990
Connecticut	Jan 1, 1990	Minnesota	Jan 1, 1988	South Carolina	Jul 1, 1987
Delaware	July 1, 1985	Mississippi	Jan 1, 1990	South Dakota	Jun 1, 2005
Dist Columbia	Jan 1, 1986	Missouri	Apr 1, 1989	Tennessee	Jan 1, 1988
Florida	Jan 1, 1989	Montana	Jul 1, 2003	Texas	Nov 1, 1983
Georgia	Oct 1, 1998	Nebraska	Nov 1, 1996	Utah	Sep 24, 2001
Hawaii	Jul 1, 1997	Nevada	July 1, 1990	Vermont	Feb 4, 1996
Idaho	May 19, 2004	New Hampshire	May 1, 2006	Virginia	Jul 1, 1989
Illinois	Feb 1, 1989	New Jersey	Apr 1, 1990	Washington	Nov 1, 1991
Indiana	Jul 1, 1985	New Mexico	Oct 10, 1995	West Virginia	Jul 1, 2003
Iowa	Feb 5, 1988	New York	Apr 1, 1975	Wisconsin	Oct 31, 1988
Kansas	Jul 1, 1993	North Carolina	May 2, 1994	Wyoming	Jan 1, 1987

ogy to convey specific molecular information in a standardized format recommended by the Human Genome Variation Society.[2] Using Human Genome Variation Society nomenclature, the predominant variant in the β-globin gene (HBB) that causes sickling is written as HBB NM_000518.4:c.20A > T (p.Glu7Val), rs334, for the primary transcript. For simplification, we will refer to the SNP described by rs334 as Glu6Val, as described historically, and we will use conventional nomenclature as follows: the descriptions of hemoglobin conditions will be referenced by letters or names with the hemoglobin of highest concentration listed first. A hemoglobin structural variant may also be shown parenthetically for clarification (ie, Hb S [β6 Glu $\rightarrow$ Val]). As examples, Hb AS denotes a carrier for the sickle variant (sometimes referred to as sickle cell trait), whereas Hb SA denotes a compound heterozygote for Hb S and β^+ thalassemia.

The term "SCD" will refer to the group of inherited blood disorders characterized by the presence of Hb S, chronic anemia, painful events, and associated complications. To avoid confusion between the terms sickle cell "anemia" and sickle cell "disease," the homozygous presence of Hb S, commonly known as sickle cell anemia, will be denoted as "sickle cell disease-SS" (SCD-SS). SCD-SS is one member of the SCD family, which also includes "sickle cell disease-SC" (SCD-SC), "sickle cell disease—Sβ^0 thalassemia" (SCD-Sβ°thal), and "sickle cell disease—Sβ^+ thalassemia" (SCD-Sβ^+thal).

The term "hemoglobinopathy" will refer to both structural variants of oxygen-carrying hemoglobin and to α- and β-thalassemias. Thalassemia defines a form of anemia caused by reduced synthesis of one of the normal globin chains, which results in an abnormal hemoglobin molecule. With structural variants, such as Hb S, the hemoglobin molecule does not function normally (qualitative), whereas with thalassemia the unbalanced globin synthesis is quantitative. That is, unbalanced globin chains can skew the hemoglobin molecule away from its normal composition, which can result in fewer normal hemoglobin molecules to carry oxygen and an aggregation of abnormal tetramers determined by the globin chain of highest concentration, such as Hb H(γ4 tetramer) or Hb Bart's (β4 tetramer). As a result, oxygen carrying efficiency may be decreased.

Background Information

Basic Biochemistry

Hemoglobin is a protein tetramer that consists of 4 heme groups and 2 pairs of polypeptide chains translated from 2 globin genes. In adults, the greatest concentration of the hemoglobin molecule (Hb AA) consists of 2 α chains (HBA gene) and 2 β chains (HBB gene), with one of each of the chains having been inherited from each parent. The globin genes are located in clusters on 2 chromosomes. HBB is in the β-globin cluster region on chromosome 11, and HBA1 and HBA2 are in the α-globin cluster region on chromosome 16. Other globin genes are expressed at various times during embryonic and fetal development.

Hemoglobinopathies are inherited disorders and account for 3 of the core conditions included in the national recommendations for NBS in each state—SCD-SS, SCD-SC, and SCD-Sβ°thal/SCD-Sβ^+thal.[3] The remaining clinically significant hemoglobin variants are included as secondary NBS targets (may be detected as a consequence of screening for the core conditions). Structural hemoglobin variants frequently occur because of a change in the molecular sequence of at least one of the globin genes, usually a single nucleotide

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polymorphism (SNP). Thalassemias, demonstrating unbalanced synthesis of the globin chains, are frequently caused by the deletion of a portion or all of one of the α- or β-globin genes, as well as by a SNP in a regulatory region.

Erythrocyte sickling may ensue when low oxygen tension occurs in the presence of an abnormal hemoglobin containing the β6 Glu $\rightarrow$ Val substitution, HBB NM_000518.4: c.20A > T. Other hemoglobinopathies which cause sickling contain a second, "double" mutation, along with the above mutation for Hb S. Sickling is more pronounced with the homozygous state (Hb SS) or when Hb S is present in combination with certain other hemoglobin variants. Sickling hemoglobin molecules tend to stick together and form long chains or polymers as oxygen is released. These rigid polymers cause distortion of the cells resulting in irregular or crescent (sickle) shapes. Anemia characteristic of this disease is a consequence of more rapid deterioration of sickle-shaped cells.

The clinical manifestations and severity of SCDs vary. Genetic modifiers that may alter the clinical severity of SCD include (1) α-thalassemia, (2) the ability to continue producing fetal haemoglobin,[4,5] (3) coinheritance of Hb S with other variants containing the same mutation (β6 Glu $\rightarrow$ Val), and (4) coinheritance of β-chain abnormalities, such as Hb E or β-thalassemia. The coinheritance of α-thalassemia is thought to ameliorate severity because it decreases the concentration of Hb S in the cell which reduces polymerization and hemolysis.[6] The red cells are more deformable, with less irreversible sickling.[7] As an additional modifier, the presence of Hb F not only dilutes the concentration of Hb S, but also directly inhibits Hb S polymerization.[8] However, other conditions predict a more severe clinical outcome than the inheritance of the trait condition for Hb S. When coinherited with 1 allele for Hb S, other sickling variants, such as Hb C-Harlem or Hb S-Travis$_s$, may increase clinical symptoms, effectively presenting a homozygous genotype for the sickling mutation.[9-13] Hb E, one of the world's most common hemoglobinopathies, is caused by the substitution of Glu $\rightarrow$ Lys at β26, HBB NM_000518.4:c.79G > A. Significantly, this mutation may lead to a thalassemia phenotype because the mutation activates a cryptic mRNA splice site, resulting in reduced and unbalanced globin synthesis with some instability.[14] β-thalassemia, depending on the causative mutation, presents with varying degrees of decreased synthesis of β-globin. In more severe cases of β-thalassemia, an accumulation of free α chains can precipitate and form inclusions in the erythrocyte precursors, resulting in ineffective erythropoesis and anemia.[15] When β-thalassemia is coinherited with Hb S, the result is an intraerythrocytic increase in the concentration of HB S with more Hb S polymer fractions.[16] The clinical presentation of SCD-Sβ°thal or SCD-Sβ^{+}thal is similar to that of SCD-SS, whereas the presentation of Hb AS has few clinical manifestations except under extreme conditions. Research continues in an attempt to better explain the spectrum of clinical manifestations of SCD and to predict severity of the diseases for earlier treatment.

Early History of Laboratory Detection of Hemoglobinopathies

Historically, laboratory tests for hemoglobinopathies have been based on 3 major observations: (1) the appearance of sickled erythrocytes on a blood smear, (2) solubility of Hb S under low oxygen tension, and (3) electrophoretic profiles. In 1910, Herrick published the first case report of SCD-SS in the United States. He described the presence of sickle-shaped red blood cells with a hypochromic, microcytic appearance.[17] It was almost 40 years before a microscopic diagnostic test for sickling hemoglobin using dithionite to produce sickling was described by Itano and Pauling in 1949.[18] They also reported the first electrophoretic separation of Hb S in the same year.[19] The dithionite sickling test was further refined by Nalbandian et al in 1970 as a more rapid and automatable solubility test for sickling hemoglobins. With this increased capability for high analytical throughput, mass screening was suggested.[20-22] By 1974, 7 commercial kits for solubility testing of sickling hemoglobins were available.[23] Today, this test alone is limited in its usefulness and is not considered sufficient either for screening newborns or for genetic counseling of SCD risk in pregnancy outcomes, with complete hemoglobin analysis of both parents recommended before genetic counseling of the latter.

Electrophoresis and chromatography were also evolving as accepted procedures for detecting hemoglobinopathies. In 1955, Smithies[24] described a protocol for zone electrophoresis in starch gels, and in 1969 Kohn[25] used cellulose acetate electrophoresis to separate hemoglobins with a run time of 25 minutes and good resolution. Several commercial electrophoresis kits for hemoglobin identification emerged, and adaptations, including secondary testing with citrate agar electrophoresis, allowed further discrimination of the hemoglobinopathies.[26] The gel filtration chromatography protocol of Clegg et al[27] in 1966 became a classic reference procedure for measuring synthesis ratios of globin genes and for diagnosing thalassemia. Isoelectric focusing (IEF) was adapted from the original tube gel method of Righetti and evolved as a single electrophoretic test with definitive results and high throughput, replacing the dual cellulose acetate/citrate agar procedure.[28,29]

Cord blood screening for Hb SS was first reported in 1972 when researchers in the United Kingdom concluded that screening at birth could be accomplished by using a combination of cellulose acetate or starch gel electrophoresis (basic pH) in combination with agar gel electrophoresis (acid pH). However, screening at 3 months was recommended because fetal hemoglobin interferences from birth were sufficiently decreased by that time to allow reliable diagnosis. Compliance in the London program, however, was only about 45%.[30] A milestone in NBS hemoglobinopathy laboratory testing occurred in 1973 when Garrick, working with Guthrie (the originator of NBS for inborn errors of metabolism), determined that eluates from dried blood spots on filter paper could be used for cellulose acetate and citrate agar electrophoresis.[31] The primary analytical challenge for NBS was the oxidative degradation that occurred within days in dried

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blood spots which resulting extra bands and smearing on analysis, complicating result interpretation. When automated punching equipment for dried blood spots became available later in the decade, NBS programs began to consider additional screening tests, including SCD. New York was the first state to require universal screening for SCD-SS[32,33] and testing was first available in April 1975.[34] As IEF techniques improved, interpretation problems diminished and NBS programs slowly embraced the technology.[35] Capillary electrophoresis has afforded higher throughput with the IEF procedure.[36] High-performance liquid chromatography (HPLC) was also refined and marketed to NBS programs as a means of facilitating qualitative and/or quantitative identification and electronic data management of hemoglobin laboratory testing.[37,38] Today, most programs use IEF because of its relatively low cost, but there is an increasing trend towards HPLC testing as NBS programs continue to seek more quantitative results and automated result.

History of Sickle Cell Disease and Newborn Screening

In 1929, Diggs began to extensively define the natural history of SCD-SS. However, it was not until 1971 that he opened the first US comprehensive sickle cell clinic in Memphis, TN.[39] Meanwhile, in 1949, Pauling documented SCD-SS as a molecular disease,[19] and Neel[40] and Beet[41] reported that the gene was recessively inherited. In 1957, Ingram[42] described the substitution of glutamic acid to valine at position 6 in the β-globin chain as the cause for Hb S. About the same time, Guthrie was beginning his work with bacterial inhibition assays for phenylalanine from blood collected on filter paper. He hoped to be able to screen newborns for phenylketonuria, as early identification and treatment was crucial to improved disease outcome. In 1963, he reported a dried blood spot assay to detect phenylketonuria shortly after birth, which ultimately led to successful implementation of nationwide NBS.[43] In addition to developing additional assays for other inborn errors of metabolism, Guthrie's laboratory studied other possibilities for optimizing NBS, which ultimately led to Garrick's work with hemoglobinopathies as described previously.

In the interim, Perutz[44] received the Nobel Prize for Physiology and Chemistry in 1967 with his description of a 3-dimensional structure for the hemoglobin molecule. Interest was growing to screen high-risk populations for Hb S. In 1970, Scott[45,46] described the disparities in federal funding for SCD, which further increased the interest in Hb S screening. He, along with other impassioned physicians and academicians, struggled to focus federal attention on SCD-SS, emphasizing the need for funds for testing, research, and education.[47] When the National SCD-SS Control Act (Public Law 92-294) was signed into law on May 16, 1972, SCD-SS and ultimately other hemoglobinopathies were publicly and politically acknowledged as significant public health issues. This was the first federal program targeting a genetic disease.[48]

The National Sickle Cell Disease Program was established in the then Department of Health, Education, and Welfare under the NHLBI in 1972. The Department of Health, Education, and Welfare funded 41 sickle cell centers and clinics, over 250 general screening programs, 69 research grants and contracts with numerous locally supported screening, education, and counseling clinics.[49] Also, in 1972, the Hemoglobinopathy Reference Laboratory was created at the CDC (with funding from the NIH) to serve as a national reference laboratory for the 34 state health departments testing for SCD and for the funded sickle cell centers and clinics. Additional foci included standardizing hemoglobinopathy testing protocols and improving laboratory testing quality through external proficiency testing and continuing education. CDC reference services soon expanded to include all hemoglobinopathies, including thalassemias and unstable hemoglobins. After NIH funding ended in 1990, CDC continued to support this program until 1993.

The Hemoglobinopathy Reference Laboratory at the CDC quickly became a national center for protocol standardization and reference testing. Standardized analytical protocols were developed for protein-based testing, including cellulose acetate and citrate agar electrophoresis, solubility testing, and quantitation of Hbs F and A_2. Two procedural manuals were developed for basic and advanced methods of hemoglobinopathy detection to standardize protocols for the numerous laboratories that were emerging to test for hemoglobinopathies.[50,51] These manuals formed the basis for at least 2 annual training courses at the CDC which included lectures on hemoglobinopathies, and a laboratory training to instill a better understanding of standardized protocols and interpretation of the test results. Completion of these training courses was required for personnel in federally funded clinics and centers. As a result, many center directors who were experts in hemoglobinopathy detection attended, and their experiences added to the discussions and improved course content. These training courses were also popular with state health department personnel, who were just beginning hemoglobinopathy detection projects as part of expanded NBS. With support from commercial vendors, additional CDC courses were often held off campus.

The Hematology Section at the CDC organized the first hemoglobinopathy proficiency testing program in December 1972. This program was intended to improve analytical laboratory services through periodic performance evaluation of unknown specimens and to provide a basis for an ongoing education and quality control program. Anticoagulated whole blood specimens were distributed quarterly. The initial program in January 1973 included 23 invited reference and 116 volunteer laboratories, which grew to 25 reference and 204 volunteer laboratories by the end of the year. The details of the first year's activities are reported elsewhere, and note that one-third of reporting laboratories misidentified a sample containing Hb AS on the first survey.[52] This program continued to expand until 1993 when the Hemoglobinopathy Reference Laboratory was discontinued and the proficiency testing program was transitioned to the Newborn Screening Quality Assurance Laboratory at

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Table 2 Errors in Domestic Laboratory Reporting of Results for Dried Blood Spot Hemoglobinopathy Proficiency Testing Specimens, Centers for Disease Control and Prevention 2000-2008. (Provided by J. Mei, Newborn Screening Quality Assurance Program, CDC, October 9, 2009)

Hemoglobin Variant(s) Assayed	2000 Specimens Assayed	2000 Phenotype Errors	2000 Clinical Assessment Errors	2001 Specimens Assayed	2001 Phenotype Errors	2001 Clinical Assessment Errors	2002 Specimens Assayed	2002 Phenotype Errors	2002 Clinical Assessment Errors
FA	200	1	1	276	2	2	306	0	0
FAC	250	1	1	230	0	0	255	1	1
FAD	50	0	0	0			0		
FAE	0			0			51		
FAG	0			0			0		
FAS	300	0	1	322	2	2	357	0	0
FE	0			0			0		
FS	100	0	0	46	0	0	51	0	0
FSC	100	3	0	46	0	0	0		
Total	1000	5	3	920	4	4	1020	1	1
	2003			**2004**			**2005**		
FA	260	1	1	270	0	0	385	1	1
FAC	208	0	0	216	0	0	220	1	2
FAD	52	1	1	0			0		
FAE	52	0	0	0			0		
FAG	0			54	7	6	0		
FAS	260	0	0	270	0	0	220	2	2
FE	0			54	0	0	0		
FS	52	0	1	162	4	3	165	1	1
FSC	0			54	0	1	110	1	1
Total	1040	2	3	1080	11	10	1020	6	7
	2006			**2007**			**2008**		
FA	260	0	0	204	0	0	147	0	0
FAC	260	1	1	204	3	1	196	0	0
FAD	50	0	0	0			0		
FAE	0			0			51		
FAG	0			0			0		
FAS	312	1	0	306	0	0	294	0	0
FE	0			0			0		
FS	156	0	0	204	0	0	98	0	0
FSC	56	3	0	102	0	0	0		
Total	1040	2	1	1020	3	1	735	0	0

Explanatory notes:

A phenotype error is a failure to detect the presence of abnormal hemoglobin in a specimen or finding abnormal hemoglobin in a specimen known to have a normal (FA) phenotype.

A clinical assessment error is a failure to report of the correct presumptive clinical classification, e.g. reporting a specimen as "normal – no abnormal hemoglobin found" when the classification should have been reported as "Hemoglobin S carrier."

Because the source of umbilical cord blood used to prepare PT specimens changed, NSQAP reduced the number of panels from four to three times a year to conserve materials.

the CDC.[53] Beginning in 1994, the NSQAP hemoglobinopathy proficiency testing program provided quarterly samples to NBS Laboratories, but in 2008 the number decreased to 3 shipments annually due to a change in the source of fetal cord blood from which proficiency testing materials are made. Table 2 gives a summary of proficiency testing results (from 2000-2008) as confirmation of the reliability of current laboratory testing interpretations.

As commercial tests were marketed, the CDC laboratory evaluated their accuracy and precision, along with that of other "home brew" procedures.[54-63] Acid and alkaline globin chain electrophoresis reference services were made available.[26] Chromatography, electrophoresis, and precipitation tests were used for additional discrimination of globin variants.[64-66] α-Thalassemias were identified using neutral pH electrophoresis on cord blood or newborn blood specimens to detect Hb H(γ_4 tetramer) or Hb Bart's (β_4 tetramer), both indicating a deficiency of α-globin chain synthesis, or definitively on adult specimens with α- to β-synthesis ratios (which are unbalanced with the thalassemias) from chromatographic separation of globin chains which had been synthesized with tritiated leucine.[27] Reference tests for unstable hemoglobins included isopropanol precipitation, heat denaturation, and brilliant cresyl blue staining for inclusion bodies.[67-70] IEF was evaluated for the diagnosis of 134 abnormal hemoglobins from the National Hemoglobinopathy Standardization Laboratory in 1975.[71] In that evaluation there were no discrepancies and the authors proposed that the protocol would be a powerful addition to other methods.

For identification of more challenging variants, the CDC Hemoglobin Biochemistry Laboratory performed additional testing. These techniques included sequencing the hemoglobin protein to determine the exact structural variation in the globin polypeptide. The elucidation of the structure and/or

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function relationships resulting from these perturbations of the hemoglobin molecule was crucial to understanding the role of the protein, particularly changes in O_2 dissociation.[72-80] Hemoglobin reference activities at the CDC resulted in over 100 published articles from 1972 through 1993, including identification and characterization of more than 45 previously unreported hemoglobins. The eighth and final printing of the basic laboratory reference manual occurred in 1984.[81] The CDC reference laboratory also prepared and distributed lyophilized hemoglobin electrophoresis standard materials to help refine laboratory testing protocols across the country. These reference standards included major abnormal hemoglobins in combination with Hb A (Hbs J, F,S, and C), which were purified, stabilized, and combined to reflect amounts approximate to those which might be observed clinically.[82]

In 1977, the NHLBI-sponsored multicenter Cooperative Study of Sickle Cell Disease (CSSCD) began.[83] This study aimed to determine the natural history of SCD with investigation by: (1) studying the effect of SCD on growth and development from birth through adolescence; (2) studying conditions or events related to the onset of painful crises; (3) obtaining data on the nature, duration, and outcome of major SCD complications; (4) determining the nature, prevalence, and age-related incidence of organ damage because of SCD; and (5) studying the role of SCD and its interaction with selected health events. Study participants were enrolled from 1978 through 1988, and data collection for the study ended in 1998.

The success of prophylaxis using penicillin in children with SCD has become one of the epic studies from the CSSCD. Among patients enrolled in the CSSCD, the annual incidence of pneumococcal septicemia in SCD-SS was found to be 7-8 per 100 person years in this population, with a at least 20% case fatality rate in childhood.[84] In 1983, the NHLBI initiated a Prophylactic Penicillin Study to assess the efficacy of oral penicillin in preventing severe bacterial infections in children with SCD-SS. This study showed such a dramatic decrease of *S. pneumoniae* bacteremia with daily administration of penicillin (84% risk reduction) in the treated vs nontreated patients that the trial was terminated 8 months before its planned ending date.[84] On the basis of the findings and implications of the Prophylactic Penicillin Study, the NHLBI recommended that: (1) newborns in high risk populations should be screened for SCD-SS; and (2) newborns diagnosed with SCD-SS should be placed on oral prophylactic penicillin by 4 months of age.[85]

In 1978, the National Genetic Disease Act was passed and provided additional funds for SCD. The NHLBI, responsible for implementing the Act, transferred funds to HRSA to develop community-based SCD education, screening, and counseling services. The NIH funded SCD research projects and established comprehensive research centers. The result was 23 comprehensive care centers (reduced to 10 study centers by 1986) and screening and education centers in 40 states and the District of Columbia by 1981. Despite the successes of the various federal efforts in comprehensive care and education, there was not widespread acceptance of NBS as an approach to early detection and treatment of SCD.[86]

Subsequently, in 1987, the NIH convened a consensus development conference (cosponsored by HRSA) on "Newborn Screening for Sickle Cell Disease and Other Hemoglobinopathies." Participants addressed: (1) effectiveness of NBS programs for SCD-SS; (2) screening techniques and their efficacies; (3) risk and/or benefit considerations in NBS programs; (4) optimal follow-up and management strategies for identified cases; and (5) directions for research. The consensus jury concluded that, "every child should be screened for hemoglobinopathies to prevent the potentially fatal complications of SCD during infancy." As part of their recommendations, the panel noted that, "The benefits of screening are so compelling that universal screening should be provided. State law should mandate the availability of these services while permitting parental refusal." Further they noted that, "Centralization of laboratory services improves efficiency and decreases the probability of error."[85]

Building on the NIH Consensus Conference recommendations and recognizing the benefits of universal NBS for SCD-SS, HRSA made supplemental funding available through grants to states for NBS expansion to include SCD. In fiscal year (FY) 1988, $1.3 million was available, which increased to $3.88 million in 1989 and $6.86 million in 1990. In the first cycle (1988), 5 states received 3 year grant funding. In the second cycle, 12 states were given 2-year grants. Six other states received federal funding from other sources so that in 1989, 23 states were receiving federal assistance for NBS expansion to include SCD. At the end of the decade, there were 16 states with universal NBS for SCD and 14 others either screening "high risk" populations or developing screening programs.[87] Over the next 7 years, there was increasing focus on the few states not requiring universal NBS for SCD, with a final push from the American Academy of Pediatrics, who provided expert consultants to transmit educational information to policy makers. The last state to adopt universal NBS for SCD did so in 2006 (see Table 1).

Other significant reports emanating from the CSSCD included the correlation of pain rate with early death among individuals with SCD over the age of 20, as well as risk factor analysis of silent cerebral infarcts in children with SCD.[88,89] With the Sickle Cell Disease Act of 2004 and appropriation of funds in 2005, the groundwork has been set in place to develop specialized sickle cell treatment centers with a National Coordinating Center for Sickle Cell Disease. Furthermore, each state can receive federal funding for patient counseling, educational programs, and community outreach projects. Through Medicaid, patients can receive matching funds for services related to SCD (http://www.sicklecelldisease.org/news/2004/treatment_act.phtml) (http://waysandmeans.house.gov/media/pdf/hr4520/hr4520confreptlegtext.pdf). Once again, attention has been given to the crucial need to provide quality healthcare to all SCD patients.[90] In 2008, the NHLBI Advisory Council recommended an initiative with CDC and other federal partners to develop and implement a Registry and Surveillance System in Hemoglobinopathies (RuSH) and a biospecimen bank. The goal

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is to collect and maintain data to describe characteristics of all hemoglobinopathies. This project is now in its formative phase.

Molecular Testing for Hemoglobinopathies

Molecular protocols for hemoglobinopathy detection began to rapidly evolve in the 1970s. In 1976, a combination of Southern blotting and restriction fragment length polymorphism (RFLP) analysis was used for prenatal SCD-SS diagnosis,[91] and in 1978, SCD-SS antenatal diagnosis was performed by similar DNA analysis of amniotic cells.[92] Using a similar protocol, Kan and Dozy[93] reported a marker outside of the β-globin gene in a HpaI site associated with sickle cell and people of African descent. Direct analysis of the Hb S mutation using the DdeI restriction enzyme for antenatal diagnosis was reported in 1981.[94] The oligomer restriction technique, as a precursor to the polymerase chain reaction (PCR), was used for enzymatic β-globin amplification and restriction site analysis for SCD-SS in 1985.[95]

Analytical procedures changed dramatically a year later when the PCR protocol was published requiring less DNA for analysis and providing analytical throughput.[96] Direct genomic sequencing of amplified single copy DNA for β-globin was reported in 1987.[97] That same year, McCabe reported the microextraction of DNA from dried blood spots and the potential application of DNA as a second-tier NBS test for SCD.[98] Primer-directed PCR for hemoglobinopathies was reported the following year, as was the diagnosis of SCD-SS and SCD-Sβ-thal with PCR using nonradioactive probes.[99,100] Step-wise advancements in molecular applications of hemoglobinopathy detection using DBSs were reported throughout the published data during this same time.[101-106] Comprehensive reviews of the various molecular techniques for the diagnosis of hemoglobinopathies through the mid-1990s, including NBS applications, have been reported elsewhere.[107,108] Currently, a limited number of NBS screening and reference laboratories use DNA testing as a second-tier mechanism for clarifying screening results and speeding the time to diagnosis and treatment.[38]

Future Directions

The human genome project has led to development of numerous accurate and precise protocols for high throughput testing of SNPs. Although mutations in many other genes have been studied using TaqMan® assays (Applied Biosystems, Inc., Foster City, CA), multiplex Luminex XMAP® and XTAG® protocols (Luminex Corporation, Austin, TX), various PCR-based protocols, and sequencing using capillary electrophoresis, few of these assays are available from affordable commercial sources for mass hemoglobinopathy testing.[109] Technical results of 2 high throughput Luminex bead-based multiplex assays for 23 cystic fibrosis mutations and 4 polymorphisms without discrepancies suggest much potential with this molecular technology.[110] Luminex technology has also been used for genotyping 4 β- and 6 α-globin chain variants in a single assay,[111] and a custom-designed Luminex assay identified Hbs S, C, and E, with no discrepancies when results were compared with sequencing results on the same samples.[112] Marligen Biosciences (Rockville, MD) currently markets an assay based on the Luminex platform, Signet Hemoglobinopathies panel 1, for interrogating for the Hb variants of S, C, and E. Within the β-globin cluster region, over 200 TaqMan assays are available, but few assays have been designed to detect clinically relevant variants.

α-Globin gene deletions influence the natural history of SCD, but testing has not been done in a public setting or in a general clinical practice because the technology has not been readily available. A commercial assay for Multiplex Ligation-Dependent Probe amplification (MLPA—available from MRC, Holland; Amsterdam, Netherlands) has been marketed for detection of deletions and/or duplications in the α- and β-globin genes. This technique is currently being used at the Mayo Clinic as a Luminex assay in a partnership between Mayo Clinic and Luminex Corporation (http://www.clpmag.com/clprime/2007-12-05_03.asp). This protocol uses copy number variation to detect thalassemia mutations resulting from deletions. Copy number variation assays can also be custom designed for the TaqMan (Applied Biosystems) Real Time PCR protocol. When these assays become available for the Open Array Genotyping System (Applied Biosystems), duplication and deletion detection should be rapid and accurate. Additionally, LightCycler® (Roche, Indianapolis, IN) technology has been used to detect 9 of the common mutations for β thalassemia along with the SNP for Hb S using a rapid screening protocol with 3 probe sets.[113] A summary of 4 of these unique, but complimentary applications which might be performed in a reference laboratory for additional discernment of modifier genes is presented in Table 3.

Although protein-based techniques work well in expert hands, the throughput is not as rapid as with some of the more comprehensive molecular assays. With stable DNA available from multiple sources using noninvasive protocols, reliable molecular techniques may become more desirable in

Table 3 Applications of Molecular Techniques for Comprehensive Characterization

Technique	Luminex	MLPA	Resequencing	TaqMan® CNV
SNP detection	Y	—	Y	—
Small insertion/deletions	?	—	Y	Y
Duplications	—	Y	—	Y
Sequence variation	Y	—	Y	—
Large deletions (Thalassemia)	—	Y	—/(?)	Y

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a clinical setting or in a central reference laboratory.[114] Fewer data entry mistakes occur when the results can be transferred automatically into a database for reporting. Most of the high throughput molecular protocols offer this capability. However, widespread, standardized commercial application of these protocols to NBS for hemoglobinopathies has not occurred.

Significant advances continue in disease management of patients with SCD. Research on genetic modifiers of SCD[5,115,116] is promising and haplotype studies in the β-globin cluster region may provide a method to predict the severity of SCD-SS.[117] SNPs in several genes, including the TGF-β/BMP super family, the NOS genes, and other genes affecting endothelial function, are being studied to explain some of the phenotypic variability with SCD.[118] Genome-wide association studies (GWAS) have been providing information on the multigenic components, especially SNPs, of vascular pathways (which seem to be risk factors for the severity of the symptoms associated with some hemoglobinopathies).[119]

The major protocols currently used in NBS for hemoglobinopathies are IEF and HPLC, but developing technologies may lead to improved screening systems in the future. Currently the only public health screening laboratories performing molecular testing as a second-tier newborn hemoglobinopathy screen are in New York, Texas and Washington State, and California utilizes a contractor to provide some molecular testing as a routine part of their screening protocol. The current screening model in California, with a diagnostic confirmatory step before in the screening process, may expand as states seek NBS program improvements. Already, NBS for α-thalassemia has been proposed to the Advisory Committee on Heritable Disorders in Newborns and Children for consideration as a core condition for inclusion in state NBS panels, and technology advancements in hemoglobin disease detection and treatment may lead to other considerations over time. It is important, therefore, that state NBS programs be aware of research developments and modifiers which affect the clinical severity of the hemoglobinopathy.[120]

Accurate diagnoses are crucial for proper health care management, with careful attention to the painful episodes, infections, and anemia observed in SCD and the anemias associated with some thalassemias. Prophylactic penicillin for SCD-SS patients has been demonstrated to reduce mortality in infants and young children.[84,121] Transfusions may be necessary in cases of severe thalassemia with careful monitoring and chelation treatment for iron overload.[122,123] Preliminary evidence suggests that the carrier condition of Hb AS has been associated with an increased risk for venous thromboembolism, and, likewise, the use of oral contraceptives among women who are carriers for Hb S may increase the risk for venous thromboembolism.[112,124] Because some hemoglobinopathies interfere with the reliable measurement of hemoglobin A_{1c} values, accurate diagnosis of a hemoglobinopathy is imperative to achieve reliable glycemic control for a patient with diabetes.[125-127] The high prevalence of osteopenia and osteoporosis among young adults with SCD presents the possibility of fracture risk.[128] The effects of a hemoglobinopathy can be pervasive, even in the carrier state as with Hb AS.[129,130]

Globally, approximately 7% of the world's population may carry a clinically significant hemoglobinopathy.[131] Since, in countries with NBS programs for SCD and other hemoglobinopathies, the prognosis and management of patients with SCD-SS and SCD-β thalassemia have improved, the need to develop programs for management of hemoglobin disorders in all countries requires increased attention and elevation in priority. Development of commercially available multiplex molecular assays to test for the hemoglobinopathies would ensure accurate diagnosis. Future goals of developed hemoglobinopathy NBS programs should include improved short- and long-term case surveillance, increased attention to carrier detection and education, adherence to standardized nomenclature for reporting variations, and better and broader education about disease genetics. Increased attention should be paid to translating research into clinically relevant applications for improved health outcomes. Coordinated, effective state NBS programs provide a continuing opportunity to provide services that result in improved health and well being for SCD patients and carriers alike.

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EDITORIAL

Sickle Cell Trait and Sudden Death—Bringing it Home

Bruce L. Mitchell, MD
Jacksonville, Florida

Sickle cell trait continues to be the leading cause of sudden death for young African Americans in military basic training and civilian organized sports. The syndrome may have caused the death of up to 10 college football players since 1974 and, as recently as 2000, was suspected as the cause of death of three U.S. Army recruits. The penal military-style boot camps in the United States and the recent death of two teenagers with sickle cell trait merits renewed vigor in the education of athletic instructors, the military and the public about conditions associated with sudden death in individuals with sickle cell trait.

Key words: exercise ■ hypoxia ■ sickle cell trait ■ sudden death

© 2007. From the Division of Community Internal Medicine, Mayo Clinic Jacksonville, FL. Send correspondence and reprint requests for *J Natl Med Assoc.* 2007;99:300–305 to: Dr. Bruce L. Mitchell, Division of Community Internal Medicine, Mayo Clinic, 4500 San Pablo Road, Jacksonville, FL 32224; e-mail: mitchell.bruce@mayo.edu

A great deal of controversy continues to surround sickle cell trait and its association with exercise-related morbidity and sudden death. Sickle cell trait has a high prevalence among African Americans, and although most individuals have minimal or no clinical sequelae, debate centers on the effects of extreme physical exertion, dehydration and relative hypoxia (typically at high altitudes). The large number of African Americans who are enlisted in the U.S. military or who participate in endurance athletics are particularly at risk, and their experiences support the characterization of this condition as a public health problem. This paper describes risks and the complex interaction of physiologic and sociologic factors involved in downplaying the clinical significance of sickle cell trait. Screening and clinical recommendations are made that will identify and improve the care of individuals with sickle cell trait.

Sickle Syndromes

Sickle syndromes include several distinct diseases that cause red blood cells to sickle in vivo. The most recognized are sickle cell anemia, sickle cell trait, hemoglobin sickle cell disease and sickle cell-ß-thalassemia. In the United States, of all the hemoglobinopathies, individuals with homozygous sickle genes (Hgb SS) have the greatest morbidity and mortality, and the disease limits their ability to participate in athletic activities. Sickle cell trait is the heterozygous condition (Hgb AS) and has a prevalence rate of 8% in African Americans and 0.046% in nonblack Americans.

When sickle hemoglobin is deoxygenated, the molecules have an increased tendency to form hydrophobic bonds and will subsequently aggregate into large polymers on the red blood cell membrane. The rate and extent of polymer formation are dependent on three factors: intracellular hemoglobin concentration, presence or absence of Hgb F and the degree of oxygenation in the cell. In sickle cell trait, the hemoglobin concentration is normal, and Hgb F is not usually present postnatally. Therefore, the predominant physiologic cause of intravascular sickling in those with heterozygous sickle genes (Hgb AS) is the low level of oxygenation in the cell.

Sickle Cell Trait and Exercise

During exercise, the pH decreases and the temperature increases at the tissue level to facilitate oxygen delivery; these changes lead to higher concentrations of deoxygenated hemoglobin. Under usual circumstances, cardiac output is likely to increase substantially and raise perfusion pressure sufficiently to overcome local tissue hypoxia and avoid the membrane polymerization that can occur with sickle cell trait. Although no study has shown sickling during rest at sea level, maximal exercise at sea level routinely produces mild intravascular sickling (typically <1%).[1,2] Venous blood from an exercising limb was measured and showed that sickling does indeed occur at low altitude (1,270 m); the sickling rate dramatically increased at high altitude (4,000 m). Even though multiple studies showed that large amounts of sickled cells formed during exercise, no measurable effects on exercise performance were noted.[3,4] Nevertheless, the formation of sickled cells supports a causal relationship between sickle cell trait and exercise-relat-

ed complications. The wide range of sickling that occurred during peak exercise (1–25%) suggests that other physiologic variables play a role.

Sickle Cell Trait and Sudden Death

The strongest evidence implicating intravascular sickling with tissue injury and even death is extreme exercise, typically to exhaustion, with dehydration and relative hypoxia (altitude). The first cases identified were four U.S. Army recruits who died during or immediately after strenuous exercise during basic training at Fort Bliss, TX (elevation 4,050 ft) between March 1968 and February 1969.[5] On the basis of autopsy specimens and morphologic criteria previously established for individuals with sickle cell disease,[6,7] the authors concluded that the soldiers died of diffuse microvascular obstruction from sickled erythrocytes, or "sickle crisis." Although hypoxemia was modest at 4,000 ft (ambient PO_2, 70–75 mmHg), investigators concluded that the decrease in arterial PO_2 levels initiated events that led to acidosis, excess lactate and intravascular sickling. They postulated that even though factors such as dehydration, increased blood viscosity and hypercoagulability may have contributed to the syndrome, the "... moderate-to-severe exercise after recent arrival at a relatively high altitude ..." ultimately led to sickle crisis and sudden death. They also concluded that the variable Hgb S concentration (30–44%) among recruits spared some with sickle cell trait but not others.

Since the description of the initial deaths, multiple reports of serious complications or sudden death during exercise for individuals with sickle cell trait have been published.[8-21] The magnitude of the problem was established after a retrospective review of all deaths among active-duty military between 1977–1981 was published by Kark et al[22] in 1987. The comprehensive review, written by Army Medical Corps physicians, concluded that the risk of sudden unexplained death in black recruits with sickle cell trait was approximately 30-fold more than that of black recruits without sickle cell trait and 40-fold more than that of nonblack recruits. Most deaths occurred during the first month of training and were associated with exertional activities requiring maximal effort. Much of the data implicated exertional heat illness, which had been somewhat arbitrarily subclassified into heat stroke, heat exhaustion and heat injury. Deaths were attributed to exertional rhabdomyolysis if muscle necrosis was a major component of the clinical picture.[19,23-25] The degree of hyperthermia present during any of the syndromes was variable, and it was not always present during exertional rhabdomyolysis. Furthermore, in many cases, the true cause of death was difficult to determine; rhabdomyolysis may have been attributable to exercise, or mild muscle necrosis may have been augmented by hypotension during resuscitative measures and cardiac arrest.

Several factors may explain the higher incidence of sudden death in military recruits with Hgb AS when compared with civilian athletes. The intensity of physical conditioning over an 8–12-week period is often implicated, and conditioning typically includes activities that raise the metabolic rate 10–14 times above the basal level. Other commonly cited factors included variable recruit conditioning, viral syndromes, water deprivation in the field and heavy protective military gear that may inhibit evaporation of sweat.

An age-associated increase in the death rate has been noted for those with sickle cell trait;[22,26] the death rate of 28–29-year-olds is eight-fold higher than that of 17–18-year-olds. The higher death rate may be attributable to the cumulative effects of renal papillary necrosis and resultant isosthenuria (Figure 1),[27] the inability of the kidneys to maximally concentrate urine. Isosthenuria is the only clinical condition consistently present in individuals with sickle cell trait; the condition tends to be intermittent and reversible initially but gradually may become structural, irreversible and cumulative.[28,29]

Even though the majority of data in the sickle syndrome literature[5,6,8,9,12,14,16,22,30,31] showed that sickle cell trait was associated with increased risk of exercise-induced sudden death (independent of pre-existing disease), the inability to discern histologically artifactual postmortem sickling from antemortem sickling has left the association open to debate. Even though a causal relationship is plausible, no direct evidence links the pathogenesis of exercise-related deaths to microvascular obstruction by rigid erythrocytes.

In the context of their study findings, in 1982, the Army Medical Corps reiterated the importance of regulations to decrease the incidence of exertion-related deaths and noticed a dramatic decrease in the death rate in individuals with sickle cell trait over the next 10 years.[32] Each branch of the military has developed its own policy. In 1996, the Army ceased screening for the sickle gene. The Marines screen all individuals and do not alter the routine of those with sickle cell trait. The Air Force screens everyone and offers the option to decline service to each recruit positive for the sickle gene. The Navy screens recruits and identifies those with sickle cell trait by a neck tag and a red belt during strenuous exercise drills. The Army was said to be reviewing its policy with respect to screening after eight deaths occurred during basic training from 1999–2000.[32] Of those soldiers, five were tested and three were positive for the sickle gene. Their review has been put on hold since the start of the Iraq War. The Sickle Cell Disease Advisory Committee of the National Heart, Lung and Blood Institute (a section of the National Institutes of Health) has since recommended that the military stop the routine screening of recruits for sickle cell trait.

Bringing It Home

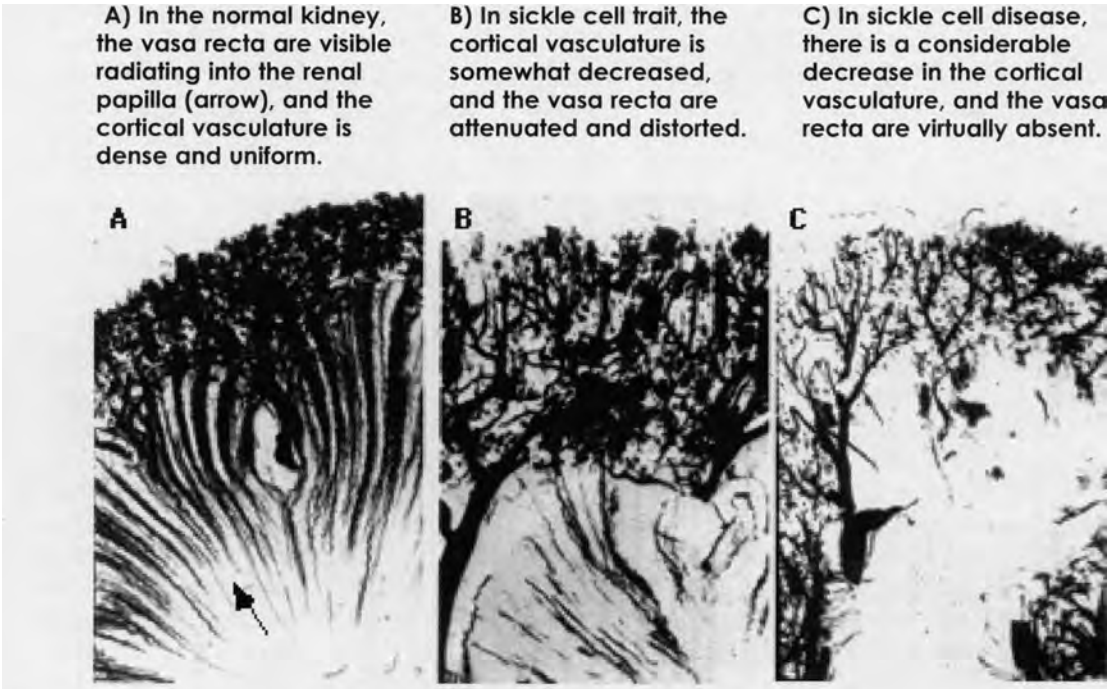
And that is where we are: inconclusive data have been accepted; inconsistent messages about screening, prevention and precaution have been relayed; and research into sickle cell trait-associated sudden death has not advanced. For many physicians, the story stops there. However, I am an African-American male with sickle cell trait, and this is a personal journey. In August 1975, I was a 17-year-old Navy recruit in basic training at the Recruit Training Center in Orlando, FL (Figure 2). I can easily recall the daily marches and drills on the "blacktop" in 105° weather. I can also recall the multiple near-syncopal episodes that, like most 17-year-olds, I tried to ignore so I would not appear weak in front of my fellow sailors. Were any of these episodes due to sickle cell trait and exertional heat illness? I will never know.

Before new regulations were implemented in 1982, the exercise-related death rate among black recruits with sickle cell trait was 30 times higher than that of black recruits without sickle cell trait.[22] Why did 13 years pass after the four deaths occurred in Fort Hood before the military implemented new regulations? Why is there still no consensus among practicing physicians on how to advise patients with sickle cell trait and their parents, athletic trainers and coaches? What roles have black scientists and physicians played in discouraging or supporting further research in this area?

I believe research has been hindered for several prevailing reasons. Many leaders in the black community were reluctant to support identification of increased actuarial risk, and several insurance companies had increased rates and refused to issue or renew policies for individuals with sickle cell trait, even when sickle cell trait was not considered an actuarial risk. My mentor, Dr. Louis Sullivan, wrote in 1987, "... there is no justification for discrimination in employment, job promotions, insurability, or other aspects of daily living among the 8 percent of the black population in the United States who have sickle-cell trait."[33] Moreover, the Health Insurance Portability and Accountability Act, enacted in 1996, prohibited issuers of health benefits from considering "genetic information" when evaluating, granting, denying or canceling an individual's healthcare coverage. Genetic information included results of genetic tests (e.g., a patient's Hgb SS or Hgb AS status) or findings of a routine history and physical examination.

Another barrier to investment in research has been the prevailing social climate in the United States during this period. Racial prejudice has undoubtedly fueled the occasional acrimonious debate. Combined with the inherent mistrust of medical research by many blacks, particularly in light of research fiascos such as the Tuskegee Syphilis Study and our recent acknowledgement of the pervasive disparities in healthcare, it is not

Figure 1. Injection microradiographs of kidneys from an individual with no hemoglobinopathy (A), sickle cell trait (B) and sickle cell disease (C). The renal cortex is at the top and the medullary pyramid is at the bottom of each picture.



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difficult to understand why the motives of anyone associated with the medical establishment are questioned.[34]

The unpredictable nature of events leading to sudden death is the main reason the true tenets of scientific method could not be consistently applied. Specifically, affected individuals need to be observed in extreme environments with all the implicated variables (e.g., increased ambient temperature, dehydration, poor conditioning, viral illnesses, etc.) to form a reasonable hypothesis. However, variability in the clinical presentation and environmental factors makes testing the hypothesis for validation nearly impossible.

After publication of the comprehensive review by Kark et al,[22] physicians generally accepted that indeed there was an increased risk of sudden unexplained death associated with physical exertion in individuals with sickle cell trait. Multiple prospective and well-controlled physiologic studies from the 1980s[3,33,35] showed that sickle cell trait alone was not an impediment to exercise tolerance or maximum performance. However, a subset of individuals with sickle cell trait obviously have an increased risk of exercise-induced complications. Most researchers and clinicians working in this area appear to have concluded that the increased morbidity and mortality for individuals with sickle cell trait result from the confluence of poor conditioning, strenuous physical exertion (especially in hot climates), dehydration and age.[22,26]

Previous studies showed that exercise to exhaustion produced some intravascular sickling at sea level,[1,2] and the amount of sickling was variable among individuals with Hgb AS.[3,4] In the absence of further evidence, we must conclude that Hgb AS and exercise-related sudden death have a cause-and-effect relationship. However, substantiation of that relationship is unlikely.

Making treatment decisions and recommendations on the basis of assumptions is not without precedent. For example, even though there has been considerable evidence for many years indicating that maintenance of euglycemia decreases most microvascular complications of diabetes mellitus (retinopathy, neuropathy and clinical nephropathy), there was no conclusive evidence that showed improved glycemic control decreased cardiovascular complications. The United Kingdom Prospective Diabetes Study[36] showed improved lipoprotein profiles with improved glycemic control, and the Diabetes Control and Complications Trial[37] did not show a statistically significant decrease in macrovascular events with improved glucose control. Nevertheless, clinicians assumed a cause-and-effect relationship between hyperglycemia and poor cardiovascular outcomes, and we continued to tell this to our patients and the public in the absence of conclusive data. Diabetic research progressed and only recently has confirmed that improved glycemic control decreases the risk of cardiovascular disease.[38]

Future Research

More attention needs to be given to the progressive and cumulative nature of the renal effects of sickle cell trait. Previous work[28,29] showed that after the fourth decade, some individuals with Hgb AS had the same diminished renal concentrating capacity as that of patients with Hgb SS (sickle cell disease), which created a clinical situation similar to nephrogenic diabetes insipidus (Figure 3).[27] Nevertheless, the prevalence and magnitude of renal dysfunction generally remain unknown. How does our lack of awareness and surveillance of renal concentrating capacity affect our ability to treat diseases such as systemic arterial hypertension and diabetes mellitus for patients with Hgb AS? In light of the potential for volume depletion, especially during physical exertion, is diuretic use ever appropriate in this population?

Under conditions of severe hypoxia, those with sickle cell trait are at increased risk of intravascular sickling. I designed a study to better understand the physiologic changes that occur in individuals with sickle cell trait while diving with a self-contained underwater breathing apparatus (SCUBA); ultimately, the goal was to calculate the risk of decompression sickness. The pilot study was funded for two years and was to test divers with sickle cell trait. Blood, drawn anaerobically and immediately fixed upon ascent from several depths, was to be evaluated for sickled red blood cells. After soliciting participation from major African-American dive organizations in the United States over several years, we were able to enroll only a few participants. The study was terminated because of lack of participation. Later discussions with potential participants revealed that most were concerned

Figure 2. Photograph of the author at the completion of Navy basic training (September 1975)

about the effect of adverse findings from the study restricting their ability to get diving insurance. Most also felt that since they had been diving thus far with no serious consequences, the possibility of intravascular sickling with diving was small or inconsequential.

CONCLUSION

All individuals who are phenotypically at risk should know their Hgb S status. In the absence of definitive studies that identify individuals with sickle cell trait who have the highest risk of sudden death, specific precautions should be reinforced. When participating in strenuous activities, care should be taken to avoid dehydration, and water must be consumed beyond the level of intake stimulated by thirst, especially in hot and humid conditions. Conditioning regimens should be slow and gradual, and older or obese individuals may need to take extra precautions. Activities requiring maximal exertion should be limited when the individual feels ill, and such activities should be limited for ≥1–2 weeks after viral syndromes or respiratory infections.

The military (and military-like institutions) should screen all recruits for the sickle gene and establish measures during basic training that would decrease the likelihood of exertional heat illness for all participants. These measures will decrease the disparity in sudden death between those with sickle cell trait and those with normal hemoglobin, and they would eliminate the need to mark and segregate individuals. Knowledge of a recruit's Hgb S status could facilitate prompt and appropriate treatment during a medical emergency. As individuals with sickle cell trait age and acquire other diseases (e.g., diabetes, hypertension, etc.), military physicians can better direct exercise and treatment regimens that are suitable for them.

Since diuretics play a major role in the treatment of hypertension in blacks, clinicians should be aware of the possible deleterious effects of this class of drugs for those with sickle cell trait. Patients with hematuria, polyuria or nocturia should be evaluated promptly.

Until further research is conducted, individuals with sickle cell trait who dive with SCUBA should limit their dives to <66 ft (2 atmospheres). They should also diligently maintain adequate hydration before and after dives.

African Americans clearly have a vested interest in understanding variables that increase the morbidity and mortality of this otherwise benign condition. New research initiatives need to be encouraged and broadly supported. We must understand the social context in which many previous studies were conducted and recognize its potential effect on further scientific research. Until conclusive data are gathered, the reasonable assumption of a causal relationship between sickle cell trait and higher morbidity and mortality must be acknowledged to develop consistent, cautionary recommendations for our patients, the military and the public.

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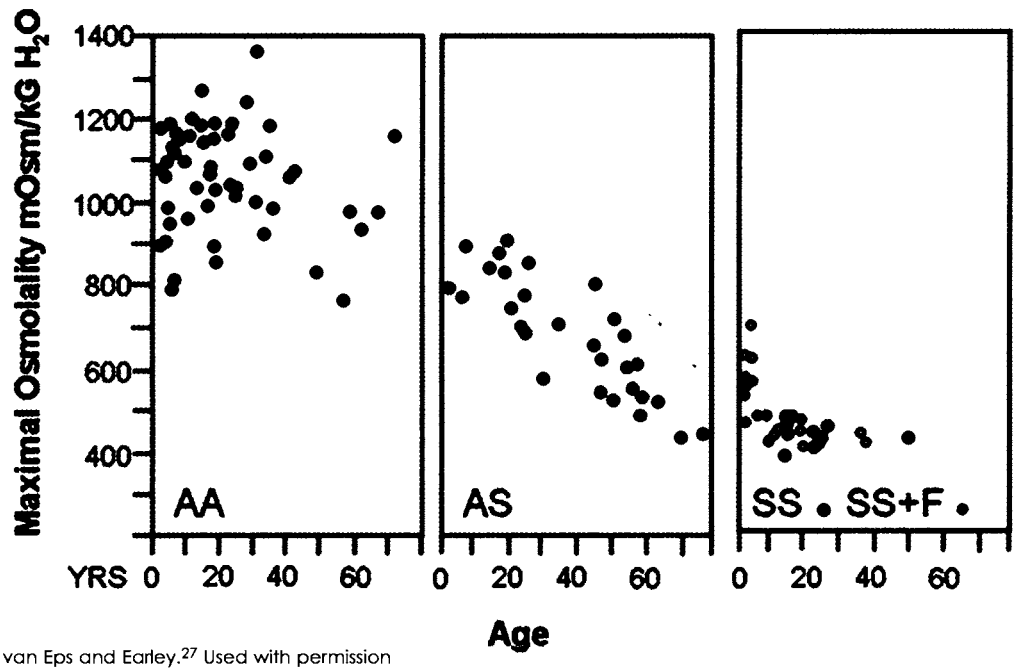
Figure 3. Relationship between maximal urinary osmolality and age in normal subjects (AA), sickle cell trait (AS), sickle cell anemia (SS, SS+F)



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Sickle Cell Trait

By Dan Martin, Ed.D., ATC

- It is estimated that eight percent of the U.S. African-American population has sickle cell trait (SCT).
- SCT does not necessarily preclude an individual from sport participation.
- Signs and symptoms of a sickling crisis must be recognized early to prevent complications, including the risk of death.
- Basic precautions will greatly decrease the risk of a sickling crisis.

SIGNIFICANCE

Sickle cell trait (SCT) is not a disease, but a description of a type of hemoglobin gene. Hemoglobin carries oxygen in the bloodstream. SCT differs from sickle cell anemia in that the trait is present when one gene for sickle hemoglobin is inherited from one parent while a normal hemoglobin gene is inherited from the other. If a sickle cell gene is inherited from each parent, the child will then have sickle cell anemia.

Sickle cell anemia is a serious disorder which typically causes severe medical problems early in childhood which continue into adulthood. People with SCT rarely have any symptoms of the condition. However, they may develop problems under extreme physical stress or with low oxygen levels (high-altitude).

People with ancestors from Africa, Mediterranean countries, India, South or Central America, and Saudi Arabia are at increased risk for having SCT. SCT occurs in about eight percent of the African-American population in the U.S.

SCT exercise-related deaths do occur in both athletics and in the military. Individuals with SCT participating in intense exercise are particularly vulnerable to the effects of heat and dehydration. The potential for a sickling collapse can be decreased if the athlete takes preventative measures. Early recognition of the signs and symptoms by the athlete, coaches and medical staff, with stopping all activity and initiating appropriate treatment will greatly reduce the potential for long-term consequences or death.

BACKGROUND

The U.S. military first linked SCT to an increased risk of sudden death during extreme physical exertion decades ago. SCT has also been linked to several deaths which have occurred during off-season conditioning in collegiate football players over the past decade. Currently, SCT does not appear to be a prominent issue in high school athletes. This is likely due to the fact that the intensity and duration of physical activity in high school athletes does not reach that seen in collegiate conditioning drills.

SCT generally does not present problems with daily activities. The vast majority of athletes with the trait compete at the high school, college, and professional levels without complications. However, there is always the possibility that a sickling collapse can occur with intense exertion, potentially resulting in death.

PHYSIOLOGY

During intense exertion, red blood cells can change from the typical donut-shaped appearance to a "sickle" or a "quarter-moon" shape. In this shape, these cells no longer carry oxygen efficiently and become rather stiff and sticky. These "sickle cells" can then stick together and block normal blood flow to any tissue or organ. This can produce pain, weakness, swelling of the arms or legs, muscle cramping and shortness of breath. Kidney and other vital organ function can also be affected.

Even what appears to be a mild exertional distress can turn lethal in an individual with SCT. The kidneys and spleen may be damaged and exercise-related rhabdomyolysis (skeletal muscle breakdown) may also occur. Asthma (see Asthma chapter), acute illness, dehydration (see Fluid Replacement and Dehydration chapter), heat stress (see Heat-related Illness chapter) and high altitude can predispose an individual with SCT to a sickling crisis during intense physical exertion.

IDENTIFYING THE ATHLETE WITH SICKLE CELL TRAIT

The preparticipation evaluation form (see Preparticipation Evaluation chapter) should have a question about the athlete's sickle cell status. If the athlete or parents are unaware of the athlete's status, they may very likely be able to find the information from their primary care provider or state newborn screening records. The NCAA currently recommends that the SCT status of all athletes be determined. Most states in the U.S. have been conducting newborn SCT screening for more than 20 years, thus many athletes may already know, or be able to find out, their status. There is currently no medical organization calling for the universal screening of SCT in high school athletes. Parents who are interested in having their child screened for SCT should discuss it with their primary care provider.

When an athlete with SCT is identified, it is important that the athlete and his or her parents are educated about SCT. It is important to not discourage the athlete from sports participation. However, the athlete must be educated on preventive measures and the potential dangers. It is vital that coaches and the sports medicine staff be aware of the athlete's SCT status, but it is also important to protect the student's privacy as much as possible.

RECOGNITION

If an athlete exhibits any signs or has symptoms of a sickling collapse, he or she must be removed from activity. Continuing to exercise will lead to worsening symptoms, additional serious internal organ damage, or even death. However, if the proper steps are taken, these symptoms are generally easy to manage and will normally subside within a few minutes. The athlete's symptoms typically resolve when he or she is hydrated and rests. During hot weather, the athlete should also be taken into a cool, controlled environment to prevent overheating. If at any time the athlete collapses, (sickling collapse) the episode must be treated as a medical emergency and Emergency Medical System activated (see Emergency Action Planning chapter).

Signs and Symptoms of a pending sickling crisis

- Appears dazed or confused
- Appears weak
- Not keeping up with other team members (undue fatigue)
- Having difficulty breathing
- Muscle pain, weakness and/or cramping

MANAGEMENT

Athletes with SCT can generally perform at the same physical level as their teammates, but may not be able to do it for an extended amount of time. For example, athletes with SCT should not run timed, sustained 100-yard sprints, or timed, sustained "suicides" or shuttle runs. The athlete with SCT can still run sprints and suicides, but must be given rest breaks between sprints. Coaches and the athlete with SCT must be aware of his or her physical limits. If the athlete is feeling exhausted, or is showing symptoms of physical distress, he or she must immediately stop, hydrate and rest.

If an athlete is known to have SCT, the following precautions are suggested during physical activity:

- Set own pace
- Engage in slow and gradual preseason conditioning regimen
- Use adequate rest and recovery between intense drills
- Stop activity immediately upon struggling or experiencing muscle pain, abnormal weakness, undue fatigue, or shortness of breath
- Stay well hydrated
- Seek prompt medical care when experiencing unusual distress

Though caution must be taken, the athlete with SCT should always be allowed to compete in all sports and should be treated the same as the other athletes. It needs to be emphasized that athletes with SCT normally do not have problems, except if put under extreme physical duress. The precautions and training modifications discussed in this chapter are intended to allow the athlete with SCT to participate in athletics as safely as possible.

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Resources

National Heart Lung and Blood Institute: http://www.nhlbi.nih.gov/health/dci/Diseases/Sca/SCA_WhatIs.html

Sickle Cell Disease Association of America: https://www.sicklecelldisease.org/about_scd/index.phtml

Sickle Cell information center: www.scinfo.org

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Sickle Cell Trait

E. Randy Eichner

Sickle cell trait can pose a grave risk for some athletes. In the past few years, exertional sickling has killed nine athletes, including five college football players in training. Exercise-physiology research shows how and why sickle red cells can accumulate in the bloodstream during intense exercise bouts. Sickle cells can "logjam" blood vessels and lead to collapse from ischemic rhabdomyolysis. Diverse clinical and metabolic problems from explosive rhabdomyolysis can threaten life. Sickling can begin in 2-3 minutes of any all-out exertion, or during sustained intense exertion – and can reach grave levels very soon thereafter if the athlete struggles on or is urged on by coaches despite warning signs. Heat, dehydration, altitude, and asthma can increase the risk for and worsen sickling. This exertional sickling syndrome, however, is unique and in the field can be distinguished from heat illnesses. Sickling collapse is a medical emergency. Fortunately, screening and precautions can prevent sickling collapse and enable sickle-trait athletes to thrive in their sports.

This article on sickle cell trait keys mainly on the features of exertional sickling and how to distinguish it from heat illnesses and other causes of collapse. Some cases of exertional sickling in athletes, especially in football, have been confused with heart disease, viral meningitis, heat cramping, or heat stroke.[1] Knowing the setting and distinguishing features of exertional sickling is vital to athletes, coaches, athletic trainers, and team physicians, because sickling collapses and deaths can be prevented with screening and precautions.

The sickle gene is common in people who come from where malaria is widespread, such as Africa. Over the millennia, carrying one sickle gene—sickle cell trait—fended off death from malaria, which resulted in one in 12 African-Americans (versus one in 2,000 to one in 10,000 White Americans) with sickle trait.

In the past four decades, exertional sickling has killed more than a dozen football players in training and many more military recruits in boot camps. In the past 6 to 7 years alone, sickling has killed nine athletes with sickle trait: five college football players in training, two high school athletes (one a basketball player), and two 12-year-old boys training for football.[2] Other recent non-fatal cases are known to exist. For example, one college football player was fortunate to survive sickling; he underwent renal dialysis for two weeks and was hospitalized for two months. Among 136 well-studied, sudden, non-traumatic, sports deaths over a decade in high school and college athletes, seven (5%) were from sickling-associated rhabdomyolysis.[3]

The author is Professor Emeritus of Medicine and Team Internist at the University of Oklahoma in Norman. E-mail: Reichner1@cox.net.

The U.S. military was the first to tie sickle trait to sudden death during the physical training of recruits. The relative risk of exercise-related death in sickle trait, unexplained by preexisting disease, was about 30. The risk plummeted, however, for career military after basic training. This observation led researchers to speculate that the risk of exercise-related death in sickle trait is mainly during intense conditioning to novel exercise or during sustained exertion for which the recruit is unprepared.[4,5]

Analysis clarified the type of death. Most collapses occurred as recruits ran 1 to 3 miles. Of 40 deaths or near-deaths from sickling collapse, some had features suggesting heat illness (but not heat stroke) and others were sudden cardiac arrhythmic death. But most deaths were delayed for hours or a day or two and were from metabolic complications of rhabdomyolysis and myoglobinuric renal failure. Indeed, acidosis and hyperkalemia from explosive rhabdomyolysis may have caused the sudden arrhythmic deaths. So the main cause of death is explosive rhabdomyolysis, and the risk of rhabdomyolysis is about 200 times greater in military recruits with sickle trait.[4,5]

Pathophysiology of Exertional Sickling

Exercise physiology helps explain why exercise in sickle trait can cause rhabdomyolysis. In sickle trait, in which about 40% of hemoglobin in each red cell is hemoglobin S, strenuous exercise evokes four forces that foster "sickling," or change of red-cell shape (when the red cell releases its oxygen) from round to quarter-moon or "sickle." The metabolic acidosis and hyperthermia "shift to the right" the oxygen dissociation curve, displacing more oxygen from hemoglobin S. Dehydration of red cells traversing the hyperosmotic mileau of working muscles increases the concentration of hemoglobin S. And severe hypoxemia because of extreme muscle oxygen uptake completes the sickling foursome.

Military researchers showed that sickle cells accumulate in venous blood draining exercising muscles *and* in blood from the arm in recruits cycling with the legs. When sickle-trait recruits cycled at high altitude, sickle cells in venous blood from the forearm rose to a mean of 9% and a maximum (in one man) of 28%. This suggests that as exercise stress and hypoxemia increase, sickle-trait athletes can accumulate sickle cells in the *arterial* circulation—sickle cells that can go to the heart, brain, and muscles.[6] If too many sickle cells accumulate, they can "logjam" blood vessels and rob vital organs of blood.

Recent research finds that dehydration can increase exertional sickling. Two men with sickle trait walked briskly for 45 minutes in the heat, once with fluids 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%.[7]

Field Studies in Africa Suggest a Pattern

Field studies in Africa suggest that elite sickle-trait runners are limited not in single sprints but in middle-distance, semi-marathon, and altitude racing. In Ivory Coast, 13 sickle-trait runners won 33 titles, but only one was at 800 meters or more. The other 32 wins were all in races of 400 meters or less.[8] Sickle-trait racers were

underrepresented among top finishers in a semi-marathon and underperformed in high-altitude stretches of a distance race.[9,10] In contrast, sickle-trait runners can be elite sprinters.[11] However, these same African field studies suggest that sickle trait can limit racing performance at altitude or at 800 meters and beyond. This pattern seems to hold in training for American college football, where repeated sprints that total about 800 meters or beyond seem to pose a sickling hazard.

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 in Colorado.[2] 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 a bit, 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."

The pattern of the most recent death, a freshman defensive back at Rice University, is similar. In the fall of 2006, he ran 16 sprints, each 100 yards, and said he felt bad and his legs hurt. Within minutes, he collapsed and became less responsive. No cardiac arrest occurred. He was rushed to a hospital. Serum creatine kinase rose sharply and overnight his kidneys failed. He died the next morning, likely from cardiac arrhythmia from hyperkalemia. The cause of death was acute exertional rhabdomyolysis associated with sickle cell trait.

It seems that at least 13 college football players have died after a sickling collapse, and although the type and proximate cause of death in some have been debated, the setting and syndrome in most are similar. Sickling football players may be on field only briefly, sprinting only 800 to 1600 meters, early in training, often the first or second day of preseason workouts. Sickling can also occur during repeat hill, ramp, or stair running, or even during intense, sustained weight-lifting or other grueling resistance drills, such as pushing a weighted sled in the "bear-crawl" position. Sickling collapse can occur if the tempo increases late in intense one-hour football drills, including "mat drills" in the winter. At the end of a long, hot football practice, if players run sprints or "gassers" with insufficient breathers, sickling can begin within 600 meters. Sickling can even occur rarely in the game, for example, when a running back is in almost constant action during a long, frantic drive downfield.

Other Sickling Situations

Sickling collapse is not limited to college football. It has killed or nearly killed several college or high school basketball players (two were females) in training, typically when "running for time," such as repeat sprints on the court ("suicide runs"), laps on a track, or a long training run. A young sickle-trait runner collapsed twice at the end of cross-country races. He survived severe rhabdomyolysis and renal failure.[12]

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 to 1200 meters) than in military

recruits running 1 to 3 miles. Some sickling can begin in only 2 to 3 minutes of sustained sprinting—or in any other sustained all-out exertion—and sickling can soon increase to grave levels if the stricken athlete struggles on or is urged on by the coach to push beyond pain and weakness. Any cramping, struggling, wobbling, or collapse in a sickle-trait athlete must be considered sickling—a medical emergency—until proved otherwise. Especially in high heat and humidity, the exertion need not be "all-out" to cause ominous sickling, as in military men who collapse at the end of a 2-mile fitness run.[4]

Heat and dehydration increase the risk of sickling, mainly because they make the drill more difficult and drive the blood oxygen lower. They also tend to worsen sickling, by shifting the oxygen-dissociation curve to the right and by increasing the concentration of hemoglobin S. Exercise-induced asthma and the thin air of altitude also increase sickling, even when the exercise is not all-out, because of lower blood oxygen.

Differential Diagnosis: Four Possibilities

This is key for athletic trainers and team physicians to know: If an athlete takes the field in good health, the four most common nontraumatic causes of sudden exertional collapse are a cardiac condition, a heat illness, asthma, or sickling. Clues can help observers distinguish among these four emergencies. In general, cardiac collapses are very sudden and without cramping and, especially with ventricular fibrillation, when the athlete hits the ground, he no longer talks and may even have seizure activity. Exertional asthma typically presents with a slow crescendo of chest tightness and undue breathlessness, sometimes with audible wheezing, usually without extremity or back pain or cramping.

It seems that sickling collapse on field is most often confused with heat cramping, heat exhaustion, or heat stroke. However, unlike heat exhaustion or heat stroke, sickling collapse often occurs early in a workout, in the first few minutes, as during wind sprints. The point is, the athlete has not been out in the heat long enough to suffer heat stroke. Rectal temperature is generally elevated only mildly and in concert with the activity. Also, with heat stroke, central nervous system changes are more profound and can range from confusion to delirium to stupor, seizures, or coma. The most telling symptom of sickling collapse is increasing pain and weakness in the working muscles, especially the legs, buttocks, and/or low back. The athlete may call the pain "cramping," but it is unique. Knowing which athletes have sickle trait avoids possible confusion over "cramping." Any cramping is considered sickling until proved otherwise.

Football players who have suffered both heat cramping and sickling can tell the difference between the two syndromes. Notable differences are the following:

1. Heat cramping often has a prodrome. 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 has no prodrome.
2. The pain is different. Heat-cramping pain is an excruciating pain of sustained, full contraction of muscles, a "lock-up." Sickling pain is milder, neither the

unbearable pain of a heat-cramp lock-up nor the "burning" pain in the thighs as at the end of a middle-distance race. Sickling pain is an ischemic pain from trying to use muscles robbed of blood supply—it is 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 major heat cramping, one can see and feel large, rock-hard muscles in full contraction, and the athlete often is writhing and yelling in pain. With sickling, the exhausted player lies fairly still and complains little, except to say that he feels bad and his legs hurt and are weak. 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 our experience in college football, most but not all sickle-trait athletes sooner or later will have problems, mild or severe, from exertional sickling. The risk may vary not only with the amount of hemoglobin S in each red cell,[2] but also with the sport, setting, drill, or fitness. Nor will all sickling athletes describe this syndrome the same way or look exactly the same. Some present with leg and low-back pain or "leg cramps" that "spread up through my body." Some complain of leg weakness more than pain. Some say "my chest muscles are tight" or "I just don't feel right" or "I can't catch my breath" and simultaneously say their legs are weak or "wobbly." They can be on hands and knees by this point, breathing very fast, with great anxiety. Some stoic players with sickling will just stop and sit or lie down, saying "I can't go on" or "my legs won't go." This self-limiting feature has likely saved the lives of many sickle-trait athletes.

A Medical Emergency

A sickling collapse is an emergency. Fast action can save a life:

- Check vital signs.
- Give supplemental oxygen by face mask.
- Cool the athlete, if necessary.
- Failing immediate improvement, call 911, attach an AED, and start an intravenous line.
- Get the athlete to the hospital quickly.
- Tell the doctors to expect explosive rhabdomyolysis and its grave metabolic complications.

Return to Play

Return to play must be individualized. Some athletes who spend a long time in the hospital with major, debilitating rhabdomyolysis and renal failure do not return to play. For example, the college football player who spent two weeks on renal dialysis and two months in hospitals was not cleared to return to play because of permanent loss of renal function. At the other extreme are exercising athletes who report early symptoms of sickling and are attended to immediately with rest, oxygen, and cooling. In our experience, these athletes can rebound quickly. Likely many or most sickle cells revert quickly to normal shape as they traverse the lungs and can feel normal again in 15 to 30 minutes. We have let such athletes return to play the next day.

Many athletes may fall between these two extremes. This has been the case for football players who develop moderate rhabdomyolysis. Indeed, we have seen troubling myonecrosis of lumbar paraspinal muscles, with a sharp rise in creatine kinase (sometimes to the range of 10,000 to 50,000 IU/L) but no worrisome rise in creatinine and no renal compromise. These athletes tend to have limiting muscle weakness and soreness for a week or two and need daily assessment to ensure prudent, gradual return to play.

Preventive Measures

How to prevent sickling collapse is detailed elsewhere[2]; in general, it involves screening and simple precautions. Over the past decade, dealing with 13 sickle-trait college football players and lesser numbers of basketball players and track athletes, these precautions seem to work for us:

- Build up slowing in training, slow the tempo, and give longer “breathers.”
- Allow no timed sprints or miles.
- Allow no sprints beyond 500 meters.
- Allow no all-out exertion of any kind for 2 to 3 minutes without a breather.
- Adjust work/rest cycles for heat, hydrate well, control any asthma, and allow no workout if ill.
- Have supplemental oxygen ready at altitude games.
- Set the tone so athletes report symptoms immediately and coaches and athletic trainers consider any struggling or cramping as sickling.
- If sickle-trait athletes can set their own pace, they seem to do well.

Screening and precautions for sickle cell trait can prevent sickling collapses and deaths and enable sickle-trait athletes to thrive in their sports.

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SECRETARY'S ADVISORY COMMITTEE ON HERITABLE DISORDERS
IN NEWBORNS AND CHILDREN

Screening U.S. College Athletes for Their Sickle Cell Disease Carrier Status

10/11/2010

This briefing paper outlines issues surrounding the screening of young athletes for their sickle cell disease carrier status (or sickle cell trait), a genetic condition. The Secretary's Advisory Committee on Heritable Disorders in Newborns and Children (SACHDNC) has developed the report to advise the Secretary of the U.S. Department of Health and Human Services about the rule of the National Collegiate Athletic Association requiring testing for sickle cell trait in all incoming Division I student athletes.

Section 1: Introduction and Background

By the end of 2009, there were approximately 4 million people in the U.S. (1.31% of the population)[1] and an estimated more than 300 million people worldwide with sickle cell trait (AS).[2] Sickle cell trait occurs in high frequency among people of African or Middle Eastern descent but it is also common among those of Indian, or Mediterranean origin.[3] For nearly three decades, screening for sickle cell disease and related hemoglobinopathies has been part of state-public health newborn screening programs in the U.S. An important aspect of the public health approach to screening is the recognition that screening for sickle cell disease, sickle cell trait, and related hemoglobinopathies, like all genetic testing, should include counseling to explain the benefits and risks of testing, and health education that explains the results and their implications.

SACHDNC shares the commitment of the athletic associations - National Collegiate Athletic Association (NCAA) and the National Athletic Trainers' Association (NATA) - to ensure the safety and health of college athletes, including those with sickle cell trait, and offers recommendations within this briefing paper that are based on the current state-of-knowledge about definite, probable, possible and unlikely associations of morbidity and mortality or health risks with sickle cell trait, and provides recommendations that may help prevent certain adverse events in all persons involved in collegiate sports, including those with sickle cell trait.

The Committee believes that it is within the public health protection and assurance responsibility of the Secretary to provide guidance to the nation and institutions, like athletic associations, on matters related to athletics and health. We recognize that the scientific evidence needed to fully judge the best practices related to the athlete with sickle cell trait has not been fully established. SACHDNC thinks informed opinion allows rational recommendations based on experience and knowledge of the pathophysiologic mechanisms underlying sickle cell conditions.

Finally, this briefing paper does not discuss implications of universal or population-based carrier screening for heritable disorders or conditions but is a limited discussion of the implications of genetic screening for a selected population presumably for its protection against physical harm in collegiate athletics.

Section 2: Research findings and reports on Health Outcomes of Sickle Cell Trait

Sickle cell trait is defined by the inheritance of a normal beta hemoglobin gene from one parent and a sickle cell gene from the other parent. In this condition, every red blood cell contains both hemoglobins A and S, and hemoglobin A is the predominant hemoglobin produced as opposed to sickle cell disease (SCD) in which sickle hemoglobin (S) is the predominant hemoglobin. Hemoglobins exist as tetramers but in their assembly, stable (alpha-beta) dimers are formed first. Sickle cell trait red cells therefore contain three main populations of hemoglobin tetramers: $\alpha^A{}_2\beta^A{}_2$ (Hb A), $\alpha^A{}_2\beta^S{}_2$ (Hb S), and the hybrid hemoglobin, $\alpha^A{}_2\beta^A\beta^S$ (Hb A/S).[4] Both Hb S and Hb A/S participate in polymerization when deoxygenated. It is the presence of excess Hb A ($\alpha^A\beta^A$ dimers) that ensures that AS cells do not sickle under physiologic conditions. It is not surprising therefore that red blood cells from individuals with sickle cell trait can be induced to sickle *in vitro* under certain conditions such as deoxygenation.[5] Furthermore, in situations or in tissues where high plasma osmolarity, severe acidosis, and/or hypoxia occur, such as the renal medulla, sickling is believed to occur in sickle cell trait. However, although sickle cell disease is characterized by chronic hemolytic anemia, vasculopathy, and acute vaso-occlusive events, sickle cell trait has not been associated with these features under <u>normal</u> physiologic conditions.

Medically, sickle cell trait is known as a powerful protector of young children from mortality and severe morbidity from acute falciparum malaria infection through reduced parasite density.[6] And, in malaria endemic populations, sickle cell trait has a broad positive effect on child survival. In other populations, there is no evidence of such a beneficial effect of sickle cell trait. In a large study of 65,514 “Black” patients admitted consecutively to 13 Veterans Administration hospital in the 1970s, 7.8% of them were found on blood tests to have sickle cell trait.[7] The inpatient records of 24,616 members of the tested cohort - 18,294 with no abnormal hemoglobins, 4,900 with sickle cell trait, and 1,422 with hemoglobin C trait – were reviewed. The study concluded that, “sickle cell trait had no effect on average age at hospitalization or death, overall mortality, length of hospitalization on medical and surgical wards, and frequency of any diagnosis, except essential hematuria and pulmonary embolism. Although, historically, sickle cell trait was thought to be an asymptomatic condition, hyposthenuria[8], hematuria[9], and an increased risk for glaucoma after traumatic hyphema are now accepted as complications of sickle cell trait.[10] In addition, splenic infarction has been reported in individuals with sickle cell trait traveling to high altitudes.[11] Over the last two decades, renal medullary carcinoma, a rare aggressive kidney tumor, has been reported to be associated with sickle cell trait.[12] These reports, combined with continual

reports of sudden death in individuals with sickle cell trait undergoing physical exertion, have fueled the controversy over the issue of the impact of sickle cell trait on the health of the individual.

There has been growing concern over the past several decades about the association of sickle cell trait and exercise-related morbidity and mortality in collegiate athletes and military recruits. The strongest epidemiological data supporting that association was a study of over 2 million military recruits in which the risk of sudden unexplained death in recruits with sickle cell trait (hemoglobin AS) was compared with that in recruits without hemoglobin S[13]. The retrospective review of 1977-1981 demonstrated that among "Black" recruits, those with sickle cell trait (AS) were 27.6 times more likely to suffer sudden unexplained death than those without hemoglobin S; and, among all recruits (“Black” and “Non-Black”), those with sickle cell trait were 39.8 times more likely to suffer sudden unexplained death than those without hemoglobin S. Over the 5-year period reviewed, there were 12 unexplained sudden deaths in 37,300 “Black” recruits with sickle cell trait, none in 1,300 “Non-Black” recruits with sickle cell trait, and 5 in 429,000 “Black” and 11 in 1,617,000 “Non-Black” recruits without hemoglobin S. All the 12 sudden unexpected deaths in recruits with sickle cell trait were related to exercise with five of the 12 classified as “sudden unexplained cardiac deaths”.

Kark’s retrospective report from the US military suggested strongly that exertional heat illness might account for increased incidence of sudden death in military recruits, especially those with sickle cell trait, who were training under extreme conditions. In a 10-year (1982-1991) follow-up prospective trial of modified training regimen that included enforced hydration and close monitoring of environmental conditions, there was overall decrease in sudden expected deaths in the 2.3 million recruits, with none occurring in 40,000 recruits with sickle cell trait.[14] Training centers not participating in the trial had rates of sudden unexpected deaths similar to those reported in the 1977-1981 review. Based on this study, testing for sickle cell trait was removed as a requirement for military accession in 1996. There have not been similar epidemiologic studies or intervention trials in collegiate or any other group of athletes.

However, although exercise related injury and death are rare generally, there continues to be concern over the concentration of deaths among athletes with sickle cell trait. The issue of how to manage exercise in people with sickle cell trait has been debated in two articles that summarize much of the debate.[15,16] There have been 15 reports of sudden deaths in college athletes with sickle cell trait over the last 15 years. These cases have received a great deal of media attention. However, because there is

no systematic reporting or registry of the exercise-related deaths, and the diagnosis of sickle cell trait is often unclear, the true incidence is unknown. Since many of these cases were settled out of court, the medical details have neither been revealed nor subjected to scientific review. Without well-designed epidemiologic studies, the number of deaths and injury, their causes, possible underlying pathophysiologic mechanisms, and true attributable risks in individuals with sickle cell trait remain unclear. Therefore, medical scientists have had great difficulty determining whether there is increased risk of sudden death in individuals with sickle cell trait.

However, there are common clinical themes. Many of the deaths occurred after strenuous exercise in unconditioned military recruits or deconditioned athletes in pre-season. Some reports suggest that milder episodes of heat-related illness occurred previously in the subjects.[17] Clinical description of fatal cases are often incomplete and anecdotal. The exercise routine leading to the acute illness is often easier to document in organized athletic activities. It has been suggested that increased risk of morbidity/mortality is related to repeated exercise with great physical effort over a short period of time rather than sustained mild to moderate physical effort over a long period.

Finally, experts do not agree on the possible underlying pathophysiologic mechanisms that may lead to sudden death in individuals with sickle cell trait who undergo strenuous exercise. Some believe that red cell sickling is the primary event leading to vascular occlusion, muscle infarction, and rhabdomyolysis. Almost all reports of unexplained death in people with hemoglobin S cite the presence of sickled red blood cells at autopsy as causation of death. However, the presence of sickled cells in post-mortem tissue specimens from a person with hemoglobin S does not imply pre-mortem sickling. In fact, the tissue hypoxia and acidosis associated with death are expected to induce deoxygenation and polymerization of sickle hemoglobin and red cell sickling soon after death. Therefore, sickling seen in post-mortem samples is most likely an artifact of death. Hyposthenuria, a known feature of sickle cell trait, has been suggested as a factor that may increase the risk of dehydration leading to rhabdomyolysis. Sickling of a few red cells has been observed in experimental subjects with sickle cell trait undergoing strenuous exercise under conditions of heat and restricted hydration.[18]

Athletes with sickle cell trait can and have performed at the highest level of sports. In reports from several countries, including the U.S., on professional and international athletes in several sports, the prevalence rates of sickle cell trait were similar to those in the general population. In addition, exercise

in individuals with mild or severe forms of sickle cell disease does not seem to be associated with serious adverse outcomes, making a biological link between sudden death and sickling much more difficult to suspect in those with sickle cell trait. However, those with the most severe forms of sickle cell disease may be incapable of or prohibited from participating in strenuous exercise to the degree where adverse outcomes may be seen.

In summary, there is evidence for the occurrence of exercise and/or heat-related acute illness in people with sickle cell trait. Some of these events are severe and fulminant enough to cause death. However, the prevalence of exercise-related illness and death in people with sickle cell trait is too small to affect the overall survival or morbidity of the condition. The possibility that an unidentified, rare, genetic defect found in a subset of people with sickle cell trait is either solely or partially responsible for the increased risk of exercise and/or heat-related death can only be ruled out through well-organized, large-scale genetic epidemiology studies.

Section 3: Public Health Implications of the National Collegiate Athletic Association Rule

NCAA Rule on Testing for Athletes for Sickle Cell Trait

In June of 2009, the NCAA Committee on Competitive Safeguards and Medical Aspects of Sports adopted the recommendation that its member colleges and universities test student-athletes to confirm their sickle cell trait status if that information is not already known. Subsequently the NCAA amended its Sports Medicine Handbook to recommend that athletics departments confirm sickle cell trait status in all student-athletes during their required medical examinations.[19] In April 2010, the NCAA Division I Legislative Council decided that effective from the 2010-11 academic year, "all incoming Division I student-athletes must be tested for sickle cell trait, show proof of a prior test or sign a waiver releasing an institution from liability if they decline to be tested." [20] This is a rule, not a recommendation.

The original 2009 legislation requiring all incoming student-athletes to be tested before participating in athletic activities was part of a legal settlement between Rice University and the parents of Dale Lloyd, a football student-athlete whose death a day after he collapsed during a workout in September 2006 was attributed to sickle cell trait.[21] The NCAA cited the 2007 recommendations of the National Athletic Trainers' Association (NATA)[22] and the College of American Pathologists (CAP) in making their 2009 recommendations. The NCAA, NATA, and CAP all state that athletes with sickle cell trait would not be disqualified.

A critical concern with both voluntary and mandated screening for sickle cell trait as a prerequisite for participation in organized sports is protection of the rights of the individual. This includes protection of privacy and protection against actions of a discriminatory nature, such as labeling or prevention from participation in competitive sports. With regard to the NCAA rule, the benefit of screening thousands of collegiate students as prerequisite for participation in sports is unproven and should be carefully weighed against the consequential risk of stigmatization, misinformation, and unwitting denial of access to potentially successful athletic careers for those who happen to have sickle cell trait. Such consequences would be prejudicial against students found to have sickle cell trait.

Prior to the 2009 NCAA recommendation, testing for sickle cell trait was not uncommon in NCAA Division 1-A colleges. In a 2006 survey reported by Clarke and colleagues in 2007, 59 (64%) of 92

participating colleges in Division 1 Football Bowl Subdivision were screening athletes for sickle cell trait. Of those, 44 colleges (76%) targeted screening to African American athletes, and 9 (21%) screened all athletes.[23] Certified athletic trainers, strength/conditioning coaches, and coaches were notified of sickle cell trait test results the majority of the time, according to Clarke's findings. Screening results were primarily used to counsel athletes with sickle cell trait about potential risks during exercise while genetic counseling was provided at a substantially lesser percentage of colleges. Of the colleges that screened, 13 (22%) reported they had directly treated athletes for a complication of sickle cell trait during exercise. Forty-seven (80%) of the screening colleges reported that they screened in order to initiate preventive measures but, of 33 schools that did not screen, 24 (73%) said there was "lack of evidence-based data supporting such screening", and 13 (39%) thought screening was cost prohibitive.

The NCAA states in its Sports Medicine Handbook "if screening is done, it may be done on a voluntary basis with the informed consent of the student-athlete and should be offered to all student-athletes, "…because sickle cell trait occurs in all populations".[19] If a screening test is positive, the student athlete should be offered counseling on the implications of sickle cell trait, including health, athletics and family planning. Confirmatory testing should be recommended to verify the screening result. Screening can be used as a gateway to targeted precautions." [5] The NCAA suggests that knowledge of a student-athlete's sickle cell trait status "should facilitate prompt and appropriate medical care during a medical emergency." However even prior to the new NCAA testing policy of April 2010, many problematic aspects of the rule began to emerge.

There are two options for providing results on sickle cell screening: provision of results from prior testing or from new testing. Based on informal reports from community physicians, sickle cell centers, sickle cell disease counseling programs and sickle cell disease community-based organizations, both options have encountered problems. In the first option, some athletic departments are encouraging families to obtain newborn screening results. Parents have been contacting their newborn-screening programs to obtain test results. Unfortunately, once the family has obtained the test results, follow-up counseling or education may be unavailable or inadequate. Research has shown that some pediatricians and family physicians do not feel competent to discuss conditions included in newborn screening panels.[24] Evaluation of the skills of pediatric residents in relaying newborn screening results has shown that, indeed, their explanations may be too complex for some parents[25] and even incorrect or

misleading.[26,27] An additional problem is that in some states, universal newborn screening for sickle cell disease may have started too late to include all children currently of college age.

The option of new testing is also presenting some difficulties. Some colleges are offering testing and, in order to save costs, they have selected inexpensive screening tests that do not provide accurate diagnosis of sickle cell trait or other common hemoglobin disorders. Athletes who test positive on the inexpensive screening test are referred for further confirmatory testing with a more definitive test. This approach will of course result in a missed opportunity to detect other hemoglobin variants in athletes testing negative for hemoglobin S, giving them the false impression of having no abnormal hemoglobins.

Other barriers encountered in the college-based testing have included unavailability of coaches and athletic trainers for education sessions about sickle cell trait and exercise-related illness, the source of payment for the testing – college or student, and targeting of testing at some athletes and sports as a cost-saving measure.

In a more organized approach, one sickle cell center partnered with a local university to provide testing, on-site counseling and education about sickle cell trait to student-athletes [28]. Testing was voluntary and included pre-test counseling with each student-athlete. Post-test telephone counseling was provided regardless of trait status. Sickle cell center staff met with coaches, athletic trainers and administrators to provide education about sickle cell trait, methods to prevent heat and exercise-related injury, and counseling to help prevent stigmatization of student-athletes found to have sickle cell trait.

Management of the Student Athlete with Sickle Cell Trait

The NCAA recommends that student-athletes with sickle cell trait should set their own pace; engage in slow and gradual pre-season conditioning; use adequate rest and recovery between repetitions; be excused from some performance tests; stop activity when struggling or experiencing symptoms such as muscle pain, abnormal weakness, undue fatigue or breathlessness; stay well hydrated, especially in hot and humid conditions; refrain from extreme exercise when ill; access supplemental oxygen at [high] altitudes when needed; and seek prompt medical care when experiencing unusual distress. These guidelines place the burden on the athlete to design his/her own training program and monitor his/her condition during strenuous activity, and gradually become acclimated to drills that are more strenuous.

However, athletes do not usually make their own training schedule or exercise routines. If athletic officials need to know the sickle cell status of athletes, they should also bear the responsibility to ensure safety of the athlete. On the other hand, the guidelines do not address the potential of the coach to limit the practice or play time of the athlete with sickle cell trait, consciously or unconsciously, out of concern for the safety of the athlete or for potential personal liability. The guidelines also do not address the culture of sports where athletes are pushed beyond their limits and those who cannot keep up are not given the same opportunities.

If guidelines similar to those guidelines instituted by the Department of Defense were applied to prevent dehydration and exercise-related illness for all student-athletes, [29] the need for sickle cell trait testing would be negated and all students would be better protected. Formal responses from the Sickle Cell Disease Association of America, Inc. and the American Academy of Pediatrics (AAP) to the NCAA rule do not support carrier screening as a means to reduce heat related illness or death. Hord and Rice's editorial in the AAP newsletter indicated that the risk of illness will be reduced for all individuals as training and fitness standards are modified. The authors noted that successful training modifications have occurred in military, firefighter, and police trainee programs. Continued assessment and modification of collegiate training protocols, development and implementation of educational and awareness programs and a clear chain of administrative responsibility to actively address illness and injury prevention will benefit all athletes.[30]

Genetic Privacy

Genetics and genomics have provided the ability to reveal the 'invisible' part of heredity at the molecular level.[31] Genetic information is different from information on cholesterol level or blood pressure. However, because genetic information discloses information beyond the perception of the individual tested, many scientists, lawyers and ethicists have described genetic information as unique and qualitatively different, deserving of "exceptional or special" consideration.

In the case of student athletes, mandatory identification of sickle cell status not only discloses information about the individual athlete, but may also disclose information about paternity and genetic carrier status of family members, raising an important question. Are an athlete's right to genetic privacy invaded if the athlete is required to undergo a test to determine sickle cell status? The result of the test

discloses genetic information that may reach beyond the individual, potentially yielding non-consensual genetic information about the athlete's family members.

Sickle cell anemia, the first "molecular disease", has provided a case study for the exploration of the legal, ethical and social implications of genetic screening programs. Mandatory screening programs, as a condition for school attendance and marriage licenses, are a part of the history of sickle cell screening. Many sickle cell screening programs in the late 1960s and 1970s were later judged shortsighted with limited or no benefit.[32] The history of sickle cell testing has been fraught with abuse of human rights, including the modern right to genetic privacy, with examples of individuals being screened for sickle cell trait without their knowledge. In response to the case of Norman-Bloodsaw versus Lawrence Berkeley Laboratory, in which employees were tested for sickle cell trait, syphilis, and pregnancy without their knowledge, and other examples of invasion of genetic privacy, many states enacted genetic privacy and non-discrimination laws.[33]

Genetic Discrimination

A patchwork of state law was enacted addressing employment and insurance discrimination and genetic privacy rights. On May 21, 2008, President Bush signed the United States Genetic Information Nondiscrimination Act of 2008 (''GINA'').[34, 35] The Congressional Findings in support of the law highlighted the mass screening of African Americans for sickle cell carrier status and the resulting discrimination that occurred during the 1960s and 1970s as a rationale for the Act:

"(3) Although genes are facially neutral markers, many genetic conditions and disorders are associated with particular racial and ethnic groups and gender. Because some genetic traits are most prevalent in particular groups, members of a particular group may be stigmatized or discriminated against as a result of that genetic information. This form of discrimination was evident in the 1970s, which saw the advent of programs to screen and identify carriers of sickle cell anemia, a disease that afflicts African Americans. Once again, State legislatures began to enact discriminatory laws in the area, and in the early 1970s, began mandating genetic screening of all African Americans for sickle cell anemia, leading to discrimination and unnecessary fear. To alleviate some of this stigma, Congress in 1972 passed the National Sickle Cell Anemia Control Act, which withholds Federal funding from States unless sickle cell testing is voluntary". [34]

The Act was the accomplishment of a "13-year legislative saga" where leaders on both sides of the aisle in Congress finally came together to draft and pass a genetic information nondiscrimination bill. The reach of GINA is limited to employment and health insurance discrimination. GINA does not address life insurance, disability insurance, or long-term care insurance. GINA specifically excludes the military, where historically there have been examples of genetic discrimination against individuals with sickle cell trait.[36] Covered entities include health insurers and employers; organizations such as the NCAA are not explicitly covered.

The Act became effective on November 21, 2009. A legal question that will likely be adjudicated is what is meant by "manifested disease or a manifested condition". As the scientific understanding of what it means to be a carrier of a genetic condition (including sickle cell trait) is developed (specifically, when there is phenotypic expression or clinical complications), the federal judiciary may be confronted with interpretation of the term "manifested" when dealing with genetic carrier status and the potential phenotypic expression pursuant to GINA.[34]

A limitation of the Act is that GINA prohibits discrimination based on genotype, but not phenotype.[37] Thus, GINA only applies to individuals who are asymptomatic (non-manifested condition). However, the current debate that sickle cell trait is associated with significant clinical indications during intense exercise can be argued as establishing a manifested genetic condition and excluded under GINA. This is only a legal theory for speculation today but may be the interpretation of the law tomorrow.

The ethical and social considerations of the NCAA rule on screening Division I athletes for sickle cell trait have recently been reviewed by Bonham et al., in a New England Journal of Medicine Perspective. [38] One of the notable points made by the authors addresses the motivation of the new NCAA rule: "The fact [that] the students can avoid the testing if they prove they have already been tested or sign a waiver releasing their university and the NCAA from liability suggests that it is designed primarily as a defensive legal measure."

Accessing Newborn Screening Results and/or its Residual Blood Spots

As of 2007, all states include sickle cell disease as part of their newborn screening panel. In one form or another, states are engaged in efforts to ensure that the results of state-mandated newborn screening

are retained securely, made known to identified medical personnel (pediatrician of record) and parents and guardians of tested newborns. With regard to sickle cell trait, short and long-term follow-up procedures vary from state to state with most states engaging only in the initial notification through letters. In some states, community-based organizations serving persons with sickle cell disease are contracted to provide counseling and education for the families of newborns with sickle cell disease or trait.

Because newborn screening for sickle cell disease in the United States is universal and an integral part of state public health systems, the cost of retrieving test results should be covered by the state. It has been argued that screening need only occur once, in the newborn period, with the understanding that states would maintain permanent databases and systems that would enable results to be retrieved at a later date. Unfortunately, databases for retrieval of the results of newborn screening have not always been implemented widely and even physicians have not always been able to access past results easily. Past results may not have been retained long enough to be available to young adults. Even if the problem of the cost of retrieving newborn screening results becomes resolved, the issue of providing appropriate education and counseling regarding the results would need to be addressed.

It is highly unlikely that student health staff, coaches, and athletic trainers will work with physicians, nurses, genetic counselors, and others trained about sickle cell trait to:

- help the student athlete and parents to understand better sickle cell trait and related genetic disorders of relevance to them;
- become educated and trained continually on how best to identify, prevent, and treat the rare exercise-related complications that may affect the athlete; and,
- ensure that the athlete with sickle cell trait is not handled in a manner that may lead to stigmatization or discrimination and that measures are in place to address such issues if they were to occur.

Section 4: Recommendations of SACHDNC to the Secretary, U.S. Department of Health and Human Services on Screening of Student Athletes for Sickle Cell Trait

It is agreed that all individuals should be given the opportunity to find out whether or not they have sickle cell trait, and they should understand what the medical and genetic consequences of having sickle cell trait are. Recent events such as the decision by the NCAA to recommend screening for sickle cell trait in athletes in its Division I schools will probably result in greater awareness of sickle cell trait than many other efforts to date. However, this awareness may unwittingly also lead to misinformation, undue alarm, stigmatization, and discrimination. Although very uncommon, persons with sickle cell trait may develop symptoms associated with heat stroke and muscle breakdown, also called rhabdomyolysis, when undergoing strenuous exercise as in military training, or sports. However, these problems, not exclusive to sickle cell trait, have simple and practical solutions that were universally applied successfully enough in the military to lead to the removal of screening for sickle cell trait as a requirement for military accession.

The recommendation by athletic organizations that student athletes be tested for sickle cell trait is not backed by empirical scientific evidence. While the value of screening athletes for sickle cell trait remains to be determined, the recommendation fails to acknowledge that testing for sickle cell trait is genetic screening and does not include clear guidelines about follow up counseling and education and mechanisms to protect the privacy of the student athlete, and prevent stigmatization and discrimination. The recommendation shows a lack of sensitivity to an historical context within which sickle cell carrier screening took place in the U.S., and a lack of sensitivity to the real possibility of stigma and loss of opportunities for student athletes who are identified as having sickle cell trait. In response to the recommendation to screen students for sickle cell trait because of concerns that those with sickle cell trait might be at greater risk of exercise or heat-related illness compared with other student athletes, SACHDNC recommends:

1. All individuals should have the opportunity to find out their risk for various medical disorders, including their carrier status for genetic conditions such as sickle cell disease.

2. Evaluation and testing for sickle cell disease and other genetic conditions should take place within the individual's medical home. That evaluation should include counseling regarding the implications of the information for the individual and assurance of the privacy of genetic information. Genetic testing should not be a pre-requisite for participation in sports, unless deemed medically necessary.

3. As part of the individual's annual medical evaluation for participation in sports, all potential athletes should receive education on safe practices for prevention of exercise and heat related illnesses.

4. The Secretary, HHS, instruct SACHDNC to work with the SCDAA, relevant federal HHS agencies, athletic associations, community based and health care professional organizations to develop guidelines and educational resources about screening for sickle cell trait in all persons, including athletes.

5. The National Institutes of Health and the Centers for Disease Control and Prevention conduct research to ascertain if some athletes with sickle cell trait are at increased risk of exercise-related sudden death.

Secretary's Advisory Committee on Heritable Disorders in Newborns and Children Sickle Cell Disease Carrier Screening Workgroup Members

- Susan Berry, M.D. – University of Minnesota, Department of Pediatrics
- Vence Bonham, M.S.- National Human Genome Research Institute, NIH
- Lorraine Brown, B.S. – Genetic Services Branch, HRSA
- Melissa Creary, M.P.H. – National Center for Birth Defects and Developmental Disabilities, CDC
- Althea Grant, Ph.D. – National Center for Birth Defects and Developmental Disabilities, CDC
- Lanetta Jordan, M.D. – Sickle Cell Disease Association of America
- Julie Luedtke, M.P.H. – Nebraska Department of Health & Human Services
- Kwaku Ohene-Frempong, M.D. - Children's Hospital of Philadelphia
- Kim Smith-Whitley, M.D.– Children's Hospital of Philadelphia
- Lenee Simon, M.P.H. – Genetic Services Branch, HRSA
- Joseph Telfair, Ph.D. – University of North Carolina at Greensboro
- Marsha Treadwell, M.D. – Children's Hospital and Research Center Oakland
- Michael Watson, Ph.D. – American College of Medical Genetics
- Andrea William, B.A. – Children's Sickle Cell Foundation, Inc.

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Rhabdo Redux: "Don't Know Much about History"

E. Randy Eichner, MD, FACSM

Introduction

Just as there are 50 ways to leave your lover, there are 50 causes of rhabdomyolysis (16), including eight commonly reported categories of trauma, muscle hypoxia, genetic defects, infections, body temperature changes, metabolic or electrolyte disturbances, drugs or toxins, and exercise (2). When it comes to exertional rhabdomyolysis (ER), however, only one common cause exists: too much, too fast, too soon of an exercise too novel. We have known this for 50 yr, since the outbreaks of the "squat jump syndrome" in U.S. Marine platoons (7). The U.S. military experience also teaches how to prevent ER (3,11,12).

Yet, with each outbreak of ER in a team sport, as in high school football last year in Oregon (4) or in college football this year in Iowa — even after a detailed investigation — some coaches say they are baffled as to how or why this can happen (1). With 50 yr of history, Shakespeare should be right: what's past is prologue — to informed prevention. Instead, Faulkner was right: the past is not even past. The University of Iowa outbreak is an example.

Hawkeye Rhabdo: The Unlucky 13

The Hawkeye outbreak in January 2011 was widely covered, and the in-house investigative report is available online via media links (8). Two days after a 3-wk winter break, the football team was put through "the hardest one-day training regimen in the Iowa…playbook" (17). Some upper body work and sled pushing was done, but by far, the hardest task was 100 timed back squats at 50% top weight for one repetition. The strength coach told the team that losing close games during the prior season should concern everyone and that the workouts ahead would determine "who wants to be here" (17).

The report pinpoints the back-squat drill as the likely culprit, saying, "[We] are as certain as possible…that the strenuous squat lifting workout…caused rhabdo in the 13 who were hospitalized…." One player had dark urine the first night. The next day was upper body work; many players had severe leg pain, making it hard to don shoes or climb stairs; five more had dark urine. The next 2 d were the weekend; no workouts took place, but legs stayed sore, stiff, and weak, and four more players had dark urine. Monday brought speed workouts, but many players said their legs hurt so much they could not jump over the low hurdles; three more had dark urine. On Monday, one player saw an athletic trainer for hypertension; he was sent to a physician who diagnosed ER and then found it in four other players, all of whom he hospitalized. Word was spread, and by Tuesday, 13 football players were hospitalized with ER. None developed compartment syndrome or problematic renal failure; all were discharged as symptoms subsided during the next few days. All were cleared to return to play, but media reports 2 months later implied that not all 13 players were up to full speed and that 1 player was considering sitting out a year to regain his strength and weight (14).

The in-house investigative report, of course, found no wrongdoing and no evidence that the workout was for punishment, although the last time a similar workout had been done (December 2007) was 1 wk after another year that, like 2010, had disappointed coaches. That 2007 workout was recalled by players as very tough, making them very sore, making it hard to don shoes or climb stairs, but none recalled brown urine then. No such workout was done in 2008 or 2009, both strong winning years.

The report of the 2011 outbreak found that those with ER were more likely to have gone to muscle failure during the squats, to think they could not finish, and to do more squats because some were not counted as full squats. The risk of ER also increased with the number of sets and time needed to finish the 100 squats. In all but the linemen, the risk of ER increased with the amount of weight lifted as a percentage of body weight.

The report also says, "We learned that the strength and conditioning coaches were very aware of heat injury and dehydration but they did not know about ER until the cluster occurred…members of the football coaching staff and the strength coaches had not had prior experience with ER." On March 28, 2011, the National Strength and Conditioning Association spoke out on the Iowa outbreak,

Address for correspondence: E. Randy Eichner, MD, FACSM, 321 Baudin Way, Sonoma, CA 95476 (E-mail: reichner1@comcast.net).

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calling for strength coaches to get educated on ER and saying that "[ER] is not an expected or acceptable outcome of any training program…and is a sign that the training program was inappropriate for those athletes at that time of year."

Do Not Know Much About History?

No strength coach should be unaware of ER. This is like a scuba instructor being unaware of decompression sickness. Many articles on ER are in the medical and fitness literature. Even the *CrossFit Journal* publishes articles about it (6). In my column on the football ER outbreak in Oregon, I covered seven other cases or team clusters of upper extremity ER (4). Lower extremity ER as in Iowa has been reported in a college football kicker who did 300 squats (9) and in more than 50 prisoners who did up to 600 to 900 squats or squat thrusts (10,15). Besides the U.S. military experience with ER (3,7,11,12), major and fatal ER has been reported in police and firefighters in training (13). Three college wrestlers died of ER (with heat injury) in the fall of 1997. Fulminant ER tied to sickle cell trait is now the leading cause of death in Division 1 National Collegiate Athletic Association football conditioning (5).

How to Fix It?

Although the investigation of Iowa by Iowa found no wrongdoing, it presented some good ideas: 1) never again use the intense, high-volume squat workout; 2) educate everyone in the athletic department about ER and how to prevent it; 3) teach all athletes the warning signs of ER, and if a few develop it after a strenuous workout, test the entire team for it; 4) improve communication between strength coaches and health care staff, so that athletes are not cleared for inappropriate drills; and 5) disseminate this report, so others can learn from our experience.

The investigative committee speculated that the players had become deconditioned to some degree, especially fast-twitch fibers, during the 3-wk break. Some players agreed. The media too got the point, saying in effect that it was not smart to conduct their hardest workout right after a 3-wk holiday break (17). The head football coach? Not so much. He implied that no "smoking gun" was found. "To me, it would have been great [to find a cause]," he said, and although he promised to drop the workout in question, he also said, "I don't think we can [back off]" (1). Then he awarded the head strength coach the "Assistant Coach of the Year." This may speak to the limits of education.

Education could begin with what the Marines learned 50 yr ago. A seminal report of 60 cases of ER in recruits from "the squat jump syndrome" appeared in 1960 (7). Other reports followed, including an outbreak of ER that hospitalized 40 Marines in one platoon. Incidence and severity of ER varied within platoons and especially between platoons, according to fitness, prior physical conditioning, and type and intensity of exercise. Especially troubling was "high-intensity, repetitive exercise." Besides attention to hydration, diet, and rest, the first tip for prevention of ER in Marine recruits was emphasis on prolonged submaximal exercise, not repetitive exhaustive exercise (3,11,12).

In short, do not try to build Rome in a day. Even CrossFit has figured it out. They encountered major ER from brief sessions, where the athletes who came down with ER turned in marginal CrossFit performances but, during and right after the workouts, showed "no signs of discomfort that were out of the ordinary." CrossFit calls this "cold rhabdo" and says, "it seems abundantly clear [they] were exposed to too much work in too short a time...their previous training…proved woefully inadequate at preparing them for sustained power output" (6).

Maybe Iowa football can learn from the Marines and from CrossFit. As Eleanor Roosevelt said, "Learn from the mistakes of others. You can't live long enough to make them all yourself."

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Blood (ASH Annual Meeting Abstracts) 2010 116: Abstract 945
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Oral Session

Hemoglobinopathies, excluding Thalassemia II

Prevention of Exercise-Related Death Unexplained by Preexisting Disease (EDU) Associated with Sickle Cell Trait (SCT) without Hemoglobin (Hb) Screening or Hb Specific Management

John A Kark, MD[1], Richard J. Labotka, MD, MS[2], John W Gardner, DM, DrPH[*,3] and Frank T Ward, MD[4]

[1] Hematology, Thomas Jefferson University, Philadelphia, PA, USA,
[2] Univ. of Illinois at Chicago (M/C 856), Chicago, IL, USA,
[3] Epidemiology, Uniformed Services University of the Health Sciences, Bethesda, MD, USA,
[4] Hematology Oncology, Texas Oncology - Tyler, Tyler, TX, USA

Abstract 945

We previously reported EDU rates 37.5 fold higher among recruits with SCT than without Hb S in military entry basic training for 1977–1981(Kark JA et al. New Engl J Med 1987;317:781-87, revised). The National Collegiate Athletic Association now requires identification of SCT athletes to reduce this risk, based on a single institution opinion untested by controlled observation, that EDU's of college football players with SCT were due to explosive rhabdomyolysis from exertional sickling unrelated to muscle heat production. A mild syndrome of "exertional sickling" was postulated, consisting of myalgia with flaccid leg weakness, usually responding to O_2 inhalation within 30 minutes. Their plan is for non-medical personnel to use SCT screening results to assign SCT athletes training runs at reduced intensity and to pull them out of exercise for myalgia more often than non-Hb S athletes, practices chosen for safety but detrimental to their sports career (Gatorade Sports Science Institute Sports Science Exchange 2006;19:1-4). The cited football EDU's with SCT were actually associated with multiple risk factors for exertional heat illness (EHI), e.g. competitive activity with incomplete acclimation to heat, sustained hot weather, high body mass index, poor fitness, and/or heat-retaining clothing. We conducted a prospective controlled trial to reduce EDU rates for all recruits by better prevention of heat retention during exercise. We compared changes in EDU rates from 1977–1981 versus 1982–2001 at test recruit centers providing our intervention to changes at control centers using prior practice (Kark JA et al. Blood 1997; 90: 447a). The intervention included adjustment of observed water intake, exercise intensity, and work-rest cycle based upon the current heat index on site, early clinical assessment with rectal temperature for recruits who failed to keep up or became symptomatic, and early aggressive medical management of EHI. We did not alter training practices, exercise performance standards, or treatment by Hb type. O_2 was given only for signs of failure to adequately oxygenate arterial blood. EDU rates at test centers were reduced 22-fold for those with SCT (0 deaths/ 29,500 recruits vs 14/37,300, significant with

P=0.0003) and 2-fold for those without Hb S (6 deaths/1.8 million vs 15/2.05 million, not significant with P= 0.1), but were not substantially reduced at control centers for recruits with SCT (4 deaths/13,500 recruits vs 14/37,300, RR= 0.8, P=0.7) or for recruits without Hb S (6 deaths/ 1 million recruits vs 15/2.1 million, RR=0.85, P=0.8). Despite not screening for SCT, recruits at Army test centers had a significant reduction of mortality with SCT (0 deaths/21,000 recruits vs 7/17,100, RR= 0.06, P=0.002) and without Hb S (4/1.1 million recruits vs 15/2.1 million, RR= 0.3, P=.048). Reducing heat retention with exercise may have eliminated excess EDU with SCT. Survival with SCT did not require prevention of ordinary exertional sickling by measures targeted to SCT. Our intervention appeared to reduce EDU's for people without Hb S by nearly 50%. We also studied all cases of recruit EHI at one basic training center using our intervention during 1979–1991. Rates of non-fatal EHI were 32 cases/5010 recruits with SCT versus 1,834 cases/263,000 recruits without Hb S, a relative risk that was not significant (RR=0.92, P=0.7). The incidence of symptomatic muscle injury, ataxia, falls, hypotension, tachycardia, elevated biochemical markers for EHI, and non-focal encephalopathy were not significantly different for SCT vs Non Hb S recruits. We did not identify a specific non-fatal syndrome associated with SCT. The predominant mechanism of EDU with SCT might involve initial severe EHI from heat retention, subsequently exacerbated by complications related to Hb S, rather than initiation of tissue damage by exertional sickling in the absence of EHI. Proof for any form of exercise might require similar descriptions of all EHI and EDU cases among at least 27,000 people with SCT and population controls during at least one hot season of heavy exercise. Prevention of this morbidity and mortality for SCT probably does not require genetic screening in the absence of effective specific remedies. It may be more effective to provide appropriate prevention and management of exertional heat illness, including isolated rhabdomyolysis, to all exercising people regardless of hemoglobin type.

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About the Author

Ed Bouchette
Email Author
Call 412-263-3878

Ed Bouchette has worked for the Post-Gazette since 1983 and has covered the Steelers as his main beat since 1985.

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On the Steelers: Clark's call to play in Denver

Wednesday, September 16, 2009
By Ed Bouchette, Pittsburgh Post-Gazette

Chris McGrath/Getty Images
Safety Ryan Clark could sit out the Steelers' Nov. 9 game in Denver.

The Steelers have lost one starting safety for a while in Troy Polamalu, and they may lose another for one game.

Ryan Clark has not decided if he will play Nov. 9 in Denver. The last time he played there, he nearly died, and there could be a risk if he returns to exert himself in Denver's high altitude.

"I've taken the test, I've taken the pictures and done all those things, so I guess we'll just cross that bridge when we come to it," Clark said.

Clark knew he had the sickle-cell trait when he traveled with the Steelers to play the Broncos Oct. 21, 2007. He did not know the problems that would cause. The high altitude caused a reaction in his blood that attacked his spleen. Over the course of a few weeks, Clark lost his spleen, gall bladder and more than 30 pounds. He did not play again that season.

"The first speech I got from the doctors in the hospital was that my lungs had filled, my kidneys were dented and my stomach was closing," Clark told the Post-Gazette last year. "My spleen had gotten enlarged and infected and it died."

Clark, though, pulled through and had the best season of his seven years in the NFL last season when he started 17 games, including the postseason. The two games he missed had nothing to do with his sickle cell but with a shoulder injury.

But now Denver and that high altitude loom again, and Clark is uncertain what to do about it.

"We haven't really talked about it too much yet, so we're not at that point," he said this week.

Clark will hold a conference with his doctors and his coaches but the decision will be his. While he cannot lose another spleen and gall bladder, he also does not want to risk something else happening from a similar reaction in his blood. He feels healthy now; missing one game to stay that way might be a good trade-off for him.

"We have a bye week before it," Clark said of the Steelers' Nov. 1 weekend off. "That's when everything will be decided."

Offensive line kudos

Let's hear it for the Steelers' offensive line!

While the debate continues about who and what prevented the Steelers from producing much on the ground in their opener, there is little debate about another important aspect of the line play -- its pass protection.

The coaching staff came away from grading the videotape of that game with one consensus about the pass protection -- it was superb.

"I thought it was solid," coach Mike Tomlin declared yesterday.

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"We got hit a couple times in some blitz-game things and some no-huddle things, but, all in all, I thought, from a matchup standpoint, they did a nice job up front."

Ben Roethlisberger was sacked four times, and, if that becomes an average, he would break either a Steelers record with 64 sacks over the course of a 16-game season or some part of his body before he can get that far. But there is other evidence that the pass protection held up well, and at least one sack of minus-19 yards should be discounted because Roethlisberger held on far too long and ran far backward before the Titans got to him.

Here are some numbers the coaches discerned from the video: The combined quarterback hurries/pressures from that game came to 13 for the Titans on 47 pass attempts by Roethlisberger (43 passes plus four sacks). That compares with the Steelers' hurries/pressures of 19 on 36 attempts by Kerry Collins (35 passes plus one sack).

The Chicago Bears are not particularly good at getting to the quarterback. They ranked 29th in the league in sacks per pass play last season when they had only 28 sacks. That compares with the Steelers' 51. And now they've lost linebacker Brian Urlacher.

They did ring up four in their 21-15 opening loss at Green Bay, and Tomlin yesterday made them sound like the 1985 version of the Bears.

"They will pursue the ball relentlessly. They will rush Ben relentlessly. They'll chase. They'll do the things that you can't coach, the detail things."

Tomlin on running game ...

In so many words, Tomlin acknowledged that his running game did not work in the opener. And he will put more emphasis on it in practice this week.

"We've got to get better in that area as a football team. For one reason and one reason only, that increases our chances of winning. That's what we're about, putting ourselves in position to win. We acknowledge that if we continue along those lines from a run-game standpoint that doesn't help us in that regard, so that's why we're focusing on it."

Tomlin said that in his experience defenses key on the run more in the early part of the season to stop it.

"I believe, particularly in September football, people make a commitment to stopping the run, and it's easier to make the commitment in September when everybody feels good and you've got all the horses in the stable.

"I think over the course of the long haul, you see who's good at it week in and week out. It's usually tough sledding early in the football season. That's been my experience. That was my intent when I was a defensive play-caller, that, in order to be a good defense, it starts there -- making people one dimensional, making people struggle if they're committed to running the football."

For more on the Steelers, read the new blog, Ed Bouchette On the Steelers at post-gazette.com/plus. Ed Bouchette can be reached at ebouchette@post-gazette.com.

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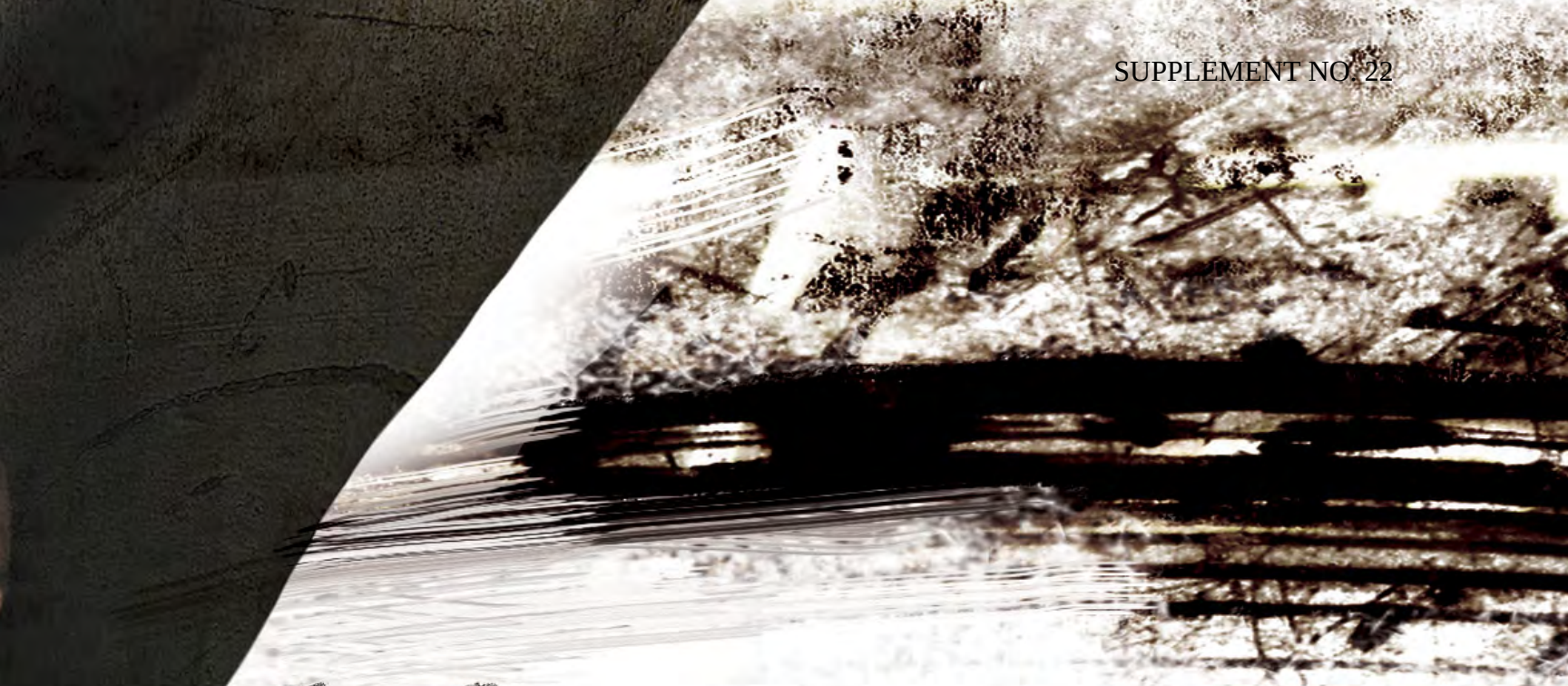

Monster within

{ By Brian Hendrickson }

Why me?

Lance **Kearse** searched nearly four years for an answer to that question, after his body turned on him in the fall of 2007 and **held him hostage**, demanding all his dreams as ransom. **The** search was painful and maddening, the distress still tainting the Eckerd basketball player's voice **as he recalls the day** his athletics career was forever altered.

It started with an intense cramp in his thigh, which steadily **crept throughout his body**, spreading to his hamstring, his abdomen and his back. He dropped **helpless** to the gym floor, locked in a 6-foot-6 full-body charley horse, too scared to move, listening to panicked voices call for 9-1-1, asking himself **if he was about to die**.

{ Photos by Matt Marriott }

Kearse spots an understanding of that pain and fear only in the eyes of other sickle cell trait carriers. The condition has emerged in high-level athletics like a boogeyman over the last decade, drawing attention from media that has described tragic tales of severe health consequences, unpredictable collapses and sudden deaths. While about 8 percent of African-Americans in the U.S. carry the trait – by far the largest percentage of carriers among ethnic groups – many are unaware because the condition is considered benign in the general population.

It becomes a threat in some carriers only when the body experiences extreme physical conditions, causing red blood cells to warp into sticky, half-moon shaped sickles less efficient at transporting oxygen or moving efficiently through the bloodstream. They can clog circulation, reducing oxygen to muscles and vital organs and eventually causing acute fatigue, cramping or even collapse.

The threat is alarming, but recognition has only recently begun to spread. Efforts to institute testing – including the mandatory testing among NCAA Division I institutions that began in 2010 and similar efforts being considered by Divisions II and III – are attempting to improve awareness, which the medical community views as the greatest form of prevention. But many athletes don't learn about their condition until they experience a severe incident.

Kearse was among them.

After reading about tragic incidents, he joined those who feared the trait's dangers. His arresting fatigue, burning muscles and full-body cramps nearly chased him from the court, where his personal "monster" always lurked. But Kearse has emerged as an example of the benefits of awareness, proper treatment and education. His playing time has returned to normal. He no longer fears the burns and cramps, or questions if he's risking his life. Closely monitoring his activity, nutrition and hydration eased concerns. And a fifth year of eligibility granted last summer by the NCAA will give him a chance to enjoy his first full season as a normal, productive player.

Kearse no longer tortures himself with that open-ended question: "Why me?"

His journey – at times terrifying, frustrating, but eventually heartening – ultimately provided a purpose, and an answer.

FROM SUPERMAN TO SIDELINED

Kearse noticed something wasn't right during his all-state career at Fort Myers High in Florida. But he never took the signs seriously.

He worked out daily, yet was the only kid in the postgame locker room exhausted and cramping while teammates laughed and celebrated. His thigh muscles would tire in games, and his combustible skills would flare out. Kearse wondered why his conditioning couldn't match his teammates', but his size and athleticism provided such an advantage that those deficiencies were easily overlooked.

In a state quarterfinal game against Dillard High, Kearse scored 17 points, grabbed 20 rebounds, blocked seven shots and had six steals. A Scout.com report described him as a "big-time athlete" and "aggressive and relentless," while his Nike Team Florida AAU coach Tom Topping gushed, "He's one of the best athletes we've ever had in our program."

It was a mighty statement. Kearse's teammates included Chandler Parsons, a 6-foot-10 forward who later earned SEC Player of the Year honors at Florida and became a second-round NBA draft selection. There was also Nick Calathes, an all-SEC first-team guard at Florida who went on to play professionally in Greece, and Florida State standout Solomon Alabi, currently a Toronto Raptor. Yet after watching lob passes routinely thrown to Kearse for easy dunks, Team Florida assistant coach Ed Hall would look at the No. 5 on the muscular guard's jersey and jokingly question if it was actually an "S."

"He's Superman," Hall would say. "We became accustomed to him doing unbelievable athletic plays."

Expectations were high when Kearse committed to VCU in 2006 to form a powerful incoming class that included eventual NBA first-round draft selection Larry Sanders – a group that as seniors guided VCU to the Final Four last spring. But as Kearse started pushing his body during preseason conditioning to meet the rigors of college basketball, he found himself struggling to keep pace. He failed conditioning benchmarks and worked to catch up with extra sessions.

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terrifying, frustrating, but eventually heartening – ultimately provided a purpose, and an answer.

Gift

Kearse busted through the wall with a painful burst, and found his personal monster lurking on the other side.

As his freshman season drew close, Kearse stepped into a hot gymnasium for his third workout in a single day, trying to close the gap in his conditioning. As the training dragged on, Kearse felt his legs tire. The muscles felt lifeless. His body seemed sluggish and heavy, like he was carrying another person. He pushed on, conceding to the athletics culture that drives athletes to push through the mental barriers urging them to stop.

It's a mental issue! It's a block! You've got to break down that barrier!

So Kearse pushed as the lifeless feeling in his legs changed to a raging burn. The muscle fibers ignited with tiny cramps, as if he were pushing through the final reps of an intense weight-training exercise.

Break down that wall!

He pressed for another 10 minutes, pushing a towel up and down the court, ignoring his instincts to stop.

Don't give in!

Kearse busted through the wall with a painful burst, and found his personal monster lurking on the other side.

He dropped to the court, quadriceps searing with a horrific cramp. He reached to massage it but experienced something he'd never encountered: The cramp kept spreading. It moved from his thigh into his hamstring, then his glutes. As he reached around, more muscles joined in – his abdomen, his back, his chest. Kearse lay on the court in agony. He was fed Gatorade, but it didn't help. Scared to move, extremities growing cold, an ambulance en route, one thought pierced the pain.

"I was scared for my life," he said. "I thought I was going to die."

The pain persisted to the hospital. Nurses inserted an IV and cautioned that his creatine kinase levels were wildly elevated, signaling the dangerous breakdown of muscle fibers called rhabdomyolysis. The process releases the fiber contents into the bloodstream, which can cause kidney damage and intense pain. The incident kept him out of workouts for a month.

But sickle cell trait was never mentioned.

Medical experts are not shocked that reaching his final diagnosis was a lengthy process, though. Awareness of sickle cell trait's dangers, they say, is still developing. And because it affects a small, niche population – typically military recruits and high-level athletes – it may not be the first option to explain symptoms that can also be attributed to poor conditioning or inadequate hydration.

Like concussions, which are now being treated at all levels of sport – from high school to the professional ranks – as serious conditions after years of receiving "shake it off" diagnoses, sickle cell trait is moving through a process in which the medical community first must recognize its potential danger, then actively work to catch and treat it.

"As with any medical condition or anything in medicine, it takes a while from the initial description to where people are recognizing it and putting it into practice," said Kevin Harris, a cardiologist at the Minneapolis Heart Insti-

tute who has studied sudden deaths in athletes related to sickle cell trait. "It takes a while, and I think that's where we're at with this."

Arnold Ramirez, the team doctor at Eckerd who diagnosed Kearse with sickle cell trait, said his experience treating the condition has revealed symptoms that are stealthy and complex. The trait presents uniquely mild or severe symptoms for each individual, depending on physiology.

So where one sickle cell trait carrier may experience uncomfortable light-headedness or weakness, others like Kearse can experience debilitating cramping in extremities, making diagnosis tricky.

"It could be any of a number of things that you're rifling through your head," said Chris Gillespie, director of athletics training education at Samford and member of a national sickle cell trait task force. "But if you know they're sickle cell trait and it's in your medical file on that athlete, then it's one of the first things you would obviously know about the situation. … Otherwise you're guessing."

It's possible Kearse's drawn-out diagnosis resulted from that lack of awareness. According to Kearse, several other options were explored: psychology, biopsies, blood tests and metabolic exams among them. He said one theory pointed to a possible myopathy – a neurological condition that causes muscle weakness and can produce cramping. But it didn't answer all the questions.

The lack of solutions was exasperating. Kearse grew frustrated and angry. He watched teammates improving while he was stuck in neutral. After a second full-body cramping episode sent him back to the hospital, Kearse became unwilling to put himself at risk, afraid he might die if he tried.

"I was scared for my life. I thought I was going to die."

A teammate nicknamed Kearse "Half-Man, Half-Unbelievable" because he could cramp one minute, throw down a dunk the next, then cramp up again shortly after. The health troubles led to inconsistent playing time and cast questions about toughness upon him. Kearse's self-image was battered. The competing pressures were maddening. Teammates called him "Tripolar" as his emotions raced the spectrum. When a physician suggested midway through his sophomore year that he should quit basketball for his own protection, Kearse holed up in his room for two days and cried.

"I realized that I may not play basketball again because my body is that bad, and I know that my body is that bad," Kearse said. "When I'm out there on the court, I feel like I'm playing against my opponents, and myself."

REGAINING CONTROL

When Ed Hall phoned during the winter of 2009 with the news that Kearse would be transferring, Eckerd coach Tom Ryan figured

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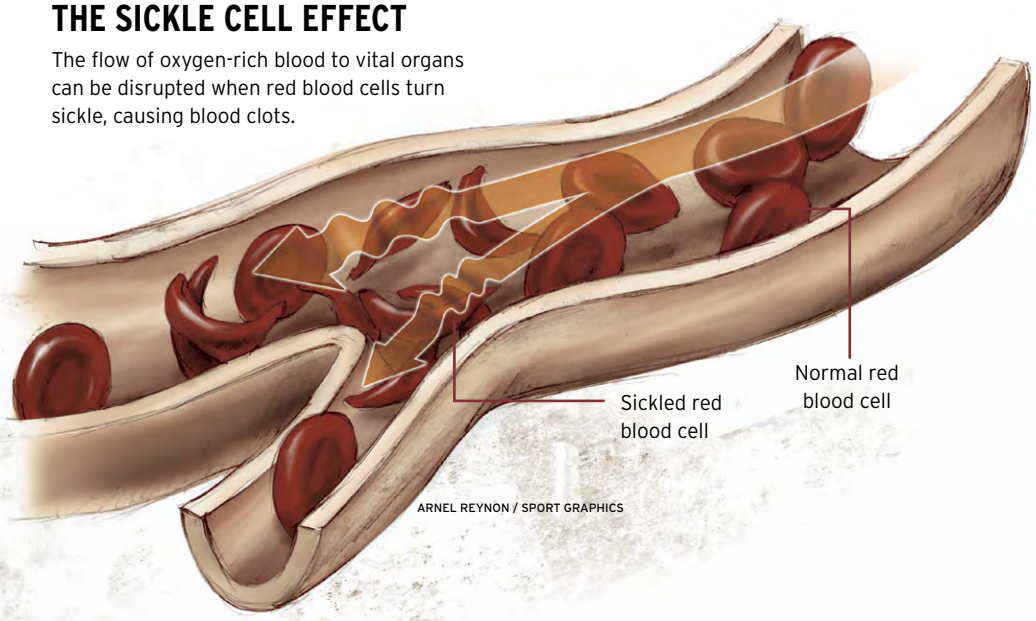
THE SICKLE CELL EFFECT

The flow of oxygen-rich blood to vital organs can be disrupted when red blood cells turn sickle, causing blood clots.



ARNEL REYNON / SPORT GRAPHICS

SIMILAR NAMES, DIFFERENT CONCERNS

Medical experts say sickle cell trait is often misunderstood and confused with its more serious cousin, sickle cell disease. But while the conditions are related, the health concerns for each are vastly different.

Sickle cell disease is caused by the inheritance of two genes for sickle hemoglobin, the oxygen-carrying component in red blood cells. The genes cause the cells to warp into stiff, sticky crescent shapes, which can block blood flow in limbs and organs, causing pain, infections and organ damage.

Sickle cell trait carriers inherit one sickle hemoglobin gene and one normal gene. Their red blood cells retain a normal round shape and flow freely through the circulatory system. In the general population, the condition is not considered life threatening.

But when some carriers of the trait experience extreme physical conditions – such as in basic-training exercises with the military or in harsh conditioning for high-level athletics – the cells can sickle and begin disrupting the flow of blood and oxygen. In some cases it can induce dangerous results, including intense cramping, loss of consciousness and even sudden death.

The potential hazards of sickle cell trait have gained awareness in recent years, but Dr. Kwaku Ohene-Frempong, director of the Comprehensive Sickle Cell Center and a sickle cell trait carrier who previously captained the Yale track team, cautions that the potential dangers should not be cast in a one-size-fits-all mold that can attach a bias against its carriers.

He points to a study of military recruits performed in the mid-1980s that showed only 1 in 3,200 trait carriers died while performing similar exercises. Ohene-Frempong said the study suggests that not all trait carriers may experience health problems from intense exercise and raises questions about whether additional factors, such as inherited defects, may also play a role in athlete deaths.

Ohene-Frempong said further studies must be conducted to fully understand the dangers related to sickle cell trait.

"We can't ignore it," Ohene-Frempong said. "Nobody should go to the athletic field as a healthy young person … and suffer a serious injury that is not an accident, or die because of something they carry with them and they didn't know or they weren't aware of. It is a serious matter. But the signs of it need to be looked at very carefully."

– Brian Hendrickson

SHUTTERSTOCK

EARLY TESTS FOR SICKLE CELL ARE LOST IN THE SYSTEM

The NCAA's decision to test all athletes for sickle cell trait beginning in 2010 has been praised by many medical experts as an important step toward preventing crisis.

But ironically, most current student-athletes already have had the test and didn't know it.

All 50 states now test for sickle cell trait as part of standard screening of newborns. The exams check for rare diseases that can put infants' health at immediate risk, including intolerance to amino acids or the more serious (and rare) sickle cell disease. But parents often are not informed of a positive sickle cell trait test for various reasons.

The parents may not have a pediatrician at the time of birth, leaving the test work to remain at the birth hospital where it never reaches a doctor. Family may relocate, but medical paperwork may not follow. Parents also may not be notified of a positive test because it is often considered a benign condition in the general population. Only niche groups like high-level athletes and military recruits tend to experience its effects.

The result: Important health information slips through life's cracks.

"What a waste to have this data and to not be able to find the data," said Dr. Stephen Rice, a sports medicine pediatrician at Jersey Shore University Medical Center. "What really needs to happen is for us to take advantage of the fact that this test was done."

Dr. Kwaku Ohene-Frempong, director of the Comprehensive Sickle Cell Center, said states don't make it a priority to notify patients of positive sickle cell trait results. Because the trait is generally considered non-life threatening, the expense and effort of informing and counseling patients is rarely supported, he said.

But a positive test carries importance beyond athletics. It can also impact reproductive decisions, where sickle cell trait carriers might need to consider the possibility of bearing a child with sickle cell disease. So Ohene-Frempong and Rice believe the medical community should work together to ensure that newborn tests can be retrieved by doctors and prevent the data from being wasted.

– Brian Hendrickson

it was a dead end. But when Kearse arranged for an official visit several weeks later, Ryan started to understand there was a reason for his interest in the Division II school. It was a two-hour drive from Kearse's Fort Myers, Fla., home and close to his support network of family and coaches – people who understood him and knew something wasn't right. Kearse needed that support because he still didn't know what was affecting his health.

Ryan and Ramirez could see the psychological toll the unanswered questions placed on Kearse. It was apparent that his frustration led to clashes with his previous coaches. It was also apparent that he'd become spooked by his own body, driving a panic that at times was paralyzing. Kearse was tired of wondering why it was fighting him.

"He was screaming for help," Ryan said. "He wanted somebody to tell him what the hell was going on with his body."

"He was screaming for help. He wanted somebody to tell him what the hell was going on with his body."

– Tom Ryan,
Eckerd head basketball coach

Eckerd's staff didn't know if they could help. But they resolved to try.

Ramirez and team athletic trainer Travis Irwin put Kearse through a torrent of tests during his official visit. He ran on a treadmill and had numerous vials of blood drawn. They checked for rare disorders, performed metabolic tests, looked for muscle myopathies, congenital enzymes and neurological conditions. They checked for any possible diagnoses that hadn't been explored. Sickle cell trait, Ramirez reasoned, was one possibility, and a test that was separate from standard blood panels. He added it to the list.

The results arrived two weeks later. Sickle cell trait was the only abnormality found, and the variety Kearse carried was believed to be severe. His monster finally had a name.

But that identity scared as much as it resolved.

His mother, Ida Kearse, was wrought with guilt. Nobody in her family had sickle cell trait, but Lance's father lived in Canada and had never been tested for the condition. Ida searched her mind, recalling Lance's birth, hospital stays and doctor's visits. She couldn't recall sickle cell trait ever being mentioned.

"I was just so down and bad on myself," Ida said. "Could I have done something?"

The news locked Kearse in a panic. He read the horror stories about athletes collapsing and dying, their conditions discovered only during the autopsy. But one particular case raised goose bumps: the story of football player Ereck Plancher. The UCF redshirt freshman collapsed and died during a spring conditioning workout a year earlier. Sickle cell trait was later blamed.

Memories instantly flashed into Kearse's mind, carrying him back to a recruiting visit to UCF's football team before his senior year of high school. Then-coach George O'Leary eyeballed Kearse's impressive physique and athleticism. "If you play football this year," O'Leary's haunting words echoed in Kearse's memory, "there's a good chance I'll offer you a scholarship."

The ironic circumstance consumed Kearse. He often feared for his life, but the diagnosis transformed possibility into reality.

"I was faced with a monster that I knew nothing about," Kearse said. "It was extraterrestrial. It was like, 'What is this that's going on?' I wanted no part with it."

Kearse tried convincing himself that life offered more than basketball. He could graduate with his business management degree. He could be successful without sports. But that talk was fear overriding desire. Basketball was an essential part of his life. It turned a shy, awkward kid into a confident, joking, social young adult. He couldn't simply let it go.

Ramirez assured Kearse that he would be safe if they carefully controlled his water intake and diet. Safely managing the condition wouldn't be difficult. But Kearse also had psychological scars to heal. Stepping onto a basketball court felt like entering a shootout unarmed. He had trouble catching his breath; anxiety paralyzed him.

I could die doing this.

It took a year of experimenting to make Kearse comfortable again. He had to learn to pull back and become a jump shooter rather than attack the basket with relentless aggression. He had to learn to take himself out of the game when his muscles started burning rather than push himself to the point of cramping, which

Once the Eckerd staff diagnosed Kearse with sickle cell trait, they were able to devise a training regimen that helped him manage the condition and experience a normal campus life, which includes being able to play basketball without fear of an incident.

could keep him sidelined for days afterward. In essence, Kearse had to relearn the game.

Ryan, too, had to restructure plans, devising a rotation in which Kearse would play for three minutes, then rest. That management of time and effort could assure that Kearse wouldn't overextend himself.

The medical staff complemented those efforts with hydration strategies and strict monitoring of his body chemistry. Before each game, Kearse receives intravenous fluids, ensuring that he will be fully hydrated by tipoff. In addition, an oxygen tank sits on the sideline for Kearse to draw from during breaks.

The strategy confronts the sickle cell trait from two ends: Hydration helps his blood flow smoothly; the oxygen helps his sickling cells recover their normal shape. The combination is like sending a delivery truck through city streets: More oxygen allows the truck to deliver more, and better hydration reduces the traffic it has to negotiate.

The process was slow, but as Kearse learned to trust Eckerd's staff and to read the messages sent by his body, he took steady steps toward a normal game. His playing time increased, from 17 minutes his first season to 20, then 23 and finally close to 30 by his senior season's close. In his final six games, Kearse averaged 16.3 points, shot 55 percent from the field, and played 26.8 minutes – a monumental leap forward. A year earlier, Kearse couldn't last half the game.

For the first time in four years, he was becoming a most efficient player.

MOVING ON

Kearse is now his body's foremost expert, driven to master sickle cell trait now that he knows it can be controlled.

It requires total lifestyle awareness. He carefully manages his diet; a good meal includes tuna and a protein shake. He won't drink alcohol or caffeine, both of which dehydrate him. He laughs more now and has let go of the frustrations experienced at his previous school ("It's not worth it because they didn't know; I didn't know," he says). He speaks casually and informatively about his condition. It no longer looms like a monster. Instead, he reminds himself of an old wisdom.

"Without failure, there is no progress," he says. "I live by that."

His progress was recognized earlier this summer. While Kearse worked out in the gym, Ryan walked in to share a text message. He'd just received word about the school's request to the NCAA to grant Kearse a fifth year of eligibility. Ryan played solemn: "I'm sorry," he said, handing Kearse the phone.

The request had been accepted.

"Coach, why are you playing with me like that?" Kearse jabbed back before the two celebrated with a laugh. It felt like victory for the entire staff: for Kearse overcoming fear and severe health scares; for the coaches who took on the challenge; and for the medical staff that devised the means to treat his condition.

Healing has come physically and psychologically. Kearse no longer fears his body. In recent months, he's become comfortable discussing his experience and advocating for the importance of awareness as the surest form of prevention. He wants others to know sickle cell trait and normal life can dance together.

It's in that renewed vitality that Kearse found the answer to that exasperating question: *Why me?*

"I know why," he says with determination. "I can help other people. I can help so many people."

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Review

Hemorheology and exercise: effects of warm environments and potential consequences for sickle cell trait carriers

Philippe Connes

Laboratoire ACTES, Département de Physiologie, Université des Antilles et de la Guyane, Campus de Fouillole, Guadeloupe, French West Indies
Corresponding author: Philippe Connes, Laboratoire ACTES, Département de Physiologie, Université des Antilles et de la Guyane, Campus de Fouillole, 97159 Pointe à Pitre, Guadeloupe, French West Indies. E-mail: pconnes@yahoo.fr

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The relationship between blood rheology and hemodynamics is complex, and the following review presents several aspects of exercise hemorheology, with the focus on both cycling and running as exercise. The scientific literature assessing the effects of warm environment on exercise hemorheology is presented and the potential consequences for a specific population, sickle cell trait carriers, are discussed.

Background: the importance of hemorheology in hemodynamics

The French physician Poiseuille (1835) was the first to study the principles of fluid flow in cylindrical tubes in order to understand blood circulation. According to the law derived from this work, vascular geometry is the most important factor of blood flow resistance and, consequently, the impact of blood viscosity and other hemorheological factors have often been ignored. Poiseuille's law has been used extensively in scientific and medical research, and critical elements of blood rheology in cardiovascular physiology continue to be studied. Blood interacts directly with endothelium, and hemorheological properties may be strong modulators of vascular geometry (Baskurt & Meiselman, 2008; Salazar Vazquez et al., 2008). For example, it was demonstrated that a rise in plasma viscosity may cause a counterintuitive and surprising decrease in vascular resistance (Tsai et al., 2005). In fact, the increase in viscosity causes a greater shear stress applied at the endothelium level, which stimulates the production of nitric oxide by the endothelial nitric oxide synthase (e-NOs) and results in compensatory vasodilatation. Baskurt et al. (2004) recently reported an effect of red blood cell (RBC) aggregation on the vasodilatory function of muscular microcirculation. They described depressed e-NOs activity and decreased NO-mediated relaxation of arterioles when RBC aggregation was increased. This effect is explained by the enhanced axial migration of cellular elements of blood, which reduces the frictional forces on the endothelial cells and down-regulates NO synthesis.

In the microcirculation, blood flow and adequate oxygen delivery to tissue are mainly dependent on RBC deformability (Kaul et al., 2008; Connes et al., 2009a). Parthasarathi and Lipowsky (1999) studied the effect of reduced RBC deformability on microvessel recruitment attendant to a reduction in tissue oxygen pressure (PO_2) in rat cremaster muscle. These authors found that impaired RBC deformability may adversely affect capillary recruitment and the physiological mechanisms that ensure adequate delivery of oxygen to the tissue. Studies on the rheological behavior of RBCs in the capillary network clearly demonstrated that capillary flux and velocity are strongly dependent on the ability of RBCs to deform in the capillaries. A decrease in RBC deformability adversely affects capillary perfusion (Driessen et al., 1980). Baskurt and Meiselman (2007) demonstrated that a 15–17% reduction in RBC deformability can cause a 75% rise in flow resistance in isolated–perfused rat hind limbs when vasoactivity is intact and >250% when vasoactivity is inhibited. Recently, experimental data obtained by Kaul et al. (2008) strongly suggested that when RBC deformability is reduced and RBC adherence to endothelium is increased, as is often the case in an inflammatory context, the flow resistance may be further increased.

Exercise hemorheology in a temperate environment

Cycling exercise

Hemorheology, in response to exercise conducted in thermo-neutral environments, has been extensively studied. Cycling exercise usually increases blood viscosity by 15–20% (Brun et al., 1993, 1998), due to essentially the increase in plasma viscosity (+5–10%) and hematocrit (+3–4 U) (Brun et al., 1998). The rise in blood viscosity has not always been interpreted as a beneficial adaptation for hemodynamics but, as discussed above, recent findings on the possible beneficial effects of hyperviscosity on vascular geometry suggest that the increase in blood viscosity (at a given shear rate) in association with the increase in blood flow during exercise is a required condition to optimally stimulate the endothelium for vasodilation (Connes et al., 2009a; Connes, 2009b). In addition, cycling exercise changes the deformability of RBCs, with the direction of change (impairment or improvement) depending on the type of exercise (maximal or submaximal) and the population (hypoxemic athletes, trained or untrained subjects, etc.) (Brun et al., 1998; Connes et al., 2004). For example, Connes et al. (2004) demonstrated that RBC deformability in endurance-trained subjects increases (+6–7%) in response to maximal exercise, whereas the opposite is usually found in less-trained populations (Brun et al., 2007). Decreased RBC deformability contributes to the increase in blood viscosity, whereas improved deformability attenuates the effects of increased plasma viscosity and hematocrit on blood viscosity. The rise in RBC deformability during exercise may be viewed as beneficial for microcirculatory blood flow and adequate capillary perfusion. We recently reported a significant negative correlation between RBC deformability and systemic vascular resistance during exercise (Fig. 1(a)), as well as significant positive correlations between RBC deformability and the arterio-venous oxygen content difference (Fig. 1(b)) and between RBC deformability and oxygen consumption (Connes et al., 2009a). Although the correlations were not very high, these data suggest that RBC deformability affects aerobic performance via oxygen delivery to tissues.

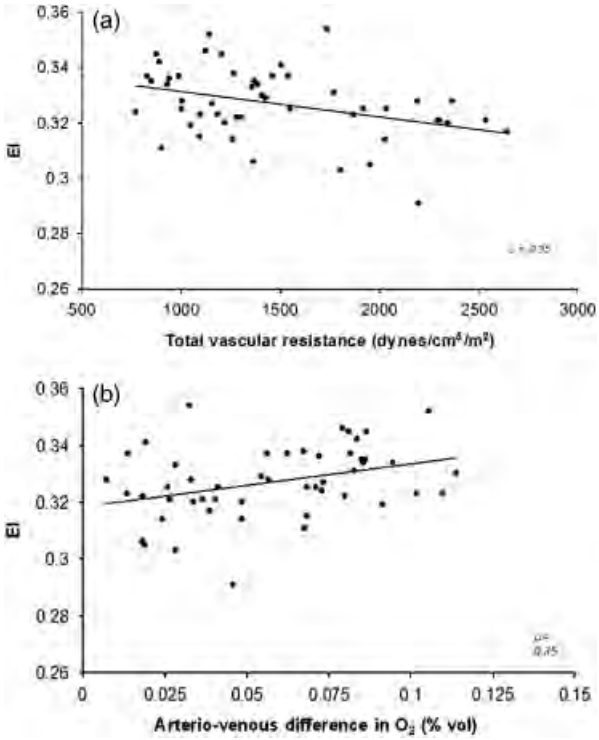

Fig. 1. Relationship between red blood cell (RBC) deformability and the total vascular resistance ($P<0.01$) (a). Relationship between RBC deformability and the arterio-venous difference in oxygen ($P<0.01$) (b). After a resting period, sportsmen underwent a submaximal cycling exercise protocol consisting of three successive 9-min levels at 50, 100, and 150 W. The RBC deformability (EI = Elongation Index) was determined by ecktacytometry at 3 Pa. Total vascular resistance was calculated by dividing the mean arterial pressure by the cardiac output, the latter being measured by transthoracic impedancemetry. The arterio-venous difference in oxygen was calculated according to the Fick equation by dividing the oxygen consumption by the cardiac output. Adapted from Connes et al. (2009a).

Running exercise

The hemorheological adaptations observed during running differ from those observed during cycling. Most of the prolonged running exercise performed in an outdoor environment, such as marathons, does not alter blood viscosity, plasma viscosity or hematocrit (Galea & Davidson, 1985; Neuhaus et al., 1992; Neuhaus & Gaehtgens, 1994). Senay and Pivarnik (1985) proposed that blood and plasma volumes are usually well maintained during such exercises, unless athletes lose a considerable amount of water (around 4–5% of total body weight). The hemorheological stability observed in the studies of Neuhaus et al. (1992) and Neuhaus and Gaehtgens (1994) was probably related to the *ad libitum* fluid replacement that is usual at outdoor events. In addition, the footstrike hemolysis that occurs during running may also be responsible for the lack of change in the hemorheological profile observed in these studies. The most rigid RBCs are destroyed during footstrike hemolysis, and the change in hematocrit is therefore partially or totally blunted.

Other studies have investigated the rheological properties of RBCs during running. Using a filtration technique, Galea and Davidson (1985) and Reinhart et al. (1983) reported a decrease in RBC filtrability after a marathon (−16.3 and −35.8% for women and men, respectively) and a 100 km race (−9.3%), respectively. However, Neuhaus et al. (1992) found

no change in RBC filterability after a marathon. Wood et al. (1991), using viscometry methods, suspected an increase in RBC deformability after a 48 km mountain run. These discrepancies have not yet been fully explained but they might be due to the great variability in (1) the methods used (i.e. filtration techniques, rotational viscometry, and ecktacytometry) and (2) the time delay for measurement after sampling [ranging from a few minutes after sampling (Connes et al., 2004) to 1 day later (Oostenbrug et al., 1997)].

Exercise hemorheology in a hot environment

Hot environments may cause greater dehydration than thermo-neutral environments, and it can thus be hypothesized that the hemorheological changes during exercise will be greater in hot conditions than in temperate conditions. Kareva and Gushchin (2009) examined the effects of thermal loading on hemorheological parameters at rest: they found that the changes depend on the duration of the exposure to thermal loading and that 30 min at 43 °C increases the hematocrit by 8%, decreases RBC deformability by 15%, and reduces RBC aggregation. Few studies have investigated the effects of thermal loading on blood rheology during exercise and it is not known whether the changes usually observed in thermo-neutral conditions are amplified. In an initial study (Tripette J, et al., 2010), nine sportsmen performed a 10 km running trial in the tropical climate of Guadeloupe (temperature: 28–30 °C; humidity: 65%). Although all the subjects were authorized to drink *ad libitum* during the race, they lost weight (-1.42 ± 0.15 kg; around 2% of total body weight). Despite the dehydration, there was no observed change in hematocrit or blood viscosity in response to the running race. We also studied RBC deformability with ektacytometry, one of the most accurate techniques available. In contrast to the results obtained by Reinhart et al. (1983) and Wood et al. (1991), but in agreement with the results reported by Neuhaus et al. (1992), Tripette et al. (2010) observed no significant change in RBC deformability, whatever the shear stress used for measurement. This finding was reinforced by the observation of no change in RBC morphology post-exercise. Moreover, the cumulative effects of a running race and tropical climate did not affect RBC aggregation. These results support previous reports that running does not greatly affect blood rheology, and it appears that hot or warm environments are not an additional strain for blood rheology during such exercise, even when dehydration occurs. The plasma shifts between intravascular and extravascular spaces may play a role in this equilibrium (El-Sayed & Omar, 2008).

In another recent study (Tripette et al., 2010), sportsmen performed a 40-min submaximal cycling exercise at moderate intensity (55% of maximal aerobic power) in two conditions: with and without *ad libitum* drinking allowed. As for the previous studies conducted in temperate conditions (Brun et al., 1998; Ernst et al., 1991), blood viscosity increased with exercise and returned to baseline within the 2 h of recovery with both conditions of fluid replacement. Perhaps, the most surprising outcome was the lack of difference observed between the hydration and dehydration conditions. These studies were conducted in conformity with our institutional ethics policy, and the sportsmen were not exercised to their extreme hydration/dehydration status limits (they were asked to drink when they felt they needed to drink).

The case of sickle cell trait (SCT) carriers

We extended the study on the effects of "hydration–dehydration on blood rheology" to a special population, SCT carriers, and it appeared that hydration was beneficial for them because dehydration elevated blood viscosity during the recovery (Tripette et al., 2010). This suggests that environmental conditions and hydration status may have different effects on blood rheology, depending on the population. This is of particular importance for SCT carriers as they may be at risk for heat illness and related vaso-occlusive epidoses during exercise that can lead to sudden death (Connes et al., 2007).

Recently, Hedreville et al. (2009) reported the case of a cyclist with SCT who developed a massive central retinal vein occlusion (CRVO) after a strenuous cycling race of 138 km conducted in hot temperature (35 °C) and elevated humidity (60%). Anecdotally, the patient felt very thirsty, not only during the race but also for several hours after the race was completed. The CRVO rapidly worsened into neovascular glaucoma. The presence of four thrombi in the left atrium at the time of the accident suggested hypercoagulability. In addition, the resting RBC rigidity of the patient was elevated. This case study and the cases of exercise-related sudden death reported in several exercising SCT carriers (Kark & Ward, 1994) implicate environmental conditions as a factor in the occurrence of clinical complications in this population. We hypothesize that the sub-clinical alterations in RBC rheological properties sometimes observed in SCT carriers, in association with marked dehydration and exposure to a very hot environment, may promote vaso-occlusive accidents in exercising SCT carriers. SCT carriers should probably drink more than sportsmen without hemoglobinopathy (i.e. more than the level defined by thirst), but they

should be made aware of the risk of hyponatremia with excessive hydration during ultraendurance exercise.

In summary, blood rheology is changed by cycling exercise but not by running exercise. RBC deformability, hematocrit, and plasma and blood viscosities are changed in cycling exercise, but RBC deformability is affected differently in athletes and non-athletes at maximal exercise. This is an important point because RBC deformability has a mild effect on systemic vascular resistance and the arterio-venous oxygen difference, which may affect aerobic performance. When a hot environment is added to the above exercise modes, there is no clear additional effect. The few available data indicate no changes in RBC deformability, hematocrit, blood viscosity or plasma viscosity during running exercise in a tropical environment, whereas increased viscosity has been observed during cycling exercise in a tropical environment. Very few studies have investigated the effects of hydration on blood rheology, but it seems that subjects with SCT are more sensitive, from a hemorheological point of view, to hydration than the normal population.

Key words: thermal loading, blood viscosity, cycling, running, hemoglobinopathy, deformability, aggregation.

Conflicts of interest: The author has no potential conflicts of interest.

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Exertional Sickling Deaths in Army Recruits With Sickle Cell Trait

LTC Kenneth Ferster, AN USA; E. Randy Eichner, MD†*

ABSTRACT Exertional sickling from sickle cell trait (SCT) can pose a grave risk for some military recruits and is a troubling cause of death in college athletes. We report the cases of two U.S. Army recruits with undetected SCT who collapsed and soon died from metabolic complications of exertional sickling as they struggled to finish in time the 2-mile run of the Army Physical Fitness Test, having failed this test on prior attempts. These cases are similar to other military cases and to recent sickling deaths in college track and football. Research shows how and why, in the face of SCT, during intense exercise bouts, sickle cells can quickly form and lead to fulminant rhabdomyolysis that can be fatal. Increasing evidence suggests that, in the military and in sports, the proximate trigger for most cases of fatal sickling collapse is intensity. If this hypothesis is correct, that sickling collapse is an intensity syndrome, it raises vital questions about how best to train military recruits with SCT.

INTRODUCTION

The first report of sickle cell trait (SCT) as a possible cause of sudden death came from the U.S. military in 1970 when it was noted that during one year of Army basic combat training (BCT) at a base in El Paso, four of 4,000 (0.1%) African-American recruits died suddenly. All four had SCT. One died of coronary heart disease, but the other three deaths seemed to fit a pattern: collapse during running, hypotension, metabolic acidosis, hyperkalemia, disseminated intravascular coagulation, oliguria, and "sudden" death 8 to 25 hours after the collapse.[1]

The pattern was clarified by the next four cases reported. From 1970 to 1974, at two large military installations, two recruits and two cadets were hospitalized for exertional rhabdomyolysis, disseminated intravascular coagulation, and acute myoglobinuric renal failure. Three collapsed after a run, the fourth had severe muscle cramping and swelling after a run. All needed early renal dialysis; one died of hyperkalemia. All four had SCT.[2]

These and other reports led to a benchmark epidemiological study by the U.S. military that strengthened the link between SCT and exercise-related sudden death. Of all deaths that occurred among 2 million recruits during BCT in the U.S. Armed Forces over the 5 years from 1977 through 1981, the risk of unexplained sudden death in black recruits with SCT was 40 times higher than in all other recruits.[3] In follow-up analysis, exercise-related death in black recruits with SCT was 30 times more common than in black recruits without SCT. It seemed that exertional heat illness (EHI) was often the initiating culprit.[4]

The conclusion that EHI was a possible trigger led to a 10-year intervention study (1982–1991) to test the hypothesis that better prevention of EHI would reduce deaths for all recruits and reduce excess deaths with SCT. Drill sergeants monitored wet-bulb globe temperature, decreased exercise intensity and increased rest cycles and hydration as wet-bulb globe temperature rose, and responded early to any sign of struggling. This program did not require SCT screening. This study, which has been presented and discussed but not published, showed that the intervention reduced or eliminated excess mortality for SCT recruits.[5] As a result, the Army stopped screening recruits for SCT in 1996, stating that most SCT-related deaths could be avoided by preventing EHI and that screening for a trait that rarely, if ever, resulted in death under normal circumstances was not cost-effective.[6]

Yet exercise-related deaths continue in Army recruits with SCT, for example, six from 1992 to 2001.[6] We report two recent such deaths and compare them to similar deaths in college football and track. These deaths raise questions on how best to safely train military recruits with SCT. The two cases we report suggest that intense exercise per se, without EHI, can cause fatal exertional sickling in Army recruits.

CASE REPORTS

Case 1

In October 2008, a 21-year-old African-American female recruit ran the 2-mile run of the Army Physical Fitness Test (APFT). She was in her ninth week of BCT and had failed the APFT run four times before. On her fourth try, she finished the 2 miles, but not in time. She began her fifth run at about 7:00 a.m. The temperature was 64ºF, relative humidity 96% (recent rain), and heat category 1. She had returned from a 4-day field training exercise in the morning of the prior day. She was able to shower, eat a hot meal, and slept in an air-conditioned barracks that night. Having passed all the other rigorous and demanding physical and mental challenges during BCT, this recruit would be considered to be in excellent physical condition. She was not on phenothiazines or anticholinergics and had no recent illness. On her fifth try, urged on by her drill sergeant running with her, she began to struggle on the final lap, fell to her knees, got up, and collapsed again, near the finish. She was carried to the side of the track. She was sitting up,

*Department of Soldier Care, Moncrief Army Community Hospital, 4555 Stuart Street, Fort Jackson, SC 29207.

†University of Oklahoma Health Sciences Center, 1100 N. Lindsay Avenue, Oklahoma City, OK 73104-5036.

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her eyes open, breathing. Emergency medical services (EMS) arrived within 7 minutes at 7:27 a.m. Paramedics said she was able to sit up and was conscious, complaining of leg weakness. The lower extremities were rigid from the knees down. Soon she became unresponsive and apneic. Cardiac monitor showed pulseless electrical activity. Paramedics began cardiopulmonary resuscitation (CPR) and transported her to a local hospital, arriving 13 minutes later at 7:49 a.m. She arrived in pulseless electrical activity. Despite continued CPR in the emergency room, she soon developed asystole and was pronounced dead at 8:20 a.m. At autopsy, sickling was seen in several organs and she was found to have SCT. Her parents did not know she had SCT and doubted that whether she knew about it. The cause of death was "cardiac arrhythmia of undetermined etiology during running," with a risk factor being "SCT." The medical examiner cited the increased risk of exercise-related sudden death in SCT recruits.

Case 2

In October 2009, at about 11:20 a.m., after doing sit-ups and push-ups, a 20-year-old African-American male began the 2-mile APFT run. He was just in from 3 days in the field, but conditions were modest, temperature 75ºF, relative humidity 87%, and heat category 1. He was in his ninth week of BCT. A miler and wrestler in high school, he had completed unit runs, but had failed the APFT 2-mile run twice. On his third try, he started fast and was first or second early-on, but began to walk on the penultimate lap. Urged on by peers, he ran again, only to collapse 300 m from the finish line. He sat down, stayed conscious, and complained that he was thirsty and his legs hurt. Witnesses said he mumbled and groaned about leg pain, once said he "couldn't feel his legs" and seemed confused and disoriented. He was hosed down, ice blanketed, and rushed to Urgent Care of the Post's Medical Treatment Facility, arriving about 12:00 p.m.

On initial assessment by a physician, he was alert and oriented, heart rate 170, respiratory rate 30, rectal temperature 102.6ºF. He complained of thigh pain, but thighs were not painful to palpation. In initial laboratory work, hemoglobin was 14.9 g dL^{-1} and white blood cell count was 11,040 mm^{-3}, but hemolyzed samples prevented further analysis. At 2:40 p.m., serum potassium was reported as 2.1 mEq L^{-1}, so he was given potassium. Serum bicarbonate was very low at 5 mEq L^{-1}, and serum creatine kinase was 202 U L^{-1}. At 3:00 p.m., blood urea nitrogen was 21 mg dL^{-1}, serum creatinine 2.8 mg dL^{-1}, serum bicarbonate still low at 12 mEq L^{-1}, but creatine kinase very high at 27,947 U L^{-1}. Urinalysis showed glucose, protein, and "large blood" (likely myoglobin). At 3:15 p.m., he was transferred to a civilian hospital, still tachycardic and tachypneic, with rectal temperature 99.7°F.

On arrival at the civilian hospital at 3:30 p.m., he was conscious but lethargic. At 3:40 p.m., hemoglobin was 18.2 g dL^{-1}, bicarbonate still low at 12 mEq L^{-1}, serum transaminases elevated (likely from muscle), and potassium 4.9 mEq L^{-1}. Soon after arrival, he deteriorated, vomited several times, and suffered a cardiopulmonary arrest. Resuscitation was unsuccessful and he was pronounced dead at 5:05 p.m.

The medical examiner said the cause of death was "complications of exertional rhabdomyolysis with SCT." Hemoglobin analysis revealed 56.9% hemoglobin A, 36.4% hemoglobin S, 4.6% hemoglobin A2, and 2.1% hemoglobin F. The Office of the Armed Forces Medical Examiner agreed that "the cause of death was clearly exertional rhabdomyolysis." They noted the "higher frequency of sudden, unexpected death – frequently from exertional rhabdomyolysis – in SCT."

DISCUSSION

We report the cases of two Army recruits who collapsed from exertional sickling as they struggled to finish in time the 2-mile APFT run. Both soon died from complications of the fulminant ischemic rhabdomyolysis that follows exertional sickling. Well-meaning drill sergeants who did not know that these recruits had SCT encouraged them to try repeatedly to pass the 2-mile run. One died on her fifth try; the other on his third try.

Another fatal sickling collapse was recently reported in a 23-year-old Army National Guard recruit with SCT who collapsed sprinting to the goal line of a 2-mile obstacle training course.[7] The evidence suggests that the proximate trigger for the fatal sickling in these Army recruits was sustained high-intensity exertion.

A similar exertional sickling death occurred recently in college track. In August 2010, in early evening, in rainy weather with temperature 82ºF, a 20-year-old sophomore who had raced well in the 300-m hurdles in high school joined a track tryout at an National Collegiate Athletic Association (NCAA) Division 1 university. After a 20- to 25-minute dynamic warm-up, he ran laps on the track. He began to struggle on the third lap and collapsed to his knees soon thereafter. He was not able to stand, even with help. He complained of leg pain. He was carried to the side of the track, where for about 10 minutes he sat, conscious, able to talk, drinking some fluid, before "slumping over." At that point, a teammate performed CPR until emergency medical services arrived. He was taken to a local hospital where serum myoglobin was elevated at 293 ng mL^{-1} (suggesting acute rhabdomyolysis) and serum potassium was elevated at 5.5 mEq L^{-1}. Despite resuscitation measures, he died soon after arrival. Autopsy findings included SCT and vascular congestion with sickling in the heart, lungs, liver, kidneys, and brain. The cause of death was "complications of SCT with physical exertion."[8]

The exertional sickling deaths of these recruits are also similar to recent sickling deaths in college football.[9] In fact, exertional sickling has become the leading killer in NCAA Division 1 football. Since the year 2000, no deaths have occurred from the play of the game in Division 1 football, but 16 deaths have occurred from conditioning for the game, 15 from sprinting or high-speed agility drills, and one from weightlifting. Alarming is that 10 (63%) of these 16 deaths are tied to SCT and exertional sickling, an excess of up to 21-fold. Most of these exertional collapses, in recruits and athletes

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alike, were similar in clinical presentation: conscious collapse during sprinting or other maximal exertion, major muscle pain or weakness, and the inability to stand or walk unaided following collapse.

The common trigger for a sickling collapse in football conditioning is maximal exertion sustained for at least a few minutes, and typically no evidence exists for EHI as a prelude.[10] It could be argued that, for Army recruits just back from a field training exercise, cumulative heat stress (CHS) may predispose to EHI and then sickling during a 2-mile run. As U.S. Army Training and Doctrine Command instructs unit leaders: "If the soldiers have been subjected to category IV or V conditions for 2–3 consecutive days, then CHS increases their chance for a heat injury on the subsequent day."[11] It seems very unlikely, however, that CHS, even if present, would pose a hazard for major EHI during just a 15- to 20-minute run on a track. Besides, no evidence exists for CHS in our Case 1 or in the recruit who ran the 2-mile obstacle course,[7] and even if our Case 2 in theory had mild CHS, he ran on a mild day and his rectal temperature after collapse was consistent with the physiologic hyperthermia of the running he had just done. It seems the trigger for exertional sickling collapse in these Army recruits was "intensity."

It is true, of course, that ambient heat increases the effort required to complete any given exercise drill, compared to completing the same drill in the same time on a cool day. In other words, heat increases the "intensity." The same applies to "thin air," so being new at altitude, even on a cool day, is a risk for exertional sickling collapse. But in all these settings—in college football at least—the proximate trigger is a bout of sustained maximal exertion, in which the athlete—desperate to make the team—and the coach—relentlessly driving his squad—fail to recognize or heed early warning signs of exertional sickling and so push beyond limits to collapse that can be fatal.

The pathophysiology also fits an intensity collapse. Football players just out of an air-conditioned locker room but sprinting all-out can collapse from sickling in only 2 to 5 minutes. Maximal exertion can cause profound metabolic changes in 2 to 5 minutes—striking hypoxemia and lactic acidosis, along with muscle hyperthermia and red cell dehydration—that can cause sickling in SCT. U.S. Army research has shown that sickling can occur within minutes, doing maximal arm cranking exercise.[12] If enough sickling occurs, the sickle cells can logjam small blood vessels and lead to fulminant, ischemic rhabdomyolysis. So in the face of SCT, maximal exertion can evoke profound metabolic changes that can foster sickling and lead to collapse in a few minutes. This is likely what happened to the two Army recruits here. Many other similar cases, including in police and firefighter recruits, have recently been summarized.[10,13]

If this hypothesis is correct—that exertional sickling is an intensity syndrome—it raises questions about how to mitigate this risk in recruits. The NCAA has mandated SCT screening for all Division 1 athletes and has suggested tailored precautions so they can thrive in their sports. The Air Force, Navy, and Marine Corps screen their recruits for SCT, but the Army does not, relying instead on universal precautions to prevent EHI and thus sickling deaths.[6] Yet some sickling deaths continue in Army recruits. It seems likely that additional measures could further decrease the risk of these deaths.

For example, no practice guidelines or algorithms exist to help health care providers recognize that conscious collapse during an intense event such as sprinting can be life-threatening sickling. Both recruits we report were treated as EHI collapse, which is common in BCT. Exertional sickling collapse is under-recognized.[10,13] The distinction is vital, because the mainstay of therapy for a major EHI is rapid cooling, whereas the mainstay of therapy for a sickling collapse is preventing grave metabolic complications of fulminant rhabdomyolysis. Greater awareness of the different collapse syndromes and a practical algorithm on how to approach and manage an exertional collapse would help drill sergeants in their critical responsibilities for the recruits under their command. Finally, we suggest that any Army recruit who struggles and fails in his or her first 2-mile APFT run should be screened for SCT.

CONCLUSION

Two Army recruits with undetected SCT collapsed and soon died from metabolic complications of exertional sickling as they struggled to finish the 2-mile APFT in the required time, having failed to pass this test on prior attempts. These cases are similar to other military cases and to exertional sickling deaths in college track and football. Evidence increasingly suggests that the trigger for most sickling collapses is intensity. Sickling can begin in 2 to 5 minutes of all-out exertion and can reach grave levels soon thereafter if the recruit or athlete struggles on or is urged on—despite warning signs—by drill sergeants, coaches, or comrades.

For several decades, the military has focused on preventing EHI and its complications. Yet since fatalities continue, it seems fair to suggest that education of trainers and providers on the exertional sickling syndrome, coupled with risk mitigation measures targeted at high-intensity exercise, may reduce SCT deaths. Clinical practice guidelines and algorithms are needed on how to recognize and treat exertional sickling. As debate and research continue on the pros and cons of screening Army recruits for SCT, we suggest that any recruit who struggles and fails in his or her first 2-mile APFT run be screened for SCT before being urged to try that run again.

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Erythrocyte Sickling During Exercise and Thermal Stress

Michael F. Bergeron, PhD, Joseph G. Cannon, PhD,† Elaina L. Hall, BS,* and Abdullah Kutlar, MD‡*

Objective: To examine effects of exercise in the heat and fluid intake on erythrocyte sickling and neutrophil activation in carriers of sickle cell trait (HbAS).

Design, Setting, and Participants: Six African American men (2 HbAS; 42% HbS, 4 HbAA; 20.7 ± 0.8 years; 87.4 ± 9.6 kg) participated in 2 randomized sessions (separate days) each consisting of 45 minutes of brisk walking (treadmill) in a hot (33°C) environment.

Intervention: Subjects consumed no fluids or fluid for 3 hours prior to (ad libitum) and during (1.02 L) testing.

Main Outcome Measurements: Core temperature, heart rate, and perceived exertion were measured. Forearm venous blood was analyzed for percent erythrocyte sickling and plasma myeloperoxidase.

Results: Time-averaged heart rate (126.6 ± 5.7 vs. 146.7 ± 5.9 bpm; $P = 0.02$) and core temperature (37.6 ± 0.1 vs. 38.1 ± 0.1°C; $P < 0.05$) responses were lower during fluid versus no fluid, with no statistically significant difference in perceived exertion (12.3 ± 0.5 vs. 13.6 ± 0.4; $P = 0.06$). Erythrocyte sickling progressively increased (to 3.5%–5.5%) for HbAS carriers during no fluid exercise only. No sickling was detected in HbAA subjects. Plasma myeloperoxidase responses to exercise were greater ($P = 0.03$) in HbAS versus HbAA.

Conclusions: Fluid ingestion at a rate sufficient to offset a body weight deficit can effectively reduce erythrocyte sickling during exercise in the heat.

Key Words: sickle cell trait, hydration, myeloperoxidase

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INTRODUCTION

Sickle cell trait has been characterized as a benign disorder,[1] with little or no risk of sickling in vivo.[2] Accordingly, sickle cell trait is often not considered to be a restriction to athletic participation,[3] a detriment to exercise performance,[4] or an independent factor to increase exercise risk.[5] However, Kark et al[6] indicated a 30-fold greater rate of unexplained exercise-related sudden death among black recruits with sickle cell trait compared with black recruits without hemoglobin S. A number of fatal and nonfatal events (e.g., nonfatal exertional collapse, heat illness, and rhabdomyolysis) have also been reported with recruits, athletes, firefighters, and children.[7–9]

A combination of extensive exercise, dehydration, and thermal strain may be enough to induce sufficient hyperthermia, hyperosmolality, acidosis, and red cell dehydration, potentially leading to significant erythrocyte sickling, inflammation, microvascular occlusion, and ultimately to exertional collapse and exercise-induced sudden death in certain HbAS carriers.[7,8,10] However, few reports on exertional-related death or life-threatening complications among HbAS carriers noted field hydration status or core body temperature[8]; therefore, the contribution of each is unclear.

We examined whether a low level of progressive sickling would occur in healthy volunteers with sickle cell trait in response to exercise and thermal stress. We also investigated whether improved hydration would lower metabolic stress and core body temperature during exercise in the heat, and thus reduce or eliminate any exercise-induced changes in erythrocyte sickling and acute signs of neutrophil activation.

METHODS

Six African American men (2 HbAS; 42% HbS, 4 normal hemoglobin [HbAA]; 20.7 ± 0.8 years; 87.4 ± 9.6 kg) participated in 2 randomized sessions (2–7 days apart), consisting of 45 minutes of walking (treadmill) in a hot environment (33°C; 45% relative humidity). For 1 session, subjects consumed no fluids (NF) for 3 hours prior to or during testing. For the other, subjects were encouraged to drink (ad libitum) noncaffeine fluids (F) throughout the morning and up to the exercise session. During F, subjects consumed water (170 mL) at the start of exercise and every 10 minutes thereafter (total, 1.02 L). This rate of fluid ingestion offset sweat-induced fluid losses during F, so that there was no body weight deficit at the end of exercise. The institutional Human Assurance Committee approved the study, and subjects provided written informed consent prior to participation.

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From the *Department of Physical Therapy, Medical College of Georgia, Augusta, GA, the †Departments of Medical Technology and Physiology, Medical College of Georgia, Augusta, GA, and the ‡Department of Medicine, Medical College of Georgia, Augusta, GA.
Reprints: Michael F. Bergeron, PhD, FACSM, Department of Physical Therapy, CH-100, School of Allied Health Services, Medical College of Georgia, Augusta, GA 30912-0800 (e-mail: mbergero@mcg.edu).
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Exercise Intensity

During familiarization (FAM) sessions (1 week earlier), treadmill speed and elevation were adjusted so that the subjects were walking briskly, eliciting a rating of perceived exertion (RPE: Borg category ratio scale) of 13 to 15 (somewhat hard to hard) for most of the 45-minute exercise period. Subjects consumed no fluids for 3 hours prior to or during the FAM exercise session (same as during NF). For the subsequent randomized F and NF sessions, the same exercise protocol and work rate (from the FAM sessions) were used for each individual. Heart rate (bpm) was continuously monitored using a Polar® monitor (Polar Electro Inc., Woodbury, NY).

Blood Collection and Analyses

Blood was collected (20-gauge catheter, antecubital vein) at the following times: pre, pre-exercise; 15 minutes, at 15 minutes; 30 minutes, at 30 minutes; IP, immediate postexercise; and 15 minutes post, after 15 minutes of standing cooldown in 22°C. For percent sickling, whole blood was drawn into EDTA tubes. The red cells were washed in saline, fixed in glutaraldehyde, and applied to a microscope slide. Slides were read on a Nikon Labophot, 2 phase-contrast microscope, in a blind, random order by an experienced hematologist. Ten fields of 100 erythrocytes each were examined, and sickled cells were counted and expressed as a percentage of total cells. Plasma from each original blood sample was separated and recentrifuged at 10,000 rpm to remove platelets and aggregates, then frozen at −80°C until assayed. Plasma myeloperoxidase (MPO) was measured by ELISA (OxisResearch, Portland, OR).

Core Temperature

Core temperature (°C) was monitored via ingestible telemetry sensors and a programmable hand-held monitor (HQ, Inc., Palmetto, FL). Each temperature sensor was ingested 5 hours prior to exercise.

Statistics

Data are reported as means ± SEMs. The influences of sickle trait and fluid status were investigated by 2-factor, repeated-measures analysis of variance using Statview statistical software (SAS Institute, Cary, NC).

RESULTS

Time averaged heart rate (126.6 ± 5.7 vs. 146.7 ± 5.9 bpm; $P = 0.02$) and core temperature (37.6 ± 0.1 vs. 38.1 ± 0.1°C; $P < 0.05$) were lower during and after (15 minutes − 15 minutes post) F compared with NF. RPE tended to be higher during (15 minutes − IP) NF (13.6 ± 0.4 vs. 12.3 ± 0.5; $P = 0.06$). Urine specific gravity indicated better ($P < 0.05$) pre-exercise hydration levels for F (1.017 ± 0.003) compared with NF (1.024 ± 0.003). Only unequivocally sickled (not merely distorted or crenated) cells were counted (Fig. 1). No sickling was detected in the HbAA subjects.

Myeloperoxidase increased in response to exercise ($P < 0.001$); however, hydration had no statistical effect ($P > 0.05$). Therefore, we collapsed the hydration groups (NF and F) together and plotted the response in plasma MPO (Fig. 2), displaying HbAS versus HbAA. Neutrophil activation, indicated by MPO responses to exercise, was greater ($P = 0.03$) in the HbAS group.

DISCUSSION

In a hot environment without fluid intake, there was progressive sickling with only 45 minutes of brisk walking. However, improved hydration effectively lowered metabolic stress and core body temperature, essentially eliminating any exercise-induced changes in erythrocyte sickling.

In the event of sickling, changes in erythrocyte structure activate the complement cascade, which in turn can activate neutrophils to release MPO. Alternatively, neutrophils may become activated by direct contact with sickled red cells. Activated neutrophils (in addition to sickled erythrocytes) can adhere to endothelium, leading to further restriction in microvascular flow. In our study, those with HbAS had greater apparent exercise-induced neutrophil activation, but the MPO data do not support a conclusion that it was a response related to sickling alone.

CONCLUSION

Many athletes with HbAS regularly participate in demanding exercise without appreciating that they may be at particular risk. Our findings suggest that fluid ingestion at a rate sufficient to offset a body weight deficit can be a simple yet effective preventative strategy to reduce erythrocyte sickling during exercise in the heat.

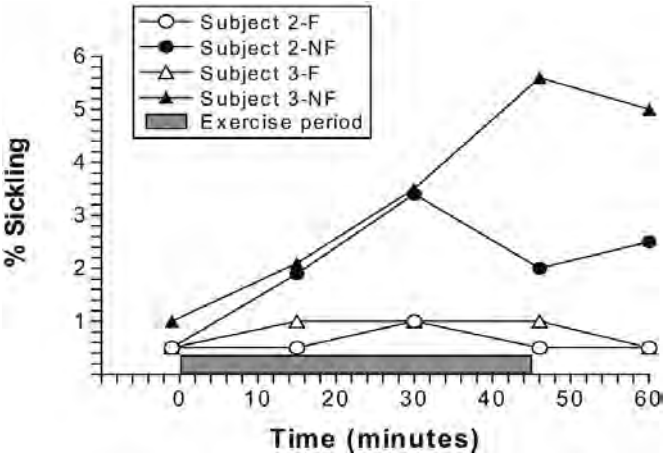

FIGURE 1. Percent (%) red blood cell sickling for the sickle cell trait (HbAS) carriers for NF and F trials.

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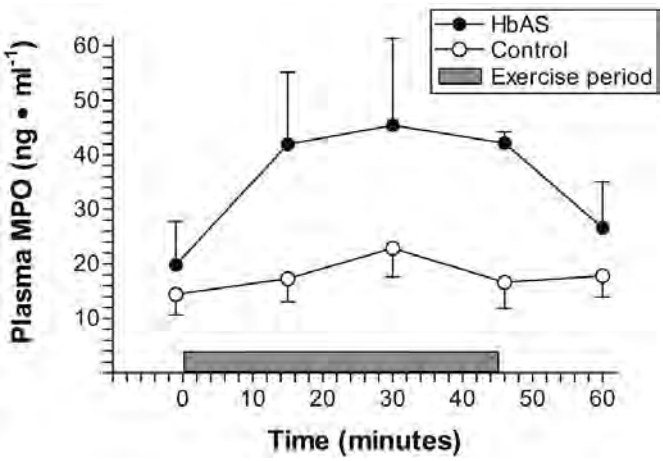

FIGURE 2. Plasma myeloperoxidase (MPO) responses for sickle cell trait (HbAS) carriers and control (HbAA) subjects with NF and F trials collapsed together. Neutrophil activation, indicated by MPO responses to exercise, was greater ($P = 0.03$) in the HbAS carriers.

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CLINICAL SCIENCES

Sickle Cell Trait and Fatal Rhabdomyolysis in Football Training: A Case Study

MARY L. ANZALONE[1], VALERIE S. GREEN[1], MAXIMILLIAN BUJA[2], LUIS A. SANCHEZ[1], RAJESH I. HARRYKISSOON[3], and E. RANDY EICHNER[4]

[1]Office of the Medical Examiner of Harris County, Houston, TX; [2]Department of Pathology, University of Texas Health Sciences Center, Houston, TX; [3]Department of Medicine, University of Texas Health Sciences Center, Houston, TX; and [4]Department of Athletics, University of Oklahoma, Norman, OK

ABSTRACT

ANZALONE, M. L., V. S. GREEN, M. BUJA, L. A. SANCHEZ, R. I. HARRYKISSOON, and E. R. EICHNER. Sickle Cell Trait and Fatal Rhabdomyolysis in Football Training: A Case Study. *Med. Sci. Sports Exerc.*, Vol. 42, No. 1, pp. 3–7, 2010. We report the athletic, the clinical, and the pathological details of a case of fatal rhabdomyolysis during training in a college football player with sickle cell trait (SCT) who collapsed minutes after running 16 successive sprints of 100 yd each. The player, 19 yr old, African American, was apparently healthy when he took the field for the conditioning run. No exertional heat illness was present. After collapsing on-field, the player soon went into coma and developed fulminant rhabdomyolysis, profound lactic acidosis, acute myoglobinuric renal failure, refractory hyperkalemia, and disseminated intravascular coagulation. Despite intensive care in the hospital, he died about 15 h after admission, likely from a hyperkalemic cardiac arrhythmia; the terminal rhythm was pulseless electrical activity. The forensic autopsy confirmed that the cause of death was acute exertional rhabdomyolysis associated with SCT. Counting this case, at least 15 college football players with SCT have died from complications of exertional sickling, as have younger football players and other athletes. In SCT, maximal, sustained exercise evokes four forces that can foster sickling: hypoxemia, acidosis, hyperthermia, and red cell dehydration. The setting, the clinical and laboratory features, and the clinicopathological correlation here suggest that the fulminant rhabdomyolysis and its fatal sequelae were from exertional sickling. These data suggest that screening and simple precautions for SCT may be warranted to prevent tragedies like this and enable all athletes with SCT to thrive in their sports. **Key Words:** LACTIC ACIDOSIS, RENAL FAILURE, HYPERKALEMIA, DIC

Sickle cell trait (SCT) is common and generally benign. More than 3 million Americans have SCT—about 1 in 12 blacks and between 1 in 2000 and 1 in 10,000 whites—and almost all live normal, healthy lives. However, for some carriers, SCT can pose clinical problems. As they grow older, many SCT carriers lose the ability to concentrate fully the urine; this usually is harmless. Two other clinical complications in SCT—splenic infarction at altitude and intermittent gross hematuria—are not life threatening and are covered elsewhere (6). Some athletes with SCT, however, develop a syndrome that can be grave or even rapidly fatal: fulminant exertional rhabdomyolysis.

Address for correspondence: E. Randy Eichner, M.D., 321 Baudine Way, Sonoma, CA 95476-5669; E-mail: reichner1@comcast.net.
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Fulminant rhabdomyolysis is an underrecognized and misdiagnosed cause of sudden collapse in sports. Among 136 well-studied, sudden, nontraumatic sports deaths in high school and college athletes over a decade, the third most common cause of death, behind cardiovascular conditions and heatstroke, accounting for 5% of the deaths, was sickling-associated rhabdomyolysis (20).

Sudden death in SCT was first reported in 1970 in four recruits undergoing Army basic combat training at an altitude of 4060 ft (10). One recruit died of what seemed to be an acute coronary event, but in three recruits, profound metabolic acidosis and early hyperkalemia (plus peaked T waves on electrocardiogram [EKG] in one) suggested that fulminant, sickling-associated rhabdomyolysis played a role in the deaths. Four similar cases were soon reported from the US Air Force, two recruits in basic training (not at altitude) and two cadets at the Air Force Academy (7200 ft). Three survived. All four had exertional rhabdomyolysis, hyperkalemia, disseminated intravascular coagulation (DIC), and acute myoglobinuric renal failure requiring dialysis (13). All seven of these recruits or cadets with SCT were exercising vigorously when they collapsed, but none had heatstroke.

Rectal temperatures were normal or mildly elevated; the highest being 102°F (10,13). The four men in the second report were the only ones hospitalized for rhabdomyolysis and renal failure, among thousands of trainees subjected to the same physical and environmental stresses, leading the authors to conclude that major exertional rhabdomyolysis may be more likely to occur in trainees with SCT (13).

Exertional sickling without major heat stress seems also a likely cause of collapse from fulminant rhabdomyolysis in some athletes with SCT. Counting this case, at least 15 college football players have died from complications associated with SCT, as have younger football players and other athletes (4,5,7). The typical SCT syndrome is demonstrated by this case of a college football player in training.

CASE PRESENTATION

A 19-yr-old African American football player in Texas collapsed during training. His medical history included seasonal allergies and "fatigue and shortness of breath with exercise" at the age of 13 yr—which may have been exercise-induced asthma—when he started playing baseball. He did not use tobacco, drugs, or alcohol. An aunt had SCT, but neither he nor the university knew he had SCT.

The collapse occurred during a late September conditioning workout. The workout began at 4:00 p.m. with weight lifting indoors. At 4:30 p.m., the athletes went outdoors for sprints. The player was apparently healthy when he took the field for the conditioning run. The climate was mild; the turf temperature was 76°F with mild wind.

The player ran 16 sprints of 100 yd each. Rest breaks were 1 min between each of the first four sprints, 2 min between each of the next four sprints, and 1 min between each of the last eight sprints. The player may have struggled and lagged behind teammates (media report) in the final sprints. By about 4:55 p.m., he completed the sprints and complained of shortness of breath and lower extremity discomfort. He was alert at first, but over the next 10 min became lethargic, so the university emergency medical service (EMS) was called. The player was conscious and breathing rapidly. His legs were too weak to support his weight, so he was loaded onto a cart and driven to the training room, where his legs were elevated and he was given supplemental oxygen and ventilated via a bag-valve mask. At 5:12 p.m., the university EMS arrived, gave 100% oxygen via a bag-valve mask, started intravenous fluids, and called the Fire Department EMS.

At 5:28 p.m., the Fire Department EMS arrived. By then, he was unconscious and unresponsive to pain, with sluggish pupils, shallow respirations, not moving extremities, and a Glascow coma score of 3. His blood oxygen saturation by pulse oximetry on ambient air was 62% and on high-flow oxygen increased to 100% within 3 min. He was nasally intubated. EKG showed sinus tachycardia and peaked T waves in leads V2–V3. He was rushed to the hospital, arriving in the emergency room at 5:52 p.m., where initial blood pressure was 151/49 mm Hg, pulse 126 min^{-1}, respirations 30 min^{-1}, and temperature 97°F (site not specified).

Initial laboratory work showed a profound lactic acidosis: arterial blood gas at 7 p.m. showed pH 7.05, PCO_2 45 mm Hg, PO_2 540 mm Hg, HCO_3 13 $mEq{\cdot}L^{-1}$, lactic acid 33 $mmol{\cdot}L^{-1}$ (upper limit of normal (ULN) = 2.2 $mmol{\cdot}L^{-1}$), and serum bicarbonate 8 $mEq{\cdot}L^{-1}$ (lower limit of normal (LLN) = 24 $mEq{\cdot}L^{-1}$). There was evidence for acute rhabdomyolysis: elevated serum myoglobin 7485 $ng{\cdot}mL^{-1}$ (ULN = 72 $ng{\cdot}mL^{-1}$), serum creatine kinase 536 $U{\cdot}L^{-1}$ (ULN = 220 $U{\cdot}L^{-1}$), serum phosphorus 12.8 $mg{\cdot}dL^{-1}$ (ULN = 4.5 $mg{\cdot}dL^{-1}$), and serum potassium on the high side at 5.2 $mEq{\cdot}L^{-1}$ (ULN = 5.1 $mEq{\cdot}L^{-1}$). There was evidence for acute renal failure (serum creatinine elevated at 2.3 $mg{\cdot}dL^{-1}$) and DIC (prothrombin time 23 s, partial thromboplastin time 84 s, fibrinogen 87 $mg{\cdot}dL^{-1}$, and fibrin split products >20 $\mu g{\cdot}mL^{-1}$). Chest x-ray was normal; EKG showed peaked T waves. A serum drug screen was negative and cerebrospinal fluid was unremarkable. White blood cell count was 9700 mm^{-3}, with a left shift in the differential count. Hemoglobin was 14.5 $g{\cdot}dL^{-1}$ (but hours later fell to a nadir of 4.3 $g{\cdot}dL^{-1}$), and platelet count was 149,000 mm^{-3}.

Consultations included cardiology, who did bedside echocardiography and thought the primary problem was not cardiac; neurology, who did the lumbar puncture and an electroencephalogram that showed no epileptiform activity; neurosurgery, who thought no surgical intervention was needed; and infectious disease, who treated him with antibiotics for possible septicemia.

Through the evening and night, he remained unresponsive and bled copiously from the nose and venipuncture sites. His lower extremities were repeatedly described as "rigid" and "stiff" and "unable to bend." He was given intravenous fluids, started on renal dialysis by nephrology consultants, and transfused with 20 U of fresh frozen plasma, 10 U of red blood cells, and 4 U of platelets. Serum creatine kinase rose to >41,000 $U{\cdot}L^{-1}$ (the top level on their test scale) by 10:27 p.m., confirming fulminant rhabdomyolysis. Hyperkalemia was refractory, with serum potassium rising to 7.1 $mEq{\cdot}L^{-1}$ by 8:27 p.m. and to 9 $mEq{\cdot}L^{-1}$ 2 h later. He died about 15 h after admission. It seems very likely that the proximate cause of death was a hyperkalemic cardiac arrhythmia. The terminal rhythm was pulseless electrical activity.

AUTOPSY FINDINGS

Generalized edema and ecchymoses were present. Evidence of DIC included bilateral serosanguineous pleural effusions and a similar peritoneal effusion, along with extensive pelvic soft-tissue hemorrhage extending from catheter sites in the thigh. Also, a small, thin interhemispheric subdural hemorrhage was grossly noted on the brain. The heart weighed 425 g (ULN about 400 g) and the left ventricular wall and interventricular septum were 15 mm thick

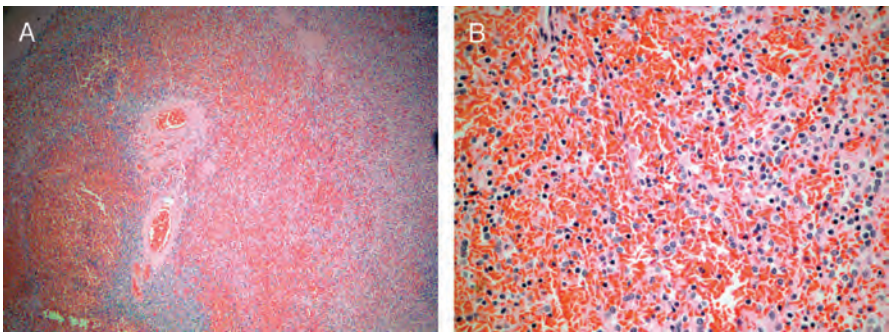

FIGURE 1—Focal packing of sickled red blood cells in the spleen. *A*, Hematoxylin and eosin (H&E), low magnification. *B*, H&E, higher magnification.

(ULN about 13 mm). Lungs were moderately congested with thickened bronchial basement membranes and occasional eosinophils, consistent with mild asthma. The 1400-g liver was congested with developing centrilobular necrosis and many sickled red cells. The kidneys showed patchy coagulative necrosis, vacuolization and hydropic change of tubular epithelium, and sickled red cells. Mild lymphocytic thyroiditis was present. Adrenal glands were normal. The spleen was enlarged at 250 g and showed red pulp congestion and focal packing of sickled cells (Fig. 1). The brain had no other abnormalities on careful sectioning; however, on microscopic examination, it showed focal ischemic neuronal changes and rare perivascular pigmented macrophages and calcification suggesting possible sickling bouts in the past. Skeletal muscle showed evidence of the acute rhabdomyolysis that in our opinion was ischemic, from exertional sickling, and led to the collapse of the player: focal vacuolization and disruption of the normal banding pattern, contraction bands, and decreased to absent staining for myoglobin and desmin in the cytoplasm of muscle cells, along with loss of staining of cell membranes for dystrophin (Fig. 2). Antemortem toxicologic testing and postmortem testing on pretransfusion antemortem samples were both negative. The heart was examined also by an expert in cardiopathology and was judged as normal, with physiologic cardiomegaly ("athlete's heart"), normal atrioventricular and sinoatrial nodes with hypertrophic myocytes, contraction band necrosis, and vacuolization of some myocytes. Most of the sickling was judged to be antemortem, not agonal, because 1) it was extensive despite considerable transfusion with normal red cells that would never sickle, and 2) the spleen was enlarged with focal packing with sickled red cells. Hemoglobin electrophoresis showed

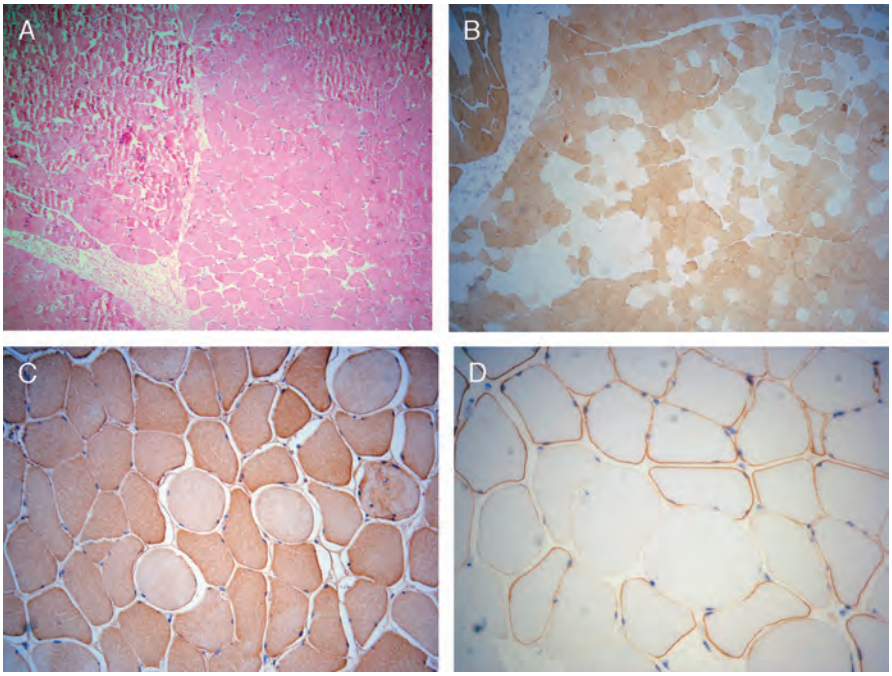

FIGURE 2—Skeletal muscle with microscopic features of acute rhabdomyolysis. *A*, Disruption of normal banding pattern and contraction bands, H&E. *B*, Decreased to absent cytoplasmic immunostaining for desmin. *C*, Decreased to absent cytoplasmic immunostaining for myoglobin. *D*, Loss of cell membrane immunostaining for dystrophin.

SCT with 55.3% hemoglobin A, 41.3% hemoglobin S, and 3.4% hemoglobin A2, whereas a normal electrophoresis would be about 97% hemoglobin A, 2.5% hemoglobin A2, and no hemoglobin S. His newborn screening record showed SCT. The cause of death was acute exertional rhabdomyolysis associated with SCT.

DISCUSSION

This college football player collapsed with fulminant rhabdomyolysis after running 16 successive sprints of 100 yd each in training at sea level in a mild climate. The setting, the clinical features, the laboratory profile, and the clinicopathological correlation strongly suggest that the fulminant rhabdomyolysis was ischemic, from exertional sickling. No exertional heat illness (EHI) was present, although the player's body may have encountered measurable heat strain. He rapidly developed profound lactic acidosis, hyperkalemia, acute myoglobinuric renal failure, and DIC. The player died 15 h after hospital admission, likely from cardiac arrhythmia from refractory hyperkalemia. No one knew he had SCT, and it seems likely that the treating physicians were initially unclear as to what may have caused his collapse.

This medical emergency, sudden collapse from fulminant rhabdomyolysis without EHI and coincident with apparent exertional sickling, is similar to the collapse syndrome first reported in seven vigorously exercising military recruits and cadets (10,13). It is also similar to the fatal sickling collapses of at least 14 other college football players with SCT, the first in 1974 (4,5,7). Although some of these football players collapsed during or immediately after intense football drills (e.g., rapid tempo, multiple station, or "mat drills"), most of them, like the player herein, had been running serial sprints (some on day 1 of summer practice), collapsed after only 800–1600 m of sprinting, and had no evidence of EHI (4,5,7,18).

Controversy exists on the role of EHI in exertional collapse for those with SCT. US Army researchers found that the risk of sudden unexplained death during basic training was 40 times more common for black recruits with SCT than for all other recruits (11). In follow-up analyses, Gardner and Kark (8) and Kark and Ward (12) found that exercise-related death in black recruits with SCT was 30 times more common than in black recruits without SCT and concluded that EHI was a culprit in SCT deaths. They outlined 30 SCT exercise collapses (21 fatal) and concluded that EHI was present in 22. However, only 2 had temperatures as high as 105°F, 2 were at 103–104°F, 16 were at 102°F or lower (7 of these were 99°F or lower), and 10 had no temperature recorded (12). Gardner and Kark (8) detailed the collapse and death of a soldier with SCT who marched 3 miles on a cool morning. The clinical course was classical for fulminant rhabdomyolysis, and rectal temperature on collapsing was only 98°F. Accordingly, the data do not support that collapse was prompted by EHI. Considering the study of the aforementioned 30 cases, although 102°F, for example, implies measurable heat strain, it seems likely that many of the SCT collapses analyzed were associated not really with EHI but only with the expected physiologic hyperthermia of strenuous exercise. These authors do note that most SCT deaths they studied were from acute rhabdomyolysis and that the risk of fatal exertional rhabdomyolysis is increased about 200-fold in SCT (8,12).

Gardner and Kark (8) and Kark and Ward (12) went on to show that measures to prevent EHI could decrease SCT-related deaths. Preventive measures included decreasing exercise intensity and increasing rest cycles and hydration as ambient temperatures rose. It seems likely that these precautions decreased SCT-related deaths in basic training, not so much by preventing EHI but more by making the exercise easier—and so decreasing the forces that foster sickling—just as breathing supplemental oxygen when exercising at altitude would decrease SCT-related deaths. Recent data suggest the SCT death rate may be increasing again in the US Army (19).

Strenuous exercise evokes four forces that in concert are proposed to foster sickling in SCT: 1) severe hypoxemia, 2) metabolic (lactic) acidosis, 3) hyperthermia in working muscles, and 4) red cell dehydration, as red cells lose water traversing the hyperosmotic milieu of the working muscle (5). Exercise physiology research shows that in SCT, during intense exertion and hypoxemia, sickle cells can accumulate in the venous blood and that systemic dehydration increases heat strain and worsens exertional sickling (1,17). These and other factors that contribute to exertional sickling collapse are detailed elsewhere (4,5,7). Field studies in Africa suggest that SCT runners are limited not in single sprints but in middle distance or altitude running (16). The pattern in American athletes seems similar.

This case study focuses on football because at least 15 college football players with SCT have died in training with apparent concomitant exertional sickling. Similar collapses have resulted in death in football players in high school and as young as 12 yr. Several nonfatal collapses with sickling in football training also exist (2). Collapse with sickling but not EHI has also occurred in distance racing or running (3,9,14) and has resulted in hospitalization or death of college or high school basketball players (two were young women) in training (4,5,7), typically during intense sprints on the court or while running laps on a track. In 2008, a Golden Gloves boxer with SCT died after a collapse (associated with sickling) evoked by a three-round bout. A near-fatal collapse associated with sickling was recently reported in a police academy cadet with SCT who tried to run 1.5 miles (15). Evidence suggests that the harder and the faster these athletes go, the earlier and the greater the apparent sickling. Symptoms of suspected sickling can begin in only 2 to 3 min of sprinting or in any other all-out, sustained exertion, and analysis of collapses in SCT athletes suggests that sickling can quickly increase to grave levels if the stricken athlete struggles on or is urged on by the coach (5,7).

In our opinion and field experience, screening and precautions can prevent sickling-associated collapse and death in sports. Precautions are detailed elsewhere (7), but in general, the evidence suggests that the SCT athlete should build up slowly in training, tempo of drills should be slowed, and longer periods of rest given. The SCT athlete should run no timed serial sprints or miles and should avoid lengthy, sustained sprints or any all-out exertion beyond 2 to 3 min without a rest. Work/rest cycles should be adjusted for heat and altitude. The athlete should stay hydrated, and any asthma must be controlled. The emphasis with coaches should be that if SCT athletes can set their own pace, they seem to do fine; moreover, if any STC athlete begins to struggle in any drill, the drill should be stopped and medical attention should be prompt. It is critical for coaches and medical support staff to appreciate that prompt action can save a life. We suggest to have the athlete lie down, check vital signs, give supplemental oxygen by face mask, cool the athlete if necessary, and failing immediate improvement, call 911, attach an automated external defibrillator (AED), start an intravenous line with normal saline solution, get the athlete to the hospital quickly, and tell the emergency room doctors to expect fulminant rhabdomyolysis and its grave metabolic complications (7).

SUMMARY

SCT can pose a grave risk for some athletes. At least 15 college football players with SCT—along with other athletes—have suffered a sickling-associated fatal exertional collapse as a result of fulminant rhabdomyolysis that occurs without evidence of EHI. To illustrate the typical syndrome, we detail the fatal collapse of a college football player in training. The clinical course and the pathological correlation strongly suggest that exertional sickling is the cause of this collapse syndrome. In athletes with SCT, it appears that sickling can begin within 2–3 min of maximal sprinting—or any other all-out, sustained exertion—and can reach grave levels very soon thereafter if the athlete struggles on or is urged on by coaches despite warning signs. Heat, dehydration, altitude, and asthma can increase the risk for and worsen exertional sickling. Collapse with sickling is a medical emergency, and fast action can save lives. In our opinion, screening and simple precautions can prevent this unique syndrome and enable SCT athletes to thrive in their sports.

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SUPPLEMENT NO. 22

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SICKLE CELL TRAIT AND THE ATHLETE

E. Randy Eichner, MD
Professor Emeritus of Medicine and Team Internist
University of Oklahoma, Norman, OK

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 sickle-trait 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 "lock-up." 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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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."

Normal red blood cell

Sickled red blood cell

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

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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Awareness and Identification of Athletes With Sickle Cell Disorders at Historically Black Colleges and Universities

J. David Jones, MS, ATC; Douglas M. Kleiner, PhD, ATC

Objective: Information regarding the incidence or complications of sickle cell disorders in athletes has not been well reported in the literature. In this study we identify the amount of exposure that athletic trainers have to athletes with sickle cell disorders at historically black colleges and universities and describe the precautions, screening procedures, and treatment techniques used to identify and manage this population.

Design and Setting: A 12-question survey was mailed to head athletic trainers asking about their exposure to athletes with sickle cell disorders.

Subjects: Athletic trainers at 94 historically black colleges and universities.

Measurements: Descriptive data were compiled as frequencies and reported as a percentage of the total responses (n = 34). The data were also grouped by NCAA division or as non-NCAA member institutions.

Results: Of the 94 surveys, 34 (36%) were returned. Respondents reported that 4.9% of their athletes had the genetic trait, yet only 12% of the schools required screening for the trait during their athletic preparticipation examinations. Three of the schools (9%) reported a total of 10 incidents of athletes having sickle cell crises at their facilities.

Conclusions: We suggest that there is a likelihood for athletic trainers to encounter athletes with sickle cell disorders. Further education regarding sickle cell disorders may be needed.

Key Words: athlete, sickle cell anemia, sickle cell disorder, sickle cell trait

Sickle cell disorders are conditions that have long been associated with African-Americans.[6,18] Individuals who carry the sickle cell trait are considered by the medical profession as being mostly "healthy persons,"[22] whereas sickle cell anemia, the more severe form of the disorder, can limit physical performance and, in extreme cases of intense physical activity, can cause sudden death.[4] There have been few reports about preventive measures taken by colleges or universities to identify or treat athletes who may have sickle cell disorders.

Sickle cell trait, the benign disorder characterized by the heterozygous Hb-AS blood type, and sickle cell anemia, the homozygous disorder (Hb-SS blood type), are both genetic conditions.[11] Collectively, these are categorized as sickle cell disorders, with each having associated complications.[9,16]

The incidence of sickle cell trait is reported to be between 8% and 10% of the American black population,[18] while worldwide the number increases to 12% to 40%.[1,5,9] Studies involving athletic populations have indicated an incidence similar to that of the general population. A survey of National Football League players indicated that approximately 7% of the black players have sickle cell trait,[13] while it is reported that 18.6% of West African college athletes have the trait.[13,23] Despite these numbers, routine screening for sickle cell trait is not currently recommended by the National Collegiate Athletic Association (NCAA) or other sports-governing bodies.[14]

In its heterozygous state, sickle cell trait represents approximately 20% to 45% of red blood cell total hemoglobin, while the majority of the hemoglobin is normal.[1] Persons with sickle cell trait are regarded as carriers of the disorder and will normally show no signs or symptoms. However, with intense physical activity, individuals with sickle cell trait can develop a greater amount of sickling.

With sickle cell anemia, 80% to 100% of the hemoglobin in the red blood cells is abnormal and the red blood cells possess the distinctive crescent-shaped cell.[1] In the United States, it is estimated that 0.2% to 1.0% of black Americans suffer from chronic sickle cell anemia, while worldwide the incidence can rise to greater than 30% depending on the region.[1,15] Even though sickle cell trait is considered much more benign than sickle cell anemia, both disorders are considered risk factors for sudden death when associated with intense physical activity.[1,9,10,12,19]

The tendency toward sickling is influenced by factors associated with intense exercise including acidosis (such as lactic acidosis), hypoxia (induced by physical exertion at altitude), and dehydration.[7,9] Exertional rhabdomyolysis, or the catabolism of muscle tissue, can be a severe complication of sickle cell disorder and is frequently present when dehydration leads to sickling.[20] The waste products that are produced as a result of rhabdomyolysis are potentially lethal to the body's systems, particularly to the liver and kidneys. Once the kidneys have been affected by rhabdomyolysis, the situation becomes more critical and can ultimately lead to death.[2,3]

It is important for athletes to understand that extreme physical exertion often creates conditions conducive to sickling.[8] Complications often associated with sickling include: muscle cramps, painful episodes of tissue ischemia, musculoskeletal pain, delayed growth and sexual maturation, chronic pulmonary dysfunction, renal disorders, orthopedic problems (such as vasocclusion, bone infarction, and avascular necrosis), and, in some instances, sudden death.[6,17,24] When the signs and

J. David Jones is an athletic trainer at Southern Therapy Services in LaGrange, Georgia.

Douglas M. Kleiner is an assistant professor and Director of the Sports Medicine-Athletic Training Education Program at the University of North Florida in Jacksonville, FL 32224.

symptoms of these associated complications are present, it is said that the athlete is in "crisis." Previous studies have suggested that exercising individuals who have sickle cell disorder may be susceptible to a crisis, and, therefore, should not participate in intense activities.[10,19] Although this practice is no longer accepted, it does stress the importance of black athletes being aware of their sickle cell status. Considering the potential for complications in athletes with a sickle cell disorder, it is also important that athletic trainers be knowledgeable regarding screening, recognition, and treatment of these disorders.[8]

The purpose of this study was to identify what exposure the athletic trainers (ATs) working at historically black colleges and universities had to athletes with sickle cell disorders. A secondary purpose was to describe the precautions, screening measures, and treatment techniques used to identify and manage this population. Finally, as descriptive information, we attempted to identify whether the athletic trainers at the historically black colleges and universities who responded to survey were certified by the National Athletic Trainers' Association (NATA).

METHODS

The head athletic trainers at 94 historically black colleges and universities were each mailed a 12-question survey. The addresses of the colleges and universities were obtained from a mailing list provided by the NCAA. Historically black colleges and universities were specifically chosen because of their greater likelihood to have athletes with sickle cell disorders.

Each instrument was accompanied by definitions of sickle cell trait and sickle cell anemia, to better enable the respondents to clearly understand the differences between these two different, but related, blood disorders. Schools not responding to the first mailing of the survey were sent a follow-up instrument.

All returned instruments were compiled by the researchers and kept on file. To ensure confidentiality, each school and each instrument was coded with a number known only to the principal investigators. Descriptive data were compiled as frequencies and are reported as a percentage of the total responses (n = 34). The data were also grouped by NCAA division or in a non-NCAA member classification.

RESULTS

Of the 94 instruments, 34 (36%) were returned and classified according to their NCAA division (Table 1). Questions were asked about the amount of exposure the ATs had to athletes with sickle cell disorders (Table 2). Three (9%) of the colleges (one from each NCAA division) reported a total of 10 incidents of athletes developing sickle cell crisis while at their institutions. When asked what percentage of their athletes they thought had sickle cell trait, 15 (44%) of the ATs at these institutions reported that it was their perception that less than 1% of their athletes had the trait. Five (15%) of the ATs believed that less than 5% of their participating athletes had the trait. Four (11%) of the respondents reported having more than 5% of their athletes test positive for sickle cell trait. These data indicate that the ATs at these historically black colleges and universities believe that a small number of athletes with sickle cell trait participate in their colleges' athletics programs or that they may be unaware of the number of athletes with sickle cell trait. Furthermore, 10 (29%) of the ATs did not respond to this particular question of the survey which might also indicate a lack of awareness.

Table 1. Number of Respondents Who Completed the Sickle Cell Survey, Grouped by NCAA-Division

NCAA Division	Frequency	Percentage
Division I	09	26.5
Division II	13	38.2
Division III	04	11.8
NCAA nonmember	08	23.5
Total population	34	100.0

Additional questions were designed to determine the health care prevention strategies used by the ATs for athletes with sickle cell trait. The ATs were asked if their schools routinely paid for the cost of sickle cell screening. In this study, 4 (12%) of the schools required blood screening for sickle cell trait as part of their athletic preparticipation examinations. However, an additional 6 athletic departments (17%) said they would pay for the cost of the screening if the athlete wanted to be tested for sickle cell trait. Fifty percent of the schools asked about sickle cell trait in the athlete's medical history. Fifty percent also asked if sickle cell trait was present in the medical history of the athlete's immediate family. In 6 (17%) of the cases, the athletes were not asked about sickle cell trait, but were referred to the student health center for their preparticipation examinations.

Another question on the instrument asked the ATs if they had ever heard of the law case of *Sorey v Kellett* involving a college athlete who died of sickle cell complications.[21] In this lawsuit, the team physician, the athletic trainer, and the coach were all sued by the family of the athlete who died because he was not properly cared for by the university's medical staff.[21] Only 3 (9%) of the respondents in our survey reported knowing of this lawsuit.

The respondents were then asked if their institutions employed a full-time athletic trainer, and if he/she was certified by the NATA. Twenty-five of the 34 respondents (74%) employed athletic trainers. Of the AT respondents, 21 (62%) reported that they were certified by the NATA. However, it is likely that this statistic is biased. It is possible that the schools which responded did so because there was an AT there to receive the survey, and those that did not employ an AT were unable or unwilling to complete the survey. Therefore, we urge caution when interpreting these results. We would also recommend that historically black colleges and universities hire full-time NATA-certified athletic trainers, if they do not already.

DISCUSSION

It is our opinion that the current practice of screening athletes for sickle cell trait may not be sufficient and should be

Table 2. Sample Questions Taken From the Instrument

1. How many intercollegiate athletes do you have at your college or university?
2. Do you require blood screening for sickle cell trait as part of your preseason participation requirements?
3. Which of the following are included as part of the athlete's preseason examination? (Check all that apply)
   _ Blood screening specifically for sickle cell trait.
   _ Asking if the athlete has sickle cell trait on a medical history form.
   _ Asking if the athlete has any immediate family members with history of sickle cell trait.
   _ Does not apply to my program.
4. Does your student health center ask about sickle cell anemia on their medical history form?
5. Does the athletic department at your university or college provide and pay for sickle cell trait screening if the athlete requests it?
6. Have you ever had an athlete that has had complications (crisis) related to sickle cell trait?
7. If your response was "yes" to number 6, how many cases (s) have you seen or do you have on record?
8. Do any athletes with sickle cell anemia participate in any of your athletic teams at your college or university?
9. What percentage of your athletes DO YOU THINK have sickle cell trait?
10. Have you heard about the case of the athletic trainer who was sued by the family of an athlete who died of a sickle cell disorder?
11. Does your institution employ a full-time athletic trainer?
12. If your response was "yes" to number 11, is the AT certified by the National Athletic Trainers Association?

scrutinized further. Screening is needed to identify sickle cell status. Individuals with sickle cell trait may not know they are carriers for the disorder and may therefore be unaware of possible complications related to their condition. We believe that, as professionals, it is the responsibility of every athletic trainer to ensure that athletes at risk are identified and educated regarding their specific conditions. We recommend that questions designed to identify sickle cell trait be included in the medical history and that screening be provided to student-athletes during their preparticipation examinations.

The data from this study suggest there is a likelihood for athletic trainers to encounter athletes with sickle cell disorders. Furthermore, the respondents in this study probably underestimated the incidence of athletes with sickle cell disorders at their institutions. It was also apparent by several of the responses that there is still confusion regarding sickle cell anemia and sickle cell trait. Because of this, further education of ATs regarding sickle cell-related complications may be needed. Without current knowledge regarding sickle cell disorders, the possibility for ATs to encounter legal action remains. It is the responsibility of each AT to continually educate himself/herself regarding all aspects of sports medicine. ATs assume liability when treating athletes with sickle cell disorder, as with all athletes, and should be knowledgeable about screening, recognition, and treatment. It is hoped that the information presented in this study will encourage all ATs to seek further education regarding sickle cell disorders and its related complications.

Additional studies including ATs from other settings (high schools, professional teams, and other colleges and universities) in addition to historically black colleges and universities are needed to determine what screening measures, prevention strategies, and treatments of sickle cell-related complications are currently being practiced. Further research and continuing education of sickle cell-related complications for athletic trainers appears to be warranted.

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Sickle Cell Trait and the Athlete

Posted July 11, 2007

Statement

The College of American Pathologists (CAP) recommends athletic programs consider confirming sickle cell trait status in all athletes' preparticipation physical examinations. Simple precautions can prevent sickling crises and enable sickle cell trait athletes to thrive in sports and physical activities. If an athlete lacks documentation, the CAP recommends that screening be considered to determine his/her status. The CAP also stresses education to create an environment that encourages trainers and athletes with sickle cell trait to immediately recognize and report any symptoms assumed to be sickling.

Information Highlights

- Sickle cell trait is an inherited blood disorder, which can cause serious problems during exercise.
- The sickle gene may be present most often in people of African, African-American, Mediterranean, Middle Eastern, Indian, Caribbean, South American, and Central American decent.
- Many people do not know whether or not they carry the sickle cell gene.
- During intense exertion, dehydration, and hypoxemia (decrease in oxygen), the red blood cells change their shape in the bloodstream, can block blood vessels, and can cause problems throughout the body.
- Sickling collapse has been mistaken for cardiac collapse or heat collapse.
- Signs of sickling include fatigue, difficulty breathing, leg or low back pain, and sudden weakness.
- Environmental heat, dehydration, asthma, illness, and altitude predispose an athlete with sickle trait to an onset of crisis in physical exertion.
- No sickle-trait athlete is ever disqualified from participating in sports.
- Education and precautions can prevent sickle crises in athletes with sickle cell trait. These include adjusting exercise for the environmental temperature, emphasizing hydration, controlling asthma, no exercise if a sickle trait athlete is ill, modifying exercise upon exposure to a new altitude.

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Public Health Implications of Sickle Cell Trait

A Report of the CDC Meeting

Althea M. Grant, PhD, Christopher S. Parker, PhD, Lanetta B. Jordan, MD, MPH, Mary M. Hulihan, MPH, Melissa S. Creary, MPH, Michele A. Lloyd-Puryear, MD, PhD, Jonathan C. Goldsmith, MD, Hani K. Atrash, MD, MPH

Abstract: Although the issue of whether sickle cell trait (SCT) is clinically benign or a significant health concern has not yet been resolved, the potential health risk to affected individuals is of vital importance and represents a tremendous challenge in protecting, promoting, and improving the health of the approximately 300 million people worldwide and 3 million people in the U.S. who possess the trait. In response to a request by the Sickle Cell Disease Association of America, in December 2009, the CDC convened a meeting of partners, stakeholders, and experts to identify the gaps in public health, clinical health services, epidemiologic research, and community-based outreach strategies and to develop an agenda for future initiatives. Through facilitated discussion and presentations in four topic areas, participants discussed pertinent issues, synthesized clinical research findings, and developed a coherent framework for establishing an agenda for future initiatives. A primary outcome of the meeting was to provide the first step of an iterative process to move toward agreement regarding appropriate counseling, care, and, potentially, treatment of people with SCT.
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Introduction

On December 17, 2009, the CDC hosted a meeting on sickle cell trait (SCT), inviting partners from the NIH, the Sickle Cell Disease Association of America (SCDAA), the Health Resources and Services Administration (HRSA), and other stakeholders and experts to discuss the scientific and public health implications of SCT. The objective of the meeting was to identify gaps in public health, health services, epidemiologic research, and community-based outreach and to develop an agenda for future initiatives. Specific objectives were to:

- review scientific literature on health outcomes associated with SCT;
- identify opportunities for prevention of adverse health outcomes associated with SCT;
- consider models for screening and health education;
- assess screening programs;
- suggest a framework for screening including health education and follow-up; and
- identify effective strategies for preventing stigma and discrimination.

Sickle Cell Trait: Summary of Presentations

To create the foundation for accomplishing the meeting's objective, several speakers were invited to give presentations on a variety of topics. The meeting commenced with a discussion of reports from the medical literature about complications of SCT.[1,2]

Investigations on the prevention of exercise-related mortality for people with SCT were presented that included a discussion of the military experience with interventions and polices to prevent SCT-related deaths among recruits. This was accompanied by a presentation on SCT and athletes, which provided a brief history of the National Collegiate Athletic Association's (NCAA) sickle cell–related policies and statistics on college athlete deaths that occurred between 2000 and 2009.

The final group of presentations focused on various screening programs and models. The NCAA experience with SCT screening among athletes, including the results from surveys of colleges about their participation in SCT testing and their practices in treating athletes for SCT-

From the Division of Blood Disorders (Grant, Parker, Hulihan, Creary, Atrash), National Center on Birth Defects and Developmental Disabilities, CDC, Atlanta, Georgia; Department of Sickle Cell Services (Jordan), the Memorial Healthcare System, Hollywood, Florida; and the Sickle Cell Disease Association of America (Jordan), Baltimore; the Office of Rare Diseases Research (Lloyd-Puryear), NIH, the Division of Blood Diseases and Resources (Goldsmith), National Heart, Lung and Blood Institute, NIH, Bethesda, Maryland

Address correspondence to: Althea M. Grant, PhD, Branch Chief, Epidemiology and Surveillance Branch, Division of Blood Disorders, National Center on Birth Defects and Developmental Disabilities, CDC, 1600 Clifton Road, NE, MS-E64, Atlanta GA 30333. E-mail: agrant@cdc.gov.
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related complications, was reviewed.[3,4] Of note, at the time of the meeting, the NCAA was considering a proposal to mandate student screening for SCT unless carrier status was already known. An overview of newborn screening (NBS) policies for sickle cell disease in the U.S. and a summary of practices with respect to the reporting of test results with regard to SCT as well as an overview of recommendations regarding prenatal screening for hemoglobinopathies from the American College of Obstetricians and Gynecologists were presented.[5,6] Finally, a presentation discussing the National Practice Collaborative on preconception health coordinated through CityMatch in Nashville outlined the results of a program designed to address sickle cell education and awareness. The program developed an education campaign that included proposing new policies for school entry and mandatory school physicals at 5th and 9th grades, during which sickle cell status could be determined and discussed.

Because there are few epidemiologic studies examining the association of SCT with exercise-related death or the effectiveness of interventions to prevent exercise-related death among individuals with sickle cell trait, it is important to present here in more detail the evidence accumulated from the experience of the military with regard to SCT testing. Unpublished information was presented that had a strong influence on the discussion and conclusions of the meeting.

Evidence on exercise-related mortality for people with SCT is difficult to definitively classify due to the inability of autopsy findings to determine whether observed sickling of red blood cells post mortem caused an event or was secondary to agonal hypoxia.[7] To address this, researchers adopted a statistical approach comparing the frequency of death among those with SCT and those without SCT from the same population. Studies performed by Dr. John Kark and colleagues at Walter Reed Army Medical Center analyzed nontraumatic deaths during basic training among two million Armed Forces recruits from 1977 to 1981.[7]

Examining the risk of nontraumatic death of African Americans with SCT compared to African Americans and others without SCT, they observed 13 exercise-related deaths in 37,300 recruits with SCT versus five deaths in 429,000 other African-American recruits without SCT, yielding a relative risk of 30. In this analysis, it was observed that half of the cases of death were due to fatal exertional heat illness (EHI) and half were idiopathic sudden death associated with cardiopulmonary arrest. EHI includes exertional rhabdomyolysis with or without hyperthermia, heat stroke, and exertional acute rhabdomyolysis.

At the meeting, Dr. Kark also reported unpublished findings from a prospective interventional trial he directed from 1982 to 1991.[8] This study examined the hypothesis that preventing EHI would reduce mortality for all recruits and in particular the excess risk of death among those with SCT. In this trial, 1.8 million recruits participated in the intervention, and 1.1 million recruits did not. The intervention educated military drill instructors to record physiologic heat index hourly, to decrease training regimens of recruits to minimal effort in hot weather, to order and observe increased water consumption, to have recruits exercise in light clothing unless special exercises prevent this, and to get an immediate rectal temperature and initiate rehydration if a recruit showed early signs of "falling out" from group training. Although 13 deaths were expected among SCT recruits if no interventions were enforced, no deaths were observed when the precautionary measures were undertaken. This was a significant decline in predicted mortality by about 22-fold ($p<0.01$). It was calculated that there were also six lives saved among those without SCT due to the interventions.

In the nonparticipant group, there were four exercise-related deaths among individuals with SCT, all during hot weather, which correlated with the expected number of deaths. The conclusion was that reducing the number of deaths does not require screening for SCT when the same prevention methods are universally applied, and EHI is a necessary and preventable factor contributing to sudden exercise-related death with SCT. In a separate analysis, Dr. Kark found that the risk of mild to moderate EHI among Marine recruits at Parris Island during the same time period was the same for those with SCT and without SCT. Thus, it appears that SCT does not affect EHI until it becomes life-threatening. This was contrary to the expected results.

Additionally there was a discussion of the evolution from the late 1960s to the present of the military's policy relative to hemoglobinopathy screening. The U.S. Department of Defense (DoD) has concluded that the evidence supports SCT as a risk factor for EHI, likely with a contribution from still-unidentified genetic polymorphisms, but that risk can be reduced by prevention measures such as modified training practices. Individuals must meet specific medical fitness standards to qualify for entry into active duty in the Armed Forces. Currently, SCT is not a disqualifying condition for entry into the Armed Forces. However, anemia that can not be corrected by therapy is a disqualifying condition for military service, and with respect to hemoglobinopathies, sickle cell disease (SCD), not SCT, is frequently the cause of the uncorrectable anemia that disqualifies individuals for service. The 2003–2004 Armed Forces Epidemiological

Board (AFEB) recommended that all applicants be screened for anemia (defined as hemoglobin <13.5 g/dL for men and <11 g/dL for women) and tested for hemoglobinopathies if anemia is present. Eligibility for military service is based on a history of anemia as reported by the applicant on their medical history form, not SCT status.[9]

Although SCT does not exclude individuals from joining the armed services, depending on the branch of armed services, recruits may be screened for SCT once they have joined the military. The Army does not screen recruits for SCT, although soldiers being considered for certain high-risk occupations (e.g., aviation, diving, and special operations) are screened, and those with SCT may be considered medically unfit for participation in those activities. The Air Force, Navy, and Marines screen recruits upon entry into basic training for SCT. Group counseling of individuals who screen positive is routine. The Navy and Marines require recruits with SCT to wear identifiers during basic training. The Air Force allows recruits with SCT to leave service if they choose. All branches have heat injury prevention policies in place for all trainees. Additionally, all military branches also screen all babies born to military personnel for sickle cell and other hemoglobinopathies as part of newborn screening.[10]

Conclusions and Recommendations

After each speaker, meeting participants highlighted the public health needs with respect to SCT in the U.S. and identified approaches to address these gaps. The areas discussed included epidemiologic and clinical research, community awareness and education, ethical and legal concerns, screening practices, and prevention of adverse health outcomes.

Research in Sickle Cell Trait

The meeting participants expressed concern that there was a lack of both evidence and systematic evaluation of evidence to assess risks of adverse health outcomes associated with SCT. Meeting participants commented that researchers should not rely on the use of the general population with different ethnic backgrounds as controls in case-control studies and should determine whether exposures are the same between cases and controls. Another methodologic issue noted in SCT studies included the failure to fully consider the effect of confounding variables and comorbidities. An example is the failure to consider the impact of genetic modifiers such as α-thalassemia trait. Participants identified the need for further research to improve understanding of several SCT issues including:

- the prevalence of SCT among athletes to accurately determine the rate or risk of adverse health outcomes;
- the incidence of exercise-related adverse health outcomes (ERAHOs) associated with SCT. In addition to exercise-related deaths, there may be other ERAHOs that are not well documented.
- the impact of preconditioning, heat acclimatization, work–rest cycles, proper hydration, and clothing on those with SCT;
- the pathophysiologic mechanism of the association of SCT with ERAHOs;
- the psychosocial effect of targeted screening on individuals; and
- the impact of SCT on less studied outcomes such as adverse pregnancy outcomes, venous thromboembolism, and others.

In addition, participants identified several resources needed to facilitate addressing SCT research questions. These include:

- longitudinal cohort genetic studies;
- databases that provide a sufficient population size to provide study power to analyze the impact of SCT;
- studies that assess the influence of comorbidities, environmental factors (e.g., altitude), and genetic and biological factors (e.g., hemoglobin S level, haplotype, α-globin genotype) on risk of ERAHOs of individuals with SCT;
- systematic analysis of current evidence supporting the association of SCT with adverse health outcomes;
- studies that improve understanding of the etiology of ERAHOs associated with SCT;
- studies to distinguish causality from association;
- research on exercise (potential and limitations) for those with SCT; and
- cross-disciplinary collaboration on research.

Communication, Education, and Awareness

The participants expressed concern that there was a lack of consistency in content and delivery of health education messages about SCT and a lack of public awareness regarding the health issues associated with SCT. Participants noted that translating medical terminology for families is difficult, particularly in a period of declining outreach/education resources. However, some resources already exist. For instance, HRSA funded several years of research and development to support communicating complex genetic messages across languages and cultures, and participants suggested that the educational materials and documents produced from those projects could serve as a resource. Additionally, the experience of the former national network of sickle cell centers can provide some "lessons learned" on reaching the community. With respect to counseling, participants noted that information

about SCT is not a one-time message from care providers; it must be repeated at various stages of life so that people have the information at times when it is most useful to them. The participants suggested the following activities are needed to move the community awareness and health education initiatives forward:

- Health education efforts should capitalize on existing resources that have been created by established organizations and/or funded by federal agencies such as HRSA.
- Education and awareness about risks associated with SCT targeted toward policymakers, general public, media, health providers—especially primary care providers—coaches, and employers and addressing needs of various populations should be improved.
- Awareness and education strategies that incorporate broad media sources and community institutions, including faith-based organizations, schools, daycare, and other community institutions, should be developed.
- Health education efforts should address variability in health literacy, cultural and linguistic diversity, and reproductive decision making, as well as adverse health outcomes for individuals.
- Health education efforts should avoid increasing stigma and unduly worrying parents/athletes/prospective parents. It was noted that it is important to inform individuals aware of their SCT that they can still choose to participate in athletics. Institutions with appropriate standards of care and training in place can minimize the risk of adverse outcomes.
- Uniform consistent SCT messages should originate from authoritative public health sources such as the CDC, HRSA, NIH, and others.
- Programs are needed to support public understanding of genetic information. Participants suggested that health education messages may need to be tailored for SCT. Participants mentioned that traditional teaching about recessive disorders implies that there is no risk of adverse health outcomes among carriers, but this may not be the case with SCT, and consideration of this is needed when selecting the terminology used in SCT education.

Screening Practices

The participants expressed concern that only reliable laboratory methods (i.e., hemoglobin electrophoresis, high-performance liquid chromatography, or DNA-based techniques) should be used for screening in order to support appropriate individual education for all abnormal hemoglobins, not just hemoglobin S. Although the cost of screening is a potential barrier, participants cautioned against the utilization of less expensive solubility tests as a part of screening programs. The participants stated false-negative results from these types of tests may lead to incorrect reassurances, including the risk of having a child with SCD or another hemoglobinopathy. The participants suggested the following are needed to address screening issues:

- accurate and cost-effective means of ascertaining trait status, including new cost-effective molecular tests and a system to make NBS results available to physicians, parents, and affected individuals throughout the lifespan of the individual;
- easy-to-read, informative, and consistent screening reports;
- adequate funding to implement uniform screening and follow-up programs (especially NBS follow-up at the local level); and
- an informed testing process, including consent to screening and appropriate genetic counseling.

Ethical and Legal Concerns

The participants expressed concern that even in the context of organized sports, athletes with SCT could experience discrimination. For example, institutions might protect athletes with SCT and the institution from litigation by excluding affected athletes from practice. Participants voiced concern that some athletes have lost their athletic scholarships due to this issue. Participants also suggested that only a neutral informed party, such as a physician or genetic counselor, should provide screening results and communicate the health implications of having SCT to an individual. Additionally, there was discussion about the implications of SCT and regulations to protect individuals against genetic discrimination using legislation such as the Genetic Information Nondiscrimination Act (GINA) of 2008.[11] Participants expressed concern that screening for SCT may lead to a focus on the primary prevention of SCD. The participants cautioned that although it is important to address concerns for those with SCT, care is needed to avoid diverting funds to SCT away from efforts to deal with SCD health issues that remain as an underserved area of health care and public health. The participants suggested the following are needed to address ethical and legal concerns regarding SCT:

- Policies that minimize discrimination, exclusion, and stigma based on SCT status: Special attention is needed to avoid unintentional discrimination or bias from "labeling" people with SCT. In addition, policies need to be universal to avoid potential for racialization and stigmatization of athletes, in particular for African Americans with SCT.
- Policies that protect privacy: Although it is important that individuals know their carrier status, care must be taken to develop policies and procedures to protect

privacy of screening information. This may particularly be an issue if programs are implemented to minimize redundant screening by broadening legal access to universal NBS data.

- Research that monitors ethical, legal, and psychosocial results of screening programs: Participants suggested a research component that would track medical, legal, and psychosocial outcomes for identified individuals.

Prevention

To prevent adverse health outcomes, particularly exercise-related health events for individuals with SCT, most participants expressed support for utilizing a universal precaution approach in athletic training similar to DoD's interventions for EHI. This approach has been demonstrated by the military to reduce exercise-related deaths among those with SCT even in the absence of SCT screening. However, participants also noted that the athletic culture makes "universal precautions" difficult to implement. Others cautioned that relying on screening alone may not be sufficient to protect athletes and that implementation of screening guidelines may be inconsistent.

Several opportunities for prevention of adverse outcomes of SCT were identified:

- universal safe training practices for all athletes;
- collaboration of the public health community with the National Football League or collegiate athletic associations on primary and secondary prevention of ERAHOs;
- provision by the states of universal follow-up and education of carriers and creation of a database of that information that is accessible to screened individuals and their healthcare providers;
- the use of a comprehensive standard sports physical examination and history that is consistent in quality and ensures that information is passed on to adolescents before the adolescent is at risk;
- tools and education materials available for people with limited English proficiency and varying levels of health literacy;
- systems for adults to be screened and to have access to that information;
- standard policies for organizations to screen for all hemoglobinopathy traits, not just SCT, so that the athletes and others will understand their risk of having children with SCD;
- uniform availability of genetic counseling for those screened; and
- models that are tailored to the cultural contexts of organizations and populations served.

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SUPPLEMENT NO. 22

FRAMING THE RESEARCH AGENDA FOR SICKLE CELL TRAIT: BUILDING ON THE CURRENT UNDERSTANDING OF CLINICAL EVENTS AND THEIR POTENTIAL IMPLICATIONS

Jonathan C. Goldsmith MD[1], Vence L. Bonham JD[2], Clinton H. Joiner MD PhD[3], Gregory J. Kato MD[4], Allan S. Noonan MD MPH[5] and Martin H. Steinberg, MD[6]

1. Blood Diseases Branch, Division of Blood Diseases and Resources, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, Maryland
2. Social and Behavioral Branch, National Human Genome Research Institute, National -- Institutes of Health, Bethesda, Maryland
3. Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio
4. Sickle Cell Vascular Disease Section, Hematology Branch, National Heart, Lung and Blood Institute, National Institutes of Health, Bethesda, Maryland
5. School of Community Public Health and Policy, Morgan State University, Baltimore, Maryland
6. Boston University School of Medicine, Boston, Massachusetts

Address all correspondence to Jonathan C. Goldsmith, MD: 6701 Rockledge Drive, MSC 7950, Bethesda, MD 20892-7950; 301-435-0050 (Phone); 301-480-1046 (FAX); goldsmithjc@nhlbi.nih.gov (EMAIL).

ABSTRACT-

Sickle Cell Trait (HbAS), the heterozygous state for the sickle hemoglobin beta globin gene is carried by as many as 100 million individuals including up to 25% of the population in some regions of the world.[1] Persons with HbAS have some resistance to falciparum malaria infection in early childhood [2,3] and as a result individuals with HbAS living in malarial endemic regions of Africa have a survival advantage over individuals with HbAA. Reports from the US emphasize possible health risks for individuals with HbAS including increased incidence of renal failure and malignancy, thromboembolic disorders, splenic infarction as a high altitude complication, and exercise-related sudden death. The National Heart, Lung, and Blood Institute, National Institutes of Health convened a workshop in Bethesda, Maryland on June 3-4, 2010, *Framing the Research Agenda for Sickle Cell Trait*, to review the clinical manifestations of HbAS, discuss the exercise-related sudden death reports in HbAS, and examine the public health, societal and ethical implications of policies regarding HbAS. The goal of the workshop was to identify potential research questions to address knowledge gaps.

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BACKGROUND

Global Perspectives on the Health Impacts of HbAS

Studies in the 1950's revealed that the presence of sickle hemoglobin in erythrocytes appeared to protect against malaria, currently responsible for more than 1 million childhood deaths per year in sub-Saharan Africa. In 1,022 children from a birth cohort in Kenya, the highest mortality in children aged 2 to 16 months occurred in those with HbSS (sickle cell anemia), followed by HbAA (normal hemoglobin) and HbAS (sickle cell trait). The study demonstrated a survival advantage for children with HbAS over those with HbAA, with a 55 percent reduction in all-cause mortality.[3]

More recently, investigators followed 800 children and adults in Kenya for clinical events. While HbAS had no impact on the prevalence of asymptomatic parasitemia, it was 50 percent protective against mild clinical malaria, 75 percent protective against hospital admission for malaria, and 90 percent protective against severe or complicated malaria. [4] In an outpatient clinic in Tanzania, the prevalence of malaria parasitemia was 0.64 for AS, 0.72 for SS, and 2.96 for AA hemoglobin.[5] These studies suggest that children in Africa with HbAS have overall improved survival and lower malaria infection rates, and the difference is largely due to HbAS. The estimates of prevalence of HbAS in Africa range from 10% to 40% across equatorial Africa and decrease to between 1% and 2% on the north African coast and <1% in South Africa.[6] In all newborns in the U.S., the HbAS prevalence is 1.3 % based on a finding of HbAS in 1 out of 12 African Americans.[7] Worldwide, more than 100 million individuals have HbAS.

Potential Clinical Complications Associated with Sickle Cell Trait

A recent review of the medical literature reported that HbAS is not completely benign and provided an evaluation of the strength and specificity of the reported associations.[8] "Definite" associations with HbAS were proposed for exercise-related sudden death, exertional rhabdomyolysis, micro- and macroscopic hematuria, loss of renal concentrating ability, protection against severe falciparum malaria, renal medullary cancer, renal papillary necrosis, and splenic infarction in the setting of low oxygen tension at high altitudes. "Probable" associations with HbAS were suggested for pregnancy-related conditions including fetal loss, low birth weight, preeclampsia traumatic hyphema, and venous thromboembolic disease.

Acute chest syndrome, asymptomatic bacteriuria in pregnancy and proliferative retinopathy were reported as "Possible" associations with HbAS. Sufficient evidence was not found to associate avascular necrosis of the femoral head, cholelithiasis, leg ulcers, priapism, or stroke with HbAS. Aspects of this review, including the authors' conclusions, were addressed by the workshop.

SUDDEN DEATH IN SICKLE CELL TRAIT: WHAT IS THE EVIDENCE?

To review current information, the National Heart, Lung, and Blood Institute, National Institutes of Health convened a workshop to review the clinical manifestations of HbAS, discuss exercise-related sudden death reports in HbAS, and examine the public health, societal and ethical implications of policies regarding HbAS. The goal of the workshop was to identify potential research questions to address gaps in knowledge. During sessions devoted to discussion of

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reports of sudden death in individuals with HbAS, three major questions and the potential role for screening were addressed.

1. Exercise Related Death
    a. Is HbAS an independent risk factor for sudden death in young athletes?
    b. What are the etiologies of sudden death?
    c. Is sudden death preventable in individuals with HbAS?
2. Can screening for HbAS prevent or mitigate associated morbidity or mortality?

Sudden Death in Athletes

From a definition perspective, exercise related sudden death in all athletes is generally viewed as an irreversible and terminal dependence upon life support implemented within one hour of the onset of symptoms. The causes of sudden death have been described as cardiac (myocardial, anatomic, electrical, and combined disease), pulmonary, neurologic (brain stem injury, vagal reactions), metabolic (rhabdomyolysis, malignant hyperthermia), endocrine, hemorrhagic, and -- thrombotic. For cardiac or idiopathic sudden death, the US National Registry of Sudden Death in Athletes [(9)] reports 115 cases per year and the 36th Bethesda Conference on Eligibility Recommendations for Competitive Athletes with Cardiovascular Abnormalities estimated a prevalence of 1 in 33,000 – 50,000 with a male predominance of 5:1 to 9:1.[(10)] The number of reported cases increased by about six percent per year with 0.3% of the deaths attributed to HbAS. Of the cardiovascular deaths, 82% were reported as exertional; 2.4% of the deaths were attributed to heat stroke. The most common cardiac etiology was hypertrophic cardiomyopathy (HCM) (36%) followed by coronary artery anomalies (17%), The average age of those affected was 18$\pm$5 years and 89% were reported to be male. Hypertrophic cardiomyopathy, inherited as an autosomal dominant trait, has been attributed to mutations in genes that encode for sarcomere proteins.[(11)] Thirteen genes account for approximately 50 percent of HCM cases. HCM is disproportionately present in African American athletes. Of the relevant electrical diseases, the long QT Syndrome is the most important. Inherited in either autosomal or dominant fashion, thirteen genes have been described that are involved in the timely execution of the cardiac action potential.[(12)] The utility of ECG screening for athletes continues to be debated and provides some parallels for the current discussions of screening for HbAS.[(13)] Issues of cost, test sensitivity and specificity, resource utilization and difficulty of program implementation are central to this discussion. [(14)]

Performance of Black Athletes

Review of the historical athletic participation of individuals of African ancestry, self identified as black or African American, adds perspective. In the 1936 Olympics, 18 black athletes won 14 medals, 25% of those won by US athletes, and dominated the track and field events. In the 1968 Olympics, nearly 19% of the athletes were black and approximately 1% (11/1265) had HbAS. There were no clinically detected adverse events at Mexico City's high altitude (7350 feet above sea level). Black athletes continue to participate in the National Football League (NFL) (60% of players are black), the National Basketball Association (NBA) (over 80% of players are black), and in college athletics (34% of football players and 45% of male and 33% of female basketball players are black). Many U.S. black athletes compete at high altitude in Denver, Colorado (5280

feet above sea level), experiencing frequent exposures to reduced oxygen tension, with rare reports of adverse events, although one NFL player with HbAS suffered splenic complications.

Conditions Favoring Sickling

Acute, intense exercise has multiple effects that could potentially trigger sickling of HbS containing erythrocytes. These effects include a decrease in plasma volume and pH and increases in sympathetic outflow, oxidative stress, heat production, microvascular perfusion, tissue hypoxia, and release of inflammatory mediators, These detrimental effects are balanced by the benefits of chronic physical training which include a reduced resting heart rate and blood pressure, improved autonomic system balance, increased maximal myocardial oxygen uptake, improved endothelial function and vascular compliance, metabolic and neuromuscular modifications and training-specific muscle adaptations.[15]

The Potential Roles of Skeletal Muscle Function in Exercise

Skeletal muscle function is fundamental to exercise and is affected by mechanical, metabolic, hormonal, and neuronal stressors. The consequences of these stressors depend on the degree of loading, the number of contractions and the thresholds of protective mechanisms for stress-induced damage. The threshold is affected by fiber type, number and oxidative content of mitochondria, energy reserves, speed and strength of fiber contraction, and fiber size The major types of muscle fibers are Type I (slow twitch), Type IIa (fast twitch), and Type IId (x) (fast twitch). Slow twitch fibers have higher aerobic capacity, myoglobin content and fatigue resistance while fast twitch fibers have higher glycolytic capacity, glycogen content and sarcoplasmic reticulum (SR) development. Contraction speeds are slower for the fast twitch fibers compared to the slow twitch fibers. The three types of fibers are further distinguished by different myosin heavy chains.

Black, African men had significantly more Type IIa fibers (49 vs. 42%) and fewer Type I fibers (33 vs. 41%) than Caucasian men.[16] The muscle activities of glycolytic pathway enzymes were 40 to 76% higher in black than Caucasian men and the average creatine kinase nearly twice as high. Obese black women have been reported to have a higher percentage of Type IIb fibers and a lower percentage of Type I fibers than Caucasian women.[17]

A study of young, military recruits with Exertional Heat Illness (EHI) in Taiwan found that 24 of 37 with EHI developed rhabdomyolysis and that the percentage of Type II fibers was 66% in controls, 75.5 % in individuals with EHI alone, and 81% in those with EHI and rhabdomyolysis.[18]

Other Genetic Factors Influencing Muscle Function

Rapid rises in cytoplasmic calcium are required for skeletal muscle contraction. The increases in calcium are the result of calcium release through type I ryanodine receptors (*RYR1*). Analysis of *RYR1* is used as a screening test for susceptibility to Malignant Hyperthermia. A possible relation between malignant hyperthermia, EHI, and rhabdomyolysis has been proposed. Small clinical series have suggested a relationship between *RYR1* variants and Malignant Hyperthermia and co-existent HbAS. [19] Other potential genetic factors contributing to sudden death in

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African Americans include polymorphisms in the cardiac sodium channel gene, *SCN5A* Y1102, associated with increased risk of sudden death and ventricular arrhythmias in black adults. [20]

Lessons from the Military Experience

Individuals with HbAS could be at increased risk for EHI due to the polymerization of sickle hemoglobin in an exercise induced hypoxic, dehydrated, acidotic, hyperthermic environment. Agonal hypoxia causes extensive sickling in these individuals and may not have a causal role. There is no evidence that antemortem sickling can be differentiated from postmortem sickling in HbAS carriers who die suddenly. To study this on a population basis, all natural deaths among approximately two million Armed Forces Recruits were reviewed from 1977-1981. The prevalence of HbAS was estimated based on the number of African American recruits (8%) plus 0.08% of the recruits from other races. Fewer than 10% of the sudden death cases were tested for hyperthermia or rhabdomyolysis. The calculated number of African American HbAS recruits had a 30-fold increased relative risk of exercise-related death compared to those without hemoglobin S ($p < 10^{-6}$).

Excess mortality related to exercise has been investigated in military recruits and may help evaluate interventions to reduce the risk for participation in college level athletics. [21] [22] [23] Half of the cases of military recruit deaths had fatal EHI with exertional rhabdomyolysis, heat stroke, and/or acute renal failure although requirements for life support were delayed. The other half suffered idiopathic sudden death in cardio-pulmonary arrest. About 1 out of 15 to 1 out of 30 of these cases were found to have acute EHI as the cause of sudden death, however only 3% were screened for EHI. The spectrum of clinical presentations described in military recruits included collapse, rapid progression of muscle pain, slowly developing painful muscle(s), and hyperthermia.

In response to these findings, an interventional program was devised when the Wet Bulb Temperature Index increased. The program included limiting exercise intensity, reducing work/rest ratios, ordering observed water consumption and use of light clothing in hot weather conditions if possible. Both Test and Control groups used prompt evaluation and triage of any recruit with early signs of EHI including failure to keep up with group activities, determination of field rectal temperature, cooling, rehydration and rapid transport to medical facilities. The intervention reduced deaths in both the HbAS cohort (relative risk for death = 0.06 [$p<0.0001$]) and the HbAA groups compared to those not receiving the intervention. Both idiopathic and EHI deaths were prevented by the intervention. [22]

In a small cohort of military recruits, presumptive alpha-thalassemia was an additional factor reducing risk for exercise related death. The lower HbS concentration in the cells of those with HbAS-alpha thalassemia may theoretically have reduced HbS polymerization. .

As of the date of the 2010 workshop, in collegiate football, 18 deaths related to sickling were reported in the lay press since 1974. Of the sixteen cases reported during the last decade, ten of the individuals had HbAS. [24] Individuals with HbAS represent 3% to 4% of National Collegiate Athletic Association (NCAA) Division I football players. The NCAA attributes the deaths to an “Intensity Syndrome,” not Exertional Heat Illness. The deaths were seen as metabolic complications and are not usually sudden (within one hour). For each reported death, there are a total of 35 non-fatal events.

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Studies in maximally exercising normal individuals demonstrate that hypoxemia and lactic acidosis occur within 5 minutes. [25] In military recruits with HbAS, the percentage of sickled cells in venous blood increased with exercise and to a greater extent with increased altitude. [26] However, performance was not affected. In contrast, sickling was virtually absent in arterial blood suggesting that sickling may have been reversed by exposure to oxygen tensions in the pulmonary microcirculation

Reports from the NCAA and Responses

The reported deaths in NCAA Division I athletes all occurred during conditioning, not during game play, either in sprinting or speed drills and in one case with weight lifting. Reports of the clinical presentation of events attributed to a proposed "sickling syndrome" suggest that affected individuals experience weakness but do not exhibit slowly evolving, painful muscle cramping, and do not lose consciousness. A syndrome of rhabdomyolysis is apparent in these individuals with typical laboratory findings of hyperkalemia, acidosis, elevated creatine phosphokinase and renal failure secondary to myoglobinemia. The hypothesis that exercise-induced sickling triggers vaso-occlusion in muscle, which in turn precipitates rhabdomyolysis, has not been demonstrated empirically. Alternative explanations were proposed, such as rhabdomyolysis triggering a reinforcing cascade of events in athletes with HbAS that intensified the rhabdomyolysis syndrome, making a fatal outcome more likely. These discussions highlighted the need for further research in this area.

Altitude with concomitant hypoxia and the potential adverse influence of dietary supplements in the setting of strenuous exercise may also play a role in poor outcomes in individuals with HbAS. Anecdotal reports of college athletes suggest that sickling precedes rhabdomyolysis. A muscle biopsy study in non-athletes revealed remodeling of the microvasculature in muscles in individuals with HbAS characterized by lower capillary density, lower capillary tortuosity, and enlarged microvessels as compared with controls. [27] In a small series, individuals with HbAS were found to have higher mean blood viscosity at rest compared to healthy controls that was normalized by hydration during exercise. [28]

Interventions to mitigate this symptom complex worthy of further study include taking longer breaks during practice and shortening intensity and length of practice sessions held in hot weather or high altitude. A known genetic predisposition to malignant hyperthermia might pose a genetic risk factor in conjunction with HbAS. HbAS phenotypes may be the result of interactions between conditioning status and genetic influences such as muscle fiber types and regulation of cellular calcium and sodium flux.

Interest in the potential risks of participation at college level athletics for individuals with HbAS was brought into focus in 2007 by the National Athletic Trainers' Association (NATA) consensus statement, "HbAS and The Athlete." [29] NATA recommended that athletes with HbAS can participate in all sports; however screening and simple precautions should be implemented to help prevent deaths. Although not an evidence based review, the case for screening was determined to be "strong" by NATA. [30] Coaches and athletes should receive education to reduce risks and to understand the response to serious complications. The Sickle Cell Disease Association of America (SCDAA) did not support the consensus statement. The NCAA recommended that member institutions test student athletes for HbAS in 2009. In April

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2010, the NCAA adopted legislation that required screening of athletes. This action required that incoming Division I athletes must be tested for HbAS, show proof of a prior test, or sign a waiver releasing an institution from liability.

Potential Role of Pathology Methods

Pathological evaluation of several individuals with delayed death, survival long enough for medical intervention, determined that all had exerted effort beyond their conditioning level and all presented with metabolic acidosis soon followed by rhabdomyolysis, renal, failure, and DIC.[31] Individuals with sudden or rapid death who experience cardiac arrest prior to medical intervention should be evaluated using toxicology, pathology, and electrocardiography when feasible. Hemoglobin determination by high pressure liquid chromatography (HPLC) is the method most commonly used to diagnose HbAS at autopsy although it may be confounded by inadequate sampling or sample degradation. Histologic diagnosis has been used to identify HbAS cases with a very high degree of sensitivity and specificity even years after the time of death.[31] Establishment of the diagnosis postmortem does not specifically address whether sickling occurred antemortem or postmortem.

Research Opportunities Related to Sudden Death in Athletes

- Conduct a large epidemiological study to address some of the most pressing public health issues of HbAS. Prospective studies could collect information about other morbid states and determine methods to establish the causes of death in athletes and the occurrence of lesser, but important, clinical events. The study should:
    - Incorporate information from the NCAA database of college athletes including outcomes data for athletic related sports
    - Assess preventive strategies such as the effects of training and hydration methods in college athletics and/or the military
- Evaluate the use and impact of a standardized content educational message about the risks of injury and death in athletics for people with HbAS.

ETHICAL, LEGAL AND SOCIAL IMPLICATIONS OF SICKLE CELL TRAIT SCREENING PROGRAMS

The identified impediments to research included under-representation of the African-American community in pre-clinical and translational research projects and limited study in health disparities research.

Ethical Considerations in Screening Athletes for Sickle Cell Trait

A potential framework was proposed for a public health ethics analysis to identify data needs and ethical implications of programs, policies, and interventions following the implementation of NCAA policy on HbAS screening of athletes.[32]

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1. What are the public health goals for the proposed intervention, policy or program? These include prevention or reduction in the risk of exercise-related morbidity/mortality associated with HbAS.
2. How effective is the intervention, policy, or program in achieving its stated goals? The more burdensome the program, the stronger the evidence that is needed.
3. What are the known or potential burdens of the program? These may include unwarranted loss of opportunities, risks to privacy and confidentiality, a potential to stigmatize the HbAS athlete, a potential to induce anxiety or fear in the athlete or other burdens.
4. How can the burdens be minimized? One method would be a universal program to reduce heat/exertional illness among athletes.
5. Is the program implemented fairly? Inequalities and programs targeted to "high-risk" groups must have strong justification.
6. How can the public health benefits and the accompanying burdens be balanced? The benefits of the program must outweigh the identified or anticipated burdens.

Bioethics, Behavioral, and Social Sciences Research

The diagnosis of the carrier state for a genetic disease may be associated with serious health problems. This has been highlighted by a study of carriers of glucocerebrosidase mutations that identified a five-fold increased risk for Parkinson's disease.[33] The potential link between HbAS and exercise related illness could represent another such association.

The passage of the National Sickle Cell Anemia Control Act in 1972 (Public Law 92-294) emphasized screening and counseling programs, development and dissemination of information and educational materials, and research in the "…diagnosis, treatment, and control of sickle cell anemia." Problems were identified in the subsequent surge in interest in sickle cell disease.[34] These problems included a potential overemphasis on prevention of births of infants with sickle cell disease as the goal of genetic counseling, the portrayal of HbAS as a personal health hazard and determination of the harms and risks of a testing program. These risks included psychological impact, stigmatization, and the potential for discrimination.[35]

Ethical and Social Considerations in Sickle Cell Counseling

HbAS/sickle cell disease counseling takes place in four contexts: premarital (required by some states), prenatal, neonatal and community based. The predictive uncertainty of genetics and genomics is illustrated by difficulties in predicting the clinical phenotype of individuals affected by sickle cell disease when the genotype is well described.[36]

Although newborn sickle cell screening is mandated in all 50 states, information about HbAS status may be poorly communicated to affected individuals.[37] Counseling and education are uncommonly conveyed outside of the family resulting in perpetuation of misinformation. Imparting genetic information affects the entire family including misattributed parenthood. Confidentiality of results and biospecimens must be considered. The prevalence of HbAS varies widely in racial-ethnic groups and affects values about health and wellness and self image of affected individuals.

Sickle Carrier Detection – Lessons Learned Over 75 Years

The diagnosis of HbAS was initially made by microscopic examination of blood. [38] More than 8% of specimens from at risk individuals had preparations compatible with HbAS. In 1949, the solubility test and use of electrophoretic mobility were introduced to determine the presence of HbS and a diagnosis of HbAS.[39]

The lack of counseling, use of poor methodologies and poorly trained personnel reduced enthusiasm for mass screening following passage of the National Sickle Cell Anemia Control Act.[40] In the 1980's, genetic counseling had limited impact on reproductive decision making.[41] Newborn screening for sickle cell disease received a boost when the results of the Penicillin Prophylaxis in Sickle Cell Disease (PROPS) trial were published. [42] This randomized trial concluded that children should be screened in the newborn period for SCD and that those with sickle cell anemia should receive prophylactic therapy with oral penicillin by four months of age to reduce pneumococcal associated morbidity and mortality. Individuals with HbAS are also detected by newborn screening.[43] In 2005, of 4,190,705 US births, 72,111 were identified as hemoglobin variant carriers including HbS (53,532), HbC (14,814) or HbE (3,695). Carrier follow-up should include notification of the carrier's parents and provision of both immediate education and counseling and when the carrier reaches reproductive age. Inadvertent side effects of carrier testing for HbAS including decreased self-image, parental overprotection and discrimination with respect to marriage, employment, or insurance have made the process more difficult.[44] Misdiagnosis of carriers as individuals with SCD may contribute to oversight of other serious medical problems.

Informing the Public Health Agenda with Data

Efforts to bring sickle cell screening to the local community in the early 1970's encountered difficulties.[45] The public health risks associated with SCD were seen as low compared to hypertension and environmental diseases. The community had concerns about discrimination and counseling messages that were insufficiently conveyed or understood. Similarly, a pilot education and counseling program did not succeed due to inadequate staffing, poor follow up, and insufficient counselor knowledge.

There are currently two Health Resources and Services Administration (HRSA) data collection efforts. These are the SCD Newborn Screening Initiative (SCD-NBS) which includes a national coordinating center and 17 grantees participating in a minimum dataset project and the SCD Treatment Demonstration Program (SCDTDP). Both programs depend on telephone interviews, requests for written information, clinic and home visits, and encounters with community-based organizations (CBO's). The expected outcomes of the data collection are increased SCD knowledge for families including affected children and carriers of HbAS and to improve NBS and carrier follow-up activities. Recommendations from the initiative include the use of multicultural-multilingual educational media outreach, collaboration with CBO's to increase culturally appropriate educational materials, and targeting diverse and emerging populations through the use of culturally diverse staff members and support groups. Unmet needs include the creation of a standardized newborn screening follow-up procedure in all states, and standardization of referral and treatment protocols for both subspecialists and primary care providers.

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The Secretary's Advisory Committee on Heritable Disorders in Newborns and Children (SACHDNC) was charged with evaluating research and findings on HbAS status and health outcomes; the impact of the NCAA recommendations on the affected population, community service providers, and public health; current guidelines on HbAS status screening; and recommendations on HbAS screening.

In a letter to the Secretary, HHS, on June 14, 2010, the Committee made several recommendations. [46]

- All individuals should know their risk for carrier status for various inherited genetic conditions such as sickle cell disease.

- Genetic testing or screening should not be a pre-requisite for participation in athletic endeavors.

- Evaluation and screening for sickle cell disease and other genetic conditions should take place within the individual's medical home. That evaluation should include and assurance of the privacy of genetic information.

- All potential athletes should receive annual education on safe practices for prevention of exercise and heat related illnesses.

- Research is needed to determine if some athletes with sickle cell trait are at increased risk of exercise-related sudden death.

Societal and Public Health Implications for Sickle Cell Trait and Community Based Participatory Research Programs

Community Based Participatory Research may enable the study of changing social, political and environmental conditions that affect health outcomes.[47][48] The study of HbAS falls within this approach that includes participation by the affected community as defined by a sense of identify and purpose, not geography. Successful education and outreach efforts in HbAS will engage the public, the athletic community, agencies and programs to effect change in the current approach. In this type of methodology the location of power is in the research process.

Important public health concerns regarding HbAS that can be addressed include:

- Protection, Non-discrimination and Stigmatization of the carrier
- Practical recommendations communicated to communities, individuals, professionals (medical and athletic) and the general public
- Knowledge, Education and Counseling
- Public, Community and Stakeholder Roles and Level of Engagement
- Harmonizing Science, Law, Policy, Values, Ethics, Clinical Utility and Service
- Monitoring and actions for new information including mortality reports and new policies

<u>Research Opportunities Related to Implications of HbAS Screening Programs:</u>

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- Assess the risks and benefits of the NCAA HbAS screening program as an example of "occupational" genetic testing.

- Assess the risks including stigmatization, ineffective communication and benefits of carrier state detection for athletes and in general.

- Evaluate implications for societal, community, health care provider and individual responses and knowledge bases

Research Opportunities Related to Behavioral, Social and Ethical Research:

- Evaluate the impact of disease construction – The impact on individuals and health care of establishing HbAS as a disease.

MEDICAL KNOWLEDGE BASE FOR SICKLE CELL TRAIT

Background information:
It is known that HbAS-containing RBC can be induced to sickle *in vitro* within minutes.[49][50] Hypoxia is required and the sickling process is promoted by acidic pH, hypertonicity, and altitude. Exercise produces conditions conducive to sickling. For example at an altitude of approximately 1300 meters, venous pO_2 falls to the mid-30 mmHg range and pH also decreases and is associated with an increased percentage (1% to 2%) of sickling red cells in the blood.[51] The effect is more marked at a height of 4000 meters. Fluid restriction during lengthy (40 minutes) treadmill exercise and thermal stress is also associated with peripheral venous blood sickling.[52] Whether or not these effects are relevant *in vivo* during the one second or less circulatory transit time in the systemic capillaries is not known.[53][54] In addition to changes in hemoglobin polymerization, HbAS carriers have higher rigidity of red cells at rest and after a brief supramaximal exercise period.[55] Exercise-induced plasma soluble vascular cell adhesion molecule-1 (sVCAM-1) increase is higher and more prolonged in athletes with HbAS.[56] This may signal microcirculatory disturbances with endothelial activation and increased potential for adhesive events. In studied subjects, alpha-thalassemia reduced the observed effects, suggesting a possible link to HbS-polymerization.

Is Sickle Cell Trait a Risk Factor for Venous Thrombosis?

In 1979, of more than 65,000 consecutively admitted black males at 13 Veterans Administration hospitals 7.8 % had HbAS. [57] No age-dependent differences in frequency of HbAS were found. HbAS did not have an effect on average age at hospitalization or death, overall mortality, or length of hospitalization. The only diagnoses in which there was a difference in frequency were "essential" hematuria and clinically diagnosed pulmonary embolism (1.5% in individuals with HbAA compared to 2.2% in those with HbAS).

In individuals with HbAS compared to HbAA controls laboratory parameters are consistent with activation of blood coagulation. D-dimer, a reliable indicator of future risk of venous thrombosis, is higher.[58] Similarly, thrombin anti-thrombin complexes (TAT) and prothrombin fragment 1.2 are elevated in individuals with HbAS compared to controls. The studies were sex matched but females represented 83% of the subject population.

A case-control study [59] identified HbAS as a risk factor for venous thromboembolism (VTE). The risk of VTE was approximately twice as high in blacks with HbAS compared to controls. HbAS also appeared to confer a four-fold increased risk of pulmonary embolism. The study concluded that, "…HbAS is a more important cause of VTE among blacks than is the prothrombin [G20210A] mutation among whites." The risk was even higher for a small sample of African American women taking oral contraceptives.[60]

A potential coagulation role for microparticles, phospholipid microvesicles of 0.05 to 1.5 micrometers in size,[61] derived from red cells, platelets, monocytes, and endothelial cells, has also been suggested.

Sickle Cell Trait and Pregnancy

The available medical literature is largely based on retrospective cohorts from single institutions. Screening for hemoglobinopathies in study populations has not been consistently thorough and complications of pregnancy had variable or incomplete definitions. Control groups were not incorporated into the studies and confounding variables such as co-existent alpha-thalassemia or iron deficiency were not evaluated.

Single case reports during pregnancy describe HbAS as the cause of pulmonary embolism, sudden death,[62] death associated with cardiomyopathy,[63] death associated with aorto-caval compression,[64] hypopituitarism,[65] and proliferative retinopathy in association with gestational diabetes and hypertension.[66]

Asymptomatic bacteriuria in women with HbAS during pregnancy has been studied in retrospective cohorts. A two-fold increase,[67] 1.4-fold increase,[68] and no increase [69] have been reported. The occurrence of pyelonephritis in the series is higher in individuals with HbAS but is associated with risk factors including prior episodes of pyelonephritis. Lower HbS concentrations are associated with a lower risk of urinary tract infection in pregnancy. [70] Screening for bacteriuria at first prenatal visit has been recommended.

In one prospective cohort study, preeclampsia occurred twice as frequently in women with HbAS although women with HbAS had a two-fold increased history of preeclampsia in a prior pregnancy.[71] Other studies failed to confirm the report.[72][73][74] Placental abnormalities including amniotic fluid infection and meconium histiocytosis have been noted to be three- to nine-fold more common in pregnant women with HbAS and associated with fetal loss after the first trimester. [75, 76] Other studies have not shown an increase in pre-term delivery rate.[70][72]

Renal Perspectives Including Malignancy

Hemoglobin S is known to polymerize at low oxygen tension, high hydrogen ion concentration, higher intracellular HbS concentration and under conditions of high osmolality. These conditions all exist in the renal medulla and have been shown to be associated with fewer vessels and a greater degree of disruption of the vasa recta in the kidney.[77]

Hematuria is possibly the most common renal manifestation of HbAS.[57] Hospitalized African American veterans with HbAS have an approximate two-fold increased risk of hematuria (2.5%). In addition, there are numerous case reports that describe episodes of papillary necrosis.

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The community prevalence of Chronic Kidney Disease (CKD) is 7 – 8%. In diabetic populations the prevalence ranges from 18 to over 30%.[78][79][80] A contributing role for HbAS in this context has been sought in a small series of patients.

A recent epidemiological study reported that HbAS prevalence is twice as frequent in African American patients with End Stage Renal Disease (ESRD)($p < 0.001$) and HbAC is ~2.5 times more prevalent ($p = 0.01$) than African American control subjects.[81] Although these relationships might reflect an effect caused by the variant hemoglobin, alternatively HbAS and HbAC might be surrogate markers for other risk factors that are associated with African ethnicity . HbAS occurred more frequently in a black population with autosomal dominant polycystic kidney disease and was associated with an earlier onset of ESRD ($p < 0.003$).[82]

Renal medullary carcinoma is a rare tumor that arises centrally in the renal medulla and grows rapidly in an infiltrative pattern to invade renal sinuses. Patients with HbAS account for nearly 90% of approximately 130 reported cases.[83, 84] The prognosis is generally poor with diagnosis made at the time of metastatic disease and poor responses to treatments.[85, 86]

Other Potential Manifestations of Sickle Cell Trait

Most information is in the form of case reports. The impact of HbAS on severe malaria in endemic areas also confounds analyses.[87]

Since the initial description of splenic infarct in HbAS in the 1940's, over 80 cases have been reported.[88, 89] The event may occur one to two days after exposure to high altitude and also may occur during unpressurized air travel. From 1986-2006, 25 cases were identified at the Colorado Sickle Cell Center.[90] All were male, ages ranged from 4 to 40 years. Thirty-six percent were African-American, 36% non-Hispanic white, 16% Hispanic and 12% unknown. The infarcts occurred at greater than 7500 feet elevation for non-Denver residents and greater than 9000 feet for Denver residents. Treatments have included return to lower altitudes and splenectomy.

More recently, there has been a report of an increased rate of invasive pneumococcal disease in children with HbAS and HbC trait.[91] Children born from 1996 to 2003 in Tennessee were followed through 2005 or to their fifth birthday, to evaluate the onset of a pneumococcal event or death. The introduction of pneumococcal conjugate vaccine (PCV7) led to reduced rates of invasive pneumococcal disease in all children and an 82% reduction in children with HbAS, but the rates in HbAS and HbC trait remained elevated (adjusted RR of ~ 1.8).[92] The differences were identified after adjusting for known high-risk conditions: age, asthma and race.

The role of HbAS as a risk factor for stroke has been debated.[93][94] A small study in Guadeloupe found that the risk of ischemic stroke in HbAS was less frequent (4%) than in controls (8.5%).[95] Potential co-factors for stroke risk include hypoxemia, acidosis, dehydration, and viscosity.

Following blood bank storage, red cells from HbAS donors survive normally in normoxemic recipients.[96] However, use of these cells for infants or patients with sickle cell disease undergoing exchange transfusion is not recommended. Removal of white blood cells by filtration is commonly practiced to reduce the risks of alloimmunization, febrile reactions, CMV infections, and potentially immune suppression. In the case of HbAS donors, leukodepletion filtration often leads to line occlusion and loss of the unit. HbS polymerization due to collection

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into an acidic citrate anticoagulant environment and low oxygen tensions appear to explain filter failures.[97] The addition of air to storage bags to increase oxygen levels greatly improves the chances of filtration success.[98]

Research Opportunities to Improve the Medical Knowledge Base:

- Are there potential genetic modifiers of phenotypic expression of HbAS? What is the impact of co-morbid conditions such as asthma or diabetes mellitus on clinical outcomes in individuals with HbAS?
- Evaluate the development of animal models for hypothesis testing.
- Determine the potential interaction of HbAS with the development of renal disease, including the significance of HbC as a risk factor for renal disease.
- Quantify the impact of known modifiers of disease severity for SCD including HbF level, alpha-thalassemia and HbC.
- What risk factors combined with HbAS result in thrombosis? Are there genetic factors? Is there a role of microparticles in HbAS?
- Viewing HbAS as a genetic polymorphism, how might it interact with other genetic polymorphisms to influence the development of clinical disease?
- Improve understanding of the protective effect of HbAS against malaria.
- Improve understanding of the potential pathophysiology of HbAS containing red blood cells, including red cell membrane abnormalities, vascular wall interactions, and impact on immune function and infectious disease.
- What is the possible impact of co-factors such as intense athletic exercise, high altitude exposure and pre-ecclampsia.

Summary and Conclusions

The purpose of the workshop was to identify research topics about potential health risks of HbAS. Various mechanisms of sudden death in young athletes were discussed and a genetic basis for some of the outcomes including the risk of rhabdomyolysis was proposed. Case reports on sudden deaths include potentially important information for consideration. However, these anecdotal reports lack of uniform definitions and add uncertainty. A more methodologically sophisticated approach is needed. However, from a practical perspective, implementation of preventive measures for EHI for all athletes seems to be warranted. Other potential complications and aspects of HbAS should be investigated. These include renal and coagulation complications and access to genetic counseling. Ethical, social, and behavioral research should be conducted in concert with investigations to improve understanding of the basic biology and the clinical implications of HbAS. This condition also provides an opportunity to conduct multidisciplinary research. NIH, through its Clinical and Translational Science Award Program, in collaboration with CDC and HRSA plans to support research on the clinical implications for individuals with Sickle Cell Trait. Research in HbAS is also in alignment with the NHLBI Strategic Plan, specifically, Goal 2; “to improve understanding of the clinical mechanisms of disease and thereby enable better prevention, diagnosis, and treatment”.[99]

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From: Klossner, David
To: NCAA Head Athletic Trainers (head_ath_trainer@mylist.net); Team Physicians
Subject: NCAA Catastrophic Insurance Plan - Division I information
Date: Thursday, May 24, 2012 2:38:00 PM

Division I Head Athletic Trainers and Team Physicians,

The following information has been shared with your Director of Athletics and Senior Woman Administrator. You are likely aware of legislation recently passed for Division I Men's Basketball relating to on-campus evaluations for prospective student-athletes. Per NCAA records regarding this legislation, the intent is as follows: "In men's basketball, an institution may conduct an evaluation of a high school or preparatory school senior or a two-year college prospective student-athlete who has exhausted eligibility or a four-year college prospective student-athlete on its campus or at a site at which it normally conducts practice or competition, as specified."

The implementation of this legislation has caused questions regarding insurance coverage for prospective student-athletes. The purpose of this correspondence is to summarize options available related to this coverage. Questions pertaining to the legislation itself should be directed through your standard compliance channels.

NCAA Catastrophic Injury Insurance Program

Among the primary financial benefits the NCAA funds to member schools is the NCAA Catastrophic Injury Insurance Program, which assists student-athletes and other athletics participants who suffer catastrophic injuries while participating in an intercollegiate athletics activity. However, the NCAA Catastrophic Injury Insurance Program is limited in its coverage of prospective student-athletes. The Catastrophic policy covers only "a prospective student that has graduated from high school and signed an irrevocable commitment to participate in a Qualifying Intercollegiate Sport (or its equivalent for cheerleading) at a Participating School." If the prospective student-athlete coming to your campus for a try-out does not meet these requirements, **they will not have coverage** under the NCAA Catastrophic program. Because the primary purpose of the Catastrophic program is to cover full-time student-athletes at member institutions, the NCAA has been careful to extend coverage only to these individuals in order to maintain the long-term viability of this critical program for the benefit of NCAA student-athletes.

While the NCAA Catastrophic program will not be extended beyond these covered individuals, the program’s insurer, Mutual of Omaha, has agreed to offer optional coverage to institutions wishing to provide catastrophic medical coverage to prospective student-athletes not already covered. Please note that coverage and limits for this program will differ from the NCAA's Catastrophic program.

This optional Prospective Student-Athlete Catastrophic coverage is available to all NCAA institutions with the exception of those located in New York (product not yet available). There is variability within the product in certain states as follows: for those institutions located in Connecticut, Louisiana, and Maryland, there are no Home Health Care/Custodial Care or Extended Care Facility benefits available. Please find following a summary of benefits for eligible institutions:

Option 1:
Benefit Maximum $5,000,000 Catastrophic Accident Medical
Total Institutional Premium $600 Per Year

Option 2:
Benefit Maximum $10,000,000 Catastrophic Accident Medical Maximum
Total Institutional Premium $900 Per Year

Benefit Period: 10 years from accident date

Deductible for each Covered Accident (see NCAA insurance certification legislation): Participating NCAA Group Basic Accident Medical Program Institutions $75,000-- All other eligible institutions $90,000

NCAA Group Basic Accident Insurance Program

The NCAA Group Basic Accident Insurance Program has historically covered prospective student-athletes and their chaperones while on "official" visits. Effective April 5, 2012, the coverage is being expanded to include unofficial visits as well. Please note that a $10,000 per injury sublimit applies with respect to chaperones only.

If you have questions regarding the information contained in this communication or would like more information about the optional catastrophic coverage, please contact Donna Hill at Summit America Insurance at 866/528-6222 or dhill@summitamerica-ins.com

Division I Basketball

- Try-outs - Effective now.
  - similar legislation has been in place for Division II
- Summer Workouts - Effective now.

Division I Men's Basketball

- In men's basketball, a student-athlete who is enrolled in **summer** school may engage in **required** weight-training, conditioning and **skill-related instruction** for up to eight weeks (not required to be consecutive weeks). Participation in such activities shall be limited to a maximum of eight hours per week with not more than two hours per week spent on skill-related instruction. An individual who is not eligible to use the exception to **summer** school enrollment (Bylaw 17.1.6.2.1.1.4.1) may participate only during the period of the institution's **summer** term or terms (opening day of classes through last day of final exams) in which he is enrolled. *(Adopted: 1/14/12)*
  - Excerpt from Rationale: This proposal will provide opportunities for both academic and athletic improvement, which will, in turn, contribute to greater retention of student-athletes. Allowing limited athletically related activities in a structured environment will not significantly impact competitive equity, but it will prove beneficial in establishing stronger relationships and enhancing the student-athlete's bond to the institution.
  - Impact on Student-Athlete's Time (Academic and/or Athletics): Up to eight hours per week of required athletics activities for up to eight weeks during the summer.

Division I Basketball

ON CAMPUS EVALUATIONS (Tryouts)

Who? High school seniors;
2 year college PSAs who have exhausted their eligibility;
4 year transfers.

When? After the PSAs season is over and before the first day of classes for fall semester.
During an Official or Un-Official visit (i.e., on our campus)

How? Two hours Maximum;
Can involve current SAs who are eligible (enrolled full time);
Must count in your 20 or 8 hour depending on whether you are in our out of season;

Number? One per PSA (like Official visits, one in high school, and one after enrollment at 2 or 4 year college.)

Health and Safety: Prior to participation in a **tryout**, a student is required to undergo a medical examination or evaluation administered or supervised by a physician (e.g., family physician, team physician). The examination or evaluation must be administered within six months prior to participation in the **tryout**. The medical examination or evaluation may be conducted by an institution's regular team physician or other designated physician as a part of the **tryout**.

- Check with your compliance office for full details and a copy of the Q&A document used for the proposal.

- There is more to the process for a tryout than just allowing it to be permissive. This includes discussions and approvals of Athletic Administration, coaches, compliance office, risk management, physicians signing off on the type physical for the tryout, waivers, medical costs due to injury, follow-up care for those injuries, and the role and liability of campus athletic trainers, parents and the recruit.

1. Does this process allow for the ability to do a physical or obtain a physical that would be adequate for your institution. Men's basketball players have the highest risk of cardiac sudden death and in addition to the SCT screening it is difficult for a Division I to account for both of these tests during the tryout period if they are normally conducting them as part of the basketball medical evaluation.

- The legislation is flexible in that institutions may use a physical that was conducted by another physician (e.g., personal physician, school physician) within six months of the on-campus evaluation or within six months of the prospect's first participation in his immediately completed season.

2. Are schools prepared to or have the ability to pay for medical care if an injury occurs. **Can an institution pay for medical care after injury.**

- The legislation permits an institution to pay for medical expenses if an injury occurs. This issue should not be any different than situations in which prospects participate in voluntary conditioning activities with strength and conditioning coaches during the summer.

3. It appears that these recruits are not covered under the NCAA Catastrophic Insurance Plan.

- NCAA is currently in discussion with carrier.
- Institutions need to evaluate their situations and make appropriate decisions.

4. What can be provided during a try-out?

- An institution may provide athletic training room and medical services as it sees fit.
- It may provide locker room and shower facilities and equipment and apparel on an issuance and retrieval basis.
- Prospects on an official visit may receive meals and snacks as is permissible pursuant to official visit regulations.
- Prospects on unofficial visits may receive water and/or electrolyte replacement drinks (e.g., Gatorade, Powerade etc.) in conjunction with the tryout.

5. Are medical staff required to be at these tryouts?

- Medical personnel are not required to be at the evaluations.

Message from NATA-CUATC for Div 1 Head AT or Basketball AT

To: College Head Athletic Trainer or Men's Basketball Athletic Trainer

RE: New NCAA Legislature allows Men's Basketball PSA to tryout on your campus.
(NOTE: Recently Adopted NCAA Legislation listed below for your reference)

The NATA-CUATC has been in contact with the NCAA and others to present you with the following information meant to clarify and aid you and your institution as a result of recent changes to the NCAA rules in regards to Men's Basketball PSA tryouts.

As a result of this recently adopted legislation, the NATA- CUATC encourages you to discuss this with your athletic administration and risk management personnel to interpret how your institution will enact this new legislation. Below is a brief summation of the recent changes and discussion points that should be considered during these discussions. The text of the legislation is also enclosed for your convenience.

ON CAMPUS EVALUATIONS (Tryouts)

Who? High school seniors;
2 year college PSAs who have exhausted their eligibility;
4 year transfers.

When? After the PSAs season is over and before the first day of classes for fall semester.
During an Official or Un-Official visit (i.e., on our campus)

How? Two hours Maximum;
Can involve current SAs who are eligible (enrolled full time);
Must count in your 20 or 8 hour CARA depending on whether you are in our out of season;

Number? One per PSA (like Official visits, one in high school, and one after enrollment at 2 or 4 year college.

Health and Safety: Prior to participation in a **tryout**, a student is required to undergo a medical examination or evaluation administered or supervised by a physician (e.g., family physician, team physician). The examination or evaluation must be administered within six months prior to participation in the **tryout**. The medical examination or evaluation may be conducted by an institution's regular team physician or other designated physician as a part of the **tryout**;

The following points should be considered by you and your institution in adoption of this legislation..

1. Most of these PSA's would not qualify for the NCAA Catastrophic Insurance. These PSA's would only qualify for the NCAA Catastrophic Insurance if they meet the following criteria.

   "a prospective student that has graduated from high school and signed an irrevocable commitment to participate in a Qualifying Intercollegiate Sport (or its equivalent for cheerleading) at a Participating School."

This information should be provided to the appropriate personnel on your campus. That may include Athletic Administration, coaches, compliance office, risk management, physician signing off on physical, athletic trainers, the parent of PSA and the PSA.

2. Each institutions' insurance provider should determine ahead of time if the PSA would be covered by the institutions athletic insurance policy if an injury occurs. This varies with each insurance plan. If the institutions' insurance does not cover the PSA, the athletic administration and/or risk management, the PSA, the parents and coaching staff should be made aware of this and understand that any costs incurred as a result of injury may be the sole responsibility of the PSA.

3. If an institution decides they would not cover medical expenses or if their insurance carries declares that the PSA would not be covered, . A "waiver" or some type of acknowledgement may need to be created to notify the PSA and their parents that any medical expense from an injury occurring would not be covered by the institution. This should be discussed with the appropriate personnel from the institution.

   If an institution decides that they would cover medical expenses it would should determine the length and extent of medical coverage and notification of such decision should be made to the PSA prior to the tryout.

4. Prior to participation in a tryout, a student is required to undergo a medical examination or evaluation administered or supervised by a physician (e.g., family physician, team physician). The examination or evaluation must be administered within six months prior to participation in the tryout. The medical examination or evaluation may be conducted by an institution's regular team physician or other designated physician as a part of the tryout;

   These physicals should be reviewed by your institutions medical staff to determine if they fill the universities requirements. It should be determined who would review these physicals. The medical examination should indicate that the PSA is cleared for exertional activities.

5. As with all student-athletes, the ability to determine Sickle Cell Trait status or allowance to Opt Out needs to be addressed by the institution. , If opting out of determining sickle cell trait status is an option the ability and time to give proper education and counseling if PSA is necessary., It is important to understand that if a SA opts out of screening for sickle cell trait all parties should follow the current recommendations and standard of care guidelines with the assumption this individual has the sickle cell trait.

6. Please remember that if a physical or treatment is being performed and the PSA is a minor, a consent to treat a minor form would have to be signed by parent or guardian.

7. It should be determined prior to the tryout if the medical staff including the athletic trainer, would be covered by the institutions malpractice insurance if they had to give care to the PSA. Likewise, the statute governing athletic training in a particular state should be considered prior to treating a student- athlete that is not officially a part of the institution's program.

8. From NCAA Representative. "This is permissive legislation so no institution is required to conduct on-campus evaluations if it has concerns about whether institutional insurance would cover such activities."

Division I Proposal 2012-2

RECRUITING -- ON-CAMPUS EVALUATIONS -- MEN'S BASKETBALL

Status: Adopted (INSERT DATE), 60-Day Override Period

Intent: In men's basketball, an institution may conduct an evaluation of a high school or preparatory school senior or a two-year college prospective student-athlete who has exhausted eligibility or four-year college prospective student-athlete on its campus or at a site at which it normally conducts practice or competition, as specified.

A. Bylaws: Amend 13.2, as follows:

13.2 Offers and Inducements.

[13.2.1 through 13.2.6 unchanged.]

13.2.7 Medical Expenses -- Men's Basketball. In men's basketball, an institution may finance medical expenses (including rehabilitation and physical therapy expenses) for a prospective student-athlete who sustains an injury while participating in an on-campus evaluation (see Bylaw 13.11.2.1) or while participating in voluntary summer workouts conducted by an institution's strength and conditioning coach with department-wide duties (see Bylaw 13.11.3.8).

[13.2.7 renumbered as 13.2.8 unchanged.]

13.2.~~8~~**9** Medical Expenses -- Sports Other Than **Men's Basketball and** Football. In sports other than **men's basketball and** football, an institution may finance medical expenses (including rehabilitation and physical therapy expenses) for a prospective student-athlete who sustains an injury while participating in voluntary summer workouts conducted by an institution's strength and conditioning coach with department-wide duties (see Bylaws ~~*13.11.3.8,*~~ 13.11.3.9 and 13.11.3.10).

[13.2.9 through 13.9.10 renumbered as 13.2.10 through 13.2.11, unchanged.]

B. Bylaws: Amend 13.11.2, as follows:

13.11.2 Permissible Activities.

13.11.2.1 On-Campus Evaluations -- Men's Basketball. In men's basketball, an institution may conduct an evaluation of a prospective student-athlete on its campus or at a site at which it normally conducts practice or competition, under the following conditions:

(a) For a high school or preparatory school senior, the evaluation may be conducted only after the conclusion of the prospective student-athlete's season and after he has exhausted high school or preparatory school eligibility in basketball;

(b) For a two-year college prospective student-athlete, the evaluation may be conducted only after the conclusion of the prospective student-athlete's season and he has exhausted his two-year college eligibility in basketball;

(c) For a four-year college prospective student-athlete, the evaluation may be conducted only after the conclusion of the prospective student-athlete's season. (See Bylaw 13.1.1.3);

(d) The on-campus evaluation may be conducted only during the prospective student-athlete's official or unofficial visit;

(e) The on-campus evaluation shall be conducted not later than the opening day of classes of the institution's fall term;

(f) Not more than one on-campus evaluation per prospective student-athlete per institution shall be permitted (applied separately to the time period in which a prospective student-athlete completes high school or preparatory school eligibility and to the time period after the prospective student-athlete enrolls full time in a collegiate institution);

(g) Before participating in an on-campus evaluation, a prospective student-athlete is required to undergo a medical examination or evaluation administered or supervised by a physician (e.g., family physician, team physician). The examination or evaluation shall include a sickle cell solubility test unless documented results of a prior test are provided to the institution or the prospective student-athlete declines the test and signs a written release. The examination or evaluation must be administered either within six months before participation in the on-campus evaluation or within six months before the prospective student-athlete's initial participation in practice, competition or out-of-season conditioning activities during his immediately completed season. In addition, the medical examination or evaluation may be conducted by an institution's regular team physician or other designated physician as a part of the on-campus evaluation;

(h) The institution's men's basketball student-athletes may participate in an on-campus evaluation, provided such participation is counted toward the applicable hourly and weekly limitations on countable athletically related activities (e.g., four hours per day and 20 hours per week during the playing season, two hours of skill instruction and eight hours per week outside the playing season). [See Bylaws 17.1.6.2-(a) and 17.1.6.2.2];

(i) The duration of the on-campus evaluation activities (other than the medical examination or evaluation) shall be limited to two hours; and

(j) The institution may provide equipment and clothing to a prospective student-athlete on an issuance-and-retrieval basis.

[13.11.2.1 through 13.11.2.4 renumbered as 13.11.2.2 through 13.11.2.5, unchanged.]

13.11.2.~~5~~**6** Medical Examinations.

13.11.2.~~5~~**6**.1 During Campus Visit. During a prospective student-athlete's official or unofficial visit to campus, a member institution, through its regular team or other designated physician, may conduct a medical examination to determine the prospective student-athlete's medical qualifications to participate in intercollegiate athletics, provided no athletics department staff member other than the athletics trainer is present, the examination does not include any test or procedure designed to measure the athletics agility or skill of the prospective student-athlete and the results of the examination are not used by the institution to deny admission of a prospective student-athlete who is otherwise qualified for admission under the institution's regular admissions criteria.

[13.11.2.5.1.1 renumbered as 13.11.2.6.1.1, unchanged.]

13.11.2.6.1.2 Exception -- On-Campus Evaluation -- Men's Basketball. In men's basketball, additional athletics department staff members (e.g., coaches) may be present during a medical examination that is conducted as part of an on-campus evaluation (see Bylaw 13.11.2.1) and the medical evaluation may include tests or procedures designed to measure the athletics agility or skill of the prospective student-athlete.

[13.11.2.5.2 renumbered as 13.11.2.6.2, unchanged.]

Source: NCAA Division I Board of Directors

Effective Date: Immediate

Proposal Category: Amendment

Topical Area: Eligibility

Rationale: This proposal was developed as part of a comprehensive review of the Division I men's recruiting model and is designed to facilitate sound recruiting decisions by both institutions and prospective student-athletes through the establishment of an on-campus evaluation opportunity. Many prospective student-athletes do not receive financial aid offers they may initially expect. Such a prospective student-athlete must continue the recruiting process after exhausting high school or two-year college eligibility. Likewise, a four-year college transfer who is not satisfied with his original institution may need to re-open the recruiting process. An institution that was not considered initially by the prospective student-athlete may have minimal information regarding the prospective student-athlete's ability and whether such an individual will be a good fit with the team. An on-campus evaluation, which might involve several prospective student-athletes, may provide valuable information for both the prospective student-athlete and the institution to make a better informed decision. Appropriate medical safeguards have been established to ensure the health, safety and well-being of the prospective student-athlete while participating in the evaluation.

Why conduct medical examinations prior to sports activity.

The health and safety principle of the National Collegiate Athletic Association's constitution provides that it is the responsibility of each member institution to protect the health of, and provide a safe environment for, each of its participating student-athletes (Bylaw 2.2.3).

Participation in intercollegiate athletics involves unavoidable exposure to an inherent risk of injury. Catastrophic sports injuries, including sudden death, are rare but tragic events.

Prior to participation in any practice, competition or out-of-season conditioning activities, the NCAA Bylaw 17.1.5 requires student-athletes who are beginning their initial season of eligibility to undergo a medical examination or evaluation administered or supervised by a physician.

The NCAA works through its Committee on Competitive Safeguards and Medical Aspects of Sports (CSMAS) to evaluate and implement relevant rules, policy and best practices as the science becomes available and medical positions statements are published.

The goal of the NCAA legislated mandatory medical examination, often referred to as a preparticipation physical evaluation (PPE), is to promote the health and safety of the athlete in training and competition, not to serve as a substitute for an athlete's regular health maintenance examination.

Goals of a sports PPE:

Primary:
1. Screen for conditions that may be life-threatening or disabling;
2. Screen for conditions that may predispose to injury or illness.

Secondary
1. Determine general health;
2. Serve as an entry point to the healthcare system;
3. Provide opportunity to initiate discussion on health-related topics.

To provide guidance in accomplishing this objective and to assist member schools in developing a safe intercollegiate athletics program, the NCAA CSMAS publishes guidelines in a Sports Medicine Handbook on topics not accessible in another easily attainable source.

These general guidelines are not intended to supersede the exercise of medical judgment in specific situations by a member institution's sports medicine staff. In all instances, determination of the appropriate care and treatment of student-athletes must be based on the clinical judgment of the institution's team physician or athletic health care team that is consistent with sound principles of sports medicine care.

Why conduct a PPE prior to exercise?

The basic intent of screening programs is to identify and withdraw from training and competition (either temporarily or permanently) those athletes perceived to be at risk in an effort to reduce the likelihood for sudden death, and to allow for preventive treatment intervention if appropriate.

What standards should the treating physician consider when conducting the medical examination?

An effective PPE screening tool identifies medical and orthopedic conditions that might affect a student-athletes ability to participate safety in a given sport. To be effective, a screening tool must identify diseases or processes that will affect the athlete, be sensitive and accurate, and be practical and affordable.

DRAFT

The SMH guideline outlining the PPE was first published in 1977. Since that time, numerous publication and resources have been made available to the membership by the medical community. Most notable is the 4th edition of the "*Preparticipation Physical Examination*" monograph setting minimal standards for the medical examination as published by six national medical associations including the American medical Society for Sports Medicine, American Academy of Family Physicians, American Academy of Pediatrics, American College of Sports Medicine, amongst others.

What does a PPE involve?

This initial evaluation should include a comprehensive health history, immunization history and a relevant physical exam, with strong emphasis on the cardiovascular, neurologic and musculoskeletal evaluation. After the initial medical evaluation, an updated history is required to be performed annually. Subsequent preparticipation physical examinations are not believed to be necessary unless warranted by the updated history or a change in the student-athlete's medical condition.

Should specific diagnostic screens be required?

Routine laboratory, cardiac, and pulmonary screening tests for PPEs remain controversial. The value of a screening test or procedure depends on two variables: (1) the predicative value of the proposed screen, which is in turn affected by the prevalence of the condition in the population being screened, and (2) the ability to reduce morbidity and mortality by identifying the condition with the screening method. The screening test should also be acceptable in terms of cost and potential side effects.

Even with advanced medical screening tools, athletes and families should still be informed by physicians and health care providers that risks still remain for athletes even after normal findings on a screening examination and that intense training and competition increase the risk for sudden cardiac death or disease progression in athletes with certain structural heart disease.

4th edition of the PPE Monograph Covers:

- Medical History
- Medications and Supplements
- Allergies and anaphylaxis
- Cardiovascular problems
- Surgical history
- Musculoskeletal injury
- Asthma
- Paired Organs
- Viral illness
- Dermatologic conditions
- Neurologic conditions
- Heat illness
- Sickle Cell Trait or disease
- Eyes and Vision
- Nutritional concerns
- Menstrual History
- Immunizations

Why mandate Sickle Cell Trait (SCT) status?

The 4th edition of the PPE does not require a sickle cell trait test. However, the NCAA has been tracking on deaths in NCAA sports since 1973 involving complications due to sickle cell trait. Division I requires student-athletes new to their campus to complete a sickle cell solubility test, show results of a prior test, or sign a written release declining the test.

Although Sickle Cell Trait screening is normally performed on all U.S. babies at birth, many student-athletes may not know whether they have the trait. The membership within Division I passed legislation based on the risk associated with sudden death related to sickle cell trait during exertional activity. SCT is associated with death in NCAA football players with a rate higher than those without. Since 1973,

experts have cited up to 19 deaths of NCAA athletes related to complications from SCT during exertion. All but two of these 19 deaths have been in Division I football players. There has been one NCAA male basketball athlete playing pick-up basketball (Division II) and one male track athlete who have reportedly died from exertional collapse associated with sickle cell trait.

In an effort to provide a more efficient and effective process for sickle cell trait (SCT) screening, the NCAA has an agreement with a national laboratory to provide three different service models for member institutions and their student-athletes to access affordable screening exams to determine SCT status as part of the student-athlete's medical examination process.

What about screening for cardiac conditions?

Sudden cardiac death (SCD) is the leading medical cause of death in NCAA athletes and represents 75 percent of all sudden death cases that occur during training, exercise or competition. In a five-year review of sudden deaths involving NCAA student-athletes, the incidence of SCD was approximately one in every 40,000 student-athletes per year.

In recent years, the NCAA has mandated medical examinations and requiring CPR, first aid, and automated external defibrillator (AED) training for coaches. Additionally, the NCAA is funding an external research grant evaluating the feasibility of additional diagnostic screening for heart conditions.

Pre-participation cardiovascular screening has traditionally been accomplished with a history (personal and family) and physical examination with further testing at the discretion of the examining physician. The American Heart Association (AHA) consensus panel on pre-participation cardiovascular screening for young competitive athletes recommended the history and physical examination combination to be the most practical and best available strategy for screening large populations. The similarities between athlete's heart and cardiac disease may identify an athlete to unnecessarily withdrawal from athletics, depriving that person of the benefits of sports. Important changes in medical status or abnormalities may require more formal cardiovascular evaluation.

The NCAA continues to monitor the science and technology involving diagnostic screening and will make appropriate recommendations as warranted.

What other components might be required during the medical examination?

Components of the PPE are decided by the medical staff of each institution. Some institutions might screen for baseline concussion assessments, mental health concerns, nutrition practices, ADHD and other medications, nutritional supplements, urine for institutional drug testing, and functional movement.

What should I do to ensure our institution is in compliance with the most recent recommendations for the PPE?

Meet with your team physician and/or medical director to discuss this process and review with our legal counsel and risk management personnel. Set up a quarterly review to ensure all student-athletes are meeting the institutional requirements prior to participating in activity and educate coaches on your expectations.

What types of diagnostic tests are on the horizon that might be added to the NCAA medical examination process?

At this time, the minimum guidelines outlined in the *Preparticipation Physical Evaluation* publication serves as standardized, systematic approach to screening for sports participation. The NCAA continues to monitor the latest scientific information available and works through its membership governance structure to discuss, proposal and adopt relevant legislative regulations. To date, additional diagnostic tests have not been identified as mandatory to screen for specific conditions that would place the athlete at an unacceptable medical risk.

How can I find out more information?

Athletics administrators can talk with their team physicians for more information. Reading the NCAA material on sickle cell trait and cardiac conditions can be a good starting point to understand the topics. The NCAA Sports Medicine Handbook will continue to be edited each year with relevant and timely recommendation to the membership on these topics.

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TO BE DISTRIBUTED

SUPPLEMENT NO. 27

Intersex Conditions and Sports Participation

Why are we talking about this –

- Postponed during transgender discussions
- CSMAS and CWA – likely targets for requests
- Media issue (Caster Semanya example)

Not creating policy – seeking statement of position to guide committees and inform our member schools

Umbrella term to describe a variety of 30 congenital conditions

- Individuals are born with sex characteristics that are not typical for either the male or female sex category.
- AKA “Disorders of Sex Development”
- 1 in 2000 people have some kind of intersex condition; though many are not identified

Note: Intersex is not the same as gender identity. Someone who has an intersex condition:

- may express their gender as identified at birth
- may identify gender different from that of birth and therefore would be considered transgender.

Almost all intersex individual identify/verify gender by age 18 and maintain birth gender assignment.

WHAT IS INTERSEX?

Intersex conditions may affect

- sex chromosomes and cause them to appear in configurations other than the typical XX (female) or XY (male).
- how sex-related hormones function in the body, such as when an individual with XY chromosomes cannot absorb or utilize testosterone.
- internal or external sex characteristics, such as cases when an individual's genitalia at birth defies easy classification as male or female.

WHAT IS THE EFFECT OF AN INTERSEX CONDITION?

- Testosterone levels and responsiveness vary widely among women, as well as among men, for reasons that include but are not limited to intersex conditions.

- Testosterone levels are just one aspect of the human body that operates to produce variation and diversity among women.
  - Women come naturally in all shapes and sizes, tall, short, large, small, strong, or weak. Some of this variation might be caused by women's diverse levels of testosterone, while some of it is caused by other influences, both genetic and environmental.

WHAT IS THE EFFECT OF TESTOSTERONE ON A FEMALE ATHLETE WITH AN INTERSEX CONDITION?

- Many intersex conditions are benign when it comes to an individual's physical characteristics that are relevant to sport.

- Some intersex conditions may cause female athletes to have atypical quantities or responses to testosterone and therefore may generate concern about competitive advantage.
  - For example, an individual with Androgen Insensitivity Syndrome (AIS) has XY chromosomes, and as a result, a body that produces testosterone. But AIS also limits—either completely or partially—the body's ability to respond to testosterone

- Other intersex conditions, however, may result in female-bodied, female-identified individuals that have more usable testosterone in their bodies than women without the condition.

DO INTERSEX ATHLETES HAVE A COMPETITIVE ADVANTAGE WHEN THEY COMPETE IN WOMEN'S SPORTS?

Position: Intersex athletes should have the same access to athletic opportunities as other athletes.

Testing female athletes to make sure their testosterone levels are "average" or "typical" for women, would be problematic and ill-advised:

- singles out testosterone
- requires every athlete to submit to testing as a condition for participation.
- testing only those athletes who "appear" masculine, would have the effect of singling out women who do not conform to stereotypes about femininity
- evokes a double standard, since widely-varying levels of testosterone among male athletes do not raise fairness concerns

SHOULD INTERSEX ATHLETES BE PERMITTED TO COMPETE?

- Lived gender identify is best policy for assignment for men or women's teams.
- physical facilities, including bathrooms, locker rooms, and showers, schools and other sports organizations should support intersex athletes seeking to use whichever facilities they are most comfortable with, and which are most consistent with their gender identity.
- Some intersex athletes may request separate facilities due to privacy concerns. In such cases, the school or sports organization should make every reasonable effort to accommodate such a request.
- If an organization accommodates other athletes' bona fide requests for privacy (such as those due to religious reasons) but does not accommodate a students' request based on an intersex condition, that organization may be liable for discrimination.

IN WHAT WAYS SHOULD INTERSEX ATHLETES BE ACCOMMODATED?

J Am Acad Dermatol. 2005 Aug;53(2):237-41.

Sunscreen use among collegiate athletes.

Hamant ES, Adams BB.

Source

University of Cincinnati College of Medicine, Cincinnati, Ohio 45267-0592, USA.

Abstract

BACKGROUND:

College athletes participating in outdoor sports are a population with extensive exposure to ultraviolet radiation during the peak hours of the day. These relatively young athletes could represent a group with alterable habits.

OBJECTIVE:

We sought to assess the level of sunscreen use in college athletes who are at high risk for ultraviolet radiation exposure.

METHODS:

An anonymous survey was administered to NCAA soccer and cross-country teams at 4 universities. Answers were analyzed for proportions of sunscreen use and associations among various host variables.

RESULTS:

Of the surveyed athletes, 85% reported no sunscreen use in the previous 7 days and only 6% reported sunscreen use at least 3 of the previous 7 days.

LIMITATIONS:

The subject population is limited to one geographic area and includes athletes from only two sports.

CONCLUSIONS:

These results identify a need for improved primary prevention of ultraviolet damage among the more than 250,000 NCAA athletes participating in outdoor sports. A preventive program involving education, provision of readily available sunscreen, and coach participation is needed to target these individuals.

Clin J Sport Med. 2006 May;16(3):253-60.

Sun-protective behavior among high-school and collegiate athletes in Los Angeles, CA.

Cohen PH, Tsai H, Puffer JC.

Source

Rutgers University Sports Medicine, Picataway, NJ 08854, USA. pcohen@aya.yale.edu

Abstract

OBJECTIVES:

To evaluate the use, knowledge, and perceptions of sun-protective behavior (SPB), among young athletes in Southern California.

DESIGN:

Cross-sectional survey, assessing various measures of SPB, sun exposure, attitudes, and knowledge.

SETTING AND PARTICIPANTS:

A total of 1006 students from 2 public high schools and 1 public university in the Los Angeles area volunteered. Athlete volunteers were recruited during sports preparticipation examinations. Nonathlete controls were recruited from physical education classes (high school) and from an introductory lecture class (university).

MAIN OUTCOME MEASURES:

Use of SPB during sports and nonsports activities, sun exposure, sun-protection knowledge, and attitudes. Results from athletes were compared with those from controls at each site.

RESULTS:

Overall rates of SPB were low. During nonsports activities, university athletes had higher rates of sunscreen (37.7% vs. 32.3%, $P = 0.03$) and hat use (34.7% vs. 17%, $P < 0.001$) than did controls. However, university athletes had lower rates of use of long shirts (27.9% vs. 39.8%, $P < 0.001$), long pants (38.8% vs. 63%, $P < 0.002$), and had higher total sun exposure (16.1 vs. 10.1 h/wk, $P < 0.001$). Similar results were seen among the high-school subjects. Despite having good knowledge of the potential dangers of ultraviolet radiation exposure, approximately 1 in 5 subjects overall had experienced a blistering sunburn within the past year.

CONCLUSIONS:

Young athletes in the Los Angeles area reported low rates of SPB, high levels of ultraviolet radiation exposure, and high rates of sunburns, despite knowing the risks of sun exposure. Specific educational and behavioral interventions should be directed toward improving SPB in this high-risk group.

J Am Acad Dermatol. 2012 Feb 2. [Epub ahead of print]

Sunscreen use and availability among female collegiate athletes.

Dubas LE, Adams BB.

Source

Department of Dermatology, University of Cincinnati, Cincinnati, Ohio.

Abstract

BACKGROUND:

Each year more than 250,000 athletes participate on National Collegiate Athletic Association (NCAA) teams. The majority of outdoor NCAA athletes, however, do not use sunscreen on a daily basis. Previous research notes that sunscreen availability represents the main barrier to use among collegiate athletes.

OBJECTIVE:

We sought to explore the effect of sunscreen availability on its application among outdoor collegiate athletes.

METHODS:

Participants (n = 83) on NCAA Division IA female golf teams were randomized to form treatment and control groups. Treatment group players placed study-supplemented sunscreen in their golf bags and locker rooms during the study period. Both groups completed self-administered surveys at the beginning and end of the 4-week investigation.

RESULTS:

The athletes for whom sunscreen was placed in the locker room and bags exhibited a statistically greater use of sunscreen at the end of the study compared with the control group (P = .01). Provision of readily available sunscreen in the locker room resulted in an average of 1.13 more days per week of sunscreen use, after adjustment for baseline use (P = .008). In addition, players with ready access to sunscreen during competitions increased their reapplication, although not significantly, by nearly 20% (P = .10). Sunscreen reapplication during practice did not change for either group.

LIMITATIONS:

Future investigations should target a larger population of both male and female outdoor collegiate athletes.

CONCLUSIONS:

The application of sunscreen increased significantly when athletes had easy access to sunscreen during practice and competition. These findings highlight a major opportunity to improve sun-protective behaviors among this vulnerable population.

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WASHINGTON − Despite high levels of sun exposure, nearly half of collegiate athletes don't use sunscreen, according to a recent survey.

The anonymous survey of 290 athletes from two universities − one on the East Coast and one on the West Coast − found that 96% of respondents believed sunscreen would help protect them from skin cancer. Yet, 43% of the athletes surveyed reported never using sunscreen, 31% reported only using sunscreen 1-3 days per week, 18% reported using sunscreen 4-6 days per week, and 8% reported using sunscreen every day of the week.

On average, the athletes reported spending 4 hours a day outdoors for 10 months a year, said Dr. Ashley Wysong, a resident in the department of dermatology at Stanford (Calif.) University.

With more than 400,000 NCAA collegiate athletes in universities across the nation, "An organized educational campaign by dermatologists, the NCAA, member universities, and individual coaches may be beneficial" in achieving better sun protection among college athletes, said Dr. Wysong, a former six-time NCAA All-American and national champion in middle distance running.

The survey, which included 13 outdoor sports, found that the percentage of athletes who wore sunscreen increased with the frequency of coaches or athletic administrators speaking with them about sun protection.

Survey respondents listed several reasons for not using sunscreen. The most common was forgetting to use it (63%), followed by inconvenience of use (41%), and "I like to be tan" (39%). Other reasons the athletes reported for not using sunscreen included: belief that they don't burn (35%), the greasy feel of sunscreen (34%), and the time it takes for application (22%).

Not surprisingly, sunburns were common among the participants. Nearly 84% reported experiencing at least one sunburn in the past year, with 28% reporting four or more, and 10% reporting a blistering sunburn.

The majority of respondents were Fitzpatrick Skin Type I and II, but all skin types were represented in the study, the authors reported.

A few factors contributed to sunscreen use: living in the West Coast, female gender, number of sunburns in the past year, belief in the risk of skin cancer, knowing someone with skin cancer, and worry about wrinkles.

A study by researchers at the University of Cincinnati reached a similar conclusion, finding "a need for improved primary prevention of ultraviolet damage" among college athletes. The survey found that 85% of athletes at four universities reported no sunscreen use during the prior week, and only 7% reported using sunscreen at least three times during that time period (J. Am. Acad. Dermatol. 2005;53:237-41).

Dr. Wysong said that sun protective behavior extends beyond just the athletes themselves, because "they are role models for younger kids."

To follow-up their study, the authors are planning to hand out educational literature to freshmen athletes and conduct before and after questionnaires on sunscreen use.

Dr. Wysong reported having no conflicts of interest.

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June 2, 2006
For Immediate Release

COLLEGE ATHLETES IN THE DARK ABOUT SUNSCREEN
Study Finds Few Protecting Adequately; Calls for Education Program

DENVER - Skin protection is not a burning issue among college athletes, according to research presented at the 53rd Annual Meeting of the American College of Sports Medicine (ACSM) in Denver. Fewer than ten percent reported using sunscreen regularly defined as at least three of the previous seven days.

Researchers Erica S. Hamant and Brian B. Adams analyzed brief, anonymous questionnaires completed by 186 athletes at four universities in late summer 2005. Sunscreen use was low among both genders at all institutions, regardless of whether respondents played soccer or ran cross-country.

"These data reveal that young athletes are needlessly risking skin damage as they age," said Hamant. "We know that unprotected exposure to the sun can lead to a host of problems, from sunburn and photoaging to skin cancer. Athletes in some sports spend many hours outdoors, and regular use of sunscreen can go a long way in preventing skin damage."

In spite of the risks, Hamant and Adams found that just nine percent of athletes age 20 and older and only two percent of 19-year-olds used sunscreen regularly. The difference is not considered statistically significant. According to the National College Athletic Association, more than 250,000 athletes in 2003 participated in sports involving sun exposure. "This large number of high-risk athletes is also an easily target-able group," said Hamant. "We believe that a preventive program is needed. This would involve educating the athletes, making sunscreen easily accessible in the locker room, and enlisting coaches to encourage sunscreen use among the athletes over whom they have significant influence."

Other data regarding this study was published in the Journal of the American Academy of Dermatology, regarding why respondents didn't use sunscreen daily. Many comments related to availability of sunscreen; others reflected a need for education on the topic, with students reporting they didn't think sunscreen was needed for their skin type.

The stakes are high, says Hamant, but solutions are at hand. Even a brief bout of sunburn can affect the competitiveness of top athletes, and those who accumulate significant sun exposure are more likely to develop skin cancer. Hamant calls for dispensers of sunscreen near the entrance to locker rooms, perhaps funded by manufacturers of sunscreen.

Studies have shown that coaches can play a large role in motivating sunscreen use among athletes. For example, coaches can require sunscreen use and prevent sunburned athletes from competing.

"This study involved a rather small population of athletes," said Hamant. More research is needed. However, it's clear that too many collegiate athletes are risking their health by not using sunscreen. Programs to promote education and availability can have a very real impact."

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NCAA Athletes Not Using Sunscreen

By: NASEEM S. MILLER, Skin & Allergy News Digital Network

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On average, the athletes reported spending 4 hours a day outdoors for 10 months a year, said Dr. Ashley Wysong, a resident in the department of dermatology at Stanford (Calif.) University.

With more than 400,000 NCAA collegiate athletes in universities across the nation, "An organized educational campaign by dermatologists, the NCAA, member universities, and individual coaches may be beneficial" in achieving better sun protection among college athletes, said Dr. Wysong, a former six-time NCAA All-American and national champion in middle distance running.

The survey, which included 13 outdoor sports, found that the percentage of athletes who wore sunscreen increased with the frequency of coaches or athletic administrators speaking with them about sun protection.

Survey respondents listed several reasons for not using sunscreen. The most common was forgetting to use it (63%), followed by inconvenience of use (41%), and "I like to be tan" (39%). Other reasons the athletes reported for not using sunscreen included: belief that they don't burn (35%), the greasy feel of sunscreen (34%), and the time it takes for application (22%).

Not surprisingly, sunburns were common among the participants.

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The majority of respondents were Fitzpatrick Skin Type I and II, but all skin types were represented in the study, the authors reported.

A few factors contributed to sunscreen use: living in the West Coast, female gender, number of sunburns in the past year, belief in the risk of skin cancer, knowing someone with skin cancer, and worry about wrinkles.

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Despite knowing that sunscreen helps protect against skin cancer, 43% of NCAA athletes surveyed said they never use it.

A study by researchers at the University of Cincinnati reached a similar conclusion, finding "a need for improved primary prevention of ultraviolet damage" among college athletes. The survey found that 85% of athletes at four universities reported no sunscreen use during the prior week, and only 7% reported using sunscreen at least three times during that time period (J. Am. Acad. Dermatol. 2005;53:237-41).

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Dr. Wysong reported having no conflicts of interest.

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calendar

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May 25 - 28 Orlando, FL	Florida Society of Dermatology & Dermatologic Surgery (FSDDS): Annual Meeting
Jun 1 - 3 Hilton Head Island, SC	Georgia Society of Dermatologists (GSD): Annual Meeting
Jun 1 - 3 Dana Point, CA	Summit in Aesthetic Medicine 2012
Jun 6 - 10 Verona,	European Academy of Dermatology and Venereology (EADV): Spring Symposium
Jun 7 - 8 New York, NY	New York University (NYU): Advances in Dermatology
Jun 12 - 16 Malmo,	European Society of Contact Dermatitis (ESCD): 11th Congress
Jun 15 Albany, NY	Albany Medical College: Annual Dermatology Teaching Day
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Sun Safety

We all need some sun exposure; it's our primary source of vitamin D, which helps us absorb calcium for stronger, healthier bones. But it doesn't take much time in the sun for most people to get the vitamin D they need, and repeated unprotected exposure to the sun's ultraviolet rays can cause skin damage, eye damage, immune system suppression, and skin cancer. Even people in their twenties can develop skin cancer.

Most kids rack up a lot of their lifetime sun exposure before age 18, so it's important that parents teach their children how to enjoy fun in the sun safely. With the right precautions, you can greatly reduce your child's chance of developing skin cancer.

Facts About Sun Exposure

The sun radiates light to the earth, and part of that light consists of invisible ultraviolet (UV) rays. When these rays reach the skin, they cause tanning, burning, and other skin damage.

Sunlight contains three types of ultraviolet rays: UVA, UVB, and UVC.

1. **UVA** rays cause skin aging and wrinkling and contribute to skin cancer, such as melanoma. Because UVA rays pass effortlessly through the ozone layer (the protective layer of atmosphere, or shield, surrounding the earth), they make up the majority of our sun exposure. Beware of tanning beds because they use UVA rays as well as UVB rays. A UVA tan does **not** help protect the skin from further sun damage; it merely produces color and a false sense of protection from the sun.

2. **UVB** rays are also dangerous, causing sunburns, cataracts (clouding of the eye lens), and effects on the immune system. They also contribute to skin cancer. Melanoma, the most dangerous form of skin cancer, is thought to be associated with severe UVB sunburns that occur before the age of 20. Most UVB rays are absorbed by the ozone layer, but enough of these rays pass through to cause serious damage.

3. **UVC** rays are the most dangerous, but fortunately, these rays are blocked by the ozone layer and don't reach the earth.

What's important is to protect your family from exposure to UVA and UVB, the rays that cause skin damage.

Melanin: The Body's First Line of Defense

UV rays react with a chemical called melanin that's found in skin. Melanin is the first defense against the sun because it absorbs dangerous UV rays before they do serious skin damage. Melanin is found in different concentrations and colors, resulting in different skin colors. The lighter someone's natural skin color, the less melanin it has to absorb UV rays and protect itself. The darker a person's natural skin color, the more melanin it has to protect itself. (But both dark- *and* light-skinned kids need protection from UV rays because *any* tanning or burning causes skin damage.)

Also, anyone with a fair complexion — lighter skin and eye color — is more likely to have freckles because there's less melanin in the skin. Although freckles are harmless, being outside in the sun may help cause them or make them darker.

As the melanin increases in response to sun exposure, the skin tans. But even that "healthy" tan may be a sign of sun damage. The risk of damage increases with the amount and intensity of exposure. Those who are chronically exposed to the sun, such as farmers, boaters, and sunbathers, are at much greater risk. A sunburn develops when the amount of UV exposure is greater than what can be protected against by the skin's melanin.

Unprotected sun exposure is even more dangerous for kids with:

- moles on their skin (or whose parents have a tendency to develop moles)
- very fair skin and hair
- a family history of skin cancer, including melanoma

You should be especially careful about sun protection if your child has one or more of these high-risk characteristics.

Also, not all sunlight is "equal" in UV concentration. The intensity of the sun's rays depends upon the time of year, as well as the altitude and latitude of your location. UV rays are strongest during summer. Remember that the timing of this season varies by location; if you travel to a foreign country during its summer season, you'll need to pack the strongest sun protection you can find.

Extra protection is also required near the equator, where the sun is strongest, and at high altitudes, where the air and cloud cover are thinner, allowing more damaging UV rays to get through the atmosphere. Even during winter months, if your family goes skiing in the mountains, be sure to apply plenty of sunscreen; UV rays reflect off both snow and water, increasing the probability of sunburn.

With the right precautions, kids can safely play in the sun. Here are the most effective strategies:

Avoid the Strongest Rays of the Day

First, seek shade when the sun is at its highest overhead and therefore strongest (usually 10 a.m. until 4 p.m. in the northern hemisphere). If kids must be in the sun between these hours, be sure to apply and

reapply protective sunscreen — even if they're just playing in the backyard. Most sun damage occurs as a result of incidental exposure during day-to-day activities, not at the beach.

Even on cloudy, cool, or overcast days, UV rays travel through the clouds and reflect off sand, water, and even concrete. Clouds and pollution don't filter out UV rays, and they can give a false sense of protection. This "invisible sun" can cause unexpected sunburn and skin damage. Often, kids are unaware that they're developing a sunburn on cooler or windy days because the temperature or breeze keeps skin feeling cool on the surface.

Make sure your kids don't use tanning beds at any time, even to "prepare" for a trip to a warm climate. Both UVA and UVA/UVB tanning beds produce sunburn. And there is an increase in the risk of melanoma in people who have used tanning beds before the age of 35.

Cover Up

One of the best ways to protect your family from the sun is to cover up and shield skin from UV rays. Ensure that clothes will screen out harmful UV rays by placing your hand inside the garments and making sure you can't see it through them.

Because infants have thinner skin and underdeveloped melanin, their skin burns more easily than that of older kids. But sunscreen should **not** be applied to babies under 6 months of age, so they absolutely must be kept out of the sun whenever possible. If your infant must be in the sun, dress him or her in clothing that covers the body, including hats with wide brims to shadow the face. Use an umbrella to create shade.

Even older kids need to escape the sun. For all-day outdoor affairs, bring along a wide umbrella or a pop-up tent to play in. If it's not too hot outside and won't make kids even more uncomfortable, have them wear light long-sleeved shirts and/or long pants. Before heading to the beach or park, call ahead to find out if certain areas offer rentals of umbrellas, tents, and other sun-protective gear.

Use Sunscreen Consistently

Lots of good sunscreens are available for kids, including formulations for sensitive skin, brands with fun scents like watermelon, long-lasting waterproof and sweat-proof versions, and easy-application varieties in spray bottles.

What matters most in a sunscreen is the degree of protection from UV rays it provides. When faced with the overwhelming sea of sunscreen choices at drugstores, concentrate on the SPF (sun protection factor) numbers on the labels.

For kids age 6 months and older, select an SPF of 30 or higher to prevent both sunburn *and* tanning. Choose a sunscreen that states on the label that it protects against both UVA and UVB rays (referred to as "broad-spectrum" sunscreen). In general, sunscreens provide better protections against UVB rays than UVA rays, making signs of skin aging a risk even with consistent use of sunscreen. To avoid possible skin allergy, don't use sunscreens with PABA; if your child has sensitive skin, look for a product with the active ingredient titanium dioxide (a chemical-free block).

To get a tanned appearance, teens might try self-tanning lotions. These offer an alternative to ultraviolet exposure, but only minimal (or no) protection from UV light.

For sunscreen to do its job, it must be applied correctly. Be sure to:

- Apply sunscreen whenever kids will be in the sun.
- Apply sunscreen about 15 to 30 minutes before kids go outside so that a good layer of protection can form. Don't forget about lips, hands, ears, feet, shoulders, and behind the neck. Lift up bathing suit straps and apply sunscreen underneath them (in case the straps shift as a child moves).
- Don't try to stretch out a bottle of sunscreen; apply it generously.
- Reapply sunscreen often, approximately every 2 hours, as recommended by the American Academy of Dermatology. Reapply after a child has been sweating or swimming.
- Apply a waterproof sunscreen if kids will be around water or swimming. Water reflects and intensifies the sun's rays, so kids need protection that lasts. Waterproof sunscreens may last up to 80 minutes in the water, and some are also sweat- and rub-proof. But regardless of the waterproof label, be sure to reapply sunscreen when kids come out of the water.

Keep in mind that every child needs extra sun protection. The American Academy of Dermatology recommends that all kids — regardless of their skin tone — wear sunscreen with an SPF of 30 or higher. Although dark skin has more protective melanin and tans more easily than it burns, remember that tanning is also a sign of sun damage. Dark-skinned kids also can develop painful sunburns.

Use Protective Eyewear for Kids

Sun exposure damages the eyes as well as the skin. Even 1 day in the sun can result in a burned cornea (the outermost, clear membrane layer of the eye). Cumulative exposure can lead to cataracts (clouding of the eye lens, which leads to blurred vision) later in life. The best way to protect eyes is to wear sunglasses.

Not all sunglasses provide the same level of ultraviolet protection; darkened plastic or glass lenses without special UV filters just trick the eyes into a false sense of safety. Purchase sunglasses with labels ensuring that they provide 100% UV protection.

But not all kids enjoy wearing sunglasses, especially the first few times. To encourage them to wear them, let kids select a style they like — many manufacturers make fun, multicolored frames or ones embossed with cartoon characters. And don't forget that kids want to be like grown-ups. If you wear sunglasses regularly, your kids may be willing to follow your example. Providing sunglasses early in childhood will encourage the habit of wearing them in the future.

Double-Check Medications

Some medications increase the skin's sensitivity to UV rays. As a result, even kids with skin that tends not to burn easily can develop a severe sunburn in just minutes when taking certain medications. Fair-skinned kids, of course, are even more vulnerable.

Ask your doctor or pharmacist if any prescription (especially antibiotics and acne medications) and over-the-counter medications your child is taking can increase sun sensitivity. If so, always take extra sun precautions. The best protection is simply covering up or staying indoors; even sunscreen can't always protect skin from sun sensitivity caused by medications.

If Your Child Gets a Sunburn

A sunburn can sneak up on kids, especially after a long day at the beach or park. Often, they seem fine during the day but then gradually develop an "after-burn" later that evening that can be painful and hot and even make them feel sick.

When kids get sunburned, they usually experience pain and a sensation of heat — symptoms that tend to become more severe several hours after sun exposure. Some also develop chills. Because the sun has dried their skin, it can become itchy and tight. Sunburned skin begins to peel about a week after the sunburn. Encourage your child not to scratch or peel off loose skin because skin underneath the sunburn is vulnerable to infection.

If your child does get a sunburn, these tips may help:

- Have your child take a cool (not cold) bath, or gently apply cool, wet compresses to the skin to help alleviate pain and heat.
- To ease discomfort, apply pure aloe vera gel (available in most pharmacies) to any sunburned areas.
- Give your child an anti-inflammatory medication like ibuprofen or use acetaminophen to lessen the pain and itching. (Do **not**, however, give aspirin to children or teens.) Over-the-counter diphenhydramine may also help reduce itching and swelling.
- Apply topical moisturizing cream to rehydrate the skin and treat itching. For the more seriously sunburned areas, apply a thin layer of 1% hydrocortisone cream to help with pain. (Do **not** use petroleum-based products, because they prevent excess heat and sweat from escaping. Also, avoid first-aid products that contain benzocaine, which may cause skin irritation or allergy.)

If the sunburn is severe and blisters develop, call your doctor. Until you can see your doctor, tell your child not to scratch, pop, or squeeze the blisters, which can become easily infected and can result in scarring. Keep your child in the shade until the sunburn is healed. Any additional sun exposure will only increase the severity of the burn and increase pain.

Be Sun Safe Yourself

Don't forget: Be a good role model by consistently wearing sunscreen with SPF 30 or greater, using sunglasses, and limiting your time in the sun. These preventive behaviors not only reduce your risk of sun damage, but teach your kids good sun sense.

Reviewed by: Kate M. Cronan, MD
Date reviewed: August 2010

Note: All information on KidsHealth® is for educational purposes only. For specific medical advice, diagnoses, and treatment, consult your doctor.

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Centers for Disease Control and Prevention
CDC 24/7: Saving Lives. Protecting People.™

Prevention

Protection from ultraviolet (UV) radiation is important all year round, not just during the summer or at the beach. UV rays from the sun can reach you on cloudy and hazy days, as well as bright and sunny days. UV rays also reflect off of surfaces like water, cement, sand, and snow. Indoor tanning (using a tanning bed, booth, or sunlamp to get tan) exposes users to UV radiation.

The hours between 10 a.m. and 4 p.m. daylight savings time (9 a.m. to 3 p.m. standard time) are the most hazardous for UV exposure outdoors in the continental United States. UV rays from sunlight are the greatest during the late spring and early summer in North America.

CDC recommends easy options for protection from UV radiation—

- Seek shade, especially during midday hours.
- Wear clothing to protect exposed skin.
- Wear a hat with a wide brim to shade the face, head, ears, and neck.
- Wear sunglasses that wrap around and block as close to 100% of both UVA and UVB rays as possible.
- Use sunscreen with sun protective factor (SPF) 15 or higher, and both UVA and UVB (/cancer/skin/basic_info/index.htm#uv) protection.
- Avoid indoor tanning.

Shade

You can reduce your risk of skin damage and skin cancer by seeking shade under an umbrella, tree, or other shelter before you need relief from the sun. Your best bet to protect your skin is to use sunscreen or wear protective clothing when you're outside—even when you're in the shade.

Clothing

Loose-fitting long-sleeved shirts and long pants made from tightly woven fabric offer the best protection from the sun's UV rays. A wet T-shirt offers much less UV protection than a dry one. Darker colors may offer more protection than lighter colors.

If wearing this type of clothing isn't practical, at least try to wear a T-shirt or a beach cover-up. Keep in mind that a typical T-shirt has an SPF rating lower than 15, so use other types of protection as well.

Hats

For the most protection, wear a hat with a brim all the way around that shades your face, ears, and the back of your neck. A tightly woven fabric, such as canvas, works best to protect your skin from UV rays. Avoid straw hats with holes that let sunlight through. A darker hat may offer more UV protection.

If you wear a baseball cap, you should also protect your ears and the back of your neck by wearing clothing that covers those areas, using sunscreen with at least SPF 15, or by staying in the shade.

Sunglasses

Sunglasses protect your eyes from UV rays and reduce the risk of cataracts. They also protect the tender skin around your eyes from sun exposure.

Sunglasses that block both UVA and UVB rays offer the best protection. Most sunglasses sold in the United States, regardless of cost, meet this standard. Wrap-around sunglasses work best because they block UV rays from sneaking in from the side.

Sunscreen

The sun's UV rays can damage your skin in as little as 15 minutes. Put on sunscreen before you go outside, even on slightly cloudy or cool days. Don't forget to put a thick layer on all parts of exposed skin. Get help for hard-to-reach places like your back.

The United States Food and Drug Administration has announced significant changes to sunscreen product labels (http://www.fda.gov/Drugs/ResourcesForYou/Consumers/BuyingUsingMedicineSafely/UnderstandingO-the-CounterMedicines/ucm239463.htm) (http://www.cdc.gov/Other/disclaimer.html) that will help consumers decide how to buy and use sunscreen, and allow them to protect themselves and their families from sun-induced damage more effectively.

How sunscreen works. Most sun protection products work by absorbing, reflecting, or scattering sunlight. They contain chemicals that interact with the skin to protect it from UV rays. All products do not have the same ingredients; if your skin reacts badly to one product, try another one or call a doctor.

SPF. Sunscreens are assigned a sun protection factor (SPF) number that rates their effectiveness in blocking UV rays. Higher numbers indicate more protection. You should use a sunscreen with at least SPF 15.

Reapplication. Sunscreen wears off. Put it on again if you stay out in the sun for more than two hours, and after you swim or do things that make you sweat.

Expiration date. Check the sunscreen's expiration date. Sunscreen without an expiration date has a shelf life of no more than three years, but its shelf life is shorter if it has been exposed to high temperatures.

Cosmetics. Some make-up and lip balms contain some of the same chemicals used in sunscreens. If they do not have at least SPF 15, don't use them by themselves.

Avoid Indoor Tanning

Using a tanning bed, booth, or sunlamp to get tan is called "indoor tanning (indoor_tanning.htm)." Indoor tanning has been linked with skin cancers including melanoma (the deadliest type of skin cancer), squamous cell carcinoma, and cancers of the eye (ocular melanoma).

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Centers for Disease Control and Prevention 1600 Clifton Rd. Atlanta, GA 30333, USA
800-CDC-INFO (800-232-4636) TTY: (888) 232-6348 - cdcinfo@cdc.gov

New Chapter

Guideline XX. Sun Protection. June 2012.

Exposure to sunlight or ultraviolet (UV) light has a profound effect on the skin causing skin damage, premature skin aging , eye damage, immune system suppression and skin cancer. Repeated unprotected exposure to the sun's UVA and UVB rays cause sun burns, can lead to cataracts (clouding of the eye lens), photoaging, skin wrinkling and can contribute to skin cancer.

Skin cancer is the most common type of cancer and is thought to account for half of all cancers. About 3.5 million cases of non-melanoma skin cancer (basal cell or squamous cell cancers, the two most common types of skin cancer) are diagnosed each year. Melanoma, the third most common type of skin cancer accounts for less than five percent of skin cancer cases but causes a majority of skin cancer deaths.

Risk Factors

Skin cancer is largely preventable by limiting exposure to the primary source of ultraviolent (UV) radiation, sunlight. Reducing exposure to the sun's UV rays can decrease the risk of sun burn, skin cancer and photoaging. Persons with high levels of exposure to UV radiation are at an increased risk for all three major forms of skin cancer. The US Environmental Protection Agency (EPA) estimates that the sun causes 90 percent of non-melanoma skin cancers. Approximately 65 percent to 90 percent of melanomas can be attributed to exposure to UV radiation, and because a substantial percentage of lifetime sun exposure occurs before age twenty, UV light exposure during childhood and adolescence plays an important role in the development of skin cancer. Other risk factors include lighter natural skin color and skin that burns, freckles, reddens easily, or becomes painful in the sun; appearance of moles (particularly an increased number of moles or an atypical mole or changing mole); family history of skin cancer, increasing age, and use of artificial UV radiation (e.g. tanning beds). Environmental factors that increase the amount of UV radiation exposure are also of importance to note including, latitude (closer distance to the equator), higher altitude, light cloud coverage (UV rays can penetrate clouds, windshields, windows and light clothing) and the presence of materials that reflect the sun (e.g. snow, pavement, water and sand).

Text Box / Table: Risk Factors

Primary Risk Factors
Exposure to UV radiation is the most important risk factor for developing skin cancer. Other risk factors include:

- Those who have had at least one severe, blistering sunburn.
- A lighter natural skin color and skin that burns, freckles, reddens easily, or becomes painful in the sun.
- Appearance of moles (particularly, an increased number of moles or an atypical or changing mole).
- Total amount of sun exposure over a lifetime.
- Use of artificial UV radiation (e.g. tanning beds).
- Family history of skin cancer and increasing age.

Environmental Risk Factors
The hours between 10 a.m. and 4 p.m. daylight savings time (9 a.m. to 3 p.m. standard time) are the most hazardous for UV exposure outdoors. Environmental factors that increase the amount of UV radiation exposure include:

- Latitude (closer distance to the equator).
- Higher altitude.
- Light cloud coverage (UV rays can get through clouds, windshields, windows, and light clothing).
- The presence of materials that reflect the sun (e.g. snow, pavement, water and sand).

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Prevention

Unprotected skin can be damaged by the sun's UV rays in as little as fifteen minutes. Reducing exposure to the sun's UV rays can decrease the risk of skin damage and developing skin cancer. Wearing broad spectrum (UVA and UVB) sunscreens and/or clothing to protect as much skin as possible when exposed to the sun is a key component of a comprehensive skin protection program. Sunscreens help to prevent UV radiation from reaching the skin; however, no sunscreen provides complete and total protection. Avoiding the sun during the midday hours provides additional defense against skin damage. However, if the sun cannot be avoided, implementation of alternative sun safety measures (e.g. seeking shade; and wearing a hat, protective clothing, sunglasses or sunscreen) is paramount. With the right precautions student-athletes and athletics staff can participate safely in outdoor athletics activities. Primary protective measures that help reduce the risk for skin cancer:

- Avoiding the sun between 10 a.m. and 4 p.m.
- Wearing sun-protective clothing when exposed to the sun.
- Using a broad spectrum sunscreen with a sun-protection factor (SPF) greater than 30.
- Avoiding tanning beds and other artificial sources of UV light.

Research suggests a need for improved primary prevention of UV damage among NCAA student-athletes. According to research conducted by the University of Cincinnati College of Medicine, fewer than ten percent of collegiate student-athletes reported using sunscreen regularly defined as at least three of the previous seven days Fifty-three percent of US adults were "very likely" to protect themselves from the sun by practicing at least one. A study conducted by Stanford University Department of Dermatology found via an anonymous survey of 290 student-athletes from two NCAA universities – one on the East Coast and one on the West Coast – that 96 percent of respondents believed sunscreen would help protect them from skin cancer. Yet, forty three percent of the student-athletes surveyed reported never using sunscreen, thirty one percent reported only using sunscreen one to three days per week, eighteen percent reported using sunscreen four to six days per week, and eight percent reported using sunscreen every day of the week.

More than 250,000 NCAA student-athletes participate in outdoor sports. Sun protection measures should not reduce student-athlete participation. Institutions may find it difficult to avoid scheduling activities around the midday hours or when UV radiation is most damaging. Institutions should focus their efforts on promoting other safety measures (e.g. seeking shade; and wearing a hat, protective clothing, and sunscreen) which can be implemented without compromising athletics participation while gradually making feasible scheduling changes.

Research suggests improved availability of sunscreen increases usage among collegiate athletes. A study conducted at the University of Cincinnati of collegiate women's golfers reported the application of sunscreen increased significantly when athletes had easy access to sunscreen during practice and competition; highlighting an opportunity to improve sun-protective behaviors. The Stanford University survey, which included thirteen NCAA outdoor sports, found that the percentage of athletes who wore

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sunscreen increased significantly with the frequency of coaches or athletic administrators speaking with them about sun protection.

Text Box / Table:

Personal Sun Safety Recommendations

- Liberally and evenly apply a golf ball sized amount (palm full) of a broad spectrum sunscreen evenly over all exposed areas. For those with thin or thinning hair, apply sunscreen to the scalp as well.
- Apply a broad spectrum sunscreen with a SPF greater than 30 before going outdoors and reapply often.
- Always wear protective clothing and sunscreen before going outside:
    - Long sleeve shirts or sun sleeves.
    - A wide-brimmed hat to shade the face, head, ears, and neck (at least a 2- to 3-inch brim all around is ideal).
    - Sunglasses that wrap around and block as close to 100 percent of both UVA and UVB rays as possible.
- Reapply sunscreen every two hours and after swimming, perspiring, and toweling off.
- Do not use tanning beds or other artificial sources of UV light.
- Maintain proper hydration.

Recommendations for Athletics Healthcare Providers and Athletics Administrators

- Serve as a sun safety role model.
- Promote a culture of sun safety and awareness.
- Educate student-athletes and athletics staff about UV protection and skin cancer prevention.
- Use the PPE as an opportunity to educate student-athletes and parents about skin damage and skin cancer prevention.
- Assess athletics staff and each team's sun exposure patterns and reinforce sun safe behaviors.
- Provide sun safety resources to student-athletes, coaches, athletics staff, administrators and institutional staff.
- Advocate for sun safety policies and practices with appropriate athletics and institutional administrators.

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- Improve access to sunscreen for student-athlete use at every outdoor practice and competition.
- Encourage sunscreen and protective clothing (e.g. long sleeve shirts, hats and sunglasses) as indispensable during outdoor practice as sports equipment.
- Consider access to ultraviolet protection factor (UPF) clothing for outdoor practice and competition uniforms.
- Stay in the shade whenever possible and assist athletics staff in choosing locations with shade for outdoor activities. Seek alternative methods of shade such as tents, umbrellas and shade from buildings.
- Make sun safety behaviors routine so that wearing protective gear and taking time out to reapply sunscreen become as much a part of athletics practices and competitions as water breaks.

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SPORTS SUN SAFETY

GENERAL PREVENTION TIPS

Exposure to sunlight for student-athletes and athletics staff members is a common part of athletics activities and daily life. Athletics ~~activities~~ often occur during the midday hours ~~or~~ when ultraviolet (UV) radiation is most damaging. ~~Institutions should focus their efforts on promoting sun safety measures that can be implemented without compromising athletics participation and consider feasible scheduling changes when available.~~ When in the sun, seek shade, cover up, get a hat, wear sunglasses, and use sunscreen (CDC, 2012).

RISK FACTORS

Exposure to UV radiation is the most important risk factor for developing skin cancer. Other risk factors include:

- Those who have had at least one severe, blistering sunburn.
- A lighter natural skin color and skin that burns, freckles, reddens easily, or becomes painful in the sun.
- Appearance of moles (particularly, an increased number of ~~atypical moles or a changing mole~~).
- Total amount of sun exposure during a lifetime.
- Use of artificial UV radiation (tanning beds).
- Family history ~~and age~~.

ENVIRONMENTAL RISK FACTORS

The hours between 10 a.m. and 4 p.m. daylight saving time (9 a.m. to 3 p.m. standard time) are the most hazardous for UV exposure outdoors. Environmental factors that increase the amount of UV radiation exposure include:

- Latitude (distance to the equator).
- Higher altitude.
- Light cloud coverage (UV rays can get through clouds, windshields, windows and light clothing).
- The presence of materials that reflect the sun (e.g., snow, pavement, water and sand).

PERSONAL SUN SAFETY TIPS

- Apply a broad spectrum sunscreen with an SPF greater than ~~15~~ before going outdoors and reapply often.
- ~~Liberally and evenly apply broad spectrum (UVA and UVB) sunscreens over all exposed areas.~~
- Generously apply a golf ball-sized amount (palm full) of sunscreen to cover the entire body (including neck, ears and lips).
- For those with thin or thinning hair, also apply sunscreen to the scalp.
- Reapply sunscreen every two hours and after swimming, perspiring, and toweling off.
- Stay in the shade whenever possible and choose locations with shade for outdoor activities.
- Always wear protective clothing when outside.
- Wear a wide-brimmed hat to shade the face, head, ears, and neck (at least a 2- to 3-inch brim all around is ideal).
- Wear sunglasses that wrap around and block as close to 100 percent of both UVA and UVB rays as possible.
- Do not use tanning beds or other artificial sources of UV light.
- Maintain proper hydration.

SKIN CANCER FACTS

- If you see a spot, check the spot.
- Early detection is crucial; perform self-exams and visit the dermatologist regularly.
- All skin cancers – basal, squamous cell and melanoma – are caused by exposure to ultraviolet (UV) radiation.
- Skin cancer is the most common cancer with more than 3.5 million cases diagnosed in the United States each year.
- Skin cancer is largely preventable by limiting exposure to sunlight, the primary source of UV radiation.
- Unprotected skin can be damaged by the sun's UV rays in as little as 15 minutes.
- About 90 percent of non-melanoma skin cancers ~~are associated with exposure to UV radiation.~~
- ~~About 65 percent of melanoma cases can be attributed to UV radiation from the sun.~~
- Melanoma accounts for less than five percent of skin cancer cases, but causes a majority of skin cancer deaths.
- Sunscreens help to prevent UV radiation from reaching the skin. However, no sunscreen provides complete and total protection.

ADDITIONAL TIPS FOR COACHES AND ATHLETICS HEALTHCARE PROVIDERS:

- Serve as a sun safety role model.
- Improve access to sunscreen for student-athlete use at every outdoor practice and competition.
- Encourage sunscreen and protective clothing as indispensable during outdoor practice as sports equipment.
- Consider access to UPF protective clothing for practice and competition uniforms in outdoor sport athletes.
- Make sun safety behaviors routine, so that wearing protective clothing and taking time out to reapply sunscreen become as much a part of athletics practices and competitions as water breaks.

LOGOS TK FROM CLIENT

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2011 NCAA ATHLETICS HEALTHCARE SURVEY: FINDINGS

I. Introduction.

The purpose of this survey was to help the NCAA to better understand the state of athletics healthcare access within each NCAA division. The results of the study will help the NCAA make appropriate policy decisions in each division and identify best practices that institutions can use to support the health and safety principle of the NCAA.

Athletic trainers and other key athletics administrators at each active and provisional NCAA member institution were solicited via email to participate in the web-based survey. The identity of each respondent and institution has been kept confidential and the findings are being reported in aggregate form.

II. Sample.

Table 1: Overall responses.

	n
Division I	302
Division II	184
Division III	305
Overall	**791**

Table 2: Institutions responding.

	n	**%**
Division I (335)	209	62.4
Division II (302)	139	46.0
Division III (447)	235	52.6
Overall (1,084)	**583**	**53.8**

Table 3: Institutions with multiple responses.

	n	**%**
Division I (209)	68	32.5
Division II (139)	28	20.1
Division III (235)	52	22.1
Overall (583)	**148**	**25.4**

Responses were provided by personnel at more than half of NCAA member schools. Overall, multiple responses were received from one-quarter of responding schools. Findings in this report are based on the overall sample which does include multiple responses from one-quarter of the responding schools.

Athletic trainers accounted for 92 percent of responses. Responses from athletics administrators accounted for the other eight percent.

III. Emergency action plans.

Does your institution have on file an emergency action plan for each athletics venue?

Table 4: Emergency action plans on file for each athletics venue.

	%
Division I	98.7
Division II	93.5
Division III	95.7
Overall	**96.3**

Do you require all athletics healthcare providers and coaches review and practice their respective action plan at least annually?

Table 5: Emergency action plans reviewed and practiced at least annually.

	%
Division I	70.4
Division II	56.0
Division III	66.8
Overall	**65.7**

While nearly all respondents indicated emergency action plans on file for each athletics venue on campus, approximately one-third reported the plans were reviewed and practiced on an annual basis.

IV. Conference requirements.

Does your athletics conference require visiting teams to travel with a certified athletic trainer to away competitions?

Table 5: Conference requirement for visiting teams to travel with a certified athletic trainer to away competitions.

	%
Division I	12.5
Division II	1.6
Division III	1.7
Overall	**5.7**

While 12.5 percent of Division I respondents indicated there was a requirement for visiting teams to travel with a certified athletic trainer to away competitions, fewer than two percent of Division II and III respondents reported such a requirement.

V. Certifications and training.

Does the athletic department require current first aid certification, CPR certification and AED training to any of the following coaches?

Table 6: Coaches required to have current first aid certification, CPR certification and AED training (percent).

	DI	**DII**	**DIII**
All head coaches.	46.7	77.2	92.8
All employed full-time coaches.	60.3	84.8	58.0
Strength and conditioning coaches.	85.4	40.8	36.1
Wrestling coaches.	16.2	14.7	16.4
Volunteer coaches.	34.4	28.8	19.7
Managers.	5.3	1.1	2.0
Cheer/spirit advisors.	32.1	25.5	12.5

93 percent of respondents from Division III schools indicated all head coaches were required to have current first aid certification, CPR certification and AED training. 77 percent of Division II respondents and 47 percent of Division I respondents reported this about their head coaches.

85 percent of Division II respondents indicated all their employed full-time coaches had these requirements. This rate was 60 percent at Division I and 58 percent at Division III.

While 85 percent of Division I respondents indicated their strength and conditioning coaches maintained these requirements, 41 percent of Division II respondents and 36 percent of Division III respondents indicated this about their strength and conditioning coaches.

VI. Team physicians.

Do you have a designated team physician/medical director authorized to oversee the medical services for injuries and illnesses incidental to a student-athlete's participation in intercollegiate athletics?

Table 7: Designated team physician/medical director authorized to oversee medical services for student-athletes.

	%
Division I	98.7
Division II	96.2
Division III	94.1
Overall	**96.3**

Does the athletic department employ the team physician?

Table 8: Athletic department employs team physician.

	%
Division I	32.2
Division II	16.8
Division III	28.8
Overall	**27.3**

How many physicians do you regularly use for providing services in intercollegiate athletics?

Table 9: Average number of physicians regularly used for providing services.

	DI	**DII**	**DIII**
Medical	2.8	2.0	1.6
Orthopedic	2.9	1.9	1.9

While nearly all respondents indicated they have a designated team physician/medical director authorized to oversee medical services for student-athletes, less than one-third employ a team physician. Division I respondents reported the highest average number of physicians regularly used – 2.8 medical and 2.9 orthopedic.

VII. Certified Athletic Trainers.

Does the athletic department employ certified athletic trainers?

Table 10: Athletic department employs certified athletic trainers.

	%
Division I	97.0
Division II	93.4
Division III	96.3
Overall	**95.9**

Do you identify a physician to supervise the practice of employed certified athletic trainers?

Table 11: A physician identified to supervise certified athletic trainers.

	%
Division I	87.7
Division II	82.3
Division III	78.7
Overall	**83.0**

Enter total full time equivalencies for athletics trainers (GA's count as ½ FTE for 20 worked hours. Students in athletic training education for not count).

Table 12: Average total full time equivalencies.

DI	DII	DIII
8.1	3.4	2.8

Nearly all respondents indicated their athletic department employs certified athletic trainers. 83 percent reported a physician was identified to supervise them. Division I respondents indicated the highest average full time equivalencies for athletic trainers at 8.1 while Division II and III respondents reported levels significantly lower at 3.4 and 2.8 respectively.

Have any certified athletic trainers been required to take a furlough in the last 12 months?

Table 13: Certified athletic trainers required to take a furlough in the last 12 months.

	%
Division I	11.6
Division II	6.1
Division III	6.7
Overall	**8.4**

Has the institution/athletics department eliminated any full time certified athletic trainer positions in the last 12 months?

Table 14: Certified athletic trainers eliminated in the last 12 months.

	%
Division I	4.0
Division II	3.7
Division III	1.4
Overall	**2.9**

Has the institution/athletics department decreased the months of certified athletic trainers?

Table 15: Certified athletic trainers decreased.

	%
Division I	0.4
Division II	4.8
Division III	5.3
Overall	**3.3**

Overall, less than ten percent of respondents indicated certified athletic trainers were required to take a furlough in the last 12 months. Additionally, less than five percent of respondents overall reported the elimination or decrease in certified athletic trainers. There are some small differences by division.

Has the institution/athletics department added certified athletic trainer positions in the last 12 months?

Table 16: Certified athletic trainers added in the last 12 months.

	%
Division I	40.8
Division II	24.1
Division III	18.1
Overall	**28.1**

Has the institution/athletics department increased 10 month contract certified athletic trainers to 12 months due to NCAA legislation on summer workouts?

Table 17: Certified athletic trainers' contract increased from 10 to 12 months.

	%
Division I	19.6
Division II	5.2
Division III	0.4
Overall	**7.4**

More than a quarter of respondents overall indicated certified athletic trainer positions had been added in the last 12 months. There were differences by division where 41 percent of Division I respondents reported the addition of positions while 24 percent of Division II and 18 percent of Division III reported additions. At the same time, 20 percent of Division I respondents indicated contracts being increased from ten to 12 months where five percent of Division II and less than one percent of Division III respondents reported contract increases.

VIII. Campus Student Health Center.

Does your institution have a campus student health center?

Table 18: Institution has a campus student health center.

	%
Division I	98.6
Division II	93.3
Division III	90.8
Overall	**94.4**

For those that do not have a campus health center, is your institution affiliated with a medical service which is considered the "campus student health center" but is located off-campus?

Table 19: Student health center located off-campus (institutions that do not have campus health centers).

	%
Division I	75.0
Division II	41.7
Division III	50.0
Overall	**50.0**

Nearly all respondents reported there institution has a campus health center. Of the few who do not have a campus health center, half reported their institution was affiliated with a medical service off-campus.

IX. Roles and Responsibilities.

Indicate under whose employment and supervision the following institutional staff utilized for athletics healthcare deliver fall.

Table 20: Employment and supervision for institutional staff (percent, Division I).

	Athletics Department	**Campus Health Center**	**Non Institutional Entity**	**Not Applicable**
Team Physician	21.8	20.4	52.6	5.2
Physician Assistant	4.7	12.3	22.5	60.5
Certified Athletic Trainers	93.8	1.7	3.1	1.4
Physical Therapist	15.7	5.7	32.0	46.6
Nurse Practitioner	3.3	43.6	8.8	44.3
Licensed Psychologist	14.1	43.8	17.8	24.3
Mental Health Counselor	7.9	57.9	9.7	24.5
Registered Dietician	23.4	27.7	20.9	28.1
First Responders	28.5	4.7	16.1	50.7

Table 21: Employment and supervision for institutional staff (percent, Division II).

	Athletics Department	**Campus Health Center**	**Non Institutional Entity**	**Not Applicable**
Team Physician	19.4	12.0	64.0	4.6
Physician Assistant	4.7	10.5	30.2	54.7
Certified Athletic Trainers	90.9	2.3	4.0	2.9
Physical Therapist	3.5	1.2	34.1	61.2
Nurse Practitioner	0.6	63.6	9.2	26.6
Licensed Psychologist	2.3	38.6	13.5	45.6
Mental Health Counselor	2.3	60.5	8.7	28.5
Registered Dietician	1.2	12.9	18.7	67.3
First Responders	22.8	2.9	28.1	46.2

Table 22: Employment and supervision for institutional staff (percent, Division III).

	Athletics Department	Campus Health Center	Non-Institutional Entity	Not Applicable
Team Physician	24.8	13.8	53.4	7.9
Physician Assistant	2.5	15.0	25.0	57.5
Certified Athletic Trainers	91.6	3.1	3.8	1.4
Physical Therapist	3.5	2.8	29.1	64.5
Nurse Practitioner	1.4	67.4	6.3	24.9
Licensed Psychologist	1.1	45.4	12.3	41.2
Mental Health Counselor	2.8	61.6	7.7	27.8
Registered Dietician	1.1	19.2	15.7	64.1
First Responders	31.8	8.1	14.8	45.2

Employment and supervisory structures for team physicians, physician assistants, nurse practitioners, first responders, mental health counselors and certified athletic trainers are similar across divisional lines. Where Division I responses diverged from Division II and III are in physical therapists, licensed psychologists, and registered dieticians where they report greater levels of alignment with the athletics department. Division I respondents did report these three staff positions were aligned with campus health centers and non-institutional entities as well. These particular services appear to be a more central feature of athletics at Division I than at Division II and III.

Does the head athletic trainer/director of training services serve on the Director of Athletics' management team and attend administrative meetings?

Table 23: Head athletic trainer/director of training services serves on Director of Athletics management team.

	%
Division I	63.4
Division II	66.9
Division III	64.8
Overall	**64.8**

Approximately two-thirds of respondents reported that the head athletic trainer or director of training services serves on the Director of Athletics management team. These rates are similar across divisions.

In your athletics department, do you track…?

Table 24: Athletics department tracking.

	DI	**DII**	**DIII**
Number student-athlete visits in the athletics healthcare facility each year.	70.8	72.7	74.7
Number injury evaluations performed each year.	70.7	70.9	76.5
Number treatments performed each year.	75.9	73.8	73.3
Number surgeries occur each year.	88.6	79.1	78.9
Number hardship waivers processed each year.	88.5	76.7	81.9
Number insurance claims processed each year.	84.9	76.2	69.3
Number certified athletic trainer staff hours worked each year.	29.3	33.9	42.3

There is a similar pattern of athletics department tracking across divisions in the areas of student-athlete visits to the athletics healthcare facility, injury evaluations performed, treatments performed, surgeries and hardship waivers processed. The pattern diverges in the areas of insurance claims and certified athletic trainer staff hours worked. For insurance claims, 85 percent of Division I respondents reported their athletic department tracks this as opposed to 76 percent of Division II respondents and 69 percent of Division III respondents. 42 percent of Division III respondents indicated the athletics department tracks certified athletic trainers staff hours worked as opposed to 34 percent of Division II respondents and 29 percent of Division I.

Health and Safety

A Discussion Regarding Division II Student-Athlete Access to Health and Well-Being Support Services.

David Klossner
Mary Wilfert

NCAA

NCAA Membership Principles

Principle of Institutional Control and Responsibility

- It is the responsibility of each member institution to control its intercollegiate athletic program in compliance with the rules and regulations of the Association.

Principle of Student-Athlete Well-Being

- Health and Safety: It is the responsibility of each member institution to protect the health of and provide a safe environment for each of its participating student-athletes.

Impacts of Staff Size

- Response to NCAA Requirements and Best Practices - Drug testing short notice, concussions, ADHD medication documentation, tryout medical exams, sickle cell trait results.
- Varying levels of Care and Coverage.
- Staff Life/Work Balance – Retention – Continuum of Care

NCAA Health and Safety Actions

1986 – NCAA Drug Testing and Drug Education Program

1998 – Wrestling Weight Management

2003 – Football Preseason Practice

2007 – Mandated Medical Examinations

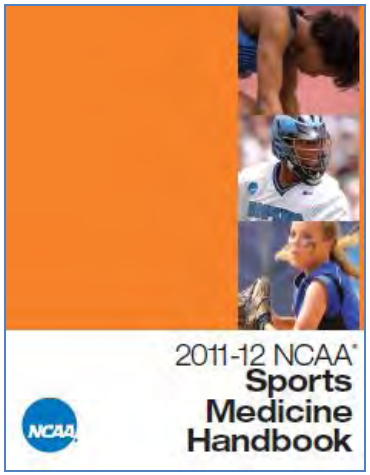
2009 – Mandatory Coaches First Aid, CPR certification and AED training

2010 – Concussion Management Plans

2010 – Sickle Cell Solubility Test

NCAA Policy Impact on Workload

- Sports Medicine Handbook
- Drug Testing Handbook
- Student-Athlete Insurance
- Sport Rules Books (18)
- NCAA Bylaws
- Championships Handbooks



What does the provision of healthcare services include at NCAA institutions.

- Medical Examinations
- Prevention care
- Event Coverage
- Medical Services
- Playing Rules
- Drug Testing
- Drug Education
- Medical Exception paperwork - ADHD
- Nutritional Supplements
- Tobacco use
- Coach Training and Education
- Championships Hosting
- Recruiting
- Clinical Teaching
- Research
- Injury Surveillance Reporting
- Counseling
- Meals/hydration
- Rehabilitation
- Concussions
- Strength and Conditioning
- Protective Equipment
- Insurance
- Surgery
- Hardship Waivers
- Consent Forms
- Costs/vendors
- Parents
- Travel
- Medical Records

Division II Injury Rates

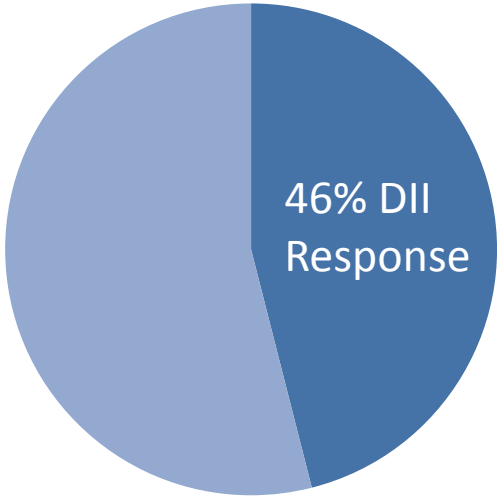
	DII Indicators of Injury Incidence Report								
	Total for Eight Sports (Men's Football, Men's Soccer, Women's Soccer, Women's Volleyball, Men's Basketball, Women's Basketball, Men's Baseball, & Women's Softball)								
	Competition			Practice			Overall (Competition & Practice)		
	Injuries	Exposures	Injury Rate	Injuries	Exposures	Injury Rate	Injuries	Exposures	Injury Rate
2004-2005	10,146	842,708	12.0	12,002	2,681,685	4.5	22,147	3,524,393	6.3
2005-2006	9,636	850,671	11.3	13,682	3,033,392	4.5	23,318	3,884,064	6.0
2006-2007	11,819	929,776	12.7	16,028	3,051,410	5.3	27,848	3,981,188	7.0
2007-2008	7,732	948,099	8.2	9,761	3,267,250	3.0	17,493	4,215,349	4.1
2008-2009	7,651	940,816	8.1	8,940	3,182,734	2.8	16,591	4,123,550	4.0

DATALYSCENTER
Sports Injury Research and Prevention

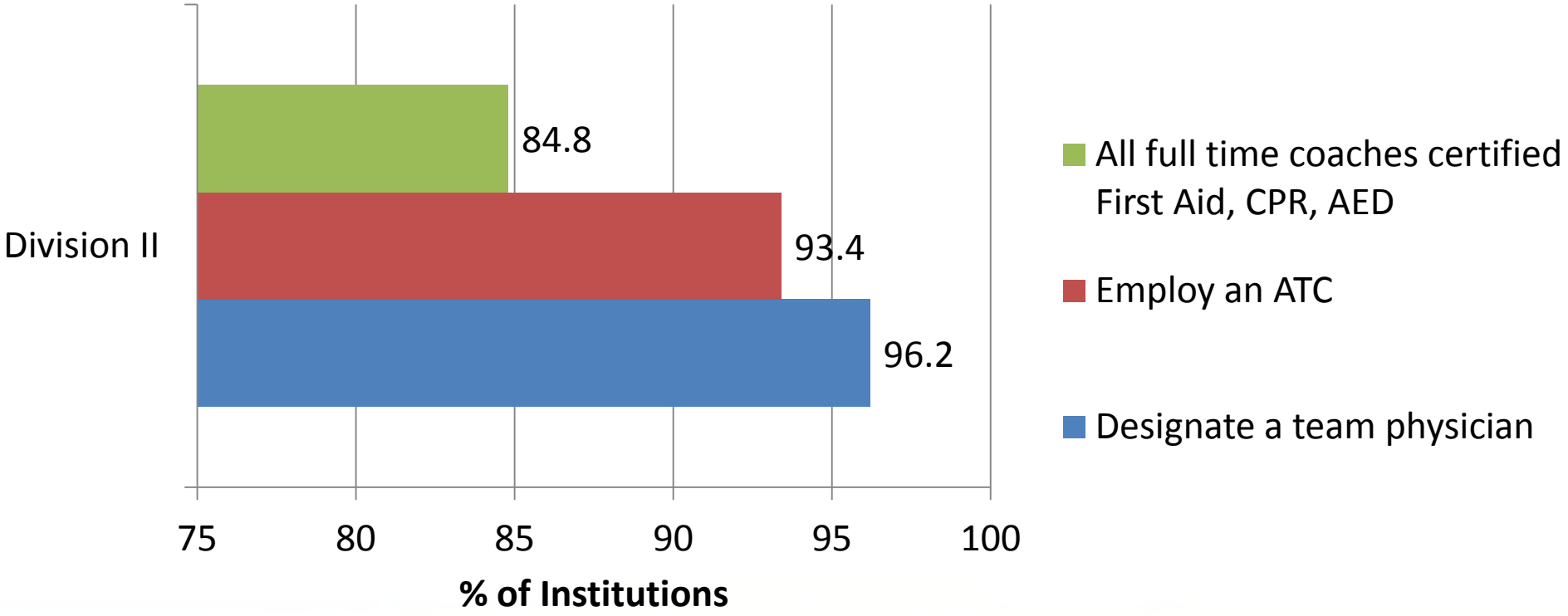
NCAA

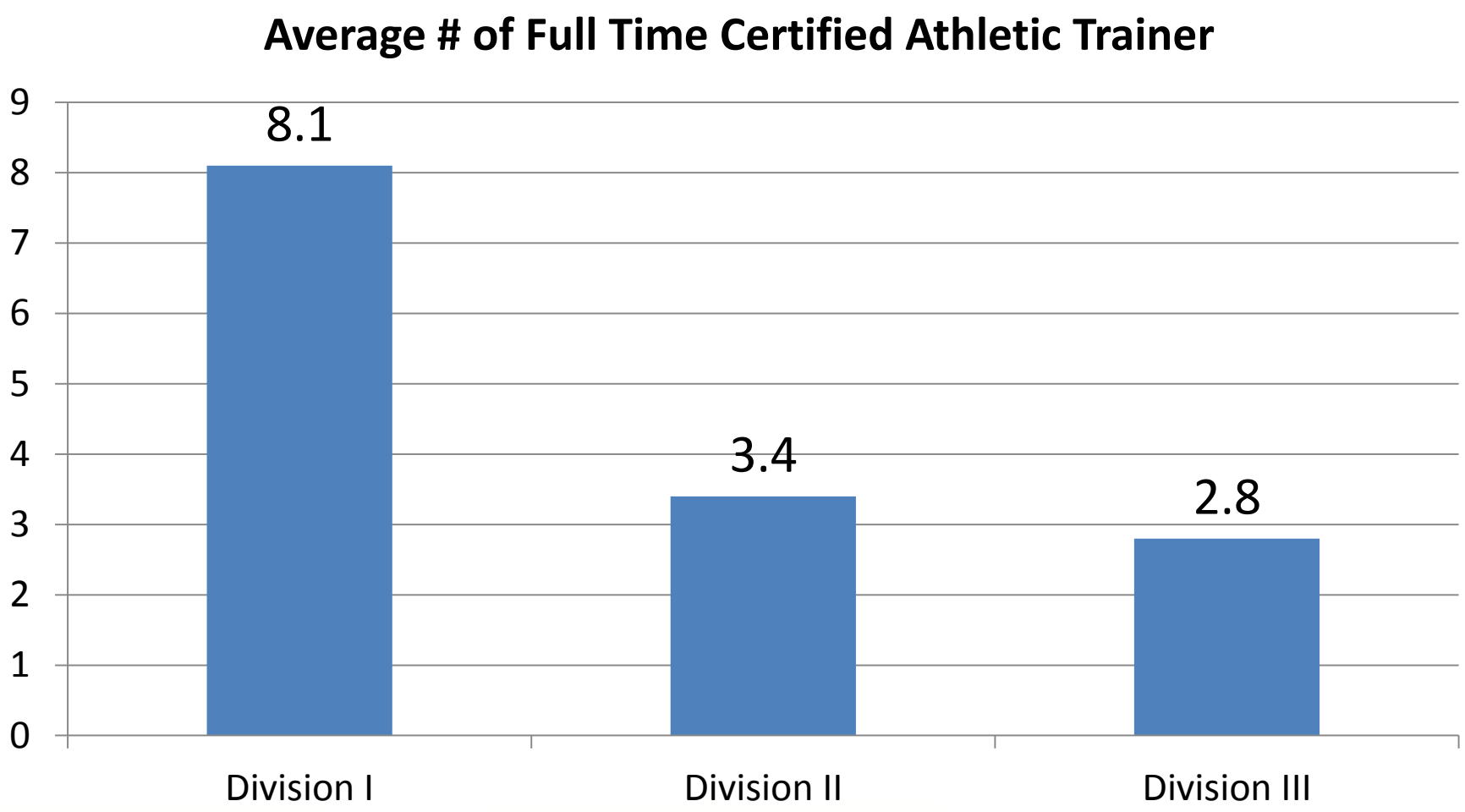
2011 Athletics Healthcare Study

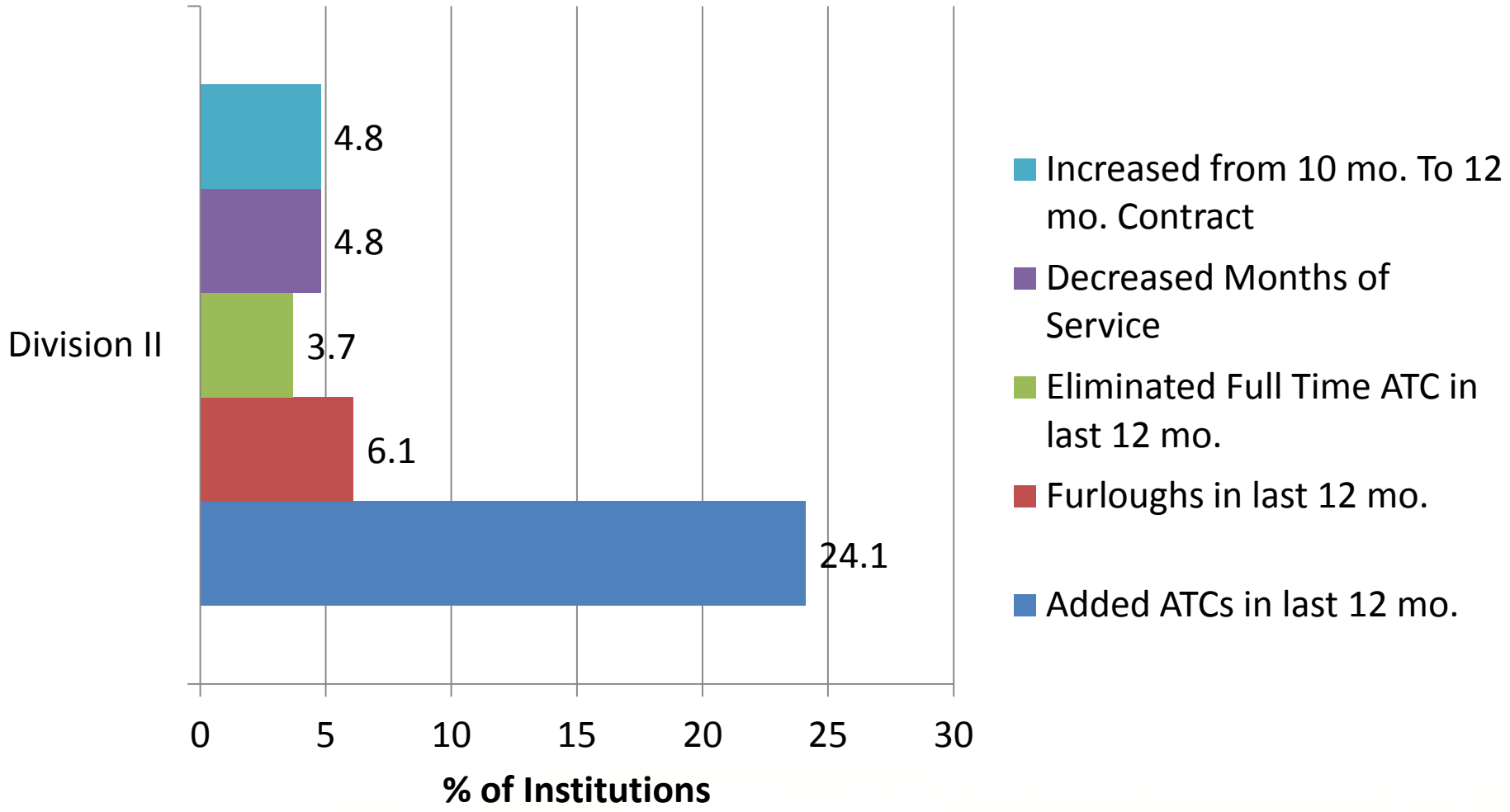
- To help the NCAA better understand the state of athletics healthcare access within each NCAA division.
- The results of the study are assisting the NCAA to make appropriate policy decisions in each division and to identify best practices that institutions can use to support the health and safety principle of the NCAA.



Medical services for injury and illnesses incidental to a student-athlete's participation in intercollegiate athletics.



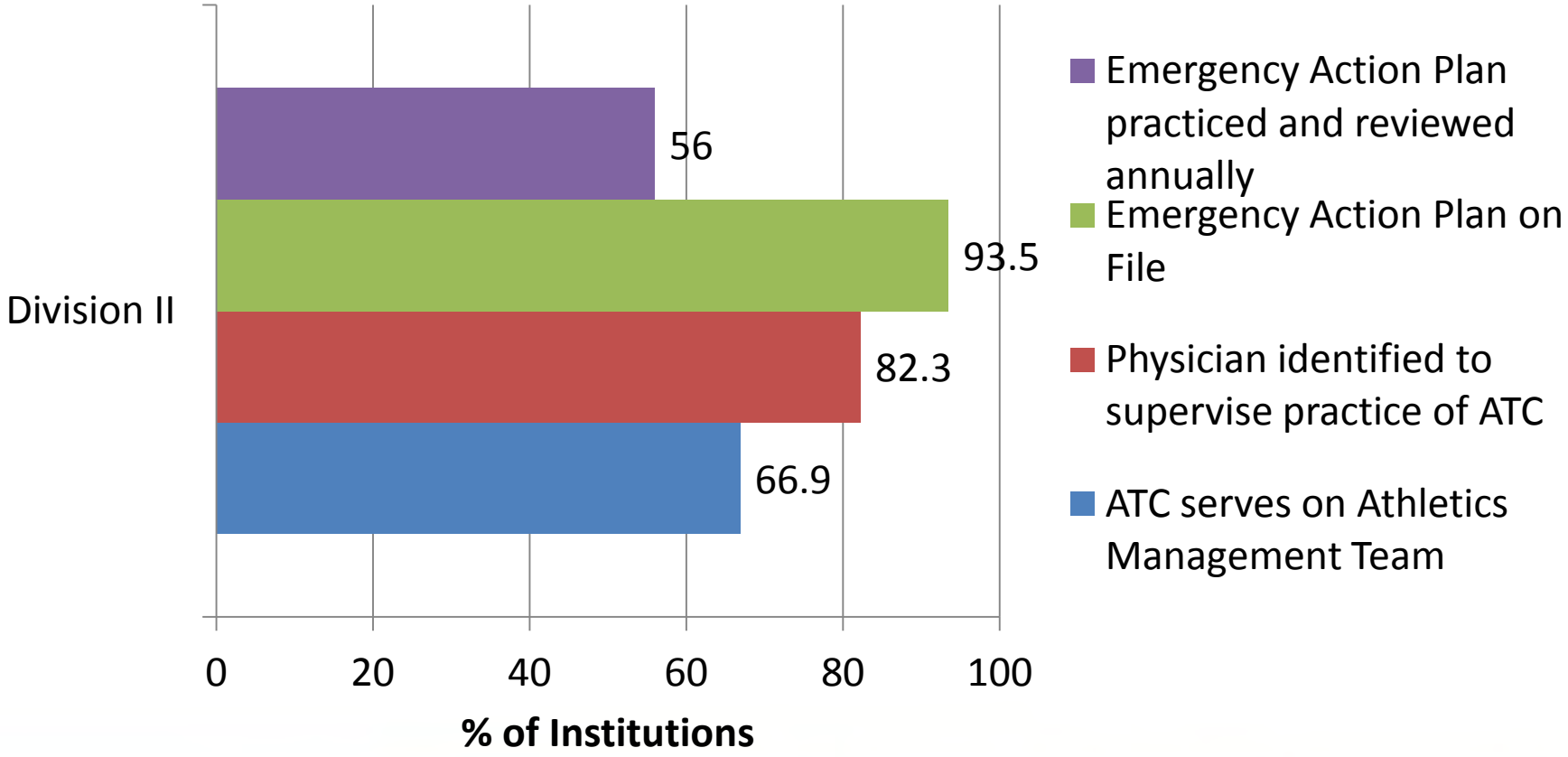

Average # of Full Time Certified Athletic Trainer
9
8
7
6
5
4
3
2
1
0
8.1
3.4
2.8
Division I
Division II
Division III


Division II
4.8
4.8
3.7
6.1
24.1
0
5
10
15
20
25
30
% of Institutions
Increased from 10 mo. To 12 mo. Contract
Decreased Months of Service
Eliminated Full Time ATC in last 12 mo.
Furloughs in last 12 mo.
Added ATCs in last 12 mo.

- 37% reported considering leaving NCAA Athletics do to time demands
- 74% work more than 50 hrs/wk

2006 Study

Administrative



Division II Reporting Structures and Employment

	Athletics Department	Campus Health Center	Non Institutional Entity	Not Applicable
Team Physician	19.4	12.0	64.0	4.6
Physician Assistant	4.7	10.5	30.2	54.7
Certified Athletic Trainers	90.9	2.3	4.0	2.9
Physical Therapist	3.5	1.2	34.1	61.2
Nurse Practitioner	0.6	63.6	9.2	26.6
Licensed Psychologist	2.3	38.6	13.5	45.6
Mental Health Counselor	2.3	60.5	8.7	28.5
Registered Dietician	1.2	12.9	18.7	67.3
First Responders	22.8	2.9	28.1	46.2

The Collegiate Surveillance Network

Division II has a historically LOW participation rate with NCAA Injury Surveillance Program

29% of Division II institutions report they do not electronically record student-athlete injuries.

Physician Care

- *Pre 2010 legislation* - A physician is *required* to see all student-athletes with a concussion. Overall, < 1/2 reported that they are.

Division I FBS	Division I FCS	Division I – No FB	Division II	Division III
80%	57%	74%	35%	36%
Division I Total = 71%				

Baseline Concussion Assessments

- *Pre 2010 best practices* - Overall, **66%** of respondents indicated their institution performs a baseline concussion assessment on their student-athletes. There were significant divisional differences:

Division I FBS	Division I FCS	Division I – No FB	Division II	Division III
86%	72%	67%	49%	66%
Division I Total = 77%				

Recommendations and Guidelines for

Appropriate Medical Coverage of Intercollegiate Athletics

NATA's Recommendations and Guidelines for Appropriate Medical Coverage of Intercollegiate Athletics (Revised) offer college and university health care providers a system by which they can evaluate their current level of coverage for student-athletes. These recommendations have been created for the safety of student-athletes competing at the collegiate level. To that end, certified athletic trainers in this setting must have a thorough understanding of the recommendations before implementing the system.

Useful Links

- Online Version of Worksheet
- Strategies for Using this Document
- Frequently Asked Questions
- Mentors

Overview

DEFINITION OF APPROPRIATE MEDICAL COVERAGE

Appropriate medical coverage involves more than basic emergency care during sports participation. It encompasses the provision of many other health care services for the student-athlete. While emergency medical care and event coverage are critical, appropriate medical coverage also includes activities of ongoing daily health care of the student-athlete, such as:

Considerations

1) How much of the budget is allocated for the Health Care of the student-athletes?
2) Are policies focused on event coverage or a more robust health care for their S-As?
3) How are the reporting lines structured to ensure that ATCs are not "controlled" by coaches?
4) Do team physicians and ATCs have final say on all health care related decisions and return to play?
5) Do the team physician(s) and ATCs annually review their "standing orders" or "plan of care"?
6) How do you decide sport coverage? Risk – Coach – Availability.

Dietary/Nutritional Supplement Products Sponsorship Survey - DRAFT

1. Division
    a. Division I FBS
    b. Division I FCS
    c. Division I No Football
    d. Division II

2. Institution – Drop Down Menu (Skip logic employed after #1 to a divisional institution list)

3. Does a dietary/nutritional supplement product company sponsor your institution in the form of providing **products** for your student-athletes? (Products could include electrolyte drinks, energy bars, vitamins, carbohydrate boosters, etc.)
    a. Yes
    b. No (Automatic skip to #6)

4. From the list below, please select those products that are being provided to your institution. (Select all that apply)
    a. Carbohydrate/electrolyte drinks (eg. Gatorade, PowerAde, Nunn)
    b. Carbohydrate boosters (eg. Ensure, Boost, Gu Gel)
    c. Carbohydrate energy bars (eg. PowerBar, Cliff Bar, Hammer Bars)
    d. Vitamins and minerals (eg. Centrum, One-A-Day)
    e. Pre work-out products (eg. Generation UCAN)
    f. Post work-out/recovery products (eg. Muscle Milk Collegiate, Shamrock Farms Rockin Refuel)
    g. Other_____
    h. None of the above

5. From the list below, please select those companies that sponsor your institution in the form of **providing products**. (Select all that apply)
    a. Cytosport
    b. Advocare
    c. Generation U-CAN
    d. MusclePharm
    e. Universal Nutrition
    f. BSN
    g. Herbal Life
    h. Powerbar
    i. FRS
    j. Max Hydration
    k. Onnit Labs
    l. Other _____

DRAFT - 5/26/12

6. How likely is it that your institution would support a recommendation from the NCAA Committee on Competitive Safeguards and Medical Aspects of Sports (CSMAS) **prohibiting** supplement product company sponsorships that provide **products** (*other than electrolyte drinks*) to institutions?
    a. Very likely
    b. Likely
    c. Somewhat likely
    d. Somewhat unlikely
    e. Unlikely
    f. Not at all likely

7. Please provide additional information regarding your response to #6.

8. What percentage of your overall student-athlete supplement use is provided free-of-charge from supplement product companies?
    a. 0%
    b. 1-25%
    c. 26-50%
    d. 51-75%
    e. 76-99%
    f. 100%

9. Does a dietary/nutritional supplement product company sponsor your institution in the form of **financial incentives**?
    a. Yes
    b. No (Automatic skip to #12)

10. From the list below, please select those companies that provide financial incentives to your institution. (Select all that apply)
    a. Cytosport
    b. Advocare
    c. Generation U-CAN
    d. MusclePharm
    e. Universal Nutrition
    f. BSN
    g. Herbal Life
    h. Powerbar
    i. FRS
    j. Max Hydration
    k. Onnit Labs
    l. Other_____

DRAFT - 5/26/12

11. How much does your institution receive annually in dietary/nutritional supplement company financial incentives? (Note: This could be monies directed to the athletic department or the general university fund.)
    a. Less than $1,000
    b. $1,000-5,000
    c. $5,001-15,000
    d. $15,001-30,000
    e. $30,001-50,000
    f. $50,001-75,000
    g. More than $75,000

12. How likely is it that your institution would support a recommendation from CSMAS **prohibiting** supplement product company sponsorships that provided **financial incentives** to institutions?
    a. Very likely
    b. Likely
    c. Somewhat likely
    d. Somewhat unlikely
    e. Unlikely
    f. Not at all likely

13. Please provide additional information regarding your response to #12.

14. Which of the following nutritional supplement products does your institution provide during **practices**? (Select all that apply)
    a. Carbohydrate/electrolyte drinks (eg. Gatorade, PowerAde, Nunn)
    b. Carbohydrate boosters (eg. Ensure, Boost, Gu Gel)
    c. Carbohydrate energy bars (eg. PowerBar, Cliff Bar, Hammer Bars)
    d. Vitamins and minerals (eg. Centrum, One-A-Day)
    e. Pre work-out products (eg. Generation UCAN)
    f. Post work-out/recovery products (eg. Muscle Milk Collegiate, Shamrock Farms Rockin Refuel)
    g. Other_____
    h. None of the above

15. Which of the following nutritional supplement products does your institution provide during **games**? (Select all that apply)
    a. Carbohydrate/electrolyte drinks (eg. Gatorade, PowerAde, Nunn)
    b. Carbohydrate boosters (eg. Ensure, Boost, Gu Gel)
    c. Carbohydrate energy bars (eg. PowerBar, Cliff Bar, Hammer Bars)
    d. Vitamins and minerals (eg. Centrum, One-A-Day)
    e. Pre work-out products (eg. Generation UCAN

DRAFT 5/26/12

f. Post work-out/recovery products (eg. Muscle Milk Collegiate, Shamrock Farms Rockin Refuel)
g. Other_____
h. None of the above

16. Which of the following nutritional supplement products does your institution provide during outside of practices or games on an **as-needed basis**? (Select all that apply)
    a. Carbohydrate/electrolyte drinks (eg. Gatorade, PowerAde, Nunn)
    b. Carbohydrate boosters (eg. Ensure, Boost, Gu Gel)
    c. Carbohydrate energy bars (eg. PowerBar, Cliff Bar, Hammer Bars)
    d. Vitamins and minerals (eg. Centrum, One-A-Day)
    e. Pre work-out products (eg. Generation UCAN)
    f. Post work-out/recovery products (eg. Muscle Milk Collegiate, Shamrock Farms Rockin Refuel)
    g. Other_____
    h. None of the above

17. Does your institution have different policies on the provision of supplement products based on sport group (e.g., are the policies for the provision of supplement products to football student-athletes different from soccer student-athletes)?
    a. Yes
    b. No (Automatic skip to #19)

18. You responded that your institution has different policies on the provision of supplement products based on sport group. Please provide further explanation below.

19. How much did your institution (including monies from the athletic budget or general university fund) spend on nutritional supplement products during the 2011-12 academic year?
    a. Less than $1,000
    b. $1,000-5,000
    c. $5,001-15,000
    d. $15,001-30,000
    e. $30,001-50,000
    f. $50,001-75,000
    g. More than $75,000

NOTE: Potential changes to Bylaw 16.5.2 are currently being discussed that would permit an institution or conference to provide, as a benefit incidental to participation, food to student-athletes at any time or during specified time periods. The manner in which this would potentially be implemented (e.g., application to all student-athletes, playing and practice periods only, etc.) is also being discussed.

DRAFT 5/26/12

20. If deregulation of food for all student-athletes (including walk-ons) is passed, which of the following is the likely response from your institution:
    a. Status quo – Will not change food provisions to student-athletes
    b. Provide more food to student-athletes but continue providing the same level of supplements
    c. Provide more food to student-athletes and ease reliance on supplements

21. How much do you agree or disagree with the following statement: Providing food to student-athletes is a healthier way of fulfilling their dietary needs than providing nutritional or dietary supplement products.
    a. Strongly Agree
    b. Agree
    c. Somewhat Agree
    d. Somewhat Disagree
    e. Disagree
    f. Strongly Disagree

22. If you have any additional comments regarding any of the topics covered in this survey, please provide those here.

DRAFT - 3/26/12

SUPPLEMENT NO. 33

2012 - 2013 NCAA NATIONAL STUDY OF SUBSTANCE USE HABITS OF COLLEGE STUDENT-ATHLETES

Sponsored by: The National Collegiate Athletic Association
Indianapolis, Indiana

Dear Student:

This survey of college students is part of the 2012-13 NCAA National Study of Substance Use Habits of College Student-Athletes. This nationwide study of college student-athletes seeks to assess your opinions and experiences with alcohol and other drugs. The results of the study will be used to develop programming and educational initiatives that will assist all of us associated with college athletics.

If this study is to be helpful, it is important that you answer each question thoughtfully and honestly. If you find a question that you feel you cannot answer honestly, we would prefer that you leave it blank. This information is being collected anonymously. We will not ask you to identify yourself or your school. That means nobody, including the NCAA or the researchers, will know your identity or be able to match your responses to your team or your school. Results that are reported will be in terms of sport trends or national averages; an individual's responses will never be reported in isolation.

Your participation in this study is voluntary. You can omit answers to any question or discontinue your participation in the study at any time without penalty. Your voluntary completion of this questionnaire constitutes your informed consent to participate in the study.

If you have questions regarding the study or survey, please contact Nicole Bracken, study coordinator, at 317/917-6328 or nmbracken@ncaa.org. For questions or concerns about how the study was carried out at your school, please contact Mike Miranda, research review board coordinator, at 317/917-6304 or mmiranda@ncaa.org.

Thank you very much for your participation in this important national study!

GLOSSARY OF TERMS

Alcohol: a nervous system depressant. Examples: beer, wine, distilled spirits

Amphetamines: any of a group of drugs that stimulate the central nervous system. Examples: Adderall, Benzedrine, Dexedrine, Ecstasy, Methamphetamine, Preludin, Ritalin. Also called: Speed, Dexies, Uppers, Black Beauties, Bennies, Stackers, Yellow Jackets, Mini-Thins

Anabolic steroids: any of a group of testosterone or its synthetic derivatives. Examples: Anavar, Andro or Norandro products, Boldenone, DHEA, Dianabol (D-bol), Nandrolone, Stanozolol, Testosterone

Cigarettes: a slender roll of cut tobacco enclosed in paper and meant to be smoked and does not include cigars, pipes, or flavored tobacco

Cocaine: a stimulant obtained from coca leaves that is used illicitly for its euphoric effects

Ephedrine: a stimulant usually used as a bronchodilator and decongestant. Examples: ephedra, ma huang

Marijuana: the dried leaves and flowering tops of the pistillate hemp plant that yield tetrahydrocannabinol (THC). Examples: hashish, cannabis

Narcotics: a drug that in moderate doses dulls the senses, relieves pain and induces profound sleep, but in excessive doses causes stupor, coma or convulsions. Examples: codeine, heroin, morphine, opium, oxycodone, percocet, tylenol with codeine, vicodin

Nutritional/dietary supplements: herbs, botanicals or any ingredient or product OTHER than ephedrine intended to supplement the diet. Examples: amino acids, creatine products, thermogenics, chromium, protein products, nitric oxide products, energy drinks, multivitamin and mineral products

Spit tobacco: products derived from tobacco plants that are absorbed through mucous membranes which contain the stimulant nicotine. Examples: Copenhagen, Skoal, Red Man

INSTRUCTIONS

Read each question carefully. For most questions you are asked to mark only one answer. If more than one answer is required, you will be asked to "mark all that apply" or be given other instructions. Please reference the "GLOSSARY OF TERMS" on page 1 for definitions of the substances listed.

Do not write your name or your school's name on the survey form.

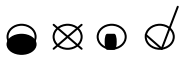
When you are finished, return your completed survey to the individual responsible for collecting them. This survey will take approximately **15 – 25** minutes to complete. If you have any questions, please ask the faculty member who distributed the survey.

Mark the circle that indicates your answer. Please stay within the circle and do not mark spaces between the circles. Please use only pencil or black ink.

Example:

Correct mark

Incorrect mark



SECTION 1: Institutional and Background Information

The following demographic information is collected to allow for the analysis of national trends by sex, year in school, sport and so on. Such information has proven valuable for NCAA policy makers in previous years.

1. What is your sex? (Mark ONE)
- ☐ Female
- ☐ Male

2. What is your current age? (Mark ONE)
- ☐ 18-20
- ☐ 21-23
- ☐ 23+

3. What is your current academic standing? (Mark ONE)
- ☐ Freshman
- ☐ Sophomore
- ☐ Junior
- ☐ Senior
- ☐ Graduate student

4. How do you describe yourself? (Mark ALL that apply)
- ☐ American Indian or Alaskan Native
- ☐ Asian
- ☐ Black or African American
- ☐ Hispanic or Latino
- ☐ Native Hawaiian or Pacific Islander
- ☐ White
- ☐ Other

5. What is your school's NCAA Division? (Mark ONE)
- ☐ Division I
- ☐ Division II
- ☐ Division III

6. What is the main sport for which you are currently or were a NCAA varsity team member? (Mark ONE)
- ☐ Baseball
- ☐ Basketball
- ☐ Bowling
- ☐ Cross country
- ☐ Fencing
- ☐ Field hockey
- ☐ Football
- ☐ Golf
- ☐ Gymnastics
- ☐ Ice hockey
- ☐ Lacrosse
- ☐ Rifle
- ☐ Rowing
- ☐ Skiing
- ☐ Soccer
- ☐ Softball
- ☐ Swimming/diving
- ☐ Tennis
- ☐ Track and field - indoor and/or outdoor
- ☐ Volleyball
- ☐ Water polo
- ☐ Wrestling
- ☐ Other

7. Which of the following best describes your current overall grade point average on a 4.0 scale? (Mark ONE)
- ☐ A (3.84-4.00)
- ☐ B+ (3.17-3.49)
- ☐ B- (2.50-2.83)
- ☐ C (1.84-2.16)
- ☐ D or below (< 1.50)
- ☐ A- (3.50-3.83)
- ☐ B (2.84-3.16)
- ☐ C+ (2.17-2.49)
- ☐ C- (1.50-1.83)
- ☐ I have not yet received any grades

8. What is your school's NCAA Division? (Mark ONE)
- ☐ Residence hall or other campus housing (not a fraternity/sorority)
- ☐ Fraternity/sorority house
- ☐ Division I No Football (formerly I-AAA)
- ☐ Off campus apartment/house (within walking distance of the college)
- ☐ Off campus apartment/house (not within walking distance of the college)
- ☐ With parents or family
- ☐ Other

SECTION 2: Substance Use Experience

9. Please indicate the age you first used the following substances: (Mark ONE for each substance)

	Never used	Under 10	10 – 11	12 – 13	14 - 15	16 - 17	18 - 20	21+
Alcohol.....								
Amphetamines....								
Anabolic steroids.								
Cigarettes.....								
Cocaine.....								
Ephedrine.....								
Marijuana.....								
Spit tobacco.....								

10. Please indicate your experience with the following substances: (Mark ONE for each substance)

	Never used	Used in the last 30 days	Used in the last 12 months	Used, but not in the last 12 months
Alcohol.....				
Amphetamines....				
Anabolic steroids.				
Cigarettes.....				
Cocaine.....				
Ephedrine.....				
Marijuana.....				
Spit tobacco.....				

11. Within the last 12 months, about how often have you used the following substances: (Mark ONE for each substance)

	Never used	Have not used in the last 12 months	Less than once a month	Less than once a week	1 - 2 days per week	3 - 4 days per week	5 - 6 days per week	Every day of the week
Alcohol.....								
Amphetamines....								
Anabolic steroids.								
Cigarettes.....								
Cocaine.....								
Ephedrine.....								
Marijuana.....								
Spit tobacco.....								

12. Please indicate the MAIN REASON you use(d) the following substances: (Mark ONE for each substance)

	Never used	To improve athletics performance	For sports related injuries	Reasons not related to sports	Pain management	Anxiety or depresseion	To aid sleep
Alcohol.....							
Amphetamines....							
Anabolic steroids.							
Cigarettes.....							
Cocaine.....							
Ephedrine.....							
Marijuana.....							
Spit tobacco.....							

13. Please indicate when you use(d) the following substances: (Mark ONE for each substance)

	Never used	Only during the competitive season	Only during the off season	During BOTH the off season and competitive season
Alcohol.....				
Amphetamines....				
Anabolic steroids				
Cigarettes.....				
Cocaine.....				
Ephedrine.....				
Marijuana.....				
Spit tobacco.....				

14. Please indicate when you use(d) the following substances: (Mark ONE for each substance)

	Never used	Use(d) before practice and/or competition	Use(d) during practice and/or competition	Use(d) after practice and/or competition
Alcohol.....				
Amphetamines....				
Anabolic steroids				
Cigarettes.....				
Cocaine.....				
Ephedrine.....				
Marijuana.....				
Spit tobacco.....				

15. Please indicate how often you have experienced the following because of your drinking during the last 12 months: (Mark ONE for each line)

	Never	Once	Twice	3 – 5 times	6 – 9 times	10 + times
Had a hangover						
Got nauseated or vomited						
Performed poorly on a test or important project						
Missed a class						
Performed poorly on a test or important project						
Showed up late or missed practice or game						
Performed poorly in practice or game						
Been hurt or injured						
Got into an argument/fight						
Had a memory loss						
Done something I later regretted						
Been criticized by someone I know						
Thought I might have a drinking or other drug problem						
Had feelings of depression, feeling sad for two weeks or longer						
Tried unsuccessfully to stop drinking						
Damaged property, pulled fire alarm, etc						
Been in trouble with police or other college authorities						
Drove a car while under the influence						
Been arrested for DWI/DUI						

16. When you drink alcohol, typically how many drinks do you have in one sitting? (Mark ONE)

- ☐ I do not drink alcohol
- ☐ 1
- ☐ 2
- ☐ 3
- ☐ 4
- ☐ 5
- ☐ 6
- ☐ 7
- ☐ 8
- ☐ 9
- ☐ 10+

One drink:
One 12 oz. beer
One 4 oz. glass of wine
One 12 oz. beer wine cooler
One mixed drink (w/ 1 shot)
One shot glass of liquor

17. Mark the MAIN REASON why you have never used or have stopped using the following substances: (Mark ONE for each substance)

	I still use this substance	Concerned aoubt what it might do to my health	Against my beliefs/ values	No desire to experience the effects	Hurt my athletic performance	Afraid of the consequences of being caught	Fear of getting drug
Alcohol							
Amphetamines							
Anabolic steroids							
Cigarettes							
Cocaine							
Ephedrine							
Marijuana							
Spit tobacco							

SECTION 3: Drug Testing

18. Have YOU ever been drug tested by any of the following: (Mark ONE for each line)

	Yes	No
Your college's drug testing program		
The NCAA		
A national or international sports governing body		

19. To your knowledge, have other members of YOUR TEAM been drug tested by any of the following: (Mark ONE for each line)

	Yes	No
Your college's drug testing program		
The NCAA		
A national or international sports governing body		

20. There has been a lot of discussion about whether athletes should be tested for drug use. Please indicate your level of agreement with the following statements: (Mark ONE for each line)

	Strongly agree	Agree	Disagree	Strongly disagree	No opinion
All professional athletes should be tested...					
All Olympic athletes should be tested.....					
All college athletes should be tested by their school.....					
All college athletes should be tested by the NCAA.....					
Drug testing by individual colleges has deterred college athletes from using drugs.....					
Drug testing by the NCAA has deterred college athletes from using drugs.....					
Imposing team penalties (such as disqualification of the team when a member tests positive for banned drugs) would be fair and appropriate.....					

SECTION 4: Substance Information

21. What are your sources of information about each of the following: (Mark ONE for each substance)

	Athletics staff	Teammate or other athlete	Internet or magazine	Nutritionist of dietician	Local gym or retail store	Resource Exchance Center (The R.E.C.)	None of the above
Amphetamines.....							
Anabolic steroids.....							
Cocaine.....							
Dietary Supplements...							
Ephedrine.....							
Marijuana.....							

22. Where do (did) you get the following substances: (Mark ONE for each substance)

	Never used	Athletic trainer or team physician	Coach or strength coach	Teammate, friend or family	Internet or magazine	Nutritionis or detician	Local gym or retail store	None of the above
Amphetamines.....								
Anabolic steroids.....								
Cocaine.....								
Dietary Supplements...								
Ephedrine.....								
Marijuana.....								

SECTION 5: Prescription Drugs and Other Supplements

23. Within the last 12 months, have you taken any attention deficit-hyperactivity disorder (ADHD) medications with or without a prescription? (Mark ALL that apply)

- ☐ I have not taken attention deficit-hyperactivity disorder (ADHD) medications with or without a prescription
- ☐ I have taken Adderall OR Ritalin **with** a prescription
- ☐ I have taken Adderall OR Ritalin **without** a prescription
- ☐ I have taken OTHER attention deficit-hyperactivity disorder (ADHD) medications

25. Within the last 12 months, have you taken any of these asthma medications with or without a prescription? (Mark ALL that apply)

- ☐ I have not taken asthma medications with or without a prescription
- ☐ I have taken Albuterol (e.g.Proair, Proventil) **with** a prescription
- ☐ I have taken Albuterol (e.g.Proair, Proventil) **without** a prescription
- ☐ I have taken OTHER prescription asthma medications

26. Within the last 12 months, have you taken any of these pain management medications with or without a prescription? (Mark ALL that apply)

- ☐ I have not taken pain management medications with or without a prescription
- ☐ I have taken Tylenol with Codeine, Vicodin, Oxycontin OR Percocet **with** a prescription
- ☐ I have taken Tylenol with Codeine, Vicodin, Oxycontin OR Percocet **without** a prescription
- ☐ I have taken OTHER prescription pain management medications

27. Have you continued use of pain management medications beyond their intended use? (Mark ONE)

- ☐ Yes
- ☐ No

28. For what purpose have you continued use of pain management medications beyond their intended use? (Mark ALL that apply)

- ☐ I have not taken these medications beyond their intended use
- ☐ Recreation
- ☐ Sports injury
- ☐ Injury outside of sports
- ☐ Surgery due to athletics injury
- ☐ Surgery due to non-athletics injury
- ☐ Other

29. Which of the following ergogenic aid (i.e. muscle building aids) have you taken while in college? (Mark ALL that apply)

- ☐ I have not taken any of the items listed below
- ☐ Andro or norandro product
- ☐ Beta-methyl butyrate (HMB)
- ☐ Clenbuterol
- ☐ Dehydroepiandrosterone (DHEA)
- ☐ Epitestosterone
- ☐ Erythropoietin (EPO)
- ☐ Gamma hydroxbutyrate (GHB)
- ☐ Human chorionic gonadotrophin (HCG)
- ☐ Human growth hormone (HGH) - Injected form
- ☐ Human growth hormone (HGH) - Oral form
- ☐ Insulin
- ☐ Testosterone
- ☐ Testosterone boosters

30. Which of the following dietary supplements have you taken while in college? (Mark ALL that apply)

- ☐ I have not taken any of the items listed below
- ☐ Amino acids
- ☐ Chromium
- ☐ Creatine
- ☐ Glucosamine
- ☐ General multivitamin
- ☐ Multivitamin with caffeine
- ☐ Multivitamin and mineral with other additives

31. Which of the following have you taken while in college? (Mark ALL that apply)

- ☐ I have not taken any of the items listed below
- ☐ Bath salts
- ☐ Diuretics (e.g. water pills)
- ☐ Energy boosters (e.g. energy drinkgs/shots)
- ☐ Protein products
- ☐ Sleep aid (e.g. Ambien)
- ☐ Synthetic marijuana (e.g. Spice, K2)
- ☐ Weight gain products
- ☐ Weight loss products

THANK YOU!

This completes the questionnaire. Thank you for your participation in this study.

To ensure your anonymity, please put your completed questionnaire in the collection envelope.

BACKGROUNDER – NCAA Drug Use Deterrence and Drug Testing

The NCAA employs a comprehensive approach to deter drug use, consisting of strong policy and serious sanctions, effective education and short notice random drug testing. Drug use deterrence is a shared responsibility between the National office and NCAA member institutions, which are required to educate student-athletes about banned substances and the products that may contain them. Reports of substance use received by the National office are sent to the director of athletics to follow up according to institutional policy. Student-athletes are subject to testing at any NCAA championships, and Division I and II programs are subject to NCAA drug testing at any time.

The NCAA bans drugs by class; the NCAA banned drug classes are: stimulants, anabolic agents, alcohol and beta blockers (rifle only), diuretics and urine manipulators, street drugs, peptide hormones and analogues, anti-estrogens and beta-2 agonists. Any substance chemically related to the banned drug class is banned unless exempted.

The NCAA began testing at its championships in 1986, and added year round testing in DI and DII in 1990. In addition to NCAA administered drug testing, according to the 2011 NCAA Survey of Institutional Drug Education and Testing, 90 percent of Division I, 68 percent of Division II and 24 percent of Division III schools also conduct an institutionally administered drug-testing program. Two athletic conferences currently conduct independent conference administered drug testing. The NCAA spends approximately $4.5 million each year to collect and analyze approximately 13,500 samples through its national drug-testing program, and it provides more than $1,500,000 each year in funding assistance for drug-education programs to NCAA colleges and universities.

The NCAA sponsors two national drug testing programs – at NCAA championships and on campus throughout the year.

- The NCAA guarantees that every championship is tested at least once every five years, and some championships are tested every year. About 2,500 student-athletes are tested annually in the championships program, which tests for steroids, diuretics and masking agents, stimulants, peptide hormones, anti-estrogens, beta-2 agonists, beta blockers and street drugs.

- NCAA year-round testing focuses heavily on football, but any Division I or Division II student-athlete in any sport may be tested. About 11,000 student-athletes are randomly selected from squad lists and tested by the NCAA on their campuses August through July. The year-round program tests for steroids, diuretics and masking agents and peptide hormones. The NCAA extended year-round testing into the summer months in 2006.

NCAA drug-test samples are collected and processed by an independent certified drug-testing collection agency. All NCAA samples are collected and analyzed under a strict, published protocol using laboratories certified by the World Anti-Doping Agency (WADA), which establishes Olympic anti-doping policies.

Sanctions for a positive drug test are swift and automatic. After the first positive test, a student-athlete cannot compete in any intercollegiate sport for one year, and the student-athlete loses one of his or her four years of eligibility. After the second positive test for a performance-enhancing drug, a student-athlete loses all remaining eligibility and is permanently banned from intercollegiate competition. The NCAA affords an appeal process for a positive drug test that may reduce or eliminate the sanction. A medical exception procedure is available to review the use of prescribed medications that contain a banned substance in all classes except for street drugs.

A student-athlete under a drug-testing suspension from a national or international sports governing body that has adopted the WADA code may not participate in NCAA intercollegiate competition through the duration of that suspension.

Below are snapshots of the number of positive tests for the past 9 years:

2002-03 -- 85 of 10,679 tests were positive for anabolic steroids
2003-04 -- 46 of 10,541 tests were positive for anabolic steroids
2004-05 -- 51 of 11,610 tests were positive for anabolic steroids
2005-06 -- 52 of 12,671 tests were positive for anabolic steroids
2006-07 -- 30 of 13,049 tests were positive for anabolic steroids
2007-08 -- 64 of 13,586 tests were positive for anabolic steroids
2008-09 – 53 of 13,569 tests were positive for anabolic steroids
2009-10 – 52 of 13,543 tests were positive for anabolic steroids
2010-11 – 54 of 12,585 tests were positive for anabolic steroids

The NCAA conducts research to identify reported use and trends. Every four years approximately 20,000 NCAA student-athletes representing all sports complete an anonymous survey regarding drug use. Following are percentages for reported steroid use by baseball, basketball, football and ice hockey student-athletes from the NCAA drug use studies:

	Baseball	Basketball	Football	Ice hockey	
1989			9.7%		
1993	0.7%	2.6%	5.0%		
1997	1.9%	0.6%	2.2%	1.2%	
2001	2.3%	1.4%	3.0%	1.6%	
2005	2.3%	1.5%	2.3%	1.2%	
2009	2.7%	0.9%	2.0%	1.7%	

H.R.3724
Latest Title: Medical Controlled Substances Transportation Act of 2011
Sponsor: Rep Sessions, Pete [TX-32] (introduced 12/16/2011) Cosponsors (None)
Latest Major Action: 1/6/2012 Referred to House subcommittee. Status: Referred to the Subcommittee on Crime, Terrorism, and Homeland Security.

SUMMARY AS OF:
12/16/2011--Introduced.

Medical Controlled Substances Transportation Act of 2011 - Amends the Controlled Substances Act to authorize a physician registered to dispense, or conduct research with, schedule II, III, IV, or V controlled substances to enter into an agreement with the Attorney General that authorizes the physician to transport controlled substances from a practice setting to another practice setting or to a disaster area and that requires the physician to: (1) notify the Attorney General in advance of any such transport; (2) identify the controlled substances to be transported, the areas from which and to which the substances will be transported, and the intended dates, mode, time, and duration (not to exceed 72 hours) of transport; and (3) maintain records in the physician's primary practice setting on the dispensing of any substance transported.

MAJOR ACTIONS:

NONE

ALL ACTIONS:
12/16/2011:
Referred to the Committee on Energy and Commerce, and in addition to the Committee on the Judiciary, for a period to be subsequently determined by the Speaker, in each case for consideration of such provisions as fall within the jurisdiction of the committee concerned.
12/16/2011:
Referred to House Energy and Commerce
12/16/2011:
Referred to the Subcommittee on Health.
12/16/2011:
Referred to House Judiciary
1/6/2012:
Referred to the Subcommittee on Crime, Terrorism, and Homeland Security.

TITLE(S): (*italics indicate a title for a portion of a bill*)

- SHORT TITLE(S) AS INTRODUCED:
  Medical Controlled Substances Transportation Act of 2011

- OFFICIAL TITLE AS INTRODUCED:
  To amend the Controlled Substances Act to authorize physicians, pursuant to an agreement with the Attorney General, to transport controlled substances from a practice setting to another practice setting or to a disaster area.

COSPONSOR(S):

NONE

NCAA Medical Exception Documentation Reporting Form
to Support the Diagnosis of Attention Deficit Hyperactivity Disorder (ADHD)
and Treatment with Banned Stimulant Medication

- Complete and maintain (on file in the athletics department) this form and required documentation supporting the medical need for a student-athlete to be treated for ADHD with stimulant medication.
- Submit this form and required documentation to Drug Free Sport in the event the student-athlete tests positive for the banned stimulant (see Drug Testing Exceptions Procedures at www.ncaa.org/drugtesting).

To be completed by the Institution:

Institution Name:_____

Institutional Representative Submitting Form:
Name_____
Title_____
Email_____
Phone_____

Student-Athlete Name_____
Student-Athlete Date of Birth_____

To be completed by the Student-Athlete's Physician:

Treating Physician (print name):_____
Specialty:_____
Office address_____
Physician signature:_____ Date_____

Check off that documentation representing each of the items below is attached to this report.

- o Diagnosis.
- o Medication(s) and dosage.
- o Blood pressure and pulse readings and comments.
- o Note that alternative non-banned medications have been considered, and comments.
- o Follow-up orders.
- o Date of clinical evaluation: _____
- o **Attach written report summary of comprehensive clinical evaluation:**
- o The evaluation should include individual and family history, address any indication of mood disorders, substance abuse, and previous history of ADHD treatment, and incorporate the DSM criteria to diagnose ADHD. Attach supporting documentation, such as completed ADHD Rating Scale(s) (e.g., Connors, ASRS, CAARS) scores.
  The evaluation can and should be completed by a clinician capable of meeting the requirements detailed above.

Deleted: Physician documentation (letter, medical notes) to include the following information:

DISCLAIMER: The National Collegiate Athletic Association shall not be liable or responsible, in any way, for any diagnosis or other evaluation made, or exam performed, in connection herewith, or for any subsequent action taken, in whole or in part, in reliance upon the accuracy or veracity of the information provided hereunder.

SUPPLEMENT NO. 37

P.O. Box 6222
Indianapolis, Indiana 46206
Telephone: 317/917-6222

Shipping/Overnight Address:
1802 Alonzo Watford Sr. Drive
Indianapolis, Indiana 46202

www.ncaa.org

April 11, 2006

MEMORANDUM

TO: Directors of Athletics]
Compliance Officers] of NCAA Division I and II Member Institutions

FROM: Dr. Michael Krauss, chair of the Committee on Competitive Safeguards and Medical Aspects of Sports

SUBJECT: Year-Round Drug Testing to Include Summer Testing – Effective Immediately

The NCAA Committee on Competitive Safeguards and Medical Aspects of Sports, the Association-wide committee charged by the NCAA Executive Committee with oversight of NCAA drug testing, has directed The National Center for Drug Free Sport (Drug Free Sport), the NCAA third party drug-testing administrator, to include summer drug testing in the year-round program, pending final budget approval. Drug testing provides a health and safety benefit to all student-athletes by deterring banned drug use and supporting student-athletes in their efforts to compete fairly. As the summer months continue to represent a time of training, conditioning, and sanctioned sport league play, student-athletes' susceptibility to external influence and pressure to use performance-enhancing substances increases, thus posing a greater need to close the gap in the NCAA year-round drug-testing program. NCAA Bylaws 18.4.1.5.2 and 21.2.2.2 provide authority for this action, and the Student-Athlete Statement and Drug Testing Consent forms provide notice to and consent by student-athletes in the signing of the statement/consent form, in that it shall be in effect from the date the document is signed and shall remain in effect through August 31 of the following year.

Later this month, you will receive further details and instructions from Drug Free Sport on the steps your institution will need to take to accommodate summer drug testing. As NCAA drug testing is a compliance responsibility codified in NCAA legislation, assurance of the assignment of the summer drug-testing site coordinator falls to the office of compliance. The person selected to perform these duties should be an institutional staff member who is employed year round, so as not to present undue time demands on those who are not scheduled to perform duties throughout the summer months.

The first year of summer drug testing will focus primarily on Division I football and baseball. Programs to be tested will be randomly selected from the entire pool of Division I institutions, with summer site coordinators notifying randomly-selected student-athletes, and advising Drug Free Sport of their contact information. However, all Division I and II student-athletes, whether they are on-campus or away from campus, are subject to this testing requirement. The current drug-testing protocol will be followed, with strict chain-of-custody, confidentiality, penalty and appeal processes maintained.

For your reference, an information article on this issue was published in the NCAA News March 27, 2006. You can access that article by going to www.ncaa.org/health-safety, under the Hot Topic. If you have questions please contact Mary Wilfert, with the NCAA at 317/917-6319 or Andrea Wickerham, with Drug Free Sport at 816/474-8655 ext. 118.

MK:clm

cc: Conference Commissioners

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- Practice Hard!

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INSERT LOGO

w/Name: The Coalition of Higher Education Associations for Substance Abuse Prevention

Why and How Campuses Should Address Substance Abuse Prevention
Focus: Non-Medical Use of Prescription Drugs

CoHEASAP (www.collegesubstanceabuseprevention.org) brings together representatives of over 20 diverse higher education organizations to set a national agenda for substance abuse prevention on campus The coalition model implies a shared, campus-wide responsibility , with input and active involvement from the many offices and individuals committed to the health and safety of students (see www.higheredcenter.org,). An integrated model of campus coalition can be found at www.caph.gmu.edu/PromPrac/htdocs/2005/TaskForcePlannerGuide.pdf

An Ongoing Problem

Substance abuse has been identified by college presidents as the number one issue impeding student success. Studies show that students perform better socially, emotionally and academically when they make more positive choices related to substance use – see a snapshot of high-risk drinking consequences at www.collegedrinkingprevention.gov/NIAAACollegeMaterials/TaskForce/Intro_03.aspx

Healthy living practices – stress management, good nutrition, adequate rest and active lifestyle – all improve when alcohol and other drug use is minimized, and conversely campuses that provide comprehensive support for healthy behaviors reduce heavy drinking and its negative consequences [Jim Grizzell data here? http://www.csupomona.edu/~jvgrizzell/hc2010/] .
More information about these issues and Best Practices can be found at the Department of Education's Higher Ed Center, www.higheredcenter.org and at www.CollegeDrinkingPrevention.gov .

Hot Topic: Non-Medical Use of Prescription Drugs

An integrated and comprehensive campus approach to reduce the negative outcomes of substance abuse includes incorporating new information about emerging issues that arise on the campus scene. Once such growing concern is related to the non-medical use of prescription drugs.
Easy access and perceptions that misuse of these substances do not carry the same risks as street drugs has led to cavalier attitudes around sharing others' medications without proper medical monitoring. Though reported misuse is around 10 percent, this represents an upward trend. Campus health and wellness efforts should include information about the risks of non-medical use of prescription drugs, and provide students with best practices to avoid negative consequences associate with misuse.
Those resources and more details regarding this emerging issue can be found on the CoHEASAP website at www.collegesubstanceabuseprevention.org (Stacey Andes PowerPoint, OSU Rx website, etc.)

Deleted: ¶

DRAFT

ACHA 2012
ACHA Theme: Innovative and effective practices that promote health and well-being on our campuses, addressing critical issues in college health –and address successful advocacy efforts at the local, state and federal level.

Title: A Whole New Ballgame: How an Interdisciplinary Approach to Student-Athlete Health Care Benefits the Broader Campus.
Wednesday May 30, 1:45 PM - 3:15 PM

Workshop Description:
Sports medicine and student health professionals will discuss why and how to integrate and replicate an interdisciplinary and collaborative approach to health promotion and health care of college athletes, and how partnership and shared resources can contribute to overall campus health and safety. The panel will identify challenges, best practices to achieve a more efficient and effective delivery of services, and present existing successful models, and resources developed in collaboration among national advocacy groups.

Topic relevance: Student-athletes represent a significant number of students on our college campuses, are seen as student leaders, and many utilize the services provided by student health. Many health issues that impact student-athletes also have relevance to the health and well-being of the general student body – such as injury, alcohol and other drug use, mental health -- and finding ways to partner/collaborate between sports medicine and student health creates efficiencies and advances in health promotion and health care.

Workshop **Learning Objectives:**
The participant in this workshop will be able to

1. Recognize personal health and medical issues, including mental health, of today's collegiate athlete
2. Identify the benefits to the broader campus of engaging student health and counseling services and sports medicine in collaborative health care
3. Explore (replicate) various campus models that integrate student health and sports medicine services

Incorporated Data and Resource: ACHA-NCHA, NCAA Health Care Survey, NCAA Sports Medicine Handbook, CDC initiatives, other

Presenters:
Kenneth Chew, ISU
Lori DeWald, NATA liaison to ACHA
Tiandra Finch, NCAA Intern and ATC
Jerry Hilker, TAMUCC
Mary Wilfert, NCAA

College/University Athletic Trainers' Committee
Report to CSMAS June, 2012

Melinda Larson

Inter-Association Task Force for Preventing Sudden Death in Collegiate Conditioning Sessions Best Practices Recommendations
This report was supported by CUATC chair Charlie Thompson. It will be published in the June or July Journal of Athletic Training

Education Modules
We are continuing work on developing template presentations to distribute to our constituents. Examples of topics included are Asthma, Emergency Planning, and Concussions. The presentations are currently being sent to content experts for review and then will be published by the NATA.

Value Model
The NATA Committee on Revenue is partnering with various practice setting groups to develop an educational tool informing both ATs and their stakeholders regarding the value of athletic trainers. The collegiate value model will focus on their entire athletics environment and not just a reimbursement model. The focus will be student-athlete well-being and success. This model will include identifying the multi-faceted role of the collegiate athletic trainer and key metrics that can be measured. This document is near final editing stages and will be reviewed by the CUATC during their June meeting.

Athletic Training facility certification
Work continues in collaboration with the NATA Board of Certification regarding various standards.

Sickle Cell
Communication from the CUATC should support the NCAA position and focus on education of administrators and coaches.

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Facility and Patient Care Principles – Summary

Executive Summary

The delivery of healthcare in the United States is heavily regulated. Numerous organizations, local, state and federal, are responsible, often overlapping, for ensuring the quality of facilities where healthcare services are delivered through the promulgation of regulations and standards. Practice administrators, risk management professionals, and practitioners are very aware of these standards as they work to maintain the accreditation of their facilities. This accreditation is given by third parties such as the Joint Commission, Center for Medicare Services, Health Departments, etc.

Athletic healthcare services are not commonly delivered in an orthodox healthcare facility; those governed by third party accreditation such as The Joint Commission, the Accreditation Association for Ambulatory Health Care or the Centers for Medicare Services. Most commonly it is the athletic training facility that serves as this point for healthcare. Many administrators are unaware of the myriad of federal, state, and local regulations and standards which govern the delivery of athletic healthcare. A review of possible regulations on the national level indicates that no less than eight Federal agencies have regulations that can impact athletic healthcare delivery. In addition to the Federal regulations, local, state and national non-governmental bodies have issued rules, regulations and position statements that must be considered.

The BOC has identified a need for the development of a document for those involved in athletic healthcare delivery. This document will help ensure the administration and oversight boards are aware of these standards. In July of 2011 the BOC, as part of the joint Vision Quest project of the BOC, CAATE and the NATA, pledged the formation of an inter-organizational ad hoc group. The working group is charged with reviewing the existing regulations and recommending the development of educational materials and tools to assist those involved in delivery of athletic healthcare. These materials and tools will provide the means for secondary and post-secondary educational institutions and organizations to self-assess their policies, procedures and facilities to ensure the safe, effective and legal provision of athletic healthcare.

In August of 2011 a panel of subject matter experts (Appendix IV) was chosen to begin working on developing educational materials and/or tools to fulfill the goal of enhancing the delivery of athletic healthcare by allowing self assessment of their facility and principles of patient care.

Appendix IV – Committee Bios

Name	Affiliation
David Klossner, PhD, ATC	NCAA
Randy Cohen	NATA-CUATC
Jerry Diehl	BOC Board of Directors
Doug Killgore	NIAAA
Steven Danon	Risk Manager
David Edell	NATA-SSATC
Brian Michelotti	NFHS

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1. ***The importance of the topic to the field and to the NCAA (30 points)***

a. **Is the topic worthwhile?**
b. **What literature already exists on this topic?**
c. **Is the proposed research duplicating current NCAA research initiatives?**
d. **Will this study contribute to the NCAA?**
e. **Does the research have implications for understanding or improving the student-athlete experience?**
f. **Will the study findings inform current or future policy or best practices? Will the field learn anything**

2. ***The strength of the methodological approach (25 points)***

a. **Is the student's research question clearly defined?**
b. **Is it likely that the researcher can accomplish the proposed research?**
c. **How does the methodology relate specifically to the research question?**
d. **Does the researcher have the necessary expertise and prior experience to accomplish the work?**
e. **Will the researcher be able to recruit enough subjects?**
f. **Is the conceptual basis of the research well-developed, empirically sound and likely to enhance**
g. **Is the researcher using the best measures available?**

3. ***The appropriateness of proposed analyses (20 points)***

a. **Is the proposed data analysis technically sound and does the researcher have the technical expertise**
b. **Has the researcher incorporated the important factors in the area into the design, conceptualization,**

4. ***The contribution to the field (15 points)***

a. **Does the research break new conceptual or methodological ground in its scientific field?**
b. **Will the knowledge gained be very new or different from existing knowledge in the field?**
c. **Has similar research already been conducted in the field?**
d. **Does the research extend previous research and provide new and updated results?**

5. ***The acceptability of the proposed budget (10 points)***

a. **Will the money help the principal investigator work towards finalizing the research?**
b. **Can the study be completed on the proposed budget?**
c. **Are the financial resources indicated in the budget description being used appropriately?**

Developing a webinar on the topic of Severe Weather.

Co-branded with NOAA and some new material hosted in Indianapolis.
Hopefully by the end of the fall we increase the number of schools with NOAA recognition status.

Webinar
Fact sheet
Poster
Large Venue Safety Plan

Serves as a best practice extension of our Sports medicine handbook guideline.

Slides 8, 9, 16, 18-21 of the presentation may be of use in the fact sheet/poster

Lightning Safety

Learning Objectives

What you Need to Know: Learn the Facts and Risks of lightning strikes.
You and Your Team:
- When Thunder Roars, Go Indoors.
- Understand the flash-to-bang method for determining when to seek shelter and/or resume athletics activity.
- Identify the characteristics of a safe shelter and location of safe shelter sites of common athletics venues.
- Understand the criteria for Return to Activity following a lightning delay.
- Understand the proper response if someone is struck by lightning.

Safety Plans.
- Personal safety/team practices.
- Large Venue.
- Use of electronic monitoring systems.

Dave,

We spoke last year about taking initiatives across the NCAA in regards to ensuring each institution has a lightning safety plan in place. I developed a lightning safety plan that large venues are being approached with by the National Weather Service. In 2011, Georgia Tech became the first university to complete this program. I have attached the press release and a picture of the recognition. Does the NCAA have any publications or forums where this could be shared? I was happy to see Georgia Tech embrace this safety plan for lightning and severe weather. I'm hoping others will follow.

If there's anything I can do to help out the NCAA from a National Weather Service and lightning safety perspective in the future let me know.

Thanks,
Charlie
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Charlie Woodrum
National Weather Service
Meteorologist
WFO Pittsburgh, PA
http://www.weather.gov/pbz

NOAA NATIONAL OCEANIC AND ATMOSPHERIC ADMINISTRATION
UNITED STATES DEPARTMENT OF COMMERCE

Contact: Barry Gooden
770-486-1133

FOR IMMEDIATE RELEASE
December , 2011

Georgia Tech's Bobby Dodd Stadium receives National Weather Service lightning safety recognition

Georgia Tech football fans have something new to cheer about. Officials at the team's home field – Bobby Dodd Stadium in Atlanta – have developed a lightning safety plan for large outdoor venues based on NOAA's new lightning safety toolkit. Home to Georgia Tech football games for nearly 100 years, the historic sports facility is now safer for the nearly 60,000 patrons who gather there with the distinction of being the nation's first stadium to earn this achievement.

The volunteer program is part of a National Weather Service lightning safety campaign for large outdoor venues.

The stadium's lightning safety plan includes a locally run lightning detection system on site, a written lightning safety plan with specific instructions for contacting local emergency management officials and the National Weather Service, procedures to notify patrons that a lightning threat exists, an emergency operations plan to evacuate the venue and identify locations of available shelters and a way to advise patrons of lightning safety procedures and the appropriate response to a lightning threat.

Safety managers of large outdoor venues are welcomed and encouraged to use the National Weather Service lightning safety toolkit to develop a lightning safety plan. The toolkit is available at: http://www.lightningsafety.noaa.gov/resources/large_venue.pdf.

"This toolkit offers outdoor venue managers a great opportunity to develop new plans or enhance existing plans for severe weather," said Charlie Woodrum, a meteorologist with the National Weather Service. "Georgia Tech sets a great example of how universities throughout the nation can benefit from the toolkit."

"The threat of lightning-related injuries or fatalities to sports fans in large outdoor venues is a growing concern for all of us," said Lans Rothfusz, meteorologist-in-charge of the National Weather Service forecast office in Atlanta. "This program is designed to help reduce the risk through improved preparedness on the part of event staff and emergency managers and increased public awareness of the hazards associated with thunderstorms and lightning."

For more information on lightning safety, visit: http://www.lightningsafety.noaa.gov.

The National Weather Service is the primary source of weather data, forecasts and warnings for the United States and its territories. It operates the most advanced weather and flood warning and forecast system in the world, helping to protect lives and property and enhance the national economy. Working with partners, the National Weather Service is building a Weather-Ready Nation to support community resilience in the face of increasing vulnerability to extreme weather. Visit us online at weather.gov and on Facebook.

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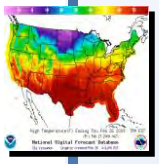
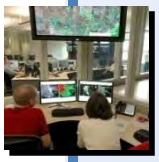
National Weather Service

Protecting Lives and Property





Using a Lightning Safety Toolkit for Outdoor Venues

NOAA Lightning Safety Team

Charlie Woodrum, NWS Pittsburgh, PA

Topics to be Covered

- Motivation
- Recent Lightning Delays
- Safety Toolkit
- Recognition Ceremonies
- Proactive Examples
- Outreach

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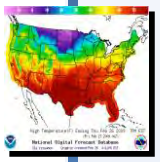

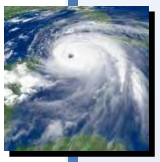

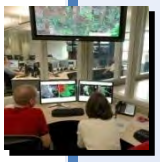

Motivation

- NWA Conference 2008, Louisville, KY
- West Virginia @ Auburn – 1 hour, 3 minutes

The message displayed on the video scoreboard during the weather delay. (Photo courtesy of Andrew McKaughan, NOAA/NWS, Tampa, FL)

Game operations crew monitoring the weather during the weather delay. (Photo courtesy of Susan McCallister, Auburn University Emergency Management)

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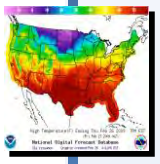

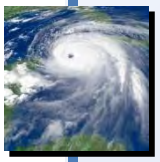

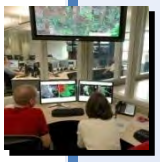

South Florida @ Notre Dame

September 3, 2011 – 2 delays → 2 hours, 33 minutes

Cloud to ground lightning strike just behind Notre Dame stadium.
(Photo courtesy of NBCSports)

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Western Michigan @ Michigan

September 3, 2011 – 2 delays → 1 hour, 20 minutes

Radar imagery displayed on the video board at Michigan Stadium.
(Photo courtesy of the Associated Press)

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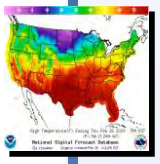

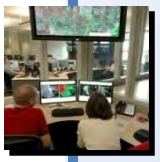

Oklahoma State @ Tulsa

September 17, 2011 – 3 hours, 6 minutes

Cloud-to-cloud lightning strikes over H.A. Chapman Stadium.
(Photo courtesy of Mike Simons/Tulsa World)

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Oregon @ Tennessee

September 11, 2010 – 1 hour, 10 minutes

- **Southeastern Conference (SEC) and NCAA rule**
  - *6 mile radius*
- **Too close for a 100,000 seat stadium!**

Spectators cram into the stadium concourses (Photo courtesy of Terry Taylor/RV Goddess.com)

Cloud-to-ground lightning strike during the delay at Neyland Stadium
(Photo courtesy of http://www.wbir.com/news/local/story.aspx?storyid=133870&provider=rss)

"It's just a hard situation when you have 100,000 people that we have space for in the seating areas, but not necessarily in all the concourses…," Elliott said.

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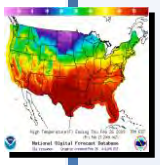

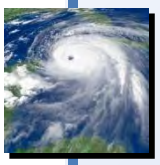

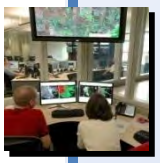

Lightning Delays at Football Games

August-December 2010

*Data gathered via media articles, some events may not be documented.

Lightning Delays at Football Games

August-December 2011

*Data gathered via media articles, some events may not be documented.

Voluntary Recognition Program

- **Motivate for venues to pursue a lightning safety plan**
  - *BE PROACTIVE*

- **Utilizing the Toolkit**

- **Safety Plan Submission**
  - *Local WCMs*

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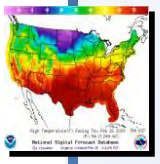

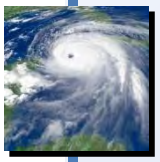

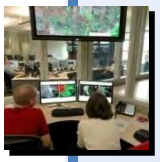

Eligible Venues

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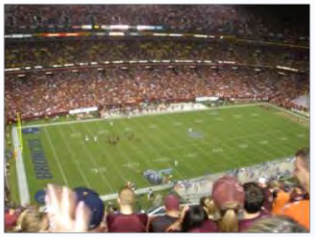

Outdoor Stadiums

Raceways

Amusement and Water Parks

Golf Courses

Concert Venues

Little League Facilities

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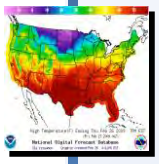

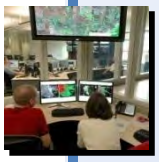

Keys to Preparing a Venue

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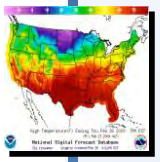
Information Reception

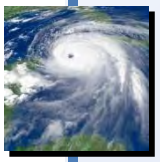
Decision Support Standards

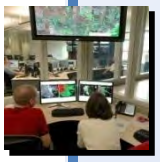
Public Notification Plan

Protection Program

Education

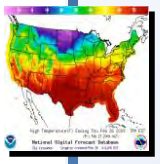


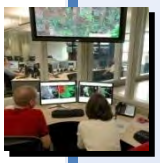




Toolkit

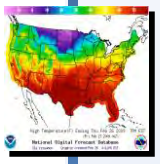
- **Before the Event**
  - *Weather watcher designated*
  - *Situational Awareness*
- **During the Event**
  - *Weather Monitoring*
  - *Decision Points – Lightning Radii*
- **After the Event**
  - *Review of Operations*

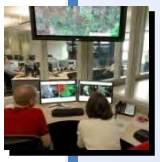




During the Event Activity

- *Weather Monitoring*
  - **Internet, TV, weather radio, lightning detection service**
- *Educate Spectators*
  - **Potential for a delay, what to do…**
  - **Develop different weather-related announcements**
  - **Include information in programs**
  - **Inform spectators on re-entry procedures**
- *Decision Points – Lightning Radii*





Lightning Radii

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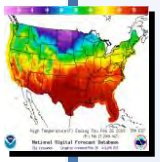

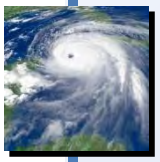

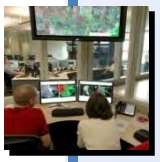

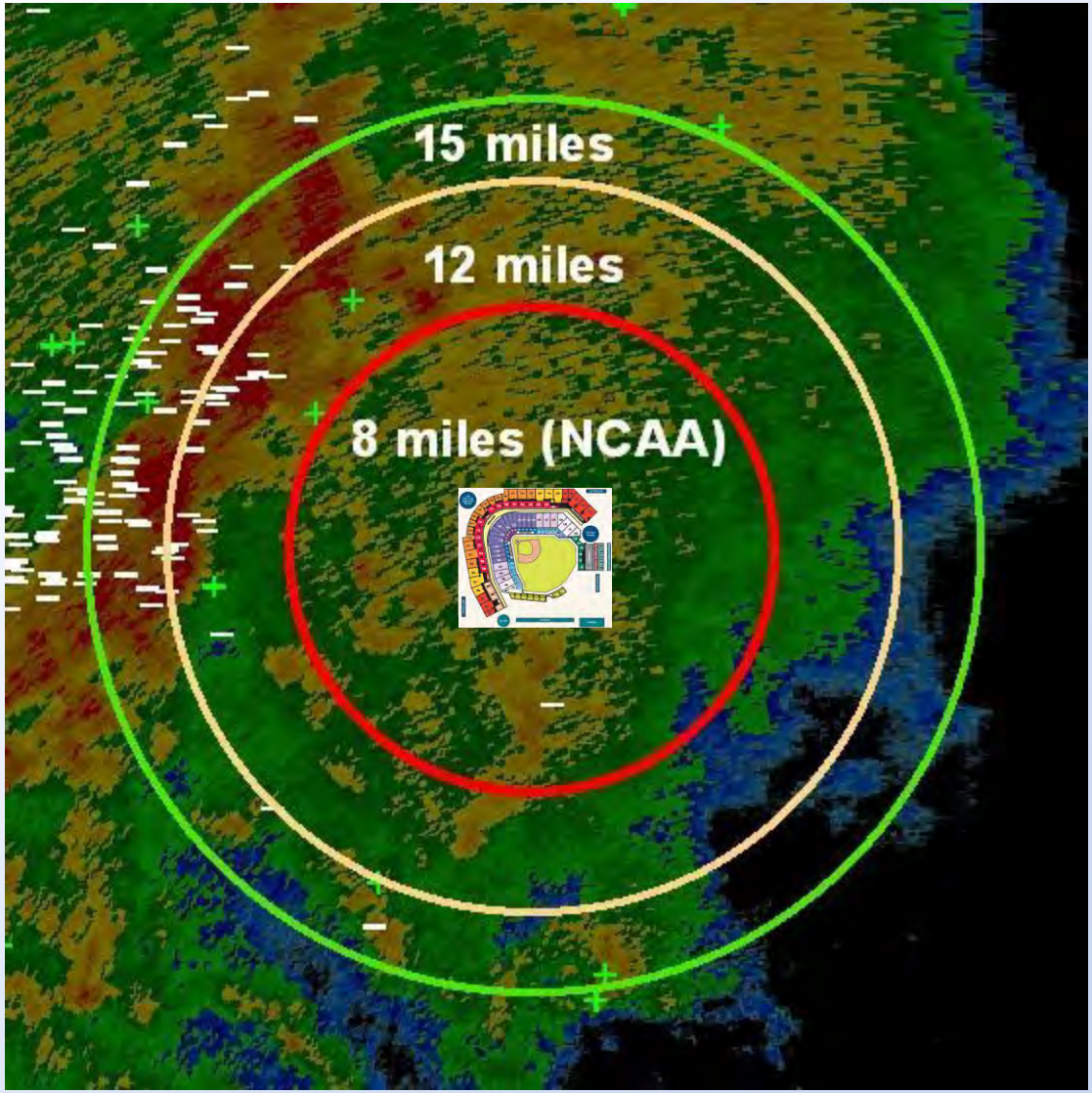

Recommended radii for lightning delay decisions

Lightning Observation Guidelines

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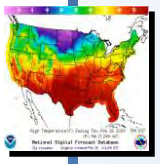

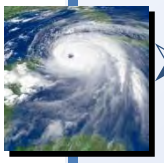

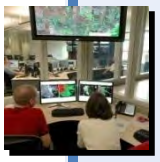

- **Observations-** The best course of action is to practice *"When Thunder Roars, Go Indoors!"*

- ***The Flash/Bang method-*** *To use this, the observer begins counting at the sight of a lightning flash. Counting is stopped at the sound of related thunder. The count is then divided by 5 to determine the distance (miles) that the lightning strike is from the venue. Large venues with long evacuation times should consider a longer count.*

- ***30-30 Rule-*** *Once lightning has been observed within a defined radius of the venue (30 seconds= 6 miles using flash/bang rule), officials should not resume play until 30 minutes following the final observed or detected strike.*

Evacuation Plans

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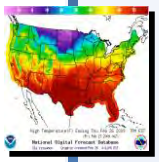


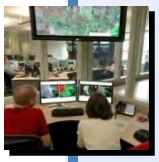
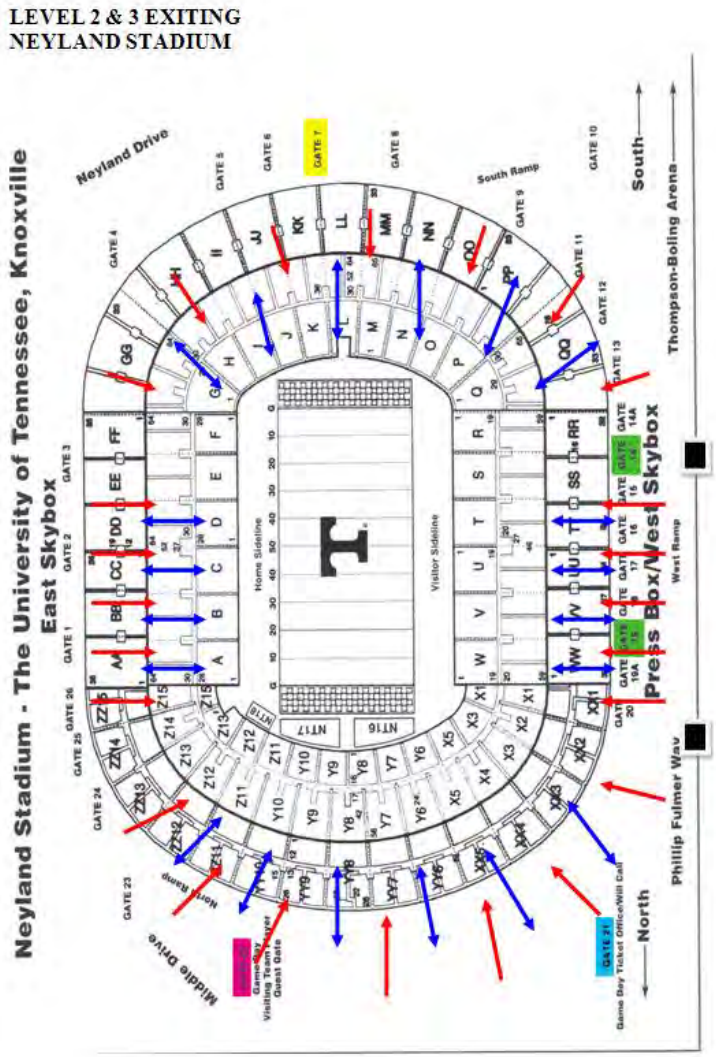
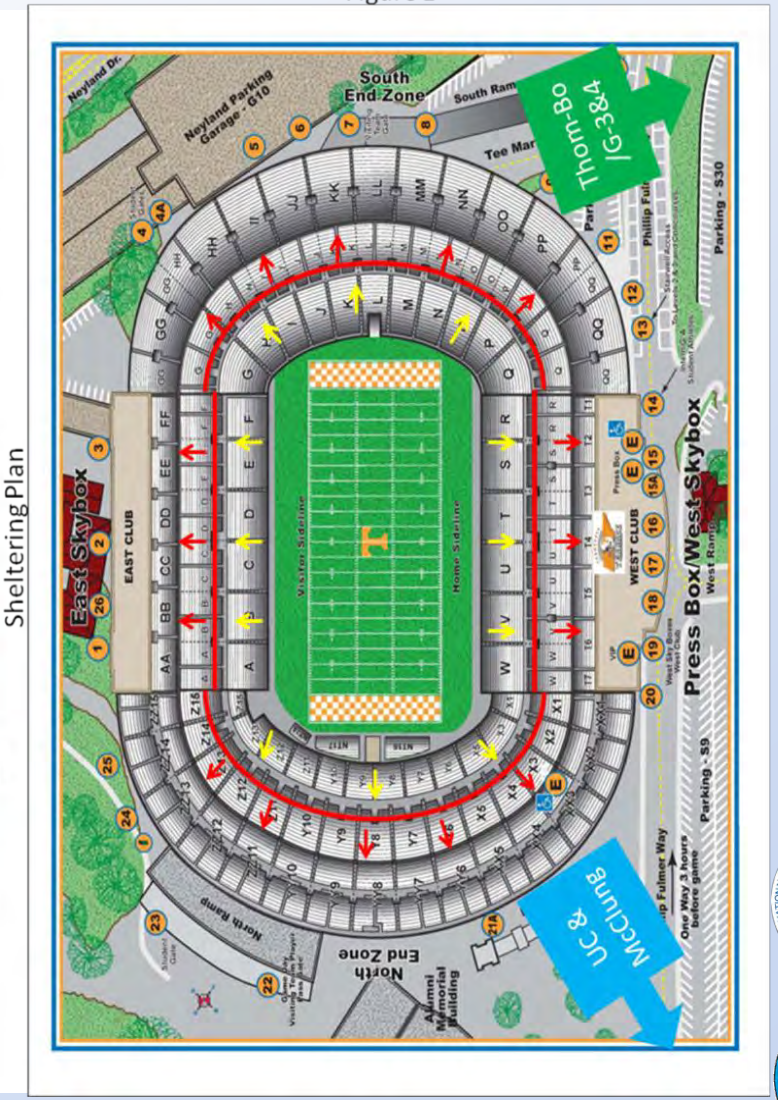


Figure 1



Sheltering Plan



Neyland Stadium evacuation routes (Graphics courtesy of Brian Gard)

Safe Structures

- Large substantial building
- Where people frequently live or work
- Have grounded wiring and plumbing, and metal structural members
- Surrounds people inside with a Faraday cage-like effect

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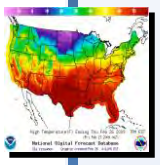

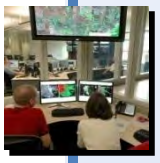

Sometimes Safe Structures

- Unsafe from Lightning - has the word ‘shelter’ attached – sun, beach, golf, picnic, or rain shelter

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Completely Unsafe Structures

- **Dugouts**
  - *Open side*
  - *Small*
  - *No conducting material around people inside*
  - *Nearby tall utility pole*
  - *Water on ground*

- **Tents**
  - *Invisible to lightning*

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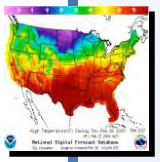
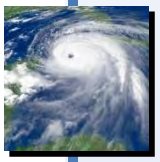
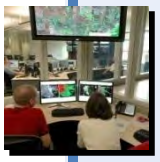
Safe/Unsafe Vehicles

National Weather Service

Protecting Lives and Property







Safe

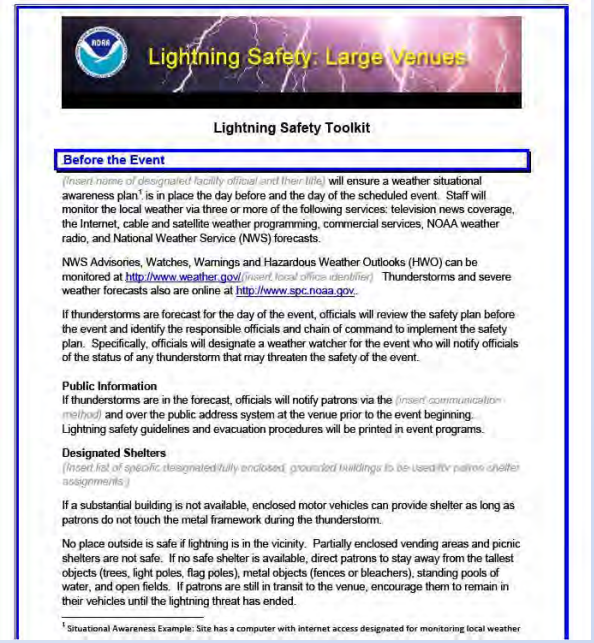
- Fully enclosed metal-topped vehicle of any type

Unsafe

- Convertible
- Fiberglass body
- Cloth top

Accessing the Toolkit

- **www.lightningsafety.noaa.gov**
  - **Third bullet down (Toolkit for Large Venues)**
- *PDF document*
- *Word template*
- *Fill in the Word template and submit it to your local National Weather Service office for review*



Lightning Safety: Large Venues

Lightning Safety Toolkit

Before the Event

(Insert name of designated facility official and their title) will ensure a weather situational awareness plan[1] is in place the day before and the day of the scheduled event. Staff will monitor the local weather via three or more of the following services: television news coverage, the Internet, cable and satellite weather programming, commercial services, NOAA weather radio, and National Weather Service (NWS) forecasts.

NWS Advisories, Watches, Warnings and Hazardous Weather Outlooks (HWO) can be monitored at http://www.weather.gov/(insert local office identifier). Thunderstorms and severe weather forecasts also are online at http://www.spc.noaa.gov.

If thunderstorms are forecast for the day of the event, officials will review the safety plan before the event and identify the responsible officials and chain of command to implement the safety plan. Specifically, officials will designate a weather watcher for the event who will notify officials of the status of any thunderstorm that may threaten the safety of the event.

Public Information

If thunderstorms are in the forecast, officials will notify patrons via the (insert communication method) and over the public address system at the venue prior to the event beginning. Lightning safety guidelines and evacuation procedures will be printed in event programs.

Designated Shelters

(Insert list of specific designated/fully enclosed, grounded buildings to be used/fill out shelter assignments.)

If a substantial building is not available, enclosed motor vehicles can provide shelter as long as patrons do not touch the metal framework during the thunderstorm.

No place outside is safe if lightning is in the vicinity. Partially enclosed vending areas and picnic shelters are not safe. If no safe shelter is available, direct patrons to stay away from the tallest objects (trees, light poles, flag poles), metal objects (fences or bleachers), standing pools of water, and open fields. If patrons are still in transit to the venue, encourage them to remain in their vehicles until the lightning threat has ended.

[1] Situational Awareness Example: Site has a computer with internet access designated for monitoring local weather

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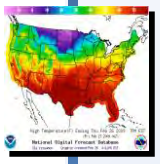
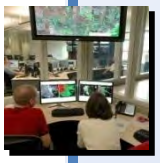
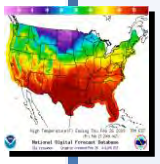


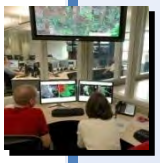


Preparation Checklists

National Weather Service

Protecting Lives and Property





Venue Preparation Checklists

1. **Data Reception Sources-** The venue should investigate the installation of a locally run lightning protection system with a display unit on site or subscribe to a commercial notification system. In addition, the facility should have a NOAA weather radio on location.

☐ NOAA Weather Radio	☐
☐ NOAA Weather Wire (subscription)	☐
☐ Scanner	☐
☐ Amateur Radio	☐
☐ Pagers (warning reception)	☐
☐ Television (Local network or Cable TV)	☐
☐ Radio Station (AM/FM) - EAS Reception	☐
☐ Internet (subscription for alerts)_____	☐
☐ Commercial Data Service_____	☐
☐ Lightning Detection System_____	☐
☐ Other-_____	☐

Recognition Ceremonies

- **Toolkit Review**
- **Safety Plan Submission**
  - *Local WCMs*
  - *With or without StormReady® recognition*
- **Approval of Plan**
- **Media Event**

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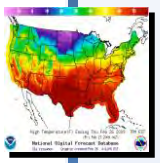

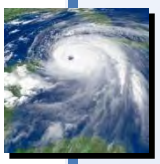

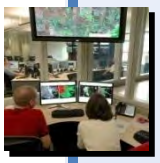

Georgia Tech First to Complete Toolkit

National Weather Service

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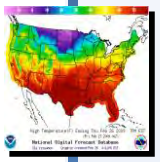

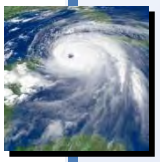

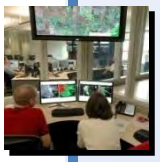

Officials pose with the “When Thunder Roars, Go Indoors” signs at Bobby Dodd Stadium in Atlanta, GA.
(Photo courtesy of Georgia Tech.)

Proactive Examples

University of Texas

West Virginia University

- *"Weather Watcher"*
- *NWSChat*
- *Briefings from 911 Center*
  - **Law enforcement , ushers, and EMS**
- *Authority to call for "shelter in place"*
- *Weather Log*

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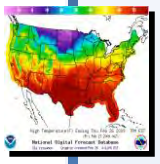
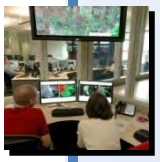




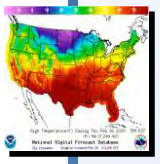
Marshall @ West Virginia

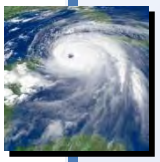
September 4, 2011 – 4 hours, 22 minutes

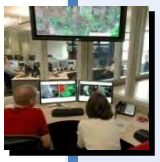
West Virginia players head to the locker rooms.
(Photo courtesy of the Charleston Gazette)

National Weather Service

Protecting Lives and Property







Marshall @ West Virginia

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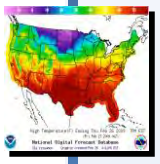
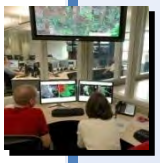
A weather message on the video board at Milan Puskar Stadium during the 3rd quarter delay.
(Photo courtesy of the Charleston Gazette)

National Weather Service

Protecting Lives and Property





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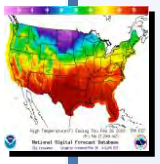
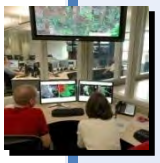
Fans take shelter in the adjacent practice facility.
(Photo courtesy of April Kaull)

National Weather Service

Protecting Lives and Property





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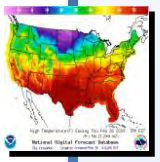

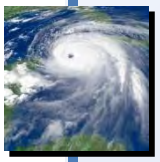

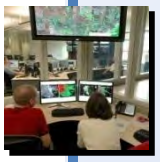

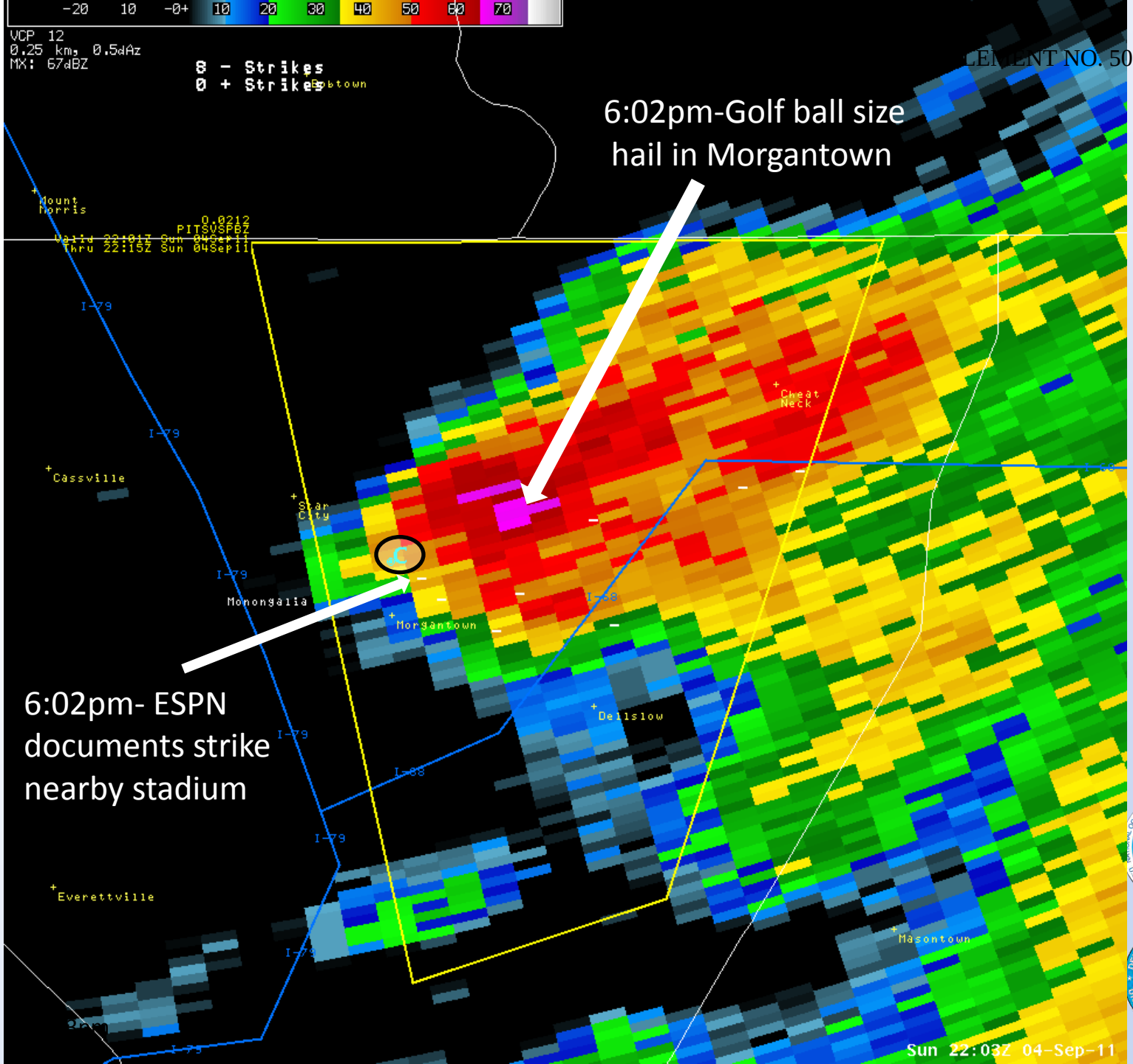

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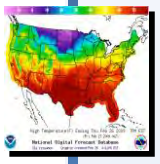
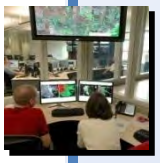
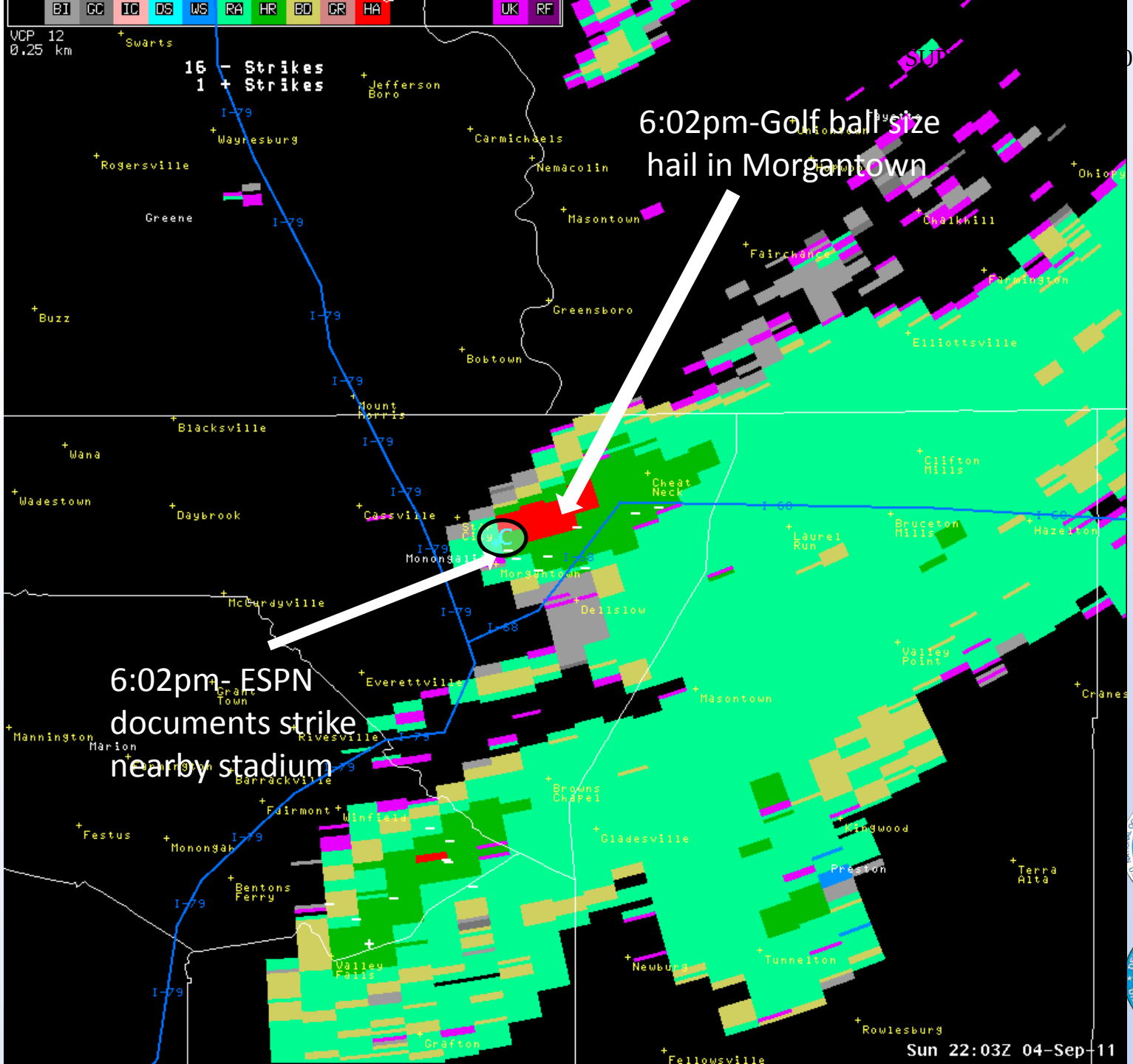






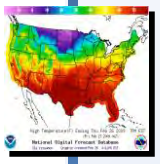
Marshall @ West Virginia

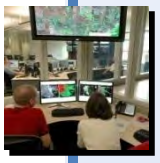
September 4, 2011 – 4 hours, 22 minutes

Lightning strikes behind Milan Puskar Stadium.
(Permission to use photos granted by ESPN)

National Weather Service

Protecting Lives and Property





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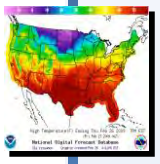

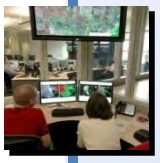

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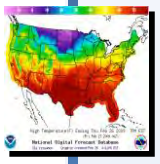
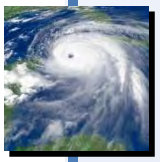
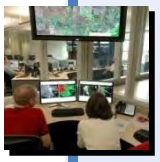




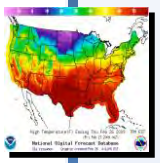


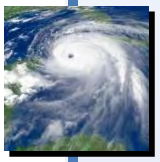
Summary

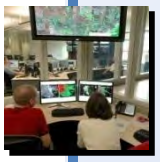
- **This program is just taking off…**
  - *Georgia Tech University*
    - First to complete the recognition for Bobby Dodd Stadium
  - *University of Tennessee and Florida State University*
    - Currently working toward submitting toolkits
  - *National Collegiate Athletic Association*

More outreach needs to be done…

National Weather Service
Protecting Lives and Property

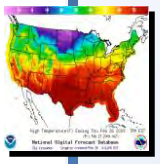


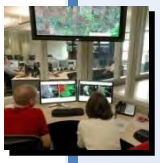




Expansion Plan

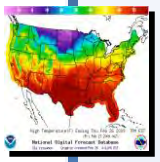
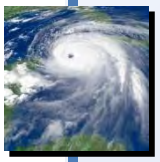
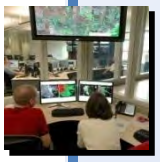
- **Initial Phase**
  - *Large Outdoor Venues – NCAA*
  - *Professional Stadiums*
- **Second Phase**
  - *Fairgrounds*
  - *Amusement Parks, Waterparks*
  - *Golf Courses, Concert Venues*
- **Third Phase**
  - *High School Facilities*
  - *Little League Organizations*





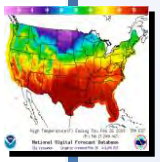
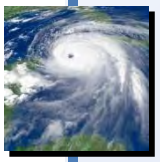
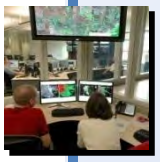
What can be done now?

- **Fair and Venue Managers**
  - *Pinpoint your vulnerable venues and events*
  - *Work with your local NWS office to develop plans*
  - *Complete Lightning Safety Toolkit*
  - *Remain flexible with the toolkit*
    - *Make edits and customize*
  - *Share plans completed with us*
  - *Request signs → Recognition Ceremony*

Special Thanks

- **Lightning Safety Team**
  - *Donna Franklin- OCCWS, Silver Spring, MD*
  - *Ron Holle- Vaisala, Tucson, AZ*
  - *Mary Ann Cooper- University of Illinois-Chicago*
  - *Melody Magnus- OCCWS, Silver Spring, MD*
- **Outdoor Venue Team**
  - *Mary Fairbanks- OCCWS, Silver Spring, MD*
  - *Tim Troutman- WFO Morristown, TN*
  - *Jeff Evans – WFO Tallahassee, FL*

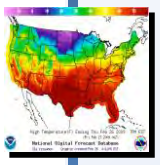




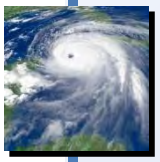
Thank You!

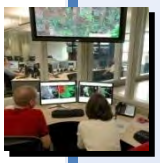
SUPPLEMENT NO. 50

National Weather Service

Protecting Lives and Property







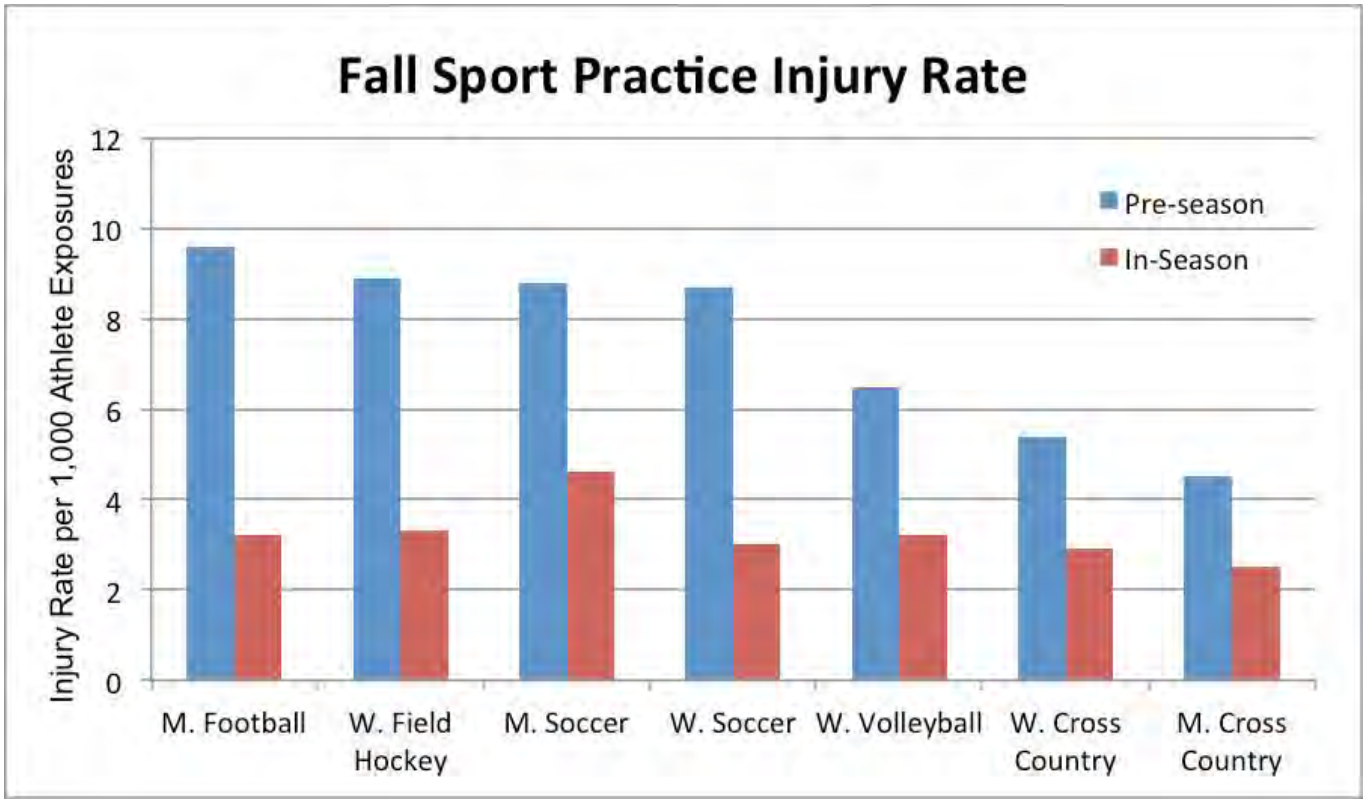
Email: Charles.Woodrum@noaa.gov
Donna.Franklin@noaa.gov

Visit our web site:
http://www.lightningsafety.noaa.gov

Best practices developed to address heat illness in fall sports

Apr 20, 2012 11:54:38 AM

By Brian Hendrickson
NCAA.org



As the NCAA's football programs prepare for the fall season, all will follow a strict road map to guide them past the potential hazards that can accompany premature exposures to intense exercise under the hot summer sun.

NCAA policies lay down specific regulations for acclimatizing football players to the rigors of preseason conditioning. This includes regulating the number of on-field practices each day, the length of those sessions, at which points of the preseason certain pieces of equipment can be worn, and even the frequency of multiple-practice days. The policies are designed to protect the student-athlete's

health and avoid exposure to a rapid acceleration of activity before their body's physical conditioning is ready to handle the load. The risk of sudden death is highest when athletes begin any new practice period, especially within the first few days. And sport injuries can be three times higher during the preseason than in the regular-season practice periods. Conditioning sessions are also a higher risk activity than the normal sport drills and skills sessions.

Other fall sports face similar challenges when starting up following the summer period, though they do not have specific practice policies to guide them. They must design appropriate practice plans that address heat, hydration, conditioning and rest periods that balance the needs for protecting the health of their student-athletes while at the same time preparing them for competition.

As institutions search for the best approaches, the NCAA Committee on Competitive Safeguards and Medical Aspects of Sports developed these practices that should be followed to develop an effective preseason conditioning strategy:

- **Develop a preseason practice plan:** Athletes cannot be conditioned into shape over a two-week period. It takes much more time to reach competitive-level condition. Coaches and student-athletes should anticipate a period of six to eight weeks for preseason preparation, during which the type, frequency and intensity of exercises is progressively increased and ample recovery time is provided. As part of this preparation, student-athletes should be encouraged to prepare for preseason activities independently with a progressive strength and conditioning plan at least four weeks before the start of the preseason period. This will prepare their bodies for the increased activity in practices, which coaches often conduct at contest-level intensity.
- **Start slow:** During the first three days of preseason workouts, practices should be limited to one session per day that lasts no more than two hours. This will give student-athletes enough time to steadily build up their endurance without the risks of overtraining. Freshmen, transfers and walk-on student-athletes may need additional time to acclimate to the rigors of collegiate sports, so extending this period to six or seven days would be appropriate to consider. This can also apply to any athlete returning from an injury or illness and those beginning after a school break period.
- **Limit daily practice sessions:** After the initial acclimatization period, limit practice sessions to no more than two per day and avoid scheduling multiple practice sessions on consecutive days. Instead, schedule multiple practice sessions every other day, with a single workout in between, until the academic year begins. Each practice should last no more than two hours.
- **Plan for a heat acclimatization period:** Student-athletes should gradually increase exposure to hot, humid summer weather for a minimum of 10 to 14 days at the beginning of preseason activities. Similar to the preseason conditioning plan, activity during each exposure to heat should gradually increase in intensity and duration until the exercise is comparable to the exposure likely to be experienced in competition. During periods of intense heat, practice times should be reduced and scheduled during cooler times of the day.
- **Phase in exertional activity.** Coaches should consider allowing student-athletes to become accustomed to heat and exercise intensity before conducting highly exertional activities such as timed runs, max lifting sessions, repeat sprints or competitive running. An example of a progressive training plan is to start at 50 percent of total workload and intensity, and then move up incrementally over a set number of days. Workout sessions should not be used for punishment or disciplining behavior,

academic or athletic performance.

- **Take time off between practices**: On days when multiple practice sessions are held, provide at least three hours of continuous recovery time between the end of the first practice and the start of the next. During that time, student-athletes should not attend meetings or engage in physical activities related to their sport. However, time spent receiving medical treatment or eating meals can be included as part of the recovery period.
- **Walk-through after a break**: Walk-through sessions can be used as a teaching opportunity during the preseason to supplement the physical practice sessions, but just like multiple-practice days, student-athletes should also be given a rest period of at least three hours between practice and the walk-through. During the walk-through, student-athletes should not use equipment related to the sport or perform conditioning exercises.
- **Take a day off:** Student-athletes should not participate in practice sessions for more than six consecutive days. This includes any organized physical activity related to the sport, such as warm-ups, stretching, scrimmaging, weight lifting, fitness testing, conditioning, yoga, pilates, cool downs, non-medically related rehabilitation or captain's practices.
- **Communicate:** Encourage coaches to get involved with medical staff in the development of a conditioning plan. All personnel should be aware of the potential impact acclimatization, hydration, medications and drugs, existing medical conditions, nutritional supplements, clothing and equipment have on student-athletes' health while participating in strenuous workouts. When determining if a student-athlete is ready to return to competitive activities following an injury or illness, there should be a joint evaluation between the sports medicine staff and the strength and conditioning staff when making a judgment on the best return-to-play model for each individual.
- **Have an emergency plan on file:** On-field personnel should review, practice and follow their venue emergency plan and be trained in administering first aid, cardiopulmonary resuscitation and automated external defibrillator use.

http://www.ncaa.org/wps/myportal/ncaahome?WCM_GLOBAL_CONTEXT=/ncaa/ncaa/ncaa+news/ncaa+news+online/2012/association-wide/best+practices+developed+to+address+heat+illness+in+fall+sports

DRAFT Concepts

Southern Conference

Acclimatization Policy, Summer and Off Season

Freshmen/Transfer

- Acclimatization 5-7 days
- Heat, Exercise, Work/Rest ratio
- Highly motivated activity is not allowed for 5 days
    - 16-110's
    - Timed run
    - Competitive running
    - No Max lifting
- During this time, athletes may not participate in more than 1 conditioning session per day.
- Factors to also consider
    - Previous Heat Illnesses
    - Fluid Accessibility
    - Clothing Color
    - Ambient Temperature
    - Weight Room Activity

<u>Protocol</u>

Day 1

- 50% of max lifting only allowed
- Exercise in the heat will last for no more than 30 total minutes in the heat
    - 5 minutes of rest per 10 minutes of exercise

Day 2

- 50% of max lifting only allowed
- Exercise in the heat will last for no more than 30 total minutes in the heat
    - 5 minutes of rest per 10 minutes of exercise

Day 3

- 50% of max lifting only allowed

- Exercise in the heat will last for no more than 45 total minutes in the heat
    - 5 minutes of rest per 10 minutes of exercise

Day 4

- 75% of max lifting only allowed
- Exercise in the heat will last for no more than 45 total minutes in the heat
    - 5 minutes of rest per 10 minutes of exercise

Day 5

- 75% of max lifting only allowed
- Exercise in the heat will last for no more than 60 total minutes in the heat
    - 5 minutes of rest per 10 minutes of exercise

Day 6

- Lifting as tolerated
- Exercise in the heat will last for no more than 60 total minutes in the heat
    - 5 minutes of rest per 10 minutes of exercise

SUPPLEMENT NO. 53

Recommended Practices for the Maintenance of Infill Surfaces for NFL Games

August 5, 2011

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Introduction

This document describes four recommended practices (RPs) for the maintenance of *infill* turf surfaces used for NFL games. The RPs are intended solely for *infill* turf surfaces used in the NFL and may not be applicable to *infill* turf surfaces used at other levels of play or for other sports. The RPs are not applicable to natural grass surfaces. The RPs are not an exhaustive description of the *infill* turf maintenance practices that are or should be used by individual clubs. Nor are the RPs intended to replace any relevant industry standards or the applicable manufacturer recommendations for care and maintenance of a particular *infill* surface.[1]

As set forth in detail below, RPs are provided for (1) impact hardness, (2) *infill* depth and evenness, (3) visual inspection of the field surface, and (4) the use of anti-bacterial agents. Supporting documents and references for each RP are attached at Appendices A though E.

Documentation of compliance with the RPs shall be completed by the home club prior to any pre-season, regular season, or post-season NFL game. The timing and frequency of compliance with the RPs and the instructions for compliance certification are specified in each RP. A compliance certification form is provided in Appendix D. All compliance documentation should be maintained by each club and will be subject to inspection by the League Office at any time.

If you have any questions about the RPs, please contact

Michael Kensil
212-450-2084 (office)
917-257-1152 (mobile)
mike.kensil@nfl.com

[1] The RPs were developed by Richard Kent, PhD, University of Virginia, Andrew McNitt, PhD, Penn State University, Barry Myers, MD, PhD, Duke University, Don Follet, Baltimore Ravens, and James Daniel, National Football League and are based on relevant industry standards, including those listed in Appendix E. The authors and the NFL acknowledge and are grateful for the assistance of the Synthetic Turf Council, FIFA, ISA, IRB, A-Turf, AstroTurf, FieldTurf, Shaw Sportexe, UBU Sports, Joel Forman, MD, Mt. Sinai School of Medicine, Daryl Johnston, and Donald McPherson, who contributed to the development of the RPs.

RP 1: Impact Hardness

Recommendation

The game-day field should produce a g-max of less than 100 g (as measured by the device specified below) in all locations tested according to the methods described below. Any field locations that fail the impact hardness test must be remediated in accordance with the applicable turf manufacturer's recommendations and all such locations should be retested and must pass the impact hardness test prior to the commencement of game-day play.

Testing Device and Methods

Device

The device to be used for impact hardness testing is the Clegg impact tester using the 2.25 kg missile (See ASTM F1702 Sections 1 through 6.). The device is shown below. A Clegg impact tester will be provided to you by your club prior to the commencement of the season.

Figure 1. Clegg impact tester (Clegg hammer).

The procedure for using the device is as follows:

Set up the Clegg impact tester and prepare it for use in accordance with its instructions.

After ensuring that each test location point (as specified below) is free of debris, position the test apparatus and conduct the drop test in accordance with the device instructions.

Record the g-max measurement using the form provided in Appendix A.

Record basic data related to the test site and environmental conditions using the form provided in Appendix A.

Field Test Locations

The following locations on the field should be tested (see ASTM F1936):

Point 1—Goal Line, End A, center of field;
Point 2—10 Yard Line, End A, 63 ft from center of field to Side C;
Point 3—25 Yard Line, End A, 40 ft from center of field to Side C;
Point 4—Center of the field;
Point 5—25 Yard Line, End B, 63 ft from center of field to Side D;
Point 6—12 Yard Line, End B, center of field;
Point 7—A test point selected by the tester, with the objective of identifying and testing a high-wear area located outside the in-bounds lines;
Point 8—A test point selected by the tester, with the objective of identifying and testing an area (including the in-bounds area) that may have different impact attenuation performance (for example, a location that appears to be harder) than points previously tested;
Point 9—6 ft from the Goal Line to the back of the End Zone, End A, center of field;
Point 10—6 ft from the back of the End Zone to the Goal Line, End B, center of field.

Actual drop sites may be located anywhere within a 36 in. (91 cm) radius of a described test point location. An illustration of the above test locations is shown below in Figure 2 (see ASTM F1936, Section 8.1 and 8.2). Test points 7 and 8 are shown for information purposes only. Actual locations are selected at the time of the test and may vary from those illustrated.

In addition to the above-specified test locations, all areas of noticeable damage, previous repair, or non-standard fill should be tested. These include but are not limited to *inlays*, areas of paint build up, seams, recent heavy load bearing (for example, high-traffic locations at recent events such as concerts), and areas that previously failed the impact location test during the current season.

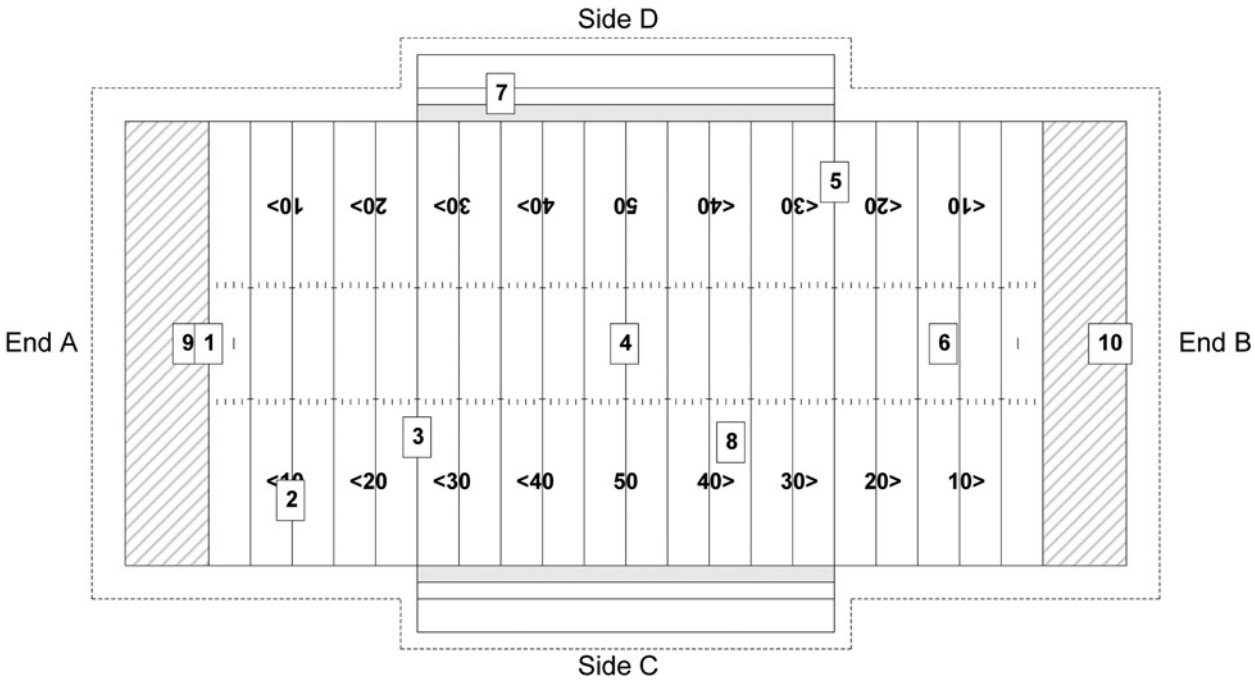

Image from ASTM F1936.

Figure 2. Field test locations.

Timing and Frequency

All fields should be certified as having passed the impact hardness test within 72 hours preceding the commencement of every game. The required certification form is attached as Appendix D. Initial testing should be conducted sufficiently in advance of game day to allow for remediation prior to commencement of play.

If the field is hosting an event that imposes significant traffic or surface loads (for example, a concert), then impact certification should occur after the event and prior to the NFL game.

RP 2: Infill Depth and Evenness

Recommendation

The depth and evenness of *infill* turf surfaces should be maintained in accordance with the below specifications of the applicable manufacturer.

Each manufacturer of *infill* turf surfaces used for NFL games has specified three characteristics of the *infill* depth for its surface:

1. The target depth of the *infill*, ***D***,
2. An allowable deviation from the target depth of the *infill*, ***d***,
3. An evenness target in terms of a *coefficient of variation*, ***C***, of the *infill* measurements specified in this RP.

The manufacturer's specifications should be attained and recorded in Appendix B.

Testing Device and Methods

Device and Measurement Method

Infill depth should be measured using the Floortest FT 50 calibrated thickness gage shown below in Figure 3. This device will be provided to you by your club prior to the commencement of the season.

The thickness measuring probes should be inserted into the *infill* until they contact the ground or hard surface beneath the turf backing. The sliding barrel portion of the gauge should then be adjusted until it is in contact with the top surface of the *infill*. The thickness measurement can then be read off of the calibrated barrel. Care should be taken to ensure that the device sits on a level under-surface (without cracks, defects, or depressions) so that both the barrel and the probes make accurate measurements.

Floortest FT 50

Figure 3. Calibrated thickness gages.

Field Measurement Locations and Compliance Procedure

Infill depth should be measured at the 10 locations specified above in **RP 1: Impact Hardness** as illustrated in Figure 2 above. In addition, 10 randomly selected locations on *inlays* and 20 locations randomly distributed over the entire *playing field* and at least 5 yards from any other measurement location should be measured.

For each of the 40 locations specified above, *infill* depth should be measured once and recorded along with the other requested information on the form provided in Appendix B.

The Excel spreadsheet [infill depth and evenness calcs.xlsx] included as an electronic file attachment to the RPs should be used to determine whether the above measurements demonstrate compliance with the applicable *infill* turf manufacturer's specifications for *infill* depth and evenness. To do so, enter into the yellow boxes in the electronic spreadsheet the applicable turf manufacturer's specifications identified above and the 40 depth measurements. If the recorded measurements demonstrate compliance, a green box with the word "COMPLIANCE" will appear. If the recorded measurements demonstrate non-compliance, a red box with the word "NON-COMPLIANCE" will appear.

In the event of non-compliance, *infill* should be added, removed, or smoothed (depending upon the measurement readings) in accordance with the applicable turf manufacturer's recommendations. The above-specified procedures should be repeated until the recorded measurements demonstrate compliance.

Timing and Frequency

All fields should be certified as in compliance with the applicable manufacturer's specifications before every game. Certification should be made no earlier than seven days before game day. Initial testing should be conducted sufficiently in advance of game day to allow for remediation prior to commencement of play. If the field is hosting an event that imposes significant traffic or surface loads (*e.g.,* a concert), then *infill* depth and evenness certification should occur after the event and prior to the NFL game.

RP 3: Visual Inspection

Recommendation

Prior to game time, the field surface should be free of any defects apparent through the visual inspection methods set forth below.

Methods

Prior to game time, the field manager should:

1. Visually inspect the *infill* turf in all locations where paint has been applied. If paint buildup is excessive, old paint should be removed and new paint should be applied. Follow the applicable turf manufacturer's recommendations for the removal of paint. Excessive paint buildup may be indicated by significantly increased g-max values or paint layering to the extent that it affects cleat penetration and traction. It may also be indicated by a change in *infill* depth measurements.
2. Visually inspect all seams and *inlays* to make sure that no sections of an *inlay* have become separated from surrounding turf and that seams have not separated wider than 3 mm. Any *inlay* or seam in question during a visual inspection should undergo additional examination by the field manager. To do so, remove sufficient *infill* to inspect the condition of the seam at the backing. If, at the backing, the seam has become loose and has separated more than 3 mm, the seam should be remediated in accordance with the applicable manufacturer's recommendations.
3. Visually inspect all special turf sections (*e.g.,* end-zone inserts) to make sure that they fit well with adjacent turf areas, leaving no seam separations wider than 3 mm. Use the procedures set forth in number 2 above. If seams have separated more than 3 mm, the seams should be remediated in accordance with the applicable manufacturer's recommendations.
4. Visually inspect turf sections inserted to cover baseball areas such as mound, plate area, and bases, to make sure that they fit well with adjacent turf areas, leaving no seam separations wider than 3 mm. Use the procedures set forth in number 2 above. If seams have separated more than 3 mm, the seams should be remediated in accordance with the applicable manufacturer's recommendations.
5. Visually inspect the *infill* turf for any other problems, such as wrinkles or burns. All such problem should be remediated in accordance with the applicable manufacturer's recommendations.
6. Visually inspect the field to make sure that the turf is swept, that surface debris and other foreign objects are removed from the field, and that no dirt or debris remains among the *synthetic grass fibers*.
7. Visually inspect the field to make sure that the surface is free of pooled water, ice, or snow.

Timing and Frequency

All fields should be certified as in compliance with the above recommendations on game day and prior to the time when the players come on to the field. Initial inspections should be performed sufficiently in advance of game time to allow for remediation and reinspection. Visual inspection to ensure that the playing field is free of surface debris and other foreign objects should be done after all in-game events, including pre-game and half-time.

RP 4: Anti-Bacterial Agents

Recommendation

Clubs should not use antimicrobial agents on *infill* turf surfaces. The medical and scientific communities have not documented any benefit of using such agents on *infill* turf surfaces and there may be potential disbenefit to doing so.

The Centers for Disease Control and Prevention (CDC) does not recognize *infill* turf as a significant source of MRSA infections. [See Appendix E] As the CDC has stated "[t]here is a lack of evidence that large-scale use (*e.g.,* spraying or fogging rooms or surfaces) of disinfectants will prevent MRSA infections more effectively than a more targeted approach of cleaning frequently-touched surfaces." [See Appendix E] This RP should not be interpreted as preventing the use of cleansing agents such as detergents and soaps to clean *infill* turf surfaces provided that such cleaning is performed in accordance with the recommendations of the applicable turf and cleaning agent manufacturers.

Appendix A: Documentation form for RP 1: Impact Hardness

Date and Time of Testing:	Name of Tester:
Field Name/Location:	Ambient Air Temperature:
General Weather Conditions:	Identify End A (per ASTM 1936 9.4.2): N NE E SE S SW W NW

Test location	Description of location	g-max	Comments (e.g., abnormalities, unexpected results)
ASTM 1936, point 1	[redacted]		
ASTM 1936, point 2			
ASTM 1936, point 3			
ASTM 1936, point 4			
ASTM 1936, point 5			
ASTM 1936, point 6			
ASTM 1936, point 7			
ASTM 1936, point 8			
ASTM 1936, point 9			
ASTM 1936, point 10			
Other			
Other			
Other			
Other			
Other			
Other			
Other			
Other			
Other			
Other			
Other			
Other			
Other			
Other			
Other			

Narrative

(Describe any testing done prior to certification and any remediation taken, including the dates of testing and remediation).

Appendix B: Documentation form for RP 2: Infill Depth and Evenness

Target Infill Depth (mm) (Manufacturer's Specification):	
Allowable Deviation from Target Infill Depth (mm) (Manufacturer's Specification):	
Allowable Coefficient of Variation (Manufacturer's Specification):	

Date and Time of Measurement:	Measurements Taken By (Name):
Field Name/Location:	Ambient Air Temperature:
General Weather Conditions:	Identify End A (per ASTM 1936 9.4.2): N NE E SE S SW W NW

Test location	Description of location	Depth (mm)	Test location	Description of location	Depth (mm)
ASTM 1936, point 1			Random 1		
ASTM 1936, point 2			Random 2		
ASTM 1936, point 3			Random 3		
ASTM 1936, point 4			Random 4		
ASTM 1936, point 5			Random 5		
ASTM 1936, point 6			Random 6		
ASTM 1936, point 7			Random 7		
ASTM 1936, point 8			Random 8		
ASTM 1936, point 9			Random 9		
ASTM 1936, point 10			Random 10		
Inlay location 1			Random 11		
Inlay location 2			Random 12		
Inlay location 3			Random 13		
Inlay location 4			Random 14		
Inlay location 5			Random 15		
Inlay location 6			Random 16		
Inlay location 7			Random 17		
Inlay location 8			Random 18		
Inlay location 9			Random 19		
Inlay location 10			Random 20		

Narrative

(Describe any measurements taken prior to certification and any remediation taken, including the dates of measurement and remediation).

Appendix C: Documentation form for RP 3: Visual Inspection

Date and Time of Inspection:	Name of Inspector:
Field Name/Location:	Ambient Air Temperature:
General Weather Conditions:	

Inspection Item	Completed	Comments (e.g., abnormalities, unexpected results)
1. Uniformity, no defects		
2. Painted areas		
3. Seams and inlays		
4. Special turf sections		
5. Non-football turf sections		
6. Maintenance		
7. Water		

Narrative

(Describe any inspections done prior to certification and any remediation taken, including the dates of inspection and remediation).

Appendix D: Certification

I, _____(name) , confirm that

._____(field name)

meets the three recommended practices listed below.

Date: _____.

Initial and date to certify:

RP 1: Impact hardness: _____.

RP 2: Infill depth and evenness _____.

RP 3: Visual inspection _____.

Appendix E: References and Technical Details

Standards and Test Methods

ASTM International (2010) Standard Specification for Impact Attenuation of Turf Playing Systems as Measured in the Field. ASTM F1936-10. DOI: 10.1520/F1936-10.

ASTM International (2010) Standard Test Method for Measuring Impact Attenuation Characteristics of Natural Playing Surface Systems Using a Lightweight Portable Apparatus. ASTM F1702-10. DOI: 10.1520/F1702-10.

British Standard (2000) Surfaces for Sports Areas – Determination of Thickness of Synthetic Sports Surfaces. BS EN 1969:2000. ICS 97.150; 97.220.10, European Committee for Standardization, Brussels.

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Waninger et al. 2011. Community-associated methicillin-resistant Staphylococcus aureus survival on artificial turf substrates. Med Sci Sports Exerc. 43(5):779-84.

RP 2: Infill Depth and Evenness Technical Details

The Excel spreadsheet provided with this document makes all of the calculations required to determine whether the 40 measurements indicate compliance with the recommended practice. The inputs required from the user are the three specifications provided by the manufacturer (the target depth of the *infill*, $\boldsymbol{D}$, an allowable deviation from the target depth of the *infill*, $\boldsymbol{d}$, and an evenness target in terms of a *coefficient of variation*, $\boldsymbol{C}$) and the 40 measurements specified in the RP.

Five sets of calculations are performed by the spreadsheet. First, the average measured *infill* depth over the entire surface, μ, is determined as the mean of the 40 *infill* depths. Second, the standard deviation of the *infill* depth over the entire surface, σ, is determined as the standard deviation of the 40 *infill* depths. Third, the coefficient of variation of the *infill* depth is determined as $C_{meas} = \sigma/\mu$. Fourth, the *infill* depth is considered to be in compliance if $D - d \leq \mu \leq D + d$. Fifth, the *infill* evenness is considered to be in compliance if $C_{meas} \leq C$.

Terminology and Definitions

Base – The bottom layer of an *infill* artificial turf that serves as the mounting substrate (e.g., crushed stone and/or concrete).

Coefficient of variation – The ratio of the standard deviation to the mean.

Playing field – The entire surface of the *infill* artificial turf that a player may be expected to interact with during play, including both end zones and the sidelines as described "Plan of Playing Field" and "Playing Field Specifications" published in the Policy Manual For Member Clubs, Vol. II, Game Operations.

Infill – A granular substance simulating soil (e.g., sand and/or ground rubber) that surrounds the synthetic grass fibers in an *infill* artificial turf.

Infill artificial turf – A layered artificial turf surface composed of synthetic fibers attached to a textile or other surface laid on or adhered to a base (e.g., crushed stone and concrete) and surrounded by an *infill* mixture (e.g., sand and ground rubber) simulating soil.

Inlay – Any area where a portion of the synthetic turf surface has been removed and replaced with an separate piece of turf. These include but are not limited to hash marks, numbers, logos, and damaged areas where a patch has been installed.

Synthetic grass fibers – Plastic blades (e.g., polyethylene and polypropylene) simulating grass in an *infill* artificial turf.

STC Guidelines for Synthetic Turf Performance

Published December 2011

Background

- **Introduction:** The purpose of these voluntary guidelines is to enable owners, buyers and specifiers of multi-purpose sports fields to better understand the quality and performance of their system during design, selection, after installation and throughout its useful life.

- **Objectives:**
  - The STC and its members understand that performance and quality of a synthetic turf sports field are critical factors.
  - By establishing objective guidelines, the STC is providing an essential guidance to synthetic turf owners, buyers and specifiers at all levels which has been unavailable until now.
  - These voluntary STC Performance Guidelines help answer many user questions about the surfaces and industry. They provide:
    - More objective measurements when selecting a provider
    - An evaluation method to determine the quality of a sports field
    - A method for understanding the type of maintenance required for improving field performance and playability
    - Information as to when maintenance no longer can enable the desired performance – i.e. an indication that the field is nearing the end of its useful life, and will need to be replaced

STC Approach

- The STC spent months gathering information. It received input from its members' experiences and technical knowledge, ASTM standards, and input from two managers of FIFA.
- The STC determined that the ASTM standards provide useful information regarding product identification, turf fabric testing and *g*-max testing, but identified opportunities for greater attention to sport performance testing.
- The STC reviewed guidelines researched and established by international governing bodies.
  - **FIFA** - the world's governing body of soccer established the FIFA Quality Concept for Football Turf in 2001 – millions of dollars has been invested by FIFA in research, testing, medical and player feedback.
    - The FIFA Quality Concept is based upon natural grass benchmarks for sport performance plus a wide range of durability and quality assurance guidelines
    - Since 2001, the FIFA Quality Concept has been proven to provide user benefits:
      - F-MARC study of 10,000 injuries – no notable differences in type and number of injuries between "football turf" and natural turf
      - Player study on fatigue – no noticeable change in heart rates and blood lactate levels between "football turf" and natural turf
      - The benchmarks in both studies were "football turf" systems that would pass FIFA Quality guidelines for durability and performance

        - FIFA is supportive of the STC's use of the FIFA Quality Concept, provided the STC maintains the label of FIFA test methods and communicates the entire array of proper testing and quality assurance protocols.
    - **IRB** - The International Rugby Board (IRB) shares similar concerns with the North American market with high impact plus lower extremity issues and elected to combine high impact shock absorption (HIC) testing with FIFA testing.

- **Conclusion** – As a result of its analysis and consultation with industry experts, the STC published its Performance Guidelines to include the following components:
    - **Product identification** – to ensure the supplied system is the same as proposed
    - **Quality and Durability testing** – to reflect certain ASTM testing plus the FIFA UV and Lisport Testing
    - **Sport Performance testing** – to include *g*-max plus the FIFA performance testing
    - **Frequency of testing** – to provide clients with the information needed at certain intervals to make proper decisions.
    - **Future developments** – to allow for gains in synthetic turf system and testing technologies, feedback from users and industry stakeholders, follow-up evaluation and inclusion of maintenance guidelines. It is noted that proper maintenance is essential for the performance and quality of any synthetic turf system.

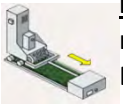
STC Guidelines for Synthetic Turf Performance

The Synthetic Turf Council hereby recommends the following guidelines as a desired range for multi-purpose sport surfaces. Soccer-specific users including clients that wish to obtain FIFA certification for their playing surfaces should refer to the FIFA Quality Concept (www.fifa.com/footballturf) for further information and guidelines.

Test Method(s)	Property	Community Field	Stadium Field
Identification Guidelines			
ASTM D1907	Fiber Denier	+/- 10% of Specification	+/- 10% of Specification
ASTM D3218	Fiber Microns	+/- 10% of Specification	+/- 10% of Specification
ASTM D5823	Pile Height	+/- 1/8" of Specification	+/- 1/8" of Specification
ASTM D5793	Stitch Gauge	Same as Specification	Same as Specification
ASTM D5848 or FIFA/ISO 8543	Pile Weight	+/- 10% and no more than minus 2 oz/sq. yd. of Specification	+/- 10% and no more than minus 2 oz/sq. yd. of Specification
ASTM D5848 or FIFA/ISO 8543	Primary Backing	+/- 10% of Specification	+/- 10% of Specification
ASTM D5848 or FIFA/ISO 8543	Secondary Backing	+/- 10% and no more than minus 2 oz/sq. yd. of Specification	+/- 10% and no more than minus 2 oz/sq. yd. of Specification
ASTM D5848 or FIFA/ISO 8543	Total Weight	+/- 10% of Specification	+/- 10% of Specification
FIFA/EN 1969	Infill Depth	+/- 15% of Specification	+/- 15% of Specification
FIFA/EN 13041	Infill Bulk Density	+/- 15% of Specification	+/- 15% of Specification
ASTM F2765-09	Lead Content	below 100ppm	below 100ppm
Quality Guidelines			
ASTM D1335 or ISO 4919	Tuft Bind (Without Infill)	> 6.8 lbs or 30N	> 6.8 lbs or 30N
FIFA 09/EN 13672	Simulated Wear/Abrasion	Shock Absorption, Vertical	Shock Absorption, Vertical
	Resistance	Deformation, Ball Rebound and	Deformation, Ball Rebound and
		Rotational Resistance - pass after	Rotational Resistance - pass after
		20,200 Lisport cycles	**5,200** Lisport cycles
FIFA/EN ISO 20105-A02	Artificial Weathering (3000 hrs UVA)	> Gray Scale 3	> Gray Scale 3
	Turf Color Change		
FIFA/EN 13864	Artificial Weathering (3000 hrs UVA)	Reduction of no more than 50%	Reduction of no more than 50%
	Pile Yarn Tensile Strength		
FIFA/EN ISO 20105-A02	Artificial Weathering (3000 hrs UVA)	> Gray Scale 3	> Gray Scale 3
	Infill Color Change		
FIFA/EN 13036	Surface regularity	< 10mm difference over 3m	< 10mm difference over 3m
		straight edge	straight edge
Surveyor's level	Slope	< 1%	< 1%
ASTM F1551 or FIFA/EN 12616	Water Permeability (after install)	> 10 inches per hour	> 10 inches per hour
Performance Guidelines			
ASTM F1936	Impact Attenuation (*g*-max)	below 165	below 165
FIFA 04 and FIFA 09	Shock Absorption	55% to 70%	60% to 70%
FIFA 05 and FIFA 09	Vertical Deformation (foot stability)	4mm to 9mm	4mm to 8mm
FIFA 06 and FIFA 09	Rotational Resistance (traction)	25n to 50n	30n to 45n
FIFA 07	Linear Friction - Deceleration	3.0g to 6.0g	3.0g to 5.5g
FIFA 07	Linear Friction - Slide	120 to 220	130 to 210
FIFA 08	Skin Abrasion (dry)	< 30%	< 30%
FIFA 08	Skin/Surface Friction (dry)	0.35 to 0.75	0.35 to 0.75
FIFA 01 and FIFA 09	Vertical Ball Rebound (Soccer)	60cm to 100cm	60cm to 85cm
FIFA 02	Angled Ball Behavior (Soccer)	45% to 70%	45% to 60%
FIFA 03	Ball Roll (Soccer specific)	4 to 10 meters	4 to 8 meters
Testing Frequency Guidelines - according to Client requirements and budget			
Client requirements	Laboratory testing	Pre-Purchase and as QC Measure	Pre-Purchase and as QC Measure
Client requirements	Field Testing	After Installation	After Installation
Client requirements	Field Testing - Follow-up	Every Four Years	Every Year

Selected Test Descriptions

Durability testing (ASTM and/or FIFA)

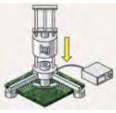


Heavy Wear and Abrasion Resistance - The surface is artificially abraded (simulation of multiple years of wear) and tested for the following: Shock Absorption, Vertical Deformation, Vertical Ball Rebound, and Rotational Resistance.

Climatic Resistance – Yarn and Infill - UV / Water / Heat - Measures the colour change, appearance change and yarn tensile strength changes – *(infill properties change after UV exposure)*

Sport Performance

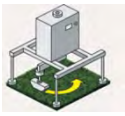
ASTM F1936 ***g*-max** – gives an indication of high impact shock absorption



Shock Absorption and Vertical Deformation – Measures the impact absorption provided by synthetic turf to a player running (lower extremity impact) or falling on as well as the foot stability of the surface as a player runs across it. Excess deformation of a surface could lead to over strained joints and fatigue.

Rotational Resistance – ***Traction*** - Measures the interaction between the shoe sole and the surface of artificial grass relating to the ability of a player to change direction.

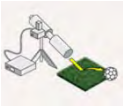
Slip Resistance Scale and Deceleration - Measures the ability of studs to slide through the surface without causing the player to slip over. Slip resistance deceleration measures the deceleration experienced by the players shoe as it makes contact with the surface. If the deceleration is too high, damages to joints and ligaments may occur.



Skin Abrasion/ Skin Friction - Measures the abrasiveness and friction of artificial turf on the skin of the player when sliding.

Vertical Ball Rebound - Measures how high the ball bounces when falling vertically onto a synthetic turf field. *(Although a method for soccer, this also gives an indication of consistent infill levels throughout the playing surface in a low cost tool)*

Ball Roll - Measures how far the ball rolls onto synthetic grass compared to natural grass. *(Although a method for soccer, this also gives an indication whether or not grass piles are standing up in a low cost tool)*



Angled Ball Behavior – *Soccer specific* - Measures how the ball rebounds from an artificial turf surface when striking it at a shallow angle under dry and wet conditions.

Test methods should be performed according to the applicable standards. The FIFA test methods and FIFA Quality Concept are the intellectual property of FIFA and should only be used within this framework.

For additional technical guidance and information, please visit www.SyntheticTurfCouncil.org.

DISCLAIMER: The *STC Guidelines for Synthetic Turf Performance* are voluntary. This document does not, in any way, imply, suggest or guarantee that a warranty, environmental, or performance issue could not arise if the system, product or component meets the suggested guidelines; nor does it imply or suggest that if any of the guidelines are not met that the product will fail to perform. These guidelines are not standards and are not to be used as the basis for warranty or other claims. The performance guidelines have been suggested to enhance the physical use of synthetic turf multi-purpose sports surfaces; however, they are not intended to be, and are not, safety standards and this document does not imply that an injury is less likely to occur if the synthetic sports surface meets the conditions and guidelines contained herein.

SUGGESTED GUIDELINES

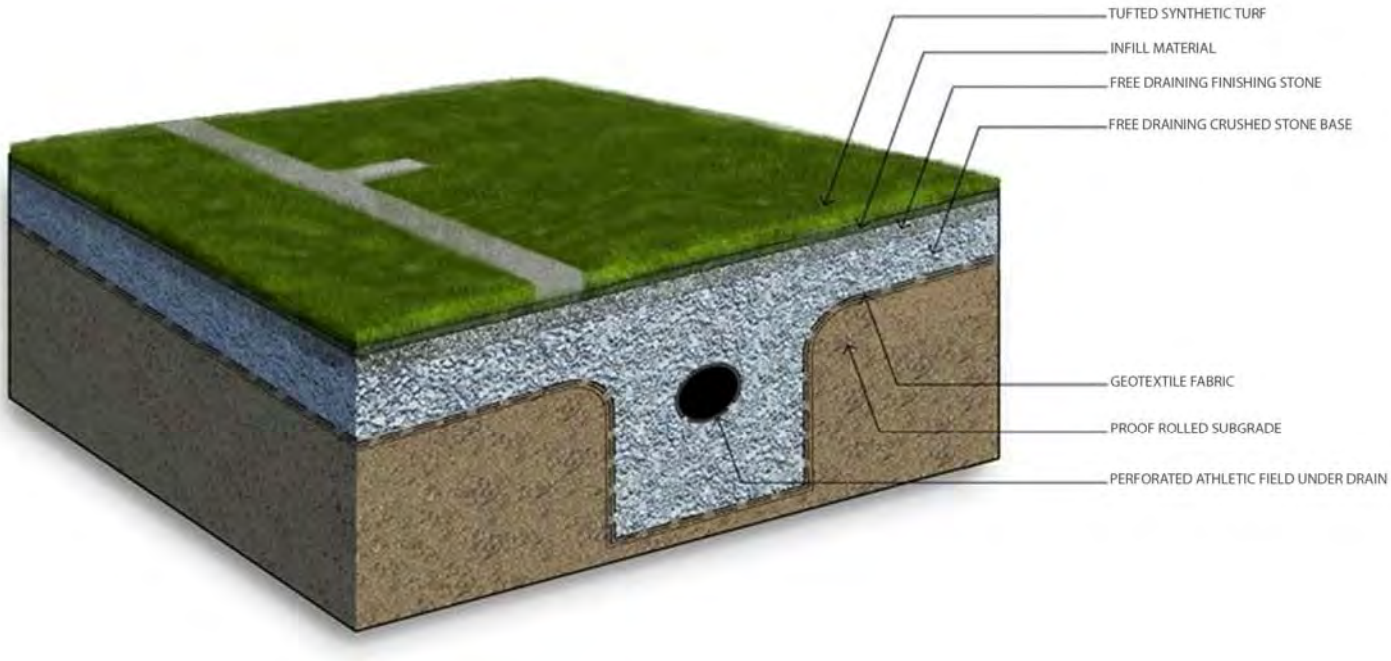
for the

ESSENTIAL ELEMENTS

of

SYNTHETIC TURF SYSTEMS

Revised November 2011



SUGGESTED GUIDELINES

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400 Galleria Parkway, Suite 1500
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PREFACE

The Synthetic Turf Council (STC) was formed to guide the selection and use of synthetic turf systems. The STC is a non-profit association dedicated to addressing the desire of the industry to enhance its reputation for quality. It hopes to earn support by developing documents that facilitate communication and good business practice between all parties to the synthetic turf systems including owners, selection teams, architects, designers, specifiers, consultants, testing laboratories, suppliers, manufacturers, installation and maintenance contractors, etc. Every attempt will be made to dispense information that is unbiased, objective, and in the best interest of all the parties and the user/owner in particular. The STC also suggests the utilization of standards and test methods that already exist, whenever applicable, to generate greater understanding in the selection and use of synthetic turf systems domestically and internationally. The STC offers to serve as a neutral platform where all elements of the industry can discuss, mediate or resolve issues.

The initial undertaking by the Synthetic Turf Council has been the development of guidelines for essential elements to be included in an objective non-proprietary specification for synthetic turf systems. Minimums suggested in these guidelines seek to improve the level of confidence in the process of selecting synthetic turf systems by owner/user/clients, selection teams and design professionals. This document facilitates a clearer understanding by all the parties of their expectations and the delivery of systems, products, and services in order to promote genuine satisfaction by the user/owners of synthetic turf systems. These guidelines developed by the STC are a continual work in progress and may be changed at any time through an orderly process that accommodates consideration of documented input. While this document contains minimums, it promotes the use of all systems that not only meet or exceed those minimums, but also encourages innovation.

The development of documents like these suggested guidelines is intended to facilitate constructive input and forge a cooperative atmosphere between users and producers. The STC invites users, professionals, manufacturers, risk managers, school officials, sports authorities, contractors and/or the representative associations of these segments to utilize these suggested guidelines.

Synthetic Turf Council

For more information, contact:
Synthetic Turf Council
400 Galleria Parkway, Suite 1500, Atlanta, GA 30339
Telephone (678) 385-6720; Fax (678) 385-6501; Email office@syntheticturfcouncil.org
Website: www.syntheticturfcouncil.org

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SYNTHETIC TURF SYSTEMS

INTRODUCTION

This document intends to provide reliable guidelines that address the need for essential elements to be included in an objective non-proprietary specification for synthetic turf systems. The minimums suggested in these guidelines seek to improve the level of confidence of the owner/user/client, selection team and design professional in the specifying and selection of synthetic turf systems. Adoption of the provisions in this document is voluntary; however, when utilized, they can assist all parties involved with the selection, design, supply and service of the systems, to generate a clearer understanding of the expected performance, the declared use, the inclusions and exclusions and the warranty coverage of synthetic turf systems. While these suggested guidelines contain minimums, they are not intended to stifle innovation. Enhancements, quality upgrades and proven innovations are encouraged to generate features that enhance the performance characteristics of the system.

1. GENERAL

1.1 These suggested guidelines provide reliable tools for the selection of synthetic turf systems relative to their declared use and expected performance.

1.2 The document is not intended to replace detailed plans and specifications. The provisions contained herein must be augmented by details specific to the project design, the job site, local environmental factors and all applicable laws, regulations and codes before the final specification can be considered comprehensive.

1.3 Those adopting or utilizing the provisions in this document have the obligation to determine their full compliance with all applicable international, federal, state and local laws, regulations, and codes. These suggested guidelines are not intended to modify that obligation or to serve as a substitute for compliance.

1.4 All Providers of the components of the systems should maintain a comprehensive quality control program to assure that the components meet the suggested guidelines.

1.5 These suggested guidelines are a continual work in progress and may be changed without notice to accommodate documented information justifying such changes.

1.6 Terminology definitions are specific to this document.

- 1.6.1 Base Materials: Materials that provide porosity and stability such as crushed aggregate or porous pavement.
- 1.6.2 Denier: The weight in grams of 9000 meters of fiber
- 1.6.3 Drainage System: A method of removing surface and subsurface moisture/water.
- 1.6.4 Fiber: A specific form of fibrous textile material that has a length at least 100 times its diameter or width.

1.6.5 Fiber Thickness: A measurement in microns (metric) or mils. (U.S.) of the thinnest cross section of a fiber.

1.6.6 *g*-max: A measurement of impact (shock absorption) in terms of gravity units as a ratio of deceleration.

1.6.7 Infill: Loosely dispersed materials that are added to the synthetic turf system, typically sand, rubber, other suitable material, or a combination thereof.

1.6.8 Knitted: A process in which the yarn fibers of the pile are tied to the backing which was simultaneously constructed by transforming continuous strands of multi-filaments into a series of interlocking loops, each row of such loops hanging from the row immediately preceding it.

1.6.9 Water Permeability: The rate at which water flows through a surface or system cross-section or components of the cross-section.

1.6.10 Planarity: Uniformity of the surface as compared to certain fixed predetermined points or prescribed slopes.

1.6.11 Primary Backing System: A single or multiple layers of woven or non-woven materials, into which the fiber is either tufted or knitted, to provide the initial construction of the synthetic turf.

1.6.12 Secondary Backing System: A coating and/or woven or non-woven fabric layer(s) applied to the primary backing after the fiber pile has been tufted or knitted into place, which serves to enhance tuft bind and provide additional structural integrity.

1.6.13 Shock Absorbing System: Component(s) that add resiliency to the system.

1.6.14 Sub-grade: A stabilized foundation onto which the base materials and field systems are installed.

1.6.15 Synthetic Pile Fiber: Grass-like blades made of synthetic materials.

1.6.16 Synthetic Turf Systems: These systems are comprised of (a) synthetic grass-like surface piles, tufted or knitted into a primary backing system to which a secondary backing system has been applied; with or without infill material(s); (b) a shock absorbing system, and (c) suitable base materials with an appropriate drainage system.

1.6.17 Tufted: A process by which the fiber yarns that form the pile are inserted into a previously prepared blanket-like primary backing.

1.6.18 At Installation: A period of time close to the completion date of the turf installation, but not to exceed 30 days.

1.7 Abbreviations referenced in the body of this document.

1.7.1 ASTM: American Society for Testing and Materials

1.7.2 DIN: Deutsches Institut für Normung

1.7.3 FIFA: Federation of International Football Association

1.7.4 FIH: International Hockey Federation

1.7.5 FIRA: Fédération Internationale de Rugby Amateur

1.7.6 IAAF: International Amateur Athletic Federation

1.7.7 NCAA: National Collegiate Athletic Association

DISCLAIMERS

The Suggested Guidelines for the Essential Elements of Synthetic Turf Systems are voluntary. This document does not, in any way, imply, suggest or guarantee that a warranty, environmental, or performance issue could not arise if the system, product or component meets the suggested guidelines, nor does it imply or suggest that if any of the guidelines are not met that the product will fail to perform. These guidelines are not standards and are not to be used as the basis for warranty or other claims. The guidelines have been suggested to enhance the use of synthetic turf sports surfaces; however, they are not intended to be, and are not, safety standards and this document does not imply that an injury is less likely to occur if the synthetic sports surface meets the conditions and suggested guidelines contained herein.

2. SCOPE

The makeup of the components and the elements to be included, or not to be included, are determined by the design, use and expected performance of the system.

2.1 Synthetic Turf Types: There are several different types of synthetic turf available. They are distinguishable through the use of different fibers and different construction. Differentiated by construction are the tufted or the knitted synthetic turf systems. Both systems are comprised of synthetic fibers with primary and secondary backing systems and a resilient shock absorbing system. The shock absorbing system can consist of infill, a padding system, or a combination of both.

2.2 Fiber: Typically, the fiber used in synthetic turf is textured and/or non-textured polypropylene, polyester, polyethylene, nylon or other suitable performing hybrid or copolymer in tape form or monofilament. Minimum fiber sizes are 50 microns for polypropylene or polyester, 100 microns for tape form (slit-film) polyethylene, 140-300 for monofilament polyethylene (shape dependent) and 500 denier for nylon. Fiber sizes for hybrids or copolymer will comply with the most closely related fiber type. Ideally, all fibers should be of the same chemical composition, shape, and texture. Fibers should be compliant to ASTM guideline for total lead content.

Monofilament

Tape form (slit-film)

2.3 Primary Backing Systems Material: The primary backing materials are of a woven or non-woven fabric in one or more layers which are utilized in the tufting process, or of high strength polyester multi-filament fiber utilized in the knitting process. This backing material provides the initial dimensional stability for the system.

2.4 Secondary Backing Systems Material: The secondary backing materials are applied through a coating process with a single or multiple applications of one or various materials.

2.4.1 Typically, knitted turf fabric receives an initial acrylic coating followed by different options of polyurethane, latex, other suitable coatings or fabrics in various weight and thickness configurations, depending on individual system design. The secondary backing utilized in knitted turf systems provides additional structural integrity to the system.

2.4.2 A tufted fabric typically receives a suitable coating of polyurethane, latex, other coatings or fabrics in various weight and thickness configurations, depending on individual system design. The secondary backing provides an additional level of tuft bind, bundle encapsulation and structural integrity to the synthetic turf. Should an increased level of system performance be desired, multiple layers of secondary backing materials with different physical characteristics could be applied.

2.5 Perforations: Depending on the final construction of the turf system, the system may or may not be permeable to water. Perforations are typically required of fully coated system backings to provide adequate vertical drainage throughout the system. Some turf systems may allow for drainage without perforations by employing a process of partial coating or other system designs. Developments in coating systems have provided for lighter weight and aqueous permeable chemicals; however, the drainage criteria must be met.

2.6 Infill Materials: The most recent generation of synthetic turf systems utilizes a long pile height and needs to be supported with infill materials for directional stability and structural integrity, as well as resiliency. The infill materials commonly used are sand, rubber, other suitable materials, or combinations thereof.

2.6.1 **EPDM** (Ethylene Propylene Diene Monomer) is a polymer elastomer with high resistance to abrasion and wear and will not change its solid form under high temperatures. Typical EPDM colors are green and tan. EPDM has proven its durability as an infill product in all types of climates. Its excellent elasticity properties and resistance to atmospheric and chemical agents provide a stable, high performance infill product.

2.6.2 **TPE** (Thermo plastic elastomer) infill is non-toxic, heavy metal free, available in a variety of colors that resist fading, very long lasting, and 100% recyclable and reusable as infill when the field is replaced. TPE infill, when utilizing virgin-based resins, will offer consistent performance and excellent *g*-max over a wide temperature range.

2.6.3 **Organics:** There are several organic infills available in the North American market, all utilizing different organic components, such as natural cork and/or ground fibers from the outside shell of the coconut.

These products can be utilized in professional sports applications as well as for landscaping. At the end of its life cycle it can be recycled directly into the environment.

2.6.4 **Silica Sand**: Pure silica sand is one of the original infilling materials utilized in synthetic turf. This product is a natural infill that is non-toxic, chemically stable and fracture resistant. Silica sand infills are typically tan, off-tan or white in color and - depending upon plant location – may be round or sub-round in particle shape. As a natural product there is no possibility of heavy metals, and the dust/turbidity rating is less than 100. It can be used in conjunction with many other infills on the market to provide a safe and more realistic playing surface. The round shape plays an integral part in the synthetic turf system. It is important that silica sand have a high purity (greater than 90%) to resist crushing and absorption of bacteria and other field contaminants. Silica sand can either be coated with different materials as a standalone product or can be used to firm up in combination with traditional crumb rubber infill systems.

2.6.5 **Coated Silica Sand:** This class of infill consists of coated, high-purity silica sand with either a soft or rigid coating specifically engineered for synthetic turf. These coatings are either elastomeric or acrylic in nature (non-toxic) and form a bond with the sand grain sealing it from bacteria to provide superior performance and durability over the life of a field. Coated sand is available in various sizes to meet the application's needs.

2.6.6 **Crumb Rubber**: Crumb Rubber is derived from scrap car and truck tires that are ground up and recycled. Two types of crumb rubber infill exist: Ambient and Cryogenic. Together these make up the most widely used infill in the synthetic sports field and landscape market. Crumb rubber infill is substantially metal free, and, according to the STC Guidelines for Crumb Rubber Infill, should not contain liberated fiber in an amount that exceeds .01% of the total weight of crumb rubber, or .6 lbs. per ton.

2.6.7 **Coated Rubber**: Both ambient and cryogenic rubber can be coated with colorants, sealers, or anti-microbial substances if desired. Coated rubber provides additional aesthetic appeal, reduction of dust by products during the manufacturing process and complete encapsulation of the rubber particle.

2.6.8 **Hybrid**: Constitutes the use of sand, rubber, or other suitable materials in various combinations. (This should not be confused with hybrid carpet systems that consist of a combination of fiber types.)

3. PERFORMANCE EVALUATION OF SYNTHETIC TURF

(For Tests and Criteria – Refer to Appendix A, Tables 1-7)

3.1 Player-Surface Interaction: Player-surface interaction describes the performance characteristics of the field that relate to footing, shock absorbency, surface abrasion, and surface stability, for example. These characteristics are determined through testing for vertical deformation, force reduction, traction, slip resistance,

energy restitution, and abrasiveness, among others. Proper shoe selection is an important factor in the way a player interacts with the playing surface.

3.1.1 Traction: The surface should provide good traction in all types of weather with the use of conventional athletic type shoes applicable to the sports and/or activity specified.

3.1.2 Rotational Resistance: The surface should allow for twisting movements as is common in athletic activities. Rotational resistance measures the ability of the user to perform twisting motions when in contact with the surface.

3.1.3 Slip Resistance Component: The system should enable a predictable range of movement between the user and the surface uniformly throughout. The surface should balance traction and slippage by way of the sliding coefficient.

3.1.4 Surface Abrasiveness: The field surface should have fibers and infill materials that minimize skin abrasions.

3.1.5 Impact Attenuation (*g*-max): The field surface should have the ability to adequately absorb player impact with the surface. The *g*-max and force reduction tests are two tests typically used. The current industry standard for *g*-max is a maximum value of 200 at each test point. *g*-max values may vary from location to location on a playing surface. Such variances shall be taken into account when setting maximum test values. A maximum, not-to-exceed limit, should be specified for the life of the warranty. *g*-Max should be below 165 for the life of the field.

3.1.6 Surface Stability (vertical deformation): The surface should provide adequate stability so that the athlete can maintain body control to help prevent or properly control contact between athletes. This is an important consideration that should be balanced with the surfaces' ability to absorb impact. If the surface is too soft, the stability provided by the field may not be optimal for player movement and body control.

3.2 Ball-Surface Interaction: Ball-surface interaction describes the performance characteristics of the field that relate to the ways in which the ball reacts to the surface. The field surface should provide consistent and predictable ball performance reaction characteristics.

3.2.1 Surface Uniformity: The synthetic turf playing field should be as level as practical. The synthetic surface shall provide a true and uniform playing surface throughout.

3.2.2 Ball Bounce: The synthetic turf field should provide a ball bounce as close to the optimal playing characteristics of the sport or sports. The published standards for the regulatory organizations as applicable for each sport should be referenced. The choice of fiber type and infill combination can affect ball bounce and in some cases promote greater "infill splash" which may not be desirable depending on the activity and level of competition.

3.2.3 Ball Roll: The synthetic turf field should provide a ball roll as close to optimal playing characteristics of the intended sport or sports. The choice of fiber type and infill combination as well as the level and type of maintenance can affect ball roll. The published standards for the

regulatory organizations as may be applicable for each sport should be referenced.

3.3 Appearance: Unless otherwise dictated by design, the synthetic turf should have a consistent color, texture, and shade without significantly noticeable streaks or other irregularities when observed in any direction.

3.4 Quality Control: The synthetic turf systems builder's quality control program should be evaluated with the system. There should be an understanding between the owner and the synthetic turf systems builder relative to the initial installation and ongoing testing responsibility, methods, and protocol. At the owner's option an independent third party testing agency or laboratory experienced with synthetic turf should test for compliance and acceptance.

3.5 Warranty: All of the proposed warranty documents should be obtained and the content thoroughly reviewed to ensure adequate coverage is contained therein.

3.5.1 Warrantor: The history and warranty performance of each warrantor should be objectively researched.

3.5.2 Important considerations in reviewing synthetic turf system warranties are:

- Who will be honoring the warranty when the field is completed?
- Is the warranty pro-rated or not pro-rated based on the age of the field?
- What is the duration of the warranty (3 yrs., 5 yrs., other)
- Has each system component been clearly defined and/or described in the specification and warranty?
- What is specifically included or excluded and what is not mentioned or covered?
- Are there any limitations as to notice, expiration, extenuating circumstances or nature of the remedy?
- Can final warranties be changed unilaterally without mutual consent?
- Have the possibilities of known risks to the users of the system been stated in writing?
- What conditions can void the warranty; e.g., the lack of routine, incorrect maintenance, or the use of unapproved maintenance equipment?
- What monitoring measures are acceptable to all parties?

3.5.3 Warranties for the synthetic turf field systems should be clearly understood and may include the following:

- Acceptable uses for the field
- Expected number of yearly hours of use of the field
- Type of shoes used
- Fading
- Color match within specifications
- Excessive fiber wear
- Acceptable loss of pile height over time
- Wrinkling and panel movement
- Shock absorbency (*g*-max)
- Seam integrity

- Drainage
- Response time for required repairs/replacement
- Approved maintenance equipment
- Other items deemed relevant

3.5.4 Several parties may be involved in providing the finished system. There may be blanket warranty coverage or each party; i.e., vendor, supplier, manufacturer, installation and maintenance contractor may provide separate and limited coverage that should be clearly defined. It should also be clear exactly who covers what, with special attention given to the coverage of the turf fiber as it relates to possible damage during the tufting, coating, installation and maintenance processes.

3.5.5 Qualification: Consideration should be given to the experience and qualification of the manufacturer and installation contractor of the synthetic turf system, and their maintenance and repair personnel.

3.6 Maintenance: Maintaining a synthetic turf field is essential for optimum appearance, safety, playing performance, and field longevity. A regular schedule of maintenance should include surface cleaning, debris removal, grooming, and infill replenishment, redistribution, and de-compaction. The maintenance procedures and equipment, as specified by the synthetic turf system builder and required for the system, should be evaluated during the selection process so that the appropriate budget resources for manpower and equipment may be allocated. **Note:** Refer to the Synthetic Turf Council's ***Suggested Guidelines for the Maintenance of Infilled Synthetic Turf Surfaces***, April 2007, for additional information.

3.7 Other Considerations: Provisions that could prevent a breakdown in communication or a delay in the process should be included.

3.7.1 Review and approvals prior to work: The synthetic turf systems builder, without liability or legal responsibility for the base (unless the base is part of their scope of work) should perform an inspection of the field planarity base on to which the synthetic turf system is to be installed and to examine the finished surface for required compaction, water permeability, and grade tolerances. After any discrepancies between the required materials, application and tolerance requirements noted have been corrected, the owner's representative (architect/engineer) should review and approve for compliance with documents. The acceptance of the base construction should be included in the certification for warranty validation.

3.7.2 Extra Materials: Upon request and agreed compensation prior to the manufacturing of the field, the synthetic turf manufacturer and installation contractor can provide extra sections of synthetic turf material for future repairs. If necessary, this should include materials for all colors used with any lines, markings, and logos. Quantities to be predetermined. This allows for materials from the same manufacturing run to be utilized for minor repairs.

- Storage: Extra Materials should be stored unrolled outside so that any fading as a result of UV exposure will be consistent with the installed material.

4. EVALUATION OF SYSTEM COMPONENTS

(For Tests and Criteria -- Refer to Appendix A, Tables 1-7)

4.1 Drainage System: An efficient and effective underground drainage system is an integral component of a synthetic turf system, and is designed to carry away the water that percolates through the turf. The system chosen will depend on the use of the field, climate, amount of rainfall and other factors.

4.1.1 Components: The drainage system may include the synthetic turf, pad, base materials and collector pipes that collect and remove storm water from the playing field. The design of the drainage system is dependent upon local conditions, climates, and site constraints. The Rational Method, Hydrograph Analysis, or Time Series Method may be used to determine the rainfall runoff that must be accommodated by collector pipes.

4.1.2 Site Conditions: Rainfall duration intensity curves can be developed from the National Weather Service Technical Paper TP-40 *Rainfall Frequency Atlas for the United States* or coordinated with the local weather statistics at the location of the project site. Otherwise, unless agreed to by the end user, the design storm frequency should be as required by local regulations. Where no local regulation exists, a minimum 5-year design storm frequency is recommended for playing fields at grade. For fields requiring pump stations, a more conservative design frequency that is compatible with the design capacity of the pump station should be used.

4.1.3 Flow Time: The time interval for water to flow through the complete system to the collector pipes is based on permeability tests conducted in the laboratory for the design of the complete system. Flow through the base material can be enhanced by the use of composite drainage materials or lateral drain pipes that intercept the normal flow of water in the complete system and flow directly to the collector pipes. Flow rate into the lateral drainage system is dependent on the amount of available open space for water to enter the pipe. The geotextile cover on many composite drains can have varying effects on how fast water can enter the system over time. Care should be taken in evaluating these products and how the chosen base materials can affect water in-flow over time.

4.1.4 Collector Pipes: Collector pipes are typically perforated polyvinyl chloride (PVC) or polyethylene (PE) pipes. Size and type of perforations are dependent upon the size of the pipe. If perforations are larger than the smallest aggregate in the base material then a geo-textile sock filter may be used to encapsulate the pipe – care should be taken to ensure that the openings in the geo-textile fabric are compatible with the granular smaller components so that they do not block the pores and reduce water

flow. A qualified civil or geotechnical engineer should be consulted to determine the suitability of using a product with a geo-textile sock in conjunction with the selected base materials as they can clog the sock over time and severely impede the performance of the system. Additionally the compressive strength of various systems can differ greatly and care should be taken to keep construction traffic off of the systems until enough stone has been placed and compacted.

4.1.5 Drainage: The expected performance evaluation and the systems used should undergo an independent engineering analysis.

4.2 Base Materials: The aggregate base on which the synthetic turf is installed provides a structurally sound foundation for field construction, and a media for drainage of the field. The base materials are critical to the performance of the entire system and should contain the necessary components and characteristics to satisfy local conditions. A good geotechnical report will provide essential information for a firm and stable base for the synthetic turf. A base that is properly designed and constructed should give the owner several years of use and last through several turf replacements. The use of design professionals and builders with demonstrated expertise and success in the development of synthetic turf systems is highly recommended.

4.2.1 Soil Separator: Depending on the local site conditions, a geo-textile fabric may be placed over the entire sub-grade and within the pipe trenches prior to the installation of the base materials to minimize contamination of the aggregate and possible clogging of the perforated drainage pipes. Where soil conditions warrant, a polyethylene, PVC, or other impermeable sheet liner may be used in lieu of the geo-textile to inhibit storm water infiltration into the subsoil.

4.2.2 Aggregate: The aggregate materials utilized to construct the field base must be a properly graded washed crushed stone to provide a balance between stability and permeability. A highly fractured material is desirable to provide the surface stability required for the synthetic turf surfacing, supplemental padding or porous paving as applicable. The graded aggregate particle sizes must be tightly controlled to fall within the bandwidth for all specified sieve sizes with just enough fines to provide stability while still allowing for sufficient drainage. Minimum stability and permeability requirements should be determined and confirmed by an independent certified laboratory prior to construction of the base course.

4.2.3 Compaction: The base materials should be thoroughly compacted to prevent differential settlement across the field area. Minimum compaction levels typically should not be less than 95% density as measured by a standard proctor test. Special attention should be given to backfill compaction of any utility trenches that cross the field area. Care should also be taken not to over compact, which could affect drainage.

4.2.4 Pavement: If pavement is required by design, the base materials may be porous or conventional asphalt. This material is installed over a permeable aggregate base and a subsurface drainage system. The porous

pavement material must be manufactured with tight quality control on asphalt content, as well as the gradation of the aggregate used in the mix. This aggregate should have a limited amount of fines to allow for efficient water permeability. Use of conventional asphalt paving will require a sloped field with either a crown or a cross slope. Consideration should be given to the use of a drainage mat or an elastic layer pad system between the turf backing and the surface of the pavement. This, along with the installation of periodic interceptor drains, should allow for horizontal water movement below the field's surface. Without the use of these materials, the infill layer will become saturated during periods of heavy rainfall and there may be migration of the infill materials with the surface water movement.

4.2.5 Water Permeability: Water permeability rates for both the field's surfacing and the field base materials should be designed to accommodate the local weather patterns and storm water management regulations. The permeability of both the field surface and the base materials will typically decrease over the life of the field. An adequate factor of safety should be utilized to provide initial infiltration rates for the completed field above those required by the local weather conditions.

4.3 Shock Absorbing Resilient Underlayment Systems: The shock absorbing elements, as part of the overall synthetic turf system, should meet or exceed the performance of the design and specification.

4.3.1 *In situ* Cushion Layer (elastic layer pad): If included in the design, these cushion systems should be installed in place with specialized paving equipment.

4.3.1.1 Physical Characteristics: These systems are typically comprised of SBR rubber granules bound with a single component polyurethane binder. Small rounded pea gravel aggregate or other suitable materials can also be incorporated with the rubber and urethane materials. The firmness of the system can be adjusted with the size and the proportions of the rubber granules and aggregate materials, as well as with the amount of polyurethane binder used and the thickness of the layer.

4.3.1.2 Performance Characteristics: The selection of the cushion layers should be closely coordinated with the performance characteristics of the synthetic turf utilized. The cushion layers should provide shock absorption without compromising footing and surface stability.

4.3.1.3 Water Permeability Rate: The *in situ* cushion-layer systems are typically permeable. The percolation rate for the *in situ* cushion layers should be well in excess of precipitation rates.

4.3.2 Prefabricated Cushion Layers (Pad): If included in the design, these cushion layers are a manufactured product comprised of rolls or tiles of resilient material installed under and occasionally adhered to the synthetic turf backing.

4.3.2.1 Physical Characteristics: Prefabricated cushion layers are typically comprised of rubber, polyurethane foam, or other suitable materials. The rubber pads are SBR rubber fibers or granules bound together with a polyurethane binder and usually come as roll or piece goods and should be permeable. The foam cushion layers are typically polyurethane or polyvinyl chloride and should be water permeable for drainage.

4.3.2.2 Performance Characteristics: The selection of the cushion layers should be closely coordinated with the performance characteristics and requirements of the synthetic turf system utilized. The cushion layers should provide shock absorption without compromising footing and/or surface stability.

4.3.2.3 Water Permeability Rate: Depending on the final construction of the pad system, the system may be or may not be permeable to water. Unless the system is permeable by design with adequate drainage, perforations should be put through all of the cushioning layers to provide for adequate drainage through the system as specified.

4.3.3 Infill Materials: Infill materials are comprised of rubber, sand, elastomers, organics and/or other suitable materials, or combinations thereof which are placed on top of the synthetic turf backing system and between the synthetic surface fibers. This is needed for resiliency as well as structural integrity and directional stability. This material is utilized today in the vast majority of synthetic turf systems with the exception of the traditional knitted synthetic turf systems. (Also addressed in Section 4.6)

4.4 Irrigation System Introduction: The installation of a manual or automatic irrigation system can be considered for synthetic turf installations. Guidelines on whether synthetic fields are watered are determined by factors such as region, climate, turf material, player traffic type and level of games played.

4.4.1 Player Comfort: High field temperatures can prove challenging to players throughout warmer climates. Watering enables the field to be cooled. Tests show approximate reduction in surface temperature of 46 degrees Fahrenheit with 10 minutes of applied watering using a high volume long range pop up sprinklers. (Results derived from a height of 2 inches above infilled synthetic turf.)

4.4.2 Field sanitation: Field cleansing and sanitation can be improved with watering from an irrigation system, particularly in climates that experience very little rainfall for natural cleansing.

4.5 Synthetic Turf: The synthetic turf surface should provide the performance characteristics, components, and construction that meet the needs of the declared use and/or functions. The synthetic turf system and all of its components should be resistant to moisture, rot, mildew, bacteria, fungus growth, ultraviolet ray degradation at all field locations, and meet local code and environmental requirements.

4.5.1 Synthetic Turf Construction and Components should be non-toxic and not cause commonly known allergic reactions. Each synthetic turf system should be constructed to provide dimensional stability and resist damage from wear and tear during athletic and recreational usage.

4.5.2 Fibers for Tufted or Knitted Systems: Typically the fiber used in synthetic turf is textured and/or non-textured polypropylene, polyethylene, or nylon in tape form or monofilament. Minimum fiber sizes are 50 microns for polypropylene and polyester, 100 microns for tape (slit film) form polyethylene, 140-300 for monofilament polyethylene (shape dependent) and 500 denier for nylon.

4.5.3 Fibers for Knitted Systems: Typically the fibers used for knitted systems are Nylon 6.6 or Nylon 6 of 500-650 denier, texturized monofilaments.

4.5.4 Primary Backing Systems: The primary backing materials should be either high strength polyester multi-filaments utilized in the knitting process, or woven, non-woven, or other suitable materials in one or more layers, utilized in the tufting process.

4.5.5 Secondary Backing Systems: The secondary backing materials should be applied through a coating process that can be single or multiple applications of one or several different materials. A knitted turf fabric should receive an initial acrylic coating and could be followed by different options of suitable polyurethane, latex and/or other coatings in various weights and thickness configurations, depending on individual system design. A tufted turf fabric should receive a suitable polyurethane or latex pre-coat or an acceptable performance-based equal, which then can be followed by an attached cushion or a laminated secondary backing utilizing a suitable polyurethane, latex, or acceptable performance-based equal. The purpose of the secondary backing is to enhance the tuft bind and structural integrity of the turf components. In cases where an increased level of system performance is desired, multiple layers of secondary backing materials of different physical characteristics can be applied.

4.5.6 Water Permeability Rate: Depending on the final construction of the turf system, the system may be or may not be permeable to water. Unless the system is permeable by design with adequate drainage, perforations should be put through all of the backing coatings to provide for adequate drainage through the system as specified.

4.5.7 Seams: New synthetic turf carpets are manufactured in panels or rolls that are typically a nominal 15 feet wide. Each panel or roll should be attached to the next with a seam to form the playing substrate of the field. Seams should be glued with a supplemental backing material or sewn with high strength sewing thread. The bonding or fastening of all system material components should provide a permanent, tight, secure, and hazard-free athletic playing surface.

4.5.8 Adhesive: Industrial adhesives, products not found in home supply stores, are used to bond synthetic turf seams and inserts, and, in some applications, for a total glue down of the synthetic turf to the base. Synthetic turf adhesives should be applied by experienced, professional

installers. The adhesives should provide a strong, hazard-free, and durable bond between the adjacent turf panels or sections and to be usable for installation under variable weather conditions. The adhesive should also be resistant to water, fungus, and mildew. Synthetic turf adhesives include: one-part adhesives (urethanes), two-part (epoxy or urethane), hot melt, and water-based (latex).

4.5.9 Seaming Tape: Seaming tape is commonly used for seams and/or inlaid lines and markings. The tape is comprised of a fabric that should be installed below the backing material on both sides of a seam or inlay. The fabric used for seaming tape should provide dimensional strength and enough surface texture and width to bond well with the adhesive and the turf backing material on each side of the seam.

4.6 Infill Material: Infill materials are comprised of rubber, sand, elastomers, organics and/or other suitable materials, or combinations thereof which are placed on top of the synthetic turf backing and between the synthetic surface fibers.

4.6.1 EPDM (Ethylene Propylene Diene Monomer) is a polymer elastomer with high resistance to abrasion and wear and will not change its solid form under high temperatures. Typical EPDM colors are green and tan. EPDM has proven its durability as an infill product in all types of climates. Its excellent elasticity properties and resistance to atmospheric and chemical agents provide a stable, high performance infill product.

4.6.2 TPE (Thermo plastic elastomer) infill is non-toxic, heavy metal free, available in a variety of colors that resist fading, very long lasting, and 100% recyclable and reusable as infill when the field is replaced. TPE infill, when utilizing virgin-based resins, will offer consistent performance and excellent *g*-max over a wide temperature range.

4.6.3 Organics: There are several organic infills available in the North American market, all utilizing different organic components, such as natural cork and/or ground fibers from the outside shell of the coconut. These products can be utilized in professional sports applications as well as for landscaping. At the end of its life cycle it can be recycled directly into the environment.

4.6.4 Silica Sand: Pure silica sand is one of the original infilling materials utilized in synthetic turf. This product is a natural infill that is non-toxic, chemically stable and fracture resistant. Silica sand infills are typically tan, off-tan or white in color and - depending upon plant location – may be round or sub-round in particle shape. As a natural product there is no possibility of heavy metals, and the dust/turbidity rating is less than 100. It can be used in conjunction with many other infills on the market to provide a safe and more realistic playing surface. The round shape plays an integral part in the synthetic turf system. It is important that silica sand have a high purity (greater than 90%) to resist crushing and absorption of bacteria and other field contaminants. Silica sand can either be coated with different materials as a standalone product or can be used to firm up in combination with traditional crumb rubber infill systems.

4.6.5 Coated Silica Sand: This class of infill consists of coated, high-purity silica sand with either a soft or rigid coating specifically engineered for synthetic turf. These coatings are either elastomeric or acrylic in nature (non-toxic) and form a bond with the sand grain sealing it from bacteria to provide superior performance and durability over the life of a field. Coated sand is available in various sizes to meet the application's needs.

4.6.6 Crumb Rubber: Crumb Rubber is derived from scrap car and truck tires that are ground up and recycled. Two types of crumb rubber infill exist: Ambient and Cryogenic. Together these make up the most widely used infill in the synthetic sports field and landscape market. Crumb rubber infill is substantially metal free, and, according to the STC Guidelines for Crumb Rubber Infill, should not contain liberated fiber in an amount that exceeds .01% of the total weight of crumb rubber, or .6 lbs. per ton.

4.6.7 Coated Rubber: Both ambient and cryogenic rubber can be coated with colorants, sealers, or anti-microbial substances if desired. Coated rubber provides additional aesthetic appeal, reduction of dust by products during the manufacturing process and complete encapsulation of the rubber particle.

4.6.8 Hybrid: Constitutes the use of sand, rubber, or other suitable materials in various combinations. (This should not be confused with hybrid carpet systems that consist of a combination of fiber types.)

4.7 Lines and Markings: Construction and materials used should be harmonious with the synthetic surface.

4.7.1 Installation: Lines and markings should be installed on the synthetic turf surface in one of three methods: with paint, with colored fiber that is either tufted or knitted into the synthetic turf panels, or installed as inlays. Tufted-in or inlaid lines and markings are a permanent part of the surface.

4.7.2 Permanency: Painted lines and markings installed with either permanent or temporary paint require maintenance. Even permanently painted lines require additional paint on a periodic basis.

4.7.3 Consistency: Synthetic turf and fibers utilized for the tufted or inlaid lines and markings should be similar to that used in all other areas of the field and installed to the same tolerances.

4.8 Inserts: They are typically used on multi-sport fields. They can include covers for goal sleeves and anchors and conversion of baseball infield clay areas to synthetic turf.

4.8.1 Consistency: The synthetic turf used for the inserts should be similar to that used in the area adjacent to the insert.

4.8.2 Installation: The inserts should be anchored securely to the surrounding areas so that they cannot be displaced by the activities occurring on the field and installed to the same tolerances.

5. CONSTRUCTION AND INSTALLATION

5.1 Inspection: Synthetic materials should be inspected prior to installation for:

- Damaged or defective goods
- Missing goods or quantities
- Correct fiber type
- Correct turf pile height and weight
- Proper tuft bind
- Correct backing perforation diameter and spacing, if applicable
- Materials out of tolerance with the specification

5.2 Sub-Grade Preparation: The sub-grade should provide a stabilized foundation upon which base materials and subsequent components of playing field systems will be installed.

5.2.1 Function: It should also provide the pitched surface on which storm water is directed toward the active drainage system for evacuation.

5.2.2 Shape and Compaction: Prior to placement of base materials, the sub-grade should be shaped to an appropriate profile and compacted by proof rolling to obtain a firm even surface. Depressed areas should be filled and unsuitable materials removed and replaced with clean fill or aggregate. Compaction should be performed to achieve a minimum of 95% in accordance with ASTM D698 Standard Proctor Method. The appropriate moisture content must be maintained in the field sub-grade to allow for optimal levels of compaction.

5.2.3 Sub-Grade (Rough) Planarity: The tolerances for the finished sub-grade should not exceed one-half (1/2") inch as measured by a 10-foot straight edge (13mm in 3m). Grading of the sub-grade shall minimize ponding to the extent practical. The use of laser guided and controlled equipment is highly recommended for sub-grade preparation.

5.3 Aggregate: Installation of the aggregate base should provide a close, evenly textured surface meeting the required tolerances.

5.3.1 Construction: Extreme care should be taken to ensure that there is no disturbance to the sub-grade and that there is no displacement of the soil separator. All disturbed, displaced, or damaged material is to be repaired or replaced.

5.3.2 Placement: The aggregate base should be placed in a manner that will produce a uniform and evenly graded mass to the specified depth. The material should be placed and spread by the appropriate equipment and methods in successive horizontal layers not exceeding six (6) inches in depth. Care should be taken to avoid overworking the material, which can affect gradation and uniformity, resulting in detrimental performance and drainage characteristics. Pockets that occur as a result of stone segregation during installation should be removed and replaced. After correct placement, each lift shall be uniformly compacted with a self-propelled roller to achieve the specified density.

5.3.3 Compaction: The field base materials should be thoroughly compacted to prevent any significant differential settlement across the area of synthetic

turf surfacing. Typical minimum compaction levels are 95% Standard Proctor for the base materials. The appropriate moisture content must be maintained in the base materials to allow for optimal levels of compaction.

5.3.4 Finish-Grade Planarity (surface tolerances): Irregularities in the surface of the base materials are typically reflected in the finished field surface. Therefore, it is important to install the base materials to controlled tolerances. The use of laser guided and controlled equipment is highly recommended for sub-grade preparation. The local deviation of the finished surface of the base stone should not exceed 1/4 in. in any direction when measured beneath a 10-foot long straight edge (6mm in 3m). Hollows and depressions, which may have developed during the process of compacting the base, should be filled with acceptable material and re-compacted.

5.4 Shock Absorbing Resilient Underlayment System: The design of these systems varies and each approach should be carefully installed to meet the requirements of the user.

5.4.1 *In situ* Cushion-Layer (Elastic Layer Pad) Installation: If required by design, the in situ cushion layers should be installed with specialized paving equipment used only for *in situ* pad or rubberized running tracks. The paving machine should be operated by a minimum of two skilled technicians at all times. All paving seams should be hand rolled and troweled. All cold joints in the pad should be pretreated with a polyurethane primer. The specified thickness of the *in situ* pad should be continuously monitored for consistency. The components of the *in situ* cushion layers should be thoroughly mixed. The mixing ratios should also be monitored for consistency. The cushion-layer system should be securely placed on the field base materials. The *in situ* cushion surface should not vary more than 1/4 in. in 10 feet as measured in any direction with a string line or straight edge (6mm in 3m).

5.4.2 Seam Installation: If required by design, prefabricated cushion-layer systems are typically installed as roll or piece goods. The head seams at

the end of each roll should be staggered across the field. When required by the padding manufacturer, all glued cushion-layer seams should be butted together and a permeable or mesh type fabric should be adhered to the surface of the cushion layer at all seam locations to bridge the cushion-layer joints. (This does not apply to sewn seams).

5.4.3 Resilient Infill: If required by design, the infill material should be applied when in a dry condition and should not be applied unless the synthetic turf is also dry. The infill material should be applied in consistent layers with multiple applications. It is critical to insure that synthetic fibers are not trapped underneath the infill. After application of each layer, the synthetic turf should be dragged and/or brushed according to the manufacturer's recommendations in order to lift the fibers and distribute the infill material into the turf system in a consistent manner. Equipment is however available that can install the infill materials and simultaneously brush them into the carpet pile in a single pass.

5.5 Irrigation System Design Considerations: When the inclusion of an irrigation system is deemed necessary or appropriate for a particular field installation it is highly recommended that it be designed, reviewed, and approved by a recognized irrigation consultant/designer. An experienced irrigation designer can provide necessary support, advice and forsee any pit falls resulting in costly re-work and an under performing system.

5.5.1 Requirements: A field watering system may be required to meet the demands of the game, such as high level field hockey competition. Each project and situation is unique. Factors such as the time allocated to irrigate before, during and sometimes after a match need consideration, this practice is common to ensure the players are offered a safe and controllable game.

5.5.2 Sprinkler Head Layout: Sprinklers located inside the field of play are not generally considered acceptable. Players falling on sprinklers through tackles and other play activities are subject to injury risk. The installation of mutiple infield sprinklers is and can affect the turf's adhesion to the field base. This can have a negative affect on the completed turf planarity and consistency. Use of agricultural, long range sprinkler guns mounted on riser posts has until recently been a common practice. Manufacturers now produce long range perimeter pop-up sprinklers. Buried at grade level, the pop-up sprinklers provide discrete, low level, unobtrusive, long range performance with the ability to throw beyond the half way line.

5.5.3 Layout Considerations: Typical sprinkler installation design would allow for minimum spray disruption for slight wind conditions. Consideration for the spectators should be met to ensure they do not become unnecessarily wet during a watering cycle caused by incorrectly adjusted arcs or ill-positioned sprinklers.

5.5.4 Control valves: High flow, low pressure loss electric solenoid valves should be installed, these are designed to ensure sprinkler performance is not prohibitied and necessary for high volume perimeter sprinklers.

5.5.5 Controls: Conventional irrigation scheduling is not often necessary with artificial field watering. Manually operating the irrigation system proves

more popular than traditional timers. A simple controller mounted in a cabinet with buttons programmed to switch on solenoid valves for a pre-set time or sequence is adequate for the ground personnel. Remote control is useful for field operation and allows the operator to ensure the field is clear of personnel hazard prior to operation.

5.5.6 Adequate safety precautions on the field such as warning notification sound and visual safety measures should be observed to ensure players, spectators and service personnel are not at risk from the water jet as the sprinklers are activated.

5.5.7 As an additional safety precaution, a security key restricting access to the control panel will ensure authorized use only of the watering system.

5.6 Synthetic Turf Material Production Quality Assurance/Quality Control: Testing of materials should be performed prior to shipment of product to the job site to avoid additional costs or delay.

5.6.1 Quality Assurance Testing: Prior to shipment of the synthetic turf and components to the job site, the synthetic turf rolls should be randomly sampled and tested by the manufacturer who will certify that they meet the specification.

5.6.2 Relevant Characteristics: Testing to be conducted should be a provision in the agreement between the parties and may include pile composition, pile weight, total weight, pile height, tuft bind (without infill), and grab/tear strength.

5.6.3 Labeling: The manufacturer, at his option, should convey in writing the test results of the relevant characteristics and certify that they meet or exceed the specification requirements.

5.7 Synthetic Turf Installation: All synthetic turf systems should be installed to provide stability that will prevent panels from shifting or bunching.

5.7.1 Seaming Method: The synthetic turf panels should be securely fastened together for the warranted life of the system. These seams are typically glued or sewn, the method for which varies from system to system. Specialized synthetic turf systems with are periodically removed and replaced may have seams, which are comprised of Velcro or other easily attachable materials. Seam gaps should be minimal and uniform. For tufted infill systems the gap between the fibers should not exceed the gauge of the tufting. For other synthetic turf systems, the seam gaps should not exceed 1/16 in. (2mm).

5.7.2 Edge Anchoring: The anchor may consist of a concrete curb, a treated wood header, a composite material or a trench drain. These may vary by design and region, but should always provide a secure anchor.

5.7.3 Inlaid Lines and Markings: Inlaid lines and markings should consist of synthetic turf with contrasting colored fiber installed in lieu of painted fiber. Inlay gaps should be uniform. For tufted systems, the gap between the fibers should not exceed the gauge of the tufting. Lines and markings must conform to the appropriate association or organization suggested guidelines for the intended level of use.

5.7.4 Temperature: Care should be taken during installation to account for rapid fluctuations in temperature to avoid expansion and/or contraction which can affect the final installation. Temperature extremes should also be carefully monitored. The carpet should never be rolled or unrolled when frozen, which can cause cracking and irreparable damage to the secondary backing.

5.8 Infill Material Installation: Correct installation is critical to performance of these systems and should follow the manufacturer's recommendations.

5.8.1 Environmental Conditions: It is recommended infill materials should be installed under dry field conditions.

5.8.2 Method of Application: The infill material should be installed uniformly. The equipment used for the application of the infill materials should erect the fiber, place the infill materials, and should incorporate a metering method to provide consistent distribution. The equipment utilized should not distort or displace any base materials or damage the system in any way.

5.8.3 Infill Depth: The depth of infill can be measured by taking the depth from the top of the primary backing to the top of the infill or subtracting the length of exposed fiber from the known pile height.

5.8.4 *g*-max Testing: *g*-max testing should always be performed by an independent testing company or lab.

5.9 Fiber Conditioning: It is essential to maintain the integrity and uniformity of the fiber throughout the manufacturing, shipping and handling, installation and maintenance processes in order to prevent damage, which could alter the specified performance and void the warranty.

6. MAINTENANCE

Maintaining a synthetic turf field is essential for optimum appearance, safety, playing performance, and field longevity. A regular schedule of maintenance should include surface cleaning, debris removal, grooming, and infill replenishment, redistribution, and de-compaction. The maintenance procedures and equipment, as specified by the synthetic turf system builder and required for the system, should be evaluated during the selection process so that the appropriate budget resources for manpower and equipment may be allocated. **Note:** Refer to the Synthetic Turf Council's ***Suggested Guidelines for the Maintenance of Infilled Synthetic Turf Surfaces***, April 2007, for additional information.

6.1 The synthetic turf installation builder should provide detailed written maintenance instructions, suggested guidelines for the system, and training of maintenance personnel. Maintenance of the systems typically consists of cleaning, stain removal, minor seam repair, dragging or redistribution of any infill material, and management of infill compaction. A primary goal of grooming maintenance is to keep the fibers standing in an upright position, which minimizes wear and UV degradation and can have a beneficial effect on performance, playing characteristics and longevity of the field. Specialized equipment is typically required for the maintenance of the surface and should be included with the field contract. Utilizing this equipment as recommended by the installation builder will generate the proper maintenance in relation to any future warranty claims.

6.2 Maintenance Monitoring: The owner/user shall be responsible to maintain a log of maintenance performed on the field as recommended and warranted by the builder.

6.3 Cleaning: The periodic use of a vacuum or a sweeper should be applied to keep the synthetic surface clean. This equipment should be compatible with synthetic turf fields. This typically means wider tires and softer nylon type brushes. The cleaning activities should conform to the written maintenance suggested guidelines provided by the synthetic installation builder.

6.4 Irrigation System Maintenance: As with any irrigation system, maintenance and winter draindown and preparation in cold climates should be observed.

6.5 Stain Removal: Stains such as tobacco, gum, etc. should be removed as soon as possible as per the installation builder's recommendations.

6.6 Brushing: The infill material and fibers should be periodically brushed depending on use to even out any low areas of infill materials and to brush the fibers back to a more vertical alignment. The sand and rubber infill materials can also have the compaction reduced with the use of metal tines. The brushing activities should conform to the written maintenance suggested guidelines provided by the installation builder.

6.7 Seam Repair: Seams that open or become loose may require some immediate and temporary gluing until they can be inspected and corrected by the installation builder. The gluing should conform to the written maintenance suggested guidelines provided by the synthetic turf vendor.

7. POST INSTALLATION TESTING

7.1 Schedule: It is recommended that a minimum schedule for on-going testing be included and understood by the parties to be at least at the end of year one and at the end of year three. Testing thereafter is at the owner's option.

7.2 The *g*-max should be tested in accordance with the above schedule.

7.3 Inspection of the seams and other installation features should be mutually agreed to by the parties but should be completed no less than one time per year or as stated in the manufacturer's warranty.

7.4 Governing Body Certification: Additional post construction performance testing may be required for fields used for high level competition. Contact the respective governing bodies for the current guidelines.

▪ APPENDIX A – TESTS AND GUIDELINES (TABLES 1 – 7)

A. Test Protocols and their respective STC minimums are for specific purposes such as the following.
   - Performance of the system and/or its essential components is the primary objective.
   - Quality control and delivery of the materials as specified.
   - Failure diagnosis, preventative measures, comparative evaluations, safety, appearance, and compliance with codes and regulations, etc.

B. Not all tests are required for every installation. A design professional can best advise on what site tests should be conducted under given circumstances.

C. Tests in the laboratory and in the field are to be utilized for a specific purpose and their protocols should be followed as published by their respective standards organizations.

D. Architects, design professionals, engineers, and consultants have the responsibility to refer only to the tables and charts in this Guideline document that apply specifically to the needs of the design, site, and the requirements of the intended use as indicated by the owner/end user and/or as may be required to validate the warranties offered.

E. All minimum values should be evaluated as they relate to the system performance.

F. Site testing shall be at ambient shaded air temperature of 40 – 100°F. Laboratory testing shall be at ambient indoor temperature unless otherwise specified by the test method.

G. Unless otherwise specified, field test measurements shall be made at a minimum of 6 locations (must avoid areas where 2 glued seams cross). Test locations shall conform as closely as possible to the test sites specified in ASTM F1936 (field used primarily for North American Football) or FIFA Handbook 3-06 (fields marked for Soccer).

H. All minimum and maximum values take into account acceptable industry manufacturing tolerances of +/- 2% of the variance.

TABLE 1 – BASE MATERIALS

Test Property	Method of Determination	STC Guidelines	Lab/Field Test
Size of particle mix	ASTM D422 Particle size analysis	As per specification	Lab (on site material)
Drainage	ASTM F1551/DIN 18035:6 Permeability to water* ASTM D2434 Permeability of Granular Soils (Constant Head)	Min. of 0.01 cm/s (14 in. per hour)	Lab or Field **
Compaction (Density)	ASTM D698 Compaction Using Standard Effort ASTM D2922 Compaction of Soil In Place by Nuclear Methods	To set criteria for ASTM D2922 Min. 95% Standard Proctor	Lab Field
Final Grade	ASTM F2157 Test method for Base Material Evenness	Less than 1/4 in. over 10 ft 6mm over 1m	Field

* Determination in the lab: It is necessary to seal the test ring to the base of the sample. The edges of the sample must also be sealed to prevent any water from flowing around rather than through the sample.

** Determination in the field: An exact seal is typically not attainable and the test is not as accurate/ reproducible due to the lateral flow of water and the problems of determining the areas through which the water is flowing.

TABLE 2 – TURF CHARACTERISTICS FOR TUFTED INFILL SYSTEMS (Typical for High School, Collegiate, and Professional Playing Fields)

Test Property	Method of Determination	STC Guidelines	Lab/Field Test
Manufacturer of System (name)	Manufacturer Declaration	Not Specified	N/A
Pile Fiber ID	Manufacturer Declaration	Not Specified	N/A
Primary Backing System ID	Manufacturer Declaration	Not Specified	N/A
Secondary Backing System ID	Manufacturer Declaration	Polyurethane/Latex/Fabrics	N/A
Pile (face weight)	ASTM D5848	Min. 30 oz./sq. yd. (2" product) Min. 38 oz./sq. yd. (2.5" product)	Lab
Primary Backing System Weight	ASTM D5848	Min. 5.5 oz./sq. yd.	Lab
Secondary Backing System Weight	ASTM D5848	Min. 16 oz./sq. yd.	Lab
Pile Height	ASTM D5823	Sport specific or as specified	Lab/Field
Pile Height above infill	Measurement	Must meet system specs	Lab/Field
Yarn Thickness	ASTM D3218	Min. 100 microns (slit film) Min. 130 microns (monofilament)	Lab tested
Yarn Denier	ASTM D1577	Min. 500 (nylon)	Lab tested
Grab Tear Strength	ASTM D5034	Min. 150 lbs.	Lab
Tuft Bind	ASTM D1335	>6.8 lbs. or 30N	Lab only
Flammability	ASTM D2859 Pill Burn	Passing result tested as installed	Lab
Color Uniformity	Visual	No significant change	Lab & Field

TABLE 3 – TURF CHARACTERISTICS FOR KNITTED TURF SYSTEMS

Test Property	Method of Determination	STC Guidelines	Lab/Field Test
Manufacturer of System (name)	Manufacturer Declaration	Not Specified	N/A
Pile Fiber ID	Manufacturer Declaration	Not Specified	N/A
Primary Backing System ID	Manufacturer Declaration	Not Specified	N/A
Secondary Backing System ID	Manufacturer Declaration	Glued: Acrylic Loose laid: Polyurethane, or Acrylic	N/A
Pile (face weight)	ASTM D5848	Min. 55 oz./sq. yd.	Lab
Primary Backing System Weight	ASTM D5848	Min. 8 oz./sq. yd.	Lab
Secondary Backing System Weight	ASTM D5848	Glued: Min. 3 oz./sq. yd. Loose laid: 1/4 in. (6mm) pre-coat & attached cushion weight combined is min. 50 oz./sq. yd.	Lab
Pile Height	ASTM D5823	Min. 0.5 in.	Lab/Field
Pile Height above infill	Measurement	N/A	Lab/Field
Yarn Denier	ASTM D1907	Min. 500 (nylon)	Lab
Yarn Thickness	ASTM D3218	Min. 100 microns PE Min. 50 microns PP	Lab
Grab Tear Strength	ASTM D5034	Min. 350 pounds	Lab
Tuft Bind	ASTM D1335	>6.8 lbs. or 30N	Lab
Flammability	ASTM D2859 Pill Burn	Passing result tested as installed	Lab
Relative Abrasiveness	ASTM F1015	Measurement	Lab
Color Uniformity	Visual	No significant changes	Lab & Field

TABLE 4 – TURF CHARACTERISTICS FOR TUFTED POLYPROPYLENE (PP), POLYETHYLENE (PE), OR NYLON SYSTEMS (NON-INFILL SYSTEMS)

Test Property	Method of Determination	STC Guidelines	Lab/Field Test
Manufacturer of System (name)	Manufacturer Declaration	Not Specified	N/A
Pile Fiber ID	Manufacturer Declaration	Nylon 6 or 6,6; PP, PE	N/A
Primary Backing System ID	Manufacturer Declaration	Not Specified	N/A
Secondary Backing: System ID	Manufacturer Declaration	Polyurethane	N/A
Pile (face weight)	ASTM D5848	Min. 48 oz./sq. yd.	Lab
Primary Backing Weight	ASTM D5848	Min. 6 oz./sq. yd.	Lab
Secondary Backing System Weight	ASTM D5848	Min. 16 oz./sq. yd.	Lab
Pile Height	ASTM D5823 or D6859	Min. 0.45 in.	Lab/Field
Pile Height above infill	Measurement	N/A	Lab/Field
Fiber Conditioning	Manufacturer Declaration	Texturized	N/A
Yarn Denier	ASTM D1907	Min. 500 Nylon	Lab
Yarn Thickness	ASTM D3218	Min. 75 microns PE Min. 50 microns PP	Lab
Yarn Elongation	ASTM D2256	N/A	Lab
Grab Tear Strength	ASTM D5034	Min. 150 pounds	Lab
Yarn Breaking Load (tensile strength)	ASTM D2256	Mfr. recommended spec	Lab
Tuft Bind	ASTM D1335	>6.8 lbs. or 30N	Lab
Flammability	ASTM D2859 Pill Burn	Passing result tested as installed	Lab
Color Uniformity	Visual	No significant changes	Lab & Field

TABLE 5 – INFILL PROPERTIES

Test Property	Method of Determination	STC Guidelines	Lab/Field Test
Material Identification	Manufacturer Declaration	Must meet system specifications	N/A
Grain Size (Particle Size)	ASTM D422 (soil) ASTM D5644 (rubber)	Must meet system specifications	Lab
Depth	Measurement from top of infill to surface of fabric	Must meet system specification at all locations 3/8 in. (±9mm)	Lab/Field
Flammability	ASTM D2859 Pill Burn	Passing result tested as installed	Lab
Color Uniformity	Visual	No significant changes	Lab/Field

TABLE 6 – SHOCK PAD LAYER PROPERTIES

Test Property	Method of Determination	STC Guidelines	Lab/Field Test
Material Identification	Manufacturer Declaration	Must meet system specifications	N/A
Mix Design	Manufacturer Declaration	Must meet system specifications	N/A
Drainage	ASTM F1551/DIN 18035-6 Water Permeability	Min. of 14 in. per hour	Lab/Field
Components Size Rubber / Stone (gravel)	ASTM F1508 Sieve Analysis	Must meet system specifications	Lab
Evenness	ASTM F2157: Test method for Base Material Evenness	Less than 1/4 in. over 10 ft. (6mm over 3m)	Field
Thickness	Measurement	Meet system spec at every point measured (+6mm/-0mm) (+1/4 in./-0) cushion layer	Lab/Field

TABLE 7 – PERFORMANCE GUIDELINES

The Synthetic Turf Council's Performance Guidelines are included by reference. For a copy of the *STC Guidelines for Synthetic Turf Performance*, please go to Guidelines and Professional Standards at http://www.syntheticturfcouncil.org.

▪ APPENDIX B – REFERENCE SPECIFICATIONS, TYPICAL (CHARTS 1-5)

A. The reference specifications noted in Appendix B are "typical" examples of minimums that are most commonly encountered and have fulfilled reasonable expectations for successful performance. Deviations from these minimums can be expected due to product innovations or quality upgrades and can be considered when properly justified in terms of their expected performance.
B. All tests prior to, during, or after installation are to be specifically listed and understood by all parties as to their execution and financial responsibility.
C. Environmental Conditions: Suitable weather conditions are important for the successful installation of the systems.
D. In the event of questionable conditions, the manufacturer's recommendation should be obtained to prevent the possible voiding of any warranties (particularly as it applies to adhesives).

CHART 1 – INFILLED SYNTHETIC TURF SPECIFICATION

TYPICAL MINIMUMS

FIBER:	
Material	PE, PP, Nylon 6, or Nylon 6.6
Denier	Must meet system specs
BACKING (primary/secondary):	
Weight primary	Not less than 5.5 oz. per sq. yd.
Weight secondary	Not less than 16 oz. per sq. yd.
Additional backings	Optional
FABRIC:	
Width	12 ft. to 15 ft.
Tuft bind	>6.8 lbs. or 30N
Pile height	Sport specific or as specified
Pile weight	Not less than 30 oz. per sq. yd. (must meet system specs)
Grab tear strength	Not less than 150 lbs.
Pill burn test	Passing results tested as installed
INFILL SYSTEM:	Depending on Manufacturer's recommendation, refer to definition section.
Depth of infill	Nominal, per Manufacturer's recommendation.
Impact Attenuation	Current industry standard for *g*-max is a maximum value of 200 at each test point. *g*-max values may vary from location to location on a playing surface. Such variances should be taken into account when setting maximum values. *g*-Max should be below 165 throughout the life of the field.
Water Permeability	Turf cushion layer – min. 10 in. per hour Base materials – min. 14 in. per hour

CHART 2 – KNITTED SYNTHETIC TURF SPECIFICATION – SHORT PILE

TYPICAL MINIMUMS

FIBER:	
Material	Nylon 6, Nylon 6.6, PE or PP
Denier	Min. 500 nylon
Thickness	Min. 75 microns PE or PP
Pile height	Sport specific or as specified
Pile weight	Not less than 55 oz. per sq. yd.
BACKING (primary/secondary):	
Ground yarn	Polyester multi-filaments
Weight primary	Not less than 8 oz. per sq. yd.
Acrylic	Not less than 3 oz. per sq. yd.
Polyurethane attached cushion	Optional
FABRIC:	
Width	Typically 15 ft.
Tuft bind	N.A.
Grab tear strength	Not less than 350 lbs.
Pill burn test	Passing results tested as installed
Total weight	Not less than 66 oz. per sq. yd. (without attached cushion)
SYSTEM:	
Impact Attenuation	Current industry standard for *g*-max is a maximum value of 200 at each test point. *g*-max values may vary from location to location on a playing surface. Such variances should be taken into account when setting maximum values. *g*-Max should be below 165 throughout the life of the field.
Water Permeability	Turf/cushion layer – min. 10 in. per hour Base materials – min. 14 in. per hour

CHART 3 – TUFTED SYNTHETIC TURF SPECIFICATION – SHORT PILE

TYPICAL MINIMUMS

FIBER:	
Material	PP, PE, or Nylon 6.6, Nylon 6
Denier/Thickness	Not less than 500 Nylon Not less than 75 microns thickness PE, and 50 microns thickness PP
Pile height	Sport specific or as specified
Pile weight	Not less than 48 oz. per sq. yd.
BACKING (primary/secondary):	
Woven PP/non-woven	Single or multiple
Weight	Not less than 6 oz. per sq. yd.
Scrape coat	Not less than 16 oz. per sq. yd.
Attached cushion	Not less than 32 oz. per sq. yd. (as required)
Secondary and/or cushion	
FABRIC:	
Width	12 ft. to 15 ft.
Tuft bind	>6.8 lbs. or 30N
Pill burn test	Passing results tested as installed
Grab tear strength	Not less than 150 lbs.
Total weight	Depending on individual construction
SYSTEM:	
Impact Attenuation	Current industry standard for *g*-max is a maximum value of 200 at each test point. *g*-max values may vary from location to location on a playing surface. Such variances should be taken into account when setting maximum values. *g*-Max should be below 165 throughout the life of the field.
Water Permeability	Turf/cushion layer – min. 10 in. per hour Base materials – min. 14 in. per hour

CHART 4 – SHOCK PAD – PREFABRICATED PAD SYSTEMS SPECIFICATION

TYPICAL MINIMUMS

Typical Requirement	Specification
Thickness	.375 in. ± 10%
Density	4.0 lbs./cu. ft. ± 10%
Weight	38 oz./sq. yd.
Width	4 ft.
25% Compression Resistance (ASTM D-1667)	10-12 psi
Tensile Strength (ASTM D-412)	Typically 75 psi
Elongation to Break (ASTM D-412)	Typically 125%

CHART 5 – SHOCK PAD – *IN SITU* SYSTEMS SPECIFICATION

TYPICAL RANGES

Thickness	35 mm	25mm	20mm
Density	2 lbs./cu. ft.	1.5 lbs./cu. ft.	1.2 lbs./cu. ft.
Weight	56 lbs./sq. yd.	40 lbs./sq. yd.	32 lbs./sq. yd.

Component	**SBR**	**Aggregate**	**PU Binder**
	1-5mm	1-3mm	
Percentages (all percentages by weight)	60-63%	30-32%	5-10%

Note: Typically the mix design is determined first, to satisfy the needs of the field in relation to its declared use. The mix design then will determine the weight, density, and thickness which should fall within the parameters indicated.

- SBR granules to be dust free, no elongated particles are allowed.
- Aggregate to be washed/clean, preferably round (pea gravel).
- Application to be performed by the use of continuous mixing device and suitable paving equipment.

▪ LEGEND – PROVIDERS OF SYNTHETIC TURF SYSTEMS

INDEPENDENT PROFESSIONAL AND CONSULTANT

Firms with legally registered or licensed design professionals who are officially designated as such as well as those with non-licensed practitioners or consultants that represent or advise the owner/end-user with surface and systems selection information, technical specifications, system designs, bid and construction documents, etc. Examples include:

- Landscape Architect
- Civil Engineer
- Consultant

BUILDER AND INSTALLER, INCLUDING SUB-BASE CONTRACTOR

Companies whose primary responsibility is installing synthetic systems either directly or indirectly through a subcontractor or distributor. Builders often have turn-key responsibility for the synthetic turf installation and provide the overall warranty for the installation. It is recognized that some companies in this category may be manufacturers. Examples include:

- Builder – Installer of sports fields (provides overall warranty on the installation)
- Builder – Installer of landscape and recreation synthetic grass (provides overall warranty on the installation)
- Installer – Installer of sports fields (does not provide product warranty)
- Installer – Installer of landscape and recreation synthetic grass (does not provide product warranty)
- Sub-base contractor (does not install synthetic turf)

MATERIAL MANUFACTURER OR SUPPLIER

Companies whose primary business is to provide materials or manufacturing services to the synthetic turf industry. These organizations do not provide turn-key installation of the synthetic turf systems nor provide the overall warranty for an installation. Examples include:

- Adhesives
- Components of Synthetic Turf Systems
- Infill of all types
- Drainage Systems
- Logos and Field Graphics
- Pigments and Masterbatches
- Shock Pads and Underlayments
- Synthetic Turf
- Yarn

SPECIALTY MANUFACTURERS AND SERVICE COMPANIES

Maintenance organizations, equipment manufacturers, professional service organizations, and other companies, typically independent, which sell their products and services primarily to the buyer or end-user. Examples include:

- Field Marking and Paint
- Field Removal
- Insurance - 3rd Party Warranty Ins., Bonding, & Other
- Irrigation Systems
- Maintenance Equipment
- Repairs and Maintenance
- Business & Scientific Consultant
- Protective Field Covers
- Sports Field Hardware & Equipment
- Testing Equipment
- Synthetic Turf and Yarn Extrusion Equipment

TESTING LABORATORIES

Independent testing organizations capable of testing synthetic turf and its components.

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SUPPLEMENT NO. 53

Incidence, Mechanisms, and Severity of Game-Related College Football Injuries on FieldTurf Versus Natural Grass

A 3-Year Prospective Study

Michael C. Meyers,* PhD, FACSM
From the Department of Health and Human Development, Montana State University, Bozeman, Montana

Background: Numerous injuries have been attributed to playing on artificial turf. More recently, FieldTurf was developed to duplicate the playing characteristics of natural grass. No long-term studies have been conducted comparing game-related collegiate football injuries between the 2 playing surfaces.

Hypothesis: Collegiate athletes do not experience any difference in the incidence, mechanisms, and severity of game-related injuries between FieldTurf and natural grass.

Study Design: Cohort study; Level of evidence, 2.

Methods: Twenty-four universities were evaluated over 3 competitive seasons for injury incidence, injury category, time of injury, injury time loss, player position, injury mechanism, primary type of injury, grade and anatomical location of injury, type of tissue injured, trauma (head, knee, and shoulder), and environmental factors.

Results: In sum, 465 collegiate games were evaluated for game-related football injuries sustained on FieldTurf or natural grass during 3 seasons. Overall, 230 team games (49.5%) were played on FieldTurf versus 235 team games (50.5%) played on natural grass. A total of 2253 injuries were documented, with 1050 (46.6%) occurring during play on FieldTurf, and 1203 (53.4%) on natural grass. Multivariate analysis per 10 team games indicated significantly lower total injury incidence rates, $F(3, 2249) = 3.468$, $P = .016$, $n - \beta = 0.778$, on FieldTurf, 45.7 (95% confidence interval [CI], 44.2-46.3), versus natural grass, 51.2 (95% CI, 49.8-51.7). Significantly lower minor injury incidence rates, 38.0 (95% CI, 36.9-38.5) versus 39.9 (95% CI, 39.1-40.0, $P = .001$), substantial injury incidence rates, 5.0 (95% CI, 4.3-5.6) versus 7.2 (95% CI, 6.6-7.7, $P = .020$), and severe injury incidence rates, 2.7 (95% CI, 2.1-3.3) versus 4.1 (95% CI, 3.5-4.1; $P = .049$), were documented on FieldTurf versus natural grass, respectively. Multivariate analyses also indicated significantly less trauma on FieldTurf when comparing injury time loss, injury situation, grade of injury, injuries under various field conditions, and temperature. No significant differences in head, knee, or shoulder trauma were observed between playing surfaces.

Conclusion: FieldTurf is in many cases safer than natural grass. It must be reiterated, however, that the findings of this study may be generalizable to only this level of competition. Because this study is still in the early stages, investigation is ongoing.

Keywords: artificial surface; knee; head; trauma

For more than 40 years, numerous studies have attributed a greater risk and incidence of articular and concussive trauma to playing on artificial turf when compared with natural grass.[21,32,55] Over the past decade, however, a new generation of synthetic surface was developed to duplicate the playing characteristics of natural grass: FieldTurf (Montreal, Quebec, Canada) is composed of a polyethylene fiber blend stabilized with a graded silica sand and cryogenically ground rubber infill. Although FieldTurf has been recommended as a viable option to natural grass in the prevention of high school football injuries,[40] research into its long-term effects on injuries at the collegiate level, during actual game conditions over several seasons of competition, has not been published in the scientific literature.

More than 1 million athletes play competitive football.[23,43] The number of knee surgeries is rising, and their

*Address correspondence to Michael C. Meyers, PhD, Adjunct Professor, Department of Health and Human Development, 139 Reid Hall, POB 172940, Montana State University, Bozeman, MT 59717-2940 (e-mail: meyersgroupinc@gmail.com).

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cost plus rehabilitation is reaching into the millions of dollars each year.[12,20,24,35,48,54] Coupled with this is the psychological trauma and setbacks in training typically experienced by athletes after a significant injury.[41] As such, efforts to address ways to minimize predisposition to injury are warranted. Therefore, the purpose of this study was to quantify the incidence, mechanisms, and severity of game-related collegiate football injuries on FieldTurf versus natural grass. It was hypothesized that collegiate athletes would not experience any difference in the incidence, mechanisms, and severity of game-related injury between FieldTurf and natural grass.

MATERIALS AND METHODS

Population

Twenty-four universities, classified Division IA (FBS) by the National Collegiate Athletic Association, were evaluated for game-related football injuries sustained on FieldTurf and natural grass during a 3-year period (2006-2008). School selection was based on the availability of both playing surfaces during the competitive season, uniformity of sport skill level, and the presence of a full-time certified athletic training (ATC) staff, thereby minimizing the potential for injury reporting bias.[11,49] The study started with 11 universities over the first year and added 13 in year 2, yielding a total of 520 seasonal games. With the exception of deleting games played on other artificial surfaces (n = 55), selection bias was avoided by reporting all remaining games and subsequent injuries on either FieldTurf or natural grass. This resulted in a total of 465 games over the 3-year period played on either FieldTurf (n = 230) or natural grass (n = 235).

Various stadiums using FieldTurf were used by all 24 schools during home and away games involving conference play in the Atlantic Coast, Big 12, Big East, Conference USA, Mountain West, Western Athletic Conference, and Pac-10. FieldTurf surfaces were considered high-quality surfaces by the ATCs. Different natural grass fields were used across the same geographical region, with similar quality and environmental influences. All teams had the opportunity to practice on either FieldTurf or natural grass.

To quantify the history and potential influence of prior injuries, all athletes underwent preparticipation physical examinations under the care of their team physicians/orthopaedic surgeons. Criteria for exclusion included (1) any known preexisting congenital or developmental factor that predisposed an athlete to potential injury and (2) the acknowledgment, complaint, or observed evidence of any medical or orthopaedic problem severe enough to compromise an athlete's performance or endanger his health as determined by self-response, medical history, and interview.[40]

Procedures

Based on paradigms suggested in prior research,[4,19,33,39,63] this research used a multifactorial approach that encompassed teams playing on both surfaces during the same period. This approach provided several advantages, including gaining a greater comparison of the nuances of each surface's influence on injury, avoiding limitations in data collection (eg, seasonal variation, subject randomization by surface), and minimizing difficulties that former studies have had in analyses and interpretation of findings.[4,47] For this prospective cohort study, a 2-sided single-page injury surveillance form was developed, based on criteria recommended and established in the literature (available as an appendix at http://www.ajs.sagepub.com/supplemental).[13,25,33,40,42,44] The form includes the following: athletic identification number; athletic trainer; date of injury; athlete weight; university; type of playing surface; surface quality; temperature at game time; year and skill level of athlete; where the injury occurred; weather and field conditions; injury category; time of injury; injury classification; injury time loss; position played at time of injury; injury situation; injury mechanism; personnel determining the injury; injury site location; principle body part; primary type of injury; grade of injury; occurrence of external bleeding; injury because of illegal action; head, eye, knee, shoulder, and thoracic/abdominal diagnosis; surgical intervention and time; and musculoskeletal, joint, or organ location of injury. The injury surveillance form was e-mailed to the head ATCs during the summer before the start of the football season. Communication was maintained to discuss potential concerns and ensure accuracy of collection, comprehensiveness of information, and ease of application.

The respective ATCs for each university were approached because of their daily interaction with the athletes and coaches during and after sport trauma and because of their expertise in injury recognition.[11,40] During the summer before the football season, all ATCs were provided with an overview of the purpose, procedures, benefits, time demands, and importance of the study. They were also provided with copies of the injury surveillance form and detailed instructions for completion to avoid the potential for performance and detection biases.[47,49] After full explanation, all ATCs appeared enthusiastic and agreed to participate in the data collection. The protocol was approved by the institutional review board at the university in which the study was based, and it was conducted in accordance with the guidelines for use of human participants as stipulated by the American College of Sports Medicine.[2]

All regular season conference and nonconference games and postseason bowl games were included. Injury data were recorded after game completion, with support from ATC notes to avoid lapse of memory leading to inaccuracy or response distortion.[40,47] All game-related injuries were evaluated by the attending head athletic trainer and team physicians on-site and, subsequently, in the physician's office when further follow-up and treatment were deemed necessary. Any sport trauma that occurred toward the end of the competitive schedule was monitored beyond the player's specific season to determine date of recovery and functional return to play.[22,40]

Completed injury surveillance forms were faxed to us within 5 working days after a game and were entered into the database before the next game. A follow-up telephone visit was used to obtain any additional information

pertaining to any changes or additions in diagnosis, treatment, or time to return to play. To avoid the potential for on-the-field detection bias,[49] a single-blind outcome approach was maintained throughout the study period, with total data collection, compilation, and analyses limited to the data coordinator.

Definitions

Although any definition of injury and level of trauma lacks universal agreement and has its shortcomings,[11,24,44,47] this study attempted to define injury based on a combination of functional outcome, observation, and treatment.[11,22,40,44,60] A *reportable injury* was thus defined as any game-related football trauma that resulted in (1) an athlete missing all or part of a game, (2) time away from competition, (3) any injury reported or treated by the athletic trainer or physician, and (4) all cranial/cervical trauma reported.[24,40] Although some authors have recommended omitting minor injuries,[44,47] others have expressed a need to quantify and track these typically overlooked minor traumas to avoid underreporting of injury and to monitor those that may turn into chronic or overuse problems.[14,25,42,66] Prior studies have also revealed that 42% to 60% of competitive trauma results in minimal time loss and medical cost.[24,40,63] Therefore, a definition that included functional outcome, observation, and treatment on all injuries might more clearly quantify the unique nuances or trauma observed with each playing surface and so reduce the individual and player bias that allegedly influences injury reporting based solely on time loss.[40,47]

Injury time loss was based on the number of days absent from practice or game competition and was divided into 0, 1-2, 3-6, 7-9, 10-21, and 22 days or more of recovery time. Not surprisingly, a review of the literature revealed high subjectivity in the determination of what constitutes moderate or severe injury. Whereas any injury resulting in time loss of approximately 7 to 28 days has been considered moderate trauma and a time loss of $\geq$21 days has been defined as severe,[16,26,27,63] others have defined severe injury as $\geq$28 days before return to play[15] or any injury resulting in $\geq$7 days of time loss.[25,33,46,53] Furthermore, what constitutes a moderate injury in one athlete (eg, elbow injury in an offensive lineman) may be considered severe when diagnosed in another (eg, the throwing arm of a quarterback).[5,53] Therefore, as previously described, any trauma that required 0 to 6 days of time loss was defined as a *minor injury*; an injury that required 7 to 21 days of time loss, resulting in the athlete's being unable to return to play at the same competitive level, was a *substantial injury*; and trauma that required 22 or more days of time loss was a *severe injury*.[40] The delineation and subsequent analysis of minor, substantial, and severe injury served to minimize potential time loss bias.[11,40,60]

Injury category was quantified by player-to-player collision, player-to-turf collision, injuries attributed to shoe-surface interaction during player contact, injuries attributed to shoe-surface interaction without player contact, and muscle-tendon overload. Time of injury by pregame and game quarter of play was documented to delineate the influence of fatigue over time from the potential surface influence on injury occurrence.[40,63,66]

Acute trauma was delineated from recurrent and overuse injury according to criteria previously published,[36,39,40,62] with acute trauma linked to an incidence that occurred during a competitive game versus repetitive exposure resulting in symptoms and injury to the same location during the season (recurrent). An *overuse injury* was defined as repetitive exposure resulting in trauma and sequelae with no definitive onset.[39,66]

To enhance optimal cell size and interpretation, the 23 player positions were condensed and analyzed by offense, defense, and special teams, as well as by power and skill positions (quarterback, backfield, offensive line, tight end, receiver, defensive line, linebacker, secondary).[8] *Mechanism of injury* was defined as that occurring while a player was blocked above or below the waist, tackled above or below the waist, blocking, tackling, impacting with the playing surface, stepped on, fallen on or kicked, sprinting or running with no player contact, catching/blocking a pass, clipped, experiencing heat illness, or injured from overuse. *Injury situation* was defined as trauma occurring during a specific play or event, such as warm-up, rushing, passing, pass catching, pass protection, pass rush, pass defense, kickoff return, point after touchdown, field goal, kickoff, punting, punt return, or fumble recovery.

To optimize analyses, primary type of injury was combined into the following categories: surface/epidermal (abrasion, laceration, puncture wound), contusion, concussion, inflammation (bursitis, tendinitis, fasciitis, synovitis, capsulitis, apophysitis), ligament sprain, ligament tear, muscle strain/spasm, muscle tear, cartilage tear, tendon strain, hyperextension, neural (burner, brachial plexus), subluxation/dislocation, and fracture (standard, epiphysial, avulsion, stress, osteochondral). Injuries were also defined according to grade (1, 2, or 3). Anatomical location of injury was combined from 40 physical areas and analyzed by type of trauma (cranial/cervical, upper extremity, thoracic, and lower extremity) and further analyzed by type of tissue injured (bone, joint, muscle, neural, other). Cranial/cervical trauma included grade 1 to 3 concussion, hematoma, post-concussion and second-impact syndromes, neurological sequelae (eg, stingers/burners, transient quadriplegia), vascular or dental injury, or associated fractures, sprains, and strains.[40] Neural trauma was restricted to any injury involving only concussion, associated syndromes, and neurological sequelae. Because of growing concerns addressing excessive head, knee, and shoulder trauma in football,[11,20,40,47] these areas were identified for further analyses (see Appendix 1, available in the online version of this article at http://ajs.sagepub.com/supplemental/).

Although studies have associated a greater rate of injury during competition under both dry and normal surface conditions,[3,52] there has been limited information on factors such as weather and the effect of playing under surface conditions that influence injury frequency.[1,3,20,31,40,61] Therefore, environmental factors, such as field conditions and temperature, were obtained before game time by each team's respective ATC and/or through the local airport climatic data center to ascertain the potential influence on

injury from changes in weather and surface conditions throughout the season.[45]

Statistical Analyses

Because of variations in the frequency of injury within several categories potentiating inadequate cell size, statistical power, and limitations on analysis, some data were combined during the 3-year period based on prior recommendations in the literature.[33,40,44] This step resulted in the following categories: injury category, time of injury, injury classification, injury time loss, position played at time of injury, injury mechanism, injury situation, injury site location, primary type of injury, grade of injury, anatomical location of injury, type of tissue injured, head diagnosis, knee diagnosis, shoulder diagnosis, specific lower extremity joint and muscle trauma, and environmental factors. Tabular-frequency distributions were computed for data in each category using SPSS 15.0. For ease of interpretation, the percentages of total injuries within each category that occurred on the playing surface were calculated, and 95% confidence intervals (95% CIs) were determined as described elsewhere.[51]

Because most universities schedule a similar number of games each season, exposure to injury was defined in terms of team games, as previously recommended.[40,63] Based on this definition, and to approximate a standard season, injury incidence rate (IIR) was expressed using (1) injuries per 10 team games = (number of injuries/number of team games) $\times$ 10 and (2) injuries per team game = number of injuries/number of team games.

To achieve a more thorough understanding beyond traditional frequency analyses and to eliminate the possibility of irrelevant sources of error,[37,39] following the season, data were numerically recoded, grouped by playing surface (FieldTurf, natural grass), and subjected to multivariate analyses of variance and Wilks lambda criteria using general linear model procedures.[37] Data screening indicated no violations of multivariate normality, linearity, outliers, homogeneity of variance, multicollinearity, or singularity.[59] When significant main effects were observed, univariate post hoc procedures were performed within each dependent variable based on the total percentage of injuries reported on each playing surface. An experiment-wise type I error rate of .05 was established a priori, and least squared means procedures were required because of the uneven number of observations on which to compare differences between variables. Statistical power analyses ($n - \beta$; n-size calculations) were performed at the P value selected to establish significance in this study.

RESULTS

Injury Incidence

A total of 465 collegiate games were evaluated for game-related football injuries sustained on FieldTurf and natural grass during 3 seasons (Table 1). Overall, 230 team games were played on FieldTurf (49.5%) versus 235 team games played on natural grass (50.5%). A total of 2253 injuries were documented, with 1050 (46.6%) occurring during play on FieldTurf as compared to 1203 (53.4%) on natural grass.

When IIRs were compared between types of playing surface, there was a significant main effect, $F(3, 2249) = 3.468$, $P = .016$, $n - \beta = .778$, between surfaces by injury level. Total injuries per 10 team games, 45.7 (95% CI, 44.2-46.3) versus 51.2 (95% CI, 49.8-51.7), and injuries per team game, 4.6 (95% CI, 4.4-4.6) versus 5.1 (95% CI, 5.0-5.2), were documented on FieldTurf versus natural grass, respectively. When minor trauma (injuries requiring 0 to 6 days of rehabilitation) was compared, a significantly lower incidence of minor injuries ($P = .0001$) per 10 team games, 38.0 (83.3%; 95% CI, 36.9-38.5) versus 39.9 (78.0%; 95% CI, 39.1-40.0), and injuries per team game, 3.8 (95% CI, 3.7-3.9) versus 4.0 (95% CI, 3.9-4.0), was documented on FieldTurf versus natural grass, respectively. When substantial trauma (injuries requiring 7 to 21 days of injury rehabilitation) was compared between type of playing surface, a significant lower incidence of substantial injuries ($P = .020$) per 10 team games, 5.0 (95% CI, 4.3-5.6) versus 7.2 (95% CI, 6.6-7.7), and injuries per team game, 0.50 (95% CI, 0.43-0.56) versus 0.72 (95% CI, 0.66-0.77), was documented on FieldTurf versus natural grass, respectively. When severe trauma (injuries requiring 22 or more days of injury rehabilitation) was compared between type of playing surface, a significantly lower incidence of severe injuries ($P = .049$) per 10 team games, 2.7 (95% CI; 2.1-3.3) versus 4.1 (95% CI, 3.5-4.7), and injuries per team game, 0.27 (95% CI, 0.21-0.33) versus 0.41 (95% CI, 0.35-0.47), was documented on FieldTurf versus natural grass, respectively.

The majority of trauma comprised acute injuries on both FieldTurf (88.7%; IIR = 40.5; 95% CI, 39.5-40.8) and natural grass (88.0%; IIR = 45.1; 95% CI, 43.6-45.7). Only 119 of 1050 injuries reported on FieldTurf (11.3%; IIR = 5.2; 95% CI, 4.5-5.8) and 144 of 1203 reported on natural grass (12.0%; IIR = 6.1; 95% CI, 5.5-6.7) were classified as recurrent trauma or complications from prior injury. As expected, upperclassmen received the majority of trauma on both playing surfaces. On FieldTurf, 330 injuries occurred to seniors (31.4%; IIR = 14.3; 95% CI, 13.5-15.0), 371 to juniors (35.3%; IIR = 16.1; 95% CI, 15.3-16.7), 240 to sophomores (22.9%; IIR = 10.4; 95% CI, 10.0-10.8), and 109 to freshmen (10.4%; IIR = 4.7; 95% CI, 4.1-5.4). On natural grass, 417 injuries were reported among seniors (34.7%; IIR = 17.7; 95% CI, 17.0-18.2), 378 among juniors (31.4%; IIR = 16.1; 95% CI, 15.2-16.7), 309 among sophomores (25.7%; IIR = 13.1; 95% CI, 12.4-13.8), and 99 among freshmen (8.2%; IIR = 4.2; 95% CI, 3.6-4.9).

Head, Knee, and Shoulder Trauma

As shown in Appendix 1, there was no significant main effect, $F(5, 2247) = 0.871$, $P = .500$, $n - \beta = .316$, between surfaces by head injury when combined by all sources of

TABLE 1
Incidence of Game-Related Collegiate Football Injuries Between FieldTurf and Natural Grass[a]

Variable	FieldTurf	IIR	95% CI	Natural Grass	IIR	95% CI	Total or Mean
Team games evaluated							
n	230			235			465
%	49.5			50.5			100.0
All injuries							
n	1050	45.7	44.2-46.3	1203	51.2	49.8-51.7	2253[b]
%	46.6			53.4			100.0
Per 10 games	45.7			51.2			48.5
Per game	4.57			5.12			4.85
Minor injuries							
n	875	38.0	36.9-38.5	938	39.9	39.1-40.0	1813[c]
%	83.3			78.0			80.5
Per 10 games	38.0			39.9			39.0
Per game	3.80			3.99			3.90
Substantial injuries							
n	114	5.0	4.3-5.6	169	7.2	6.6-7.7	283[d]
%	10.9			14.0			12.6
Per 10 games	5.0			7.2			6.1
Per game	0.50			0.72			0.61
Severe injuries							
n	61	2.7	2.1-3.3	96	4.1	3.5-4.7	157[e]
%	5.8			8.0			7.0
Per 10 games	2.7			4.1			3.4
Per game	0.27			0.41			0.34

[a]IIR, injury incidence rate; CI, confidence interval. Injuries per 10 team games = (number of injuries/number of team games) × 10. Injuries per team game = number of injuries/number of team games. Minor injury, 0 to 6 days of injury time loss; substantial injury, 7 to 21 days; severe injury, 22 or more days.
[b]$P = .016$.
[c]$P = .001$.
[d]$P = .020$.
[e]$P = .049$.

trauma, as well as no significant main effect, $F(10, 2242) = 0.461$, $P = .916$, $n - \beta = .246$, between surfaces by knee injury. A similar nonsignificant main effect, $F(7, 2245) = 1.543$, $P = .148$, $n - \beta = .653$, between surfaces by shoulder injury was also observed.

Injury Category

As shown in Appendix 2 (available at http://ajs.sagepub.com/supplemental/), multivariate analysis indicated no significant playing surface effect by injury category, $F(5, 2247) = 0.494$, $P = .781$, $n - \beta = .187$. Confidence intervals, however, indicated a lower incidence of injuries attributed to player-to-player collision ($P = .784$) on FieldTurf (54.3%; IIR = 24.8; 95% CI, 23.7-25.4) versus natural grass (54.9%; IIR = 28.0; 95% CI, 27.1-28.5), as well as a lower incidence of injuries attributed to shoe-surface interaction during contact ($P = .520$) on FieldTurf (21.8%; IIR = 10.0; 95% CI, 9.8-10.0) versus natural grass (22.9%; IIR = 11.7; 95% CI, 11.1-12.3).

Time of Injury

No significant main effect between playing surface was observed across time of injury, $F(5, 2247) = 0.833$, $P = .526$, $n - \beta = .303$. Confidence intervals (Appendix 2), however, indicated a lower incidence of injuries occurring during the second quarter of play ($P = .186$) on FieldTurf (28.3%; IIR = 12.9; 95% CI, 12.2-13.5) versus natural grass (30.8%; IIR = 15.8; 95% CI, 14.9-16.4), as well as a lower incidence of injuries occurring during the third quarter ($P = .609$) on FieldTurf (28.9%; IIR = 13.2; 95% CI, 12.4-13.8) versus natural grass (29.8%; IIR = 15.3; 95% CI, 14.4-15.9). Incidence rates for both FieldTurf and natural grass surfaces revealed that a limited number of injuries occurred during the pregame, increased from the first to second quarter, but declined from the third to the fourth quarter of play.

Injury Time Loss

Findings indicated a significant playing surface effect by injury time loss, $F(5, 2247) = 2.480$, $P = .030$, $n - \beta = .783$, with subsequent post hoc analysis (Appendix 2) revealing a significantly lower incidence of injuries resulting in 7- to 9-day time loss ($P = .017$) on FieldTurf (6.3%; IIR = 2.9; 95% CI, 2.3-3.5) versus natural grass (9.0%; IIR = 4.6; 95% CI, 4.0-5.2). A significantly lower incidence of injuries resulting in 22 days or more of time loss ($P = .044$) was also reported on FieldTurf (5.8%; IIR = 2.7;

95% CI, 2.1-3.3) versus natural grass (8.0%; IIR = 4.1; 95% CI, 3.5-4.7).

Position Played at Time of Injury

Although confidence intervals indicated lower incidences of offensive, defensive, and backfield injuries on FieldTurf, from a multivariate standpoint, no significant playing surface effect was observed between surfaces by overall player position (offense, defense, special teams), $F(2, 2250) = 0.300$, $P = .741$, $n - \beta = .098$, or by skill position, $F(9, 2243) = 0.538$, $P = .848$, $n - \beta = .271$.

Injury Mechanism and Situation

Although no significant main effect, $F(12, 2240) = 1.091$, $P = .363$, $n - \beta = .646$, between surfaces by injury mechanism was observed, there was a significant main effect, $F(14, 2238) = 2.170$, $P = .007$, $n - \beta = .971$, between surfaces by injury situation. As shown in Appendix 3 (available at http://ajs.sagepub.com/supplemental/), post hoc analyses indicated a significantly lower incidence of injuries occurring during rushing plays ($P = .040$) on FieldTurf (34.8%; IIR = 15.9; 95% CI, 15.0-16.5) versus natural grass (34.6%; IIR = 17.7; 95% CI, 16.9-18.2), as well as a significantly lower incidence of pass defense injuries ($P = .023$) on FieldTurf (14.2%; IIR = 6.5; 95% CI, 5.8-7.1) versus natural grass (17.7%; IIR = 9.1; 95% CI, 8.6-9.4). Further analyses revealed significantly lower incidence of trauma reported during punting ($P = .020$) on FieldTurf (1.0%; IIR = 0.5; 95% CI, 0.3-0.8) versus natural grass (2.3%; IIR = 1.2; 95% CI, 0.8-1.7), as well as a significantly lower number of injuries reported following pile-on ($P = .011$) on FieldTurf (0.3%; IIR = 0.1; 95% CI, 0.0-0.4) when compared to natural grass (1.2%; IIR = 0.6; 95% CI, 0.4-1.0).

Primary Type of Injury

As shown in Appendix 4 (available at http://ajs.sagepub.com/supplemental/), a significant main effect, $F(14, 2238) = 1.771$, $P = .042$, $n - \beta = .907$, by primary type of injury was noted between the 2 surfaces, with subsequent post hoc analysis revealing a significantly lower incidence of ligament tears ($P = .024$) reported on FieldTurf (2.7%; IIR = 1.2; 95% CI, 0.9-1.7) versus natural grass (4.6%; IIR = 2.3; 95% CI, 1.9-2.9). A significantly lower incidence of muscle tears ($P = .002$) was also reported on FieldTurf (0.3%; IIR = 0.1; 95% CI, 0.0-0.4) when compared to natural grass (1.7%; IIR = 0.9; 95% CI, 0.6-1.3).

Grade and Anatomical Location of Injury

There was a significant main effect, $F(2, 2250) = 12.337$, $P = .0001$, $n - \beta = .996$, between surfaces by injury grade (Appendix 4). A significantly lower incidence of second-degree injuries ($P = .0001$) was reported on FieldTurf (13.8%; IIR = 6.3; 95% CI, 5.7-6.9) versus natural grass (19.5%; IIR = 10.0; 95% CI, 9.8-10.0), as well as a significantly lower incidence of third-degree injuries ($P = .007$) on FieldTurf (8.9%; IIR = 4.0; 95% CI, 3.4-4.7) versus natural grass (12.4%; IIR = 6.3; 95% CI, 5.7-6.9)

In regard to location of injury, there was no significant main effect, $F(3, 2249) = 1.675$, $P = .170$, $n - \beta = .442$, between surfaces. Confidence intervals, however, indicated a lower incidence of upper extremity injuries ($P = .045$) reported on FieldTurf (30.4%; IIR = 13.9; 95% CI, 13.1-14.5) versus natural grass (34.2%; IIR = 17.5; 95% CI, 16.7-18.0).

Type of Tissue Injured

There was no significant main effect, $F(5, 2247) = 0.559$, $P = .732$, $n - \beta = .208$, between surfaces by tissue type. Confidence intervals, however, indicated a lower incidence of joint injuries ($P = .969$) reported on FieldTurf (46.2%; IIR = 21.1; 95% CI, 20.3-21.6) versus natural grass (46.1%; IIR = 23.6; 95% CI, 22.6-24.2), as well as a lower incidence of muscle injuries ($P = .997$) reported on FieldTurf (36.7%; IIR = 16.7; 95% CI, 15.9-17.3) versus natural grass (36.7%; IIR = 18.8; 95% CI, 18.1-19.1). When lower extremity joint trauma was analyzed, this study found a significant playing surface effect, $F(7, 224 = 2.310$; $P = .024$, $n - \beta = .852$, involving a significantly higher incidence ($P = .001$) of distal tibiofibular ligament sprains on natural grass (12.9%; IIR = 1.8; 95% CI, 1.4-2.4) when compared to FieldTurf (4.7%; IIR = 0.6; 95% CI, 0.4-1.0).

Environmental Factors

The attempt to quantify environmental conditions at time of injury revealed that the majority of injuries occurred during dry weather (see Appendix 5, available at http://ajs.sagepub.com/supplemental/). In regard to field conditions, there was a significant main effect, $F(2, 2249) = 5.450$, $P = .001$, $n - \beta = .939$, between surfaces, with a significantly lower incidence of injuries during no precipitation–dry field conditions ($P = .003$) reported on FieldTurf (86.3%; IIR = 39.4; 95% CI, 38.4-39.6) versus natural grass (81.0%; IIR = 41.4; 95% CI, 40.3-42.0), as well as a significantly lower incidence of injuries during no precipitation–wet field conditions ($P = .0001$) reported on FieldTurf (3.9%; IIR = 1.8; 95% CI, 1.3-2.3) versus natural grass (8.5%; IIR = 4.3; 95% CI, 3.7-5.0).

When analyzing data by cold days (eg, $\leq$69°F) as compared to hot days (eg, $\geq$70°F) as suggested by others,[40,45] there was a significant main effect, $F(1, 2251) = 82.360$, $P = .0001$, $n - \beta = 1.000$, between surfaces by environment temperature. A significantly higher incidence of injuries during cold days ($P = .0001$) was reported on FieldTurf (54.3%; IIR = 24.8; 95% CI, 23.7-25.4) versus natural grass (35.4%; IIR = 18.1; 95% CI, 17.4-18.6). On hot days, a significantly lower incidence of injuries ($P = .0001$) was also reported on FieldTurf (45.7%; IIR = 20.9; 95% CI, 20.2-21.3) versus natural grass (64.6%; IIR = 33.1; 95% CI, 31.9-33.7).

DISCUSSION

The purpose of this prospective cohort study was to quantify the incidence, mechanisms, and severity of game-related collegiate football injuries on FieldTurf versus natural grass. It was hypothesized that collegiate athletes would not experience any difference in the incidence, mechanisms, and severity of game-related injury between FieldTurf and natural grass. Although similarities did exist between FieldTurf and natural grass, unique differences in sport trauma were observed between the 2 playing surfaces.

Injury Incidence

Over the 3-season study, 2253 game-related injuries, or 31.3 injuries per university per season, were recorded among 24 universities competing on both surfaces, indicative of the athletic speed, strength, and subsequent other opportunities for trauma at the collegiate level of competition, when compared to 2.4 to 15.7 injuries per high school per season reported in prior studies.[5,9,40] The incidence of acute injury (88.3%) was similar to findings in earlier studies, ranging from 72% to 94%.[40,47] The incidence of substantial and severe trauma recorded in this study was similar to seasonal trauma reported elsewhere in football but in contrast to nonsignificant surface differences in the severity of trauma in soccer, which may be a function of the level of trauma/impact potential between the 2 sports.[9,15,16,40,56] Although the large variation in injury definition among these studies prevents an accurate comparison, both the total number and the number of minor, substantial, and severe injuries recorded in this study reflect the typical level of trauma observed at the collegiate level of play.

In addition to acute injury, repetitive or recurrent trauma is considered a major contributor to future trauma.[18,21,27,40,65] The incidence of recurrent cases over 3 seasons in this study ranged from 11.3% on FieldTurf to 12.0% on natural grass, higher than the 4.8% previously reported on FieldTurf at the high school level[40] but similar to the 13% to 17% of recurrent trauma reported in collegiate and professional high-contact field sports during a single season[17,62] and substantially lower than the 57% of prior injury recently reported in high school football on natural grass over a 3-year period.[27] Whether recurrent trauma was observed over the same surface in all studies is not known. The increased interest but paucity of studies that address recurrent trauma prevents further discussion at the collegiate level of play.

The higher incidence of injury to upperclassmen on both surfaces is solely attributed to greater playing exposure and subsequent predisposition to injury typically observed at this level of play, in which lower classmen receive less playing time.[27,40] With regard to foul play, the incidence (0.5%) of injury attributed to illegal action was negligible. This is lower than the 0.8% to 5.7% occurrence reported in prior collegiate and high school football[24,40] but in far contrast to the 25% to 62% reported among other intercollegiate and senior sports.[14,46,67] In summary, the lower rate of overall injury documented on FieldTurf may be attributed to the lower incidence of substantial and severe trauma on the artificial surface when compared to natural grass.

Head, Knee and Shoulder Injuries

There were no significant differences in head, knee, or shoulder trauma between the 2 surfaces. But when compared with that of high school football studies,[11,40] the greater incidence of first-degree and total concussions combined, as well as the greater incidence of anterior cruciate ligament–involved trauma, reiterates the level of severe trauma observed during collegiate competition on both surfaces (Appendix 1). The nonsignificant level of traumatic injury on FieldTurf is consistent with prior findings indicating similar nonsignificant playing surface effect.[16,56] These findings are in contrast to prior studies indicating a higher incidence of severe injury on artificial surfaces,[20,53] a reflection of earlier synthetic materials as opposed to the newer generation of artificial surfaces being installed today.

Injury Category

Results of this study indicate no significant differences between playing surfaces across injury categories. Although prior efforts have noted a greater incidence of muscle-tendon overload injuries on FieldTurf during high school games and contributing findings as a function of faster play on a more compliant, elastic surface than that observed with natural grass,[21,40] this was not observed at this level of competition. Others have reported similar nonsignificant findings between artificial and natural grass surfaces.[15,16,56] As shown in Appendix 2, any univariate differences in player-to-player collisions and shoe-surface interaction during player contact between FieldTurf and natural grass were attributed to total injuries incurred rather than surface influence (P = .520-.784).

Time of Injury

Increasing fatigue over time and declines in available energy substrate and coordination predispose an athlete to injury.[62,65] The nonsignificant differences within and between playing surfaces in this study indicated minimal influence on injury incidence from pregame through the fourth quarter of play. As previously noted, the acute differences in the composition and quality of surfaces may have influenced the type and severity of trauma but did not affect the time of injury observed over the 3-season period. Again, univariate differences in second- and third-quarter trauma may be attributed to total injuries as opposed to actual surface differences (P = .186-.609). Findings may also be reflective of the score and subsequent play calling of coaches.[5,6,27,40]

Injury Time Loss

As similarly noted when discussing severity of injury, the polyethylene nature of FieldTurf, promoted as a nonabrasive

surface with a natural-earth feel, resulted in a significantly lower incidence of minor injuries requiring 0 to 6 days of time loss. Of primary concern is the significantly greater incidence of injuries ranging from 7 to 9 days of time loss and 22 days or more of time loss associated with competing on natural grass. In this study, increased incidence of injury resulting in extensive time loss ($\geq$22 days) is consistent with prior findings on natural grass at the high school level of competition.[40] Whether these findings with the natural grass surface are a function of decreasing turf quality with high temperatures (Appendix 5), lower surface compliance, and a higher coefficient of restitution observed following noncontact injuries on natural grass,[13,51] overuse because of multipurpose use, or simply the lack of resiliency of natural grass as the season progresses, is not clear and is beyond the control of this study.

Position Played at Time of Injury

Whether data were grouped by generalized positions (eg, offense, defense, special teams) or by specific skill positions as described by others,[18,19] multivariate analyses indicated no significant effect of playing surface on position played at the time of injury. Although prior studies have expressed concerns with the greater impact forces and incidence of injuries among special team, offensive, defensive, and offensive backfield players while competing on artificial surfaces,[21,40] this study did not support those concerns. Unfortunately, at this time, the limited frequency of injury among some positions (eg, quarterback, tight end) may have prevented further in-depth analyses and discussion of potential injury differences and position susceptibility.

Injury Mechanism and Situation

Prior authors have surmised that the more consistent artificial composition enhances the speed of the game[5,40] but allows for greater opportunity for injury owing to overextension and greater fatigue potential of muscles and a greater rate of acceleration, speed, and torque.[36,40,55] Nonetheless, there were no significant differences in injury mechanism between playing surfaces in this study (Appendix 3). In regard to the injury situation, the significantly lower incidence of injury on FieldTurf versus natural grass observed in situations involving rushing, pass defense, punting, and pile-on may be simply influenced by the quality of the playing surface or by several factors noted in the literature.[4,7,18,32,36,39,40,65] Risk factors repeatedly mentioned in the literature have included pivoting, change of direction, direct contact with an opposing player, deceleration, unfortunate mishaps (eg, moving pileup), or being jolted during an uncontrolled or compromised movement.[5,37,56] Others have identified equipment (eg, shoe/cleat design), the abrasive nature and variations in playing surfaces, and various anatomical and biomechanical influences.[4,5,7,58]

Primary Type of Injury

The significantly lower incidence of ligament and muscle tears, as well as the lower incidence of ligament sprains documented on FieldTurf, is consistent with prior findings indicating lower extremity sprains on artificial turf,[15] which may be related to the lower shoe-surface traction usually associated with a more consistent, compliant surface.[40,44] This is consistent with earlier summations noting an inverse relationship between the incidence of ligament trauma and surface compliance.[51] Although others have reported greater shoe-surface peak torque and rotational stiffness with artificial surfaces,[34,64] these studies were conducted under noncompetitive, laboratory conditions using traditional mechanical simulations lacking environmental variability, player contact, and the anatomical and neuromuscular complexities during actual sports performance, thus limiting comparison to on-the-field sports activity.[28] Further investigation into the biomechanics of the shoe-surface interaction beyond the laboratory setting will be necessary to elucidate more definitive causes.

Grade and Anatomical Location of Injury

The significantly lower incidence of second- and third-degree injuries on FieldTurf (Appendix 4) is in contrast to nonsignificant findings on similar surfaces during high school football and soccer competition,[40,56] a level of play where the degree of speed, power, and subsequent impact trauma is lower than that observed at the collegiate or professional level of sport.[5] Findings may more clearly reflect the higher impact attenuation/shock absorbency of the more compliant turf surface at this level of play.[15,40]

In regard to anatomical location, the nonsignificant differences between playing surface are in contrast to earlier studies indicating a lower concussion rate on natural grass when compared to the earlier generation of artificial surfaces.[13,20] The overall incidence of cranial/cervical trauma, as well as the incidence of both upper and lower extremity injury on both surfaces in this study, were greater than that previously reported among high school, college, and professional athletes.[9,13,20,21,29,40,47] Results may reflect the level of athletic size and prowess, when comparing the time that these studies were conducted.[5]

Type of Tissue Injured

This study did not establish the coefficient of restitution or degree of rebound; however, when compared to the polyethylene/cryogenic rubber composition of FieldTurf, lower extremity findings on natural grass seemed to reflect a less compliant surface and lower energy absorption at ground impact. The energy of impact is subsequently transferred back—in this case, to the lower extremity region—increasing the potential for trauma.[65] This may be reflected in the significantly higher incidence of distal tibiofibular ligament sprains on natural grass when compared to FieldTurf. Although others have reported a significantly greater incidence of ankle sprains, combining data derived from 8 brands of artificial turf,[15] the authors did not control for length and time of collection or variation in turf type or quality, methodological concerns that may have benefitted from further analyses.

Environmental Factors

Limited attention has been directed toward the potential influence of weather conditions on injury during competition.[21,40,45] In this study, the majority of play and injuries occurred during conditions of no precipitation, therefore minimizing the opportunity to thoroughly ascertain possible influences under various field conditions. The significantly lower incidence of injury on FieldTurf during play on wet fields may reflect the more consistent surface that the turf provides during inclement weather. The significantly lower incidence of injuries on FieldTurf when temperatures remained above 70°F is in contrast to findings previously reported on other surfaces,[40,45] although those surfaces were an earlier type of turf or natural surfaces under drier conditions when compared to today's highly managed collegiate grass surfaces. The more consistent FieldTurf surface may not have impeded normal performance (eg, speed, impact) during games under cold weather conditions, thus contributing to the significant increase but lower total number of injuries when compared to injuries occurring with higher temperatures.

Contrary to that of prior studies on the original artificial turf surfaces, the significantly greater incidence of injury during hot days on natural grass supports prior findings that indicated enhanced shoe-surface interaction potentiating articular trauma with increasing turf temperature,[45,60] as well as reports of greater frequency of knee trauma with higher temperatures.[44] In summary, these findings are of clinical concern and warrant further investigation for optimal natural grass management practices.[58]

Limitations

Several potential limitations to the study may have influenced the type and number of injuries reported. These included the inability to determine and control the inherent random variation in injury typically observed in high-collision team sports[8,37]; the strength and conditioning status of the athletes and variations in the type of equipment used[4,5,7,25,31,36,59,68]; the weather conditions and variations in field conditions[1,7,22,30,56]; the differences in postural/joint integrity, musculoskeletal structure, and biomechanics of movement[4,7,10,31,38,66]; the time of year[7,15,31]; the coaching style, experience, and play calling[5,6,21,27,36,38]; the quality of officiating and foul play[64]; the player's position and actual versus average time to exposure to injury[5,11,22,25,27,32]; the sport skill level, intensity of play, and fatigue level at time of injury[20,27,31,32,50,59,65,68]; the athlete's ephemeral response to help seeking, injury, and subsequent pain[36,38,40,41,47]; the unreported congenital/developmental factors predisposing an athlete to additional injury[5,10,31,36,56,57,65]; or, simply, any unforeseen mishap.[37,40] Also, there is always the opportunity for an injury to go unreported despite the comprehensive nature of any reporting system.[5,24,36]

Key strengths of the study included the opportunity to follow several universities during the 3-year period, which prevented seasonal injury fluctuations and individual team effects and which enhanced the ability to identify differences and trends in surface effect. In addition, the combined method of assessing functional outcome, time loss, direct observation, and treatment records, as well as the daily interactions of ATCs and players evaluated in this study, minimized the potential for transfer bias and unreported injuries throughout the season.[40,62] The daily evaluation and follow-up telephone visits also increased the opportunity to quantify and track typically overlooked minor indices that often evolve into chronic or overuse problems.[40,62]

Note that the percentage of influence from risk factors other than simply surface type cannot be overlooked. Because of the inherent challenges of collecting data on multiple indices and on numerous teams and players over an extended period, the degree of influence from these risk factors remains a limitation that can only be acknowledged at this time.[27,39,65] The prospective cohort multivariate design did enhance sample size, result in randomization of play on both surfaces, control for seasonal and team variation, and allow for greater insight into significant and subtle differences between a new generation of artificial turf and natural grass.

Finally, the lack of a universally accepted definition of sport injury will continue to be a challenge and subsequent influence on injury interpretation.[40,44] With the concomitant difficulty in subjectively determining a plethora of surface conditions and quality of natural grass,[40,58] any attempt to interpret the injury-surface interaction with any degree of accuracy will continue to pose concerns.

CONCLUSION

Although similarities did exist between FieldTurf and natural grass over the 3-year period of competitive play, there were significant differences in injury incidence, severity of injury, injury time loss, injury situation, grade of injury, injuries under various field conditions, and temperature. No significant differences in head, knee, or shoulder trauma were observed between playing surfaces. Both surfaces, from a statistical and clinical standpoint, exhibited unique injury mechanisms that need to be addressed to reduce the number of game-related collegiate football injuries. The hypothesis that collegiate athletes would not experience any difference in the incidence, mechanisms, and severity of game-related injury between FieldTurf and natural grass was not supported. FieldTurf is in many cases safer than natural grass. However, the findings of this study are generalizable to only this level of competition. Because this study is still in the early stages, investigation is ongoing.

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SUPPLEMENT NO. 53

Incidence and Risk Factors for Injuries to the Anterior Cruciate Ligament in National Collegiate Athletic Association Football

Data From the 2004-2005 Through 2008-2009 National Collegiate Athletic Association Injury Surveillance System

Jason L. Dragoo,*† MD, Hillary J. Braun,† BA, Jennah L. Durham,† BA, Michael R. Chen,† MD, and Alex H.S. Harris,† PhD
Investigation performed at the Department of Orthopaedic Surgery, Stanford University, Redwood City, California

Background: Injuries to the anterior cruciate ligament (ACL) are common in athletic populations, particularly in athletes participating in football, soccer, and skiing.

Purpose: The purpose of this study was to analyze the National Collegiate Athletic Association (NCAA) Injury Surveillance System (ISS) men's football ACL injury database from the playing seasons of 2004-2005 through 2008-2009 to determine the incidence and epidemiology of complete injury to the ACL in NCAA football athletes.

Study Design: Descriptive epidemiology study.

Methods: The NCAA ISS men's football database was reviewed from the 2004-2005 through 2008-2009 seasons using the specific injury code, ''Anterior cruciate ligament (ACL) complete tear.'' The injury rate was computed for competition and practice exposures. Ninety-five percent confidence intervals (CIs) for the incident rates were calculated using assumptions of a Poisson distribution. Pairwise, 2-sample tests of equality of proportions with a continuity correction were used to estimate the associations of risk factors such as event type, playing surface, season segment, and football subdivision. Descriptive data were also described.

Results: The ACL injury rate during games (8.06 per 10,000 athlete-exposures [AEs] 95% CI, 6.80-9.30) was significantly greater than the rate during practice (0.8 per 10,000 AEs 95% CI, 0.68-0.93). Players were 10.09 (95% CI, 8.08-12.59) times more likely to sustain an ACL injury in competition when compared with practices. When practice exposures were analyzed separately, the injury rate was significantly greater during scrimmages (3.99 per 10,000 AEs 95% CI, 2.29-5.94) compared with regular practices (0.83 per 10,000 AEs 95% CI, 0.69-0.97) and walk-throughs (0 per 10,000 AEs 95% CI, 0-0.14). There was an incidence rate of 1.73 ACL injuries per 10,000 AEs (95% CI, 1.47-2.0) on artificial playing surfaces compared with a rate of 1.43 per 10,000 AEs (95% CI, 1.24-1.63) on natural grass. The rate of ACL injury on artificial surfaces is 1.39 (95% CI, 1.11-1.73) times higher than the injury rate on grass surfaces.

Conclusion: Between 2004 and 2009, NCAA football players experienced a greater number of ACL injuries in games compared with practices, in scrimmages compared with regular practices, and when playing on artificial turf surfaces. This latter finding will need to be confirmed by additional studies.

Keywords: anterior cruciate ligament; ACL; injury; football; NCAA; artificial turf

Football is one of the most popular sports in the United States and has been played competitively at the collegiate level for over 100 years. Football is a high-impact sport, with injuries occurring via both contact and noncontact mechanisms, and a complex sport, requiring that athletes perform position-specific skills and activities. It is also the leading cause of sport-related injuries, with an injury rate almost twice that of basketball.[20,24] The demands of the game are such that players' physical characteristics (eg, height and weight) vary widely, both within a team and among National Collegiate Athletic Association

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(NCAA) divisions. The large number of athletes involved in NCAA football combined with the variability in their positions and individual characteristics reveals interesting injury patterns.

Over 100,000 injuries to the anterior cruciate ligament (ACL) occur annually in the United States,[8] frequently during activities involving cutting, jumping, and pivoting.[13] Sports such as soccer, football, and skiing have been characterized as high-risk activities, with several authors reporting the risk of ACL injury in these sports to be as much as 10 times higher than in other activities.[3,9,28] In football players, ACL injuries are relatively common and can often have devastating career consequences. Data from the National Football League (NFL) Combine show that approximately 8% of participants have a history of ACL injury,[7] ACL injuries represent 35% of all knee injuries previously sustained by participants,[4] and ACL injury is the number one reason for receiving a medical failure grade.[6] Brophy et al[6] reported that a previous ACL injury was associated with a decreased probability of playing in the NFL, as only 45.9% of patients reporting an ACL injury at the NFL Combine went on to play at least 1 game. Furthermore, injury to the ACL also places patients at an increased risk for development of posttraumatic osteoarthritis, regardless of concomitant meniscal injury[18,19] or whether they undergo surgical reconstruction of the ruptured ligament.[16] It is important to understand the incidence and causes of ACL injuries in this vulnerable population.

The NCAA Injury Surveillance System (ISS) was developed in 1982 to provide current and reliable data on injury trends in intercollegiate athletics. The purpose of this study was to analyze the NCAA ISS men's football ACL injury database from the playing seasons of 2004-2005 through 2008-2009 to determine the incidence and epidemiology of complete injury to the ACL in NCAA football athletes. Our goal was to determine the injury rate and risk factors associated with ACL injuries (eg, position, playing surface, activity at time of injury, injury mechanism, game vs practice, and season segment). No formal hypothesis testing was proposed at the time this survey was initiated, so all findings represent post hoc analysis for specific comparisons.

MATERIALS AND METHODS

Sampling and Methods

Collegiate institutions sponsoring varsity football programs voluntarily participated in the fall NCAA ISS by collecting data annually during the 5-year period from 2004-2005 through 2008-2009. The ISS is a surveillance system that collects a sample of data from a larger population as opposed to a registry that records every event's occurrence. The ISS uses a convenience sample—the online database permits any institution from any of the 3 NCAA divisions to submit data. This creates a deterministic rather than a random sample but has been deemed an acceptable manner of data collection for monitoring injury trends and patterns because it maintains a representative cross-section of NCAA participants while being courteous to the needs of the volunteering institutions. Since 1982, the ISS has employed stable, basic system definitions and recording methods, served as the basis for NCAA policy, and emerged as a powerful resource for monitoring injuries in NCAA sports.

During the 2004-2005 through 2008-2009 seasons, the ISS collected data on injuries and exposures in NCAA football players that occurred during organized practices and competitions from the first day of preseason to the final postseason competition. Injury data collection was achieved via an online submission form that asked questions pertaining to the basic mechanism of injury, time and place of injury, body part injured, type of injury, severity of injury (time loss and surgical interventions required), and sport-specific questions such as player position and equipment involved. Exposures were collected through a web-based system as they were entered by participating institutions through a 1-page exposure form summarizing the number of practices and competitions, the average number of participants per activity, season (preseason, regular season, or postseason), type of playing surface, and location of competition (home or away).

The Men's Football Injury Data Set for this 5-year period (2004-2005 to 2008-2009 seasons) was examined using the specific injury code "Anterior cruciate ligament (ACL) complete tear." The number of participating institutions varies each year, but during the time period of this investigation, the overall number of participating institutions ranged from 8.9% to 10.1% of schools sponsoring NCAA intercollegiate football teams. Injuries were reported to the ISS by the team athletic trainers when the injury satisfied 3 criteria: (1) occurred as a result of participation in an organized NCAA intercollegiate practice or competition, (2) subsequently required medical attention, and (3) resulted in restriction of the student-athlete's participation in practice or competition for at least 1 calendar day beyond the initial day of injury. We also looked at the Men's Football Exposure Data Set 2004-2005 to 2008-2009 to determine the number of athlete-exposures (AEs) to be used as the denominator when calculating injury rates. A reportable AE was defined as 1 student-athlete participating in 1 practice or competition in which there was the possibility for athletic injury, regardless of the duration of that participation. Of note, preseason intrasquad scrimmages were classified as practices rather than games. Athletic trainers submitted data starting on the first official day of preseason practice,

*Address correspondence to Jason L. Dragoo, MD, Department of Orthopaedic Surgery, Stanford University, 450 Broadway Street, Pavilion C, 4th Floor, Redwood City, CA 94063-6342 (e-mail: jdragoo@stanford.edu).
†Department of Orthopaedic Surgery, Stanford University, Redwood City, California.
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and data collection extended to and included the final day postseason competition.

Once the injury and exposure variables contained in the Men's Football Injury Data Set 2004-2005 to 2008-2009 and the Men's Football Exposure Data Set 2004-2005 to 2008-2009 were identified, we included them in the statistical analysis described below to establish the incidence and epidemiology of ACL injuries and the risk factors associated with ACL injury in NCAA football players.

Statistical Methods

Incidence. To be able to make inferences regarding all NCAA football exposures from the convenience sample reflected in these data, the NCAA has created sampling weights, which were used in all analyses. The incidence of injury, that is, the injury rate (the number of injuries divided by the number of AEs), was computed per 10,000 AEs for competition and practice exposures. The AEs for the period of interest were established by looking at the AEs contained in the Men's Football Exposure Data Set 2004-2005 to 2008-2009. Ninety-five percent confidence intervals (CIs) for the incidence rates were calculated based on assumptions of a Poisson distribution using the standard large-sample formula as implemented in the ci:poisson() function in the epicalc package of the R statistical program.[27]

Rate ratios were also calculated, as they are useful for determining whether there is an increased rate of injury associated with 1 particular participation type over another. For instance, if the competition injury rate were 14.0 times higher than the practice injury rate, then the competition-to-practice rate ratio would be 14.0. Ninety-five percent CIs were computed for all rates and rate ratios using standard large-sample formulas.

Descriptive Data on Injuries. Descriptive data were also described, such as player's activity at time of injury, athlete's position, side of body, and injury mechanism. These variables cannot be considered risk factors because no data on the relevant denominators were available (eg, how many players at the same field location were not injured).

RESULTS

During the 5-year period between 2004-2005 and 2008-2009, a total of 318 injuries to the ACL occurred in NCAA football players during 2,222,155 AEs, accounting for 0.23% of all injuries sustained during this 5-year surveillance period. Thus, the overall incidence rate of ACL injuries in NCAA football players was 1.42 per 10,000 AEs (95% CI, 1.27-1.59). The injury rate was highest during the postseason at 1.55 injuries per 10,000 AEs (95% CI, 0.76-2.57), but this rate was not significantly different from the rates of ACL injury in season (1.45 per 10,000 AEs 95% CI, 1.25-1.66) or preseason (1.34 per 10,000 AEs 95% CI, 1.08-1.63). When divided based on competition type, the injury rate during games (8.06 per 10,000 AEs 95% CI, 6.80-9.30) was greater than the rate during practice (0.8 per 10,000 AEs 95% CI, 0.68-0.93). Players were 10.09 (95% CI, 8.08-12.59) times more likely to sustain an ACL injury in competition when compared with practices. When practice exposures were analyzed separately, the injury rate was significantly greater during scrimmages (3.99 per 10,000 AEs 95% CI, 2.29-5.94) compared with regular practices (0.83 per 10,000 AEs 95% CI, 0.69-0.97) and walk-throughs (0 per 10,000 AEs 95% CI, 0-0.14). Players were 4.8 (95% CI, 3.01-7.68) times more likely to sustain an ACL injury in a scrimmage when compared with a regular practice. The ACL injury rate was highest in Division III football players at 1.56 per 10,000 AEs (95% CI, 1.28-1.87), but this was not statistically different from the rates in Division I (1.45 per 10,000 AEs 95% CI, 1.22-1.71) or Division II (1.17 per 10,000 AEs 95% CI, 0.88-1.48).

Playing surface had a significant effect on injury rates, as there was an incidence rate of 1.73 ACL injuries per 10,000 AEs (95% CI, 1.47-2.0) on artificial surfaces compared with a rate of 1.43 per 10,000 AEs (95% CI, 1.24-1.63) on natural grass. The rate of ACL injury on artificial surfaces is 1.39 (95% CI, 1.11-1.73) times higher than the injury rate on grass surfaces. When artificial playing surfaces were further subdivided, the rate of injury on third-generation surfaces, documented as "artificial surfaces with fill" (1.77 per 10,000 AEs 95% CI, 1.5-2.07), was significantly greater than the injury rate on first- and second-generation artificial surfaces, "artificial surfaces without fill" (1.43 per 10,000 AEs 95% CI, 0.8-2.2) and natural grass (1.43 per 10,000 AEs 95% CI, 1.24-1.63).

Athlete positions were only recorded for game exposures. As a result, athlete positions were not recorded in 185.9 (51%) of weighted exposures and were therefore excluded from analysis. Of the remaining injuries for which a position was known, linebackers (7.73%), running backs (7.32%), special teams players (6.32%), wide receivers (5.35%), cornerbacks (3.51%), and quarterbacks (3.2%) were most frequently injured (Figure 1). Injuries to safeties (2.8%), defensive tackles (2.68%), defensive ends (2.55%), offensive guards (2.26%), offensive tackles (2.23%), centers (1.17%), tight ends (0.28%), kickers/punters (0.28%), long snappers (0.28%), and holders (0%) composed the remaining ACL injuries during this 5-year surveillance period. In evaluating these numbers, it is important to remember that exposure varies by the number of players at each position during a play.

The basic injury mechanism was reported as contact with another player in 53.03% of injuries, while acute noncontact (40.13%) and contact with the playing surface (4.38%) accounted for the majority of the remaining injuries (Figure 2). The specific mechanism of injury was undefined, or other, in 45.78% of injuries. Being tackled (20.05%), blocking (11.31%), being blocked (9.66%), tackling (6.6%), being stepped on/fallen on/kicked (5.64%), or contact with a blocking sled/dummy (0.96%) accounted for the remaining injuries. Athletes were most likely to be participating in offensive passing (19.79%), offensive

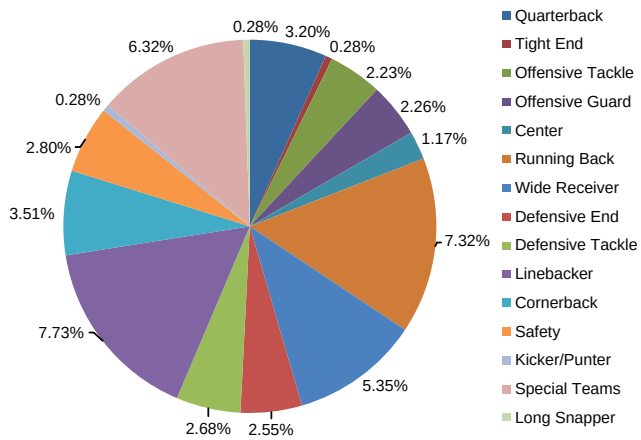

Figure 1. Distribution of anterior cruciate ligament injuries based on player position.

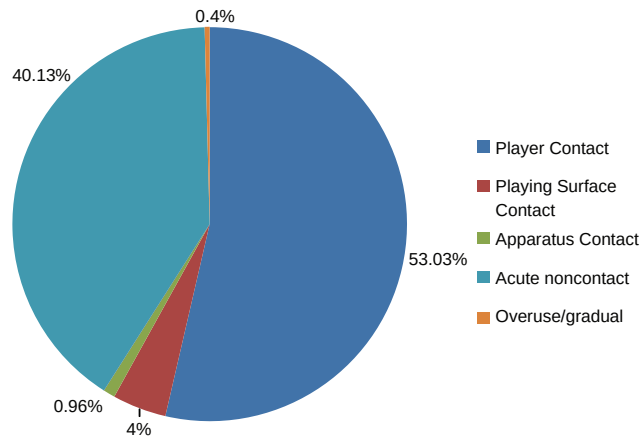

Figure 2. Basic mechanism of anterior cruciate ligament injuries.

running (18.78%), defensive passing (13.65%), or defensive running (12.75%) plays at the time of injury. General play (10.11%), kick-off coverage (6.42%), blocking drills (4.02%), punt return (3.74%), kick-off return (3.62%), punt coverage (2.93%), tackling drills (1.82%), point after touchdown (PAT)/field goal attempt (0.81%), and PAT/field goal defense (0.40%) account for the remainder of the injuries. There was no difference with regard to laterality, with 48.76% of injuries occurring on the left and 51.24% occurring on the right.

DISCUSSION

Our analysis of the NCAA ISS Men's Football Injury Data Set from 2004-2005 to 2008-2009 revealed several important findings: (1) players are over 10 times more likely to sustain an ACL injury in games versus practices, (2) players are significantly more likely to sustain an ACL injury in scrimmages compared with regular or walk-through practices, and (3) the rate of ACL injury sustained on artificial turf is significantly greater than on natural grass. We also observed that the vast majority (90.16%) of ACL injuries are caused by player contact or acute noncontact mechanisms and that player position may influence the likelihood of sustaining an ACL injury.

Injuries to the knee are widely recognized as the most common form of musculoskeletal injury sustained by American football players.[4,10] Dick et al[10] evaluated the NCAA ISS from the 1988-1989 through 2003-2004 seasons and found that knee internal derangement injuries were the most commonly sustained injury in both games and practices. A recent investigation by Bradley et al[4] examined the incidence of knee injury in collegiate football players at the NFL Combine and determined that 53% of the population had sustained a knee injury and an average of 1.3 knee injuries were sustained per player. In this participant population, ACL injuries were the third most common type of knee injury, behind medial collateral ligament and meniscal injuries, and surgical reconstruction of the ACL was the second most common type of surgery, behind arthroscopic meniscectomy.[4]

Our results indicate that ACL injuries occur over 10 times more frequently in competition than in practice, a finding that is consistent with the current literature. Dick et al[10] reported that the rate of knee internal derangement injuries in NCAA football players was 13 times greater in games when compared with practices. Shankar et al[24] reported injuries were over 6 times more prevalent in both high school and collegiate football games compared with practices, while Bradley et al[5] found that twice as many ACL injuries occurred during competition compared with routine practices. A plausible explanation for this significant trend observed in our analysis and in previous studies is that game conditions involve a less predictable playing environment and greater speeds and intensity. The increase in magnitude and frequency of collisions may increase the risk of injury, particularly given our finding that player contact mechanisms account for over half of all ACL injuries. This theory may likely also account for the increased risk of injury in practice scrimmages compared with regular or walk-through practices; we observed an almost 5-fold increase in ACL injuries in scrimmages compared with regular practices, which was consistent with a previous literature report of increased injury in football scrimmage practices in the Big Ten Conference.[1] Given that the objective of a practice scrimmage is to simulate game-like playing conditions, this similarity in injury rates is expected.

Recent literature reviews have categorized the varying types of artificial playing surfaces, investigated their biomechanical properties, and documented distinct injury patterns.[11,25,29,30] Although it is becoming increasingly clear that playing surface is an important factor in injury incidences and mechanisms, the exact role and ideal surface have yet to be determined. Significantly higher overall injury rates in football have been reported on artificial surfaces compared with natural grass.[12,14,15,21,22] However, current literature examining the rates of ACL injury

specifically on artificial surfaces and natural grass has revealed mixed results. Two reports documented no difference in ACL injury rates on artificial turf and grass surfaces,[5,17] while a different study found increased rates of noncontact ACL injuries on grass compared with artificial turf.[23] Others still have reported an increased overall risk of ACL injury on turf when compared with grass.[12,14,23] This lack of consensus may be because of the shortage of studies specifically designed to test this hypothesis, an issue further complicated by the widely variable nature of artificial surfaces. First- and second-generation surfaces consist of short nylon fibers, with variable padding, while third-generation surfaces are characterized by longer fiber lengths, rubber synthetic infill, and increased shock absorbency. Our analysis of the data in this survey indicates a greater incidence of ACL injuries in NCAA football players on artificial playing surfaces when compared with grass. Furthermore, the rate of injury on artificial surfaces with fill (third-generation) is significantly greater than both artificial surfaces without fill (first- and second-generation) and natural grass surfaces. A potential cause of this difference might be the unique shoe-surface interaction associated with artificial surfaces. Extensive research has been conducted to evaluate the mechanical properties of artificial turf. Previous studies have indicated that playing surface plays a significant role in peak torque, rotational stiffness, and foot loading.[30] Compared with grass, artificial turf surfaces showed higher peak torques and rotational stiffness, potentially explaining the mechanism by which artificial turf might contribute to knee injuries.[26] While there are many potential reasons for the observed increase of injuries on artificial turf compared with natural grass in the present study, it should be noted that the CIs for injury rates do overlap, and consequently, future studies are necessary to confirm these findings.

A large percentage of injury reports omitted specific data such as player position or injury mechanism. However, our analysis of the available data revealed that linebackers, running backs, special teams players, wide receivers, cornerbacks, and quarterbacks most frequently sustained an ACL injury (Figure 1). Shankar et al[24] reported similar findings for overall injury rates, noting that linebackers and running backs had the highest rates of injury at both the high school and intercollegiate levels. Similarly, Dick et al[10] found that NCAA running backs, quarterbacks, and linebackers most frequently sustained injuries in games. With regard to the ACL specifically, a review of these injuries in the NFL also reported running backs and linebackers to be among the positions most frequently injured.[5] Additionally, our study revealed that ACL injuries occurred most frequently during offensive running and passing plays. When combining offense and defense, running plays accounted for 31.53% of all injuries, while passing plays accounted for 33.42% and special teams play for 17.92%. These results were similar to those reported by Bradley et al[5]; in the NFL, ACL injury most frequently occurred during passing plays (32.4%), special teams plays (31.7%), and running plays (28.2%).

Analysis of the data set revealed that the majority of ACL injuries occurred via player contact (53.03%) and noncontact (40.13%) mechanisms (Figure 2). Given the high-impact nature of football, it is unsurprising that the majority of injuries were caused by player contact mechanisms. Interestingly, though, a large proportion of injuries were caused by noncontact mechanisms. Previous reports have identified a plethora of variables that may contribute to noncontact ACL injuries, including biological, biomechanical, and environmental risk factors.[2] Environmental risk factors include weather, footwear, and shoe-surface interaction. Of the injuries sustained on natural grass, 36.12% were noncontact, while 44.29% of injuries sustained on artificial turf were caused by noncontact mechanisms. This difference, combined with the structural and mechanical differences of natural grass and artificial playing surfaces, suggests the possibility of a playing surface–associated environmental risk factor at play in noncontact ACL injuries.

We did not observe a difference in ACL injury rates between preseason, regular season, and postseason. This observation was in contrast to previous literature reports, which found the rate of preseason ACL injury to be greater than regular season injury rates[5] and the overall rate of NCAA football injuries to be greater during preseason than regular season.[10] We also did not observe a significant difference in ACL injury rates between Divisions I, II, or III, which was in contrast to a previous study that reported a significantly greater rate of overall injury in Division I compared with Division II.[10]

The results of this study suggest 3 areas for future investigations and possible interventions aimed at reducing the number of ACL injuries in NCAA football players. The increased injury incidence on third-generation artificial turf may suggest that this surface is not an adequate substitute for natural grass surfaces and should be further investigated. Additionally, both the increased incidence of injury in games versus practices and the large percentage of injuries due to noncontact mechanisms suggest it may be necessary to alter training programs to improve factors such as strength, proprioception, and decision making in response to unexpected stimuli in order to improve overall and game-time biomechanics.

The NCAA ISS is a well-established and sophisticated injury reporting system. Nonetheless, the current findings are subject to several limitations. First, as with any study relying on reported information, it is possible that ACL injuries at participating institutions were unreported or underreported and are therefore missing from analysis. Second, the NCAA ISS pools data from up to 15% of NCAA institutions sponsoring intercollegiate football teams and uses sampling weights to determine overall injury and exposure values. Because a large percentage (~85%) of schools are not included in this system, actual rates and incidences of ACL injuries may vary from those that are ultimately reported. Finally, nearly 50% of reported injuries were missing data for some categories such as player position or specific injury mechanism. The trends reported here were based off the remaining subset of available information; however, it is still worth noting the large percentage of missing data, which may have ultimately skewed the results. Despite these limitations, we believe the information presented here is the best means

of classifying and evaluating incidence and risk factors for ACL injury in NCAA football players.

CONCLUSION

Between 2004 and 2009, NCAA football players experienced a greater number of ACL injuries in games compared with practices, in scrimmages compared with regular practices, and when playing on artificial turf surfaces. The majority of injuries were caused by contact with another player or acute noncontact mechanisms. The distribution of ACL injury varied with position type and was most often reported in linebackers, running backs, and special teams players.

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Impact Attenuation Values and Prevention of Head Injuries in Children's Playgrounds

Rolf Huber, B. Comm.
Canadian Playground Advisory Inc.
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Abstract

There are more than 225,000 playground related injuries per year in the United States that result in an emergency room visit, and over 16,000 are traumatic brain injuries (TBI) and approximately 350 of those with TBI are admitted to hospital. It is suggested that TBI, especially in children is underreported by as much as 10 fold. A concussion for a child may have long-term debilitating effects and a second concussion prior to the complete recovery from the first could be life altering.

Cadaver, large animal and subhuman primate studies were initially used by the military and automotive safety engineers to provide initial impact prevention thresholds related to maximum acceleration. Initial studies resulted in the Wayne State Tolerance Curve (WSTC), leading to the Severity Index (SI) and Head Injury Criteria (HIC) during the 1960s and 1970s. These were used to establishment values exceeding of 200 g's and 1000 HIC_{max} as being a risk to life. These values were being recommended by the Consumer Product Safety Commission and later became the impact attenuation thresholds of ASTM F1292 in the early 1990s.

During the 1990's, developments in automotive safety continued to improve the performance measures, injury consequences and impact attenuation values for the protection of vehicular occupants from risk of injury with an abbreviated injury score (AIS) >4. In 2000 new threshold values were set by the National Highway Traffic Safety Administration (NHTSA) for HIC_{15} at <700 for all crash test dummies 6 years and older and HIC_{15} <570 for the 3 year old crash test dummy. This is a model for the threshold of a life-threatening head injury for the relevant age group.

During the first decade of this century, concussions in sports, both amateur and professional gained prevalence. Through injury studies and media attention, the issue has achieved a raised profile. Studies of twenty-five concussed NFL players through reconstruction of the circumstances of the injury and ongoing studies using the Symbex Head Impact Telemetry System (HIT system) would suggest that concussions occur in professional, college and high school athletes in a range from 98 to 117 g's.

Based on these studies, it would suggest the current thresholds for ASTM F1292 and the recommendations of the Consumer Product Safety Commission for impact attenuation values for children's playground surfaces should get another look by injury prevention specialists and advocates. The playground industry is still operating in the 1970s to 1980s with threshold values of Gmax not exceeding 200 and HIC_{max} not exceeding 1000 for the determination of critical height or establishing the limited below which a serious injury is not likely to occur. These values do not recognize advances in research on injury prevention. Today there is credible evidence that these standards should move into the new century with thresholds substantiated by current research and injury data.

There are a number of factors that have come to light in relation to the ASTM F1292 and CPSC Public Playground Safety Handbook. The primary concern is the consideration of the Gmax and HIC values that

are to become the threshold for compliance to the standard. Although most of the impact intervals related to playground surfaces are generally less than 10ms as in the automotive studies, there are some surfaces designed for very low Gmax and HIC at above 10' (3m) that have demonstrated intervals in the 16 to 18ms and there must be a determination if the HIC calculation shall be constrained to 15ms for HIC_{15}. Additionally it might be time to consider a different HIC_{15} threshold for playground surfacing under play structures intended of children under 3 based in the HIC_{15} threshold for the 3 year old dummy of 570 and the HIC_{15} value for the 6 year old dummy being 700. There is injury data to support a reduction for the Gmax to a maximum of 125g and consideration should be given to reducing this to 110g. The separation of impact attenuation values by age group would be consistent with both ASTM F1487 Standard and the CPSC Handbook as age and anthropological differences are used as factors in the rationale that determined the technical performance requirements for play structures targeting the development needs of each age appropriate user group.

Conclusion: Strong consideration should be given to changing the HIC duration, HIC value and Gmax value in ASTM F1292 to the following;

1. HIC changed from HIC_{max} to HIC_{15}
2. HIC threshold changed from 1000 to HIC_{15} threshold 700 for play structures intended for children 5 to 12 years and 570 for play structures for children 2 to 5 years old.
3. Gmax value changes from 200 to 105

The following paper will explore the new research supporting these conclusions and begin to define the limited financial impact that these recommendations will have on the public playground and the benefits overall to playground injuries.

A Discussion of the Current Head Injury Prevention Strategies as it Relates to Concussion and Head Injuries in Children's Playgrounds

A concussion is a serious head Injury that often occurs on playgrounds. Annually playgrounds rank 4th with 226,091 emergency room visits for all playground injuries and 3rd with 16,130 traumatic brain injuries for all ages and for the 5-18 year age group 160,621 injuries requiring emergency room visits and 10,414 traumatic brain injuries for sport and recreation injuries in the years 2001 to 2005. Admissions to hospital during 2001 to 2005 for traumatic head injuries sustained in playgrounds were 529 for all age groups and 349 for ages in the 5-18 range annually.[1] In the single year between July 2000 and June 2001 220,000 playground nonfatal injuries were treated in emergency departments in the United States[2]. It has been suggested that up to 3.8 million children and adults sustain a concussion each year (Langlois, Rutland-Brown, & Wald, 2006) rather than the previously suggested 300,000 (Thurman, Branche, & Sneizek, 1998) and this might be an underestimate due to the challenges in identifying

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concussion, particularly in young children (Gioia, Collins, et al., 2008; Williamson & Goodman, 2006)[3]. In the early part of the 2000s, by far motor vehicle accidents resulted in the greatest number of head injuries and deaths (61%)[4]. Consequently the bulk of the testing programs and studies related to the prevention of all related injuries and in particular head injuries have been done by this industry. More recent information from the United States Center for Disease Control and Prevention (CDC) (2010) indicates that motor vehicles and traffic are now responsible for 17.3% of TBI and 31.8% of deaths due to head injuries and head injuries due to falls have increase to 35.2%[5].

On the playground, the surface is the major contributing factor to impact of the falling child, whether the impact is to the head or directly to a part of the body with sufficient force to transfer to the skull and brain. The following pages are a compilation of various research studies and a thorough discussion of the issues. It is the intent of this paper to present conclusions and recommendations to those responsible for; public health, injury prevention, regulators, and standard writers as to changes that should be made to existing documents for better prevention of severe injuries.

Overview of Head Injury

Statistics indicate that the head injuries and specifically the traumatic brain injury are happening at an alarming rate on playground and athletic surfaces. TBIs are both complex physiological and neurological events that are under reported to a high degree mainly due to the lack of outward physical injury that would involve the traditional ABC's or "airway, breathing and circulation of any first aid course. The complexity of diagnosis of head injury and potential for head trauma on the playground lack of or misdiagnosis of the initial damaging impact, let alone compounded threat of more serious damage caused by secondary or recurring impacts, are very difficult to detect by the untrained persons or even trained persons with the assistance of scientific instruments. To better understand the issue, it will be important to understand and define what TBI is, how to recognize it, and how to treat it. Additionally some of the history related to and advances made in impact attenuation and its relationship to the protection of humans needs to be reviewed. This will involve a closer look at past analysis methods from cadavers and primate studies to more current and sophisticated football helmets studies and injury reconstruction analysis. In the end there must be a universally accepted premise that no one wants to unknowingly seriously injure a child. Standards writing organizations, along with industry and injury prevention organizations must look to set impact attenuation thresholds that protect the child from unreasonably serious injury. It is anticipated that review of the current research and the a conclusions of future research will result in the conclusion that current thresholds for impact attenuation need to be significantly lowered to provide the level of protection provided by the current NHTSA Standards.

Traumatic Brain Injury

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[5] US CDC, What are the Leading Causes of TBI?, www.cdc.gov/TraumaticBrainInjury/causes.html., June, 2010

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The consensus statement from the Zurich, Switzerland International Symposium on Concussion in Sport (CIS) in 2008 defines concussions as "a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces".[6] Concussions have five major features;

1. Concussion may be caused either by a direct blow to the head, face or neck or elsewhere on the body with an "impulsive" force transmitted to the head.
2. Concussion typically results in the rapid onset of short-lived impairment of neurologic function that resolves spontaneously.
3. Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury.
4. Concussion results in a graded set of clinical symptoms that may or may not involve loss of consciousness (LOC). Resolution of the clinical and cognitive symptoms typically follows a sequential course; however, it is important to note that in a small percentage of cases, postconcussive symptoms may be prolonged.
5. No abnormality on standard structural neuroimaging studies is seen in concussion.[7]

The biokinetics that induce a concussion consist primarily of acceleration, deceleration and rotation forces.[8] Although the direct impact with the head is a high risk for a concussion, significant impacts between the body and the impacting surface can also lead to the concussion. This is particularly the case in the activities of children in playground falls and sport related injuries resulting in impact and rotational forces being transferred to brain within the cerebral fluid within the skull. Ultimately the concussion occurs when the brain is moving about inside the spinal fluids due to the force of an impact. The brain can hit the skull from the inside, and that can tear some blood vessels, injure some nerves and even leave some bruises on the brain.[9]

There are three different kinds of concussions that are related to severity and they are labelled as grades of concussions as follows;

- Grade 1 (Mild concussion) – Confusion, dizziness or other symptoms that last less than 15 minutes
- Grade 2 (Moderate concussion) – The symptoms last longer than 15 minutes, but there is not loss of consciousness (LOC)
- Grade 3 (Severe concussion) – There is LOC, even if it lasts only a few seconds or minutes[10]

Obviously this will have a determination of medical treatment sought immediately after the injured child or athlete is stabilized. This could range from total and quiet rest for 15 minutes to immediate transfer to a medical facility or hospital.

[6] Halstead ME, Walter KD and the Council on Sports Medicine and Fitness, Clinical Report – Sport –Related Concussion in Children and Adolescents, Pediatrics, The American Academy of Pediatrics, August 30, 2010
[7] Halstead ME, Walter KD and the Council on Sports Medicine and Fitness, Clinical Report – Sport –Related Concussion in Children and Adolescents, Pediatrics, The American Academy of Pediatrics, August 30, 2010
[8] Halstead ME, Walter KD and the Council on Sports Medicine and Fitness, Clinical Report – Sport –Related Concussion in Children and Adolescents, Pediatrics, The American Academy of Pediatrics, August 30, 2010
[9] Nagy K, What Happen Inside a Child's Brain When Force Brings a Head and an Object to Meet, Ezeinearticles.com
[10] Concussion Facts – causes, diagnosis, and treatment, Pediatrics for Parents, April 2001

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Since concussions in children in the play environment are not likely to be immediately detected and that a concussion is a serious head injury, it is important to recognize the symptoms to provide as prompt treatment as possible. First and foremost it is estimated that up to 90% of concussions do not involve loss of consciousness (LOC), however; all concussions result in some constellation of physical, cognitive, emotional, and/or sleep-related symptoms[11]. Secondly it is important to realize that children who are constantly changing developmentally and without good communication skills can sustain a concussion in a play environment (playground, hills, tree climbing, running, jumping, etc.) and are not likely to have the event causing the concussion witnessed by an adult. As a result, the symptoms might be the only determinant that a concussion has occurred. Additionally a concussion by itself cannot be seen on x-rays, CT scans or MRIs[12]. As a result checking for the following symptoms is important;

- Mild to moderate headache
- Blurred vision
- See stars or flashes of light or saw black
- Loss of balance and dizziness
- Nausea and vomiting
- Change in mood, such as laughing, crying or getting angry easily and inappropriately
- Change in the way your child performs in school
- Trouble thinking, does not know time, date, surroundings or what happened before and after
- Short term loss of learned skills such as toilet training
- Decreased energy
- Sudden alteration of sleep
- Paleness
- Easily distracted and poor concentration[13,14]

There are two recognized concussion assessment tools available to parents, caregivers, teachers, coaches and medical professionals. On the international level there is the SCAT2 tool and card that are directed primarily at the athlete aged 10 and over. Alternatively, the US Department of Health and Human Services Centers for Disease Control and Prevention with the program "Heads Up to Schools: Know your Concussion ABDs", provides a Concussion Signs and Symptoms Checklist. This provides a timed assessment of the injured child and recorded resolution of the injury. A critical part of the assessment process and very difficult in children is determining a baseline for the child prior to the injury in the play environment. It is further complicated that young children are constantly changing and baseline data may not be valid for more than 6 months at a time. As a result it is important that the initial assessment is made by a person very familiar with the child. In some activities such as Canadian minor hockey, involving children, there have been serious discussions of performance of baseline assessments prior to the playing season to facilitate, to the extent possible, the symptoms and changes in a concussed child.

[11] Gioia GA, Isquith PK, Schneider JC, Vaughan CG, New Approaches to Assessment and Monitoring of Concussion in Children, Topics in Language Disorders, pp. 226-281, Lippincott Williams & Wilkins, July-September 2009
[12] Sport – Related Concussion: Guidelines for Parents and Caregivers, ThinkFirst-SportSmart Concussion Education and Awareness Committee, May 2010
[13] Jonathan Pitts, Signs of Concussions in Children, HealthGuideance.org,
[14] Sport – Related Concussion: Guidelines for Parents and Caregivers, ThinkFirst-SportSmart Concussion Education and Awareness Committee, May 2010

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For medical professionals there is the Pediatric Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT), which is the paediatric version of the ImPACT battery of tests developed for the Pittsburgh Steelers (Lovell, Collins & Maroon, 2002)[15]. The paediatric version of ImPACT consists of seven subtests that tap several key areas of cognitive functioning related to concussion, including episodic learning, memory for verbal and visual information, working memory, and reaction time/processing speed[16]. The main reason that this is only used by medical professionals is that it requires computers and trained staff to administer the tests thereby creating another problem in the timely diagnosis of the serious head trauma.

Once a child has been assessed as having a concussion, the next step is treatment. First and foremost is "brain rest" and at least 15 minutes of total quiet for the Grade 1 concussion. It may be likely that the child will have to be removed from all activity and stay home from school and other activities for some time and until symptoms resolve. One concern for parents might be that a child "home from school" without physical injury would be like a holiday, however; total quiet and "brain rest" includes no texting, television, video games, computers or physical activity, etc. They will need to be protected from a second head impact while they are recovering and it is important to inform the child's teachers, coaches or childcare providers about the injury and the precautions that are to be taken. Additionally it will be critical to the child's future that the teachers be informed that the child will likely need some remedial help or catching up on lessons missed while they are recovering and no testing should take place during recovery as this will not reflect the ability of the child.

Recovery will be in steps that will take a minimum of one step per day and would be as follows;

- Step 1 Complete rest until all symptoms have improved.
- Step 2 Light exercises, such as walking or stationary cycling, for 10 to 15 minutes.
- Step 3 Sport-specific activity or open play for 20 to 30 minutes without risk of impact, rotation or jarring.
- Step 4 Move to "on field" practice or play without any physical contact or in the case of the play environment, no jumping or other activities that would generate an impact through the body.
- Step 5 Once cleared by a doctor, move to practice on the field with body contact or in the play environment, open play with normal activity.
- Step 6 Game play or back to the play environment[17].

Should at any time during the stepwise recovery, the child suffer any of the symptoms of the concussion, the child should be removed back to total rest for a minimum of 24 to 48 hours and the recovery process begun again after symptoms are gone. The concern always is the occurrence of a second head injury before the child has fully recovered from the first.

[15] Gioia GA, Isquith PK, Schneider JC, Vaughan CG, New Approaches to Assessment and Monitoring of Concussion in Children, Topics in Language Disorders, pp. 226-281, Lippincott Williams & Wilkins, July-September 2009
[16] Gioia GA, Isquith PK, Schneider JC, Vaughan CG, New Approaches to Assessment and Monitoring of Concussion in Children, Topics in Language Disorders, pp. 226-281, Lippincott Williams & Wilkins, July-September 2009
[17] Healthy Active Living Committee, Identification and management of children with sport-related concussion, Canadian Paediatric Society (CPS), Paediatr Child Health 2006: 11(7):420-8, revised January 2010

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Where the concussion is a Grade 2, or 3, the recovery may take weeks or months. In some high profiles sports cases in hockey and football, participant's concussions have been season and career ending. In the case of a child studies have shown that recovery from concussions are slower and take a longer duration for full recovery than adults. Studies comparing NFL players with high school football players indicate that the NFL players returned to base line within a week and many returned to normal performance two days after the injury, while high school athletes demonstrated slower injury recovery and longer lasting neuropsychological effects[18]. Since younger children are changing in ability, behaviour and personality the determination of full recovery is often complicated.

The effects of multiple and cumulative effects of concussions have been studied in organized sports rather than play environments. The risk and consequences of multiple concussions on children in a play environment are difficult due to the injury not being regularly witnessed and when they occur they are significantly under reported. In sports and athletics there is the ability to have trainers and coaches capture the data and record the events as they occur. High School athletes with three or more prior concussion were more likely to experience on-field positive loss of consciousness, anterograde amnesia, and confusion following a subsequent cerebral concussion[19]. One of the authors of the foregoing study, Dr. Michael Collins, director of the University of Pittsburgh Medical Center's Sports Medicine Concussion Program states that six to eight high school football players die each year because of "second impact syndrome."[20] Repeated concussions without full recovery are considered to have led to clinical depression, mental illness and premature death in former football players and boxers. The study involving 2,488 retired NFL players with an average age of 58, and average NFL career of 6.7 year, 61% had sustained at least one concussion and 30% had suffered more than three. Of these 2,488 athletes, 263 (11%) had been diagnosed with clinical depression.[21] Professional boxers receive repeated blows at approximately 52 g's which has been equated to being hit with a 13lb (5.9kg) bowling ball traveling at 20 mph (32kph).[22]

Current Guidance for Playground Safety Surfacing Requirements

The fact that concussions occur in playgrounds and are serious head injuries with the potential of life-changing consequences for the child is irrefutable. Concussion symptoms are ten times more likely to go underreported and left untreated in instances where there is no external physical manifestation as perceived by the child's caregiver. Familiarity with the child's moods, habits and other behaviours may be the critical to form a baseline in determining a change that could be related to a concussion. As a result it is important to investigate the forces that cause a concussion and methods of measurement in the field In order to minimise the frequency and severity that may result from the playground fall and subsequent injury.

[18] NFL Players Show More Rapid Recovery from Concussions than High School Players, University of Pittsburgh Medical Center, UPMC News Bureau, January 23, 2006
[19] Collins MW, Lovell, Iverson GL, Cantu R, Maroon J, Field M, Cumulative Effects of Concussion in High School Athletes, Neurosurgery, 2002
[20] Craig M, Medical Field Helping NFL to Learn About Concussions, Birf.info, August 2006
[21] Kuwana E, Long-Term Effect of Consussion in Football Players, Faculty of Life Science, NYMU
[22] Igou S, How Can the Brain be Injured, BrainInjury.com, Montana Publishing, 2011

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Determination of Impact Attenuation Values in Standards

To start the discussion a summarization of various playground standards' scope in relation to injury protection statements, testing procedures and performance requirements must be considered. The US Consumer Product Safety Commission (CPSC) was the first to publish a value for impact attenuation in the 1981 "Handbook for Public Playground Safety, vol.1&2" with the recommendation that from a maximum estimated fall height, the Gmax should not exceed 200 g's to an ANSI headform.[23] In the 1980s the ASTM F08 Committee on Sports Equipment and Facilities created a task group on playground surfacing, which evolved to the F08.63 Subcommittee on Playground Surfacing. Their first standard, the ASTM F1292 standard was published to outline the laboratory and field the test methods for playground surfacing and the determination of a critical height. In 1991, CPSC revised the "Handbook for Public Playground Safety" and included a recommendation that not only is peak acceleration (Gmax) important, but so too is the duration of the acceleration. Subsequently, the 1993 revision of ASTM F1292 included both a Gmax of 200 and HIC of 1000 as the maximum threshold for determination of critical height for playground surface materials/systems.

It should be noted that the selection of the Gmax 200 is based on primates, large animals and cadaver studies. Although these subjects have been used throughout medical history and continue today, the early studies using cadavers and sub-human primates in the study of head impact dates back to World War II. Many studies investigated the relationship between linear accelerations and damage to the skull or brain. In one such study in the late 1950's the US Air Force performed studies on anesthetised hogs, which indicated that impacts with duration of 4 to 8ms and impacts up to 125g showed reversible injuries, while impacts ranging up to 220g and the same duration resulted in serious to fatal injuries.[24] Similar studies at Wayne State University resulted in the development of the Wayne State Tolerance Curve (WSTC), a roughly logarithmic curve that describes the relationship between the magnitude and duration of the impact acceleration and the onset of skull fractures, in the early 1960s[25].

The WSTC has formed the basis of mathematical modeling for development of injury tolerance curves and better understanding the injury data that are now being produced by the human surrogate (crash test dummies). The first major advance in the development of mathematical models was the plotting of the WSTC on log paper by Gadd, resulting in an approximately straight line function for the weighted impulse criterion and this became known as the Gadd Severity Index (SI).[26] In 1972, the National Highway Traffic Safety Administration (NHTSA) defined the relationship between the WSTC and the SI as modified by Versace in 1971 as the Head Injury Criteria (HIC)[27]. Prasad and Mertz compared a collection of skull fracture and brain injury data with their corresponding HIC values and suggested that

[23] Shiffer RC, ASTM F-1292 as tool for playground injury severity reductions, Head and Neck Injuries in Sports, ASTM, 1994
[24] Lt. Col. John Stapp, M.C., Human Tolerance to Deceleration, American Journal of Surgery, Volume 93, April 1957
[25] Shorten M, PhD, Himmelsbach JA, Sports surfaces and the risk of traumatic brain injury, BioMechanica, LLC, Portland, Oregon,
[26] McHenry BG, Head Injury Criterion and the ATB, ATB User's Group, 2004, pages 3 to 8
[27] McHenry BG, Head Injury Criterion and the ATB, ATB User's Group, 2004, pages 3 to 8

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at a HIC value of 1000 there is a 16% risk of life-threatening brain injury in the adult population[28]. The HIC value of 1000 has been used as the pass/fail in a number of performance standards for prevention of life-threatening injury[29,30,31] in the measurement of impact attenuating surfaces in playgrounds, without consideration of the 1000 HIC value being the tolerance threshold for the adult male. Although there should have been consideration of scaling the value to the age group of the playground, 18 months to 12 years[32,33] the selection of the 1000 value was based on HIC becoming a dominant injury prevention mechanism and from the view point of the ASTM F08 Committee developing the F1292 standard the request to utilize the HIC 1000 threshold was at the request of the CPSC[34]. The use of the 1000 HIC in these standards had been perpetuated by tradition and has only begun to be called into question in the ASTM F08.63 sub-committee in 2010. It has also been believed that HIC of 1000 was the threshold for life-threatening, when it is actually 16% beyond.

For the last 30 years, the playground industry has been dominated by the companies who design and produce play structures, making the issue of setting appropriate protective surfacing a side issue to be taken into consideration by the owner later in the design process. Requirements for playground surfacing performance in the field after installation has followed two tracks of development. The first track has brought about the evolution of technical requirements for performance to a test procedure for impact attenuation, initially recommended by the CPSC[35]. The second was to adopt the presumably parallel guidance provided in the CPSC. The 1991 revision of the pubic playground safety handbook provides a table of commonly used loose-fill surfacing materials and the depths (9 inch compressed and uncompressed) at which they were tested which resulted in establishing each surface material's critical heights[36]. The table was republished in subsequent revisions of the same handbook[37] through November 2010. This table has gained general consumer use as a substitute for ASTM F1292 and was so popular as a measure of performance that it has been adopted as part of the technical requirements of Canadian Standards Association (CSA) Z614-98[38]. Use of the table by the general public has put the focus was on the critical height table and not the descriptive information of the samples that were tested. It was later discovered that the materials as described were not necessarily commercially available in all juristictions and this resulted in the removal of the surfacing Table 1 from the CSA Z614-03.[39] A version of this table was revised and published by the CPSC base upon nine (9) inches of compacted loose-fill materials. This

[28] Cozy CZ, Jones MD, Development of a simulation system for performing in situ tests to assess the potential severity of head impact from alleged childhood short falls, Forensic Science International, 2006
[29] ASTM F1292-93, Standard Specification for Impact Attenuation of Surfacing Materials Within the Use Zone of Playground Equipment, ASTM, 1993
[30] CSA Z614-98, Children's Playspaces and Equipment, Canadian Standards Association, 1998
[31] En1177:1997, Impact attenuating playground surfacing – test methods, CEN, 1997
[32] CSA Z614-98, Children's Playspaces and Equipment, Canadian Standards Association, 1998
[33] ASTM F1487-93, Standard Consumer Safety Performance Specification for Playground Equipment for Public Use, ASTM, 1993
[34] Shiffer RC, ASTM F-1292 as tool for playground injury severity reductions, Head and Neck Injuries in Sports, ASTM, 1994, p340
[35] US CPSC, A Handbook for Public Playground Safety, Volume 2: Technical Guidelines for Equipment and Surfacing, US CPSC, 1981, p22
[36] US CPSC, Public Playground Handbook for Safety, US CPSC, 1991, p21
[37] US CPSC, Public Playground Handbook for Safety, US CPSC, 1997. P5
[38] CSA, CAN/CSA-614-98, Children's Playspaces and Equipment, 1998, p97
[39] CSA, CAN/CSA-614-03, Children's Playspaces and Equipment, 2003, p46

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chart continues to mislead the general public who fails to evaluate and compare the characteristics of these common materials against those used to create the CPSC Table 1. Failure to remove or adequately revise this table and move their recommendation to a technical compliance with ASTM F1292 will continue to perpetuate this non-technical track and potentially result in continued severe head injuries.

Generally speaking, it has been thought that any changes in recommendations to improve the impact attenuation performance of the protective surface resulted in an increase in the cost of the compliant protective surfacing. This has had the effect of increasing the percentage of the playground budget being spent on the protective surfacing. In the 1970s and early 80s a grass, sand or pea gravel surface was less than 1% of the total playground budget, whereas in 2010 there are playgrounds being installed with sophisticated rubber surfacing systems that consume more than 50% of the total budget for the playground. Protective surfacing critics believe the new emphasis on protective surfacing has had a negative impact on the overall number of play areas and the magnitude of play components sold thereby significantly afftecting the financial performance of the corporations who make them.

At present time there are very few large play structure manufacturing entities that supply both play structures along with a full range of playground protective and/or accessible surfacing systems. Generally the more traditional economic business model is that the manufacturer builds a nationwide network of dealers and distributors. These dealers then establish independent relationships with providers of a diverse range of surfacing systems. As a result the profit related to the growing proportion of the overall playground budget going to surfacing is in the hands of the dealer rather than the manufacturer. This model is likely to change as competition, performance requirements, sophistication of installation requirements, warranties and economies of scale increase. Owner/operators and manufactures will have to pay greater attention to the protective surfacing primarily since 68 % of the injuries that occur on public playground equipment involve falls, primarily to the surface below the equipment[40]. Additionally, the Americans with Disabilities ACT, now a U.S. federal law (DOJ 2010 Standard for Accessible Design), specifically requires playgrounds create an accessible route within the use zone of the actual playground and its use zone must comply with and be maintained to ASTM F1292 and ASTM F1951.

All playground standards throughout the world make statements with regard to promoting challenging play within a “safe” environment or have a stated intent of protecting the child from “unforeseen hazards”. While it is important to make a statement regarding the “safety of children” or providing “safe play conditions” or protection from “unforeseen hazards”, there is a failure to clearly define the words safe, safety, unforeseen consequence, or hazard in relation to injury prevention or what is an acceptable severity of injury. In North America, both the ASTM and CSA standards make statements within their scopes with regard to the standard of injury prevention intended through the use of their performance specifications. For the CSA Z614-07, section 1.3 of the scope states “This Standard contains recommendations on technical requirements and practices applicable to the design, manufacture/construction, installation, maintenance, and inspection of public-use playground equipment and playspaces. The specifications laid out in this Standard are intended to minimize the

[40] Tinsworth DK, McDonald JE, Special Study; Injuries and Deaths Associated with Children’s Playground Equipment, US CPSC, April 2001, piii

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likelihood of debilitating and/or life-threatening injuries.[41]" ASTM F1487 in section 1.1 of the scope states "This consumer safety performance specification provides safety and performance standards for various types of public playground equipment. Its purpose is to reduce life-threatening and debilitating injuries."[42] with the 1000 HIC being 16% above the threshold for life-threatening, let alone debilitating.

Alternatively the F08.63 sub-committee on Playground surfacing has been very careful in the injury prevention statements being made in its standards and provides guidance to the user as to the severity of the potential injury and better practices. ASTM F1292, Section 1.1 of the scope, only makes statements to the effect that this specification establishes minimum performance requirements for the impact attenuation of playground surfacing installed within the use zone of playground equipment". It clearly states in section 4.4.3 that the owner/operator may choose to have the surface they are considering to install to have better performance than the minimums in the Standard. To reinforce this, statement the ASTM F08.63 sub-committee has since 2003 published and actively maintains the ASTM F2223, Standard Guide for ASTM Standards on Playground Surfacing which in section 7.2 (impact attenuation) states, "lower values of g-max and HIC signify better performance for impact absorption"[43]. Additionally, ASTM F1292 requires that the owner/operator stipulate the drop height for testing in the field and encourages that that drop height be greater than the minimums set in the relevant play structure standards for fall height. There is also a statement in 4.4.3 that, "the specifier is permitted to specify additional impact attenuation performance requirements, providing that such additional performance requirements are more stringent than the performance requirements of this specification"[44]. As warning and instruction, ASTM F1292 provides a requirement that should a surface within the use zone not comply with the Standard "the play structure shall not be permitted to be used until the playground surface complies."[45] As a result the F0.63 sub-committee has been the provider of much greater technical requirements, interpretation, best practice and clear statements as to the consequences associated with the failure of the playground surface to comply.

ASTM F1292 mission is directed at reducing the 68% of total playground injuries related to falls to the surface, yet the standards, ASTM F1487, CSA Z614, and guides, CPSC document #325, are the predominant resources on playground environment for manufacturers and teaching organizations, leading to concentration on the structures and play components from a technical, performance inspection and testing viewpoint. As an example the most well-known teaching organization for playground compliance in the United States, the National Recreation and Parks Association (NRPA), Certified Playground Safety Inspector (CPSI) course is based on the ASTM F1487, ASTM F2223, and the CPSC Handbook for Public Playground Safety[46]. Although they provide two training modules on performance requirements and use zone requirements it is difficult to understand why there is not more course content devoted to the performance or compliance requirements within these documents addressing the more prominent risk factor of injury from falls. Effectively this leaves the playground owner/operator relying on compliance and therefore injury prevention based on the pass/fail results and values of a one-time product samples successfully tested in a laboratory controlled environment.

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[41] CSA Z614-07, Children's Playspaces and Equipment, Canadian Standards Association, 2007
[42] ASTM F1487-07a, Standard Consumer Safety Performance Specification for Playground Equipment for Public Use, ASTM, 2007
[43] ASTM F2223-03, Standard Guide for ASTM Standards on Playground Surfacing, ASTM, 2003
[44] ASTM F1292-09, Standard Specification for Impact Attenuation of Surfacing Materials Within the Use Zone of Playground Equipment, ASTM, 2009, p4
[45] ASTM F1292-09, Standard Specification for Impact Attenuation of Surfacing Materials Within the Use Zone of Playground Equipment, ASTM, 2009, p4
[46] NRPA, CPSI Course Materials, 2011

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ASTM F1292 is required in ASTM F1487 Standard within the following sections;

- 9.1.1 Layout a use zone with obstacle free surfacing that conforms to F1292,
- 11.2.2 Install protective surfacing in the use zone compliant with F1292
- 13.2.1 Maintain the protective surfacing in the use zone to comply with F1292
- 14.3 Apply a label to inform, warn, and educate purchasers, installers and those supervising children who play on the equipment about the ongoing danger of installing equipment over hard surfaces and to serve as a constant reminder to provide and maintain appropriate surface as identified by F1292

Essentially these sections are not instructive as to the injuries (life-threatening head injury) that ASTM F1292 is set to prevent or that there might be a better practice with regard to injury prevention.

With regard to the most recent revision of the CPSC Public Playground Safety Handbook (document #325), this clearly states in the definitions section that critical height is "the fall height below which a life-threatening head injury would not be expected to occur.[47] Further in section 2.4 on surfacing it provides that;

> "Testing using the methods described in ASTM F1292 will provide a "critical height" rating of the surface. This height can be considered as an approximation of the fall height below which a life-threatening head injury would not be expected to occur. Manufacturers and installers of playground protective surfacing should provide the critical height rating of their materials. This rating should be greater than or equal to the fall height of the highest piece of equipment on the playground. The fall height of a piece of equipment is the distance between the highest designated play surface on a piece of equipment and the protective surface beneath it."[48]

The Handbook differs from ASTM F1487 in a number of material ways; first that non-compliance could have life-threatening consequences, and secondly that the drop height for any structure within the playground as a minimum should be performed at the fall height of the highest structure within the playground or the highest portion of a composite structure. This usually provides more impact attenuation for the play equipment with a lower fall height than the highest play component in the playground. This scenario would still place the performance of the surfacing system for the highest structure at the life-threatening level. Generally the highest fall height of the playground will be 8' to 10' depending upon the selection of swings, slides and climbers.

It could be that persons with some training and general understanding, of ASTM F1487 and the CPSC Handbook are of the belief that the performance of testing to ASTM F1292 is not important or their responsibility and they can rely upon the manufacturer of the surface system or can replace the need for field testing their surface system with the critical height table in the CPSC Handbook or they may be making an assumption that their existing surfaces continue to provide the required protection as required in ASTM F1292 years after installation. This has resulted in significantly fewer people having in their possession the technical documents of the ASTM F08.63 sub-committee. Unfortunately the impact attenuation in playgrounds in California in 2006 "found a 69% failure rate for rubberized California playground surfaces using attenuation standards.[49] This failure was most often relate to the surface test performance at the fall heights of the play equipment with the highest fall heights, generally greater than eight (8) feet. It is anticipated that awareness of ASTM F1292 and ASTM F1951 will rise as they

[47] US CPSC, Public Playground Safety Handbook, CPSC, 2011, p3
[48] US CPSC, Public Playground Safety Handbook, CPSC, 2011, p8

[49] Evaluation of Health Effect of Recycled Waste Tires in Playground and Track Products, Integrated Waste Management Board, 2007, p3

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have now been mandated as two of many performance requirements for the installed ground level accessible route on the playground surface[50].

Defining the Measurement of the Life-Threatening

The development of the impact attenuation criteria used to measure impacts on playground surfaces have almost exclusively been based on the crash tests data produced in the automotive industry with a minor confirming reliance on some military testing. There is significant evidence that a direct impact with the head or an impact to the body with sufficient energy to transmit the force of the impact to the head, combined with sufficient linear or rotational acceleration within an interval of duration in milliseconds, will result in a severe head injury. Historically this was determined with animal and cadaver studies with the result being the WSTC, the Gadd SI and the HIC. This has remained the commonly accepted threshold since the 1970s and 80s for reducing the risk for a life-threatening or permanently life threatening head injury. This HIC value of 1000 was at the time based on the large adult male. There were no other data or scaled values available. Additionally the HIC calculation was not constrained as to the duration of the interval of the impact. Even so this calculation was then translated to ASTM F1292[51]. Since 1993 there have been significant advances in the evaluation of head injury and the impact threshold values related to an acceptable injury severity.

In October of 1986, the National Highway Traffic Safety Administration (NHTSA) recommended the calculation of HIC be constrained to 36 milliseconds (HIC_{36}) since longer intervals had not shown low probabilities of injury in voluntary human subject tests and the HIC of 1000 for the unconstrained HIC would be roughly equal to HIC_{36} 1000. Additionally the test data for head impacts studied by Prasad and Mertz in 1985, Hodgson in 1973 and 1997, Got in 1978 and Tarriere, were all found to have short duration impacts, which were less than 12 milliseconds[52]. As a result both the HIC_{15} and HIC_{36} could be applied to the data. It was determined by the industry group making recommendation to the NHTSA that the limit values more related to the HIC_{15} and should be applied with the maximum HIC value for the mid-sized male being <700[53]. Through scaling it was determined that the HIC_{15} values for various dummy sizes would be as in Table 1[54]. The values in Table 1 have been adopted by the NHTSA as of the final rule making in March of 2000.[55]

Dummy Type	Large Adult Male	Mid-Sized Male	Small Female	6 year old child	3 year old child	1 year old child

[50] U.S. Department of Justice, 2010 ADA Standards, September 15, 2010, page 10
[51] Shiffer RC, ASTM F-1292 as tool for playground injury severity reductions, Head and Neck Injuries in Sports, ASTM, 1994, p340
[52] Eppinge R, Sun E, Bandak F, Haffner M, Khaewpong N, Maltese M, Development of Improved Injury Criteria for the Advancement of Automotive Restraint Systems – II, National Transportation Traffic Safety Administration, Nov. 1999, p 2-1
[53] Eppinge R, Sun E, Bandak F, Haffner M, Khaewpong N, Maltese M, Development of Improved Injury Criteria for the Advancement of Automotive Restraint Systems – II, National Transportation Traffic Safety Administration, Nov. 1999, p 2-9
[54] Eppinge R, Sun E, Bandak F, Haffner M, Khaewpong N, Maltese M, Development of Improved Injury Criteria for the Advancement of Automotive Restraint Systems – II, National Transportation Traffic Safety Administration, March 2000, 2-2
[55] Eppinger R, Sun E, Kuppa S, Supplement: Development of Improved Injury Criteria for the Advancement of Automotive Restraint Systems – II, National Transportation Traffic Safety Administration, March 2000, pS1-1

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HIC_{15} Limit	700	700	700	700	570	390

Table 1: HIC_{15} limits for Crash Dummies of various types

To provide a measure of understanding for Table 1 it is instructive to review the Abbreviated Injury Scale (AIS) and the HIC_{15} thresholds. The AIS was first developed in 1971 to aid vehicle crash investigators in quantifying injury severity and assigning a numeric value and these values are applied to 5 body regions, including the head. AIS injury score runs from 1 to 6 as outlined in Table 2.[56]

Injury Severity	**Abbreviated Injury Score**
Minor injury	1
Moderate injury	2
Severe, but not life-threatening	3
Potentially life-threatening, but survival likely	4
Critical with uncertain survival	5
Unsurvivable injury (maximum possible)	6
Severity unknown	9

Table 2: Abbreviated Injury Scale (AIS)

Although the AIS is applied to 5 portions of the body and then modified to provide the Injury Severity Score for the trauma patient we will only be considering the descriptive symptoms associated with the head and neck and the score from 3 to 5 in table 3.[57]

Severity of Injury Symptom	Body part	AIS
Cerebral injury with or without skull fracture, with unconsciousness more than 15 minutes, without severe neurological signs, brief post-traumatic amnesia (<3 hours	Head and neck	3
Displaced closed skull fracture without unconsciousness or other signs of intracranial injury	Head and neck	3
Loss of eye	Head and neck	3
Avulsion (forcible detachment) of optic nerve	Head and neck	3
Displaced facial bones fractures or those with antral or orbital involvement	Head and neck	3
Cervical spine fracture without cord damage	Head and neck	3
Cerebral injury with or without skull fracture with unconsciousness of more than 15 minutes, with definite abnormal neurological signs; post-traumatic amnesia 3-12 hours	Head and neck	4
Compound skull fracture	Head and neck	4
Cerebral injury with or without skull fracture, with unconsciousness of more than 24 hours; post traumatic amnesia more than 12 hours	Head and neck	5
Intracranial Hemorrhage	Head and neck	5
Signs of increased intra-cranial pressure (decreasing state of consciousness, bradycardia under 60, progressive rise in blood pressure or progressive pupil inequality	Head and neck	5

Table 3: Head and neck injury symptoms for AIS 3 and 4

[56] Abbreviated Injury Scale (AIS) and Injury Severity Score (ISS), Medal Military Medicine, Medal.org Ltd, p1
[57] Abbreviated Injury Scale (AIS) and Injury Severity Score (ISS), Medal Military Medicine, Medal.org Ltd, p 2-3

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During the 1990s, automotive researchers made significant advances in the understanding of injury assessment reference values (IARVs), which were originally developed by Mertz in 1978 and revised in 1993 for response measurement of the Hybrid III small female and large adult male test dummies[58]. Other IARVs were developed for Hybrid III child test dummies. Prasad and Mertz have published injury risk curves for skull fracture and for AIS ≥4 brain injury due to forehead impacts based on the HIC15 criterion and for skull fracture based on peak g center of gravity acceleration.[59] The result is the HIC_{15} value of 700 is a conservative estimate of the 5% risk level of AIS ≥4%[60], the current automotive standard in the United States and Canada.

Present playground surface performance is also partially based on the Gmax of 200, which is based on the US CSPC recommendation for eliminating life-threatening head injuries if that surface could be maintained below this threshold value. It important to review the risk assessment associated with this value of Gmax. Studies by Mertz et al (1997) estimated a 5% risk of skull fractures for a peak acceleration of approximately 180g and a 40% risk of a skull fracture for a peak acceleration of 250g.[61] A study by Chan et. al. determined that there is a 15% risk of skull fracture at 124g, with a 95% confidence level[62], while Haddadin et al, found a 5% risk of an AIS ≥3 at 72g and a risk of 20% of an AIS ≥3 at 88g[63]. Another factor that might be considered in evaluating the use of 200 g's as the threshold limit for ASTM F1292 is that data has indicated that on average, a Gmax score of 150 is equivalent to a HIC score of 1000.[64] This could well be a factor in the final selection for Gmax for ASTM F1292.

Clearly concussions and AIS >4 thresholds are not the same. It must be determined as to whether the goal is the prevention of the AIS >4 or the TBI before a thresholds for both Gmax and HIC_{15} can be established in a standard for children in a playground. Concussion studies have mostly been related to data collected from teenagers, college athletes and reconstructions of concussions sustained by NFL players. Children pose a unique challenge, because their brains are still developing and more susceptible to the effects of concussion[65] and children and high school athletes recover less rapidly than NFL players[66].

There have been two major studies, termed the NFL study, using a small sample of injured players and specific concussion incidents, and the HITS (head impact telemetry system) studies, involving a large number of athletes and thousands of head impacts. Although the conclusion of concussions in football

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[58] Mertz HJ, Injury Risk Assessment Based on Dummy Response, Nauham and Melvin 2nd Edition, Accidental Injury, Chapter 5, Springer 2002, pp 89

[59] Mertz HJ, Injury Risk Assessment Based on Dummy Response, Nauham and Melvin 2nd Edition, Accidental Injury, Chapter 5, Springer 2002, pp 89

[60] Mertz HJ, Injury Risk Assessment Based on Dummy Response, Nauham and Melvin 2nd Edition, Accidental Injury, Chapter 5, Springer 2002, pp 90

[61] Kleiven S, Finite Element Modeling of the Human Head, Department of Aeronautics, Royal Institute of Technology, 2002, p15

[62] Chan P, Lu Z, Rigby P, Takhounts E, Shang J, Yoganandan N, Pintar F, Development of a Generalizes Linear Skull Fracture Criterion, 2007, 910

[63] Haddadin S, Albu-Schaffer A, Hirzinger G, Safety Analysis for a Human-Friendly Manipulator, Springer Science & Business Media, 2010, p241

[64] Shorten M, Phd, Discuss impact Performance of Playground Surfaces on November 8, Standardization News, ASTM , Movember 2002,

[65] Halstead et. al., Clinical Report – Sport-Related Concussion in Children and Adolescents, American Academy of Pediatrics, Sept. 2010, p597

[66] Manko S, NFL Player Show More Rapid Recovery from Concussion than High School Players, University of Pittsburgh Medical Center, Jan. 2006

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are somewhat different, they both consider the incidence of the concussion to be well below the 200 Gmax thresholds for turf in ASTM standards, Interestingly both of these studies have consistently included the HIC values as very important in understanding the incidence of concussion.[67] This might be a surprise to some involved with the athletic turf, either natural or synthetic as turf is only tested to Gmax with either a the "Clegg Hammer" or the "F355-A device"[68]. These devices can only record Gmax, due to the flat shape of the impacting surface. It has been shown that the "F355-A device" is bias in favour of thin soft surfaces, whereas when the data is adjusted to provide an HIC, these thin systems demonstrate a significantly higher injury risk than the thicker systems with infill and/or padding underlayment.[69]

The NFL study, as it has become to be known, reconstructed 31 NFL collisions of which the 25 impacts involved concussions[70]. These impacts were modelled and impact data generated at the laboratories of Biokinetics and Associates in Ottawa, Canada using helmeted Hybrid III test devices, with concussions occurring at 98g ±28g and HIC of 250[71]. These results were tested as the object of the study by Zhang et.al.(2004) in which they used finite element (FE) modeling technique and the Wayne State Brain Injury Model (WSUBIM) to determine Gmax, HIC_{15} and rotational acceleration. Their results were that for the injured athlete the mean acceleration was 103 ±30g, the HIC_{15} 441 ±224 and the mean rotational acceleration was 7354 ±2897 rad/s^2.[72] Criticism of using these values is that they have subsequently become known as the injury threshold for concussion rather than exclusively descriptive of the values specifically related to known concussions. This may well be a valid argument, but the evidence demonstrates concussions are taking place at these values for elite athletes with exceptional protective equipment, training and conditioning. This would not likely be the case for the child in the playground or the high school or college athlete.

Another perspective on the risk of concussion is the analysis of injury and impact data collected through the deployment of the HITS data collection devices in football helmets of a large number of athletes. These studies have the ability to collect thousands of impacts and provide data for Gmax, HIC_{15} and rotational acceleration for each impact. At some point there is the inevitable occurrence of the concussion and the impact data prior to the concussion is available for statistical analysis. Through the comparison of impact values for those sustaining concussion and those not, it is possible to determine that there is a 10% risk of concussion for Gmax of 165 and HIC_{15} of 400[73]. This could lead to the conclusion that for some athletes other factors allow them to sustain higher values of peak acceleration without sustaining a concussion; however in a study of 1712 impacts[74], in which no concussions were sustained, there were only 3 impacts greater than 100 g's and only 1 of these was over 120 g's. Other

[67] J. R. Funk, S.M. Duma, S. J. Manoogian, S. Rowson, Biomechanical Risk Estimates for Mild Traumatic Brain Injury, Association for the Advancement of Automotive Medicine, October 2007, p1

[68] Shorten M PhD, Himmelsback JA, Sport surfaces and the risk of traumatic brain injury, Biomechanica LLC, 2002, p59

[69] ShortenM PhD, Himmelsback JA, Sport surfaces and the risk of traumatic brain injury, Biomechanica LLC, 2002, p63

[70] Pellman EJ, Viano DC, Tucker AM, Casson IR, Waeckerle JF, Concussion in professional football – Recontruction of game impact and injuries, Neurosurgery, 2003, PP799-814

[71] Pellman EJ, Viano DC, Tucker AM, Casson IR, Waeckerle JF, Concussion in professional football – Reconstruction of game impact and injuries, Neurosurgery, 2003, PP799-814

[72] Zhang L, Yang KH, King AI, A Proposed Injury Threshold for Mild Traumatic Brain Injury, ASME, April 2004, p228

[73] Rowson S, Brolinson G, Goforth M, Dietter D, Duma S, Linear and Angular Head Acceleration Measurements in Collegiate Football, ASME, June 2009, p1

[74] Rowson S, Brolinson G, Goforth M, Dietter D, Duma S, Linear and Angular Head Acceleration Measurements in Collegiate Football, ASME, June 2009, p3

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than these 3 impacts the study would tend to lend credibility to NFL values to for value below which no concussion is anticipated. This same study shows a high correlation between increasing peak acceleration[75] and increases in the angular acceleration, indicating that if rotational acceleration is a factor in concussions, the increasing g value could be a predictor of the angular acceleration.

These studies introduce an additional criterion to the traditional linear acceleration for Gmax and impact duration of the HIC, with the rotational acceleration. Rotational acceleration recognizes that most impacts, particularly between athletes that the impact will be from a side or glancing and there will be an impact that is not strictly coup/countercoup, but will allow the brain to move rotationally within the skull and can cause shearing of the neurons axon, damaging the protective myelin coating and disrupting communication between cells[76].

The HITS instrumented helmets have increased the data available to coaches, trainers and team physicians in the monitoring of potential risk of head injury to their athletes. Data collected over the 4 year period from spring 2005 to fall 2008 resulted in 71,390 impacts with a total of 55 concussions being caused at Gmax 107 ±31, HIC_{15} 272 ±213 and 7,079 rad/s^2 ± 3,408 rad/s^2.[77] In a presentation to the Federal Interagency Conference on TBI, Beckwith presented additional data for the six years 2005 to 2010 in which there are now 90 concussions and they are occurring at a peak g of 117 ±33 and 7266 rad/s^2 ±3,400.[78] The HITS helmets indicate that there are impacts greater than the mean g's for those sustaining concussions. These results must give all professionals; particularly those in states that have a legal responsibility for the prevention of concussions in their athletes, an exceptional opportunity meet their responsibilities.

The data, whether it is from the NFL study or the HITS results, shows that a threshold exists between 70 and 150 g's where concussions are occurring, and the median is likely to fall in the 105 to 110g range. These values are significantly lower than the threshold for the playground surface as required in ASTM F1292 of 200 g's.

On the basis of the data, this would translate that the Gmax of a play surface when tested should be less than between 105 and 110 and from the automotive requirements for HIC_{15} should be less than 570 for the structures intended for children 2-5 years and less than 700 for children 5-12 years. These values would prevent the AIS >4 head injury.

Implications for Long Bone Injuries

Beyond the discussion of concussion, there is a documented need "to evaluate various protective surfaces in terms of reducing fractures to the wrist, lower arm and elbow"[79] by Tinsworth (2001). Long bone injuries are common on playgrounds. The ASTM F08.63 sub-committee has frequently stated that these types of injuries would be a realistic expectation given the current allowable impact values. The F2223 Guide states in Section 7 that is directed at the F1292 impact attenuation Standard that "It should

[75] Rowson S, Brolinson G, Goforth M, Dietter D, Duma S, Linear and Angular Head Acceleration Measurements in Collegiate Football, ASME, June 2009, p3
[76] Ogilvie M, The Brain, Why a Blow to the head is a big deal, The Toronto Star, April 9, 2011. pIN 1 & 5
[77] Beckwith JG, Chu JJ, Crisco JJ, McAllister TW, Duma SM, Brolinson PG, Greenwald RM, Severity of Head Impacts Resulting in Mild Traumatic Brain Injury, p2
[78] Beckwith JG, Severity of Head Impact Preceding Diagnosis of MTBI, Federal Interagency Conference on TBI, June 2010, abstract
[79] Deborah K. Tinsworth, Joyce E. McDonald, Special Study; Injuries and Deaths Associated with Children's Playground Equipment, US CPSC, April 2001, piii

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be recognized that serious injuries (for example, long bone injuries, etc.) might occur even though the playground surfacing system meets the requirements of Specification F1292."[80] A recent study of children 1 to 17 years and running from 2003 to 2006 in Washington, DC, concluded that "Playground falls and minor trauma are implicated in the majority of childhood fractures" and "the prevention strategies should target playground safety."[81] As a result it can be expected that long bones will continue to the broken unless changes are made to the cause of the injury, the surface upon which the child lands.

Although it is not within the scope of this paper to provide a long investigation of the causes and impact attenuation factors related to long bone injuries, it might be instructive to review some of the literature to determine if the changes to impact attenuation standards in playgrounds would also have a positive outcome in relation to long bones. Studies related to primarily arm fracture in the playground and taking impact attenuation into consideration have primarily been in Canada and Australia, while there is a wealth of biomechanical fracture information in the United States within the study of injuries related to child abuse. It is interesting that depending upon the circumstances of the location of the injury taking place it could be considered as "just another playground injury" or child abuse. In 1995 in Montreal, Canada impact values were recorded at 102 playgrounds where 110 serious injuries to the head and fractures (AIS 2 or 3) were investigated. The Gmax that was recorded and the risk of serious injury was 3 times greater for impact values above 200g than for values as compared to surfaces yielding a Gmax <150 and 1.8 times greater in the 150-199g category as compared to the <150 category[82]. This resulted in a recommendation that playground standards should consider reducing the Gmax threshold from 200g to 150g or less[83]. Sherker and Smith (2004) found that of the arm fractures studied that >86% occurred in playgrounds that were compliant to playground impact standards. Additionally the mean impact values were 119g and the HIC was 615.[84] Sherker et. al. (2005) found that peak acceleration of 100G and above represented an arm fracture risk approximately 1.5 times that of less than 100g for tests conducted using both an instrument headform compliant with standards and an anthropometric child arm load dummy.[85] Fiissel et. al. (2007) indicated that >85% of major fractures in children occur in playgrounds seen at the Hospital for Sick Children in Toronto, Canada for the years 1995 to 2002 and the injury risk was 3.91 times greater for a fall from playground equipment than from standing height on the playground.[86]

Consideration of the forces and dynamics of fractures as a result of child abuse have generated considerable literature and take into consideration the same factors that are considered in playground falls to the surface when considering the load applied to the bone prior to fracture. Pierce et. al. (2003) found that softer surfaces result in less energy available for the injury, while harder surfaces are more efficient in transmitting energy to the body and that fracture morphology is a direct reflection of the

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[80] [80] ASTM F2223-03, Standard Guide for ASTM Standards on Playground Surfacing, ASTM, 2003, p2

[81] Ryan LM, Teach SJ, Searcy K, Singer A, Wood R, Wright JL, Chamberlain, JM, Epidemiology of Pediatric Forearm Fractures in Washington, DC, Journal of Trauma-Injury Infection & Critical Care, October 2010, pS200

[82] S. Laforest, Y Robitaille, D Lesage, D Droval, Surface characteristics, equipment height and the occurrence and severity of playground injuries, Injury Prevention 201; 7;35-40, p5

[83] S. Laforest, Y Robitaille, D Lesage, D Droval, Surface characteristics, equipment height and the occurrence and severity of playground injuries, Injury Prevention 201; 7;35-40, p1

[84] Sherker S, Ozanne-Smith J, Are current playground safety standards adequate for preventing arm fractures, MJA, June 2004, p564-565

[85] Sherker S, Ozanne-Smith J, Rechnitzer G, Grzebieta R, Out on a limb: risk factors for arm fractures in playground equipment falls, Injury Prevention, 2005, 120-124

[86] Fiissel D, Pattison G, Howard A, Severity of playground fractures: playground equipment versus standing fall heights, Injury Prevention, 2005, P338

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degree and direction of forces and the ability of the tissue to resist those forces.[87] This area of research for long bone injury is very similar to the automobile information relating to the head injury and is a valid source of information, data and performance requirements that could be applied to the playground injury.

The conclusion that can be made from the studies on long bone fractures, primarily in playgrounds, is that a reduction of both the Gmax to below 105 to 110g and the HIC to below 570 for children structures under 5 and below 700 for play structures intended for 5-12 year olds would have a beneficial outcome on the incidence of long bone injuries. However, these changes would not eliminate this type of injury, but result in a measureable reduction with added benefit towards head.

Impact Velocity and Free Fall Height

For all of the twenty-five NFL players, the average impact velocity was 9.3 ± 1.9 m/sec[88] or the equivalent of a 4.4m (14.4') free-fall drop. The average velocity at impact for the three concussions sustained as "head to surface" without prior player impact was 6.8 m/sec[89] or equivalent to a 2.36M (7.75') free-fall drop. The average Gmax for the twenty-five helmeted football players was 98 g's and for the head to turf players was 123g's. The 200g threshold that the unprotected child is currently required to endure on a playground surfaces when compared to football players is unreasonable. It must be remembered that when a child falls from a 9' slide deck, that child would be travelling at 7.3m/sec (24 ft/sec) or 16.4mph from the deck height and 8.5m/sec (27.7ft/sec) or 18.61 mph at the time they hit the surface. The only protection at that point is the ability of the surface to absorb the impact to the Gmax of <105 and HIC of less than 700.

Discussion around a new threshold based on AIS ≥4 will have to include a consensus decision as to whether a concussion would be considered as a "debilitating injury" and therefore affect the determination of the threshold for ASTM F1292 and the CPSC document #325. An alternative would be a recommendation to users and caregivers of the user of the playground, as to what impact values could result in a concussion and that appropriate diagnosis and treatment processes can be performed.

Considerations for Implementation of Playground Surfacing Performance for ASTM F1292 and the CPSC Handbook

There are a number of factors that have come to light in relation to the ASTM F1292 and CPSC Public Playground Safety Handbook. The primary concern is the effectiveness of the current Gmax and HIC values and what the goals for any new threshold for compliance to the standard should become. Although most of the impact intervals related to playground surfaces are generally less than 10ms as in the automotive studies, there are some surfaces designed for very low Gmax and HIC at above 10' (3m) that have demonstrated intervals in the 16 to 18ms and there must be a determination if the HIC calculation shall be constrained to 15ms for HIC_{15}. Additionally it might be time to consider a different HIC_{15} threshold for playground surfacing under play structures intended of children under 3 based in the HIC_{15} threshold for the 3 year old dummy of 570 and the HIC_{15} value for the 6 year old dummy being

[87] Pierce MC, Bertocci GE, Vogeley E, Moreland MS, Evaluating long bone fractures in children: a biomechanical approach in illustrative case, Child Abuse & Neglect, January 2003, p508 & p520
[88] Viano, DC, Casson IR, Pellman EJ, Concussion in Professional Football: Biomechanics of the Struck Player – Part 14, Neurosurgery, August 2007, p318
[89] Viano, DC, Casson IR, Pellman EJ, Concussion in Professional Football: Biomechanics of the Struck Player – Part 14, Neurosurgery, August 2007, p319

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700. For the Gmax there is injury data to support a reduction as a minimum to 125g. The separation of impact attenuation values by age group would be consistent with both ASTM F1487 and the CPSC as age and anthropological differences are use as rationale factors that determine technical requirements for play structures aimed at each group.

Implications for playgrounds, owner/operators, manufactures and installers

Currently there are a number of laws and mandates in Canada and the United States that reference compliance to ASTM F1292 for the installed playground surface. Many of these such as the 2010 American with Disabilities Act Accessibility Standards reference the ASTM F1292 by specific revision date[90] and this would not affect current playgrounds and a surface complying with the new thresholds would automatically comply with the mandated document. This would be the same in Canada, where in 2007, the CSA Z614 raised the fall height for play structures with platforms to the height above the platform of the minimum barrier[91] and specifically stated that the surface testing was to take place to the standard of the day it was installed[92].

It is important to review the financial impact and availability of products related to the acceptance of the recommended changes in Gmax and HIC. Currently products such as engineered wood fibre, loose rubber nuggets, mulch and crumb, some specific sands and gravels when installed and maintained to their appropriate depth will at not or very little additional cost meet the new requirements. A review of many of the websites for these types of products indicate significantly better performance than the existing thresholds from heights that are significantly higher than the fall heights of most playground equipment at 8' to 10' (2.4 to 3M). As a result the change should not be an economic burden on the suppliers of these products or their purchasers. As always, there will continue to be a requirement to maintain the depth, cleanliness and performance of the surface to the standard over the life of the playground. For owner/operators currently budgeting and providing this maintenance, there will not be a new financial impact on their facilities. It is however recognized that the reality of playgrounds is that playground surfacing is not installed or maintained to an adequate level. This was confirmed by the Consumer Federation of America in 2002 when 75% of playgrounds reviewed were not adequate and this was an improvement over their review in 2000 when it was found that 80% were inadequate.[93]

The greatest problem for products with relation to the new thresholds will be the unitary or synthetic surfacing systems that have traditionally been used in playgrounds. A review of numerous surfacing manufacturer's websites for products supplied around the world, these products are manufactured and "rated" to provide a specific critical height, which is then closely matched to the maximum fall height of the play structures. Most manufacturer/supplier/installers offer a variety to critical height products to be matched to fall heights as lower and higher play structures are installed. There is an increasing cost to the end user of these products directly related to increase in thickness of the product and critical height. As a result the cost of synthetic surfacing under a playground will be affected by the required critical height of the surfacing. Although each surface system will have variations, the effect would be to take a surface with a current F1292 critical height and match it to a 30 to 40% lower fall height.

[90] U.S. Department of Justice, 2010 ADA Standards, September 15, 2010, page 10
[91] CSA Z614-07, Children's Playspace and Equipment, Canadian Standards Association, 2007, p48
[92] CSA Z614-07, Children's Playspace and Equipment, Canadian Standards Association, 2007, p15
[93] Weintraub R, Cassady A, Playing It Safe: The Sixth Nationwide Safety Survey of Public Playgrounds, the Consumer Association of America, June 2002, P1

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Synthetics in playgrounds have been either rubber tiles or poured-in-place surfaces. The tiles or mats are the combination of rubber granules or crumb bonded with a polyurethane binder in ambient conditions or inside a mould and pressed. These mats typically appear to be a sheet of rubber over legs or stanchions and the impact attenuation is based on the ability of the upper rubber sheet to compress and flex and the legs to compress as force is applied. Tiles are almost always manufactured in a factory and transported to the playground for installation. Poured-in-place on the other hand involves the mixing of the rubber crumb and/or shreds with a polyurethane binder and placing it in multiple layers in the playground. Detailed information with regard to the poured-in-place surfacing can be found in ASTM F2479, Standard Guide for Specification, Purchase, Installation, and Maintenance of Poured-In-Place Playground Surfacing.[94] Generally the impact attenuation these surfaces is achieved through the ability of the wear course or top mat to indent and bend and the ability of the cushion layer to allow the upper layer to penetrate under load.

As with everything, innovation has brought new synthetic surface systems to the playground. Many of these have been developed to provide critical heights in excess of the traditional playground fall heights. Some of these systems are combinations of artificial turf, rubber matting, and PVC or other sheet type materials that can be applied in multiple layers. Since these systems have been designed for both long term compliance to ASMF1292 and the ADA standard, it is likely that improve critical height technology has been built into the new design. An example of this would the Liberty Tire SMARTE product that is generally 4” in thickness with the results for the ASTMS F1292 laboratory testing shown in table 4.

Product	Drop height	Gmax	HIC
LTR Smarte	8’ (2.4M)	55	226
LTR Smarte	12’ (3.65M)	79	372
LTR Smarte	14’ (4.25M)	94	517
LTR Smarte	15’ (4.57M)	98	625
LRT Smarte	17’ (5.2M)	97	672

Table 4. Liberty Tire Recycling SMARTE ASTM F1292 test result.[95]

Some existing materials and new surface products on the market have a demonstrated capability of meeting the new performance requirements. Clearly the proposed changes to ASTM F1292 for Gmax and HIC can only benefit the reduction of risk of severe injuries due to falls to the playground, thereby improving opportunities for play while minimizing risk of injury. When considering the cost to the child of a life altering injury or the cost of rehabilitation or the cost to an already over-burdened healthcare system, the benefits resulting from making these changes should not be overlooked. These changes will also have the effect of allowing designers to design while truly protecting children from unforeseen fall hazards in the playground environment and allowing children to experience greater challenges in the playground.

Acknowledgements

[94] ASTM, ASTM F2479-07, Standard Guide for Specification, Purchase, Installation and Maintenance of Poured-In-Place Playground Surfacing, 2007

[95] IPEMA Surfacing Material Reports F1202 TUV SUD America Inc., reports QI1101998, QI1008015, QI1005055

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ASTM F355 Standard Test Method for Impact Attenuation of Playing Surface Systems and Materials, ASTM International, West Conshohocken, PA, USA, 1995, 2001, 2010

ASTM F1292, Standard Specification for Impact Attenuation of Surfacing Materials Within the Use Zone of Playground Equipment, ASTM International, West Conshohocken, PA, USA, 1991, 1993, 1999, 2004, 2009

ASTM F1487, Standard Consumer Safety Performance Specification for Playground Equipment for Public Use, ASTM International, West Conshohocken, PA, USA, 2001, 2005, 2007

ASTM F1936, Standard Specification for Impact Attenuation of Turf Playing Systems as Measured in the Field, ASTM International, West Conshohocken, PA, USA, 1998, 2007, 2010

ASTM F1951, Standard Specification for Determination of Accessibility of Surface Systems Under and Around Playground Equipment, ASTM International, West Conshohocken, PA, USA, 1999

ASTM F2223, Standard Guide for ASTM Standards on Playground Surfacing, ASTM International, West Conshohocken, PA, USA, 2003, 2009, 2010

ASTM F2479, Standard Guide for Specification, Purchase, Installation and Maintenance of Poured-In-Place Playground Surfacing, ASTM International, West Conshohocken, PA, USA, 2007, 2010

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2011-12 NCAA Drug Testing: Collection Crew Evaluation

Response Status: 364 responses out of 644 send outs = 57% response rate

SITE COLLECTION DEMOGRAPHICS

Enter the month of the Collection Date:

Month/Year	n	%	Month/Year	n	%
Aug-11	10	3%	Jan-12	32	9%
Sep-11	51	14%	Feb-12	78	21%
Oct-11	63	17%	Mar-12	38	10%
Nov-11	42	12%	Apr-12	41	11%
Dec-11	5	1%	May-12	4	1%

Enter the year of the Collection Date:

	n	%
2011	172	47%
2012	191	52%

Select the men's sport(s) tested during this collections process. (Check all that apply)

Sport	n	%	Sport	n	%
Baseball	261	74%	Rifle	0	0%
Basketball	25	7%	Rugby	0	0%
Bowling	0	0%	Sailing	0	0%
Crew	0	0%	Skiing	0	0%
Cross Country	8	2%	Soccer	19	5%
Equestrian	0	0%	Swimming/Diving	5	1%
Fencing	0	0%	Tennis	3	1%
Football	271	77%	Track and Field	14	4%
Golf	2	1%	Volleyball	1	0%
Gymnastics	0	0%	Water Polo	3	1%
Ice Hockey	8	2%	Wrestling	13	4%
Lacrosse	11	3%			

Select the women's sport(s) tested during this collections process. (Check all that apply)

Sport	n	%	Sport	n	%
Basketball	20	16%	Rifle	0	0%
Bowling	0	0%	Sailing	0	0%
Crew	4	3%	Skiing	0	0%
Cross Country	2	2%	Soccer	24	19%
Equestrian	0	0%	Softball	22	17%
Fencing	0	0%	Squash	0	0%
Field Hockey	3	2%	Swimming/Diving	10	8%
Golf	1	1%	Tennis	7	6%
Gymnastics	5	4%	Track and Field	10	8%
Ice Hockey	2	2%	Volleyball	15	12%
Lacrosse	8	6%	Water Polo	3	2%

DRUG TESTING CREW PERFORMANCE

Please rate your level of satisfaction with the following items conducted by the on-site NCAA Drug Testing Crew:

	Very Satisfied	Satisfied	Somewhat Satisfied	Dissatisfied	Very Dissatisfied
Set-up on time	83%	11%	0%	1%	6%
Knowledge of the drug testing process	83%	11%	1%	0%	6%
Professional demeanor	82%	11%	1%	1%	5%
Cohesiveness/teamwork	80%	14%	1%	0%	6%
Communication with institutional representatives	82%	11%	1%	0%	6%
Communication with student-athletes	78%	15%	1%	0%	6%
Consistency of specimen collection process	83%	11%	0%	0%	5%
Confidentiality of specimen collection process	84%	10%	0%	1%	5%
Addressing questions and concerns	81%	12%	1%	0%	6%
Overall performance of the NCAA drug testing crew	82%	11%	1%	0%	6%

Please indicate your level of agreement with the following statements about the on-site NCAA Drug Testing Crew:

	Agree	Disagree
The drug testing plan outlined prior to the on-site visit was clear.	99%	1%
The drug testing plan outlined prior to the on-site visit was practical.	98%	2%
Communication between the crew and myself prior to the on-site visit was sufficient.	99%	1%

2012-13 NCAA Banned Drugs

The NCAA bans the following classes of drugs:

a. Stimulants
b. Anabolic Agents
c. Alcohol and Beta Blockers (banned for rifle only)
d. Diuretics and Other Masking Agents
e. Street Drugs
f. Peptide Hormones and Analogues
g. Anti-estrogens
h. Beta-2 Agonists

Note: Any substance chemically related to these classes is also banned.
The institution and the student-athlete shall be held accountable for all drugs within the banned drug class regardless of whether they have been specifically identified.

Drugs and Procedures Subject to Restrictions:

a. Blood Doping.
b. Local Anesthetics (under some conditions).
c. Manipulation of Urine Samples.
d. Beta-2 Agonists permitted only by prescription and inhalation.
e. Caffeine if concentrations in urine exceed 15 micrograms/ml.

NCAA Nutritional/Dietary Supplements Warning:

Before consuming any nutritional/dietary supplement product, review the product with your athletics department staff!

- Dietary supplements are not well regulated and may cause a positive drug test result.
- Student-athletes have tested positive and lost their eligibility using dietary supplements.
- Many dietary supplements are contaminated with banned drugs not listed on the label.
- **Any product containing a dietary supplement ingredient is taken at your own risk**.

It is your responsibility to check with the appropriate athletics staff before using any substance.

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Note to Student-Athletes: There is no complete list of banned substances.
Do not rely on this list to rule out any supplement ingredient.
Check with your athletics department staff prior to using a supplement.

Some Examples of NCAA Banned Substances in Each Drug Class

Stimulants:

amphetamine (Adderall); caffeine (guarana); cocaine; ephedrine; fenfluramine (Fen); methamphetamine; methylphenidate (Ritalin); phentermine (Phen); synephrine (bitter orange); methylhexaneamine, etc.

exceptions: phenylephrine and pseudoephedrine are not banned.

Anabolic Agents – (sometimes listed as a chemical formula, such as 3,6,17-androstenetrione)

boldenone; clenbuterol; DHEA (7-Keto); nandrolone; stanozolol; testosterone; methasterone; androstenedione; norandrostenedione; methandienone; etiocholanolone; trenbolone; etc.

Alcohol and Beta Blockers (banned for rifle only):

alcohol; atenolol; metoprolol; nadolol; pindolol; propranolol; timolol; etc.

Diuretics (water pills) and Other Masking Agents:

bumetanide; chlorothiazide; furosemide; hydrochlorothiazide; probenecid; spironolactone (canrenone); triameterene; trichlormethiazide; etc.

Street Drugs:

heroin; marijuana; tetrahydrocannabinol (THC); synthetic cannabinoids (eg. spice, K2, JWH-018, JWH-073)

Peptide Hormones and Analogues:

growth hormone(hGH); human chorionic gonadotropin (hCG); erythropoietin (EPO); etc.

Anti-Estrogens :

anastrozole; tamoxifen; formestane; 3,17-dioxo-etiochol-1,4,6-triene(ATD), etc.

Beta-2 Agonists:

bambuterol; formoterol; salbutamol; salmeterol; etc.

Any substance that is chemically related to the class, even if it is not listed as an example, is also banned!

Information about ingredients in medications and nutritional/dietary supplements can be obtained by **contacting the Resource Exchange Center, REC, 877-202-0769 or www.drugfreesport.com/rec password ncaa1, ncaa2 or ncaa3.**

It is your responsibility to check with the appropriate athletics staff before using any substance.

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5. The NCAA does not restrict the grounds for an appeal, but an institution bringing an appeal must comply with the requirements set forth in Section 8.0. Any challenge must be supported by written documentation, testimonial evidence and or corroborated factual details. Appeal considerations are outlined below:

a. Procedural Challenge: Either the institution or student-athlete may challenge any procedure relating to the collection or testing of the subject samples. If the institution or student-athlete proves it is *more likely than not* that any substantiated problem with the collection or testing procedures *materially affects* a sample's integrity, the drug-test appeal subcommittee may find that no doping violation has occurred.

b. Knowledge Challenge: The student-athlete is responsible for all substances consumed. However:

i. If the institution or the student-athlete demonstrates that the student-athlete was not aware they had been administered (defined as placed into the student-athlete's system directly or through food or drink) a substance by another person that later is found to have contained a banned ingredient, then the drug-test appeal committee may determine that no violation has occurred. In this situation, the student-athlete must show that he or she both did not know *and* could not reasonably have known or suspected (even with the exercise of utmost caution) that he or she had been administered by a third party a substance that is later found to have contained a banned ingredient. or

Deleted: proves it is *more likely than not*

ii. If the institution or the student-athlete demonstrates that the student-athlete asked specific and reasonable questions about a particular substance, medication or product of the appropriate athletics administrator and the athletics administrator assured the inquiring student-athlete that the substance does not contain a banned ingredient, then the drug-test appeal committee may determine that no violation has occurred. In this situation, the student-athlete must show that he or she both did not know *and* could not reasonably could have known or suspected (even with the exercise of utmost caution) that the information provided by staff was erroneous. IN the case where the substance, medication or product reviewed and approved for use by the institution does contain a banned substance, this may result in an institutional violation.

Deleted: proves that it is *more likely than not*

c. Reduction of Penalty Based on Mitigating Factors: The following will not be considered mitigating factors in a drug test appeal: the type or amount of banned substance detected through the drug test; evidence of the student-athlete's good character; the degree of remorse demonstrated by the student-athlete; and the degree to which the banned substance may or may not affect athletic performance.

The drug-test appeal committee may reduce the current legislative sanction to the first 50 percent of the regular season in all sports if the season of competition has not yet begun for that student-athlete or a minimum of the equivalent of the next 50 percent of a season of competition in all sports if the student-athlete tests positive during his or her season of competition when circumstances might indicate a reduction is warranted. For example:

i. Where it is shown that the institution's drug education program was inadequate and such shortcomings influenced the student-athlete's judgment regarding the propriety of taking a specific product (a reduction of penalty normally will not be available in the case of admitted street drug use); or

Deleted: it is *more likely than not* that

Deleted: *more likely than not*

ii. A student-athlete's ability to discern he or she was using a banned substance was due to circumstances beyond the student-athlete's control.

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Deleted: significantly impaired

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2012-13 Drug-Testing Exceptions Procedures (Medical exceptions)

Educational Material

- NCAA Guidelines to Document ADHD Treatment with Banned Stimulant Medications -1/30/09
  - Reporting Form to Support the Diagnosis of ADHD
  - Memo - 2/03/09
  - Frequently Asked Questions: Addendum to the January 2009 Guidelines - Updated 7/1/10
  - Student-Athlete Educational Poster: Report All Medicines - 4/24/09
  - Clinicians seeking more guidance on conducting a comprehensive clinical assessment please review this Sample Evaluation Format -1/30/09
- Video- Medical Exception Policy - 3/17/08
- Medical Exception Documentation Case Examples- 3/17/08

Procedures

The NCAA list of banned drug classes (NCAA Bylaw 31.2.3) is composed of substances that are generally reported to be performance enhancing and/or potentially harmful to the health and safety of the student-athlete.

The NCAA recognizes that some banned substances are used for legitimate medical purposes. Accordingly, the NCAA allows exception to be made for those student-athletes with a documented medical history demonstrating the need for regular use of such a drug. Exceptions may be granted for substances included in the following classes of banned drugs: anabolic agents*, stimulants, beta blockers, diuretics, anti-estrogens, beta-2 agonists and peptide hormone* (see subpart 2 below).

Procedures for Exceptions

1. Alternative non-banned medications for the treatment of various conditions exist and should be considered before an exception is pursued.

2. In the event that the student-athlete and the physician (in coordination with sports-medicine staff at the student-athlete's institution) agree that no appropriate alternative medication to the use of the banned substance is available, the decision may be made to continue the use of the medication. However, the use of an ***anabolic agent or peptide hormone must be approved by the NCAA before the student-athlete is allowed to participate in competition while taking these medications**. The institution, through its director of athletics, may request an exception for use of an anabolic agent or peptide hormone by submitting to the NCAA medical documentation from the prescribing physician supporting the diagnosis and

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treatment. (contact mwilfert@ncaa.org)

3. The institution should maintain documentation that supports the use of medication in the student-athlete's medical record on campus. The documentation can be a letter or copies of medical notes from the prescribing physician that documents how the diagnosis was reached, and that the student-athlete has a medical history demonstrating the need for regular use of such a drug. The letter should contain information as to the diagnosis (including appropriate verification of the diagnosis), medical history and dosage information.

4. Unless requesting a review for the medical use of an anabolic agent or peptide hormone, a student-athlete's medical records or physicians' letters should not be sent to the NCAA unless requested by the NCAA. Also, the use of any substance need not be reported to the drug-testing crew at the time of NCAA drug testing.

5. In the event that a student-athlete is tested by the NCAA and tests positive for a substance for which the institution desires an exception, normal procedures for reporting positive test results will be followed (See NCAA Drug-Testing Program Protocol Section No. 8.0). The institution, through its director of athletics, may request an exception at the time of notification of the positive drug test (A sample) by submitting to The National Center for Drug Free Sport the prescribing physician's letter and any other medical documentation demonstrating the need for regular use of the drug (see subpart 3 above) which the institution wishes to have the NCAA consider. A medical exception will be considered by the NCAA and the student-athlete will remain eligible during this time if the institution has provided medical documentation to Drug Free Sport before the "B" sample is reported as positive, confirming the positive finding. If the institution fails to provide medical documentation to Drug Free Sport before the "B" sample is reported as positive to the institution, the student-athlete will be withheld from competition until such time the documentation is received, reviewed and the medical exception granted. (contact mbockelman@drugfreesport.com)

6. Requests for exceptions will be reviewed by the chair of the drug-testing and drug-education subcommittee and the physicians of the NCAA Committee on Competitive Safeguards and Medical Aspects of Sports.

7. The NCAA will inform the director of athletics regarding the outcome of the exception request. In the event that the exception is not granted, the institution may appeal this action according to Section 8.0 of the drug-testing protocol.

Sources:
NCAA Drug Testing Program, 2012-13

The National Collegiate Athletic Association
July 2012

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TO BE DISTRIBUTED

Form 11-3a **Academic Year 2011-12**

Student-Athlete Statement – NCAA Division I

For:	Student-athletes.
Action:	Sign and return to your director of athletics.
Due date:	Before you first compete each year.
Required by:	NCAA Constitution 3.2.4.6 and NCAA Bylaw 14.1.3.
Purpose:	To assist in certifying eligibility.
Effective Date:	This NCAA Division I Student-Athlete Statement/Drug-Testing Consent form shall be in effect from the date this document is signed and shall remain in effect until a subsequent Division I Student-Athlete Statement/Drug-Testing Consent form is executed.

Student-Athlete: _____
(Please print name)

Name of your institution: _____

This form has seven parts:

1. A statement concerning eligibility;

2. A Buckley Amendment consent;

3. An affirmation of status as an amateur athlete;

4. A statement concerning the promotion of NCAA championships and other NCAA events;

5. Results of drug tests;

6. Previous involvement in NCAA rules violation(s); and

7. An affirmation of valid and accurate information provided to the NCAA Eligibility Center and admissions office, including ACT or SAT scores, high school attendance, completion of coursework and high school grades.

If you are an incoming freshman, you must complete and sign Parts I, II, III, IV, V and VII to participate in intercollegiate competition. If you are an incoming transfer student or a continuing student, you must complete and sign Parts I, II, III, IV, V and VI to participate in intercollegiate competition.

Before you sign this form, you should read the Summary of NCAA Regulations, or another outline or summary of NCAA legislation, provided by your director of athletics or his or her designee or read the bylaws of the NCAA Division I Manual that deal with your eligibility. You are responsible for knowing and understanding the application of all NCAA Division I bylaws related to your eligibility. If you have any questions, you should discuss them with your director of athletics or your institution's compliance officer, or you may contact the NCAA at 317/917-6222.

The conditions that you must meet to be eligible and the requirement that you sign this form are indicated in the following bylaws of the Division I Manual:

- Bylaws 10, 12, 13, 14, 15, 16, 18.4 and 31.2.3.

Part I: Statement Concerning Eligibility.

By signing this part of the form, you affirm that, to the best of your knowledge, you are eligible to compete in intercollegiate competition.

You affirm that your institution has provided you a copy of the Summary of NCAA Regulations, or another outline or summary of NCAA legislation, or the relevant sections of the Division I Manual and that your director of athletics (or his or her designee) gave you the opportunity to ask questions about them. You affirm that you have knowledge of and understand the application of NCAA Division I bylaws related to your eligibility.

You affirm that you meet the NCAA regulations for student-athletes regarding eligibility, recruitment, financial aid, amateur status and involvement in gambling activities.

You affirm that you are aware of the NCAA drug-testing program and that you have signed the current NCAA Drug Testing Consent Form.

You affirm that all information provided to the NCAA, the NCAA Eligibility Center and the institution's admissions office is accurate and valid, including ACT or SAT scores, high school attendance, completion of coursework and high school grades, as well as your amateur status.

You affirm that you have reported to the director of athletics or his or her designee of your institution any violations of NCAA regulations involving you and your institution.

You affirm that you understand that if you sign this statement falsely or erroneously, you violate NCAA legislation on ethical conduct and you will further jeopardize your eligibility.

Name (please print)	Date of birth	Age
Signature of student-athlete	Home address (street or P.O. Box)	
Date	Home city, state, and zip code	
Sport(s)		

_____

Part II: Buckley Amendment Consent.

By signing this part of the form, you certify that you agree to disclose your education records.

You understand that this entire form and the results of any NCAA drug test you may take are part of your education records. These records are protected by the Family Educational Rights and Privacy Act of 1974 and they may not be disclosed without your consent.

You give your consent to disclose only to authorized representatives of this institution, its athletics conference (if any) and the NCAA, except as permitted in the Drug-Testing Consent form, the following documents:

1. This form;

2. Results of NCAA drug tests and related information and correspondence;

3. Results of positive drug tests administered by a non-NCAA national or international sports governing body;

4. Any transcript from your high school, this institution, or any junior college or any other four-year institution you have attended;

5. Precollege test scores, appropriately related information and correspondence (e.g., testing sites, dates and letters of test-score certification or appeal), and where applicable, information relating to eligibility for or conduct of nonstandard testing;

6. Graduation status;

7. Your social security number and/or student identification number;

8. Race and gender identification;

9. Diagnosis of any education-impact disabilities;

10. Accommodations provided or approved and other information related to any education-impact disabilities in all secondary and postsecondary schools;

11. Records concerning your financial aid; and

12. Any other papers or information pertaining to your NCAA eligibility.

_____

You agree to disclose these records only to determine your eligibility for intercollegiate athletics, your eligibility for athletically related financial aid, for evaluation of school and team academic success, for awards and recognition programs highlighting student-athlete academic success (e.g., Elite 88), for purposes of inclusion in summary institutional information reported to the NCAA (and which may be publicly released by it), for NCAA longitudinal research studies and for activities related to NCAA compliance reviews and athletics certification. You will not be identified by name by the NCAA in any such published or distributed information. This consent shall remain in effect as long as any issues regarding the purposes listed above exist.

You also agree that information regarding any infractions matter in which you may be involved may be published or distributed to third parties as required by NCAA policies, bylaws or procedures.

_____ _____

Date Signature of student-athlete

Part III: Affirmation of Status as an Amateur Athlete.

You affirm that you have read and understand the NCAA amateurism rules.

By signing this part of the form, you affirm that, to the best of your knowledge, you have not violated any amateurism rules since you requested a final certification from the NCAA Eligibility Center or since the last time you signed a Division I student-athlete statement, whichever occurred later.

You affirm that since requesting a final certification from the NCAA Eligibility Center, you have not provided false or misleading information concerning your amateur status to the NCAA, the NCAA Eligibility Center and the institution's athletics department, including administrative personnel and the coaching staff.

_____ _____

Name (please print) Date

_____

Signature of student-athlete

Part IV: Promotion of NCAA Championships, Events, Activities or Programs.

You authorize the NCAA [or a third party acting on behalf of the NCAA (e.g., host institution, conference, local organizing committee)] to use your name or picture in accordance with NCAA Bylaw 12.5, including to promote NCAA championships or other NCAA events, activities or programs.

_____

Name (please print)

Signature of student-athlete Date

Part V: Results of Drug Tests.

1. Future positive test – all student-athletes sign.

Should I test positive for a substance banned by the NCAA and/or by a non-NCAA national or international sports governing body; violate their drug-testing protocol; or fail to show for their drug test, at any time after I sign this statement, I acknowledge I must report the results to my director of athletics.

Name (please print) Date

Signature of student-athlete

2. Positive test by NCAA or other sports governing body -- sign either A or B.

A. No positive drug test.

I affirm that I have never tested positive for a substance banned by the NCAA and/or by a non-NCAA national or international sports governing body; or violated their drug-testing protocol; or failed to show for their drug test.

Name (please print)

Signature of student-athlete Date

B. Positive drug test.

I have tested positive for a substance banned by the NCAA and/or by a non-NCAA national or international sports governing body; or violated their drug-testing protocol; or failed to show for their drug test. Should I subsequently transfer, I am obligated to report this to the transferring institution.

_____

_____
Name (please print)

_____
Signature of student-athlete

_____	_____	_____
Date of test	Organization conducting test	Substance

Are you currently under such a drug-testing suspension? Yes _____ No _____

Part VI: Incoming Transfers – Previous Involvement in NCAA Rules Violation(s).

Have you previously attended a four-year NCAA Divisions I, II or III institution?

Yes _____ No _____

If yes, what is the name(s) of the institution(s)? _____

Are you aware of any NCAA violations you were involved in while previously attending an NCAA institution?

Yes _____ No _____

If yes, did this violation result in your being withheld from competition while attending your previous institution?

Yes _____ No _____

If you answered yes to either of the above questions, please provide an explanation.

_____

_____

_____

_____

Part VII: Incoming Freshmen – Affirmation of Valid ACT or SAT Score.

You affirm that, to the best of your knowledge, you have received a validated ACT and/or SAT score. You agree that, in the event you are or have been notified by ACT or SAT of the possibility of an invalidated test score, you will immediately notify the director of athletics of your institution. You affirm that all information provided to the NCAA, the NCAA Eligibility Center and institution's admissions office is valid and accurate, including high school attendance, completion of coursework and high school grades. You affirm that you did not fraudulently earn your qualifying ACT or SAT score by having someone else take the test for you, copying answers from another person taking the test, etc.

_____ _____
Name (please print) Date

_____
Signature of student-athlete

What to do with this form: Sign and return it to your director of athletics or his or her designee before you first compete. This form is to be kept in the director of athletics' office for **six years**.

Any questions regarding this form should be referred to your director of athletics or your institution's NCAA compliance staff, or you may contact the NCAA at 317/917-6222.

TO BE DISTRIBUTED

ASTM Meeting Brief Recap from US Lacrosse.

As first time ASTM meeting attendees, USL staff (myself, Melissa Coyne and Steve Stenersen) and guests were pleasantly surprised to join about 65 other meeting attendees at the women's lacrosse headgear meeting. Led by Co-chairs, Eric Darnell and Steve Moore (Cascade), this first ever meeting kicked off on a positive note after introductions indicated that room was filled with experts with technical/ engineering and design backgrounds, representatives from helmet and lacrosse industry, sports medicine and women's lacrosse leaders.

USL was then invited to present an overview of our position on headgear in women's lacrosse (see attached) – where we explained in great detail the differences between men and women's lacrosse – and expressed serious concerns about introducing head protection developed for other sports (football, men's lacrosse) into women's lacrosse. Dr. Trey Crisco also presented a preliminary report on his study looking at Head Accelerations in Women's Lacrosse – which provoked a very interesting discussion amongst several other attendees who had also done some preliminary lab tests of sticks hitting head forms and had some different results (B. Brine and D. Halstead).

Without a doubt, the research that Crisco, Halstead and Brine were discussing was central to our understanding of the force of impact we wanted to mitigate through an equipment standard – but the differences in their early lab testing outcomes made it clear to us that we needed to wait until the middle of the summer to see the results of Crisco's field and lab studies to move forward with establishing any sort of unique headgear standard.

We also discussed at great length the mechanisms of injury we are most concerned with in women's lacrosse: stick-to-head and ball-to-head impacts, as there are the most commonly seen in our research. We also emphasized that we are not focusing on preventing concussions (as no helmet is able to do that) or facial injuries (as the number facial injuries reported is quite small) – but on trying to lessen the severity of the head injuries caused by those two mechanisms. Based on that information, the discussion took a turn away from the head and towards the stick and the ball – as possible factors impacting the injury outcomes. In summary, the following questions, points were raised:

1) What if we were able to design and test a ball that had less mass, but retained the same size properties as the existing ball standard; in other words - can we make the ball lighter?
2) If the primary mechanism of head injury is stick-to-head contact, and if the stick were to hit the head at its most inflexible and heaviest point (the throat of the head, versus the tip of the head), why couldn't we look at developing a padded protective sleeve for that area of the stick to soften the impact, if contact is made with the head or other body part?
3) If we developed a lighter ball, would we need to revisit the eyewear standard and the goalie helmet and face shield standard for women's lacrosse?
4) Because we still have the rule allowance in the women's game for soft headgear, we want to continue to pursue a standard for that allowance (based on research) – recognizing that if we were able to develop a lighter ball, padding for sticks, and a

standard for allowable headgear – that we would not need or want to introduce helmets in women's lacrosse.

These points/questions resulted in the establishment of 2 additional working groups (ball and stick) to join the existing women's lacrosse eyewear and headgear subcommittees, and the possible creation of a women's lacrosse subcommittee to oversee the ball and stick group. US Lacrosse asked to be represented on all of these subcommittee and working groups, and we expect to continue to lead the discussion on all fronts. The next in-person ASTM meeting is not slated until November, but we expect to get word from the ASTM Headgear co-chairs with next steps and work group assignments within the next few weeks…and we'll be working actively to address the questions and points raised above throughout the summer.
Melissa and I will also be attending the June NOCSAE meeting, where women's lacrosse helmets and ball standard are slated for discussion on the agenda.

Ann Kitt Carpenetti
Managing Director of Game Administration
US Lacrosse
113 W. University Parkway
Baltimore, Maryland 21210
P: (410) 235-6882 x117
F: (410) 366-6735
acarpenetti@uslacrosse.org

www.uslacrosse.org
www.laxmagazine.com

US Lacrosse is the national governing body of lacrosse. Through responsive and effective leadership, we provide programs and services to inspire participation while protecting the integrity of the sport.

US Lacrosse Presentation for ASTM Meeting:
Head Protection in Women's Lacrosse May 9, 2012
Co-Presented by: Steve Stenersen, President & CEO
Ann Kitt Carpenetti, Managing Director of Game Administration and
Melissa Coyne, Women's Game Director

Head Injuries in Women's Lacrosse: Why we are concerned…

Most Common Injuries in Lacrosse

- Overall, **moderate risk sport**; minor strains, sprains & bruises
- Non-contact **ankle and knee ligament sprains**, sustained while cutting and dodging, are most common: girls (21%); boys (16%)
- **Knee injuries**, including ACL tears, are the leading cause of lost game and practice time in both games. (**Hootman et al JAT 07)**
- **Head & face injury**, including concussion, less frequent but an important issue for both games
  - Body-to-body or body-to-ground contact in the boys' game
  - Inadvertent stick- or ball-to-head contact in the girls' game
  - More frequent in game than practice

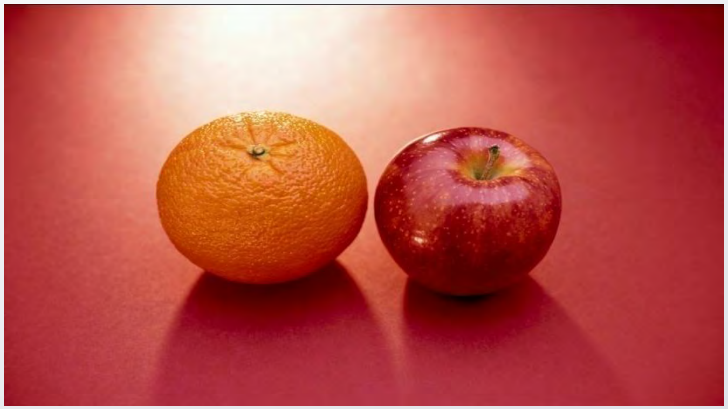
Concussion and Head Injuries

- Growing issue for both games and all youth sports
  - Media/public scrutiny
- Significant organizational focus
  - Research – injury surveillance
  - Sport Science & Safety Committee expertise
- Interventions
  - Carding Surveys (women's game)
  - Rule Changes & Enforcement (both games)
  - Education – coach, official, parent (both games)
  - Request to NOCSAE to help develop standard for women's lacrosse-specific headgear (6/11)
  - Development of ASTM Working Group on women's lacrosse headgear (1/12)

Why Does Womens' Lacrosse Need a Unique Headgear Standard?

Men's Lacrosse

Women's Lacrosse:



Two Different Games

What Do The Two Versions of Lacrosse Look Like?

- http://youtu.be/ZUQy9XV0RGg Girls

- http://youtu.be/B2oTENT1upo Boys

Women Lacrosse and Men's Lacrosse are Two Distinct Versions of the Sport

	GIRL'S RULES	BOY'S RULES
CONTACT	Aggressive stick checking and body contact are illegal	Aggressive stick checking & body contact are legal
	Cross Checking is illegal	Cross Checking is illegal
	Any contact with the head from a stick will result in a yellow or red card (time -loss) penalty	While striking an opponent in the head with a stick deliberately and viciously is illegal, contact itself doesn't not constitute a foul if contact to the head is made in an attempt to dislodge a ball, act of passing or shooting or scoop the ball, and is considered a brush.
	Body checks and slashes are major fouls and will result in a yellow or red card (time -loss) penalty.	High hits or any contact initiated by the head will result in an automatic 1, 2 or 3 minute non-releasable penalty.

Equipment Standards are Different in Men's Game vs Women's Game

	GIRL'S RULES	BOY'S RULES
EQUIPMENT	Shallow pocket rule allows for easier ball dislodgement	Deeper pocket rule requires more aggressive checking
	Protective eyewear and mouth guards are required	Mouth guards are required
	Soft head gear and nose guards are optional	Men's lacrosse helmets certified to the NOCSAE standard are required
	Close-fitted gloves are optional	Arm pads, shoulder pads and heavy gloves are required
	Goalie: helmets certified to the NOCSAE standard, chest protectors, mouth guards, gloves, shin pads are required	Goalie: helmets certified to the NOCSAE standard, chest protectors, mouth guards, gloves, shin pads are required

Current NOCSAE Lacrosse Helmet Standards Were Developed Specifically for Men's Lacrosse

- Designed for a collision sport
- Based on football helmet standard and testing (drop test)
- Designed to prevent catastrophic head injury (skull fracture)

Types of Head Injuries in Women's Lacrosse

To date, there have been no reported catastrophic or severe brain injuries in women's lacrosse.

Stick-to-players' head checks are the most common mechanism of concussive injury in women's lacrosse. Prevalence of head injuries and concussions in women's lacrosse are similar to those in women's soccer or ice hockey.

Given the lack of catastrophic head injuries in girls or women's lacrosse, use of men's lacrosse helmets that are specifically designed to meet helmet performance standards for preventing catastrophic head injury (NOCASE ND001 – 11m11 and ND041-11m11) in women's lacrosse is not supported by the epidemiological data.

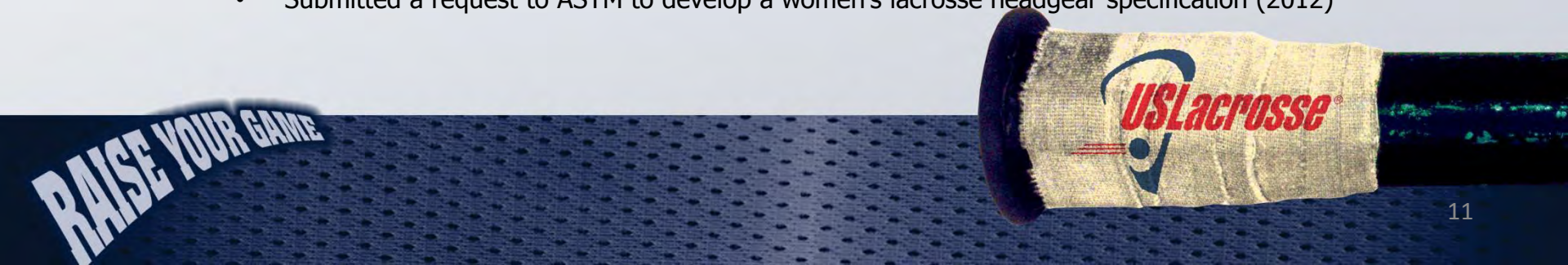
What is US Lacrosse doing to better understand head injuries in women's lacrosse?

Completed Research/Projects

- Descriptive epidemiology of scholastic lacrosse injuries
- Head, face and eye injuries in scholastic and collegiate lacrosse
- Risks and mechanisms of severe injuries among youth, secondary school, collegiate

Current Research/Projects

- Injury data collection - trends in high school lacrosse injuries (Fairfax County, VA)
- Epidemiology of TBI in boys' and girls' high school lacrosse players using video analysis (Fairfax County, VA)
- Risks and mechanisms of severe injuries among youth, high school, collegiate and post-collegiate players using US Lacrosse insurance claims data
- Trends in sports-related concussion incidence at the high school level, 1998 -2007
- RIF ball development (under consideration)
- Produced the first lacrosse-specific **Concussion Education Video**
- Effectiveness of the Women's Lacrosse Protective Eyewear Mandate in the Reduction of Eye Injuries (Lincoln) paper published in the AJSM
- Video/data collection at USL youth (U11, U13 and U15) regional events of 50 boys' and girls' games. Epidemiology of lacrosse injuries among youth players.
- Impact forces associated w/ concussive injury - video vs. mouth guard telemetry
- Submitted a request to NOCSAE to develop a women's lacrosse headgear specification (2011)
- Submitted a request to ASTM to develop a women's lacrosse headgear specification (2012)



College & High School Lacrosse Concussion Data

Trends in Concussion Incidence in High School Sports

A Prospective 11-Year Study

Andrew E. Lincoln,*† ScD, Shane V. Caswell,‡ PhD, ATC, Jon L. Almquist,§ VATL, ATC, Reginald E. Dunn,* BA, Joseph B. Norris,‖ MD, and Richard Y. Hinton,‖ MD, MPH, PT
Investigation performed at MedStar Health Research Institute, Baltimore, Maryland

Women		
Frequency, n	Percentage	Incidence Rate
111	41.4	0.32
64	23.9	0.18
56	20.9	0.16
25	9.3	0.07
12	4.5	0.03
268	100	0.77

Study evaluated mechanisms of injury in high school & college lacrosse, both boys/men and girls/women

Key Finding: Mechanic associated with concussions in women's lacrosse:

- **HS girls; 49% from stick**
- **College women; 39% contact from stick, 37% contact w/ ball, 14% contact w/ another player**

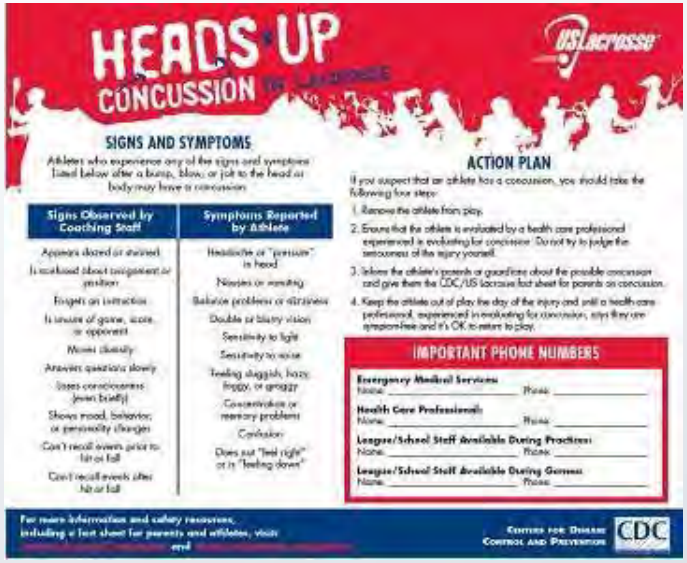
Protective Eyewear Research

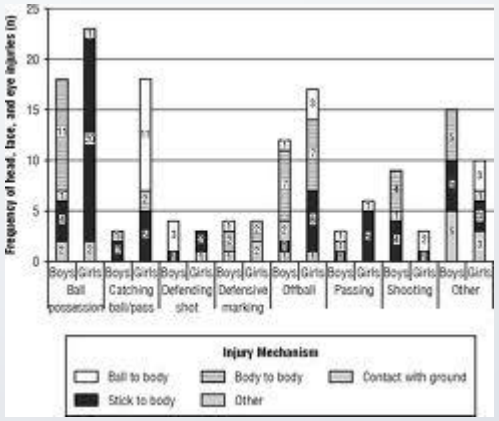
Effectiveness of the Women's Lacrosse Protective Eyewear Mandate in the Reduction of Eye Injuries

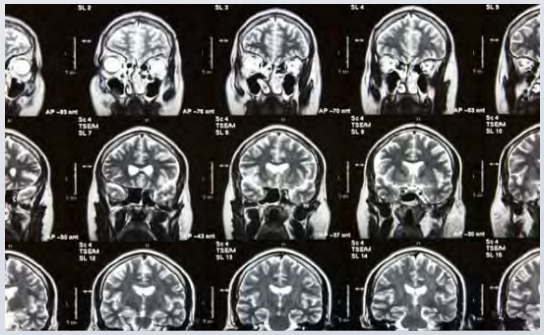
Richard Y. Hinton[1], Andrew E. Lincoln[2], Shane V. Caswell[3], Reginald E. Dunn[2], Mark Clough[1], Jon L. Almquist[4]. [1]*Union Memorial Hospital, Baltimore, MD.* [2]*MedStar Research Institute, Baltimore, MD.* [3]*George Mason University, Manassas, VA.* [4]*Fairfax County Public Schools, Falls Church, VA.*

- **Evaluated effect of eyewear mandate in HS aged athletes**
- **Rate of eye injuries decreased from 0.06 to 0.02 inj / 1000 AE**
- **Rates of other head/face injuries decreased, though rates of concussions increased likely due to increase in awareness and reporting of injury across all sports.**

What Does It All Mean?







Equipment Standards for Headgear Are Important and Needed in Women's Lacrosse

- **Equipment certification and testing standards exist for protective eyewear in women's lacrosse, for men's lacrosse helmets, but not for women's soft headgear, which is allowable by the rules for women's lacrosse.**

- **Since 2010, US Lacrosse has been pursuing the development of a <u>unique and appropriate standard</u> for women's headgear, that considers the rules, mechanism of injuries and integrity of the women's game.**

- **A women's lacrosse headgear standard should be established based on research, an understanding of the women's game and should ultimately provide additional protection to the head from the types of injuries incurred by a stick hitting/ checking a head.**

- **A women's lacrosse headgear standard should also allow for the incorporation of protective eyewear.**

Some Players, Coaches, Parents & Administrators Want Additional Head Protection in Women's Lacrosse

What Would a Women's Lacrosse Headgear Standard Look Like?

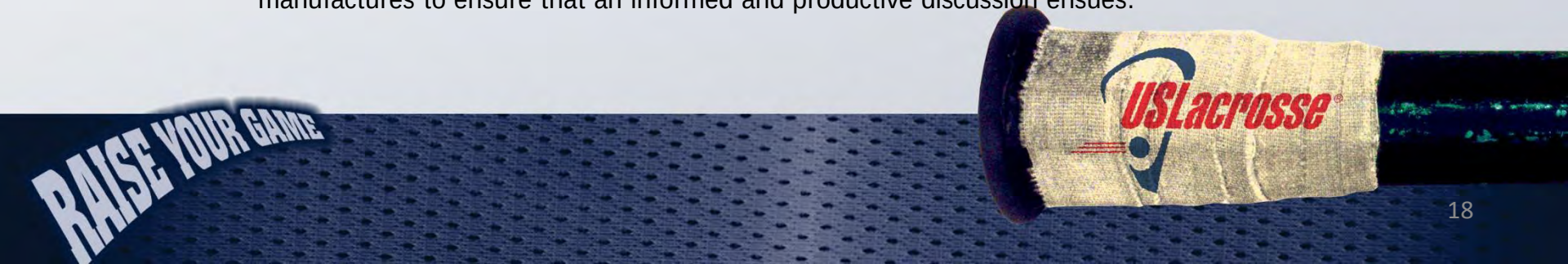
US Lacrosse Request to ASTM: Develop a Headgear Standard Designed Specifically for the Unique Rules and Culture of Women's Lacrosse

- In January of 2012, US Lacrosse turned to the American Society for Testing and Materials (ASTM) F08 Sports Equipment and Facilities Headgear Committee to encourage them to establish a Task Group to help establish a Women's Lacrosse Headgear Standard.

- ASTM has long been recognized as a leader in standards for sports equipment - and has drafted standards used in hockey, baseball, fitness and track, bicycling and skiing. They also wrote the standard for women's lacrosse eyewear.

- The ASTM Women's Lacrosse Headgear Task Group's stated goal is: **"To create a standard for women's lacrosse headgear that provides an appropriate level of protection, does not increase the risk of injuries to other players, and maintains the rules, culture and traditions of this unique women's sport."**

- The first meeting of the ASTM's Task Group on Women's Lacrosse Headgear is on Wednesday May 9, 2012 in Phoenix, Arizona, and leaders from US Lacrosse, NCAA, NFHS and the women's lacrosse and sports medicine community will join sporting goods manufactures to ensure that an informed and productive discussion ensues.



Prevention of Head Injuries and Some Concussions – IS THIS POSSIBLE?

- Enforce existing rules
- Rule changes to decrease hits to the head and increase penalties for dangerous play.
- Coach proper techniques
- Officials Consistently enforce rules
- Modified checking and limited contact in youth play
- Education regarding concussion and removal from play when signs/sx exist

Current mouth guards, helmets, and other protective equipment DO NOT prevent concussive injury but….Could a women's lacrosse specific head protection reduce the incidence and severity of head injuries, from stick to head impact?

We must answer this question.

And the $25,000 Question is… Once a Standard is Developed For Women's Lacrosse Headgear, Will They Be Mandated?

Our Commitment As the Governing Body of Lacrosse in the United States is to Balance Player Safety While Maintaining the Integrity of the Game…and We Will Keep That Commitment by Leading Efforts to:

- ✓ **Develop, Deploy and Encourage sport specific education & training of all coaches & officials**
- ✓ **Fund and monitor ongoing injury research to inform our decisions**
- ✓ **Make rule changes, where necessary**
- ✓ **Increase parent and consumer awareness about the safe-play best practices in lacrosse**
- ✓ **Pursue protective equipment interventions, as deemed appropriate and necessary**

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Thank You!

Please send any questions to
mcoyne@uslacrosse.org or acarpenetti@uslacrosse.org

Visit www.uslacrosse.org for more information on rules, equipment and player safety information on women's lacrosse

RAISE YOUR GAME

STANDARD PERFORMANCE SPECIFICATION FOR NEWLY MANUFACTURED LACROSSE BALLS

NOCSAE DOC (ND) 049-05

Prepared By

NOCSAE

NATIONAL OPERATING COMMITTEE
ON STANDARDS FOR ATHLETIC EQUIPMENT

Proposed June 2005

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1. Scope

1.1. This standard specification establishes performance requirements in the weight, compression deflection load, circumference, and coefficient of restitution for new lacrosse balls as supplied by manufacturers.

1.2. This standard has three levels of performance. *Level 1 is designated for children with the lowest skill level. Level 2 is designated for youths with moderate skill levels. *Level 3 is designated for youths and older with advanced skill levels.
*Levels 1 and 3 are being developed.

1.3. This standard does not purport to address all of the safety problems associated with its use. It is the responsibility of the user of this standard to establish appropriate safety and health practices and determine the applicability of regulatory limitations prior to use.

2. Referenced Documents

2.1. STANDARD DROP TEST METHOD AND EQUIPMENT USED IN EVALUATING THE PERFORMANCE CHARACTERISTICS OF PROTECTIVE HEADGEAR, NOCSAE DOC.001

2.2. STANDARD PROJECTILE IMPACT TEST METHOD AND EQUIPMENT USED IN EVALUATING THE PERFORMANCE CHARACTERISTICS OF PROTECTIVE HEADGEAR/PROJECTILES, NOCSAE DOC.021

2.3. STANDARD PERFORMANCE SPECIFICATION FOR NEWLY MANUFACTURED LACROSSE HELMETS WITH FACEGUARD, NOCSAE DOC.041

2.4. STANDARD PERFORMANCE SPECIFICATION FOR NEWLY MANUFACTURED YOUTH BASEBALLS, NOCSAE DOC.027

3. Sample Size

3.1 At least one dozen (12) balls of each model must be tested.

4. Conditioning

4.1 Prior to testing, condition each ball for a period of not less than 24 hrs at laboratory conditions which shall be at a temperature of 70 ± 5 °F (21 ± 5 °C) and a relative humidity of 50 ± 20 %. Record the temperature to the nearest degree and the relative humidity to the nearest percent at the time of testing on the report form for each test series.

5. Test Procedures

5.1 Ball Mass

5.1.1 Place the ball on the center of the scale and record its weight.

5.2 Ball Circumference

5.2.1 Measure the diameter of each ball twice. The readings must be taken 90° apart.

5.2.2 Compute the circumference of each ball at each diameter and divide by two, the result is the ball's average circumference.

5.3 Ball Compression (C-D)

5.3.1 Place the ball in the compression device to align the central axis of the device with the center of the ball.

5.3.2 Activate the compression press until the upper plate is in contact with the ball with less than 0.05 lbs. load applied to the ball.

5.3.3 Set the compression displacement gage reading to zero.

5.3.4 Compress the ball to a displacement of 25% at a constant rate of 1 in per minute ± 3% and record the load force applied.

5.4 Ball COR

5.4.1 The ball-throwing device is set to deliver the ball at 60.0 ± 3% mph to the strike plate. Each ball is propelled at the strike plate a minimum of 6 times and a maximum of 12 times with a 30 s rest time between impacts. The inbound reading and the rebound reading are recorded for each impact. Only those impacts with the inbound velocity within 60.0 ± 2 mph, and not deviating greater than 6 ins. while traveling through the light gates before and after impact with the strike plate, shall be valid.

5.4.2 Ball COR is calculated as the rebound velocity divided by the inbound velocity. The average of six valid COR for each ball is used to determine the individual ball COR.

6. Performance Requirements

6.1 Level 1.
*To be developed.

6.2 Level 2.

6.2.1 The ball must be of a smooth (no seams), solid, elastomeric material. Embossing of data and logos is allowed.

6.2.2 The weight value must be within 5.0 to 5.25 oz.

6.2.3 The circumference value must be within 7.75 to 8 in.

6.2.4 The C-D at 25% displacement must be within 195 lbs ± 15lbs (99.45 kg ± 6.8 kg).

6.2.5 The COR values must be within 0.60 to 0.70.

6.3 Level 3.
*To be developed.

7. Labels and Warnings

7.1 In addition to the requirements of section 9.7 of NOCSAE DOC.021, Projectile labeling, labeling must also include the following.

7.2 A permanent, replica of the NOCSAE seal must appear legibly on the exterior of the packaging.

Manufacturer Certifies

MEETS NOCSAE

LAX

STANDARD®

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7.3 The level shall be permanently indicated on each ball and on the exterior of the packaging.

7.4 The approximate (intended) circumference shall be permanently indicated on the ball and on the exterior of the packaging.

7.5 The approximate (intended) weight of the ball shall be permanently indicated on the ball and on the exterior of the packaging.

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Physical and Mechanical Properties of Various Field Lacrosse Balls

Joseph J. Crisco[1], Elizabeth I. Drewniak[2], Martin P. Alvarez[2], and David B. Spenciner[1]

[1]Brown Medical School and Rhode Island Hospital
[2]Brown University

Although the sport of lacrosse has evolved dramatically over the last few decades and is presently the fastest growing team sport in the United States, the current specifications for balls date back to 1943. The purpose of this study was to see if various commercially available field lacrosse balls meet these specifications and to determine additional mechanical properties of the ball that may more completely characterize ball performance. Eight models from several manufacturers were tested. Seven models were designated for game play, while one model was promoted as a practice ball. In accordance with the specifications, the mass, circumference, and rebound height were recorded for one dozen balls from each model. The load required to compress the balls 0.0125 m and the coefficient of restitution (COR) with an incident speed of 26.80 m/s were also determined. We found that some balls met several of the specifications, but none of the models had every ball meet all the specifications. For the two measures of ball liveliness, rebound height had a weak correlation with COR. Ball compression loads averaged about 750 N over most models, but were almost 85% less for the practice model. It appears that current governing body specifications are outdated, as no ball model we tested met these specifications. The determination of ball liveliness at more realistic speeds should also be taken into account. Since balls with low compression loads can pass through face protectors worn by lacrosse players, the sport's governing bodies may wish to consider a specification on ball compression.

Key Words: coefficient of restitution, compression, NCAA/NFHS specifications, sports

The sport of lacrosse has experienced a rapid increase in popularity since the 1970s and is one of the fastest growing team sports in the United States ("About

[1]Dept. of Orthopaedics, Brown Medical School and Rhode Island Hospital, CORO West, Suite 404, 1 Hoppin St., Providence, RI 02903; [2]Division of Engineering, Brown University, Providence, RI 02912.

Lacrosse," 2004). Referred to as the "little brother of war," field lacrosse originated as a Native American training exercise for combat. Although it was played by many Native American nations, the play of the game varied. Some played on fields that were miles long, others played on horseback, while still others competed with a stick in each hand (Fisher, 2002). In 1867 in Montreal, Dr. William George Beers first standardized the game and developed the modern playing rules. The first American intercollegiate tournament was held at Westchester Polo Grounds in New York some 14 years later. In the U.S., lacrosse remained limited to a few exclusive eastern academic intuitions until the 1970s. Field lacrosse has evolved into a high-speed game of passing, catching, and shooting for men and women of all ages. Women's lacrosse in North America appears to have originated in 1902, with rules similar to those of the men's game. In the following decades, however, men's and women's versions of lacrosse have become dramatically dissimilar. Despite differences in the rules of play, levels of contact, number of players, and field dimensions, both men's and women's lacrosse use the same ball.

Ball liveliness, typically quantified by the coefficient of restitution (COR), can have a profound influence on play and is tightly regulated in sports such as baseball and softball. Ball COR is defined as the ratio of the rebound speed to the incident speed; a COR of unity defines an ideally elastic ball, while a COR value of zero characterizes a ball that does not bounce at all. Although the rules for ball performance vary, even in the same sport, softballs and baseballs are generally required to have COR values ranging from 0.43 to 0.57, as measured with an incident speed of 26.80 m/s against a flat, rigid target (American Society of Testing Materials [ASTM], 2004a). This relatively high incident speed is used to approximate game conditions because it has been well established that the COR values of softballs and baseballs vary with incident speed, and that the values at lower speeds do not directly correlate with values at higher speeds (Adair, 1994; Heald & Pass, 1994; Hendee, Greenwald, & Crisco, 1998). Higher ball compression loads (i.e., stiffness) have also been shown to be associated with higher ball COR values and increased ball liveliness (Haake, Carre, & Goodwill, 2003). Further details on the dynamics of collisions of sports balls and COR have been presented by Cross (1999a).

The lacrosse rules of the National Collegiate Athletic Association (NCAA) and National Federation of State High School Associations (NFHS) specify ball mass (between 141.8 g and 148.8 g), circumference (between 0.186 m and 0.203 m), and rebound height (Winters, 2002). When dropped from a height of 1.829 m onto a concrete floor at a temperature of 18.3 °C to 21.1 °C, the ball must rebound to a height between 1.092 m and 1.295 m. The International Federation of Women's Lacrosse Associations (IFWLA) rules have very similar specifications (Ganzenmuller, 2003).

These specifications have remained unchanged since 1943, although the composition of the floor was changed from wood to concrete in 1978. The required drop test with an incidence speed of approximately 6 m/s is intended to quantify ball liveliness, but lacrosse shots can often exceed 40 m/s. Given the known influence of ball speed on COR and the high-speed nature of lacrosse, this low velocity measurement may not be an accurate measure of ball liveliness during actual play. To the best of our knowledge, a comprehensive study of commercially available lacrosse balls has never been undertaken, despite the age of these specifications and the known limitations in measuring ball liveliness via a rebound height test.

The purpose of this study was to determine whether various commercially available ball models conform to the current NCAA/NFHS specifications for lacrosse balls. To further evaluate the physical and mechanical properties of these balls, we measured COR values at a more realistic speed and sought to determine whether the simple drop test correlated with these values. We also measured the compression load of the balls to determine whether these values correlated with COR values. Should governing bodies address concerns of lacrosse balls penetrating facemasks, these compression loads could also serve as reference values.

Methods

The 8 ball models used in this study were either donated by the manufacturer or purchased at a local retail store. All models were described by the manufacturers as "typical" balls used in competitive lacrosse play, except for one, which was marketed as a practice ball. The balls were uniquely numbered and the manufacturer's labeling was recorded. All balls were stored in a humidity controlled chamber set to 45 to 55% and were removed for only short periods of time for testing. All tests were performed at room temperature. Every ball was tested in accordance with the NCAA/NFHS rules. Then, COR tests were conducted on 6 balls of each model and compression tests were conducted on the remaining 6 balls.

Ball mass was measured using a digital balance (Mettler, Hightstown, NJ). Three measurements were made for each ball and the average value was recorded. The circumference of each ball was measured twice, once along the mould parting line and once perpendicular to the parting line using a thin, flexible thread and a ruler. These values were also averaged.

Rebound height was measured after the ball was dropped from a height of 1.892 m onto a concrete floor as described in the ball specifications (Winters, 2002). The drop height and rebound height were measured visually to the nearest 0.006 m using a wooden ruler mounted on the wall. Measurements were made relative to the top of the ball. Each ball was dropped 5 times and the average value was reported as the rebound height. Drops that resulted in a rebound trajectory with a substantial horizontal component were discarded and repeated.

Ball COR was determined by firing each ball from an air cannon (Lansmont Corp., Monterey, CA) into a rigid steel plate using methodology similar to that used for evaluating baseballs and softballs (ASTM, 2004a). Two pairs of laser sensors (Banner Engineering Corp., Minneapolis) were positioned 0.308 m apart and 0.305 m from the rigid plate; they measured ball speed (Heald & Pass, 1994). Six rebound speeds associated with incident speeds of 26.4 to 27.3 m/s were obtained and averaged for each ball. The COR was calculated as the ratio of the rebound speed to incident speed.

Compression testing was performed on the remaining 6 balls from each model. Using test methodology similar to that used for evaluating baseballs and softballs (ASTM, 2004b), we placed each lacrosse ball between two flat rigid plates and compressed 0.0125 m at a rate of 0.025 m/min using a servo hydraulic material tester (Instron Corp., Canton, MA). The compression displacement was chosen to be twice that required by ASTM F1888-02, due to the increased flexibility of lacrosse balls as compared to baseballs or softballs. Load and displacement data were recorded at 100 Hz during this test. Each ball was compressed twice, once along the parting line and once perpendicular to the parting line. The load values at a compression of

0.0125 m were determined and then averaged for each ball. Stiffness was defined as the maximum compressive load divided by the compression distance.

Only pass/fail criteria were considered for the NCAA/NFHS specification measurements. Differences in COR and compression load values between ball models were compared using a one-way analysis of variance (ANOVA) with a Tukey-Kramer multiple comparison post hoc test. A significance level of $p < 0.05$ was set a priori. Linear regression was used across all ball models to assess the correlations between compression values and COR and rebound height and COR.

Results

Seven of the ball models possessed NCAA and/or NFHS lettering on the label. However, the exact wording on each one varied slightly (Table 1). Ball models had smooth surfaces except Models H and B, which exhibited a fine diamond texture and triangular dimples, respectively (Figure 1).

Table 1 Model, Number of Balls, Markings, and Color of Each Ball Studied

Ball model & no. of balls	Markings	Color	Surface
A 12 balls	NCAA specs	White	Smooth
B 12	Official NCAA	White	Triangular dimples
C 12	NCAA specs	White	Smooth
D 12	NCAA specs	Yellow	Smooth
E 12	NFHS, NCAA, CLA specs	White	Smooth
G 12	NCAA, NFHS specs	Pink	Smooth
H 12	NCAA, NFHS specs	White	Fine diamonds
I 12	None	Blue/green	Smooth

Note: The markings were recorded for all governing bodies listed on each ball. All were "typical" balls used in competitive lacrosse play (i.e., games and practices), although Model I was packaged as a practice ball.

Figure 1 — A typical white, smooth lacrosse ball (Model A), a ball with a fine diamond texture (Model H), and a ball with triangular dimples (Model B).

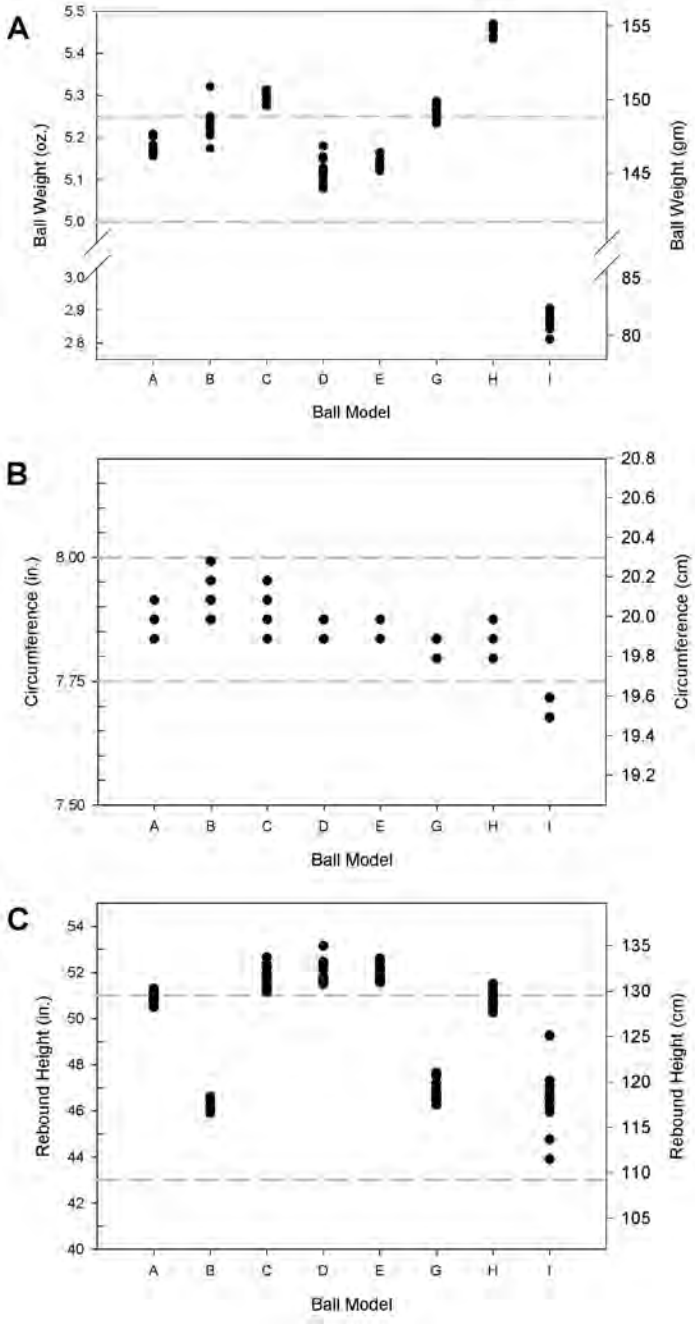

Figure 2 — All balls of 4 models exceeded the limit on ball mass (2A), while the practice model (I) was substantially lighter. All but the practice model were within the specs for circumference (2B). Models B, G, and I were within the specs for rebound height (2C). Dashed lines indicate upper and lower limits of current NCAA/NFHS specs. Data plotted is for all balls tested.

All balls of Models A, D, and E met the NCAA/NFHS mass specifications (Figure 2A). Some balls of Models B and G were within the limit, and the remaining balls were above the limit. All balls of Models C and H were above the mass limit. All balls of Model I, the practice ball, were slightly more than half the mass of the lower limit. All balls from all models, except Model I, were within the circumference limits of the NCAA/NFHS specifications (Figure 2B). All balls of Models B, G, and I were within the limits for rebound height, while only some of the balls in Models A and H were within the limits. The remaining balls of Models A and H, as well as all balls of Models C, D, and E, had rebound heights that were higher than the specified limit (Figure 2C).

The load-displacement curves generated from the compression tests were similar for all balls and were slightly nonlinear with a small hysteresis loop. The load-displacement curves of Models H and I illustrate the range of mechanical behavior from the traditional models to the practice model (Figure 3). Models B and I had compression loads that were significantly less ($p < 0.05$) by 45% and 85%, respectively, than the other 6 models (Figure 4A). The compression loads of these 6 models were approximately within a 200-N range, but most models were statistically different from one another ($p < 0.05$), with the exception of A vs. G, C vs. D, and E vs. G and H.

The average stiffness values were as follows: 594.4 ± 47.3 N/cm (Ball A); 336.3 ± 27.2 N/cm (Ball B); 543.6 ± 18.9 N/cm (Ball C); 511.4 ± 44.2 N/cm (Ball D); 632.9 ± 27.2 (Ball E); 628.2 ± 33.5 N/cm (Ball G); 662.7 ± 30.1 N/cm (Ball H); and 107.5 ± 9.5 N/cm (Ball I). The COR of all balls from Models A, C, D, E, G,

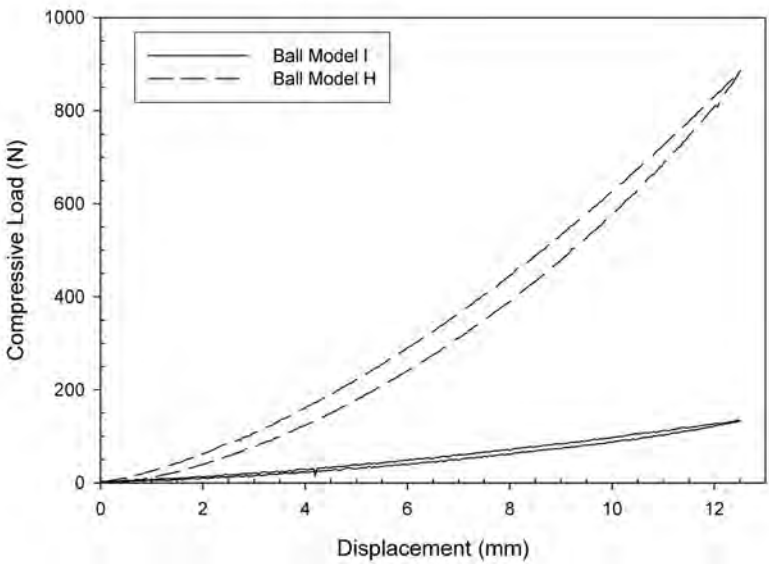

Figure 3 — Load-displacement curves for 2 ball models representing the range of compression behavior. Model H had the highest compression load, and Model I, the practice ball, had the lowest compression load of all balls tested. The reported compression value was the load required to compress the ball to a displacement of 12.5 mm.

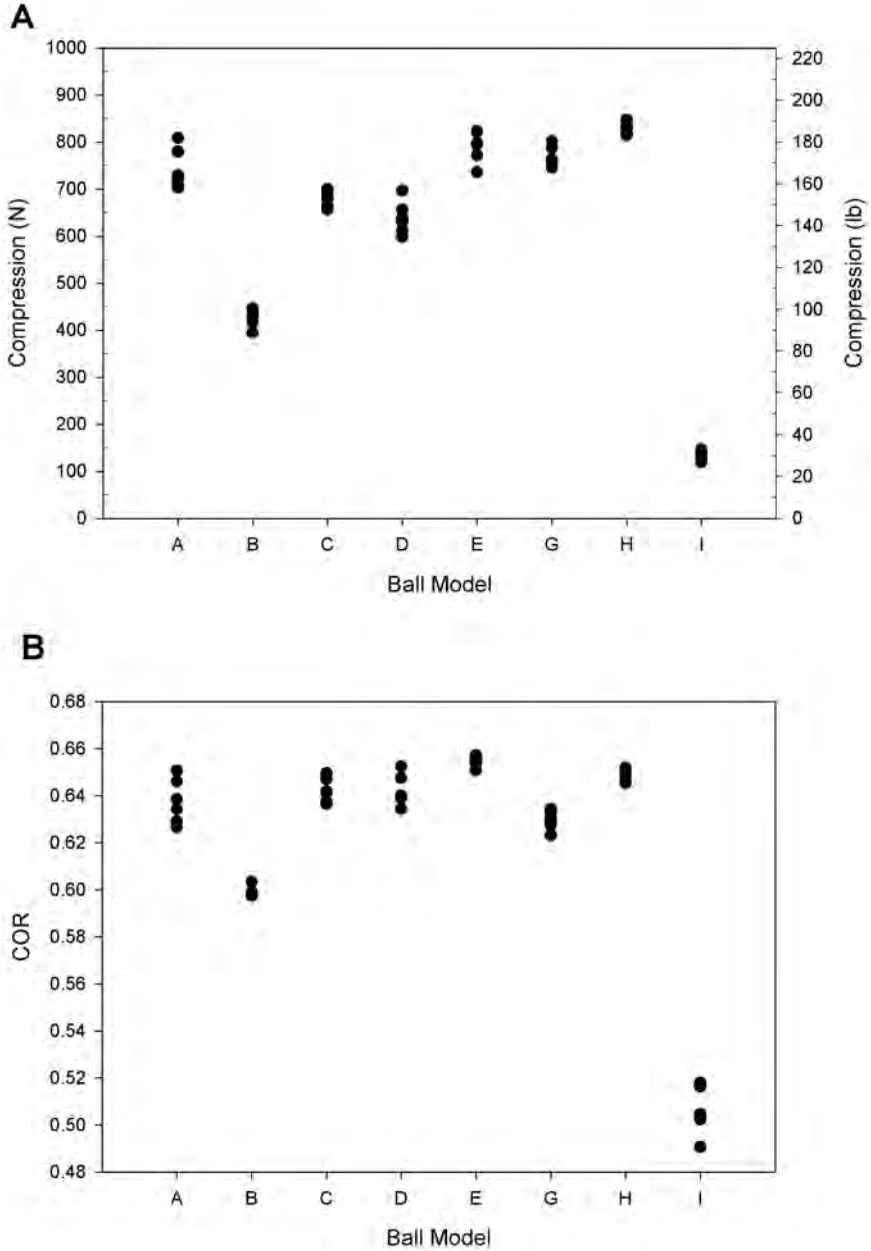

Figure 4 — Compression load of Model B was substantially and significantly less than the 7 other models by approx. 45%, while the compression load of Model I was even less, approx. 85% less than most other models (4A). The COR of the 6 models (A, C, D, E, G, and H) was within 0.62 to 0.66, while the COR of Model B was significantly less than these models and the COR of Model I was over 15% less than the other models (4B). Data plotted is for all balls tested.

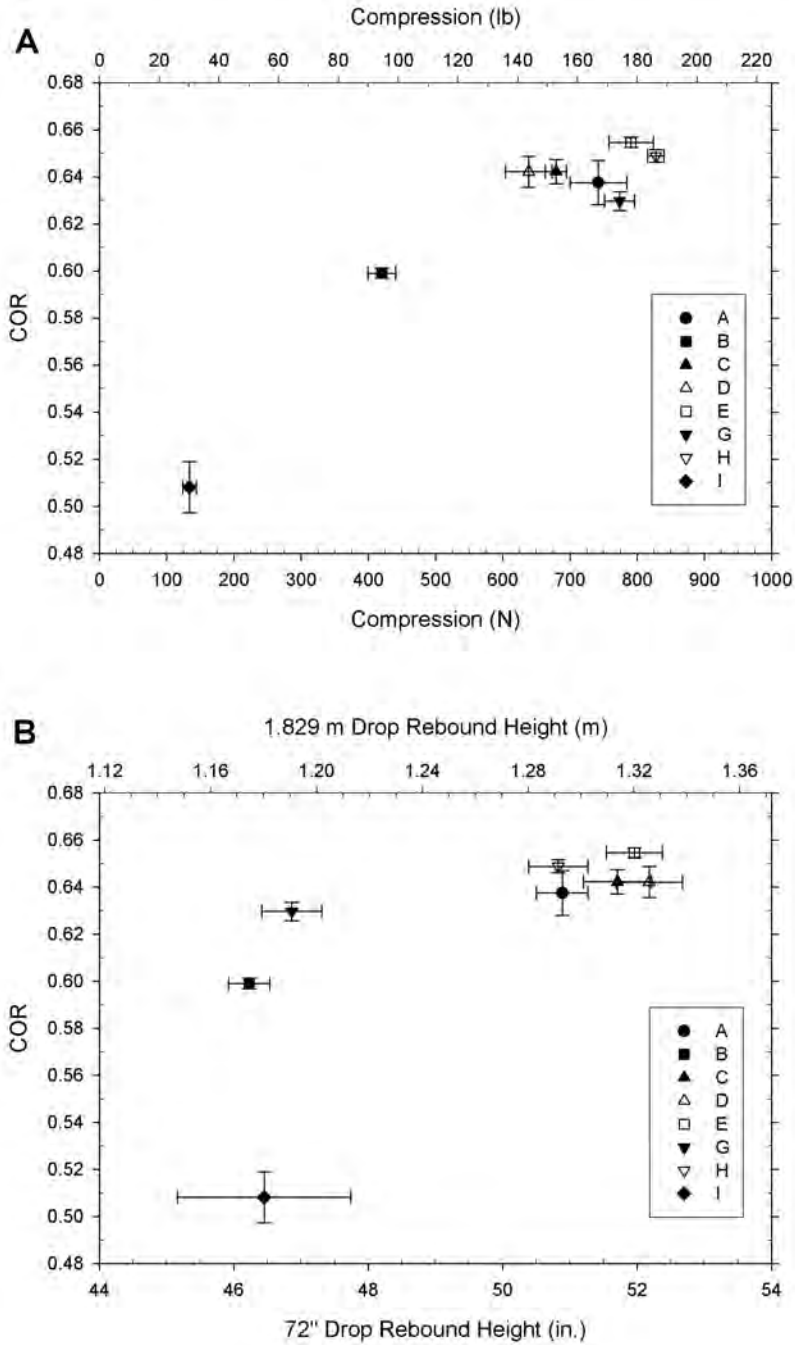

Figure 5 — Ball COR in general increased with increasing compression value (r^2 = 0.90) (5A). Overall, increasing rebound height was associated with an increasing COR (5B). When Model I, the practice ball, was excluded, the correlation was weak (r^2 = 0.73). The mean and one standard deviation of each variable are plotted for each ball model.

and H were within the range of 0.62 to 0.66 and these models were not significantly different from one another (Figure 4B). The COR value 0.599 ± 0.002 of Model B was significantly ($p < 0.05$) less than these 6 models and the COR value 0.508 ± 0.011 of Model I was more than 15% less ($p < 0.05$).

There was a strong correlation between compression load and COR when all ball models were considered ($r^2 = 0.90$) (Figure 5A). Excluding the practice ball lessened this correlation ($r^2 = 0.72$). Conversely, if all ball models were considered, then there was a poor correlation between COR and rebound height ($r^2 = 0.53$) (Figure 5B). However, if the data from the practice ball was not accounted for, the relationship between COR and rebound height was slightly stronger ($r^2 = 0.73$).

Discussion

The purpose of this study was to examine the physical and mechanical properties of lacrosse balls in a laboratory setting in order to compare the results with NCAA/NFHS ball specifications. We examined a variety of commercially available ball models, including one designated for practice play and two with textured surfaces. Current rules for NCAA/NFHS lacrosse balls do not restrict the ball surface, but they do specify limits on ball color (white, yellow, orange, or lime green), mass, circumference, and rebound height. Surprisingly, none of the models had all balls pass these specifications. Overall, the values we measured had reasonably small standard deviations, but it is unknown how other balls of these models would perform.

While the COR of both baseballs and softballs significantly decreases as incident speed increases, this trend is not predictable from a COR measurement at a single speed (Hendee et al., 1998). This finding, in addition to the fact that the drop test is an unrealistically slow speed, was our motivation for measuring the high-speed COR. The methods we used for measuring ball COR are specified in current standards for baseballs and softballs (ASTM, 2004a, 2004b). As one would expect, lacrosse balls were more lively, yet the practice ball had a COR similar to some baseballs and softballs. Rebound height, which is a low-speed measure of ball liveliness, did not correlate well with the COR at 27 m/s, a more realistic speed. Therefore, the current approach of regulating ball liveliness with a drop test may not be sufficient. We measured lacrosse ball COR at only one temperature, so it is not known whether temperature affects lacrosse ball COR. Standards for the testing of baseballs and softball also specify humidity. We studied only a single humidity value, but given the solid rubber composition of lacrosse balls it is unlikely that differing humidity conditions would affect the results.

Using methods similar to those utilized in this study, it has been determined that the static stiffnesses of traditional baseballs range from 2,000 to 3,000 N/cm, baseballs modified for youth play range from 50 to 1,000 N/cm (Hendee et al., 1998), softballs range from 161 to 952 N/cm (Heald & Pass, 1994), and tennis balls range from 109 to 143 N/cm (Cross, 1999b). Most of the lacrosse ball models we tested had stiffness values similar to those for youth baseballs and softballs (Models A, C, D, E, G, and H were all 500–700 N/cm), while Model I, with a stiffness of about 110 N/cm, was more similar to a tennis ball. Unfortunately, direct comparison with the literature is limited by differences in the definition of stiffness. A limitation to our definition of stiffness is that it provides an average value, ignoring the effect of the slack toe region.

It has been shown that the baseball compression load correlates with the ball COR (Hendee et al., 1998). In our study we found a similar correlation: as the compression load of the lacrosse balls increased, the COR did as well. This trend of increasing COR with increasing stiffness has also been reported for balls from other sports (Haake et al., 2003). This correlation is likely due the fact that energy loss is proportional to ball deformation, which is less for the stiffer balls.

Softer baseballs have been shown to reduce the risk of head injury, eye injury, and *commotio cordis* (Crisco, Hendee, & Greenwald, 1997; Heald et al., 1994; Link, Maron, Wang, et al., 2002; Vinger, Duma, & Crandall, 1999). The practice ball we tested had a compression load nearly five times less than the regular balls. Unlike baseballs, we cannot recommend the use of softer lacrosse balls (i.e., those with a lower compression load) until further research has been completed, because softer lacrosse balls could potentially pass through the facemasks and eyewear worn by players. Balls that are too soft may also pose a risk to the eye as a result of their ability to penetrate more deeply into the orbit (Vinger et al., 1999).

The rule with respect to surface texture is not clear. From our interpretation of these rules, balls of different surface textures are acceptable in NCAA/NFHS regulation play. Model B, with a triangular dimpled surface (Figure 1), had a lower compression load and lower COR than the other ball models. These lower values might be expected since this pattern places less material on the outer surface of the ball. However, the effects of the surface pattern are not conclusive since we did not control the material composition of the ball models. It is also unknown whether surface texture on the ball would alter the interaction with the stick or playing surface and affect play during a game. Specific labeling requirements for the balls are also not clearly stated in the rules. Labels such as "Official NCAA" or "Meets NCAA Specs" both suggest compliance with the requirements of the NCAA.

In summary, we found that none of the ball models we tested met all the specifications of the NCAA/NFHS. We found that the current rebound method used for quantifying ball liveliness correlated weakly with the COR measured at 27 m/s. We suggest that governing bodies consider updating their specifications to adopt more game-relevant performance tests. Specifications for the compression loads of competitive-play lacrosse balls should also be considered, but further studies are needed in order to determine the relationship between compression loads and the likelihood of facemask and goggle penetration.

Acknowledgments

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2011 CSMAS Meeting

- **Field Hockey.** Based on feedback and questions about the June 2011 CSMAS recommendation to require field hockey field player to wear American Society for Testing and Materials (ASTM) certified eye protection, further investigation was conducted through independent testing of current ASTM approved eye protection for field hockey on the market. Upon committee review and further discussion, the committee recommends placing a hold on the implementation of the recommendation for field hockey eye protector until the equipment on the market can be certified to meet the demands for the collegiate sport. The next steps identified by the committee included: (a) the need to confirm a standard ball velocity for the collegiate game; (b) the need to confirm a field hockey ball standard that describes the mechanical properties and performance criteria of the ball so that it is consistent for both testing and playing purposes; (c) the need to consider a refinement in the ball velocity requirements in the ASTM standard; and (d) confirmation that the products on the market meet the demands for the collegiate sport. The committee reaffirmed its goal to minimize the risk of eye injury. It was noted that the National Federation of State High School Associations (NFHS) required field hockey players to wear ASTM -approved protective eyewear for the high school game, effective with the 2011-12 season.

Excerpts from meetings of the
NCAA Committee on Competitive Safeguards and Medical Aspects of Sports

2011

Field hockey (June). The committee recommended NCAA field hockey players be required to wear protective ASTM-approved protective eyewear during play. The committee reviewed injury data for the sport of field hockey, the historical positions of the committee encouraging the use of protective eyewear, and the recent action by the NFHS to mandate protective eyewear. The committee believes that requiring protective eyewear during play will minimize the risk of catastrophic eye injuries. NCAA injury data noted that 25 percent of all injuries occur to the head and face with 28 percent of those head and face injuries occurring from contact with the ball and another 16 percent due to the stick. NFHS required field hockey players to wear American Society for Testing and Materials (ASTM)-approved protective eyewear, effective with the 2011-12 season.

Field Hockey (December). Based on feedback and questions about the June 2011 CSMAS recommendation to require field hockey field player to wear American Society for Testing and Materials (ASTM) certified eye protection, further investigation was conducted through independent testing of current ASTM approved eye protection for field hockey on the market. Upon committee review and further discussion, the committee recommends placing a hold on the implementation of the recommendation for field hockey eye protector until the equipment on the market can be certified to meet the demands for the collegiate sport. The next steps identified by the committee included: (a) the need to confirm a standard ball velocity for the collegiate game; (b) the need to confirm a field hockey ball standard that describes the mechanical properties and performance criteria of the ball so that it is consistent for both testing and playing purposes; (c) the need to consider a refinement in the ball velocity requirements in the ASTM standard; and (d) confirmation that the products on the market meet the demands for the collegiate sport. The committee reaffirmed its goal to minimize the risk of eye injury. It was noted that the National Federation of State High School Associations (NFHS) required field hockey players to wear ASTM -approved protective eyewear for the high school game, effective with the 2011-12 season.

2009

Field Hockey. NCAA staff and Eric Hall will work with the Datalys Center to evaluate specific injury data questions pertaining the sport of field hockey, head and eye injuries and mechanisms. These data will then be compiled in a report with the 2009 revised ASTM equipment standards that outline a new testing method specific for field hockey. The report will go the NCAA sport committee for field hockey as the NCAA does write the rules for the sport.

2007

Field Hockey. The committee continues its support for allowing protective eye wear during play, both for reasons of injury prevention and participation of student-athletes that have already incurred a significant eye injury.

2007

Field Hockey. The NCAA Field Hockey committee recommended permitting field players to use protective eyewear or head covering throughout a match at any time. The CSMAS committee

recommended conducting a survey of teams at the conclusion of the season to examine the extent to which student-athletes wore protective eyewear.

2006

Field Hockey. The committee continues its support for allowing protective eye wear during play, both for reasons of injury prevention and participation of student-athletes that have already incurred a significant eye injury. The committee noted that the addition of eye wear to women's lacrosse has eliminated eye injuries in the sport and even reduced the overall head and face injury rates. In addition, the sports of basketball and football have established permissive language that allows for student-athletes to wear polycarbonate protective eye wear during play to prevent eye injuries and have not needed to mandate their use. The committee will consider steps to allowing protective eye wear to be permissible in NCAA play.

Field Hockey. The committee reviewed a case study of severe eye injury in the sport of Field Hockey and NCAA ISS data the noted 19% of all injuries in FH occur to the head with 65% of those injuries occurring from elevated balls. In an effort to ensure student-athletes seeking to wear protective eye wear in competition are not denied the use of protective eye at the site of matches, the committee will work with the field hockey community to better communicate the position of the NCAA that protective eye wear meeting the ASTM-F803 standard for field hockey is permissible to wear during competition.

Field Hockey. The committee reviewed the process for establishing eye protection in the sport of field hockey. The committee will continue to review data from the ISS and catastrophic insurance claims and ask the medical community for specific cases of severe facial injuries in the sport to help determine incidence. It was noted that manufactures have developed field hockey specific goggles that meet the ASTM F803 safety standard established for the sport.

2005

Field hockey. The committee supported collaborative efforts with equipment standards groups such as the American Standards for Testing Materials (ASTM) and the National Operating Committee on Standards for Athletic Equipment (NOCSAE) to develop field hockey specific testing guidelines for the protective eyewear currently on the market.

2004

Field Hockey. Addressed current injury trends and suggested presenting pertinent information to the field hockey committee to encourage the use of appropriate protective eyewear during activity.

Field Hockey. The subcommittee suggested sending a letter from the committee to USA Field Hockey noting injury rates to the head. The committee suggested that the NCAA Field Hockey Committee collaborate with equipment manufacturers to develop eyewear to promote extended view to benefit student-athletes in field hockey but provide optimal protection, and to list eyewear and gloves as permissive equipment in the sport rules book.

2003

Field Hockey. The subcommittee voted to recommend the rules of field hockey be amended to include language that permits protective eyewear as permissible special protective equipment. The subcommittee requested a continued analysis of ISS data to determine the seriousness of potential head injuries in addition to the catastrophic eye injury risk in the sport of field hockey. The subcommittee has begun to explore the mandate of eyewear in the sport of field hockey based on ISS data, SMH Guideline 4b and the recommendations of other professional organizations.

2002

Field hockey. The committee recommended asking the NCAA Field Hockey Committee to clarify its stance on the wearing of protective eyewear and to add language noting the permissibility of protective eyewear.

2001

Field Hockey. The subcommittee was updated on the NFHS actions in response to a blinded eye in high school field hockey.

2000

Field hockey. The subcommittee reviewed National High School Federation actions in response to a blinded eye in high school field hockey. Concerns were expressed regarding this issue in the collegiate game although injury risk appeared very low this past year based on ISS data. The subcommittee agreed to continue to monitor this issue.

1998

Field Hockey. The subcommittee reviewed rules change in field hockey that required mouthguards for all players and required head protection for a kicking back. The subcomittee endorsed these changes but still was concerned that some of its issues raised following its January 1998 meeting had not been addressed. The subcommittee recommended revisiting this issue following a review of the fall 1998 field hockey data.

Field Hockey Issues. The subcommittee reviewed correspondence from concerned parents about the lack of head protection in field hockey. Ms. Thomas also discussed concerns raised by representatives at her institution. The subcommittee reviewed past discussions, noting the game-injury rate in the sport was relatively low, but when injuries did occur, about 15 percent were to the head or face. Primary concern was voiced about the potential injury to the eyes, nose and mouth from high sticks, harder shots and elevated balls that appear to be more prevalent in today's game. Other concerns involved a lack of developed eye protection from manufacturers and inconsistent officiating, which had been suggested by the field hockey committee as the appropriate way to control these types of injuries. Staff was directed to forward these concerns to the field hockey committee for a response. The subcommittee also

recommended developing a head injury questionnaire that could be applied to all schools sponsoring field hockey (and women's lacrosse) in an attempt to monitor all facial injuries in these activities.

1996

Field Hockey and Women's Lacrosse. The subcommittee reviewed updated ISS information regarding head and neck injuries in field hockey and women's lacrosse. It was noted that the game-head and neck-injury rates rose in the past season in field hockey and dropped in women's lacrosse. The subcommittee encouraged both sports to familiarize participants with the optional protective equipment available for use. It also encouraged the field-hockey community to work with standards agencies to develop a standard for protective eyewear in the sport.

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National Collegiate Athletic Association

Latest News

Aye for an eye?

College field hockey stares down a protective-equipment issue

By Gary Brown
NCAA.org

The collegiate field hockey community is weighing whether a recommendation to require protective eyewear is good for the game.

Citing increased rates of injuries to the face and head, the NCAA Committee on Competitive Safeguards and Medical Aspects of Sports is recommending that NCAA field hockey work toward requiring players to wear protective eyewear in the near future.

The eyewear requirement was implemented at the high school level last year. That action, combined now with the suggestion from the NCAA's primary health and safety board, is prompting a broader discussion at the collegiate, national and international levels.

The competitive-safeguards committee based its recommendation on NCAA data showing that 25 percent of all injuries in field hockey competitions occur to the head and face, with 28 percent of those being from contact with the ball and another 16 percent because of contact with the stick. Although rare, 8 percent of head injuries involve the eye (eye injuries include eyelid lacerations, corneal abrasions, orbital fractures, soft tissue contusion and others).

"We're trying to mitigate data we've seen from our Injury-Surveillance System indicating that eye injuries are a potential risk in field hockey," said Jeff Anderson, chair of the committee and director of sports medicine at the University of Connecticut.

Increased rates of injuries to the head and face has led the NCAA Committee on Competitive Safeguards and Medical Aspects of Sports to recommend required protective eyewear for field hockey players.

"Those data, along with the action the high schools took, prompted us to suggest that eyewear eventually be mandatory."

The original request from Anderson's committee last summer was for the requirement to be in place by next season, but feedback and presentations from researchers and equipment experts at the committee's most recent meeting in December prompted the group to delay the effective date of the proposal until additional testing can be done on the eyewear currently available to ensure it is appropriate for the collegiate game.

All three of the NCAA's divisional championships committees are aware of the recommendation and have referred the matter to the respective field hockey committees, which in turn are soliciting feedback from coaches and other constituents.

The early returns indicate support for enhancing player safety where appropriate, but many coaches worry that the eyewear designed to protect their players actually puts them more at risk.

The matter is complicated at the NCAA level because there's no standing playing rules committee for the sport. However, the field hockey committees in all three divisions routinely review the playing rules that are developed by the international committee and modify them for use in the college game – a process similar to other NCAA sports without a standing rules committee.

The eyewear issue at the college level may end up being decided by the NCAA's Playing Rules Oversight Panel, which has the responsibility to review and decide on rules modifications for all NCAA sports, including those without stand-alone NCAA rules committees. While the competitive-safeguards committee doesn't report to the playing rules panel, the panel may be the most logical destination within the NCAA governance structure for the matter to be resolved.

High school restrictions

The rules committee at the high school level didn't recommend eyewear, either, but the National Federation of State High School Associations nonetheless adopted the requirement last April.

The Federation's sports medicine advisory committee had been suggesting action for several years, but the rules committee routinely turned it down, based in part on not believing that data required action and that the testing to date hadn't been adequate. Last year, though, the Federation's board of directors overturned the rules committee decision, mandating that all 51 state associations require the more than 64,000 student-athletes in the sport to don eyewear that meets standards set by the American Society for Testing and Materials, the de facto regulatory body for protective equipment in the sport.

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Elliot Hopkins, the field hockey liaison at the Federation, pointed out that some state associations already had mandated eyewear (about 40 percent of players wore it already), so the board's decision wasn't that jarring to the high school community. Since then, though, Hopkins said he's received some pushback from coaches and parents.

The risk of head and eye injuries in field hockey led the National Federation of State High School Associations to adopt a requirement for protective eyewear last April.

Hopkins said he's heard some complaints about injuries that are perceived to be *caused* by the eyewear (such as a girl needing stitches after a ball forced the eyewear to gash her brow), but in defense of the Federation's action, Hopkins said, "Stitches are a lot cheaper than a prosthetic eye."

There also are complaints about how eyewear – usually some form of goggle – restricts a player's peripheral vision. Hopkins says he's aware that many college coaches don't like it, and that even USA Field Hockey seems to disagree with the mandate, but Hopkins maintains that the Federation was acting on behalf of its constituents and not concerned about what other governing bodies felt.

No momentum so far

The national and international communities have shied away from the eyewear issue in part because their rules have to be acceptable for a range of age and skill levels, including kids just starting out. So while those governing bodies might not be opposed to eyewear, it could be impractical for them to require it for their constituents.

Steve Horgan, the National Umpire and Technical Manager for USA Field Hockey, who also serves as the NCAA secretary-rules editor, said he doesn't sense any momentum from college coaches or national and international constituents to follow the high school lead. He said the protection gives players a "false sense of security" that leads to more aggressive play, and the resulting lack of peripheral vision actually puts players more at risk of head injuries in general.

"With the eyewear, we're seeing kids playing more upright rather than lower to the ground the way the game is supposed to be played," Horgan said.

Yale head coach Pam Stuper said coaches' concerns range from a negative impact on player safety to an effect on skill level. She said the false sense of security to which Horgan eluded likely will lead to an increase in concussions, as might the effect of eyewear on a player's peripheral vision.

"Try this yourself," said Stuper, who's also a member of the Division I Field Hockey Committee. "Put your pointer finger horizontally right below your eyes, as the lower bar of a

Hesitancies surrounding eyewear usage focus on a lack of peripheral vision and the reaction to play more upright rather than lower to the ground.

goggle would be, and then try to look down. If you're trying to look at a ball that's just in front of your feet, you can't see it. So what do you do? You have to tilt your head farther down, which pulls your vision more narrowly down to the ground."

The infringement on peripheral vision impacts a player's ability to make good decisions as a ball carrier, Stuper said, which in turn will affect skill level, since kids are taught to keep their head up when they carry the ball.

"National team players are talented enough so that they don't have to focus on the ball when they're carrying it, but those in high school and college need to pay attention to the ball, which means they need to have split vision," said Stuper. "And they can't if their head is down on the ball."

Division III Field Hockey Committee member Brenda Meese has similar concerns.

"It's more about the integrity of the game and the safety of the players," said the head coach and assistant AD at Wooster. "Part of that is being able to see – you need not only your peripheral vision to the sides but also up and down. Most of the goggles made don't seem to accommodate that."

Similarly, Division II committee chair Stacey Gaudette cited safety as a primary concern since the loss of peripheral vision would necessitate a change in skill. "The loss of vertical peripheral vision and the speed at which the game is played is a combination that coaches are concerned will lead to more injuries," said the head coach at Mercyhurst. "As a part of the international community, we want our skills to continually improve, but adding a barrier to the sight of the ball will hinder that skill advancement."

Many opponents of the recommendation believe more injuries could occur with the eyewear due to impaired vision.

Rules already allow players in NCAA competition to wear protective eyewear if they choose, either to address a medical condition or because they just want to. Few do so, however. Stuper and Meese both pointed out that at the recent USA Field Hockey festival in November, only a few of the hundreds of high school players there donned the eyewear, even after having played with them all season.

"Nobody wants them," Stuper said. "It has nothing to do with whether they don't look 'cool,' it's that kids can't see with them on."

Bridging the constituencies

Stuper and Meese acknowledged that the pushback from coaches and others could sound like they're not interested in player safety. Quite the contrary, they say. They're worried more

about the damage that goggles can do to the player wearing them – and to other players, too. The caged goggles in fact are specifically not allowed in NCAA or international play, even for players who need to protect a medical condition. Meese said she wouldn't be so opposed to mandatory eyewear if there was a product on the market that could be protective without impairing vision.

That's partly why the competitive-safeguards committee adjusted its recommendation from next season to "sometime in the near future." The change came after members saw preliminary testing results showing some of the wire-caged and polycarbonate models weren't always effective in protection against the velocity of the ball at the highest end of the ASTM standard, which might be more realistic to the college level of play.

"There are a lot of products on the market that meet ASTM standards," Anderson said. "We've asked for more testing on those products to see if they would be adequate for NCAA play."

Anderson also said the committee heard presentations indicating a "wide variety of mechanical characteristics" of field hockey balls, especially at different velocities, which he said dramatically affects testing. Anderson said it's tough to set a standard for eyewear when there's no standard for the ball.

To gain more clarity on the matter, Anderson said the competitive-safeguards committee will work to:

- Confirm a standard ball velocity for the collegiate game;
- Settle on a field hockey ball standard that describes the mechanical properties and performance criteria of the ball so that it is consistent for testing and playing purposes;
- Refine the ball velocity requirements in the ASTM standard; and
- Confirm that the products on the market meet these new demands for the sport at the collegiate level.

"We need to make sure we have enough information on which we can recommend changes that make a positive difference for student-athletes who play the game," Anderson said. "We're aware that many field hockey constituents are not interested in requiring eyewear, so we decided that based on the data and feedback we currently have, the best approach was to hold on our recommendation to require eyewear until we have more testing results."

The competitive-safeguards committee will continue to delve into the issue by examining characteristics of the ball.

But the delay doesn't mean that the competitive-safeguards committee might be inclined to back away from its recommendation to require eyewear.

The coaches will get a chance to talk more about the issue soon. The annual convention of the National Field Hockey Coaches Association is Jan. 12-15 in Philadelphia, and NCAA officials will be there to gather feedback. Also, all three NCAA field hockey committees have their annual meetings this month.

"People are quite opposed right now," said Stuper. "It's causing an uproar, not just because of the

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eyewear itself but also because there is a general feeling that the people making these recommendations do not understand our sport."

Anderson acknowledged at least the perceived gap between constituents who believe they understand the way the game should be played and a group that has expertise in keeping those players safe. He said that's partly why the group is seeking to understand the issue more clearly.

He said since the NCAA doesn't create standards for protective equipment, sports have to rely on external bodies like the American Society for Testing and Materials for guidance. But whether those standards are appropriate for collegiate play is worth exploring, Anderson said.

"For us, asking those questions is the appropriate next step," he said. "That's really the 'middle ground' between the field hockey people and the health and safety people, because right now, that ASTM standard is what the field hockey community and the scientific community have to work with – that's the merge right now with the two groups."

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on
Wed Jan 25 09:00:07 EST 2012

ASTM Standards for Field Hockey		
	New	Old*
ASTM designation	F-2713-09	F-803-03
Year Approved	2009	2003
Sports included in standard	Field Hockey	Field Hockey, Lacrosse, Baseball and Racquet sports
Projectile	Field Hockey ball	Lacrosse ball used for field hockey test
Projectile speed	55 MPH	60 MPH
Penetrator	Field Hockey stick toe (dimensions based on the rules of the game; they are identical to the old ASTM standard)	Field Hockey stick toe
Chairman of Subcommittee	Dr. Liz Ryan	Dr. Paul Vinger
Members of ASTM Subcommittee	Brine, Cascade, Halo,K2 (deBeer's parent company), Oakley,Reebok, STX, Warrior, consumer advocates, medical professionals and academics.	Cascade, consumer advocates, US Lacrosse, medical professionals and academics.
Approved by	Consensus vote**. Every negative vote is debated by the subcommittee, if a negative vote is found non persuasive by the subcommittee those arguments are sent to an independent nine member panel that must agree the negative vote is non persuasive. (unchanged from 2003)	Consensus vote**. Every negative vote is debated by the subcommittee, if a negative vote is found non persuasive by the subcommittee those arguments are sent to an independent nine member panel that must agree the negative vote is non persuasive.
	**Voting is by mail or online, members need not attend meetings in order to vote or participate in process. Membership in ASTM cost $75 per year (students are free)	* Field Hockey will be removed from this ASTM standard in 2009 or 2010

Deliverable 2011-06
Injuries in Women's Field Hockey attributed to contact with the ball or stick

Injury inclusion criteria:

- All injuries are for 1 or more days' time loss (released data)
- Injuries incurred during preseason, regular or postseason event types of competition or practice
- The Specific Injury Mechanisms were condensed as the question asked was to see what injuries were incurred as a result of contact with the stick or ball.

Injuries during Competition and Practice

Women's Field Hockey			
	All Injuries	Head Face Injuries	% of Head Face Injuries by Event Type
Competition	4227	1078	25.5
Practice	6561	553	8.4
*Total**	*10788*	*1631*	*15.1*

* errors in totals due to rounding

Head and Face Injuries by Basic and Specific Injury Mechanisms

Women's Field Hockey				
Event Type	Basic Injury Mechanism	Specific Injury Mechanism	Frequency	Percent
Competition	Contact with player	All Specific Mechanisms	255.0	15.6
Competition	Contact with playing surface	Not Applicable	40.2	2.5
Competition	Contact with apparatus	***WFH - Contact with stick***	***265.7***	***16.3***
Competition	Contact with apparatus	WFH - Contact with goal	21.8	1.3
Competition	Contact with apparatus	***WFH - Contact with ball (Ground = 7, Elevated = 463)***	***470.0***	***28.9***
Competition	Acute non-contact	Not Applicable	14.6	0.9
Competition	Infection	Not Applicable	10.9	0.7
Practice	Missing	Not Applicable	5.7	0.4
Practice	Contact with player	All Specific Mechanisms	81.0	5.0
Practice	Contact with apparatus	***WFH - Contact with stick***	***79.0***	***4.8***
Practice	Contact with apparatus	WFH - Contact with goal	19.8	1.2
Practice	Contact with apparatus	***WFH - Contact with ball (Ground = 29, Elevated = 296)***	***325.0***	***19.9***
Practice	Infection	Not Applicable	34.0	2.1
Practice	Other	Not Applicable	8.8	0.5
*Total**			*1632.0*	*100.0*

*errors in totals due to rounding

EXCERPTS FOR NCAA FIELD HOCKEY

FROM

NCAA INJURY SURVEILLANCE PROGRAM

COMPENDIUM REPORT

25 SPORTS

2004/05 - 2008/09 ACADEMIC YEARS

JILL CORLETTE, MS ATC

STEVE MARSHALL, PHD

DATALYSCENTER℠
NCAA® Injury Surveillance Program

1. Introduction

This reports covers injury data for twenty-five collegiate sports collected during the 5-year period when the NCAA directly operated a web-based Injury Surveillance System (the 2004/05 to 2008/09 academic years). The sports and available years of data included in this report are:

Fall Sports	**Years Available**
Men's Football	2004/05 – 2008/09
Women's Field Hockey	2004/05 – 2008/09
Men's Soccer	2004/05 – 2008/09
Women's Soccer	2004/05 – 2008/09
Women's Volleyball	2004/05 – 2008/09
Men's Cross Country	2005/06 – 2008/09
Women's Cross Country	2005/06 – 2008/09
Winter Sports	
Men's Basketball	2004/05 – 2008/09
Women's Basketball	2004/05 – 2008/09
Men's Wrestling	2004/05 – 2008/09
Women's Gymnastics	2004/05 – 2008/09
Men's Ice Hockey	2004/05 – 2008/09
Women's Ice Hockey	2004/05 – 2008/09
Men's Indoor Track	2005/06 – 2008/09
Women's Indoor Track	2005/06 – 2008/09
Men's Swimming and Diving	2006/07 – 2008/09
Women's Swimming and Diving	2006/07 – 2008/09
Spring Sports	
Men's Lacrosse	2004/05 – 2008/09
Women's Lacrosse	2004/05 – 2008/09
Men's Baseball	2004/05 – 2008/09
Women's Softball	2004/05 – 2008/09
Men's Outdoor Track	2005/06 – 2008/09
Women's Outdoor Track	2005/06 – 2008/09
Men's Tennis	2005/06 – 2008/09
Women's Tennis	2005/06 – 2008/09

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The years of available data range from a three-year period (the 2006/07 to 2008/09 academic years) to the full five-year period (the 2004/05 to 2008/09 academic years). This was because of the expansion of the sports covered by the system starting with the 2005/06 academic year. Only injuries resulting in restriction of the student-athlete's participation for one or more days beyond the day of injury are included in this report.

The statistical methodology used to produce this report included post-stratification sample weights (to adjust for year-to-year variation in the composition of the reporting sample) and adjustment for under-reporting (based on a validation study performed by Duke University).

Only injuries resulting in restriction of the student-athlete's participation for one or more days beyond the day of injury are included in this report. All injuries were limited to only athletic related injuries that occurred during practice or competition activities.

Only practice and competition events were included in the data set for each school. No weight lifting or conditioning sessions were included in the data set. Additionally, all junior varsity competitions were also eliminated as the ISS did not always allow the choosing this type of activity and not all athletic trainers entered this information.

The addition of ability to include zero-timeloss injuries was accompanied by training and education on the definition of a reportable injury, particularly during the summer of 2007. The decrease in reported injuries observed in some sports and divisions in the 2007/08 and 2008/09 academic years may be related to this training.

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2. *Overall Injury Incidence*

The competition and practice injury rates and absolute numbers of injuries (average annual national estimates) for all the sports covered by this report appear in this chapter. All injury and exposure counts in this chapter represent Average Annual National Estimates during the period covered by the individual sport report. The Average Annual National Estimates are the estimated average number of injuries per year in the NCAA in each sport. They are extrapolated from the schools that reported injury data to the ISS.

A graphical overview of the sport-specific injury rates appear in Figures 2.1 (Fall sports), 2.2 (Winter sports) and 2.3 (Spring sports). ***Injury rates*** are important, but they do not indicate the complete impact of injuries in a population of athletes. For example, a sport with a low injury rate may still generate a high number of injuries if it has a large number of sponsoring schools and/or large number of participating athletes. The ***absolute number of injuries***, as measured by ***annual average number of injuries*** is another measure of the impact of injury. The measure complements injury rate. The ***absolute number of injuries*** by sports is shown graphically in Figures 2.4 (Fall sports), 2.5 (Winter sports), and 2.6 (Spring sports).

Tables 2.1 to 2.3 present injury rates for Fall, Winter, and Spring (similar to Figures 2.1-2.4) but also include the competition-to-practice rate ratio. This rate ratio indicates the increase in the injury rate due to participation in a competition, rather than a practice. Only injuries resulting in restriction of the student-athlete's participation for one or more days beyond the day of injury are included in this report.

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Figure 2.1
Injury Rates by Sport (error bar indicates upper 95% confidence interval) – Fall Sports

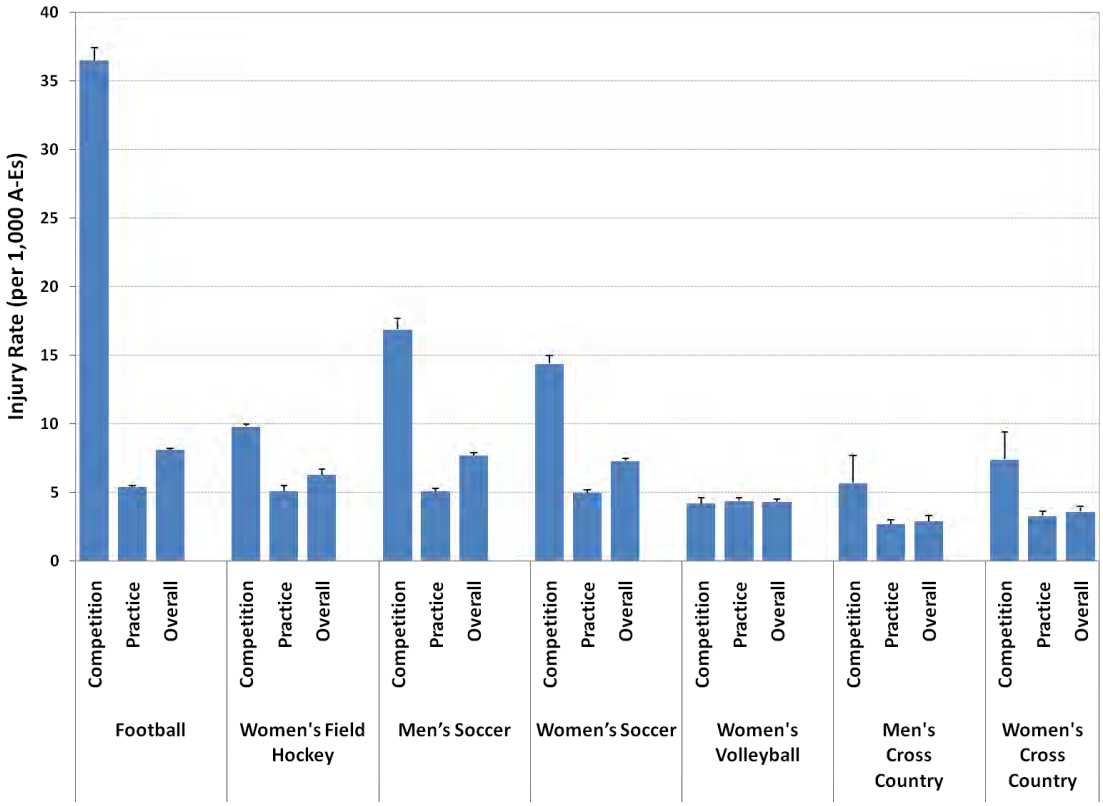

Figure 2.2
Injury Rates by Sport (error bar indicates upper 95% confidence interval) – Winter Sports

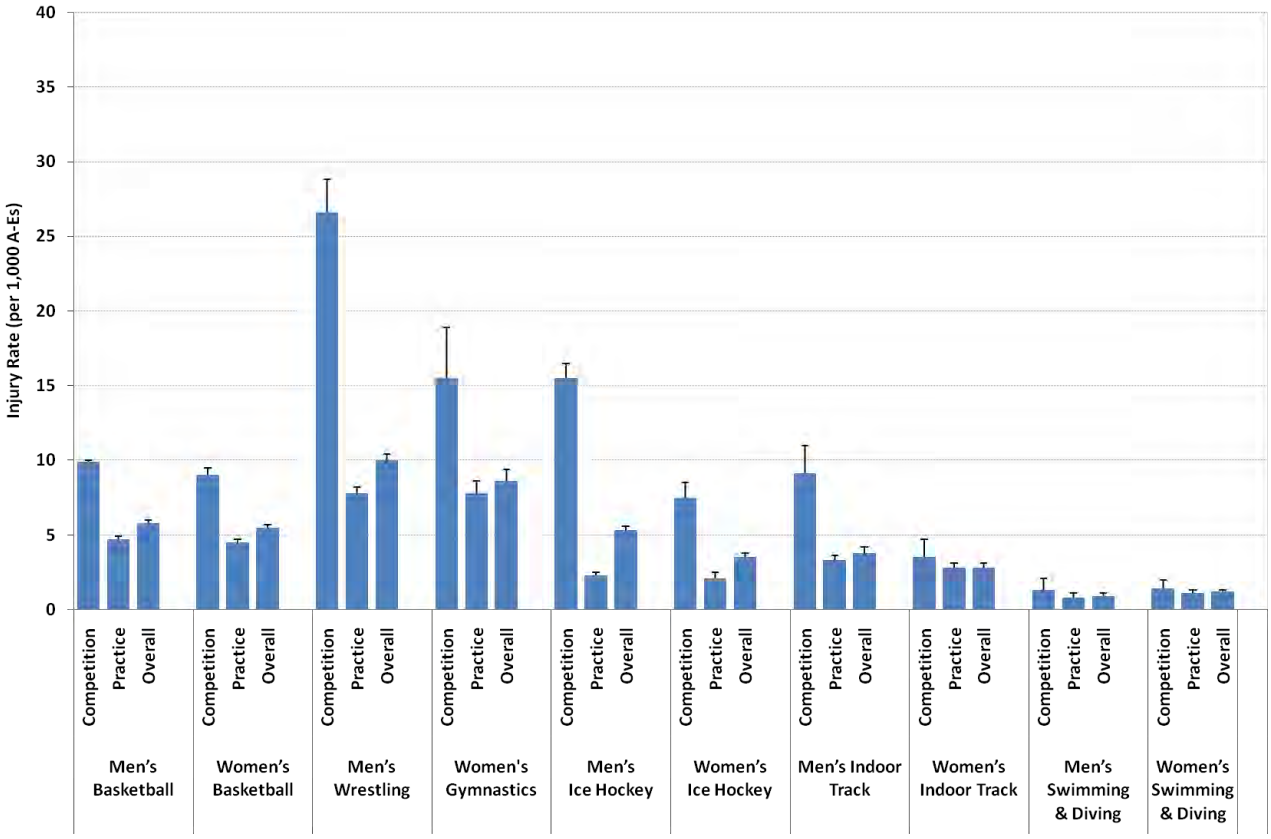

Figure 2.3

Injury Rates by Sport (error bar indicates upper 95% confidence interval) – Spring Sports

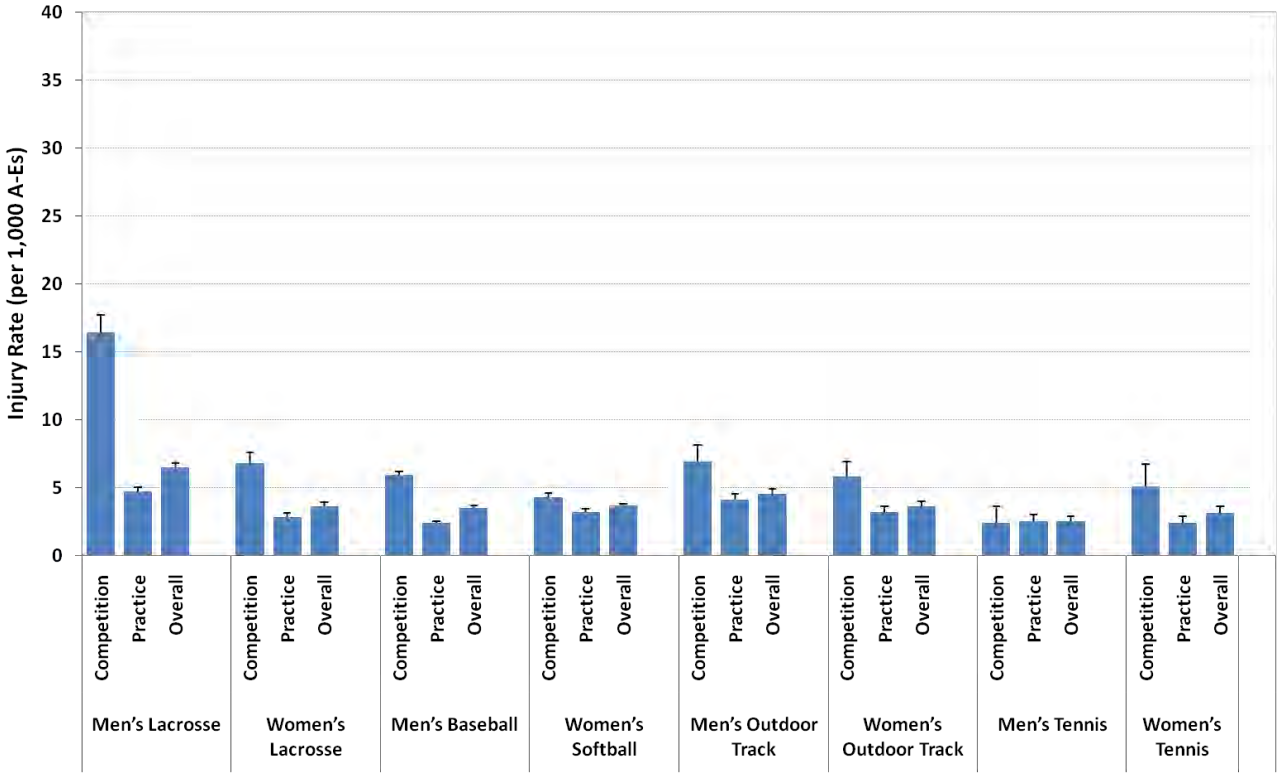

Figure 2.4 Absolute Numbers of Injuries for Competition and Practice – Fall Sports

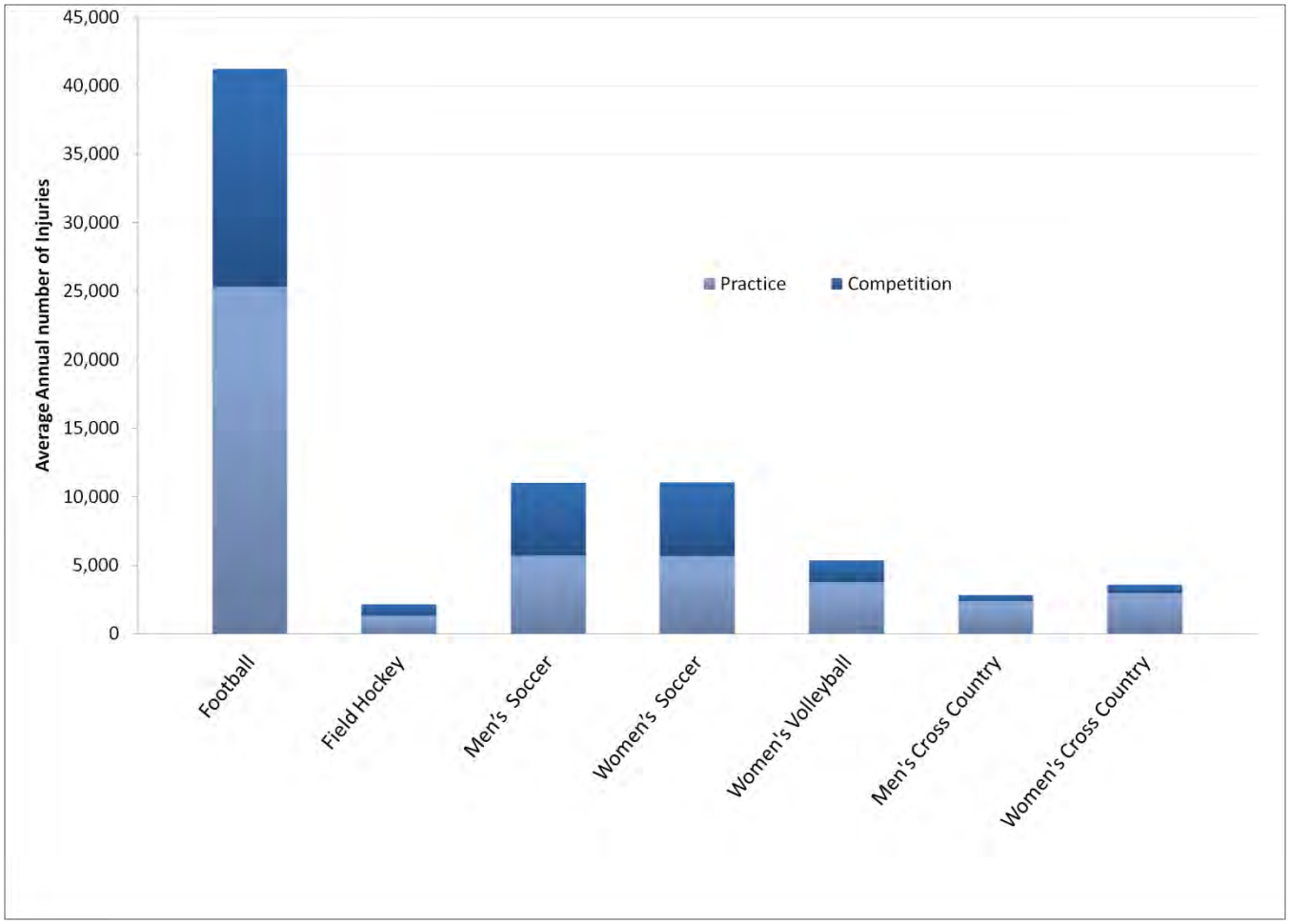

Figure 2.5 Absolute Numbers of Injuries for Competition and Practice – Winter Sports

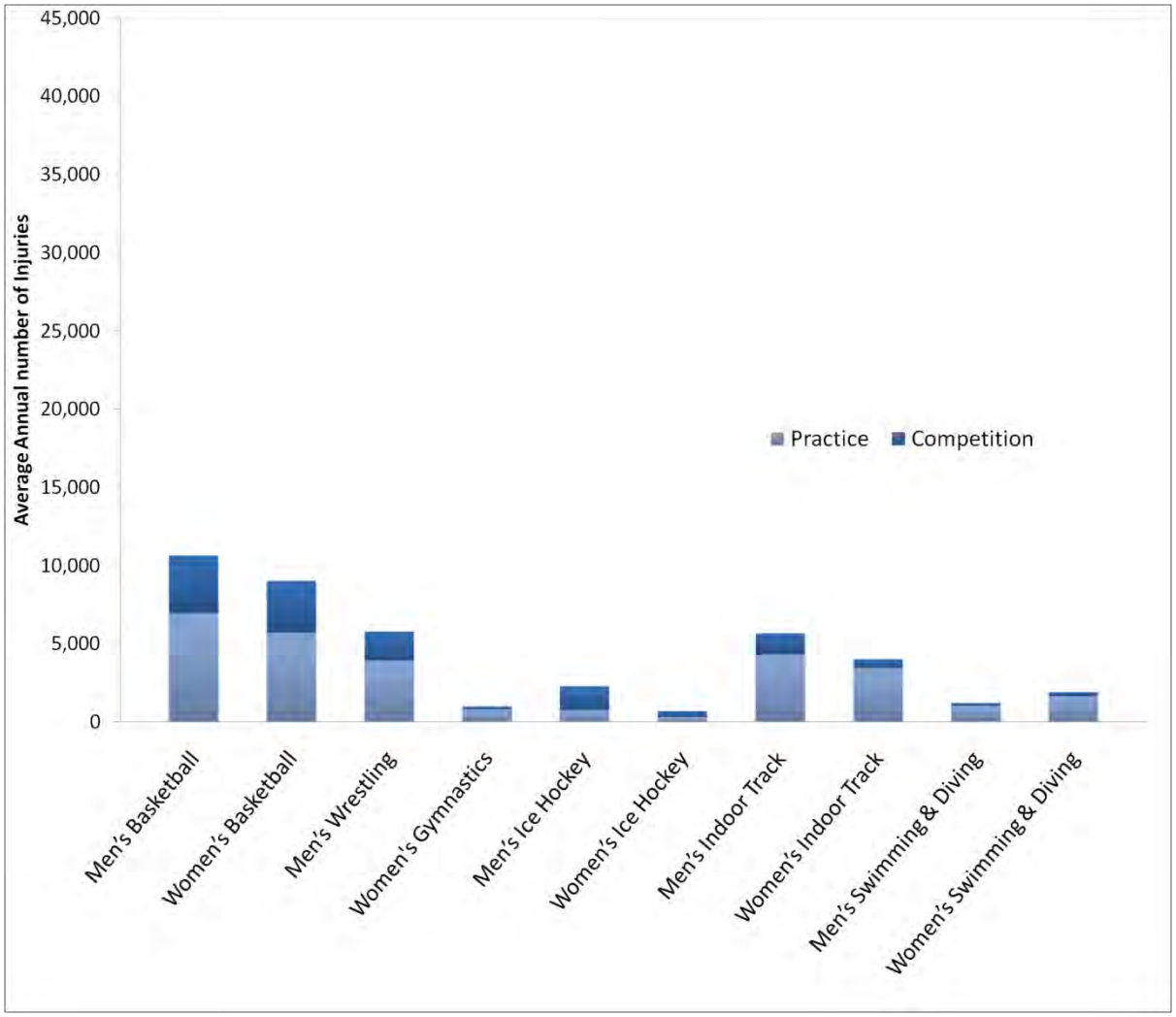

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Figure 2.6 Absolute Numbers of Injuries for Competition and Practice – Spring Sports

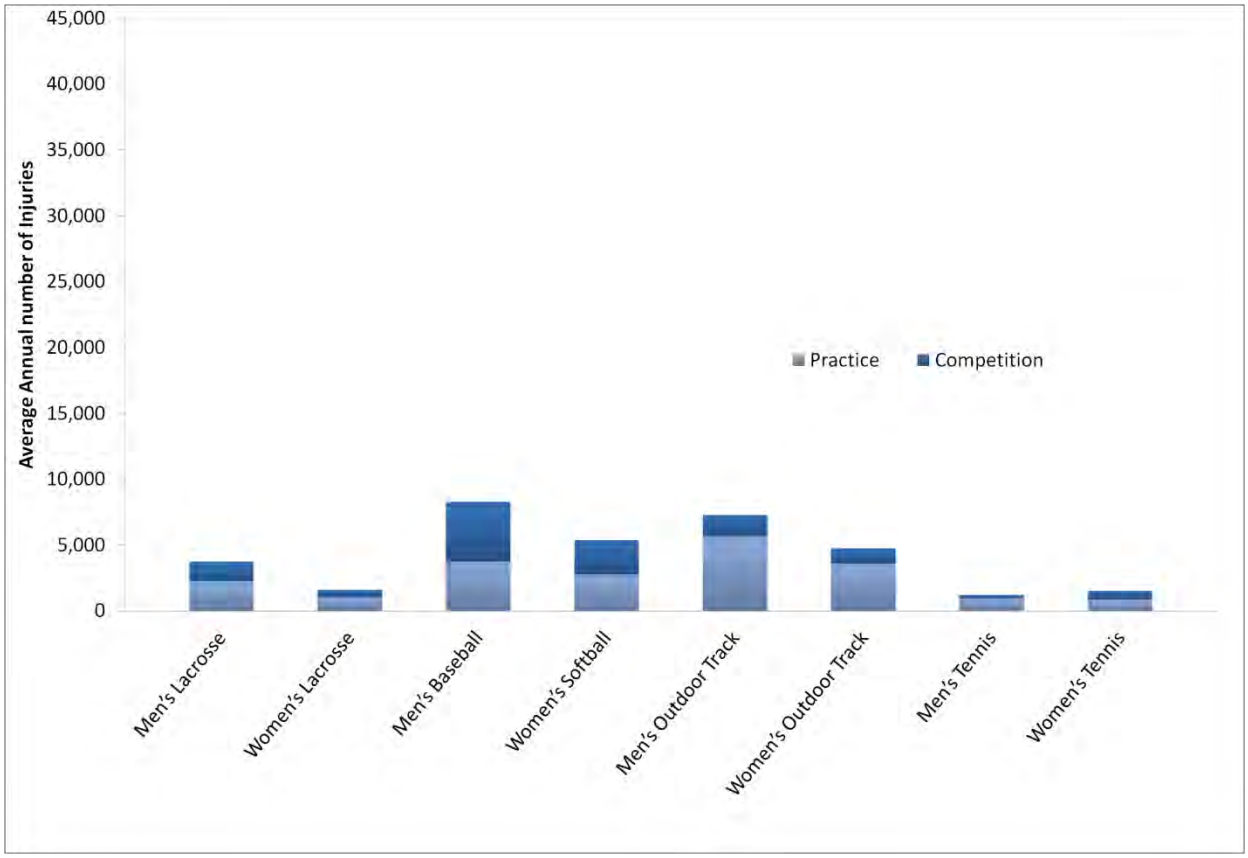

Table 2.1 Injury Rates for Fall Sports, by Sport, including Competition-to-Practice Rate Ratio

Sport	Event	Reported Injuries (all Years)	Injury Count (Average Annual National Estimate)	Athlete Exposures (Average Annual National Estimate)	Injury Rate (per 1000 Athlete Exposures)	Competition-to-Practice Rate Ratio
Football 2004/05 – 2008/09	Competition	7,020	15,907	435,262	36.5 [35.7,37.4]	6.7
	Practice	11,055	25,325	4,653,297	5.4 [5.3,5.5]	
	Overall	18,075	41,232	5,088,560	8.1 [8.0,8.2]	
Women's Field Hockey 2004/05 – 2008/09	Competition	362	845	86,135	9.8 [8.8,10.8]	1.9
	Practice	556	1,312	255,912	5.1 [4.7,5.5]	
	Overall	918	2,158	342,047	6.3 [5.9,6.7]	
Men's Soccer 2004/05 – 2008/09	Competition	1,959	5,326	314,933	16.9 [16.2,17.7]	3.3
	Practice	2,091	5,699	1,118,939	5.1 [4.9,5.3]	
	Overall	4,050	11,025	1,433,872	7.7 [7.5,7.9]	
Women's Soccer 2004/05 – 2008/09	Competition	1,965	5,361	373,118	14.4 [13.7,15.0]	2.9
	Practice	2,101	5,682	1,144,681	5.0 [4.7,5.2]	
	Overall	4,066	11,043	1,517,799	7.3 [7.1,7.5]	
Women's Volleyball 2004/05 – 2008/09	Competition	492	1,556	367,526	4.2 [3.9,4.6]	1.0
	Practice	1,222	3,789	868,920	4.4 [4.1,4.6]	
	Overall	1,714	5,345	1,236,445	4.3 [4.1,4.5]	
Men's Cross Country 2005/06 – 2008/09	Competition	39	418	72,744	5.7 [3.8,7.7]	2.1
	Practice	223	2,389	892,628	2.7 [2.3,3.0]	
	Overall	262	2,807	965,372	2.9 [2.6,3.3]	
Women's Cross Country 2005/06 – 2008/09	Competition	57	599	81,374	7.4 [5.3,9.4]	2.3
	Practice	270	2,990	916,162	3.3 [2.9,3.6]	
	Overall	327	3,589	997,537	3.6 [3.2,4.0]	

Numbers in square brackets [] indicate 95% confidence intervals

Game to practice rate ratios use practice as the reference

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Table 2.2 Injury Rates for Winter Sports, by Sport, including Competition-to-Practice Rate Ratio

Sport	Event	Reported Injuries (all Years)	Injury Count (Average Annual National Estimate)	Athlete Exposures (Average Annual National Estimate)	Injury Rate (per 1000 Athlete Exposures)	Competition-to-Practice Rate Ratio
Men's Basketball 2004/05 – 2008/09	Competition	1,334	3,697	374,686	9.9 [9.3,10.4]	
	Practice	2,489	6,960	1,467,497	4.7 [4.6,4.9]	2.1
	Overall	3,823	10,657	1,842,183	5.8 [5.6,6.0]	
Women's Basketball 2004/05 – 2008/09	Competition	1,192	3,287	366,777	9.0 [8.4,9.5]	
	Practice	2,060	5,724	1,280,500	4.5 [4.3,4.7]	2.0
	Overall	3,252	9,011	1,647,277	5.5 [5.3,5.7]	
Men's Wrestling 2004/05 – 2008/09	Competition	541	1,823	68,641	26.6 [24.3,28.8]	
	Practice	1,218	3,953	509,881	7.8 [7.3,8.2]	3.4
	Overall	1,759	5,776	578,522	10.0 [9.5,10.4]	
Women's Gymnastics 2004/05 – 2008/09	Competition	101	195	12,566	15.5 [12.2,18.9]	
	Practice	398	808	103,577	7.8 [7.0,8.6]	2.0
	Overall	499	1,002	116,142	8.6 [7.9,9.4]	
Men's Ice Hockey 2004/05 – 2008/09	Competition	943	1,524	98,295	15.5 [14.5,16.5]	
	Practice	451	756	332,240	2.3 [2.1,2.5]	6.8
	Overall	1,394	2,280	430,535	5.3 [5.0,5.6]	
Women's Ice Hockey 2004/05 – 2008/09	Competition	220	375	49,855	7.5 [6.5,8.5]	
	Practice	203	330	153,923	2.1 [1.8,2.5]	3.5
	Overall	423	705	203,778	3.5 [3.1,3.8]	
Men's Indoor Track 2005/06 – 2008/09	Competition	73	1,307	143,526	9.1 [7.1,11.1]	
	Practice	232	4,353	1,332,195	3.3 [2.9,3.6]	2.8
	Overall	305	5,659	1,475,722	3.8 [3.4,4.2]	
Women's Indoor Track 2005/06 – 2008/09	Competition	45	537	152,203	3.5 [2.4,4.7]	
	Practice	269	3,469	1,259,836	2.8 [2.4,3.1]	1.3
	Overall	314	4,006	1,412,039	2.8 [2.5,3.1]	
Men's Swimming & Diving 2006/07 – 2008/09	Competition	11	192	148,446	1.3 [0.5,2.1]	
	Practice	68	1,031	1,260,055	0.8 [0.6,1.1]	1.6
	Overall	79	1,223	1,408,501	0.9 [0.6,1.1]	
Women's Swimming & Diving 2006/07 – 2008/09	Competition	19	257	187,353	1.4 [0.7,2.0]	
	Practice	116	1,649	1,469,750	1.1 [0.9,1.3]	1.2
	Overall	135	1,906	1,657,102	1.2 [1.0,1.3]	

Numbers in square brackets [] indicate 95% confidence intervals

Game to practice rate ratios use practice as the reference

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Table 2.3 Injury Rates for Spring Sports, by Sport, including Competition-to-Practice Rate Ratio

Sport	Event	Reported Injuries (all Years)	Injury Count (Average Annual National Estimate)	Athlete Exposures (Average Annual National Estimate)	Injury Rate (per 1000 Athlete Exposures)	Competition-to-Practice Rate Ratio
Men's Lacrosse 2004/05 – 2008/09	Competition	544	1,497	91,265	16.4 [15.1,17.7]	
	Practice	886	2,251	483,530	4.7 [4.4,5.0]	3.5
	Overall	1,430	3,748	574,795	6.5 [6.2,6.8]	
Women's Lacrosse 2004/05 – 2008/09	Competition	224	607	89,464	6.8 [5.9,7.6]	
	Practice	379	1,007	355,222	2.8 [2.6,3.1]	2.4
	Overall	603	1,614	444,686	3.6 [3.4,3.9]	
Men's Baseball 2004/05 – 2008/09	Competition	1,233	4,563	775,817	5.9 [5.6,6.2]	
	Practice	995	3,722	1,576,100	2.4 [2.2,2.5]	2.5
	Overall	2,228	8,285	2,351,917	3.5 [3.4,3.7]	
Women's Softball 2004/05 – 2008/09	Competition	746	2,599	607,655	4.3 [4.0,4.6]	
	Practice	782	2,756	856,076	3.2 [3.0,3.4]	1.3
	Overall	1,528	5,355	1,463,732	3.7 [3.5,3.8]	
Men's Outdoor Track 2005/06 – 2008/09	Competition	85	1,588	231,205	6.9 [5.6,8.1]	
	Practice	239	5,679	1,370,994	4.1 [3.8,4.5]	1.7
	Overall	324	7,268	1,602,199	4.5 [4.2,4.9]	
Women's Outdoor Track 2005/06 – 2008/09	Competition	85	1,157	201,164	5.8 [4.6,6.9]	
	Practice	260	3,601	1,115,704	3.2 [2.9,3.6]	1.8
	Overall	345	4,758	1,316,868	3.6 [3.2,4.0]	
Men's Tennis 2005/06 – 2008/09	Competition	31	290	118,535	2.4 [1.3,3.6]	
	Practice	70	938	380,018	2.5 [1.9,3.0]	1.0
	Overall	101	1,228	498,553	2.5 [2.0,2.9]	
Women's Tennis 2005/06 – 2008/09	Competition	71	643	124,927	5.1 [3.6,6.7]	
	Practice	65	868	367,618	2.4 [1.8,2.9]	2.2
	Overall	136	1,512	492,545	3.1 [2.5,3.6]	

Numbers in square brackets [] indicate 95% confidence intervals

Game to practice rate ratios use practice as the reference

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Methodology & Technical Notes

Since its inception, the ISS has depended on a volunteer "convenience sample" of reporting schools. This report used post-stratification sample weights to adjust for year-to-year variation in the divisional composition of the reporting sample.

Post-stratification sample weights are a means of ensuring that the injury rates computed from the ISS sample represent the overall universe of all schools sponsoring a given sport (the "population" which is sampled, in statistical terms).

In the time period covered by this report, a reportable injury in the ISS was defined as one that: 1) occurred as a result of participation in an organized intercollegiate practice or contest, 2) required attention by certified athletic trainers or physicians, and 3) resulted in restriction of the student-athlete's participation for one or more days beyond the day of injury.

For preseason, formal team practices/competitions had to occur before first regular season contest. In season was defined as first regular season contest through last regular season contest, and post-season was defined as formal team activities following last regular season contest through last post-season contest. All injuries and exposures from outside the pre, in, or post season (such as out-of-season exposures) were excluded.

A complete 13-page methodological report is available upon request.

NCAA FIELD HOCKEY

4. Women's Field Hockey

Table 4.1 Injury Rates - Women's Field Hockey 2004/05 to 2008/09

		National Estimate, All Years			
Division	**Event Type**	**Reported Injuries All Years**	**Injury Count**	**Athlete Exposures**	**Annual Injury Rate (per 1000 Athlete-Exposures)**
All Divisions*	Competition	362	4,227	430,675	9.8 [8.8,10.8]
	Practice	556	6,561	1,279,561	5.1 [4.7,5.5]
	Overall	918	10,788	1,710,235	6.3 [5.9,6.7]

* No Division II schools submitted data

Table 4.2 Injury Rates by Event Type and Academic Year - Women's Field Hockey 2004/05 to 2008/09

		National Estimate, All Years				
Division	**Event Type**	**Academic Year**	**Reported Injuries**	**Injury Count**	**Athlete Exposures**	**Annual Injury Rate (per 1000 Athlete-Exposures)**
All Divisions*	Competition	2004-05	88	997	84,305	11.8 [9.4,14.3]
		2005-06	80	974	80,133	12.2 [9.6,14.7]
		2006-07	58	757	89,334	8.5 [6.3,10.6]
		2007-08	67	719	90,885	7.9 [6.0,9.8]
		2008-09	69	779	86,018	9.1 [6.9,11.2]
	Practice	2004-05	184	2,104	242,852	8.7 [7.4,9.9]
		2005-06	113	1,327	233,830	5.7 [4.6,6.7]
		2006-07	74	943	246,285	3.8 [3.0,4.7]
		2007-08	104	1,130	270,775	4.2 [3.4,5.0]
		2008-09	81	1,057	285,819	3.7 [3.0,4.4]
	Overall	2004-05	272	3,101	327,157	9.5 [8.4,10.6]
		2005-06	193	2,301	313,962	7.3 [6.3,8.3]
		2006-07	132	1,700	335,619	5.1 [4.2,5.9]
		2007-08	171	1,849	361,660	5.1 [4.4,5.9]
		2008-09	150	1,836	371,837	4.9 [4.2,5.7]

* No Division II schools submitted data

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Table 4.3 Injury Rates by Event Type and Season - Women's Field Hockey 2004/05 to 2008/09

				National Estimate, All Years		
Division	**Event Type**	**Season**	**Reported Injuries All Years**	**Injury Count**	**Athlete Exposures**	**Annual Injury Rate (per 1000 Athlete-Exposures)**
All Divisions*	Competition	Pre-Season	14	171	9,994	17.1 [9.2,25.0]
		In-Season	324	3,742	386,614	9.7 [8.6,10.7]
		Post-Season	24	313	34,067	9.2 [6.1,12.3]
	Practice	Pre-Season	324	3,930	442,005	8.9 [8.0,9.8]
		In-Season	220	2,485	748,447	3.3 [2.9,3.8]
		Post-Season	12	147	89,108	1.6 [.08,2.5]
	Overall	Pre-Season	338	4,101	451,999	9.1 [8.2,10.0]
		In-Season	544	6,227	1,135,061	5.5 [5.0,5.9]
		Post-Season	36	460	123,175	3.7 [2.7,4.8]

* No Division II schools submitted data

Table 4.4 Type of Injury - Women's Field Hockey 2004/05 to 2008/09

	Competition			Practice			Overall		
Type of Injury	**Annual Injury Count**	**% of Injuries**	**Median Days Lost**	**Annual Injury Count**	**% of Injuries**	**Median Days Lost**	**Annual Injury Count**	**% of Injuries**	**Median Days Lost**
Strain - muscle / tendon	95	11.3%	5	413	31.4%	5	508	23.5%	5
Contusion (hematoma)	222	26.2%	3	130	9.9%	3	351	16.3%	3
Sprain - ligament	122	14.4%	5	169	12.9%	4	291	13.5%	4
Concussion	97	11.4%	4	39	3.0%	4	136	6.3%	4
Fracture / Avulsion	102	12.0%	8	49	3.7%	6	151	7.0%	6
Miscellaneous	26	3.1%	5	82	6.2%	3	108	5.0%	3
Tendinosis	21	2.5%	2	67	5.1%	5	88	4.1%	5
Laceration	47	5.5%	4	30	2.3%	3	77	3.6%	4
Inflammation (general)	10	1.2%	2	48	3.7%	13	58	2.7%	12
Unknown	21	2.4%	4	40	3.1%	1	61	2.8%	2
Other	83	9.8%	6	246	18.7%	5	329	15.2%	6
	845	*100.0%*		*1,312*	*100.0%*		*2,158*	*100.0%*	

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Table 4.5 Body Site of Injury - Women's Field Hockey 2004/05 to 2008/09

	Competition			Practice			Overall		
Body Site of Injury	**Annual Injury Count**	**% of Injuries**	**Median Days Lost**	**Annual Injury Count**	**% of Injuries**	**Median Days Lost**	**Annual Injury Count**	**% of Injuries**	**Median Days Lost**
Thigh	63	7.4%	5	240	18.3%	6	303	14.0%	5
Knee	132	15.6%	4	213	16.2%	6	344	16.0%	5
Head/face	165	19.5%	4	76	5.8%	2	241	11.2%	3
Ankle	86	10.2%	4	136	10.3%	4	222	10.3%	4
Hand	128	15.2%	4	56	4.2%	4	184	8.5%	4
Hip	25	2.9%	11	145	11.0%	7	169	7.8%	8
Lower Leg	45	5.3%	4	123	9.4%	5	168	7.8%	5
Foot	36	4.3%	4	74	5.7%	4	111	5.1%	4
Lower back/L-spine/pelvis	13	1.6%	5	85	6.5%	3	99	4.6%	3
Shoulder	26	3.0%	6	19	1.5%	4	45	2.1%	6
Respiratory	13	1.5%	4	24	1.9%	1	37	1.7%	2
Nose	24	2.8%	7	10	.08%	4	34	1.6%	7
Wrist	8	1.0%	2	18	1.4%	4	27	1.2%	3
Chest/T-spine/ribs	18	2.1%	4	9	.07%	2	26	1.2%	3
Other	64	7.6%	3	84	6.4%	2	148	6.9%	2
	845	*100.0%*		*1,312*	*100.0%*		*2,158*	*100.0%*	

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Table 4.6 Most Common Specific Injuries - Women's Field Hockey 2004/05 to 2008/09

	Competition			Practice			Overall		
Specific Injury	**Annual Injury Count**	**% of Injuries**	**Median Days Lost**	**Annual Injury Count**	**% of Injuries**	**Median Days Lost**	**Annual Injury Count**	**% of Injuries**	**Median Days Lost**
Quadriceps partial tear	21	2.4%	3	125	9.6%	5	146	6.8%	4
Missing	50	5.9%	5	101	7.7%	4	151	7.0%	4
Concussion	97	11.4%	4	39	3.0%	4	136	6.3%	4
Hamstring partial tear	26	3.1%	6	88	6.7%	7	114	5.3%	6
Hand / finger contusion	40	4.7%	3	27	2.0%	1	66	3.1%	3
Knee contusion	37	4.4%	3	21	1.6%	3	58	2.7%	3
Iliotibial (IT) band friction syndrome	9	1.1%	2	48	3.7%	13	57	2.7%	11
Adductor (groin) partial tear	8	.09%	11	50	3.8%	7	58	2.7%	7
Iliopsoas / Sartorius (partial tear)	7	.09%	9	45	3.4%	7	52	2.4%	8
Other	551	65.1%	4	768	58.6%	4	1,319	61.1%	4
	845	*100.0%*		*1,312*	*100.0%*		*2,158*	*100.0%*	

Table 4.7 Time Lost -- Women's Field Hockey 2004/05 to 2008/09

	Competition		Practice		Overall	
Time Lost	**Injury Count All Years**	**Percent**	**Injury Count All Years**	**Percent**	**Injury Count All Years**	**Percent**
1-2 days	1,028	24.5	1,984	30.7	3,012	28.3
3-6 days	1,667	39.7	1,761	27.3	3,428	32.2
7-9 days	420	10.0	656	10.2	1,076	10.1
10-20 days	375	8.9	1,168	18.1	1,543	14.5
21 or more days	642	15.3	757	11.7	1,399	13.1
Other	64	1.5	133	2.1	197	1.8
	4,196	*100.0*	*6,458*	*100.0*	*10,654*	*100.0*

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Table 4.8 Injury Event Details - Women's Field Hockey 2004/05 to 2008/09

		Competition		Practice		Overall	
Event	**Details**	**Annual Injury Count**	**% of Injuries**	**Annual Injury Count**	**% of Injuries**	**Annual Injury Count**	**% of Injuries**
Season Segment	Pre-Season	34	4.0%	786	59.9%	820	38.0%
	In-Season	748	88.5%	497	37.9%	1,245	57.7%
	Post-Season	63	7.4%	29	2.2%	92	4.3%
		845	*100.0%*	*1,312*	*100.0%*	*2,158*	*100.0%*
Activity	General play	385	45.6%	568	43.3%	953	44.2%
	Conditioning	2	.03%	298	22.7%	300	13.9%
	Defending	190	22.5%	68	5.2%	258	12.0%
	Goaltending	35	4.2%	120	9.2%	155	7.2%
	Ball handling / dribbling	59	7.0%	75	5.7%	134	6.2%
	Shooting	21	2.5%	52	4.0%	73	3.4%
	Blocking shot	47	5.6%	8	.06%	55	2.6%
	Loose ball	34	4.0%	21	1.6%	55	2.5%
	Other/Unknown	9	1.1%	45	3.4%	54	2.5%
	Tackling	32	3.8%	15	1.1%	47	2.2%
	Receiving Pass	19	2.2%	27	2.1%	46	2.1%
	Passing	12	1.4%	16	1.2%	27	1.3%
		845	*100.0%*	*1,312*	*100.0%*	*2,158*	*100.0%*
Injury Mechanism	Acute Non-contact	220	26.2%	631	48.7%	851	39.8%
	Contact with ball (elevated)	194	23.0%	133	10.2%	327	15.3%
	Gradual/overuse	31	3.7%	248	19.1%	279	13.0%
	Contact with stick	146	17.4%	57	4.4%	203	9.5%
	Direct (e.g., knee injury results from direct player contact to knee))	82	9.8%	32	2.5%	115	5.4%
	Contact with ball (on the ground)	47	5.5%	56	4.3%	102	4.8%
	Illness/infection	26	3.1%	67	5.2%	93	4.4%
	Playing Surface	39	4.6%	39	3.0%	78	3.6%
	Player Contact (Other)	29	3.4%	23	1.7%	51	2.4%
	Unknown	15	1.8%	.	.	15	.07%
	Indirect (e.g., knee injury results from landing following contact to head)	6	.08%	6	.05%	13	.06%
	Contact with goal	7	.08%	4	.03%	11	.05%
		841	*100.0%*	*1,296*	*100.0%*	*2,137*	*100.0%*

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Table 4.9 Event Details for Competition Injuries - Women's Field Hockey 2004/05 to 2008/09

Event	Event Details	Annual Injury Count	% of Injuries
Competition Type	Away	469	55.5%
	Home	376	44.5%
		845	*100%*
Competition Time	Warm-up	63	7.4%
	First half	249	29.5%
	Second half	385	45.5%
	Overtime	16	1.9%
	Other	3	.04%
	Unknown	129	15.3%
		845	*100%*
Location	Other	37	4.6%
	Unknown	217	26.4%
	Middle of field	170	20.7%
	Within 25 yd line (defense)	127	15.5%
	Goal circle (defense)	118	14.4%
	Within 25 yd line (offense)	81	9.9%
	Goal circle (offense)	71	8.6%
		821	*100%*
Competition Position	Other	4	.05%
	Forward (Attack)	315	39.5%
	Midfield	244	30.6%
	Defense	201	25.3%
	Goalkeeper	33	4.1%
		797	*100%*

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Table 4.10 Mechanism of Injury for Competition Injuries - Women's Field Hockey 2004/05 to 2008/09

Competition Time	Injury Mechanism	Annual Injury Count	% of Injuries
Warm-up	Playing Surface	4	.05%
	Acute Non-contact	18	2.1%
	Direct (e.g., knee injury results from direct player contact to knee))	3	.03%
	Contact with ball (on the ground)	7	.08%
	Contact with stick	4	.04%
	Contact with ball (elevated)	26	3.1%
		62	*7.20%*
First half	Player Contact (Other)	11	1.4%
	Playing Surface	11	1.3%
	Acute Non-contact	61	7.2%
	Gradual/overuse	3	.03%
	Illness/infection	11	1.3%
	Direct (e.g., knee injury results from direct player contact to knee))	21	2.5%
	Indirect (e.g., knee injury results from landing following contact to head)	3	.04%
	Unknown	4	.05%
	Contact with ball (on the ground)	12	1.5%
	Contact with stick	43	5.1%
	Contact with ball (elevated)	67	8.0%
		247	*29.50%*
Second half	Player Contact (Other)	14	1.7%
	Playing Surface	16	1.9%
	Acute Non-contact	77	9.2%
	Gradual/overuse	7	.08%
	Illness/infection	6	.07%
	Direct (e.g., knee injury results from direct player contact to knee))	42	5.0%
	Indirect (e.g., knee injury results from landing following contact to head)	3	.03%
	Unknown	8	.09%
	Contact with ball (on the ground)	24	2.9%
	Contact with stick	85	10.1%
	Contact with goal	7	.08%
	Contact with ball (elevated)	94	11.1%
		383	*45.40%*

...continued

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Competition Time	Injury Mechanism	Annual Injury Count	% of Injuries
Overtime	Playing Surface	2	.02%
	Acute Non-contact	4	.05%
	Gradual/overuse	2	.03%
	Direct (e.g., knee injury results from direct player contact to knee))	3	.03%
	Contact with ball (on the ground)	3	.03%
	Contact with stick	2	.02%
		16	*1.80%*
Other	Player Contact (Other)	1	.01%
	Illness/infection	2	.03%
		3	*.04%*
Unknown	Player Contact (Other)	2	.03%
	Playing Surface	5	.06%
	Acute Non-contact	60	7.2%
	Gradual/overuse	19	2.3%
	Illness/infection	7	.09%
	Direct (e.g., knee injury results from direct player contact to knee))	13	1.5%
	Unknown	3	.03%
	Contact with stick	12	1.5%
	Contact with ball (elevated)	7	.08%
		128	*15.40%*

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Table 4.11 Mechanism of Injury by Activity for Competition Injuries - Women's Field Hockey 2004/05 to 2008/09

Activity	Injury Mechanism	Annual Injury Count	% of Injuries
Defending	Contact with ball (elevated)	62	7.9%
	Contact with stick	42	5.3%
	Acute Non-contact	25	3.2%
	Direct (e.g., knee injury results from direct player contact to knee))	19	2.4%
	Contact with ball (on the ground)	16	2.1%
	Player Contact (Other)	9	1.2%
	Playing Surface	6	.08%
		180	*22.7%*
General play	Acute Non-contact	115	14.5%
	Contact with stick	58	7.3%
	Contact with ball (elevated)	53	6.6%
	Direct (e.g., knee injury results from direct player contact to knee))	32	4.0%
	Gradual/overuse	29	3.6%
	Illness/infection	24	3.0%
	Playing Surface	23	2.9%
	Contact with ball (on the ground)	19	2.3%
	Player Contact (Other)	12	1.5%
	Unknown	7	.09%
	Indirect (e.g., knee injury results from landing following contact to head)	2	.02%
		373	*46.9%*
Other	Acute Non-contact	66	8.4%
	Contact with ball (elevated)	57	7.2%
	Contact with stick	41	5.2%
	Direct (e.g., knee injury results from direct player contact to knee)	29	3.7%
	Playing Surface	9	1.2%
	Player Contact (Other)	8	1.0%
	Unknown	8	1.0%
	Contact with ball (on the ground)	7	.09%
	Contact with goal	7	.08%
	Indirect (e.g., knee injury results from landing following contact to head)	4	.06%
	Gradual/overuse	2	.03%
	Illness/infection	2	.03%
		242	*30.4%*

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Table 4.12 Event Details by Practice Segment and Type for Practice Injuries - Women's Field Hockey 2004/05 to 2008/09

Event	Event Details	Annual Injury Count	% of Injuries
Practice Segment	Team drills	566	48.0%
	Conditioning	258	21.9%
	Unknown	183	15.5%
	Individual drills	98	8.3%
	Other	41	3.4%
	Warm-up	34	2.9%
		1,179	*100%*
Practice Type	Regular	1,099	93.3%
	Scrimmage	54	4.6%
	Walk-through	26	2.2%
		1,179	*100%*

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Table 4.13 Injury Laterality, Recurrence, Chronicity, and Surgery - Women's Field Hockey 2004/05 to 2008/09

		Competition		Practice		Overall	
Type	**Details**	**Annual Injury Count**	**% of Injuries**	**Annual Injury Count**	**% of Injuries**	**Annual Injury Count**	**% of Injuries**
Side of Body	Left	138	36.2	214	39.5	352	38.2
	Right	148	42.5	221	39.8	369	40.9
	Both	7	1.6	48	8.3	55	5.7
	Not Applicable	69	19.6	73	12.4	142	15.2
		362	*100.0*	*556*	*100.0*	*918*	*100.0*
Injury Recurrence	Recurrence (prior to college)	2	.06	24	4.6	26	3.0
	Recurrence (previous academic year)	12	3.6	47	8.8	59	6.8
	Recurrence (this academic year)	7	1.8	23	3.9	30	3.1
	New	340	93.5	461	82.6	801	86.9
	Other	1	.04	1	.02	2	.03
		362	*100.0*	*556*	*100.0*	*918*	*100.0*
Chronic Injury	Yes	6	1.9	25	4.5	31	3.5
	No	356	98.1	531	95.5	887	96.5
		362	*100.0*	*556*	*100.0*	*918*	*100.0*
Surgery Resulted from this Injury	Yes	24	7.0	34	5.8	58	6.2
	No	338	93.0	520	94.2	858	93.8
		362	*100.0*	*554*	*100.0*	*916*	*100.0*

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Table 4.14 Mechanism of Injury by Activity for Competition Concussions - Women's Field Hockey 2004/05 to 2008/09

Activity	Injury Mechanism	Annual Injury Count	% of Injuries
Defending	Direct (e.g., knee injury results from direct player contact to knee)	11	12.3%
	Contact with ball (elevated)	4	4.5%
	Playing Surface	3	3.8%
	Contact with stick	1	1.6%
		20	*22.2%*
General play	Direct (e.g., knee injury results from direct player contact to knee)	13	14.6%
	Contact with stick	9	10.4%
	Contact with ball (elevated)	7	7.3%
	Player Contact (Other)	5	5.2%
	Playing Surface	3	3.0%
		36	*40.5%*
Loose ball	Contact with ball (elevated)	9	9.5%
	Player Contact (Other)	2	2.6%
		11	*12.1%*
Other	Contact with stick	7	8.3%
	Direct (e.g., knee injury results from direct player contact to knee)	7	7.9%
	Contact with goal	4	4.8%
	Contact with ball (elevated)	4	4.2%
		23	*25.3%*

Table 4.15 Qualified and Sponsoring Schools - Women's Field Hockey 2004/05 to 2008/09

Division	Average Annual Number of Qualified Schools	Average Annual Number of Sponsoring Schools	Percent of Sponsoring Schools that are Qualified
All Divisions*	22	230	9.6%
Division I	11	76	13.9%
Division III	12	154	7.5%

* No Division II schools submitted data

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Descriptive Epidemiology of Collegiate Women's Field Hockey Injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 Through 2002–2003

Randall Dick, MS, FACSM*; Jennifer M. Hootman, PhD, ATC, FACSM†; Julie Agel, MA, ATC‡; Luzita Vela, PhD, ATC§; Stephen W. Marshall, PhD‖; Renee Messina, MS, ATC¶

*National Collegiate Athletic Association, Indianapolis, IN; †Centers for Disease Control and Prevention, Atlanta, GA; ‡University of Minnesota, Minneapolis, MN; §West Chester University, West Chester, PA; ‖University of North Carolina at Chapel Hill, Chapel Hill, NC; ¶Pennsylvania State University, University Park, PA

Objective: To review 15 years of National Collegiate Athletic Association (NCAA) injury surveillance data for women's field hockey and identify potential areas for injury prevention initiatives.

Background: Field hockey is one of the most popular sports worldwide and is growing in participation in the United States, particularly among women. From 1988–1989 to 2002–2003, participation in NCAA women's field hockey increased 12%, with the largest growth among Division III programs. In 2002–2003, 253 colleges offered women's field hockey and 5385 women participated.

Main Results: Game injury rates showed a significant average annual 2.5% decline over 15 years, most likely fueled by drops in ankle ligament sprain, knee internal derangement, and finger fracture injuries. Despite this, ankle ligament sprains were common (13.7% of game and 15.0% of practice injuries) and a frequent cause of severe injuries (resulting in 10+ days of time-loss activity). Concussion and head laceration injuries increased over this same time, and the risk of sustaining a concussion in a game was 6 times higher than the risk of sustaining one during practice. Overall, injury rates were twice as high in games as in practices (7.87 versus 3.70 injuries per 1000 athlete-exposures, rate ratio = 2.1, 95% confidence interval = 2.0, 2.3). Most head/neck/face (71%) and hand/finger/thumb (68%) injuries occurred when the player was near the goal or within the 25-yd line and were caused by contact with the stick or ball (greater than 77% for both body sites); for 34% of head/neck/face injuries, a penalty was called on the play.

Recommendations: Equipment (requiring helmets and padded gloves) and rule changes (to decrease field congestion near the goal) as well as evidence-based injury prevention interventions (eg, prophylactic ankle taping/bracing, neuromuscular balance exercise programs) may be viable prevention initiatives for reducing injury rates in women's collegiate field hockey players.

Key Words: athletic injuries, injury prevention, knee injuries, ankle injuries, concussions

The National Collegiate Athletic Association (NCAA) conducted its first women's field hockey championship in 1981. In the 1988–1989 academic year, 225 schools were sponsoring varsity women's field hockey teams, with 4905 participants. By 2002–2003, the number of varsity teams had increased 12% to 253, involving 5385 participants.[1] Participation growth during this time was apparent primarily in Division III.

SAMPLING AND METHODS

Over the 15-year period from 1988–1989 through 2002–2003, an average of 17.6% of schools sponsoring varsity women's field hockey programs participated in annual NCAA Injury Surveillance System (ISS) data collection (Table 1). Field hockey data were not collected in 2003–2004 as a result of pilot testing for conversion to a Web-based system. The sampling process, data collection methods, injury and exposure definitions, inclusion criteria, and data analysis methods are described in detail in the ''Introduction and Methods'' article in this special issue.[2]

RESULTS

Game and Practice Athlete-Exposures

The average annual numbers of games, practices and athletes participating for each NCAA division, condensed over the study period, are shown in Table 2. Division I annually averaged 7 to 10 more practices and 1 to 2 more games than did Divisions II and III. Average numbers of game and practice athletes were similar in all divisions.

Injury Rate by Activity, Division, and Season

Game and practice injury rates over years combined across divisions with 95% confidence intervals (CIs) are presented in Figure 1. A significant average annual decrease in game (−2.5%, $P < .01$) injury rates and a small, nonsignificant average annual increase in practice (0.3%, $P = .74$) injury rates occurred over the sample period. Over the 15 years of the study, the rate of injury in a game situation was more than 2 times higher than in practice (7.87 versus 3.70 injuries per

Table 1. School Participation Frequency (in Total Numbers) by Year and National Collegiate Athletic Association (NCAA) Division, Women's Field Hockey 1988–1989 Through 2002–2003*

Academic Year	Division I Schools		Division II Schools		Division III Schools		All Divisions		
	Participating	Sponsoring	Participating	Sponsoring	Participating	Sponsoring	Participating	Sponsoring	Percentage
1988–1989	18	78	4	22	24	125	46	225	20.4
1989–1990	12	76	2	20	21	123	35	219	16.0
1990–1991	14	76	3	13	22	128	39	217	18.0
1991–1992	13	74	5	13	24	126	42	213	19.7
1992–1993	14	71	3	20	23	120	40	211	19.0
1993–1994	18	71	5	22	20	121	43	214	20.1
1994–1995	20	71	3	22	23	128	46	221	20.8
1995–1996	15	73	2	23	24	135	41	231	17.7
1996–1997	19	71	3	25	26	132	48	235	20.4
1997–1998	19	72	1	27	23	135	43	234	18.4
1998–1999	7	73	1	26	10	141	18	240	7.5
1999–2000	15	75	1	24	28	140	44	239	18.4
2000–2001	8	76	5	25	22	147	35	248	14.1
2001–2002	15	77	2	26	16	149	33	252	13.1
2002–2003	21	77	5	26	24	150	50	253	19.8
Average	15	74	3	22	22	133	40	230	17.6

*''Participating'' refers to schools that provided appropriate data to the NCAA Injury Surveillance System; ''Sponsoring'' refers to the total number of schools offering the sport within the NCAA divisions.

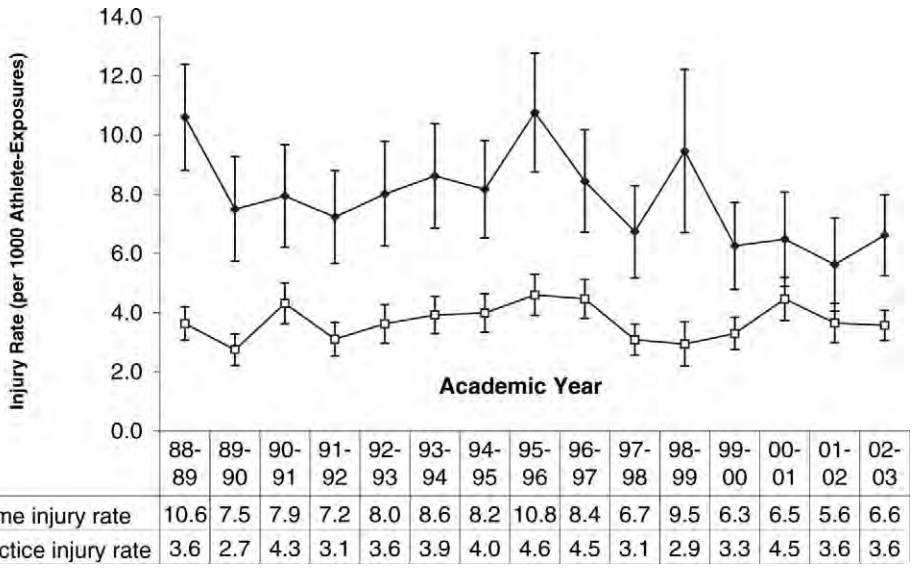

Figure 1. Injury rates and 95% confidence intervals per 1000 athlete-exposures by games, practices, and academic year, women's field hockey, 1988–1989 through 2002–2003 (n = 1220 game injuries and 2066 practice injuries). Game time trend *P* < .01. Average annual change in game injury rate = −2.5% (95% confidence interval = −4.3, −0.6). Practice time trend *P* = .74. Average annual change in practice injury rate = 0.3% (95% confidence interval = −1.5, 2.1).

1000 athlete-exposures [A-Es] (data not shown), rate ratio = 2.1, 95% CI = 2.0, 2.3).

The total number of games and practices and associated injury rates condensed over years by division and season (preseason, in season, and postseason) are presented in Table 3. Over the 15-year period, 1220 injuries from 10 358 games and 2066 injuries from 26 740 practices were reported. The game injury rate did not differ among divisions ($P = .25$) or within seasons ($P = .34$). The practice injury rate did not differ among divisions ($P = 0.71$) but did differ within season ($P < 0.01$). Injury rates were almost 3 times as high in preseason practice as in regular-season practice (6.37 versus 2.21 injuries per 1000 A-Es, rate ratio = 2.9, 95% CI = 2.7, 3.1) and were 4 times higher than postseason practice rates (6.37 versus 1.63 injuries per 1000 A-Es, rate ratio = 3.9, 95% CI = 2.8, 5.4).

Body Parts Injured Most Often and Specific Injuries

The frequency of injury to 5 general body parts (head/neck, upper extremity, trunk/back, lower extremity, and other/system) for games and practices with years and divisions combined is shown in Table 4. More than 40% of all game injuries and 60% of all practice injuries were to the lower extremity. Head/neck (25.3%) and upper extremity (20.7%) injuries were also common game injuries. In practices, the trunk/back (16.2%) was the second most commonly injured area.

The most common body part and injury type combinations for games and practices with years and divisions combined are displayed in Table 5. All injuries that accounted for at least 1% of reported injuries over the 15-year sampling period were included. In games, ankle ligament sprains (13.7%), knee internal derange-

Table 2. Average Annual Games, Practices, and Athletes Participating by National Collegiate Athletic Association Division per School, Women's Field Hockey, 1988–1989 Through 2002–2003

Division	Games	Athletes per Game	Practices	Athletes per Practice
I	19	15	52	21
II	18	15	45	21
III	17	15	42	21

Table 4. Percentage of Game and Practice Injuries by Major Body Part, Women's Field Hockey, 1988–1989 Through 2002–2003

Body Part	Games	Practices
Head/neck	25.3	8.4
Upper extremity	20.7	8.1
Trunk/back	7.1	16.2
Lower extremity	43.2	60.2
Other/system	3.8	7.1

ment injuries (10.2%), concussions (9.4%), upper leg muscle strains (7.0%), and finger fractures (6.5%) were the 5 most common injuries. In practices, upper leg muscle strains accounted for 26.9% of all reported injuries, whereas ankle ligament sprains (15.0%), pelvis-hip muscle strains (9.9%), and knee internal derangements (7.8%) were also common injury categories. A participant had 6 times the risk of sustaining a concussion in a game as in a practice (0.52 versus 0.09 per 1000 A-Es, rate ratio = 6.1, 95% CI = 4.3, 8.7), almost 3 times the risk of sustaining a knee internal derangement in a game as in a practice (0.57 versus 0.20 per 1000 A-Es, rate ratio = 2.9, 95% CI = 2.2, 3.8), and twice the risk of sustaining an ankle ligament sprain in a game as in a practice (0.76 versus 0.37 per 1000 A-Es, rate ratio = 2.0, 95% CI = 1.6, 2.6).

Mechanism of Injury

The 3 primary injury mechanisms—player contact, other contact (eg, with balls, sticks, ground), and no contact—in games and practices with division and years combined are presented in Figure 2. The majority of game injuries (approximately 60%) resulted from other (stick and ball) contact mechanisms. Approximately 26% of game injuries were associated with no contact and 13% with player contact. Most practice injuries (64%) involved no contact.

Severe Injuries: 10+ Days of Activity Time Loss

The top injuries that resulted in at least 10 consecutive days of restricted or total loss of participation and their primary injury mechanisms combined across divisions and years are shown in Table 6. Time loss of 10+ days was, for this analysis, considered a measure of injury severity. Approximately 15% of game injuries and 13% of practice injuries restricted the athlete's participation for at least 10 days. In games, knee internal derangements (23.1%), finger fractures (9.1%), and ankle ligament sprains (9.1%) accounted for the largest percentage of severe injuries. Concussions represented 5.4% of severe game injuries. In practices, knee internal derangements (15.3%), lower leg stress fractures (9.7%), and ankle ligament sprains (8.2%) were the top 3 severe injury categories.

Table 3. Games and Practices With Associated Injury Rates by National Collegiate Athletic Association Division and Season, Women's Field Hockey, 1988–1989 Through 2002–2003*

	Total No. of Games Reported	Game Injury Rate per 1000 Athlete-Exposures	95% Confidence Interval	Total No. of Practices Reported	Practice Injury Rate per 1000 Athlete-Exposures	95% Confidence Interval
Division I						
Preseason	131	4.99	1.90, 8.08	4075	5.87	5.36, 6.39
In season	3762	8.45	7.69, 9.21	7103	2.18	1.95, 2.42
Postseason	258	5.93	3.45, 8.41	506	2.20	1.28, 3.12
Total Division I	4159	8.19	7.48, 8.90	11 699	3.53	3.29, 3.76
Division II						
Preseason	12	10.70	0.00, 25.53	702	6.75	5.42, 8.08
In season	711	10.48	8.55, 12.41	1013	1.99	1.39, 2.59
Postseason	29	9.69	0.19, 19.19	42	0.00	0.00, 0.00
Total Division II	753	10.62	8.73, 12.51	1757	3.84	3.21, 4.48
Division III						
Preseason	184	7.26	4.16, 10.37	4511	6.74	6.22, 7.25
In season	4847	7.20	6.58, 7.81	8093	2.26	2.03, 2.49
Postseason	389	7.84	5.53, 10.16	633	1.29	0.66, 1.93
Total Division III	5446	7.25	6.66, 7.82	13 284	3.83	3.60, 4.06
All Divisions						
Preseason	327	6.49	4.28, 8.71	9288	6.37	6.02, 6.72
In season	9320	7.96	7.49, 8.42	16 209	2.21	2.05, 2.37
Postseason	676	7.19	5.51, 8.88	1181	1.63	1.11, 2.15
Total	10 358	7.87	7.43, 8.31	26 740	3.70	3.54, 3.86

*Wald χ^2 statistics from negative binomial model: game injury rates did not differ among divisions ($P = .25$) or within seasons ($P = .34$). Practice injury rates did not differ among divisions ($P = .71$) but did differ within seasons ($P < .01$). Postseason samples sizes are much smaller (and have higher variability than preseason and in season sample sizes because only a small percentage of schools participated in the postseason tournament in any sport, and not all of those were a part of the Injury Surveillance System sample. Numbers do not always sum to totals because of missing division or season information.

Table 5. Most Common Game and Practice Injuries, Women's Field Hockey, 1988–1989 Through 2002–2003*

Body Part	Injury Type	Frequency	Percentage of Injuries	Injury Rate per 1000 Athlete-Exposures	95% Confidence Interval
Games					
Ankle	Ligament sprain	118	13.7	0.76	0.62, 0.90
Knee	Internal derangement	88	10.2	0.57	0.45, 0.69
Head	Concussion	81	9.4	0.52	0.41, 0.64
Upper leg	Muscle-tendon strain	60	7.0	0.39	0.29, 0.49
Finger(s)	Fracture	56	6.5	0.36	0.27, 0.46
Nose	Fracture	45	5.2	0.29	0.21, 0.38
Face	Laceration	36	4.2	0.23	0.16, 0.31
Head	Laceration	31	3.6	0.20	0.13, 0.27
Patella	Patella or patella tendon injury	31	3.6	0.20	0.13, 0.27
Unspecified†	Unspecified	29	3.4	0.19	0.12, 0.26
Foot	Contusion	25	2.9	0.16	0.10, 0.22
Lower leg	Contusion	25	2.9	0.16	0.10, 0.22
Upper leg	Contusion	25	2.9	0.16	0.10, 0.22
Pelvis, hip	Muscle-tendon strain	24	2.8	0.15	0.09, 0.22
Hand	Contusion	22	2.5	0.14	0.08, 0.20
Knee	Contusion	21	2.4	0.14	0.08, 0.19
Thumb	Fracture	20	2.3	0.13	0.07, 0.19
Lower back	Muscle-tendon strain	18	2.1	0.12	0.06, 0.17
Eye(s)	Laceration	15	1.7	0.10	0.05, 0.15
Face	Contusion	15	1.7	0.10	0.05, 0.15
Mouth	Laceration	15	1.7	0.10	0.05, 0.15
Thumb	Contusion	14	1.6	0.09	0.04, 0.14
Ankle	Contusion	13	1.5	0.08	0.04, 0.13
Finger(s)	Contusion	12	1.4	0.08	0.03, 0.12
Hand	Fracture	12	1.4	0.08	0.03, 0.12
Knee	Muscle-tendon strain	12	1.4	0.08	0.03, 0.12
Practices					
Upper leg	Muscle-tendon strain	376	26.9	0.67	0.61, 0.74
Ankle	Ligament sprain	209	15.0	0.37	0.32, 0.43
Pelvis, hip	Muscle-tendon strain	138	9.9	0.25	0.21, 0.29
Knee	Internal derangement	109	7.8	0.20	0.16, 0.23
Unspecified†	Unspecified	74	5.3	0.13	0.10, 0.16
Lower back	Muscle-tendon strain	73	5.2	0.13	0.10, 0.16
Patella	Patella or patella tendon injury	58	4.2	0.10	0.08, 0.13
Lower leg	Muscle-tendon strain	51	3.7	0.09	0.07, 0.12
Head	Concussion	48	3.4	0.09	0.06, 0.11
General body	Heat illness	42	3.0	0.08	0.05, 0.10
Lower leg	Stress fracture	36	2.6	0.06	0.04, 0.09
Buttocks	Muscle-tendon strain	33	2.4	0.06	0.04, 0.08
Knee	Tendinitis	30	2.1	0.05	0.03, 0.07
Finger(s)	Fracture	27	1.9	0.05	0.03, 0.07
Knee	Muscle-tendon strain	25	1.8	0.04	0.03, 0.06
Heel/Achilles tendon	Tendinitis	24	1.7	0.04	0.03, 0.06
Lower leg	Inflammation	23	1.6	0.04	0.02, 0.06
Knee	Inflammation	20	1.4	0.04	0.02, 0.05

*Only injuries that accounted for at least 1% of all injuries are included.
†"Unspecified" indicates injuries that could not be grouped into existing categories but that were believed to constitute reportable injuries.

Game Injuries

Starting with the 1996–1997 academic year, value changes for several ISS variables (ie, mechanism of injury, field location when the injury occurred, player position, and penalty play status), made additional game analyses possible. Figures 3 through 5 and Tables 7 through 10 report game-only data for those years.

Game injury mechanisms in more detail are displayed in Figure 3. Contact with the ball (29%) and contact with the stick (19%) were the injury mechanisms for almost half of the game injuries. Player contact accounted for 14% of game injuries.

Game field location at time of injury is presented in Figure 4. More than two thirds of the game injuries occurred inside the 25-yd line (41%) or in the area around the goal (26%).

The weighted percentage of game injuries (1996–1997 through 2002–2003) by player position is presented in Figure 5. Percentages were weighted based on the player position distribution on the field during typical play: goalie = 1, defense (backfielders) = 3, midfielders = 3, and forwards = 4. Rates could not be calculated by player position because of lack of game exposure data by position. Most game injuries occurred among midfielders (28%), whereas the re-

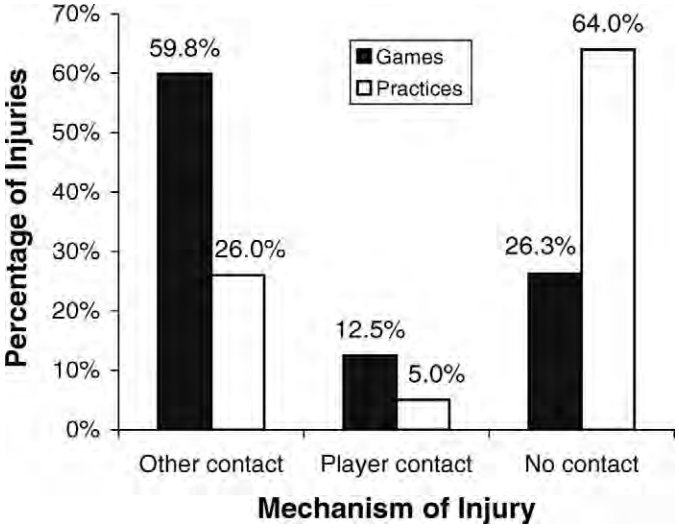

Figure 2. Game and practice injury mechanisms, all injuries, women's field hockey, 1988–1989 through 2002–2003 (n = 1220 game injuries and 2066 practice injuries). "Other contact" refers to contact with items such as balls, sticks, or the ground. Injury mechanism was unavailable for 2% of game injuries and 6% of practice injuries.

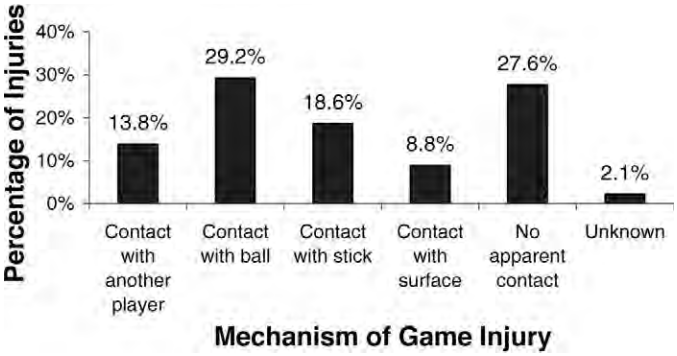

Figure 3. Sport-specific game injury mechanisms, women's field hockey, 1996–1997 through 2002–2003 (n = 1220). Reliable information on game injury mechanism was collected starting in 1996–1997.

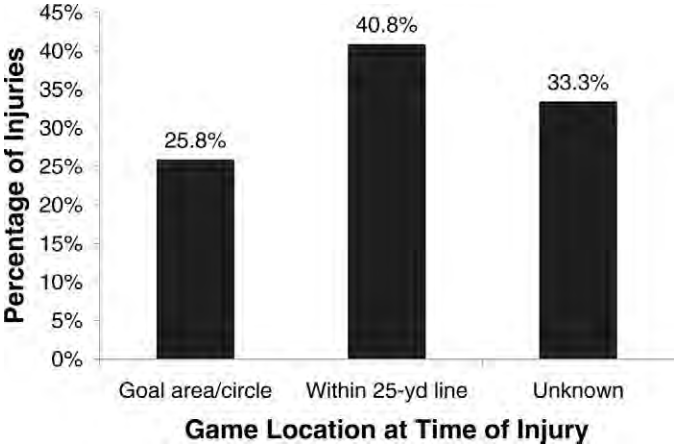

Figure 4. Game field location at time of injury, women's field hockey, 1996–1997 through 2002–2003 (n = 480). The goal area/circle is exclusive of the area within the 25-yd line on a regulation-size field. Reliable information on game field location was collected starting in 1996–1997.

maining injuries were distributed similarly among forwards (22%), backfielders/defense (24%), and goalies (20%).

The frequency of specific game above-the-neck injuries and their top 2 mechanisms are shown in Table 7. Above-the-neck injuries represented approximately one fourth (24%) of all game injuries. Most above-the-neck injuries resulted from contact with the ball or stick.

The frequency of specific hand and finger injuries and their top 2 mechanisms are displayed in Table 8. Ten percent of all game injuries were associated with the hand. The majority of these injuries resulted from stick contact.

The top reported game injury sites and types, injury rates, and confidence intervals for 2 select time periods based on the phasing in of offsides and corner-hit rule changes in the

Table 6. Most Common Game and Practice Injuries Resulting in 10+ Days of Activity Time Loss, Women's Field Hockey, 1988–1989 Through 2002–2003

Body Part	Injury Type	Frequency	Percentage of Severe Injuries	Most Common Injury Mechanism
Games (15.0% of all injuries required 10+ days of time loss)				
Knee	Internal derangement	43	23.1	No contact
Finger(s)	Fracture	17	9.1	Other contact*†
Ankle	Ligament sprain	17	9.1	No contact
Thumb	Fracture	10	5.4	Other contact*†
Head	Concussion	10	5.4	Player contact, other contact*
Other		89	47.8	
Total		186		
Practices (13.0% of all injuries required 10+ days of time loss)				
Knee	Internal derangement	41	15.3	No contact
Lower leg	Stress fracture	26	9.7	No contact
Ankle	Ligament sprain	22	8.2	No contact, other contact‡
Upper leg	Muscle-tendon strain	14	5.2	No contact
Other		165	61.6	
Total		268		

*Indicates contact with ball.
†Indicates contact with stick.
‡Indicates contact with surface.

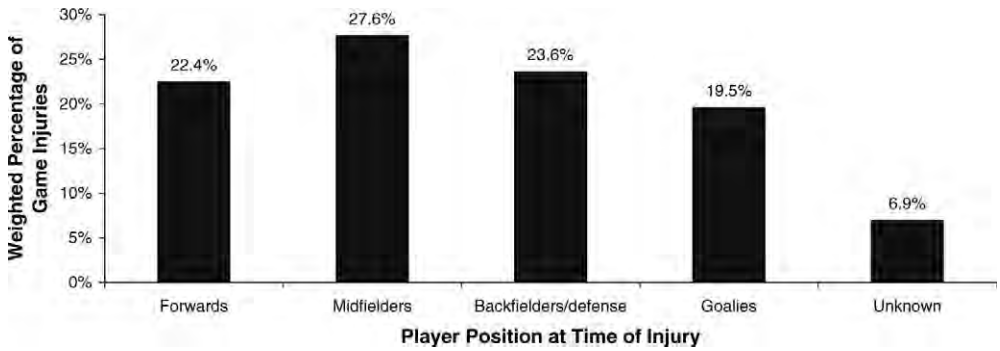

Figure 5. Game injuries by player position, weighted percentage, women's field hockey, 1996–1997 through 2002–2003 (weighted n = 182). Percentages are weighted based on the distribution of player positions on the field during typical play: goalie = 1, backfielders = 3, midfielders = 3, and forwards = 4. "Unknown" position was given a weight equal to 1. Reliable information on player position at time of game injury was collected starting in 1996–1997.

sport during the 1995–1996 year are presented in Table 9. The overall game injury rate declined significantly from 8.65 (95% CI = 8.03, 9.28) to 6.91 (95% CI = 6.29, 7.53) per 1000 A-Es from the first to the second time periods. Numbers and rates of ankle ligament sprains, knee internal derangements, and finger fractures declined after this rule change took effect. However, these decreases were not statistically significant, as the 95% CIs overlapped between the 2 time periods. Also not statistically significant, but potentially important, is the fact that the numbers and rates of concussions and head lacerations increased from the first to the second time period. In fact, in the latter time period, concussions replaced ankle ligament sprains as the most common game injury in terms of both number and rate.

Select game injuries by player position, field location, injury mechanism, and penalty play status at the time of injury are broken down in Table 10. Above-the-neck game injuries were almost equally distributed among positions, with the exception of goalies, but 69.3% occurred within the 25-yd line or near the goal, and 77.4% were caused by contact with the stick or ball. In addition, 33.6% of above-the-neck injuries occurred during a play in which a penalty was called. Most hand/finger/thumb game injuries occurred to backfielders (39.0%), and almost all (94.9%) were caused by contact with the stick or ball.

COMMENTARY

Even though field hockey is one of the most popular sports worldwide, very little recent descriptive injury epidemiology has been published on the sport.[3–6] These data from the NCAA ISS constitute an important contribution to the literature and provide critical information that can be used to promote injury prevention efforts in the sport. Overall, these data indicate that injury rates declined in the sport over these years, and catastrophic injuries were rare. Specifically, game injury rates have shown a significant average annual 2.5% decline over 15 years, and practice injury rates did not change in either direction. However, these data also highlight several issues that may be amenable to injury prevention initiatives, making the sport even safer and, subsequently, more enjoyable.

These NCAA data on field hockey injuries are unique, in that no other authors have used standardized case definitions for in-

Table 7. Above-the-Neck Game Injuries With Most Common Injury Mechanisms, Women's Field Hockey, 1988–1989 Through 2002–2003*

Body Part	Injuries, No.	Most Common Mechanism of Injury	Injuries at Each Body Part Due to Each Mechanism, %
Ear	3	Contact with ball	66
		Contact/collision with another player	33
Eye	22	Contact with stick	45
		Contact with ball	45
Face	54	Contact with ball	63
		Contact with stick	31
Head	120	Contact with stick	41
		Contact/collision with another player	29
Teeth	7	Contact with stick	71
		Contact with ball	29
Nose	61	Contact with ball	56
		Contact with stick	27
Mouth	17	Contact with ball	53
		Contact with stick	41
Jaw (temporomandibular joint)	7	Contact with stick	43
		Contact with ball	43
Total	291		

*Above-the-neck injuries accounted for 24% of all game injuries during this interval.

Table 8. Game Finger and Hand Injuries With Most Common Injury Mechanisms, Women's Field Hockey, 1996–1997 Through 2002–2003*

Body Part	Injuries, No.	Most Common Mechanisms of Injury	Injuries at Each Body Part Due to Each Mechanism, %
Finger/thumb	87	Contact with ball	49
		Contact with stick	48
Hand	39	Contact with stick	51
		Contact with ball	31
Total	126		

*Finger and hand injuries accounted for 10% of all game injuries during this interval.

Table 9. Most Common Women's Field Hockey Game Injuries Before (1988–1989 through 1995–1996) and After (1996–1997 through 2002–2003) Select Rule Changes*

Body Part	Injury Type	Number	Percentage of All Injuries	Rate per 1000 Athlete-Exposures	95% Confidence Interval
Game injuries, 1988–1989 through 1995–1996					
Ankle	Ligament sprain	78	10.54	0.91	0.71, 1.11
Knee	Internal derangement	52	7.03	0.61	0.44, 0.77
Finger	Fracture	38	5.14	0.44	0.30, 0.59
Head	Concussion	38	5.14	0.44	0.30, 0.59
Upper leg	Muscle-tendon strain	32	4.32	0.37	0.24, 0.50
Nose	Fracture	23	3.11	0.27	0.16, 0.38
Face	Laceration	23	3.11	0.27	0.16, 0.38
Upper leg	Contusion	19	2.57	0.22	0.12, 0.32
Patella	Patella or patella tendon injury	19	2.57	0.22	0.12, 0.32
Lower leg	Contusion	17	2.30	0.20	0.10, 0.29
Total injuries		740	100.00	8.65	8.03, 9.28
Total exposures		85 508			
Game injuries, 1996–1997 through 2002–2003					
Head	Concussion	43	8.96	0.62	0.43, 0.80
Ankle	Ligament sprain	40	8.33	0.58	0.40, 0.75
Knee	Internal derangement	36	7.50	0.52	0.35, 0.69
Upper leg	Muscle-tendon strain	28	5.83	0.40	0.25, 0.55
Nose	Fracture	22	4.58	0.32	0.18, 0.45
Finger	Fracture	18	3.75	0.26	0.14, 0.38
Head	Laceration	15	3.13	0.22	0.11, 0.33
Face	Laceration	13	2.71	0.19	0.09, 0.29
Patella	Patella or patella tendon injury	12	2.50	0.17	0.07, 0.27
Pelvis/hip	Muscle-tendon strain	11	2.29	0.16	0.06, 0.25
Total injuries		480	100.00	6.91	6.29, 7.53
Total exposures		69 478			

*In 1996, provisional new rules (placement of corner shots and no offsides) were implemented in collegiate women's field hockey. The rules became permanent in 1998.

juries or exposure classification in a clearly defined population of collegiate athletes for such a prolonged period of time. Earlier researchers reported on mixed populations (ie, recreational, scholastic, and elite players) and the findings, therefore, may not be directly comparable. Despite this, comparisons with other field hockey injury studies can be made with caution. Not surprisingly, injury rates per 1000 A-Es in US high school girls' field hockey[4] are lower (practices = 1.4, games = 4.9) than reported here for collegiate field hockey players (practices = 3.7, games = 7.9), but the body-site distribution and major types of injury are similar. In a study of ankle injuries in 4 sports among 901 high school and collegiate athletes, Beynnon et al[7] reported that inversion ankle ligament injuries accounted for a lower proportion of practice injuries (4.8%) than in these ISS data (10.2% for ankle ligament sprains) but a higher rate (0.97 per 1000 person-days versus 0.41 per 1000 A-Es in the ISS). Junge et al[6] reported competition-only injuries for the 2004 Olympic Games; however, only 8 injuries were reported and, thus, it is difficult to compare injury rates based on these small numbers. One notable finding, however, is that 6 of the 8 Olympic Games injuries were to the head or hand and finger regions, both of which are frequently injured body sites in NCAA field hockey. Differences among the studies may be related to the different exposure classifications used (eg, person-day, A-E, player-hour), different case definitions (time loss, required medical treatment, etc), small sample sizes, and possible true variations in injury rates among collegiate and elite field hockey programs.

The most comprehensive and recent study on field hockey injuries is from a Canadian sample of 161 players, including high school, club/recreational, collegiate, and national team members.[3] Despite differences in injury definition (defined as ''acute injuries'' without mention of time loss or need for medical attention) and exposure classifications (athlete-year), the results are strikingly consistent with the ISS findings in that head and face injuries, upper extremity fractures, and ankle sprains were common. Murtaugh[3] was also the first to high-

Table 10. Breakdown of Select Women's Field Hockey Game Injuries by Position, Location on the Field, Injury Mechanism, and Penalty Play Status, 1996–1997 Through 2002–2003*

	Above the Neck		Hand/Finger	
	Frequency	Percentage	Frequency	Percentage
Player position				
Forward	45	32.8	17	28.8
Midfield	43	31.4	14	23.7
Defense/backfield	40	29.2	23	39.0
Goalie	4	2.9	0	0.0
Unknown	5	3.6	5	8.5
Location at time of injury				
Goal	41	29.9	15	25.4
Within 25 yd-line	54	39.4	27	45.8
Other	42	30.7	17	28.8
Mechanism of injury				
Contact with another player	27	19.7	0	0.0
Contact with ball	64	46.7	29	49.2
Contact with stick	42	30.7	27	45.8
Contact with goal	1	0.7	0	0.0
Contact with surface	2	1.5	3	5.1
Unknown	1	0.7	0	0.0
Penalty called				
Yes	46	33.6	10	16.9
No	91	66.4	49	83.1
Total	137		59	

*"Above the neck" includes injuries to the head, face, eye, ear, teeth, nose, mouth, and jaw. "Hand/finger" includes injuries to the hand, fingers, and thumb. Reliable information on game injuries regarding field location, player position, mechanism, and whether a penalty was called on the play during which the injury occurred was collected starting in 1996–1997.

light different injury rates by player position, with goalies, midfielders, and multiposition players having the highest injury rates. We could not calculate injury rates by position because exposure data by player position were lacking, but backfielders/defense (24%) and midfielders (28%) had the highest weighted proportion of all injuries. (Weighted percentages were based on the distribution of player positions during typical play: goalie = 1, backfielders = 3, midfielders = 3, and forwards = 4.) In both studies, however, ankle injuries accounted for a large number of injuries per year; thus, they are an important target for injury prevention initiatives in the sport of field hockey. In contrast to the ISS data reported here, Murtaugh[3] noted a much higher frequency of low back pain: 54% of players reported low back pain anytime during the season. This may be because (1) the data collection instrument Murtaugh[3] used had a separate, specific question regarding low back pain and injury, and (2) the NCAA ISS may not be sensitive enough to consistently detect all chronic/overuse injuries such as low back syndrome, medial tibial stress syndrome, etc. Chronic overuse injuries may not always restrict the player from playing or practice for at least 1 day, a requirement of the case definition used in the NCAA ISS.

A statistically significant drop in game injury rates occurred from the early years (1988–1989 through 1995–1996) to the later years (1996–1997 through 2002–2003). This time period coincides with important rule changes, the beginning of technological advances in material design for sticks and protective equipment, and the increase in play on artificial surfaces in field hockey. Elimination of the offsides rule and a change in the long corner-shot location (from the corner to the sideline) were designed to encourage offense, leading to a more fast-paced game with fewer stops and starts, and have allowed the referees to keep the game moving. Both of these rules were experimentally introduced in 1996 and became permanent in 1998.[8] During the 1990s, traditional wood hockey sticks were replaced with lighter and stronger sticks made from composite materials. In addition, the shin guards, which used to be made only of molded foam, are now covered with a hard plastic shell. Play on faster and smoother artificial surfaces, together with rule changes (offsides and corner hits) and equipment advances, however, may have contributed to an increase in the tempo of the game, which some experts speculate may actually lead to higher injury rates.[3,9] Another contributing factor may be that with the faster play and technology advances in equipment, the ball is being elevated off the field more now than 10 to 12 years ago. In contrast, the elimination of offsides may have resulted in a more wide-open field, effectively reducing field congestion[10] and possibly reducing injury rates. Although the overall game injury rate declined after these rules were introduced, possibly fueled by drops in the rates of ankle (ligament sprain), knee (internal derangement), and finger (fracture) injuries, the rates of head injuries (concussions and lacerations) did increase over time and may be related to the increased pace of the game, resulting from rule and equipment technology changes (Table 9).

Overall, and across all 3 divisions, preseason practice injury rates were approximately 3 times higher than practice injury rates during the regular and postseason time periods. This may reflect a true difference in injury rates, possibly related to lack of appropriate conditioning and fitness at the beginning of the season for some players. Even though many players may participate in running and weight lifting in the summer prior to preseason practice, because of the specificity of the sport of field hockey (bent-over posture, sprinting, cutting, etc), this conditioning may not adequately prepare them for the intensity and quantity of preseason practice activities.

Although researchers have expressed concern about the relatively high frequency of ocular and dental injuries in the sport,[3,11,12] these specific injuries do not seem to be overrepresented in the NCAA athlete population. However, the total number and rate of injuries occurring to the head and face, including concussions, are of concern. Not only did concussions account for more than 5% of severe game injuries (10+ days of time loss), the risk of sustaining a concussion in a game was 6 times higher than in practice (rate ratio = 6.1, 95% CI = 4.3, 8.7). Covassin et al[13] also reported a similar disparity in concussion rates between field hockey games and practices from 3 years of NCAA data. This disparity may be related to the intensity of game play compared with practice play: specifically, greater frequency of contact among players during games. Field congestion, within 25 yd of the goal, has been implicated as a potential contributor to the frequent head and face injuries.[3] Although NCAA data on field location of injuries have only been reliably available since 1996–1997, these data support this suggestion: 87% of all injuries and 69% of head and face injuries occurred while the player was in the goal area/circle or within the 25-yd line. Yard and Comstock[12] also highlighted the preponderance of facial injuries in field hockey; 26% of injuries were to the head and/or face in their study of sport-related injuries treated in US emergency departments.

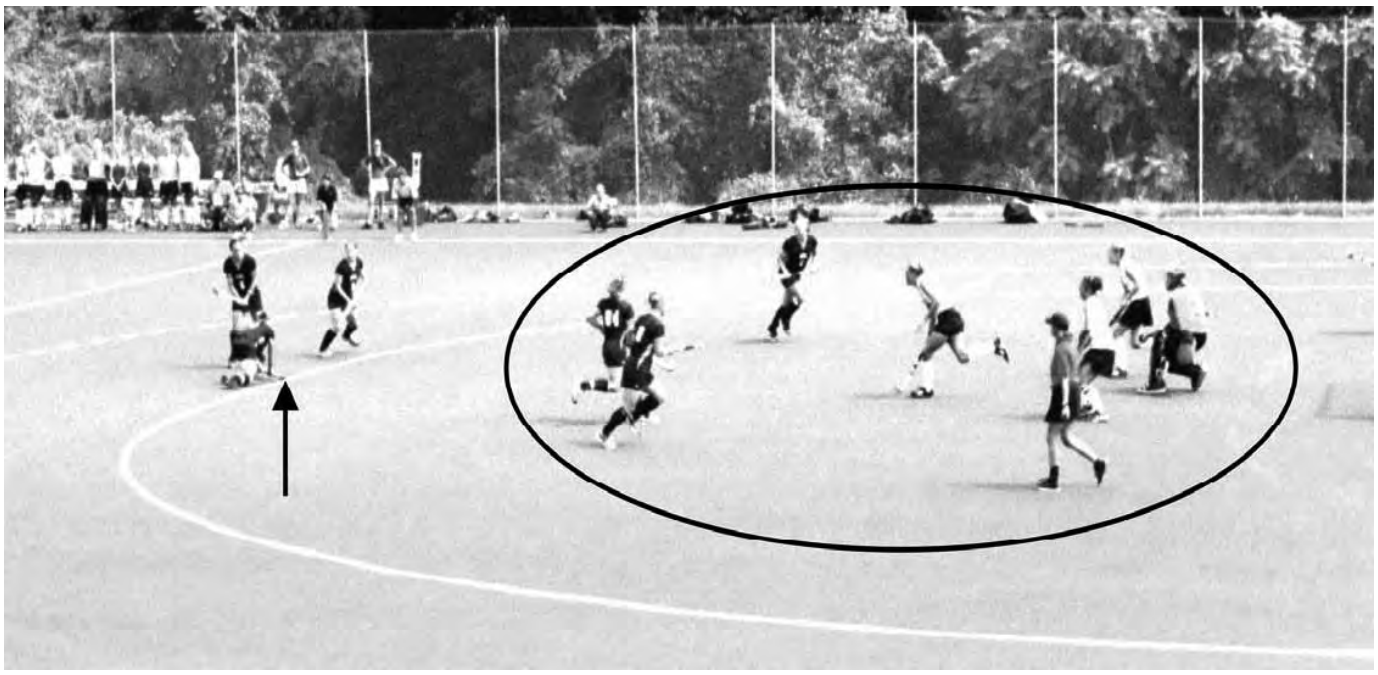

Figure 6. A typical corner shot during field hockey play showing the field congestion confined to a small part of the field in front of the goal and within the 25-yd line (circle) and a player trapping the ball with her stick for a shot-on-goal attempt (arrow). Field congestion and the practice of trapping the ball have been speculatively associated with an increased risk of injuries, particularly to the head/face and hand/finger.

Because of the potentially serious long-term or permanent effects (eg, reduced vision or blindness, loss of permanent teeth, disfigurement from facial lacerations, memory deficits), identifying potential prevention strategies for head and facial injuries is an important goal. Currently, the only above-the-neck equipment required for nongoalie collegiate field hockey players is a mouth guard. Some experts have suggested helmets become standard equipment for field hockey, whereas others argue this would change the nature of the game, possibly increasing player-to-player contact, speed, and intensity, which could ultimately increase the injury rate.[3] As of 2005, women's lacrosse has mandated protective goggles in an attempt to reduce the relatively high rate of eye injuries in the sport (http://www.uslacrosse.org/news/2004/eyewearupdate.phtml). Because these data indicate that the entire head and face are at risk for injury in field hockey, rather than just the eyes, a helmet with a full face mask, as opposed to goggles only, may have the potential to effectively reduce these injuries. However, helmets cannot completely protect against concussion, an injury on the rise in field hockey players. It is unclear if the increase in concussions is a true increase or if it is related to better diagnosis and advances in head injury assessment. Resistance to mandated head and facial protective equipment within the sport is likely to be considerable,[14] and the benefits of injury reduction will need to be weighed against the potential negative effects (eg, increased ''roughness'' and reduced vision) before a rule change will be feasible. In February 2007, the NCAA Field Hockey Committee recommended allowing (but not requiring) appropriately tested protective eyewear for all field players starting in the 2007–2008 season.

It is still difficult to tease out the possible effects of the offsides and corner-hit rule and equipment changes on injury rates. The fact that most above-the-neck game injuries occurred near the goal or within the 25-yd line (69.3%) and were caused by contact with the ball or stick (77.4%) indicates that field congestion may still be a factor related to these types of injuries. Another issue is that 33.6% of these same above-the-neck game injuries occurred during a play in which a penalty was called. Unfortunately, information in the NCAA ISS is insufficient to determine what types of penalties were called at the time an injury occurred. This information is essential for determining the need for possible rule changes. Future authors should focus on identifying the specific types and instances of injuries that may be preventable through rule and equipment changes.

In this report, finger/thumb fractures alone accounted for almost 15% of severe game injuries, and most of these were caused by contact with the ball or stick. These hand and finger injuries are important in that they may be underestimated in terms of severity and long-lasting sequelae. Fractures and tendon injuries of the hand resulting from any trauma (not just sports injuries) can result in significant long-term and permanent disability and have been associated with permanent loss of motion and function, osteoarthritis, work sick leave, lost productivity and disability, reduced general health status, and high societal costs.[15,16] Thus, it is important to understand the relatively high rates of hand and finger injuries in the sport of field hockey, particularly in terms of preventability.

Field hockey players may be more prone to hand injuries than players in some other sports because of selected inherent aspects of the game. In field hockey, the bent-over posture is used for dribbling and shooting the ball. This posture may place the player's hands (the head and face, too) closer to the ground, which may make it easier to sustain trauma from other players' sticks or cleats or a ball during normal play. In areas of congestion, such as in front of and near the goal, players may easily have their hands crushed between 2 sticks, a player and a stick, or a player and the ground (Figure 6). Before 2003, game rules required that during a penalty corner, the

ball come to a complete stop outside the circle before a shot on goal,[8,9] which led players to use the stick to ''trap'' the ball against the ground to stop it. This technique may still be used in other parts of the game and puts the hands and fingers at considerable potential for injury. Large, full-coverage gloves, such as those worn by ice hockey and men's lacrosse players, are not feasible in field hockey because they may negatively affect the grip and stick-handling ability. However, close-fitting, posteriorly padded gloves are an option that could potentially prevent some finger and hand injuries in field hockey. Except for goalies, no equipment is mandated to protect the hands in field hockey, although wearing gloves is allowed. To date, the effectiveness of wearing padded gloves to reduce hand and finger injuries is not known. Interestingly, no hand/finger injuries were recorded by goalies for the years when position information is available, indicating that they may be protected from severe hand injuries by wearing gloves. Given the high rates of severe hand injuries reported here, future consideration of mandating hand protection for all positions in field hockey may be warranted.

Ankle ligament sprains accounted for 14% of game injuries and 15% of practice injuries and also were the third most-common injury for both practices and games resulting in 10+ days of activity time loss (Tables 5 and 6). Neuromuscular and balance training programs and prophylactic ankle taping or bracing are both effective in reducing the risk of ankle injury, by as much as 50%, particularly for athletes with a history of a prior ankle sprain.[17,18] Despite the known effectiveness of these interventions, how completely they are implemented in collegiate athletes is unknown. Identifying athletes at high risk for repeated ankle injuries through preseason injury history surveys, orthopaedic screening, and functional field testing for neuromuscular deficits and implementing a comprehensive ankle injury prevention program should be the standard of care for any athletic population participating in running, jumping, and twisting sports. At the minimum, athletes with a history of ankle sprains should be offered prophylactic taping or bracing and appropriate conditioning programs that incorporate balance training exercises. Prophylactic ankle taping or bracing may be most important for game situations, because these data indicate that the risk of ankle injury in games was twice as high as during practices.

Field hockey slowly continues to grow in popularity in the United States, both at the high school and collegiate levels. Although some risk of injury is inherent with sport participation, an important goal is to make sports as safe and exciting as possible through injury prevention. The NCAA ISS is an excellent foundation from which to develop and test evidence-based injury prevention strategies. These data indicate possible areas for future injury prevention initiatives in field hockey, including potential rule changes (eg, to prevent field congestion), equipment modifications (eg, mandating helmets and padded gloves), and additional conditioning programs (eg, neuromuscular balance training).

DISCLAIMERS

The findings and conclusions in this article are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention.

The conclusions in the Commentary section of this article are those of the Commentary authors and do not necessarily represent the views of the National Collegiate Athletic Association.

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Randall Dick, MS, FACSM; Jennifer M. Hootman, PhD, ATC, FACSM; and Julie Agel, MA, ATC, contributed to conception and design; analysis and interpretation of the data; and drafting, critical revision, and final approval of the article. Luzita Vela, PhD, ATC, contributed to analysis and interpretation of the data and drafting, critical revision, and final approval of the article. Stephen W. Marshall, PhD, contributed to conception and design; analysis and interpretation of the data; and drafting, critical revision, and final approval of the article. Renee Messina, MS, ATC, contributed to analysis and interpretation of the data and drafting, critical revision, and final approval of the article.

Address correspondence to Jennifer M. Hootman, PhD, ATC, FACSM, Division of Adult and Community Health, Centers for Disease Control and Prevention, 4770 Buford Highway NE, MSK-51, Atlanta, GA 30341. Address e-mail to jhootman@cdc.gov.

1. Please select your NCAA Division.

	Response Percent	Response Count
Division I	33.3%	36
Division II	8.3%	9
Division III	**58.3%**	63
	answered question	**108**
	skipped question	**2**

2. Did you know that: Field players are permitted to wear throughout a game only a smooth preferably transparent or white but otherwise dark plain colored face mask which fits flush with the face, soft protective head covering or eye protection in the form of plastic goggles (i.e., goggles with a soft-covered frame and plastic lenses. A caged frame cannot be worn).

	Response Percent	Response Count
Yes	**66.1%**	72
No	33.9%	37
	answered question	**109**
	skipped question	**1**

3. Did any of your field players wear protective facemasks or eyewear at any point in the season?

	Response Percent	Response Count
Yes	40.4%	44
No	**59.6%**	65
	answered question	**109**
	skipped question	**1**

4. What positions wore protective eyewear/facemask?

	Response Percent	Response Count
Forward (Attack)	**36.5%**	19
Midfield	32.7%	17
Defense	**36.5%**	19
Other	23.1%	12
	answered question	**52**
	skipped question	**58**

5. Did the field players prefer wearing:

	Response Percent	Response Count
Protective eyewear	6.2%	5
Protective facemasks	17.3%	14
Both	3.7%	3
Neither	**72.8%**	59
	answered question	**81**
	skipped question	**29**

6. Why did players wear protective eyewear?

	Response Percent	Response Count
prevention	6.9%	6
following an injury	19.5%	17
N/A	**73.6%**	64
	Other (please specify)	6
	answered question	**87**
	skipped question	**23**

7. How long would a student-athlete wear the protective eyewear?

	Response Percent	Response Count
a week	7.6%	6
a month	3.8%	3
remainder of the season	8.9%	7
only during practice	1.3%	1
N/A	**79.7%**	63
Other (please specify)		11
	answered question	**79**
	skipped question	**31**

8. Why did players wear protective facemasks?

	Response Percent	Response Count
prevention	10.1%	9
following an injury	30.3%	27
N/A	**62.9%**	56
Other (please specify)		6
	answered question	**89**
	skipped question	**21**

9. How long would a student-athlete wear the protective facemask?

	Response Percent	Response Count
a week	5.0%	4
a month	10.0%	8
remainder of the season	15.0%	12
only during practice	1.3%	1
N/A	**71.3%**	57
Other (please specify)		13
answered question		**80**
skipped question		**30**

10. What type of eyewear/facemask are your student-athletes wearing?

	Response Percent	Response Count
Polycarbonate	20.7%	19
Wire Cage	4.3%	4
Custom made	9.8%	9
ASTM Certified	5.4%	5
Do not know	5.4%	5
None	**58.7%**	54
Other (please specify)		8
answered question		**92**
skipped question		**18**

11. Other Comments.		Response Count
		25
	answered question	**25**
	skipped question	**85**

Protective Eyewear Mandated for Field Hockey

FOR IMMEDIATE RELEASE

Contact: Elliot Hopkins

INDIANAPOLIS, IN (April 15, 2011) — High school field hockey players will be required to wear protective eyewear, effective with the 2011-12 season.

The National Federation of State High School Associations (NFHS) Board of Directors, in its April 13 meeting in Indianapolis, voted to mandate the use of protective eyewear that meets the current American Society for Testing and Materials (ASTM) standard for field hockey.

Acting on a recommendation from the NFHS Sports Medicine Advisory Committee, the Board agreed that the potential risk of injury warranted the requirement of protective eyewear for the 64,000 student-athletes participating in high school field hockey.

“While serious eye injuries in field hockey are rare, the NFHS Board of Directors has concluded that an eyewear requirement is the right step,” said Elliot Hopkins, NFHS director of educational services and field hockey rules editor.

NFHS field hockey rules previously allowed – but did not require – the wearing of eyewear that meets the current ASTM standard. Six NFHS-member state associations had previously passed more stringent rules and already mandate the use of protective eyewear.

The revised language to mandate protective eyewear will appear in the 2011-12 NFHS Field Hockey Rules Book.

The Mechanisms and Prevention of Sports Eye Injuries

The ideal scenario for a sports (or any other) eye injury is for it never to have happened. Prevention, effective in terms of both injury reduction and cost savings to society, [1-6] should be part of the core curriculum of anyone who prescribes, manufactures, or dispenses eyewear, as well as those in the capacity of formulating and implementing rules in the athletic environment. [7] According to the Centers for Disease Control and Prevention, "Injury is probably the most unrecognized major health problem facing the nation today, and the study of injury presents unparalleled opportunities for reducing morbidity and mortality and for realizing significant savings in both financial and human terms—all in return for a relatively modest investment." [8] By following guidelines for specific sports, eye care professionals, sports officials, and participants will significantly reduce the risk of eye injuries without changing the essential nature or appeal of sports.

Epidemiology

Without some knowledge of the incidence and severity of sports-related eye injuries, it would be difficult to attempt injury reduction since there would be no way to determine whether preventive methods were indicated or if they had an adverse or beneficial effect. A major potential pitfall in studying epidemiologic data is that the data obtained may reflect only the risk of a sport and not the benefits that may or may not justify the risk. [9] The objective is to reduce preventable eye injuries to the minimum consistent with retaining the benefits—the fun and appeal that draw participants into the sport. It is possible to achieve this goal most of the time after the incidence and mechanism of eye injuries are ascertained and a committee representing all concerned with the sport—athletes, coaches, officials, and the medical profession—meets to solve the problem.

In 1991, the National Institutes of Health Conference on Surveillance Strategies for Sports Injuries in Youth recommended the creation of a universal data base, [10] yet a decade later there still is no system that records all sports eye injuries with a numerator (injuries, injury details, and use of protective equipment) and a denominator (participants, exposures, player skill) from which the injury risk to both the individual and society can be calculated. In the absence of such a system, the incidence of sports eye injuries and the effect of injury prevention programs must be approached either from studies that emphasize the risk to society and attempt to measure the total number and severity of injuries in a given population or from the study of a small controlled population from which risks to the individual can be estimated.

It is essential to realize that injuries are not accidents. Instead, they have definite patterns and distinct nonrandom characteristics. [11,12] By carefully evaluating the underlying mechanisms, patterns, and rates of injury in a given sport, it is possible to design and implement extremely effective preventive programs.

Risk to society

Although incomplete, the data show that eye trauma is a major public health problem, [13-15] of which sports comprise a significant proportion. [16] Sports and recreation accounted for 10% of all hospital-treated eye injuries in Dane County, Wisconsin, in 1979 [17] and 65% of all eye injuries to children in Israel from 1981 to 1983. [18] Sports-related injuries were responsible for 60% of hyphemas and 10% of open globe injuries in 3184 patients seen in the Massachusetts Eye and Ear Emergency Room over a 6-month period. [19] Approximately one fourth of all trauma admissions to the Manchester Royal Eye Hospital in 1987 and one sixth of all trauma admissions to the Wills Eye Hospital over a three year period were secondary to sports-related injuries. [20-22] Sports-related injuries (BB gun [2], [23] golf [1], basketball [1]) resulted in four enucleations in Olmstead County, Minnesota, between 1956 and 1988. [24] BB and other sports injuries are common in children. [22,25] In 11 to 15-year-old children, sports and recreational activities accounted for 27% of all eye injuries. [26] Injuries result in visual acuity of less than 20/200 secondary to the development of amblyopia in the injured eye in over 40% of children injured before the age of 10. [27] The vast majority of injured players were not wearing any form of protection at the time of injury. [4,28,29]

Regional injury data often reflect the local popularity of a sport and do not necessarily reflect the risk to an individual participant. Playing with bow and arrow and gilli-danda were the leading cause of sport injuries (47.2%) in northwest India, [30] but neither of these activities appear in the data from the United States and Canada. From a societal perspective, the focus of prevention must vary from one location to another. However, basic mechanisms of injury are often similar for different sports.

Data Sources

There are several sources of injury data, some more useful than others:

The *National Safety Council* system and state data-collecting systems have been of little value in the study of sports-related eye injuries because their data are difficult to obtain and are often inconsistent. Gathering of statewide data [31] from hospital records is often impeded by the method of hospital record keeping, which often fails to identify the cause of injury or the circumstances surrounding the injury.

The *National Athletic Injury/Illness Reporting System (NAIRS)* has, in the past, obtained useful data by following injury rates in participating schools. [32] However, the data are no longer available.

In 1985, *the Centers for Disease Control (CDC)* consolidated its nonoccupational injury research efforts into the Division of Injury, Epidemiology, and Control. The reports on eye injuries thus far have not been detailed enough for use in monitoring sports eye injuries.

The *National Electronic Injury Surveillance System (NEISS*) was created under a 1973 congressional mandate that established the US Consumer Product Safety Commission (CPSC) to protect the public from unreasonable risks of injury and death associated with consumer products. [33] NEISS is the core of CPSC's Bureau of Epidemiology, and currently comprises 101 hospital emergency departments that make up a stratified sample of all hospital emergency departments throughout the United States and its territories. NEISS data—categorized by body part, product, and activity—are good for estimating the total social cost of injuries that affect large segments of the population. NEISS is limited because only emergency department visits related to injuries caused by products are recorded as the basis for projections of a national probability. Since specialty eye hospitals and private ophthalmologists offices, where most of the sports-related eye injuries

Table 1 Sports and Recreational Eye Injuries, Calendar year 1998 NEISS Estimates by Age, Group, and Percentage of Total

	All Age Groups		Under 5 Years Old		5-14 Years Old		15-24 Years Old		25-64 Years Old		65 Years & Over	
	Estimate	***Percent***	***Estimate***	***Percent***	***Estimate***	***Percent***	***Estimate***	***Percent***	***Estimate***	***Percent***	***Estimate***	***Percent***
Basketball	8723	22.2%	148	0.4%	2,338	5.9%	3,856	9.8%	2,381	6.1%	0	0.0%
Water and Pool Sports	4,593	11.7%	133	0.3%	1,782	4.5%	699	1.8%	1,817	4.6%	162	0.4%
Baseball	4,029	10.3%	182	0.5%	2,195	5.6%	823	2.1%	829	2.1%	0	0.0%
Racket Sports	2,767	7.0%		0.0%	1,000	2.5%	926	2.4%	822	2.1%	19	0.0%
Hockey	1,614	4.1%		0.0%	515	1.3%	628	1.6%	471	1.2%	0	0.0%
Football	1,464	3.7%		0.0%	533	1.4%	583	1.5%	348	0.9%	0	0.0%
Soccer	1,325	3.4%		0.0%	741	1.9%	378	1.0%	206	0.5%	0	0.0%
Ball Sports	1,270	3.2%	115	0.3%	581	1.5%	375	1.0%	160	0.4%	39	0.1%
Golf	828	2.1%	7	0.0%	142	0.4%	75	0.2%	604	1.5%	0	0.0%
Combatives	448	1.1%		0.0%	56	0.1%	82	0.2%	310	0.8%	0	0.0%
Total Selected Sports	27,061	68.9%	585	1.5%	9,883	25.1%	8,425	21.4%	7,948	20.2%	220	0.6%
Other Sorts	12,236	31.1%	596	1.5%	4,273	10.9%	2,932	7.5%	4,190	10.7%	245	0.6%
Totals	39,297	100.0%	1,181	3.0%	14,156	36.0%	11,357	28.9%	12,138	30.9%	465	1.2%

Racket sports include racquetball. tennis, squash, paddleball, badminton, and handball.
Hockey includes ice, field, street, and roller hockey.
Ball sports include other and unspecified ball sports.
Combatives include boxing, martial arts, and wrestling.
Compiled by Prevent Blindness America from statistics provided by CPSC from the National Electronic Injury Surveillance system (NEISS).

are seen, are not included in the sample, NEISS data must be viewed with caution. For example, the extreme eye injury hazard of boxing is not apparent from NEISS data. Yet national trends (e.g. the large number of basketball and baseball eye injuries) are often apparent from these data.***Table 1, Appendix 1***

The *National Eye Trauma System (NETS)* is a consortium of approximately 50 regional eye trauma centers that prospectively gathers information on the etiology, treatment, and final results of open-globe eye injuries. However, most sports-related eye injuries are caused by blunt objects and do not penetrate, perforate, or rupture the globe, and thus are not recorded. Despite the fact that the consortium misses most sports-related injuries, it is astounding that 14.1% of all injuries in the NETS database are from sports. As expected, injuries caused by projectiles (38.1 % of reported recreational injuries were due to BB/air guns) lead the NETS list of perforating injuries due to sports.[2, 34]

The *United States Eye Injury Registry (USEIR)* was formed in December 1988, modeled on the Eye Injury Registry of Alabama, which began in 1982. USEIR, now a federation of 40 state registries and the United States Military Eye Injury Registry, collects and disseminates comprehensive data on the occurrence of serious (involving permanent or significant structural or functional changes to the eye) ocular injuries. USEIR provides data on a broad spectrum of eye injuries, including blunt trauma and chemical injuries that are frequently seen only in ophthalmologists' offices. Because of under reporting by ophthalmologists, USEIR captures only about 0.3% of sports and recreational eye injuries (about 400,000 in NEISS and 1,300 in USEIR over 10 years). [35] ***Table 2*** is a summary of USEIR sports eye injury data.

National data collected by the cooperating ophthalmologists of the *Canadian Ophthalmological Society (COS)* ***Appendix 2*** [4] provides a useful national database available for following trends in sports-related injury and the results of intervention with rules changes or protective devices. The COS, which freely shares its data, has maintained the longest-term prospective database of sports-related eye injuries and has played a vital role in the prevention of these injuries. Since, like USEIR, the COS system depends on the voluntary reporting of cases by individual ophthalmologists, the reported cases are an indeterminate small percentage of the actual injuries.

SGMA International compiles the most reliable estimates of sports participation in the United States. [36] Data for specific sports are included in the discussions of individual sports, to follow. SGMA International details participation trends in 103 fitness, sports, outdoor, and recreational activities, based on a nationally representative sample of 14,276 adults and children. Sports participation falls into approximately three fairly equal groups: 86.1 million participate frequently; 83.6 million participate occasionally; 81.3 million do not participate. Combining these data with the data of NEISS gives a somewhat better perspective. The fact that soccer participants increased from 2.3 million in 1990 to 4.3 million in 2001 suggests that the increase in total soccer eye injuries ***Appendix 1*** may be due to an increase in players at risk rather than a change in incidence.

Risk to the individual

More important to the athlete than the injury statistics noted above is the risk of a specific sport to an individual participant. Despite the lack of an ideal data collecting system, it is possible to ascertain the eye injury potential of various specific sports and follow the results of intervention, with rules changes or protective equipment, by means of limited, specifically designed studies. [37]

The *National Collegiate Athletic Association (NCAA) Injury Surveillance System (ISS)* was developed in 1982 to provide current and reliable data on injury trends in collegiate sports. At

Table 2. USEIR Sports and Recreation Eye Injuries December 1988 to September 1999

	Total	Open globe	Blind eyes	Shattered eyewear lens	Sex (% male)	Age (median)
Fishing	113	50	34	0/1	82.3%	27
Hunting/Shooting	59	28	29	2/4	98.3%	32
Recreation	55	29	15	1/1	89.1%	17
Baseball	104	11	15	5/8	86.5%	14
Softball	65	3	8	3/12	63.1%	29
Basketball	66	11	12	1/1	95.5%	24
Racket Sports	55	1	8	0/6	83.3%	25
Hockey	13	2	0	0/0	100%	30
Paintball	12	1	1	0/3	100%	17
Golf	29	12	12	4/6	72.4%	40
Soccer	24	0	1	0/1	66.7%	17
Ball Sports, Other	19	2	0	0/1	68.4%	12
Football	13	0	0	0/1	100%	15
Water sports	15	3	5	0/0	66.7%	21
Sports, Other	22	4	1	0/0	80.1%	22
Motor Sports	11	5	5	0/0	81.9%	22
Fireworks	21	3	4	0/1	80.1%	20.5
Bungee Cord	6	2	1	0/0	66.7%	21
Total	702	167	151	16/46	83.3%	Range 22.5-83

Blind Eyes include: Final best corrected visions: no light perception, hand motions, finger counting, less than 20/200

Sex: In three cases the sex was not stated. Calculations based on 699 cases in which sex was specified

Age: Injuries affected all age groups, with a range of 2 to 85 years. The age median cited is only to point to an age trend by sports group.

Motor Sports include: 3 wheeler (1), 4 wheeler (2), auto racing (7), go cart (1)

Hunting/Shooting include: air rifle (4), BB gun (14), hunting (31), shooting (trap, skeet) (10)

Racket Sports include: badminton (3), handball (2), racquetball (36), tennis (14)

Ball Sports, Other include: play with ball (5), dodgeball (1), kickball (1), lacrosse (1), mudball (1), stickball (1), volleyball (6), whiffle ball (3)

Water Sports include: beach (1), boating (5), sailboat (1), surfing (3), swimming (1), water skiing (2)

Recreation includes: bicycle (1), camping (6), carnival (1), church function (1), darts (2), dirt bike (1), dog bite (1), fight (2), glass car window (1), hiking (5), home (1), motor vehicke crash (1), park (1), play (11), poker (1), recreation (11), sledding (1), sling shot (1), snowmobile (1), vacation house (1), water balloon (2), water hose (1), whirly gig toy (1)

Golf includes: golf (28), putt-putt golf (1)

Sports, Other include: bow and arrow (1), boxing (4), frisbee (2), ice skating (1), karate (2), rodeo (3), roller skating (1), running (1), ice skating (1), skiing (3), wrestling (3)

first, only football data were collected, but ISS has expanded to also include wrestling (men's), basketball, soccer, lacrosse, gymnastics (men's and women's), field hockey, volleyball, and softball (women's). Participation is limited to the 977 NCAA member institutions with ISS participants picked at random to have a minimum 10% representation of each NCAA division (I, II, and III). Data from the NCAA do not record every injury, but are a sampling that is representative of the total population of NCAA institutions sponsoring a particular sport. ISS gives the eye injury rate per 1,000 exposures, [38] but it is difficult for many to judge risk unless the NCAA data are put into more understandable terms. The NCAA incidence figures can be multiplied by the average exposure (number of games and practices) to give an easily understood risk to the individual per season and per school career—8 years high school and college. ***Table 3***

It has been more than 20 years since the *National Society to Prevent Blindnes*—now called *Prevent Blindness America*—made recommendations that sports-related eye injury data gathering fulfill the following criteria: (1) to permit population-based comparisons involving a known denominator; (2) to record demographic data and details of the injury at the time of presentation to the medical facility; (3) to record the diagnosis of the physician at the time of examination; and (4) to record the final outcome of the injury. [16] As can be seen from the data gathering systems presented, data collection has a long way to go to realize these recommendations.

The analysis of input from many reporting sectors is needed to comprehend the magnitude of sports injuries, the need for protective programs, and the effectiveness of implemented programs. From the preceding and the data to follow, it is possible to approximate the eye injury risk to the unprotected participant from selected sports. ***Table 4***

Economics of Sports Eye Injuries

The social cost of eye trauma, the most common ophthalmic indication for hospitalization, is enormous. National projections estimate annual US hospital charges of $175 million to $200 million for 227,000 eye trauma hospital days. [39] Eye injuries seen in 6 months in one emergency department are responsible for direct and indirect costs totaling $5 million and a loss of 60 work-years. [19] The average societal cost for an eye injury to a child under the age of 15 playing basketball is $3,996. [40] It is estimated that of the 1.6 to 2.4 million Americans who sustain eye injuries each year, 40,000 will be legally blinded in the injured eye. About one third of these injuries result from sports. [41] Since essentially all sports-related eye injuries are preventable, the potential economic savings resulting from the prevention of these injuries is great. There is no question that prevention of traumatic sports-related eye injuries is cost-effective. [42] In 1980 dollars, the hockey face protector saves society $10 million a year by preventing approximately 70,000 eye and face injuries in 1.2 million protected players. [3]

Total US expenditures for preventive health care amounted to under 2.5% of total health care expenditures, with less than 0.5% spent on health education. [43] Despite the fact that injuries are one of the leading causes of physician and hospital visits, the amount allotted by Congress and the National Institutes of Health (NIH) for trauma research is less than 1 % of the amount allotted for cancer and heart disease. [11]

Mechanisms of sports eye injuries

The analysis of trauma is commonly expressed in International System of Units (SI), which are the worldwide standard. [44] Since it is hard for many of

Table 3 Eye Injury Risk, NCAA

	Annual Risk		8-Year Risk	
	Men	Women	Men	Women
Wrestling	1.67%		12.58%	
Basketball, men's	0.97%		7.52%	
Lacrosse, women's		0.88%		6.79%
Field hockey		0.50%		3.97%
Basketball, women's		0.50%		3.90%
Softball		0.40%		3.17%
Soccer	0.26%	0.24%	2.06%	1.94%
Baseball	0.20%		1.59%	
Volleyball		0.12%		0.99%
Football	0.11%		0.87%	
Ice hockey, men's	0.08%		0.63%	
Lacrosse, men's	0.06%		0.45%	
Ice hockey, women's		0.00%		0.00%
Gymnastics	0.00%	0.00%	0.00%	0.00%

Open-globe injuries: softball (4); football (4); baseball (2); men's basketball (1)
Mean of 5 years (1997-2002) except for women's ice hockey (2 years: 2000-2002)
Based on NCAA data, probability calculation advice courtesy of Randy Dick and Preston Fiske.

Table 4 Sports Eye Injury Risk to the Unprotected Player

High Risk:
Small, Fast Projectiles:
Air rifle/BB gun
Paintball
Hard Projectiles, "Sticks," Close Contact
Basketball
Baseball/Softball/Cricket
Lacrosse, Men's and Women's
Field Hockey
Ice Hockey
Street Hockey
Squash/Racquetball
Fencing
Wrestling
Intentional injury
Boxing
Full-Contact Martial Arts
Moderate Risk:
Tennis/Badminton
Soccer/Volleyball
Water Polo
Football
Fishing
Golf
Cycling
Low Risk:
Swimming/Diving/Water Skiing
Skiing
Non-contact Martial Arts
Eye safe:
Track and Field*
Gymnastics

*Javelin and discus have a small potential for injury that is preventable with good field supervision.

us to visualize what some SI units actually measure (Is it very dangerous to collide with a football player who weighs 200N at 5m/s?) more understandable units, such as miles per hour (mph) will be used when appropriate. It is easier for most of us to grasp the speed of a baseball when the velocity is expressed as 75mph rather than as 33.53m/s.

The severity of an eye injury is correlated with the total impact force, expressed in newtons (N), and the impact force onset rate, expressed in newtons-per-millisecond (N/ms), and the kinetic energy, expressed in Joules (j) of an impacting object. There is an eye-injury progression from chamber angle damage to peripheral vitreoretinal damage to ruptured globe as the force increases and the time to maximum force decreases. [45] If we slow the velocity of a BB (0.345 g) to 29 mph (13 m/s; 43 ft/s), the energy will be beneath the kinetic energy of 0.03 j required for an ocular contusion and there will be no eye injury. However, when fired in the horizontal direction from a height of five feet, the BB would travel only 24 feet. [46] As the BB velocity increases, the injuries get more severe: 62.3 m/s (205 ft/s) will result in injury at the vitreous base and retinal tear; 72.0 m/s (236 ft/s) penetrates the globe; 124 m/s (408 ft/s) penetrates skin, bone, and moderate tissue. [46,47]

Test devices [48] and mathematical models [45] have been devised for the laboratory testing of various products, such as toys, to assess the potential for eye injury. The force onset rate needed to produce clinically detectable contusion injury by a blunt object is approximately 750 N/ms. Some toy dart guns (896 N/ms) that propel suction cups exceed this level, while most toy ping-pong ball shooters (428 N/msec) do not. [49] Computer modeling using finite element analysis has led to better analysis and understanding of the mechanisms of eye injury ***Figure 1***. [50-53]

Many protectors (such as football helmets) for sports and some protective eyewear [54] prevent or reduce injury by decreasing the force onset rate and the peak force by spreading the total force over time. However, the injury as related to force onset rate only applies total forces in a limited range, which has not yet been determined.

Injury classification

Sports eye injuries can be classified in accordance with the Birmingham Eye Trauma Terminology (BETT) in which all terms relate to the whole eyeball as the tissue of reference. [55] Injuries may be:

1. Open-globe injury in which there is a full-thickness wound of the eyewall:

a. Rupture. Wound caused by a blunt object, eye ruptures at weakest point ("inside out" mechanism).

b. Laceration. Wound caused by a sharp, or small-high-velocity, object at site of impact ("outside in" mechanism):

1. Penetrating. Each entrance wound caused by separate agent, includes intraocular foreign body.

2. Perforating. Entrance and exit wound caused by same agent.

2. Closed-globe injury in which there is no full-thickness wound of eyewall:

a. Contusion

1. Due to *direct energy* delivery

Table 5 Globe Rupture: Correlation Among: Intraocular Pressure, Object Hardness, Size, and Kinetic Energy *

		Human Eye	Monkey Eye	Pig eye
Elevated Pressure [196]	Intraocular Saline	2,800 to 6,400 mmHg 54.1 to 123.8 psi		
Metal Rod [197]	12.5 mm diameter 303 g		12.2 ft/s (8.3 mph) 2.1 J	
Paintball [198]	17.5-mm diameter 3.55 g			290 ft/s (198 mph) 13.9 J
Golf Ball [199,200]	43.0-mm diameter 45.4 g			86 ft/s (59 mph) 15.6 J
Squash Ball [199,200]	41.0-mm diameter 24.7 g			150 ft/s (102 mph) 25.8 J
Baseball [69]	73.2mm diameter 143.9 g	80.7 ft/s (55 mph) 43.5 J		

* Approximate averages. Rupture of individual eyes varies.

by the object.

2. Due to *changes in the shape* of the globe.

b. Lamellar laceration Partial-thickness wound of the eyewall.

Open-globe injuries.

The eyewall (the cornea and the sclera) can be modeled as the union of two thin-walled (approximate 0.5mm wall thickness) spheres (radii 8mm and 12mm) with the centers 5mm apart. [56] At the junction of the optic nerve sheath, which may be represented by a cylinder (radius 2mm), the larger (scleral) sphere is perforated by multiple openings, the lamina cribrosa. [57] However, the eyewall is not of uniform thickness, [58] is significantly thinner in myopic eyes, [59] and becomes less elastic with age. [60] The eyewall tends to rupture in three specific locations: (1) where the radius of curvature changes at the limbus, (2) where the sclera is the thinnest, near the equator behind the ocular muscles, and (3) where the sclera is perforated at the lamina cribrosa. [61]

Even with the best surgical techniques, only approximately 50% of children with open-globe injuries recover good visual acuity. [62]

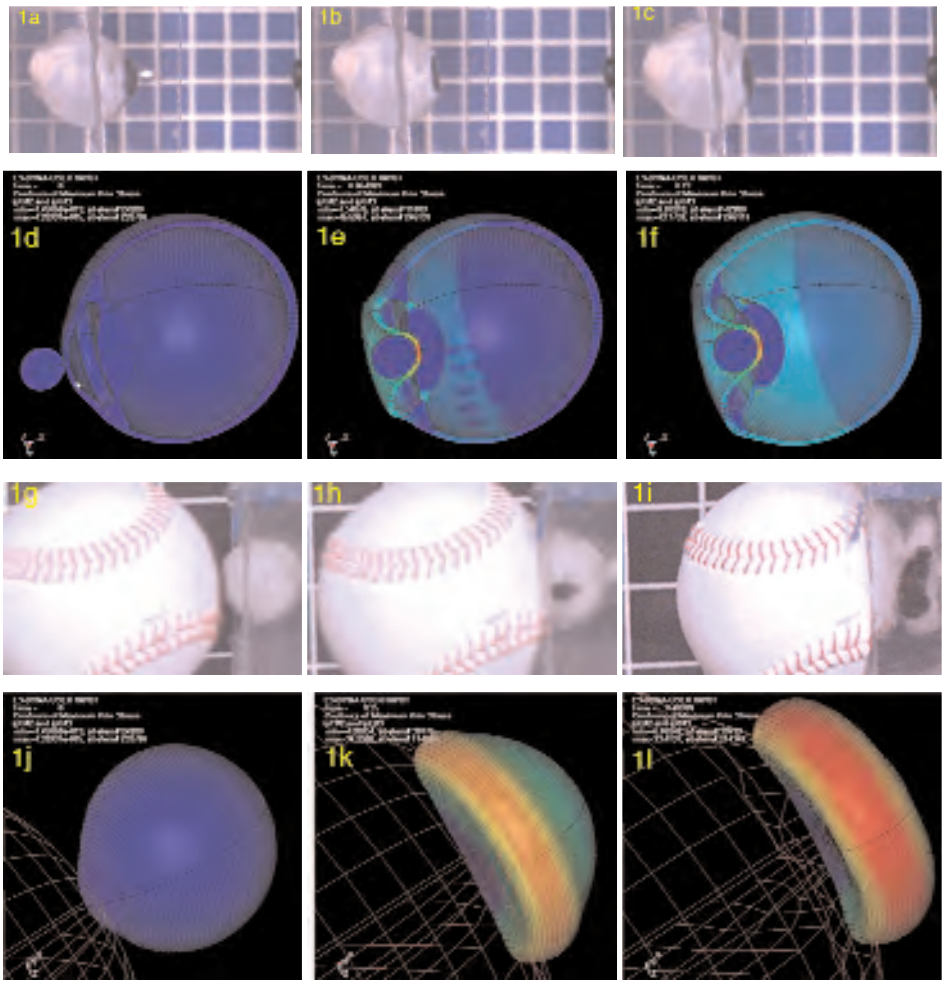


Figure 1 Finite element analysis correlated with motion analysis of impacts on human eyes.

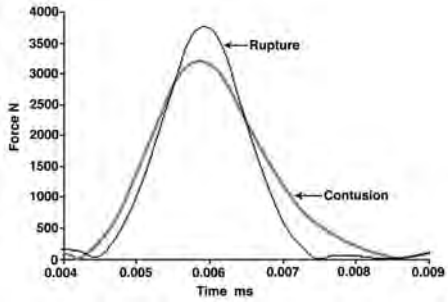
1a-c. BB, moving right to left at 92.0 m/s (301.8 ft/s; 0.58J) impacting a human cadaver eye held in an artificial orbit with clear gelatin. The sequence will be continued in the section on BB's to follow. *1 d-f* finite element analysis (FEA) of the BB impact to a point just before globe penetration. *1g-i.* Baseball, moving left to right at 41.2 m/s (92.2 mph; 124.3J) impacting a human cadaver eye held in an artificial orbit with clear gelatin.Note globe rupture (dark area) at equator of the globe starting in *1h* and enlarging in *1i*. See Figure 5 for the baseball rebound. *1 j-l* finite element analysis (FEA) of the Baseball impact to a point just before globe penetration.

This FEA model has potential usefulness as a simulationtool for ocular injury and it may provide useful information for developing protective measures against sports, industrial and traffic ocular injuries. Courtesy of Stefan Duma and Joel Stitzel. Virginia Tech Impact. Biomechanics Laboratory (duma@vt.edu)..

Rupture

Ragged rupture of the globe secondary to injury by a blunt object significantly reduces the likelihood of recovery of useful vision. [63-67] Only seven of 13 ruptured eyes regained counting fingers or better vision. [68] Rupture of the globe occurs when the intraocular pressure is greatly elevated or when a blunt external force is applied to the eye quickly. The energy required to rupture the eye varies with the dimensions and the properties of the impacting object ***Table 5***. ***Figure 2*** shows a matched pair of cadaver eyes impacted with baseballs that had similar physical properties—except for the ball hardness. Impact with a major league baseball (143.9g) at 55mph ruptures the globe at 3 microseconds. Impact with a softer ball (146.5g) at 74mph does not rupture the globe, despite the fact that the softer ball delivered more energy (80.2 joules) than did the harder ball (43.5 joules). [69] The harder baseball causes extreme flattening of the globe (2ms) immediately followed by rupture at the limbus (3ms). After the ball has totally rebounded from the eye and orbit, the ocular contents continue to be extruded by the retained energy in the globe. When the harder baseball, that ruptures the globe, is compared to the softer baseball, that does not rupture the globe, it is appar-

Figure 2. Rupture and contusion related to time in miliseconds (ms) and force onset rate



ent that the harder ball delivers energy faster, deforms the eye more, and rebounds faster. The softer ball has a lower peak force and slower force onset rate (peak force 3,208 N; onset rate 2,686 N/ms) than the harder ball (peak force 3,768 N; onset rate of 3,486 N/ms). It appears that the slower application of force allows the globe to retract into the orbit and undergo less compression than when the force is applied faster.

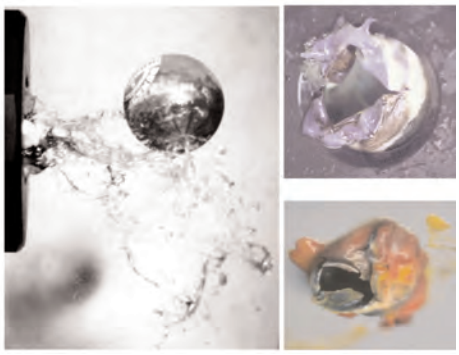


Figure 3. Total extrusion of RK eye contents.
Squash ball impact at 90 mph

The sports that cause ruptured globes to unprotected players typically use a stick with a blade end that fits into the orbit (hockey, field hockey, golf, polo), a small soft fast projectile (BB, paintball), a soft or hard ball that deeply penetrates the orbit (squash, golf), a hard projectile that partially enters the orbit with great force (hockey, baseball, softball, cricket, field hockey, polo), or a one in which there is the potential of contact with a body part that enters the orbit with force (basketball, football, soccer, rugby).

Prior surgery or eye disease. It is apparent that an eye that would have had a closed-globe injury may sustain an open-globe injury (rupture) because surgery has weakened the eyewall. [70] Deeper, longer incisions, especially in the cornea, permanently weaken the eyewall and predispose the eye to an open-globe injury. ***Table 6*** lists the approximate risk from various surgical procedures. All patients who have had surgical procedures that weaken the eyewall should be advised that eye protection is essential when there is the probability of impact. [71] The concept that a ruptured globe is a "safety valve" that prevents contusion injuries in eyes that have had radial keratotomy (RK) cannot be supported by injury data. ***Figure 3***

Laceration

Perforating injuries, in which the same agent causes an entry and an exit wound, are usually due to a high-speed projectile (air rifle/BB, firearm, shrapnel) or a slower sharp projectile (shattered eyewear, fishhook, tip of ski pole, arrow, dart). There are few data on the energy required to cause perforating injuries, which are similar to but more severe than penetrating injuries.

Penetrating injuries. The same agents that cause perforating injuries result in penetrating injuries or intraocular foreign bodies when there is sufficient energy to penetrate the eyewall, but not enough energy to exit the globe. A knife-edged missile is in a class by itself for ease of penetration. The mechanical advantage of the cutting edge is exerted until the hole it makes is the full diameter of the missile. A 20-gauge knife-edged missile penetrates the globe with a momentum of 17 mg*m/s [72] as compared to the momentum of 24,840 mg*m/s as the no-penetration value for the BB. [47]

The energy present in many sports is capable of causing severe eye injury and often exceeds the capability of ordinary eyewear to withstand the impact and protect the eye. Frequently, the lacerating instrument is a fragment of the athlete's own spectacle lens. Thus, the wrong eyeglasses can convert a potential closed-globe blunt trauma injury into an open-globe lacerating injury and permanent visual impairment [73-75] Globe laceration caused by spectacle

Table 6 Predisposition to Traumatic Ruptured Globe After Eye Surgery

High
Penetrating keratoplasty
Large incision, butt joint ICCE, ECCE
Standard RK with incisions to limbus
Hexagonal keratotomy
Moderately high
Large incision tapered joint ICCE, ECCE
Trabeculectomy or other filtration surgery
Prior repair of corneal and/or scleral laceration
Moderate
Small incision butt joint ECCE
"mini" RK
astigmatic keratatotomy
Moderately low
Small tapered incision ECCE
Scleral buckle with diathermy
No more than unoperated
Paracentesis
Scleral buckle with cryo or laser
Strabismus surgery
Lamellar keratoplasty/pterygium
LASIK*
PRK
Keratomieleusis*

*Late traumatic dehiscence of corneal flap is a potential problem

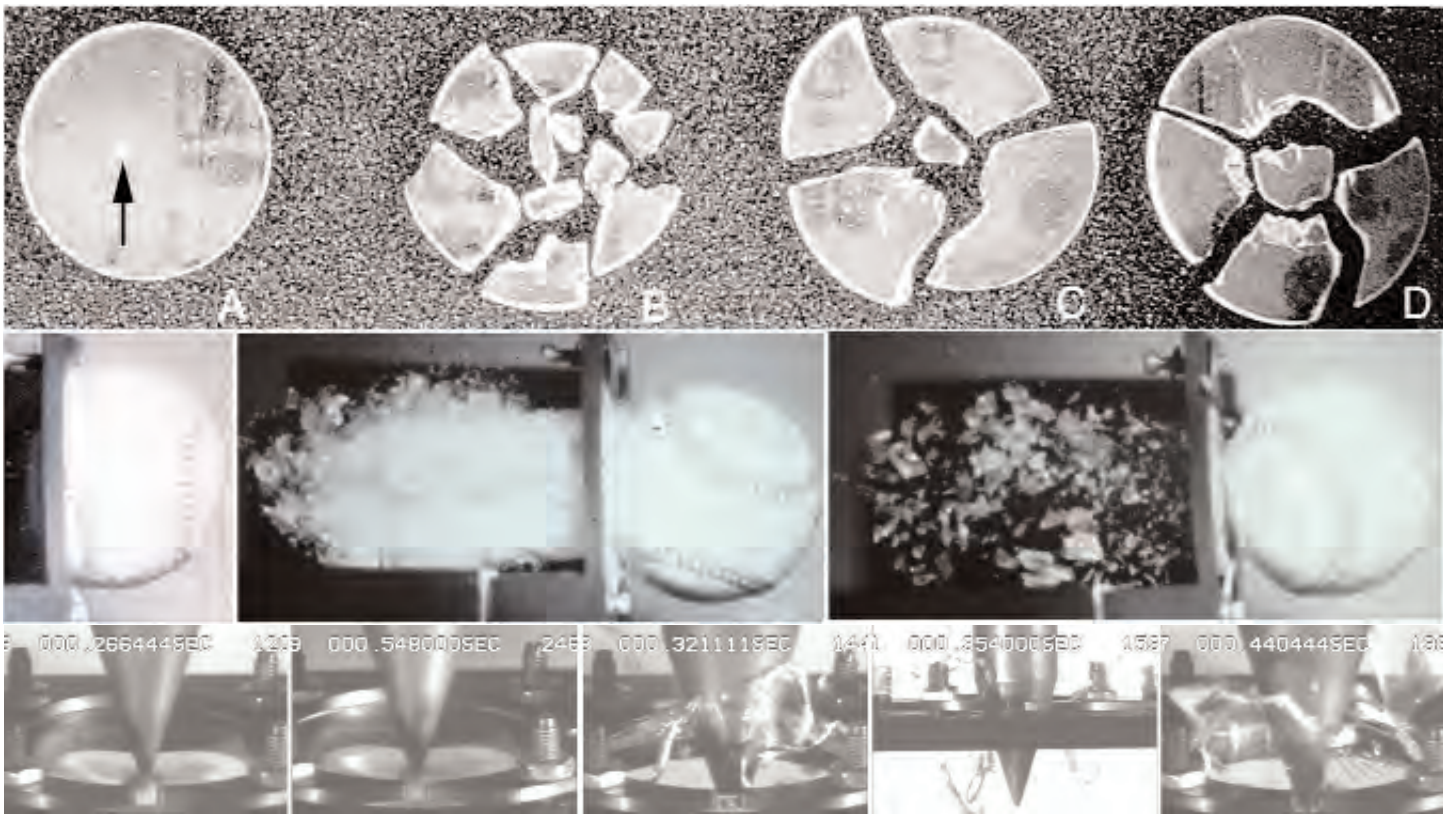


Figure 4 Impact resistance of eyewear lenses

Top row. BB impacts on (a) polycarbonate, (b) glass, (c) allyl resin plastic [CR-39], (d) high-index (1.6) plastic lenses

Center row. baseball impact on industrial safety lenses (left) polycarbonate at 169 ft/s [note flattening of baseball], (center) glass, chemically tempered at 142 ft/s, (right) allyl resin (CR-39) plastic at 137 ft/s

Bottom row. 500 g high mass Z87 test object from left to right onto Trivex, polycarbonate, CR-39, high-index (1.6), and Spectralite lenses. All lenses 2mm thick plano. Mass dropped from 75" (9.34J) onto Trivex and polycarbonate which did not shatter, and from 50" (6.23J) on the other lenses that shattered.(Bottom row courtesy of Nancy Yamasaki

At velocities expected in typical sports, glass, allyl resin, and high index plastic lenses shatter, while polycarbonate (and also Trivex) remain intact.

lens shatter has a poor prognosis and is underestimated. Keeney [76] found 491 cases of spectacle glass injuries resulting in 369 ocular injuries and 37 lost eyes. Over a 1 year period, 3.6% of 446 cases of penetrating ocular injury in Canada were due to shattered spectacle lenses—40% of the shattered lens injuries were to adult male amateur athletes. [77] Between 1978 and 1986, at least 21 racket sport players sustained serious ocular injuries when their prescription glasses (hardened glass or plastic—but not polycarbonate or Trivex) shattered. [78, 123] Of 298 eyes injured by shattered spectacle lenses in a nonindustrial setting, 157 suffered significant damage and 27 were lost. Sports accounted for 53 (17.8%) and BBs for 16 (5.4%) of the shattered eyeglasses. [79] Sixteen of 635 work-related penetrating eye injuries resulted from shattered streetwear spectacle lenses. [80] One of the two penetrating ski eye injuries reported to NETS was the result of dress spectacles shattering on impact from the handle of a ski pole. Two soccer players had significant structural changes to their lids and globes when the ball shattered streetwear glasses. [81] A homemade "potato-gun" caused a sight-threatening corneal laceration when the spectacle lens worn by a 14 year-old boy shattered. [82]

Since approximately half of the population wears eyeglasses, [83] the prescription of the appropriate spectacle lens ***Figure 4*** can protect a huge segment of the population, whereas an incorrect recommendation by the practitioner exposes the patient to the risk of a shattered lens, a perforated globe, and the good chance of permanent disability. [84]

Apart from shattered eyewear, the principal causes of intraocular foreign bodies seen in sports are BBs, shotgun pellets, and fishhooks.

Closed-globe injuries

Contusion

Nonpenetrating trauma results in a wide variety of tissue damage involving chamber angle deformities and injury to the retina, choroid, vitreous, and lens. The injury to the eye depends on the maximum force, the time to the maximum force, the area of contact, and the properties of the impacting object. The expansion of the eyeball perpendicular to the direction of impact, has been proposed as the major cause of the contusion injuries that result from blunt trauma. ***Figures 1, 2*** When a small, high-velocity object (such as a BB) hits the eye on the cornea, the entire eye deforms, and the weakest portion of the retina (upper nasal) often fails. [47,85] When a large object (such as a soccer ball) hits the eye (especially in younger players where the orbital rims are less developed) more energy is directly transmitted to the exposed temporal retina while the nasal retina is protected by the nose. A suction component [86] ***Figure 5, Table 7*** most likely adds to the distortion of the globe anatomy that causes stresses resulting in tearing of structures in the anterior [87] and posterior.[88] segments of the eye. This extreme distortion in the anatomy results in tearing of internal ocular structures (sphincter pupillae, peripheral edge of the iris, anterior ciliary body, attachment of the ciliary body to the scleral spur, trabecular meshwork, zonules, attachment of the retina to the ora ser-

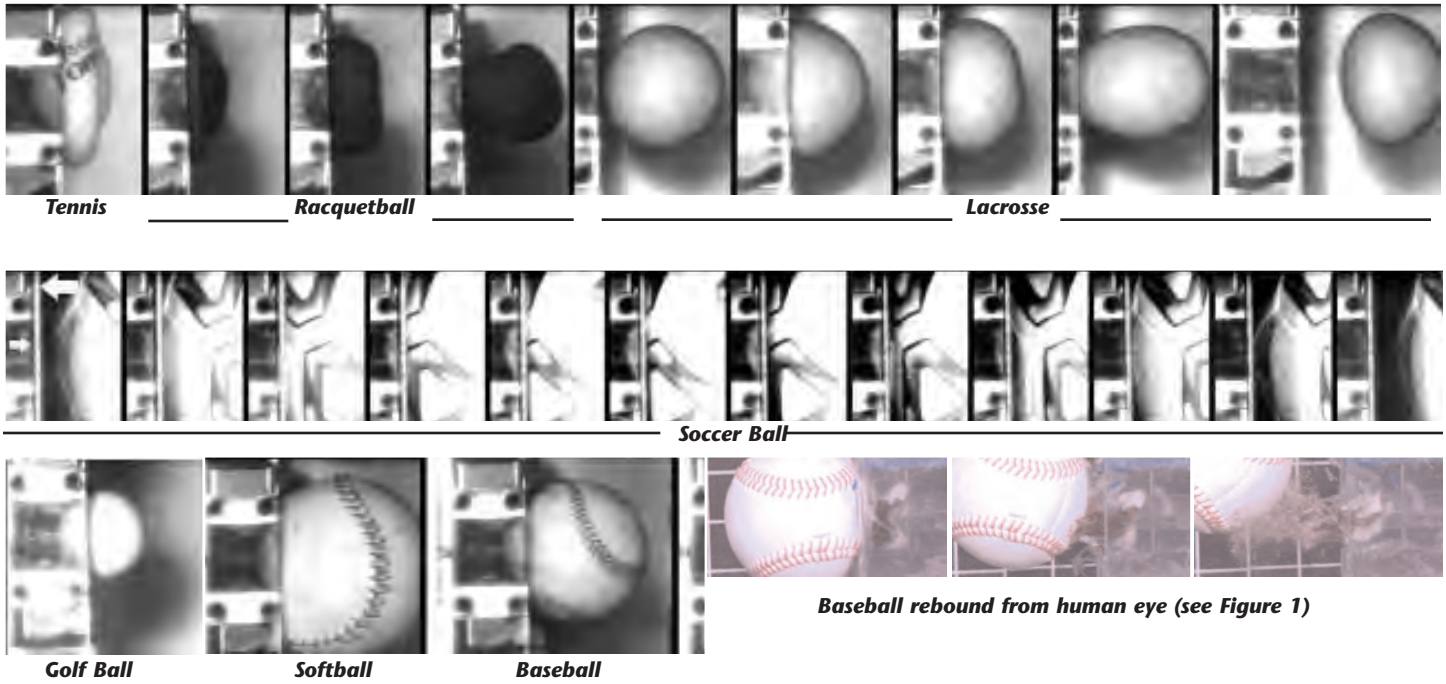


Figure 5. Orbital penetration and suction effect.

The penetration, into an artificial orbit, of tennis ball, racquetball, lacrosse ball, golf ball, softball, baseball, and soccer ball.Adjacent frames are at intervals of 1ms. The frames with the human ruptured eye are a continuation of the the sequence depedted in Figure 1 g-i. Note the suction effect of the ball on the ocular contents as ithe ball. rebounds. (All balls travelling right to left except baseball impacting human cadaver eye which is travelling left to right)

rata, and Bruch's membrane) that are resistant to stretching when the globe undergoes the deformations induced by the force of the impact. [87,89] Because there frequently is a long interval between ocular contusion and the appearance of retinal detachment, and retinal breaks are formed at the time of injury, it is essential to examine the peripheral retina of all eyes that have had a contusion injury. [90] Blunt trauma may cause transient high myopia by anterior shift of the lens-iris diaphragm and thickening of the crystalline lens. [91] Hyphema may be caused by the shock wave of a pure blast injury. [92]

A blow by a blunt object smaller than the orbital opening, such as a BB, paintball, golf ball, finger, or hockey stick, will transmit great forces to the globe. To produce eye injury, less energy is required with high-speed and small-mass missiles (BB) than with low-speed and large–mass missiles (soccer ball).

A blow by a blunt object larger than the bony opening, such as a tennis ball, elbow, or fist, has some of the energy absorbed by the surrounding bones, soft tissues, and orbital floor, which may fracture. There is a high incidence of internal ocular injury in these cases. [93] The concept that an impacting object with radius of curvature above 2 inches (4-inch diameter) rarely causes eye injury because the ball delivers most of its energy to the orbital rims [94] is incor-

Table 7. Orbital Penetration and Penetration Duration (Orbital Contact) of Sportsballs

Sports Ball	Soccer Ball psi	Weight g	Diameter cm	Velocity m/s (mph)	Impact energy J	Orbital penetration mm	Orbital contact ms
Soccer #3	9	355.9	19.6	18.0 (40)	57.5	7.5	8.7
Soccer #3	6	355.9	19.5	18.3 (41)	57.5	7.8	9.3
Soccer #3	3	355.9	19.3	17.7 (40)	57.5	7.7	10.0
Soccer #4	9	369.1	20.8	17.4 (39)	55.7	7.6	9.0
Soccer #4	6	369.1	20.6	17.4 (39)	55.7	7.6	9.0
Soccer #4	3	369.1	20.4	17.4 (39)	55.7	8.4	10.6
Soccer #5	9	435.6	21.8	18.6 (42)	75.3	8.0	9.0
Soccer #5	6	435.6	21.6	18.6 (42)	75.3	7.8	10.0
Soccer #5	3	435.6	21.4	18.6 (42)	75.3	8.7	11.0
Tennis		57.9	6.4	39.9 (89)	46.2	18.6	4.0
Racquetball		40.1	5.6	42.0 (94)	35.6	16.1	3.0
Squash, soft		23.5	4.0	41.1 (92)	23.5	completely enters orbit	stuck
Squash, hard		21.0	4.0	41.1 (92)	17.8	completely enters orbit	stuck
Lacrosse		151.7	6.3	24.4 (54)	45.4	20.0	3.0
Softball		186.8	9.6	32.6 (72.7)	103.0	10.3	1.3
Baseball		144.2	7.4	30.2 (67.3)	66.1	10.8	1
Field hockey		176.0	7.3	27.4 (61)	66.2	7.5	2
Polo		124.5	7.8	38.4 (86)	91.8	7.9	2
Golf		45.5	4.3	43.0 (96)	42.0	13.4	2

Soccer ball orbital penetration is less than for other sports balls, but the time in the orbit is longer than any other sports ball, with a slower force onset rate that rarely causes globe rupture. Soccer ball penetration is not significantly related to the size of the ball or the pressure, but softer soccer balls tend to remain in the orbit longer. Although the lacrosse ball penetrates more deeply into the orbit than a golf ball, the very hard golf ball tranmits a larger force more quickly to the globe, and tends to rupture the globe more than the lacrosse ball.

rect. Large balls (such as a soccer ball) and boxing gloves deform significantly on impact, allowing a small "knuckle" of the ball or glove, with a smaller radius of curvature, to enter the orbit and impact the globe. It is only by experiments utilizing high-speed photography, coupled with injury data, that the true mechanism of injury can be elicited and appropriate protective devices designed.

Lamellar laceration

The primary lamellar laceration potential from sports is the dislocation of a LASIK flap. All patients who have had LASIK should be warned of this potential complication [95-100] and be advised of appropriate protection, lest their flap be dislocated by a finger while playing basketball, [101] or by a tree branch. [102]

Injuries to higher visual pathways

Blows to the skull with direct or indirect injury to the visual pathways may result in permanent or temporary visual loss. [103-110] The huge forces required to produce these injuries can be encountered in many sports (e.g., collision sports, skiing, cycling, motor sports). [111-114] With high-energy loads, eye protection must be considered as part of an integrated eye/face/head/brain protection system.

Principles of preventing sports-related eye injuries

There is a natural sequence of events that will decrease the eye-injury risk of a sport to the individual player.

1. Those involved with a particular sport see a number of injuries and get an *impression* that the sport has a high risk of eye injury.

2. *Data* on the incidence and severity of injuries are collected to confirm or deny the initial clinical impression. This data collection may involve the establishment of special study groups and usually takes several years before the risk is confirmed or denied to the satisfaction of those involved with the sport.

3. If the initial impression is confirmed by the data, then a study of the sport and eye *injury mechanisms* (usually done simultaneously with no. 2) is conducted. This determines whether rules changes alone (e.g., eliminate fighting and high sticking in hockey) will reduce eye injuries to an acceptable level, or whether protective devices (e.g.,hockey face shields) are necessary.

4. If protective devices are necessary, then performance *standards* must be written to ensure that the protective devices will meet the visual requirements of the game while reducing the probability of injury to a specified level.

5. In addition to the development of standards, *certification councils* must be established to ensure that protective devices sold to the athletes meet the standard requirements.

6. If needed, *rules* changes are implemented.

7. *Data* collection is continued to document the effect of rules changes and protective equipment on eye injuries and also the effect of the changes on injuries to other areas of the body. (For example, there was concern that the use of total head protective devices for hockey players might increase injuries to the neck. Extensive studies on change of center of head mass, skating attitudes, [115,116] and analysis of all neck injuries to both protected and unprotected players have shown no increase of neck injury risk due to the protective device. [117] However, the referees and coaches must enforce the rules of the game and not allow the level of violence to offset the effects of injury prevention programs.

8. *Adjustments* in rules, standards, or protective devices are made as data collection shows the need for modification. It may turn out that serious injuries are impossible to prevent, since in protecting one area of the body injuries may be transferred to another. Then society must decide whether it is possible that the sport as it now exists presents too high a risk and should be banned. (For example, the once-popular, but dangerous, sport of jousting would not be permitted a return, but what criteria should we use in deciding whether to ban an existing sport with significant risk to the athlete, such as boxing?) [118]

Distribution of Forces

Forces are best dissipated if they are transmitted over a wide area and the duration of time over which the force is allowed to act is lengthened. [115] If possible, the best area for distribution of forces when one tries to protect the eyes is the frontal bones. These bones are the sturdiest about the orbit and have the tendency to transmit energy into the mass of the face by the lateral orbital margins. [56,119,120]

Whenever a large force is transmitted anywhere on the head the prime consideration must be the ultimate dissipation of this force as it relates to the brain. It is senseless to protect an eye if in so doing the damaging forces are transmitted directly to the brain. All protective devices for the head and face thus require two areas of consideration: (1) is the primary area of concern (e.g., eye, face, teeth) protected? and (2) is the transmission of forces such that there is no added risk to the brain? In collision sports such as hockey or football, this result is best achieved by mounting a face protector on a properly designed helmet. In this manner, the desirable goal of total head (not isolated eye, face, teeth, etc.) protection is achieved. Helmet design must be monitored by comparing predicted force [116,121] with actual measurements of injury to real players in action. [122]

Sports with less energy potential require less protection. A squash ball has little potential for injuring the brain; therefore, attention need only be directed toward protecting the eye. In this case, one might consider it acceptable to transmit the forces to the frontal bone (best) or even less desirable areas (the bridge of the nose, the lateral or infra-orbital rims) and still achieve good eye protection. One could accept a cut on the cheek, a broken nose, or even a fracture of the zygoma as far preferable to the potential loss of the eye.

Eyewear standards

The best sports standards are performance standards that specify how a protector must perform (e.g., field of view, impact resistance, distribution of forces) rather than design standards that contain certain design elements that may or may not relate to performance. By and large, design standards are unnecessarily restrictive, tend to stifle the introduction of better, more innovative protector designs, and are more likely to encounter antitrust problems than performance standards. [124]

It is clear to those who write standards that one cannot tell how a protector will perform until it is tested under game conditions or conditions that approximate game conditions. [123] If those who write standards and test protectors cannot tell how a protector will perform until the protector is tested, it is obvious that the untrained consumer will be unable to determine which prod-

Table 8 Standards and Test Energies

	Test	Energy (j)
ANSI Z80 Fashion eyewear	5/8" steel ball dropped 50"	0.2
ANSI Z87 Industrial eyewear		
glass and allyl resin Rx lenses	1" steel ball dropped 50"	0.9
polycarbonate and Trivex lenses	1/4" steel ball at 150 ft/s	1.1
	500g pointed mass dropped 50"	6.4
Military fragments		
	0.15 caliber 376 mg at 640 ft/s	7.2
	0.22 caliber 1.1 g at 550 ft/s	15.5
ASTM F803 sports eyewear		
tennis	Tennis ball at 90 mph	46.7
squash	Squash ball at 90 mph	19.4
racquetball	Racquetball at 90 mph	32.4
women's lacrosse	Lacrosse ball at 45 mph	29.6
baseball, under age 9	Baseball at 40 mph	23.7
baseball ages 9 to 15	Baseball at 55 mph	45.6
baseball over age 15	Baseball at 70 mph	70.2
	and at 85 mph	77.8

ucts will provide adequate protection with minimal impact on performance by inspection in the retail shop. Severe eye injuries in sports can be prevented by writing performance standards that specify the protector's energy attenuation and visual requirements followed by certification of the protective equipment produced by manufacturers. [125] Sports regulatory bodies must mandate the use of equipment that passes the standard requirements, and governing bodies must legislate against uncertified products gaining access to the marketplace. [126]

Test requirements of relevant eyewear standards are listed in ***Table 8***.

Standards writing organizations

ASTM International

The majority of sports eyewear standards writing in the United States comes under the jurisdiction of *ASTM International* (ASTM http://www.astm.org/). The largest of the approximately 400 standards-writing bodies in the United States, ASTM is neither a government nor a manufacturer's organization but a nonprofit corporation organized in 1898 for development of voluntary standards arrived at by consensus, with strict guidelines for due process, among all interested parties. [127,128]

ASTM committee F-8 on sports safety standards and sports safety was formed in 1968 to address the sharp increase in head and neck injuries in football. [129] ASTM F-8 now has subcommittees that write standards for many sports, including gymnastics, golf, archery, wrestling, fencing, trampolines, fitness products, racket sports, hockey, and baseball, as well as groups concerned with the more general problems of medical aspects and biomechanics, playing surfaces, headwear, footwear, padding, statistics, warning labels and signs, the female athlete, and eye safety.

Standards are designed to be revised as experience is gained. No matter how well the protector performs on paper or in the testing laboratory, it is only the use by thousands of players and continued injury monitoring that prove the protective value or demonstrate the failures of a particular design. For this reason, the ASTM mandates review of every published standard every 5 years. Other standards organizations (e.g., Canadian Standards Association (CSA), [130] American National Standards Institute,(ANSI) Deutsches Institut fur Normung [DIN], International Organization for Standardization [ISO]) operate under various bylaws.

At present, ASTM has completed the following standards for sports eye protectors:

ASTM F803 Eye protectors for selected sports (racket sports, women's lacrosse, field hockey, baseball, basketball)

ASTM F513 Eye and face protective equipment for hockey players

ASTM F1587 Head and face protective equipment for ice hockey goaltenders

ASTM F1776 Eye protectors for use by players of paintball sports

ASTM F910 Face guards for youth baseball

ASTM F659 High impact resistant eye protective devices for alpine skiing

ANSI

The *American National Standards Institute* (ANSI http://www.ansi.org/) writes standards for protective eyewear in the United States with the exception of sports eyewear. It is the central body responsible for the identification of a single, consistent set of voluntary standards called American National Standards, and is the U.S. member of international standards organizations. ANSI follows the principles of openness, due process, and a consensus of those directly and materially affected by the standards.

ANSI standards for eyewear are:

ANSI Z80.5 Requirements for ophthalmic frames

ANSI Z80.1 Prescription ophthalmic lenses-recommendations

ANSI Z80.3 Requirements for non-prescription sunglasses and fashion eyewear

ANSI Z87.1 Practice for occupational eye and face protection

The ANSI Z80 standards are for dress eyewear, also called streetwear spectacles. The test requirements are minimal and geared to the desire for a diversity of styles in fashion eyewear. Streetwear spectacles are not appropriate for work or sports with impact potential. Impact-resistant polycarbonate or Trivex lenses

Figure 6. ANSI Z87 spectacle failure with squash ball at 90mph

should be used for dress eyewear. Streetwear frames are often fragile and have poor lens retention properties. Significant eye injuries have resulted from frame failure. Yet a streetwear frame with an impact-resistant polycarbonate or Trivex lens does give protection from low impact injuries, such as thise from a fishhook or a snapping twig.

The ANSI Z87.1 Industrial eye protectors are not satisfactory for sports for which there are ASTM standard specifications ***Figure 6.*** Yet ANSI Z87+ eyewear, designed to stop small high-velocity fragments, is an excellent choice for moderate impact sunglasses and eyewear for shooting, fishing, cycling, and other activities that involve the potential of impact with a small fragment.

Department of Defense

Military eyewear will be coordinated under a single umbrella program called the Military Eye Protection System (MEPS http://www.dod.mil/) in which testing is done with fragment-simulating (T-37) projectiles ***Figure 7*** either 0.22 caliber, 17 grain (1.1g) at 168 m/s (550 ft/s) or 0.15 caliber, 5.8 grain

Figure 7. Military fragment simulators and ANSI High velocity test object

Left: military 0.15 caliber, Center: military 0.22 caliber, Right: ANSI 1/4" steel ball

(376mg) at 195 m/s (640 ft/s). Sun, wind and dust goggles (MIL-V-43511); ballistic/laser protective spectacles (MIL-PRF-44366B); and special protective eyewear with interchangeable lenses (MIL-PRF-31013) standards assure eye protection from the many hazards anticipated in military combat. Although this eyewear has not been tested for sports use, it would provide excellent protection for the hunting and shooting sports, though not for sports for which specific ASTM standards apply (such as paintball, hockey, and sports covered under ASTM F803.)

NOCSAE

The *National Operating Committee on Standards for Athletic Equipment* (NOCSAE http://www.nocsae.org) has standards for baseball, football, and lacrosse helmets; baseballs and softballs; and face shields for football and men's lacrosse.

Current NOCSAE standards include:

Standard Drop Test Method and Equipment Used in Evaluating the Performance Characteristics of Protective Headgear. NOCSAE Doc. 001-00m02

Standard Performance Specification for Newly Manufactured Football Helmets. NOCSAE Doc. 002-96m98

Standard Performance Specification for Recertified Football Helmets. NOCSAE Doc. 004-96m98

Standard Projectile Impact Testing Method and Equipment Used in Evaluating the Performance Characteristics of Protective Headgear, Faceguards or Projectiles. NOCSAE Doc. 021-98m02

Standard Performance Specification for Newly Manufactured Baseball/Softball Batter's Helmets. NOCSAE Doc. 022-98m02

Standard Performance Specification for Newly Manufactured Baseball/Softball Catcher's Helmets with Faceguards. NOCSAE Doc. 024-98m02

Laboratory Procedural Guide for Certifying Newly Manufactured Football Helmets. NOCSAE Doc. 003-96m02

Laboratory Procedural Guide for Recertifying Football Helmets. NOCSAE Doc. 005-96m02

Laboratory Procedural Guide for Certifying Newly Manufactured Baseball/Softball Catcher's Helmets with Faceguards. NOCSAE Doc. 025-98m02

Troubleshooting Guide for Test Equipment and Impact Testing. NOCSAE Doc. 100-96m97

Equipment Calibration Procedures. NOCSAE Doc. 101-00m02

Headforms

Headforms are necessary for testing and standards development. Headforms may be impacted without injury and give consistent results. Choosing the proper headform is essential to any protector design or testing. The anthropomorphic features, hardness, and energy-absorbing characteristics all affect test results. Comparison of the results on the test headform with those actually achieved on the human head are essentia1. [131,132] The Canadian headforms, which are based on actual physical and radiologic measurements of thousands of heads, [133] are better proportioned for eyewear testing and design than the commonly used US head forms (Alderson 5, 50, and 95 percentile), which are based on projections made from measuring a sample of military men. NOCSAE revised its test forms with anthromorphic measurements based on CSA data.

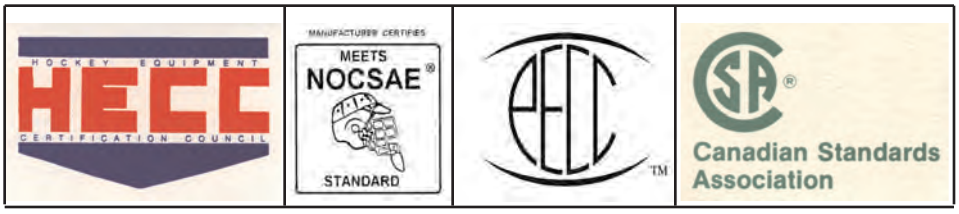
Equipment certification councils

Protectors are often certified, giving the user the assurance that the protector will afford reasonable protection. A Sports Equipment Certification Council is composed of coaches, participants, scientists, physicians, manufacturers, and administrators. Its purpose is to seek out and select codes and standards, including test methods and procedures, for equipment used in athletic, sporting, recreational, and leisure time activity. In addition, the council identifies and publishes all factors associated with safety, whether it be protective equipment, playing surfaces, rules, attitudes, officiating, training, conditioning, and administration. [132] The Council will usually have a seal ***Figure 8*** that manufacturers may affix to a protective device that is assurance to the consumer that a product meets the specifications of a performance standard. ***Figure 9***

HECC

The *Hockey Equipment Certification Council* (HECC http://www.hecc.net) is an independent, nonprofit organization that was established in 1978 through the joint efforts of the Amateur Hockey Association of the United States and a number of interested volunteers. HECC certifies ice hockey equipment, including helmets and face shields; selects codes and standards to certify playing equipment and facilities; monitors the effectiveness of its certification program; and promotes research pertaining to the prevention and reduction of ice hockey injuries. HECC is extremely effective in fulfilling its mandate of reducing

Figure 8 Certification seals



injuries in hockey.

PECC

The *Protective Eyewear Certification Council* (PECC http://www.protecteyes.org) certifies protectors complying with ASTM standards (except for ice hockey). A testing laboratory must provide evidence of the successful completion of the *American Association for Laboratory Accreditation* (A2LA http://www.a2la2.net/) evaluation process to perform the tests that are specified in the standards.

CSA

The *Canadian Standards Association* (CSA http://www.csa.ca) certifies products complying with the Canadian racquet sport and ice hockey standards, which are similar to the ASTM standards.

Figure 9. Buyer beware of uncertified products, advertised for sports, that do not protect.

Each of these products was advertised as a protective device for the sports depicted (baseball and squash). None of the major US distributors would produce evidence that the product was tested by an independent laboratory, but gave assurance that "rigid tests" were done by the manufacturer. Each product failed when tested to ASTM F803 for the advertised sport. None of the manufacturers recalled their product when informed of the test results.

Obsolescense in Protective Equipment

Protective equipment is obsolete when it no longer provides adequate protection, cannot be purchased under normal circumstances, is no longer in the desired style, is unreconditioned "hand me down" equipment, or is worn out, broken, or ill-fitting. [134] As injury data result in standard modification, certification councils must publish a list of equipment that has become obsolete by newer advances, and this obsolete equipment must be discarded.

Guidelines for Sports Participation

The American Academy of Pediatrics classifies sports into three main categories: (a)contact/collision, (b) limited contact/impact, and (c) non-contact. [135-137] and suggests that some sports are contraindicated for the one-eyed participant. The traditional contraindications to athletic participation are more appropriate to the systemic, musculoskeletal, cardiac, respiratory, paired-organ and central nervous systems than to the eye. Whereas musculoskeletal injuries and cerebral concussions are inevitable in contact/collision sports (such as rodeo) and are rare in noncontact sports (such as golf), eye injuries may be more common and severe in the "safer" sport. The recommendations of the American Academy of Pediatrics may be considered over-restrictive as society becomes more aware of the civil rights of athletes and the need to allow the handicapped to participate in sports. [138-140] It is apparent that more realistic guidelines for participation in sports by persons with various ocular handicaps and ocular diseases could be devised. Although such a list is dependent on reliable injury data that are not available at this time, there was sufficient information for the International Federation of Sports Medicine to release a position statement on eye injuries and eye protection in sports, which should help reduce eye injuries worldwide. [139]

The one-eyed athlete

Severe eye injury to a child can result in posttraumatic stress disorder, even if the vision is restored to reasonable levels after surgery. [141] The emotional, psychological, and legal impacts of severe eye injuries are often neglected but attention to them is essential in the management of all patients who have suffered severe injury—especially that involving the loss of an eye. [142,143] The risk of becoming blind is markedly higher for the amblyopic patient (1.75 ± 0.30 per 1,000) than for the general population (0.11 per 1,000 for children, 0.66 per 1,000 for adults). Trauma (work, sports, violence, accidents) causes over 50% of the resultant blindness. [144]

How can we define one-eyed? For the purpose of recommending extra safety precautions, a person is functionally one-eyed when loss of the better eye would result in a significant change in lifestyle owing to the poor vision in the remaining eye. [145] A person certainly should be considered functionally one-eyed if his or her best corrected vision in the involved eye is 20/200 or less, with the other eye found normal by an ophthalmologist. On the other hand, most of us would function fairly well with 20/40 or better vision in the remaining eye. More difficult is advising patients with between 20/40 and 20/200 best corrected vision in the affected eye. The loss of the ability to drive a vehicle legally in most states would be a handicap to most persons. The inability to drive would significantly interfere with the jobs available to a youngster when he or she is older, and studies would be more difficult throughout the school years. Therefore, a child should be considered functionally one-eyed when the best corrected vision in the poorer eye is less than 20/40, and an adult is functionally one-eyed if he or she believes the level of vision in the poorer eye would interfere with life or livelihood if the better eye were lost. [146] Functionally one-eyed athletes (and their parents in the case of minors) must be well informed of the potential long-term consequences if the better eye were lost. They should also be informed of the risks of injury (without and with various eye protectors) and the possibility of repair of injuries typically seen with the sport in question.

It is only by full discussion of the potential serious long-term consequences of injury to the better eye that the ophthalmologist, the athlete, and the parents can agree on the wisdom of participation in a particular sport as well as the level of protection necessary for the better eye. The most effective protection is possible only when the athlete understands the risks and is anxious to cooperate in the effort to protect the eyes while still allowing participation and enjoyment of the preferred sport. Having the athlete wear an occluder over the better eye for several days will allow him or her to better evaluate the ability to function with the poorer eye. Usually, if the athlete is sincere and honest with himself or herself, it is fairly easy to reach agreement among the athlete, parents, ophthal-

mologist, and sports officials as to whether the athlete is functionally one-eyed.

As protective devices improve and effective sports eye protectors are developed, more and more sports become quite safe even for the one-eyed athlete. [147] The division of sports into contact/collision (boxing, field hockey, football, ice hockey, lacrosse, martial arts, rodeo, soccer, wrestling); limited contact/impact (baseball, basketball, bicycling, diving, high jump, pole vault, gymnastics, horseback riding, ice and roller skating; cross country, downhill, and water skiing; softball, squash, handball, volleyball), strenuous noncontact (aerobic dancing, crew, fencing, discus, javelin, shot put, running, swimming, tennis, track, weight lifting), moderately strenuous noncontact (badminton, curling, table tennis) and nonstrenuous noncontact (archery, golf, riflery) [148] tells little of the risk of eye injury. From an eye injury perspective, it is far more dangerous to play badminton (moderately strenuous non-contact) without an eye protector than to play ice hockey (contact/collision) with a full face mask.

Any banning of athletic participation in certain sports should be based on guidelines using an experiential framework rather than tradition or anecdote. [149] The athlete deserves a true discussion of the risk of eye injury involved in a chosen sport. The outright ban, by some schools, of the one-eyed from participation in collision and contact sports, while the one-eyed students are permitted to play more dangerous (to the remaining eye) sports, such as tennis, is not prudent and should be reevaluated. Unless the athlete is especially gifted in a particular sport, or has psychological reasons to participate in a chosen sport, a safer sport (e.g., track and field, gymnastics) should be encouraged and will usually be chosen. The American Academy of Pediatrics recommendations now take into account that with adequate protection, the one-eyed may participate in most sports. [148] The medical/school committee should specify that the one-eyed athlete follow the safety guidelines presented in this text or modified in the future.

At this time, the only sports absolutely contraindicated for the functionally one-eyed are boxing and full-contact martial arts, since the risk of serious injury is very high and there is no known effective eye protector. Wrestling and the non-contact martial arts, while they have a lower incidence of eye injury, also do not have effective eye protection available and should be strongly discouraged for the functionally one-eyed and banned for the monocular athlete. If the player, parents, and possibly their lawyers are persistent and insistent after an informative discussion, they should be required to sign appropriate waivers as dictated by the school committee. The waiver has a dual purpose: it helps ensure that the athlete will wear appropriate protective devices for practices and games, and it often affords the only possible legal protection for school committees and members of sports-medical committees faced with the dilemma of the one-eyed athlete who insists (or whose parents insist) he or she play a sport with high risk of eye injury, such as wrestling, for which there is no adequate known protection. The best medical advice says that the functionally one-eyed athlete should not participate in a high-risk sport, such as boxing, but it seems that there may be confusion in the law. [150] The Massachusetts law reads, "The health and safety of each student must be paramount in every phase of the instructional physical education program," and also "Each school shall provide equal opportunity for physical education for all students." [151] Federal law states, "Students who can participate in regular physical education programs for all or some aspects of physical education must be placed in such programs." [152] The physician becomes hard put to prove that he or she is not discriminating against the handicapped by excluding the one-eyed student from some sports. [153]

From a performance standpoint, the one-eyed can usually function quite well in most sports. Ocularists (makers of prosthetic eyes), who deal with many one-eyed people, are aware of this fact; [154] however, ocularists should also have expertise in available eye protection and give appropriate recommendations to the patient. The reinforcement of the protective message is very important. Compliance with eye protector use is more likely if the athlete is informed of the need for protection, and given *specific* advice by the ophthalmologist, optometrist, optician, [155] and ocularist.

Protective devices

Fortunately, most sports-related eye injuries are preventable with properly designed equipment. The following is a practical guide for sports eye protection so that persons whose responsibilities involve the eye in athletics can easily determine the protective equipment they should recommend or provide. [156] A protective device should prevent damaging forces from reaching the eyes by dissipating potentially harmful forces over time and area. This theory is simple enough, but the practical application can be difficult. As soon as design is begun on a protective device for a sport with an ocular hazard, many problems arise. What forces are involved in this sport? Are they high-velocity, low-mass (hockey puck); low-velocity, high-mass (player sliding into a goal post); or a combination of high-velocity and high-mass (bicycle racer collision)? Does a protector have to be designed differently for each type of force? How? Where on the head will the forces be transmitted, and how will it be done? Will the player be killed or suffer brain damage if the force is transmitted to his or her brain through the protective device, rather than being dissipated into broken facial or orbital bones as was the case before the protector? Will the protector change the form or appeal of the game? What about the design, expense, weight, interference with vision, product liability, and full disclosure to the consumer?

In a given sport, these questions cannot be answered by any one individual, since expertise at many levels and different areas of interest is required. The best way to design and build a protective device is by the development of a performance standard as discussed above.

An experienced ophthalmologist, optometrist, optician, or athletic trainer can help an athlete select appropriate protective gear that fits and looks well. The best designed protective device, if it does not appeal to the tastes of the player, will remain on the dealer's shelf. Sports programs should assist indigent athletes in the evaluation process and in obtaining protective eyewear.

Selection criteria for protective eyewear

1. Proper fit is essential. Protective eyewear will only be worn if it is comfortable and allows good vision. Helmets must have a properly fastened chin strap for optimal protection. The optician should fit the patient with a protector that feels comfortable and fits snugly. A good test for a snug fit is to lightly run a finger around the perimeter of the protector. There should be no gaps large enough to permit the finger

Table 9. Recommended Eye Protectors for Selected Sports

	Minimal Eye Protector	Comment
Baseball/Softball		
Youth Batter/Base Runner	ASTM F910	Face guard attached to helmet
Baseball/Softball, Fielder	ASTM F803 for baseball	ASTM specifies age ranges
Basketball	ASTM F803 for basketball	ASTM Specifies age ranges
Bicycling	Helmet plus Streetwear ANSI Z80, industrial ANSI Z87.1, or sports ASTM F803 eyewear	Use only polycarbonate or Trivex lenses. There are excellent plano industrial spectacles that are inexpensive and give good protection from wind and particles
Boxing	None available. Not permitted in sport.	Contraindicated for functionally one-eyed
Fencing	Protector with neck bib	
Field hockey (both sexes)	Goalie: full face mask; others ASTM F803 for women's lacrosse	Protectors that pass ASTM F803 for women's lacrosse also pass for field hockey.
Football	Polycarbonate eye shield attached to helmet-mounted wire face mask	
Full-contact martial arts	None available. Not permitted in sport.	Contraindicated for functionally one-eyed
Ice hockey	ASTM F513 HECC or CSA certified face mask on helmet	
Goaltenders	ASTM F1587 HECC certified full face shield on helmet	
Lacrosse, Men's	NOCSAE face mask attached to lacrosse helmet	
Lacrosse, Women's	ASTM F803 for women's lacrosse	Should have option to wear helmet with attached face mask
Paintball	ASTM F1776 for paintball	
Racket Sports: (badminton, tennis, paddle tennis, handball, squash, racquetball)	ASTM F803 for specific sport	
Soccer	ASTM F803 for any selected sport	There is no specific standard for soccer. At this time, eye protectors that comply with ASTM F803 for any specified sport are recommended
Street Hockey	ASTM F513 Face mask on helmet	Must be HECC or CSA certified
Track and Field	Steetwear/fashion eyewear	Use only polycarbonate or Trivex lenses
Water Polo, Swimming	Swim goggles with polycarbonate lenses	
Wrestling	No standard is available	Custom protective eyewear can be fabricated, but no standards available. Not recommended for functionally one-eyed.

to lightly touch the eye. The user should compare several protectors for comfort, vision, and fit. Anti-fog treatment is often factory applied or may be applied by the user.

2. Protectors with clear lenses (plano or prescription) should have impact-resistant polycarbonate or Trivex lenses. If for some reason, a polycarbonate or Trivex lens cannot be used, the athlete who participates in an eye-risk sport should either: (1) wear contact lenses plus an appropriate protector as listed in ***Table 9, Figure 11***, or (2) wear an over-the-glasses eyeguard that conforms to the specifications of ASTM F803 for sports for which an ASTM F803 protector is recommended.

3. For sports requiring a face mask or helmet with an eye protector or shield, functionally one-eyed athletes should also wear lensed sports eye protectors that conform to the requirements of any sport specified in ASTM F803 to maintain some level of protection if the face guard is elevated or removed (as in ice hockey or football by some players on the bench).

4. Contact lenses offer no protection. Therefore athletes who wear contact lenses must also wear appropriate eye protection.

5. Athletes must replace sports eye protectors that are damaged or yellowed with age, because they may have become weakened.

Classification of sports eyewear

Sports have very different eye, face, and head-brain risk, and thus require specifically designed protective equipment, The equipment can be classified into:

1. A helmet with an integral face protector for sports that combine very high energy with a significant potential for eye contact (football, men's lacrosse, youth baseball batter/base runner, polo, ice hockey, automobile and motorcycle racing, downhill ski racing).

2. A helmet with separate eyewear for sports with a significant brain injury potential, but less potential eye contact (riding a bicycle, horse, or motorcycle). Note: many motorcycle, and some bicycle activities require a helmet with an integral face protector.

3. A face-supported protector for sports that have significant eye and face danger, but less potential for brain injury (paintball, fencing, baseball catcher and umpire, and youth baseball

players who wish to protect the entire face.

4. An eye protector that conforms to the requirements of ASTM F803 for sports that pose mainly an eye injury risk (racket sports, basketball, women's lacrosse, field hockey, baseball. It is recognized that baseball, women's lacrosse and field hockey also have head and face injury potential, but, other than the helmet mounted face protectors for youth baseball batters and base-runners, and some face mounted protectors for youth baseball fielders, full face protection has not been accepted by most players and sports officials of these sports.

5. Eyewear that conforms to the military fragment or the high-velocity ANSI Z87 test requirements for the shooting sports.

6. Fashion eyewear when there is negligible eye injury risk. There are several types of clear material (glass, allyl resin, high-index plastic, acrylic, polycarbonate, and Trivex) from which prescription or non-prescription (plano) lenses may be fabricated. Polycarbonate and Trivex are the most shatter resistant lens materials and are recommended for all eyewear.

Sunglasses for sports

The improper choice of sports sunglasses may be hazardous and degrade visual performance. [157] Both visible and ultraviolet light can result in eye injury, which may be minimized by the use of appropriate sunglasses. It is controversial whether or not short-wavelength (wavelength is expressed in nanometers nm) visible (<510nm, blue) light increases the tendency to macular degeneration, but there is evidence that chronic exposure to sunlight is associated with the development of early age-related maculopathy. [158-161] Exposure to ultraviolet light causes cataracts, [162-166] corneal changes (climatic droplet keratopathy, pinguecula, pterygium, and acute photokeratitis [snow blindness]),[167-169] uveal melanoma, [170,171] premature skin aging and sunburn, [172] skin cancers (basal cell carcinoma, squamous cell carcinoma, melanoma) [171,173] Even relatively brief exposure to viewing the sun when high in the sky (zenith above 60%) may result in solar retinitis due to photochemical injury from intense short-wavelength (blue) and UV radiation. [174-176] Many clinicians have the impression that herpes simplex keratitis and recurrent corneal erosion may be precipitated by exposure to sunlight.

Reflected UV light also must be considered. Fresh snow reflects about 80%, older snow over 50%, clean white sand about 30%, water 5%, and earth and grass less than 5% of the ambient ultraviolet light. Thus the greatest UV light exposure occurs at high altitude on a field of fresh snow. Mountaineers, skiers, sailors, and lifeguards, are exposed to large doses of visible and ultraviolet light, at times in situations in which there is the potential for injury from impact, in adverse conditions of high wind or dust. The inability to see well because of photokeratitis, windburn, corneal foreign bodies, or traumatic injury from shattered spectacles may be life-threatening as well as eye-threatening, so the proper choice of sunglasses is essential. Dark sunglasses permit one to be comfortable in bright light without squinting. However, one must be certain that the glasses have adequate absorption in the toxic UV and blue light ranges. [177] Wearers and those observing them should be aware that the reflection from the front surface of mirrored sunglasses may result in severe sunburn to the nose unless extra protection is used. [178]

Sunglasses are especially important for those who have had cataract surgery. Removal of the lens of the eye exposes the retina to wavelengths above approximately 300nm. In the 325nm and 350nm UV radiation range, the retina is approximately six times more sensitive to damage than to short wavelength visible radiation of 441nm. Since untreated polymethyl methacrylate intraocular lenses (IOL) absorb UV radiation only below 300nm to 320nm; [158] many intraocular lenses, classified as UV protective, offer less than optimal protection; [179] and it is not known how long the UV filter on UV absorbing IOLs lasts, it is prudent for all aphakic or pseudophakic athletes to wear sunglasses that absorb 99% of light below 470 to 500 nm.

Athletes who want maximum UV light protection should wear a hat with a brim, which reduces ocular exposure by half, [180] and close-fitting sunglasses that absorb UV when in conditions in which they could get sunburned. [181] There is considerable variability in the quality of sunglasses [182] that is of concern in children's sunglasses [183] since children frequently spend more time in the sun than adults do; damage to the lens (and possibly retina) from UV is cumulative, and the crystalline lens of children transmits more short-wavelength visible radiation and UV light to the retina than does that of the adult. [158]

Even with darkly tinted glasses, there is no way to predict by gross visual inspection which lenses effectively filter reasonable quantities of the near-infrared light (700 to 800nm) and near-UV light (300 to 400nm) that are not visible to the human eye. Cost, color, and lens composition are unreliable indicators of adequate filtration. In one study, 53% of glass and 11 % of plastic lenses had an unfavorably high near UV light transmission peak greater than 25%. [184] Eighty percent of the amount of infrared light present in daylight is transmitted to the retina. Although the infrared light present in daylight is not toxic in itself, some believe that infrared light may contribute to damage from UV light and lower wavelengths and may contribute to ocular discomfort or fatigue. Since infrared light contains no useful visual information, it is probably wise to filter it out. [158] UV light absorption is quite different for various lens materials. [185]

The vast majority of sunglasses sold for sports use are deficient in impact resistance. A sports sunglass should prevent rather than contribute to injury. The combination of lens and frame must prevent ocular contact by either the missile or the sunglass lens. Manufacturers should state the sports for which the sunglass is intended. Safety requirements are the same as for protective eyewear with clear lenses. Manufacturers should be required to provide the following information, in a statement easily understood by the consumer, on all sunglasses sold for use in sports: the standard specifications to which the sunglass conforms, the percent of visible light transmitted through the lens, the percent of UV light and infrared light (wavelengths specifically stated) transmitted through the lens, additional treatments or coatings (for example, polarization) to reduce glare.

The ideal sports sunglass should have the following characteristics:

UVB (280-315nm)—less than 5% transmittance; less than I % transmittance for wavelengths less than 310 nm.

UVA (315-400nm)—less than 10% transmittance, and absolutely less than maximal visible light transmittance; for aphakes, less than 1 % transmittance.

Blue light (400-500nm)—less than 10% transmittance and absolutely less than the maximal visible light transmittance. A blue light transmittance of 25% to 50% of the peak visible transmittance would be desirable.

Long-wavelength visible light (500-760nm)—less than 15% transmittance for bright conditions, such as sand or snow.

Infrared (above 760nm)—filtration desirable but not essential.

Allow color discrimination sufficient to recognize traffic signals.

Have a tightly-fitting "wrap around" style, or have side shields and either have a rim across the top or be used in conjunction with a brimmed hat to protect against oblique incident radiation in very bright conditions.

Have the option of polarization to decrease glare from water for fisherman and boaters.

Have aerodynamic efficiency to combat the drying effects of wind in speed and wind sports (e.g., cycling, yachting, mountaineering, skiing).

Be lightweight. Heavy sunglasses will tend to fly off the face with rapid changes in head position.

Have cosmetic acceptability.

Be impact resistant, consistent with the intended use.

These recommendations point to dark amber polycarbonate or Trivex lenses (although lighter shade lenses could be used if the user wore a brimmed hat). [176,177]

Figure 10. Safety Recommendations

1. **Eyewear should be fabricated with highly shatter-resistant polycarbonate or Trivex lenses unless there is a specific reason for another lens material. Children, functionally one-eyed people and active adults require polycarbonate or Trivex lenses.**
2. **For sports that have the potential for eye contact, use eyewear that is certified by the Protective Eyewear Certification Council (www.protecteyes.org) to ASTM F803. ASTM F803 covers the racquet sports, women's lacrosse and field hockey, baseball, and basketball. For other sports, such as soccer, protectors should meet or exceed ASTM F803 standard specifications for squash. Prescription sports eyewear requires 3-mm-thick polycarbonate lenses**
3. **Sports with high impact, such as ice hockey, men's lacrosse, and youth baseball (batter/base runner) require a face shield mounted on a helmet designed for the sport. Paintball protectors must conform to the requirements of ASTM F1776.**
4. **People working with exposure to flying chips or with power tools should use protectors that meet ANSI Z87.1 Goggles are the safest. Only polycarbonate or Trivex lenses should be used.**
5. **Many workplace activities, such as using a chain saw, require, in addition to safety glasses or goggles, a helmet with a face shield designed for the activity.**
6. **Sunglasses should conform to the above safety recommendations. Sunglasses lenses should attenuate blue light, which is potentially hazardous to the macula. Gray, amber, or brown lenses are preferred. Blue-colored sunglass lenses that transmit blue light should not be used.**

How do I know what to buy, prescribe or dispense?

It could be disastrous to buy, prescribe or dispense what you believed was protective eyewear and then have the eyewear fail. Compounding the problem is the fact that some manufacturers make "sports" eyewear that does not conform to safety standards, ***Figure 9*** and that most ophthalmologists and consumers do not know what protection standard should apply for a specific activity.

The safest way to choose an eye protector is to look for a certification seal ***Figure 8*** to assure that the protector has been tested by an accredited laboratory to a specific safety standard.

The spectacle prescription will be clear to the optician if the note *"Polycarbonate or Trivex lenses are required for children, functionally one-eyed people and active adults"* is printed on the front of the prescription.

Although a fashion eyewear frame has little impact resistance, it is far better to have a lens that is shatter resistant in front of the eye than risk a lacerated globe from a shattered lens. It is almost certain that the eye care professional who dispenses or prescribes a spectacle lens that shatters easily will be sued if the shattered lens results in significant injury. Therefore it is prudent to prescribe, dispense, and wear eyewear with extremely shatter-resistant polycarbonate or Trivex lenses. To test the strength of these lenses, try to break them with a hammer.

Safety recommendations on Rx pad

Recommendations, printed on the reverse side of all spectacle prescriptions, should help the patient choose appropriate protective eyewear. ***Figure 10***

Contact lenses

Because contact lenses offer no protection from impact, it must be stressed to patients that protective devices, where indicated, should be worn in addition to the contact lenses. Patients who request contact lenses for sports use deserve a few minutes of discussion of injury prevention. [186]

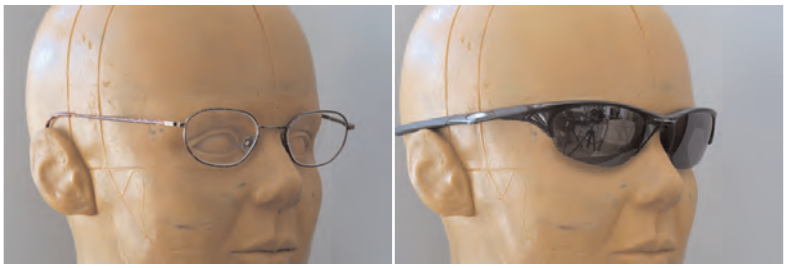
Despite the fact that contrast sensitivity may be decreased with daily wear soft lenses, [187] contact lenses, especially for people with large prescriptions, do offer advantages for many sports—better visual field, no fogging, and staying in place with rapid motion. Lens technologies that combine the excellent visual acuity of rigid gas-permeable contact lenses with the comfort and retention characteristics of soft lenses are preferred by many athletes, especially those with astigmatism. [188] Large-diameter (15.5 mm) and scleral (18-24mm) soft lenses are available for athletes who cannot wear standard soft or rigid gas-permeable lenses because of decentration with sports activity. [189-191]

Many sports are played in environments that make contact lens wear more difficult because of increased exposure to water, wind, sun, dust, and dirt. The use of wraparound polycarbonate sunglasses over the contact lenses frequently allows the mountain bicycle racer to have the benefits of contact lens vision in the face of wind and debris. For sports, such as ice hockey, in which low humidity may be encountered, low-water, low-soiling, low-dehydrating, larger-diameter, thin, soft contact lenses, seem to give satisfactory results. [192] Wind, dry air, UV light, and decreased oxygen at high altitude often cause punctate keratitis in skiers and mountaineers. [193-195] Skiers who wear contact lenses should be encouraged to wear goggles that absorb UV light and break the wind. If contact lens wear becomes impossible, spectacles could save an otherwise ruined vacation.

Figure 11. A summary of protective eyewear

ANSI Z80

Fashion eyewear
Use only with polycarbonate or Trivex lenses
OK for eye safe sports and dress eyewear



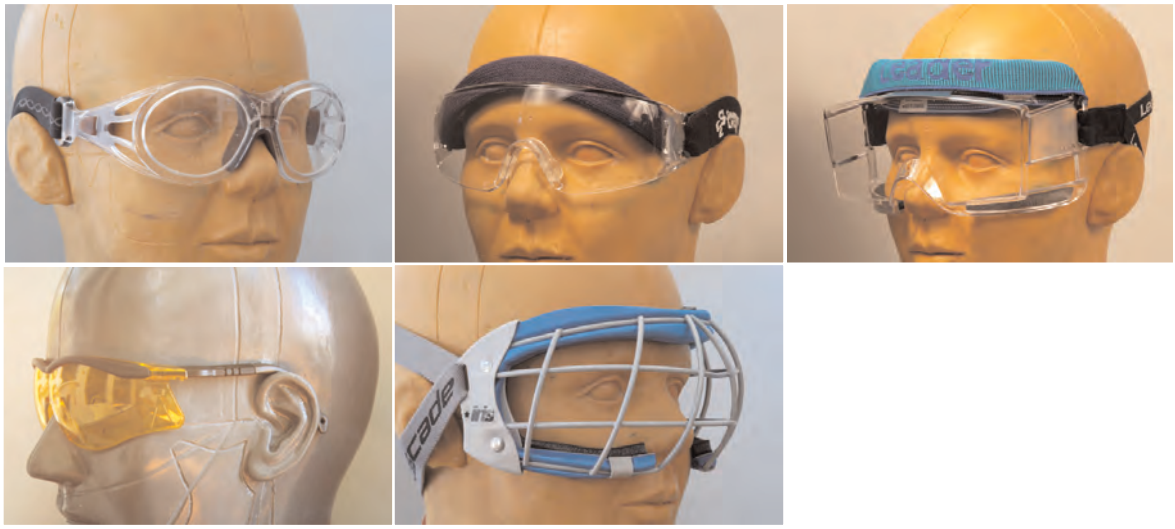
ANSI Z87 Industrial Eyewear and sports eyewear that passes theZ87 high velocity test

Must bear the Z87+ mark for work with tools and chemicals
Z87+ Goggles safer than spectacles
Z87+ spectacles or goggles ALWAYS worn under a face shield
OK for sports with no ASTM standards, such as Frisbee or cycling
A good choice for daily-wear sunglasses
NEVER for paintball

Military eyewear and industrial or sports eyewear that passes the military fragment high velocity test
Military eyewear not available to the general public but other eyewear that passes military tests good for shooting and hunting

Eyewear certified to ASTM F803 by PECC

Necessary for sports covered by ASTM F803 (racket sports, women's lacrosse, field hockey, basketball, baseball)
Also adequate for soccer
Must be certified for the specific sport to be played
Available for prescription eyewear (upper laft) for use over Z80 eyewear (upper right), as an eye shield of polycarbonate (top center) or wire (bottom center), and as a plano spectacle with interchangeable lenses (clear, yellow, and gray for sunglass use



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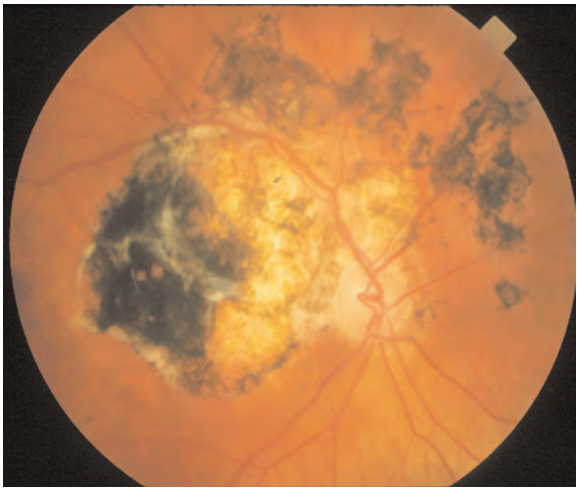
Prevention Pays



This is a photograph of a retina with a dense macular scar, a typical sports injury, that gives the same visual results as macular degeneration—loss of central vision with the inability to see the large E on a typical eye chart. After the fact, there is no treatment available to restore vision. Before the fact, had this young man been wearing a face shield (that conformed to ASTM F910) while at bat in a beasball game, his retina would be normal and his vision unaffected by the errant pitch. We hope that this discussion of the mechanisms and prevention of sports eye injuries results in more protected athletes and fewer eye injuries.

Appendix 1 NEISS sports and recreational eye injury estimated 1984-2001

	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001
Eyeglasses	2440	1818	2164	2096	2205	1329	3257	2030	3263	2189	2902	2561	1802	1601	2285	2424	4950	2668
Fishing	949	1108	586	1074	1146	478	1099	1017	856	1413	3223	1331	859	1173	925	1118	2830	1135
BB's, pellets. Gas, air or spring powdered guns	1554		1833	2320	3189	2060	2370	1984	4649	1937	2330	2625	2446	2348	2720	3221	6292	2994
Firearms				102				155	866	800		112				85	181	97
Bicycles			2298	2123	1963	2466	2590	2077	2916	2293	2721	2178	2308	2459	2566	3370	3244	1365
Baseball	1241	7205	7644	6582	6280	5518	6379	6950	8083	6136	4227	3265	3837	2270	2635	3962	2761	6193
Softball											2480	1549	1192	1290	1394	2369	1095	2414
Basketball	4256	5510	6049	7320	5492	5900	7705	6842	8304	8521	9117	8420	7852	7673	8723	8383	15714	8320
Handball			445	447	165	280		60	51	102	89	37	70	76	234	71	111	111
Squash, racquetball, paddleball		1994	2077	1411	1187	877	1291	1521	1646	1356	1252	953	1290	715	921	750	1587	773
Tennis		910	1758	1055	1301	1517	1563	1951	2413	1579	1403	702	1180	771	1613	972	1589	531
Badminton	124	109	221	81	182	0	227	448	51	146	85							
Table tennis																	153	153
Total racket sports	124	3013	4501	2994	2835	2674	3081	3980	4161	3183	2829	1692	2540	1562	2768	1793	3440	2341
Ice hockey			119	102				203	96	189	103	54	130	84	319		101	101
Field hockey			140	150	38	82	86	227			60	34	17	94	75		82	76
Street hockey		69	38	38			34	60	20	119	105	163	129	150	233	71	84	84
Hockey		285	425	438	292	69	394	563	855	638	620	556	739	280	987	289	1306	428
Roller hockey											34	19	19	148		71		
Total hockey		354	722	728	330	151	514	1053	971	946	922	826	1034	756	1614	431	1573	689
Golf			522	446	491	670	1263	927	1320	969	849	556	1076	636	827	731	2734	1079
Soccer	384	1577	852	1009	892	1181	1492	1108	1469	1319	1330	1213	1407	864	1325	1115	3800	2153
Football	569	1709	2081	1864	2090	2310	1672	2930	2105	2197	2318	2412	1470	1776	1464	1293	4349	2137
Lacrosse			119	51	81	77	308			146	165	51	34		76	95	177	13
Volleyball	401	81	428	578	172	378	514	660	785	563	550	470	197	1266	322	557	1033	655
Rugby					45		284	17	17	34	192	45	102	106	38	94	108	38
Other ball sports	216	337	388	243	172	364	266	432	395	257	126	180	180	402	237	254	851	460
Ball sports	1135	1524	1495	1788	1118	1255	545	1359	1141	1492	1073	726	913	912	1033	1206	2750	1130
Tetherball			81			45		79	43					75	87	5	152	
Swimming		1313	1461	1400	1440	977	1013	1377	2056	1669	1333	2440	848	1498	1534	1509	4833	2414
Scuba diving				102				76	17		103	43	112	89	19		68	0
Swimming pools			689	1030	1126	1114	941	1313	1338	1346	1518	1058	1148	820	1853	1557	3181	1690
Swimming pool equipmrent					126			112			189		43	21	119	67	70	17
Surfing				81	69						77	88	43	66	76	244	17	
Water skiing			183	204	188	285	224	277	205	155	189	129	224				34	17
Water polo		102					112	43	282	17		34	112	19		210	202	167
Personal watercraft, powered								112				146				308		
Diving																	253	165
Total water sports		102	2333	2817	2949	2376	2290	3310	3898	3187	3409	3938	2530	2513	3601	3895	8658	4453
Boxing			77	91	137		34	86	112	43	77	111	303	38	50	36	337	232
Wrestling			286	140	102	229	69	236	286	86	149	167	205	131	50	402	618	460
Skateboards			115	122	102	45	222				45	102	17		169	23	23	
Archery			81	207	102	132		241	158	304	64	355	421	1235	38	156	148	23
Horseback riding					102	140	408	301	308	17	310	293	168	267	537	507	73	55
Sleds			192	69	136	118	86	112	115	283	208	447	335	24	303	161	257	137
Snowmobiles			133	354	134	102	170	40	282	422	115	157	224	369	75	201	383	134
Snow skiing		204	454	45	210	295	336	490	624	275	429	591	500	315	154	143	447	239
Trampolines						38	86	0	191	259	262	391	51	107	295	161	447	239
Mountain climbing							86	43	386	146	45	301	112				552	258
Fencing																18		
All terrain vehicles		468	859	256	234	358	412	723	783	548	*59100	281	680	411	639	735	1591	460
Fireworks			1970	1673	1341	1332	1632	1542	2437	2933	2367	2406	1782	1526	1820	1938	5362	2330
Stretch cords or straps											554	323	894	788	458	735	2393	1137
Exercise equipment																103	591	410

* 1994 adjusted figure based on special study of all terrain vehicle injuries. Probably reflects total injuries, not only eye, injuries.

Appendix 2. Canadian Ophthalmological Society Survey Reported Eye Injuries in Canadian Sports, 1972-2002

	1972-3	1974-5	1976-7	1977-8	1978-9	1979-80	1980-1	1981-2	1982-3	1983-4	1984-5	1985-6	1986-7	1987-8
Hockey	287 (20)	258 (43)	90 (12)	52 (8)	4 (13)	85 (21)	68 (20)	119 (18)	115(13)	124(12)	121(18)	123(22)	93(18)	62(11)
Racket Sports			43 (3)	12 (1)	28 (1)	58 (1)	103 (4)	100 (3)	88(5)	115(6)	81(6)	83(1)	66(3)	45(4)
Baseball			19 (2)	2	2	10	15	41 (5)	68(3)	56(3)	43(2)	32(3)	34	16
Ball hockey			24 (3)	8	9 (2)	27 (2)	22 (4)	10 (2)	19(3)	25(2)	29(1)	28	18(1)	31
Football			13 (1)	2	3	1	8	4	27(1)	22(1)	15(1)	10	20(1)	12(1)
War Games											26(14)	8(2)	9(1)	2
Golf			5 (1)		1	1		5 (4)	7(2)	3(2)	4(1)	5(2)	4(1)	1
Basketball				1			2	4	3	5	1	2	7	
Skiing			1 (1)	1	3 (2)			3 (2)		2(1)	4	2(1)	6(1)	1
Volleyball			3 (3)			6	3	2	3	4(1)		2		
Broomball			2 (2)			2	2	3	3	2		1	4	3
Lacrosse			3	1	1				3	4	1	1		
Hunting & BB Guns							4 (1)			1	5(2)	1		
Snowmobile			2 (1)		1			1 (1)		1	1	2(1)	1(1)	1(1)
Other			6		1	7	3	6 (1)	6(1)	9(2)	19(2)	7(1)	5(1)	9(3)
TOTAL	287 (20)	258 (43)	211 (29)	79 (9)	92 (18)	197 (24)	230 (29)	298 (36)	342(28)	373(30)	350(47)	307(33)	267(28)	183(20)

	1988-9	1989-90	1990-1	1991-2	1992-3	1993-4	1994-5	1995-6	1996-7	1997-8	1998-9	1999-00	2000-01	2001-02	TOTAL
Hockey	37(6)	33(6)	21(3)	28(7)	32(5)	16(7)	19(5)	22(7)	12(3)	12(4)	8(5)	13(1)	6(1)	4(2)	1914 (311)
Racket Sports	62(3)	40(2)	35(1)	33	31(1)	27	14(1)	17	18(1)	12	5	11	7	1	1135 (47)
Baseball	24(2)	15(1)	14(1)	18(1)	17(1)	23(2)	17(2)	11(2)	13(1)	10	5(1)	4	4(1)		513 (33)
Ball Hockey	24(1)	14(2)	20(2)	12(2)	16(2)	11(3)	7	4	13(1)	7	8(2)	5	6		397 (35)
Football	10(1)	8	6	6	3(1)	5(1)	4	2	8	2	5	1	1	4(1)	202 (10)
War Games	6 4)	4(2)	4	3(2)	1(1)	3(3)	2(1)	3(1)	4(1)	1	1(1)	1		2	80 (33)
Golf	3	5(1)	4	4(1)	1(1)	2	1	1	4(3)	3(1)	1				65 (20)
Basketball	2	2	2	2	4 (1)	5	1	2	3	3(1)			1		52 (2)
Skiing		4	3 (1)	1	2		3(1)	1	2(1)	1			1		41 (11)
Volleyball	3		1						1		1				29 (4)
Broomball	1	2													25 (2)
Lacrosse		1	2		1			1			1				20
Hunting & BB Guns	3(1)		1	1					3			1(1)			20 (5)
Snowmobile		1								4(1)		2			17 (6)
Other	7(1)	6(2)	5	8(1)	2	3		2(1)	4	9	1	2(1)	1		128 (17)
TOTAL	182(19)	135(16)	118(8)	116(14)	110(13)	95(16)	68(10)	66(11)	85(11)	75(7)	36(9)	40(3)	27(2)	11 (3)	4638 (535)

NUMBERS IN PARENTHESES ( ) ARE BLIND EYES

Hockey: Face masks introduced 1975, note decrease in hockey eye injuries after then. Of the 311 blind eyes in ice ockey playe,: 0 were to players wearing CSA certified full face protectors, and 9 were to players wearing visors.

Injury patterns among female field hockey players

KAREN MURTAUGH

Faculty of Health Sciences, The University of Western Ontario, London, ON, N6A 3K7, CANADA

ABSTRACT

MURTAUGH, K. Injury patterns among female field hockey players. *Med. Sci. Sports Exerc.*, Vol. 33, No. 2, 2001, pp. 201–207. **Purpose:** To examine injury patterns among female field hockey players and to broaden the current base of knowledge by identifying the injury rates of different playing positions. It was hypothesized that goalkeepers would have the highest rate of injury, followed by forwards. **Methods:** High school, university, and national level female field hockey players (N = 158) completed an anonymous questionnaire. They reported personal characteristics (age, height, weight); field hockey information (level, years of experience, surface); injury history (type, site, cause, severity); and back pain information. Injury rates were calculated per athlete-year. **Results:** The most frequently injured site of the body was the lower limb (51%), followed by the head/face (34%), upper limb (14%), and torso (1%). The most prevalent types of injuries were ankle sprains, followed by hand fractures and head/face injuries. Goalkeepers had the highest rate of injury (0.58 injuries/athlete-year), whereas midfielders were the most injured field players (0.36 injuries/athlete-year). Back pain was reported by 59% of the sample, and the lower back was the most common site of this pain. **Conclusion:** There are differences in the rates of injury among playing positions in field hockey and in the types of acute injury sustained at each position. The high number of injuries to the head and face region is also cause for concern. Although most of these injuries are minor, the serious injuries that do occur can be very severe. Now that these patterns have been identified, further examination of the playing situations that lead to injury should be undertaken. **Key Words:** SPORT, TRAUMA, ACUTE INJURY, EPIDEMIOLOGY

Field hockey is one of the most popular team sports in the world, second only to soccer. In fact, the International Hockey Federation consists of five continental associations and 119 member associations. In North America, the highest level of participation is among women. In addition to junior and senior national programs, there are 240 colleges involved in the NCAA (U.S. National Collegiate Athletics Association) and 17 universities compete in the C.I.A.U. (Canadian Intercollegiate Athletics Union). These statistics barely reflect the extensive high school and club participation, which provide a feeder system for higher levels of play.

Despite the sport's apparent popularity, the data on injury rates among field hockey players are limited. The majority of the injuries reported are minor ankle sprains and contusions. More serious injuries, such as torn knee ligaments, concussions, and eye trauma leading to blindness, have also been noted (1,4,6,21). Because these studies tend to focus on elite level athletes, it has been difficult to generalize their results to the field hockey community as a whole. Some authors suggest that increased skill, conditioning and a smooth playing surface decrease the risk of injury (6,17,21,23). Others indicate that elite players not only have an increased "exposure time," but they may be prepared to take more risks (1,12).

Most of the research on field hockey was completed over 10 yr ago, and yet the game has undergone significant changes since that time. For example, since the 1976 Olympic Games, more athletes are practicing and playing on artificial turf than ever before. Also, recent advances in stick construction such as shape and new reinforcement materials allow players to manipulate the ball with more power and accuracy. Finally, there have been numerous rule changes. The offside rule has been eliminated, and alterations to the substitution guidelines allow the prompt and frequent interchange of players. Some players and coaches believe that the removal of the offside rule leads to more players around the goal area and an increased risk of injury. These changes were initiated to promote fast-paced, continuous play and have significantly decreased recovery periods for every player except the goalkeeper (2).

The role of position has received little or no attention in previous field hockey injury reports. The NCAA records the position played at the time of injury (18). However, they do not report the number of athletes who play each position or the type of injury sustained. The remaining information is based on anecdotal evidence: "Which positional role is at greatest risk is difficult to establish. However, play is most

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FIGURE 1—Many players crowd the goal area while the goalkeeper is in a vulnerable position during a high school game (photograph copyright of S. Spaulding, 1999).

intense in and near the striking circle. The goalkeeper, although normally wearing protective equipment, is called upon to come into deliberate physical contact with the ball and must therefore play a high-risk role, as do attackers who rush toward him to retrieve rebounds. Such is the nature of the game that all players are vulnerable most of the time and are involved in most of the high risk situations that arise" (6). It is not uncommon for players to crowd the goal area (Fig. 1). A more recent comment by Spedding (23) suggested that the modern game was safer for most players, except for goalkeepers who had started to lie horizontally across the goal when defending penalty corners. Moore (17) also supports the contention that goalkeepers are at the highest risk of injury, because lofted shots and rebounds off sticks are common in the goal area.

Another concern of field hockey players is back pain. A questionnaire administered by Reilly and Seaton (20) (81% response rate) revealed that 53% of field hockey players have experienced back pain at some time. There has been an increase in recent reports of low back pain of unknown origin in female players (5). Most therapists seem to classify these occurrences as overuse injuries. Field hockey is a game with an inbuilt asymmetry. All sticks are right-handed and approximately 0.91 m long. The rules require that only one side of the stick be used during play. Therefore, most ball handling and defensive skills demand a combination of trunk flexion and rotation. This semicrouched position causes greater spinal loading than normal locomotion and is thought to be ergonomically unsound (20). Previous research on back pain in field hockey players has focused on this semicrouched position as the underlying cause of injury and pain.

The purpose of this study was to examine injury patterns in women field hockey players and broaden the current base of knowledge by identifying the injury rates among different positions. Based on the available accounts, it was hypothesized that goalkeepers and forwards would have the highest rate of injury. An understanding of these patterns is crucial for developing conditioning and training programs that could be useful for injury prevention. This information could also be used to address the potential need for rule changes or additional protective equipment.

MATERIALS AND METHODS

A cross-sectional design was chosen as the most efficient method for reaching the largest and most diverse sample of female field hockey players. The major limitations of this strategy were that it relied heavily on the athletes' abilities to accurately recall their injuries and it was not possible to calculate the number of injuries per athlete exposure. For example, clinical details may not be reliable and there is a tendency for adverse events to be recalled as more recent than they actually occurred (26). However, the cross-sectional design was suitable for a descriptive study of the prevalence of injury and the relationships between injury and other factors (such as playing position) in a defined population. It was also not as subject to yearly fluctuations as the case series method (26). Focusing on acute injuries minimized the effects of recall bias. Subjects were only asked to report if they had sustained a particular injury or not. The exact timing of this injury or the severity of symptoms associated with the injury was not required.

Approval was obtained for this study from the University of Western Ontario Review Board for Nonmedical Research Involving Human Subjects. Coaches were contacted at various provincial and intercollegiate tournaments, or by correspondence, for permission to enroll their teams in the study. All of the teams and players contacted agreed to participate. The sample consisted of 161 female field hockey players. Each player signed a consent form before filling out the questionnaire. Incomplete forms (three) had to be discarded, reducing the sample size to 158. The mean age of players in the final sample was 19.8 yr (range 14–32 yr) with a mean of 6.8 ($\pm$ 3.5) yr of playing experience. Among these athletes, there were 30 with only high school experience, 23 intermediate level players (club and/or provincial experience), 75 varsity athletes, and 30 national level players.

The anonymous questionnaire was designed for quick and easy use and took approximately 15 min to complete. It included personal information such as age, height, weight, position, and playing experience. The questionnaire also inquired about injury history and back pain. The athletes reported the anatomic site of injury, number of occurrences, if medical treatment was sought, and the cause of the injury (ball, stick, player contact, or playing surface contact). Respondents were then asked whether the injury affected their performance or caused them to miss field hockey games and/or practices.

Personal information, back pain data, and the acute injuries that had occurred during a field hockey game or practice were included in the subsequent analysis. These restrictions accounted for any acute physical damage, such as contact injuries from playing equipment, another player, or the playing surface and noncontact injuries, such as muscle strains and ligament sprains. Chronic injuries due to prolonged strain, such as stress fractures, shin splints, and compartment syndrome, were not considered in this study.

The data were entered into a Microsoft Access™ database. To determine whether the differences between se-

lected variables were significant, a multiple factor analysis of variance (ANOVA) was used. A *P*-value of less than 0.05 was selected for rejecting the null hypothesis. The Student-Newman-Keuls multiple comparison *post hoc* test was performed to identify which of the variables were significantly different from one another.

The athletes were grouped according to playing position and whether they had sustained an acute injury. To determine the risk of injury to these different groups, injury rate was defined as: (no. of injuries)/((yr of playing experience) $\times$ (no. of athletes in the group)). This calculation resulted in the number of injuries per athlete-year. The sample included both injured and noninjured athletes; therefore, this method indicates the risk of injury to all players involved in the activity.

RESULTS

Of the 158 athletes surveyed, 49% reported that they regularly trained and played on artificial turf, 21% participated on grass, and 30% indicated that they used both surfaces equally. At least one acute injury that had occurred during a field hockey game or practice was reported by 74.7% of these athletes. The injured athletes had an average age of 20.4 ($\pm$3.2) yr and 7.4 ($\pm$3.3) yr of experience. The uninjured athletes ($N = 40$) were significantly younger and had less experience with an average age of 18.0 ($\pm$2.6) yr and 5.4 ($\pm$3.5) yr of experience ($P < 0.05$).

Approximately half of the 469 injuries that occurred were to the lower limb. The next most frequent site of injury was the head/face area, followed by upper limb and torso (Fig. 2). The most common type of injury was a ligament sprain (39.7% of total injuries) and most of these were ankle sprains (Fig. 3). Because these sprains were self-reported, the severity could not be assessed. Other prevalent injuries were contusions and fractures (17.1% and 16.4%, respectively). Most contusions occurred to the head/face region, whereas most of the fractures involved bones of the wrist or hand. Of the remaining 26.9% of the injuries, 9.4% were wounds, 8.1% were muscle strains, 7.7% were concussions, and, finally, 1.7% were dislocations.

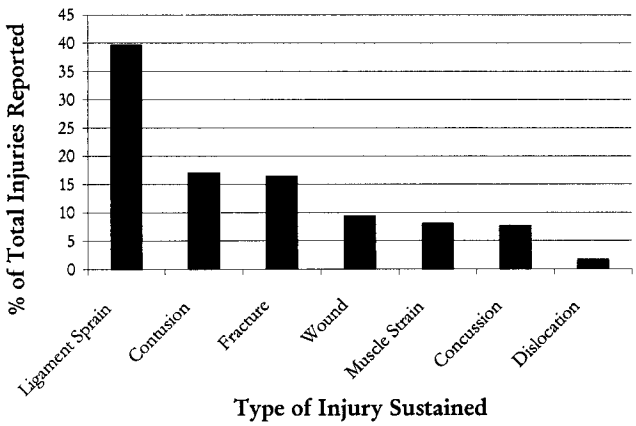

FIGURE 3—Types of acute injuries sustained by female field hockey players during a game or practice.

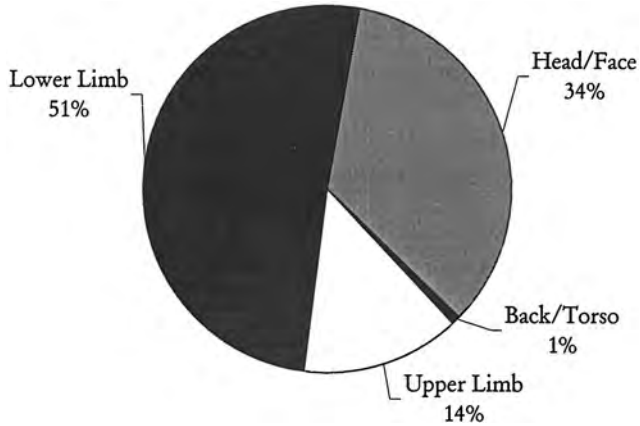

FIGURE 2—Anatomical site of acute injuries to female field hockey players that occurred during a field hockey game or practice.

The percentage of athletes who had sustained at least one injury to the head or face region was 42%. These respondents were asked whether the injury was caused by a field hockey ball, stick, player, or contact with the playing surface (Fig. 4). The most commonly reported cause was the ball, next was the stick or another player. Very few injuries to this region of the body were the result of contact with the ground (4%). This pattern varied depending on the type of head/face injury. Most of the concussions (47%) were caused by collisions with another player, whereas a stick caused most of the wounds (68%). Finally, a ball caused 60% of the black eyes.

Dividing the sample according to the position played resulted in the formation of the following five groups: goalkeepers ($N = 11$), backfield players ($N = 44$), midfielders ($N = 27$), and forwards ($N = 46$). Those who reported that they played more than one position were placed in a separate group (multiple, $N = 30$). Table 1 lists some characteristics of these groups including age, years of experience, height, and mass. The goalkeepers were the most experienced group (8.1 $\pm$ 4.0 yr), whereas the forwards had played for the least amount of time (6.3 $\pm$ 2.9 yr).

An indication of the number of injuries for each region of the body categorized according to the position played is

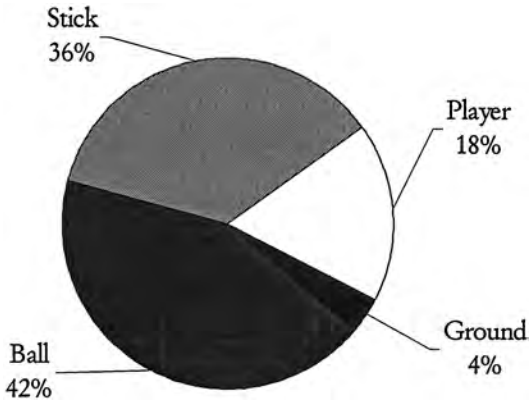

FIGURE 4—Cause of reported head or face injuries to female field hockey players that occurred during a field hockey game or practice.

TABLE 1. Personal characteristics of the female field hockey players in the sample, categorized according to the position played.

	Goal (*N* = 11)	Backfield (*N* = 44)	Midfield (*N* = 27)	Forward (*N* = 46)	Multiple (*N* = 30)	Total (*N* = 158)
Age (yr)	20.4 (3.8)	19.3 (2.9)	20.0 (3.6)	19.6 (3.0)	20.4 (3.7)	19.8 (3.25)
Experience (yr)	8.1 (4.0)	6.7 (3.6)	6.8 (2.9)	6.3 (2.9)	7.4 (4.3)	6.8 (3.5)
Height (m)	1.68 (0.05)	1.64 (0.06)	1.65 (0.05)	1.63 (0.09)	1.64 (0.07)	1.64 (0.07)
Mass (kg)	65.3 (13.0)	61.7 (9.7)	61.1 (6.1)	60.3 (5.9)	60.8 (5.8)	61.3 (7.8)

Standard deviations are in parentheses next to each mean value.

presented in Table 2. To determine the relative occurrence or risk of injury to each group, further analysis was necessary. The number of injuries per athlete-year is presented in Fig. 5. The overall rate of injury was 0.44 injuries per athlete-year. This rate included 0.22 injuries/athlete-year to the lower limb, 0.15 to the head face, 0.06 to the upper limb, and 0.005 injuries/athlete-year to the back or torso.

Overall, goalkeepers had the highest risk of injury (0.58 injuries/athlete-year), including the highest rate of upper limb (0.12 injuries/athlete-year) and back/torso injury (0.03 injuries/athlete-year). In fact, they had a rate of back and torso injury 16.7 times higher than the rate calculated for field players. The most common injuries to goalkeepers were (in order of the number of occurrences reported): concussions, wrist fractures, ankle sprains, thigh muscle strains, and knee ligament tears. The majority of these injuries occurred due to contact with other players, the playing surface, or the goal itself.

Those who played multiple positions had the highest total injury rate of the field players (0.51 injuries/athlete-year). This group also had the highest rate of lower limb injury with 0.27 injuries/athlete-year. The most common injuries were ankle sprains, contusions to the head/face region, and finger fractures.

The midfielders had the highest injury rate of the field players who played only one position on a regular basis (0.36 injuries/athlete-year). The midfielders also presented the highest rate of injuries to the head/face region (0.21 injuries/athlete-year) and the most injuries to the upper limb of all field players. The most common injuries to this group (ankle sprains, wounds, or contusions to the head/face and finger fractures) were the same as those reported by athletes who played multiple positions.

Forwards and backfield players had similar rates of injury (0.37 and 0.36 injuries/athlete-year, respectively). The top two injuries for these groups (ankle sprains and black eyes) were also the same. However, there were also notable differences between the two positions. For instance, backfield players had a higher rate of lower limb injury (0.25 vs 0.18 injuries/athlete-year). The next two most common injuries to backfield players were finger fractures and knee ligament tears, whereas forwards had more concussions and lower limb muscle strains. Finally, when compared with backfield players, forwards had a higher rate of injury to the head and face by a factor of 2.1.

Even though back pain is not necessarily related to acute injury, it appeared to be a common complaint among the players. Therefore, they were also asked some standard questions relating to back injury and pain (Table 3). Although 59% indicated that they had experienced pain at some time, only 50% reported that back pain affected them during the field hockey season. The pain was serious enough to cause 12% of athletes to miss a field hockey game or time at school or work. The lower back was the most common

TABLE 2. The number of specific traumatic injuries sustained by female field hockey players (*N* represents the number of players surveyed at each position).

	Goal (*N* = 11)	Backfield (*N* = 44)	Midfield (*N* = 27)	Forward (*N* = 46)	Multiple (*N* = 30)	Total (*N* = 158)
Head/face						
Bruise/black eye	4	11	16	21	18	70
Wound	1	3	16	9	11	40
Concussion	12	5	2	10	7	36
Broken nose	1	2	3	4	4	14
Dislocation	0	0	1	0	0	1
Total head/face	18	21	38	44	40	161
Upper limb						
Fracture	9	7	10	8	11	45
Ligament sprain	1	3	2	2	1	9
Dislocation	0	0	2	2	1	5
Bruise	1	1	0	1	0	3
Wound	0	0	1	0	1	2
Total upper limb	11	11	15	13	14	64
Torso						
Fracture	3	0	0	0	0	3
Muscle strain	0	1	0	0	1	2
Total torso	3	1	0	0	1	5
Lower limb						
Ankle sprain	6	57	21	31	43	158
Muscle strain	6	3	7	13	9	38
Knee injury	5	6	4	1	3	19
Fracture	2	5	2	4	2	15
Bruise	1	1	1	2	2	7
Wound	0	1	0	0	1	2
Total lower limb	20	73	35	51	60	239

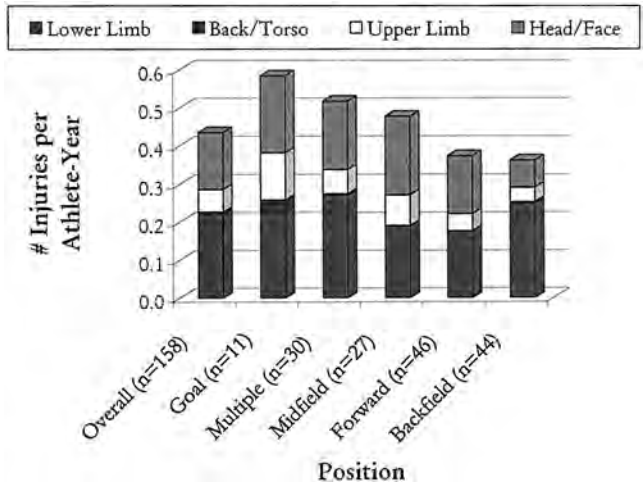

FIGURE 5—The rate of acute injuries to female field hockey players according to the site of injury and playing position.

site of pain followed by the upper back, neck, tailbone, and ribs.

DISCUSSION

The general pattern of field hockey injuries presented in this study supports the findings of other authors. The most frequent site of injury was the lower limb, followed by the head and face, upper limb, and finally the torso (8,18,19,21). An ankle sprain was the most common single injury. This result is not unique to field hockey as the lateral ankle is the most frequently injured area in the body, and, overall, an ankle sprain is the most common injury in sport (7,25). Whether the mechanism of injury in field hockey is the same as in other field sports, such as soccer, has yet to be determined. Fox (6) suggests that the stooped position used when dribbling the ball may be an unsound position for fast locomotion and could contribute to lower limb injury.

It is interesting to note that studies conducted before 1980 have reported lower incidences of upper limb injuries than more recent reports. Graham and Bruce (8) surveyed intercollegiate athletic injuries during the 1974–75 school year in Virginia and found 10.6% of total injuries to the upper limb, and Rose (21) reported 4.9% at the California State University in Long Beach from 1976 to 1979. Recent surveys of collegiate and high school field hockey (including the current study) indicate that 14.0% to 15.8% of the total injuries are to the upper limb and most of these are wrist or finger fractures (18,19). It is possible that this difference is due to changes in the game that have occurred over the past two decades. For example, improved stick construction allows players to hit the ball with greater velocity and different techniques for stopping the ball on artificial turf may have led to an increased risk of upper limb injury.

TABLE 3. Reports of back pain in female field hockey players.

	Players Reporting
Experience of pain	
At any time	59%
During season	50%
Injury	31%
Effects of pain	
Affects performance	24%
Missed games	8%
Missed practice	15%
Missed school or work	7%
Site of pain	
Neck	15%
Upper back	19%
Ribs	6%
Lower back	54%
Tail bone	12%

The second most frequent site of injury in field hockey is the head and face region. The percentage of athletes who had sustained at least one injury to this area was 42%, and the uninjured players tended to be younger and have less experience than injured players. Because only active players were surveyed, these findings do not include those who may have stopped playing due to serious or catastrophic injury. It appears that if an athletes play long enough, they are likely to experience a head or face injury. This statement is supported by a survey conducted on international athletes (1). It was found that 62% of these elite level athletes had a facial injury at least once, and the high value was attributed to their increased exposure time and tendency to take more risks.

Although no catastrophic injuries were reported in this study, the high rate of injury to the head and face region indicates that the potential for such injury still exists. Elliott and Jones (4) reported three penetrating eye injuries to female field hockey players in one season. All of these injuries were caused by a stick, as were 36% of the head and face injuries in this study. Findings such as these have led to the suggestion that a helmet with a full visor be required in field hockey. Yet, this study found that goalkeepers, who are already wearing helmets, had the highest rate of concussions. If protective equipment is deemed necessary, the impact of equipment changes on potential injuries, as well as the character and nature of the game, must be carefully considered (10,11).

With the above concerns in mind, a more immediate approach to reducing severe injury would include education about the risks of head and eye injury and strict enforcement of the current rules. For example, a recent survey indicated that parents saw a need for the mandatory use of mouthguards in field hockey (3). Mouth protectors have been shown to decrease concussions, cerebral hemorrhages, unconsciousness, jaw fractures, and lacerations of the lips and cheeks (9,16). Despite these benefits and widespread regulations enforcing the use of mouthguards, many players still do not wear them (1). The most common reason that was cited for not wearing the protector was discomfort. Also, most of the players who wore the mouthguard did so because they had already sustained an injury.

Rules governing the use of the stick and acceptable ways of playing the ball are in place to reduce dangerous play during a match. Yet, the NCAA reports that only 16.3% of total injuries in the 1998–99 season were due to illegal action (18). Clearly, most injuries occur during play that is in accordance with the current rules. This risk has severe implications for children and young players, who need to develop basic skills before progressing to a more competitive level of

FIGURE 6—The player in the white shirt chases down a player with the ball. Note that great care must be taken to avoid the stick should the offensive player decide to drive the ball (photograph copyright of J. Mahannah, 1999).

play. Modified rules and a progressive introduction to field hockey for children could reduce injuries in these players. Umpires and coaches should also be continuously trained so that there is consistent skill development and interpretation of rules to minimize unsafe play (22).

The finding that goalkeepers had the highest injury rate per athlete-year supports suggestions that goalkeepers are at the highest risk of injury in field hockey (6,17,23). However, most injuries did not occur due to contact with the ball, as proposed, but due to contact with another player, the ground, or the goal itself.

It has also been implied that the most injured field players would be forwards or defenders, who spend more time near the goal area (6). So, it was surprising to find that midfielders actually had the highest rate of injury, including the highest rate of injury to the head/face region and to the upper limb. It is possible that these injuries were due to greater fatigue or the type of defense that midfielders tend play during the game. Four positions were tested during match play using a heart rate monitor (2). It was found that the center half and inside forwards spent more time at a higher physical intensity than other positions (these positions are closest to the definition of midfielders used in this study). An increased level of physical intensity could lead to errors in play and possibly injury. Midfielders also execute most defensive skills in a recovery mode (Fig. 6). When making a chase tackle from the rear of a player about to strike a ball, great care is needed to avoid the backward swing of the stick. Otherwise, severe stick-inflicted injuries could occur. These include injuries to the nose, face, and teeth (6). In fact, up to 20% of field hockey injuries have been attributed to "tackling" (8).

One might expect to find similar rates of injury among forwards and backs as was reported in this study. Players in both positions tend to spend most of their time in the same area of the field. One attempts to score a goal and the other defends. Some coaches and players have suggested that the elimination of the offside rule has not necessarily increased the pace of the game or goal scoring. Instead, many teams have adopted a strategy of filling the striking circle with as many defenders as possible. This tactic may increase the risk of injury to forwards and defenders as more players crowd the goal area. To determine why different types of injuries occur to each player, further investigation into the mechanism of injury and the playing situation in which these injuries occur is required.

Back pain continues to be a common complaint of field hockey players. It was found that 59% of athletes have experienced back pain and the most common site of pain is the lower back. These results are similar to previous reports (20). Some authors have attempted to attribute low back pain to stresses on the spine unique to field hockey. For example, it has been shown that 7 min of dribbling a field hockey ball can cause an average spinal shrinkage of 2.73 mm (20). Yet, no obvious relationship between lumbar mobility and low back pain in field hockey players has been demonstrated (15). However, players without pain may be stronger in peak eccentric extension strength than players who experience pain (5).

It may seem as if 59% of athletes reporting back pain is a high proportion in such a young and active group. However, other sources have indicated that back pain is very prevalent in the general population (60–80%) and among athletes (50–85%) (13,24). Furthermore, by the age of 18, over 50% of girls have experienced at least one episode of low back pain (14). Thus, the possibility emerges that the pain reported by field hockey players cannot be directly attributed to the sport, but further research would be required to support or refute such a suggestion.

CONCLUSIONS

This study demonstrated that the most prevalent injuries in field hockey are ankle sprains, hand fractures, and head or face injuries. It also revealed that, not only is there a difference in the rate of injury between playing positions, but also in the types of injuries sustained at different positions. Most of these injuries are not severe. In fact, field hockey tends to have low serious injury rates when compared with other sports. In the NCAA, field hockey was fourth among the women's sports covered, behind gymnastics, soccer, and basketball (18). Of five popular high-school sports played by girls (including basketball, field hockey, soccer, softball, and volleyball), field hockey had the lowest number of injuries requiring surgery or more than 7 d of time lost (19). Yet, the nature of severe injuries in field hockey and the high rate of head/face injury indicate a serious risk. Now that these patterns have been identified, further examination of the playing situations that lead to injury should be undertaken. It seems that reducing these types of injuries will

require a multifaceted approach. This approach must include the education of players, coaches, and officials along with considering the development of rule changes and sport-specific protective equipment.

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Conclusions drawn from or recommendations based on the data provided by the National Collegiate Athletic Association are those of the author based on analyses/evaluations of the author and do not represent the views of the officers, staff, or membership of the NCAA.

Address for correspondence: K. Murtaugh, 2142 Thames Hall, The University of Western Ontario, London, Ontario, N6A 3K7, Canada; E-mail: murtaugh@julian.uwo.ca.

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SPORT-SPECIFIC ILLNESS AND INJURY

Field Hockey Injuries

Karen Murtaugh

Levy Elliott Sports Medicine Clinic, Burlington, Ontario, Canada, and Escarpment Sports Medicine Centre, Milton, Ontario, Canada

MURTAUGH, K. Field hockey injuries. *Curr. Sport Med. Rep.*, Vol. 8, No. 5, pp. 267–272, 2009. *Field hockey is a popular sport that is played throughout the world. Most of the literature on the sport has focused on describing injury patterns. This research reveals that most injuries are minor and that the most common injury is an ankle sprain. Studies also suggest that men have a higher rate of injury and that they experience severe injuries more often than women. These severe injuries include trauma to the head, face, and upper limb and usually are the result of contact with the stick or ball. Consequently, many authors suggest that all players wear face and hand protection. Current International Field Hockey Federation rules recommend minimal protective equipment (e.g., mouthguard, shin, and ankle guards), and surveys indicate that many players do not wear mouthguards regularly. Looking into the future, research should focus on developing and evaluating effective strategies for injury prevention.*

INTRODUCTION

Field hockey is played by men and women, at recreational and highly competitive levels, in 122 countries (13). In North America, there are over 5000 women competing in collegiate field hockey (8) and roughly 35,000 players registered with their national associations (12,47). Despite the sport's popularity, there are few publications that focus on field hockey injuries, and to the author's knowledge, there are no studies that critically evaluate any strategies for the prevention of injury or the effectiveness of protective equipment. However, there has been a renewed interest in the serious injuries that occur in this sport, and researchers are using more widely accepted injury definitions and standardized methods for reporting injury rates. The goal of this article is to put new information into context with what is known already about field hockey injuries. It also highlights important trends in injury patterns and suggests how the sport should develop evidence-based methods for injury prevention.

RATES AND TYPES OF INJURIES

Up to 75% of field hockey players have sustained at least one acute injury during a game or practice (35). When field hockey is compared with other team sports, such as basketball and soccer, the overall rate of injury is similar (26,39,41). Tables 1 and 2 summarize the results of studies that have reported on injury patterns in field hockey. It is difficult to compare these studies because injury definitions are varied and players from different age groups or competition levels rarely are included in the same sample. The lower limb is the most often injured site, and, like other sports, most of these injuries are ankle sprains (34,36). Beynnon et al. studied a cohort of female high school and college field hockey players (N = 138) and reported a first-time ankle injury rate of 0.9 (95% confidence interval (CI) = 0.4–1.9) injuries·1000 person-days^{-1}. This rate was lower than basketball players but not significantly different from soccer or lacrosse (2). Powell and Barber-Foss reported that only 1.2% of field hockey injuries required surgery when they analyzed the 1995–1997 National Athletic Trainers' Association High School Injury Surveillance database (41). However, a more recent analysis of the same database showed that field hockey players had the highest risk of subsequent injury (relative risk = 2.8, 95% CI = 2.3–3.4) (42). In particular, there was a significantly higher proportion of hamstring strains and anterior cruciate ligament reinjuries than new injuries (P = 0.04). This report is consistent with earlier findings that 10.5% of the acute injuries in school children are reinjuries (41). This proportion rises to 27% in recreational players and 44% in elite athletes (19,20).

The next most common site of injury is the upper limb (Table 2). It has been suspected for some time that field hockey has a high rate of upper limb injury compared with other sports (35). A recent study confirms this suspicion. Bowers et al. analyzed the NCAA Injury Surveillance System (ISS) database for injuries to the hand and phalanx in stick-handling athletes from 1986 to 2002 (4). The sports studied

Address for correspondence: Karen Murtaugh, M.Sc., M.D., CCFP, Dip. Sport Med. 474 Willard Avenue, Toronto, ON, Canada M6S 3R6 (E-mail: k_murtaugh@hotmail.com).

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TABLE 1. Types of acute injury as a percentage of the total injuries in men's and women's field hockey.

Study	Population	Sample (*N*)	Sprain	Strain	Contusion	Fracture	Wound/ Laceration	Concussion	Other/Not Reported
WOMEN									
Powell and Barber-Foss (1999) (41)	High school	442	**25.5**	20.2	–	5.9	–	–	48.4
Murtaugh (2001) (35)	High school, college, and elite	158	**39.7**	8.1	17.1	16.4	9.4	7.7	1.6
Rose (1981) (45)	College	N/A	**32.1**	18.5	**32.1**	1.2	6.2	3.7	6.2
Dick *et al.* (2007) (8) -Game	College	N/A	**23.9**	16.9	19.8	15.4	11.2	9.4	3.4
-Practice			22.8	**57.9**	–	4.5	–	3.4	11.4
Lindgren and Maguire (1985) (29)	Elite	10	19.5	**24.4**	22.0	2.4	2.4	–	29.3
Freke and Dalgleish (1994a) (16)	Elite	40	24.0	**32.0**	3.0	11.0	–	–	30.0
Freke and Dalgleish (1994c) (18)	Elite	62	8.0	**32.0**	23.0	3.0	9.0	–	25.0
Junge *et al.* (2006) (26)	Elite	N/A	13.0	–	**37.0**	–	25.0	25.0	–
MEN									
Lindgren and Maguire (1985) (29)	Elite	16	**25.0**	17.5	17.5	2.5	7.5	–	30.0
Junge *et al.* (2006) (26)	Elite	N/A	19.0	8.0	**42.0**	8.0	19.0	–	4.0
BOTH									
Roberts *et al.* (1995) (44)	Recreational	50	23.7	15.3	**26.3**	–	–	–	34.7
Sherker and Cassell (1998) (46)	ED presentations all ages	292	14.7	–	16.4	**32.5**	–	36.4	

The highest value for each study is in bold type (excluding "Other/Not Reported" injuries). ED = emergency department; N/A = not applicable. (Reprinted from Murtaugh K. Field hockey. In: Caine E, Harmer P, Schiff M, editors. *Epidemiology of Injury in Olympic Sports – Encyclopaedia of Sports Medicine* (in press). Copyright © 2009 Blackwell Publishing. Used with permission.)

were women's field hockey, men's ice hockey, men's lacrosse, and women's lacrosse. Field hockey was the only sport that did not require the use of gloves, and the athletes had a significantly higher risk of hand injury (odds ratio [OR] = 2.12, 95% CI = 1.86–2.41).

The types of injuries that occur in this sport are influenced by the playing surface, equipment, and other athletes. High-intensity training on artificial turf may be a major contributing factor in chronic hockey injuries such as exercise-related leg pain (29,43). Falling on this surface also is responsible for the high rate of abrasions (up to 9% of acute injuries) (18). Next, it is known that a large percentage of hand and face injuries are caused by impact from the ball or stick. In fact, Dick *et al.* found that approximately 60% of total game injuries were from stick and ball contact mechanisms (8). There are rules governing control of ball and stick that help to prevent dangerous play (24). However, these rules can be violated intentionally or by accident. In the 1988–89 NCAA field hockey season, 16.3% of total injuries were due to illegal action (38). At the 2004 Olympics, the team physician felt that 8 of 42 injuries were caused by foul play (26). Finally, although deliberate physical contact is not permitted in field hockey, incidental contact seems be contributing to a larger proportion of injuries in recent studies. Almost 20 yr ago, contact was responsible for only 2.2% of injuries to recreational players, but in a 2007 report it caused 13% of U.S. collegiate injuries (8,19). It is unclear whether this difference is caused by reporting methods, changes in the game, or differences in the level of play.

SEVERE INJURIES

The most serious injuries in field hockey are from blows to the head or face from the hockey stick or ball. In 1977, Graham and Bruce (20) found that field hockey had the highest rate of head and face injuries of the nine intercollegiate sports they studied. This trend has continued as a recent survey of female players from the English Hockey Association found that 68% of players had sustained at least one injury to the head or face region (21). In fact, in a study of pediatric sport injuries, the field hockey stick caused more male facial injuries than all other male and female sports combined (49).

Most of these injuries (65%) are mild and consist of lacerations and contusions or hematomas; catastrophic injuries are possible (22,46). However, facial injuries often affect the teeth or eyes, and head injuries can cause ongoing disability. In elite players, more than half of the dental injuries require a visit to a physician and/or dentist (3,10,46,49). Field hockey also had the second highest incidence of dental injury for women at the 1989 Canada Games, accounting for 1.3% of total dental injuries (28). Next, the aggressive nature of the sport, almost universal absence of facial protection, and a stick that permits orbital penetration is a dangerous combination that contributes to the incidence of serious eye injuries (10). Field hockey had the fourth highest incidence of eye injuries among the 16 sports followed by the NCAA Injury Surveillance System from 2000 to 2004, and 11% of all head and facial injuries

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TABLE 2. Site of injury as a percent of the total injuries in men's and women's field hockey.

Study	Population	Sample (*N*)	Head/Face	Upper Limb	Back/Torso	Lower Limb	Other/Not Reported
WOMEN							
Powell and Barber-Foss (1999) (41)	High school	442	16.6	15.8	4.9	**58.8**	3.3
Murtaugh (2001) (35)	High school, college, and elite	158	34.0	14.0	1.0	**51.0**	0.0
Fuller (1990) (19)	Recreational	N/A	10.4	20.0	8.9	**60.7**	0.0
Rose (1981) (45)	College	N/A	11.1	4.9	4.9	**79.0**	0.0
Dick *et al.* (2007) (8) -Game	College	N/A	25.3	20.7	7.1	**43.2**	3.8
-Practice			8.4	8.1	16.2	**60.2**	7.1
Lindgren and Maguire (1985) (29)	Elite	10	1.7	5.0	16.7	**76.7**	0.0
Freke and Dalgleish (1994a) (16)	Elite	40	0.0	11.6	21.1	**65.3**	2.1
Freke and Dalgleish (1994c) (18)	Elite	62	4.3	13.0	23.2	**59.4**	0.0
Junge *et al.* (2006) (26)	Elite	N/A	27.0	20.0	9.0	**47.0**	0.0
MEN							
Lindgren and Maguire (1985) (29)	Elite	16	6.1	8.2	12.2	**69.4**	4.1
Junge *et al.* (2006) (26)	Elite	N/A	22.0	20.0	8.0	**50.0**	0.0
BOTH							
Eggers-Ströder and Hermann (1994) (9)	Recreational	322		19.0		**62.0**	19.0*
Yard and Comstock (2006) (49)	ED presentations (2–18 yr)	1356	28.0	**39.4**	0.0	**28.2**	4.4
Sherker and Cassell (1998) (46)	ED presentations all ages	292	**32.8**	31.7	N/A	**12.7**	22.8

The highest value for each study is in bold type. Eggers-Ströder and Hermann (1994) combine head and trunk injuries. ED = emergency department; N/A = not applicable. (Reprinted from Murtaugh K. Field hockey. In: Caine E, Harmer P, Schiff M, editors. *Epidemiology of Injury in Olympic Sports – Encyclopaedia of Sports Medicine* (in press). Copyright © 2009 Blackwell Publishing. Used with permission.)

also affected the eye (6). Indeed, there are cases of players who lost vision in one eye after they were hit with an opponent's stick (10,37). Finally, despite the noncontact nature of the sport, concussions account for 1.7%–7.7% of total acute injuries (5,8,14,35). In Australia, head injuries were responsible for 5.1% of hockey emergency room presentations, with a 10.5% admission rate (46). Clearly, face and head injuries are common in field hockey, and researchers have documented several cases of significant trauma.

GENDER DIFFERENCES IN INJURY PATTERNS

Most studies of field hockey injury focus solely on women, and this situation limits the analysis of injury rates between genders. However, the available reports suggest that males have a higher rate of injury and that they sustain severe injuries more often than females (14,26). For example, at the 2004 Olympic Games, elite men had 3.2 times the risk of a game injury than elite women (26). Men also were more likely to sustain a time-loss injury in a match (OR = 6.0). Of injured women, 75% did not miss any days of competition, compared with only 52% of injured men (26). Further information comes from an analysis of the Victorian hospital emergency department (ED) database (1996–1997) (46). Men presented to the ED 1.7 times more than women for field hockey injuries (*N* = 292 field hockey-related ED presentations). Note that more men are registered as field hockey players in Australia than women (1.3 men:1 woman). Finally, Yard and Comstock found that pediatric boys had more lacerations, upper limb injuries, and facial injuries than girls (49).

INJURY AND PLAYING POSITION

Information on the types and severity of injuries sustained at different playing positions is sparse. Murtaugh reported that goalkeepers had the highest rate of injury (0.58 injuries·athlete-year^{-1}) and 16.7 times more back and torso injuries than field players (35). The most common injury in goalkeepers was a concussion, and it occurred from contact with other players, the playing surface, or the goal itself. Midfielders had the highest injury rate of field players (0.46 injuries·athlete-year^{-1}) and the highest rate of injuries to the head/face and upper limb of all field players. Finally, forwards and defenders had a similar rate of injury (0.37 and 0.36 injuries·athlete-year^{-1}, respectively). However, defenders had a higher rate of lower limb injury (0.3 vs 0.2 injuries·athlete-year^{-1}), while forwards had 2.1 times more injuries to the head and face. Another study reported weighted percentages of injuries per playing position. Midfielders had the highest percentage of injuries (27.6%),

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followed by defenders (23.6%), forwards (22.4%), and goalkeepers (19.5%) (8). The findings of other authors support these trends (15,33). But a more recent study of elite female athletes found that the forwards had the highest rate of injury (1.30 injuries·player^{-1}·season^{-1}) and that goalkeepers had the lowest rate (0.75 injuries·player^{-1}·season^{-1}) (36). This difference may be a result of different injury definitions. The latter study had the highest threshold for defining an injury by requiring pain and/or inability to play for at least 5 d. Unfortunately, none of the studies evaluated their results for statistical significance.

More research is needed to explain why these differences exist. It has been proposed that midfielders have the highest rate of injury among field players because they are often involved in tackling situations (35). Tackling is an attempt to gain possession of the ball, and it requires that players risk contact from the stick, ball, or each other (15,23). It also has been shown that up to two thirds of injuries occur inside the 25-yd line (8). Again this finding may be caused by tackling, set plays, or increased risk-taking as players attempt to score on goal. Further details would provide insight into the situations that lead to injury.

INJURY PREVENTION

Based on the injury patterns in this sport, the first priority should be promoting the use of protective equipment. A recent metaanalysis indicated that the overall risk of an orofacial injury is 1.6–1.9 times higher if a mouthguard is not worn during a sport with a risk of facial injury (27). Consequently, the American Dental Association and the International Academy of Sports Dentistry recommend the use of mouthguards when playing field hockey (1). Parents agree when surveyed, and they cite field hockey, along with football, boxing, ice hockey, wrestling, and karate, as sports in which mouthguard use ought to be mandatory (7). Despite the benefits, players do not wear mouthguards consistently because of perceived ineffectiveness, discomfort, or interference with breathing (3,21). In fact, they often wait several years into their career before starting to wear one, and most are prepared to play without a mouthguard (21). The current International Field Hockey Federation (FIH) rules state that "field players are *recommended* to wear shin, ankle, and mouth protection" (24). This recommendation should become a formal rule, and it should be enforced. The National Collegiate Athletic Association in the United States already mandates that all field players are required to wear mouthguards at all times. Individual associations should bring this matter to the attention of their National Association and the FIH Rules Board. To improve compliance, umpires at all levels should check athletes before games for mouthguards and enforce penalties for not wearing the proper equipment.

Several authors have suggested that hockey players wear gloves along with eye and/or face protection (6,25,49). In 2007, the FIH rules were modified to allow all players to use a face or head protector during short corners (Fig.). It can be worn during regular field play if there are documented medical concerns (24). The NCAA extended the option of using face protection to all field players at all times. However, there is no known research on the appropriate design or effectiveness of the devices in use. Furthermore, it is not known how the sporadic use of these protectors will alter the nature of the game or perhaps even put surrounding players at risk of injury. Gloves have been used in indoor field hockey for some time, but if athletes are considering the use of head or face protection, it should be the priority of the sporting association to ensure that this equipment is safe.

Figure. An athlete wearing facial and hand protection during a short corner at the 2008 Samsung Champions Trophy in Monchengladbach, Germany. © Copyright International Field Hockey Federation (FIH).

The next step in preventing injury is to reduce recurrent injury. Other sports have developed structured warm-up routines that incorporate specific strength and neuromuscular training. These programs have reduced lower limb injuries in soccer and hand ball (31,40). The evidence is growing to support the effectiveness of using specific exercise programs to prevent the most common injuries in sport. Therefore, field hockey coaches and athletes should consider incorporating proprioceptive and strength exercises into their training regimen.

Recurrent lower limb and back injuries are common in hockey (17,34,42). It is accepted that balance training and ankle bracing reduces the rate of recurrent ankle sprains and that they may reduce first-time injuries (48). Recently, it also was shown that poor peak dorsiflexor torque at the ankle was

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associated with an increased incidence of ankle injuries in elite female field hockey players (36). The authors propose that increased dorsiflexor strength may assist the ankle in preventing the inversion responsible for most lateral ankle sprains. When the ankle is in an inverted and plantar flexed position, the everter and dorsiflexor muscles act eccentrically. Weak dorsiflexors allow excessive movement, placing additional stress on the lateral ligaments of the ankle joints (36). Next, prophylactic core strengthening helps to prevent back injury, and it has had success in reducing back complaints among field hockey players (11,30). The game of hockey requires players to be in a forward flexed and semirotated position for prolonged periods. Many players start their careers as children and are involved in high volumes of training. As a result, many develop asymmetries in posture (30). There is no evidence to show that more flexible field hockey players have a lower incidence of back pain (30). However, one study suggested that pain-free athletes were stronger in peak eccentric lumbar extension (11). The sport would benefit from more research into the effectiveness of specific training programs that are meant to reduce injury.

Finally, it is important to treat injuries in a way that minimizes their negative effects and prevents any related complications. Now that the injury patterns have been identified, sporting bodies can focus on promoting the care of the most common and most severe injuries. At a minimum, medical personnel in attendance at field hockey competitions should be comfortable with concussion management, splinting for hand injuries, tooth preservation, and the application of an eye patch. They also should be aware of local emergency and dental facilities (21). The only catastrophic injuries in field hockey have involved head trauma (10,37). However, a recent Australian study found that treatment of head injury was variable and subjective in nature. The most common management procedure was to remove the player from the sporting field, and unless an ambulance was required, it was considered the player's responsibility to attend a medical facility. The use of evidence-based guidelines for the initial management of head and brain injury and return-to-play decisions after mild traumatic brain injury in sport should be encouraged (32). Because of the high incidence of lacerations and abrasions (Table 1), leagues should consider requiring documentation of up-to-date tetanus prophylaxis before allowing their athletes to participate. Athletes themselves can be taught the basic techniques for wound cleaning and dressing, along with the signs and symptoms that might indicate the onset of infection. Incorporating these standard procedures could reduce the morbidity associated with the most common injuries in field hockey.

The rate of injury is highest in games, and more serious injuries seem to occur at the highest levels (Table 1). Thus it is reasonable to ensure that a physician is in attendance at major tournaments, and FIH already has this recommendation in its advice for team medical personnel. However, it is not realistic to have a physician at all games. When a certified athletic trainer is not available, the responsibility falls to the coaching staff. To increase coaches' comfort with the treatment of common and severe injuries, coaching certification programs could expand their instruction on the initial treatment of skin, head, eye, hand, and ankle injuries.

CONCLUSION

The most common field hockey injuries are lower limb sprains and contusions, while the most serious injuries involve direct trauma to the upper limb and head or face. Newer studies have described the risk of head/face and upper limb injury in more detail (4,21,22). However, there continues to be a lack of information on the differences in injury rates among genders and positions. Currently, the literature on injury prevention in field hockey consists primarily of expert opinion. The common advice suggests adhering to and enforcing the rules of the game, having sensible precautions regarding protective equipment, and having a commitment to physical preparation (15,33,45,46). Although this advice is reasonable, further research is needed into the mechanism of injury, situations that lead to injury, and the effectiveness of protective equipment. This information should be collected prospectively as new rules and new equipment are introduced into the game. It will help to guide the development of appropriate preventative strategies.

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SUPPLEMENT NO. 64

NCAA®

Field Hockey Face and Eye Protection

David Klossner, PhD, ATC

Director of health and Safety

Liaison to the NCAA Committee on Competitive Safeguards and Medical Aspects of Sports

January 26, 2011

NCAA

2011 CSMAS Recommendation.

- The committee recommended NCAA field hockey players be required to wear protective ASTM-approved protective eyewear during play to minimize the risk of eye injury in the sport.

2011 CSMAS Recommendation

The committee set a hold on the implementation of the recommendation for field hockey eye protector until the equipment on the market can be certified to meet the demands for the collegiate sport.

(a) the need to confirm a standard ball velocity for the collegiate game;

(b) the need to confirm a field hockey ball standard that describes the mechanical properties and performance criteria of the ball so that it is consistent for both testing and playing purposes;

(c) the need to consider a refinement in the ball velocity requirements in the ASTM standard; and

(d) confirmation that the products on the market meet the demands for the collegiate sport.

Additional Future Actions

- Review recent rule changes believed to reduce injury in Field Hockey and compare with injury data
- Field of vision assessment
- Face masks assessments

- Interim: Allow permissive wire cage that meets ASTM standard

Injury Prevention

- In some stick and ball sports, where the players are in close proximity, using a stick to propel the ball, there is eye injury potential from both the ball and the stick.
- Currently, a national ASTM standard exists and products are available for Field Hockey - F-2713-09
- NFHS has mandated protective eyewear.

Injury Prevention

- Injuries to the head and face are common in field hockey
- Catastrophic injuries are rare but do occur.
- Facial injuries often affect the teeth or eyes, and head injuries can cause ongoing disability.
- Stick that permits orbital penetration is a dangerous combination that contributes to the incidence of serious eye injuries

Injury Prevention

- Inadvertent raised balls during play,
- Deflections off sticks,
- Deflections of goalie pads,
- Unsuspecting and suspecting players being struck by ball and stick.

Injury Prevention

- Several authors have suggested that field hockey players wear eye and/or face protection.
- NFHS has mandated ASTM approved eyewear 2011-12.
- NCAA Committee on Competitive Safeguards and Medical Aspects of Sports has encouraged and recommended Field Hockey eye protection since 1996, yet few if any NCAA players wear them during play.
- Those that want to wear protection are wearing flush polycarbonate material face masks yet no standards exist.

Moving Forward

Swing Speed

- Partnering with NFHCA
- Seek proposal for live swing speeds of collegiate Field Hockey Players
- Analyze ball exit speeds

Field Hockey Ball

- Partnering with NFHCA
- Identity the preferred playing ball.
- Test for variability within same ball.
- Determine characteristics.
- Create standard (2 year process).
- Adopt Standard.
- Current ball rules:
  - is spherical
  - has a circumference of between 224 mm and 235 mm
  - weighs between 156 grams and 163 grams
  - is made of any material and coloured white (or an agreed colour which contrasts with the playing surface)
  - is hard with a smooth surface but indentations are permitted.

NCAA Injury Surveillance Program
Field Hockey
Head and Eye Injury Report

Dataset 2004/05-2008-09 (5-years)

DATALYSCENTER
Sports Injury Research and Prevention

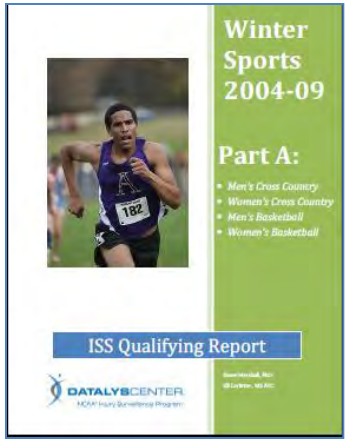
NCAA

NCAA Injury Surveillance

15 Sports

1988 – 2004
Paper Collection

15+ Sports



2004 – 2009
Web-Based

25 NCAA Sports

2009 - Forward
Web-Based + Commercial
Electronic Record Systems

Overall Summary

- Head/Face, Nose, Eye, Mouth Account for 15.1% of all Injuries
- The Eye Accounts for 1.2% of all injuries
- On Average, there are 27 Eye Injuries each Year
- On Average, there are 2 Orbital Fractures each Year
- In Competitions, Elevated Balls Accounted for 23% of all Injuries

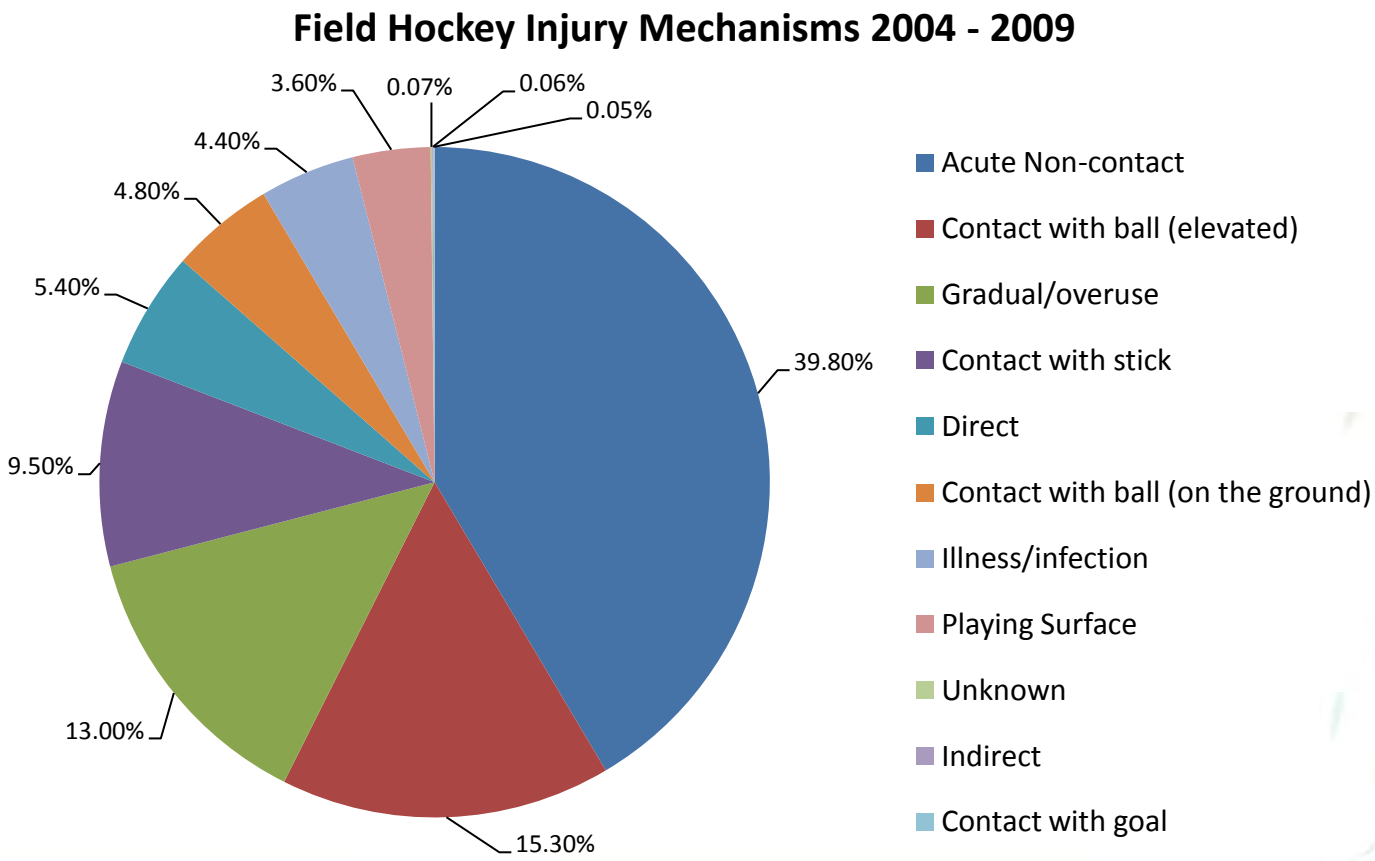
Field Hockey

Ten Most Commonly Injured Body Sites in FH Competition (%)	
Rank	NCAA (04/05-08/09)
1	Head/Face (19.8%)
2	Hand/Finger (15.5%)
3	Knee (14.0%)
4	Ankle (8.9%)
5	Thigh (7.5%)
6	Lower Leg/Achilles (6.4%)
7	Foot/Toe (4.0%)
8	Shoulder/Clavicle (3.7%)
9	Lower Back/Sacrum/Pelvis (2.6%)
10	Nose (2.5%)

Field Hockey

Ten Most Common Specific Injuries in Field Hockey Competition (%)	
Rank	NCAA (04/05-08/09)
1	Concussion (8.7%)
2	Hand/Finger Contusion (6.1%)
3	Knee Contusion (5.1%)
4	Facial Contusion (5.0%)
5	Ankle Ligament Partial Tear (4.8%)
6	Facial Laceration (4.4%)
7	Finger Fracture (3.7%)
8	Hamstring Partial Tear (2.6%)
9	Quadriceps Partial Tear (2.3%)
10	Foot/Toe Contusion (1.9%)

Field Hockey



NCAA Injury Surveillance

Table 7. Above-the-Neck Game Injuries With Most Common Injury Mechanisms, Women's Field Hockey, 1988–1989 Through 2002–2003*

Body Part	Injuries, No.	Most Common Mechanism of Injury	Injuries at Each Body Part Due to Each Mechanism, %
Ear	3	Contact with ball	66
		Contact/collision with another player	33
Eye	22	Contact with stick	45
		Contact with ball	45
Face	54	Contact with ball	63
		Contact with stick	31
Head	120	Contact with stick	41
		Contact/collision with another player	29
Teeth	7	Contact with stick	71
		Contact with ball	29
Nose	61	Contact with ball	56
		Contact with stick	27
Mouth	17	Contact with ball	53
		Contact with stick	41
Jaw (temporomandibular joint)	7	Contact with stick	43
		Contact with ball	43
Total	291		

*Above-the-neck injuries accounted for 24% of all game injuries during this interval.

2004-2009

Injuries during Competition and Practice

Women's Field Hockey			
	All Injuries	Head Face Injuries	% of Head Face Injuries by Event Type
Competition	4227	1078	25.5
Practice	6561	553	8.4
*Total**	*10788*	*1631*	*15.1*

* errors in totals due to rounding

50

Head and Face Injuries by Basic and Specific Injury Mechanisms

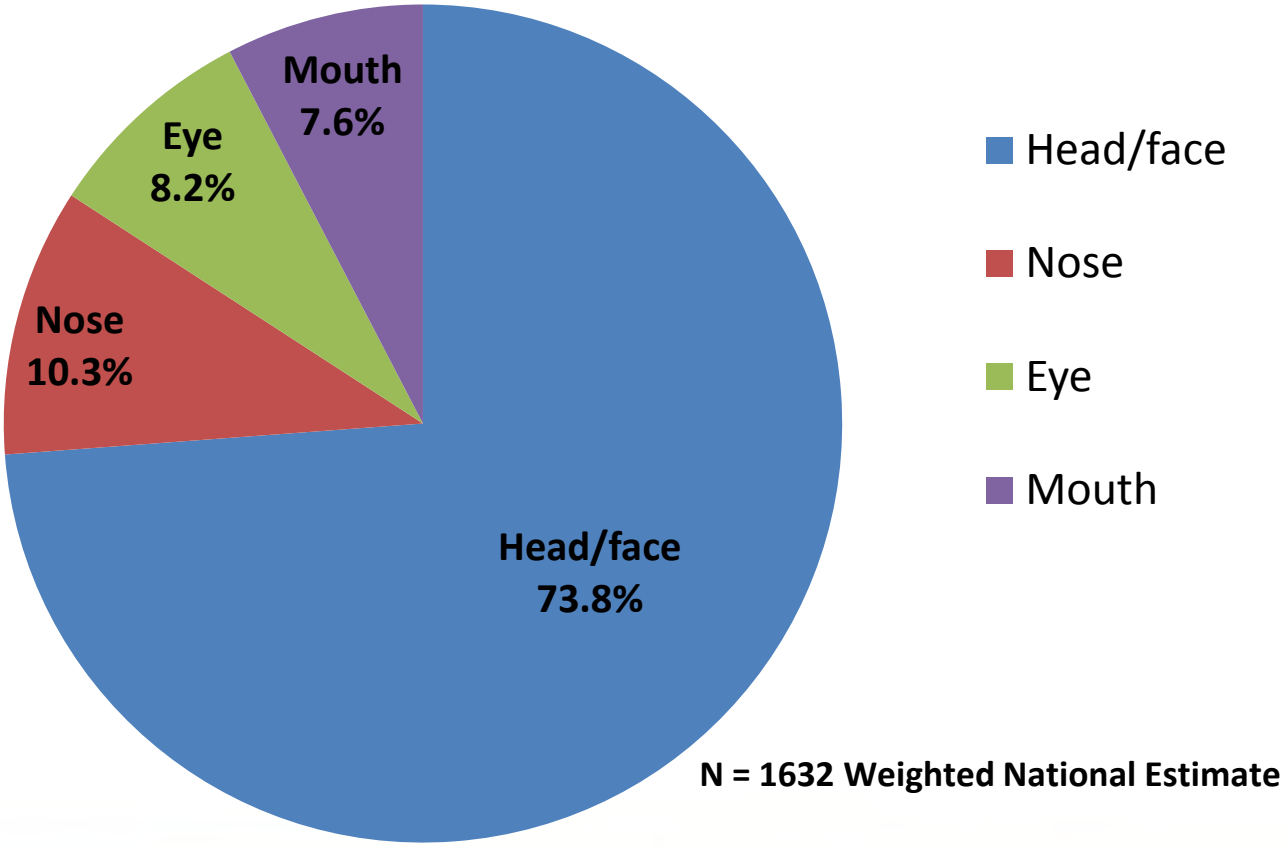
Women's Field Hockey				
Event Type	Basic Injury Mechanism	Specific Injury Mechanism	Frequency	Percent
Competition	Contact with player	All Specific Mechanisms	255.0	15.6
Competition	Contact with playing surface	Not Applicable	40.2	2.5
Competition	Contact with apparatus	***WFH - Contact with stick***	***265.7***	***16.3***
Competition	Contact with apparatus	WFH - Contact with goal	21.8	1.3
Competition	Contact with apparatus	***WFH - Contact with ball (Ground = 7, Elevated = 463)***	***470.0***	***28.9***
Competition	Acute non-contact	Not Applicable	14.6	0.9
Competition	Infection	Not Applicable	10.9	0.7
Practice	Missing	Not Applicable	5.7	0.4
Practice	Contact with player	All Specific Mechanisms	81.0	5.0
Practice	Contact with apparatus	***WFH - Contact with stick***	***79.0***	***4.8***
Practice	Contact with apparatus	WFH - Contact with goal	19.8	1.2
Practice	Contact with apparatus	***WFH - Contact with ball (Ground = 29, Elevated = 296)***	***325.0***	***19.9***
Practice	Infection	Not Applicable	34.0	2.1
Practice	Other	Not Applicable	8.8	0.5
*Total**			*1632.0*	*100.0*

*errors in totals due to rounding

How are Injuries to the Head Distributed

Injuries to the Region of the Head

(Practice and Competition Combined)

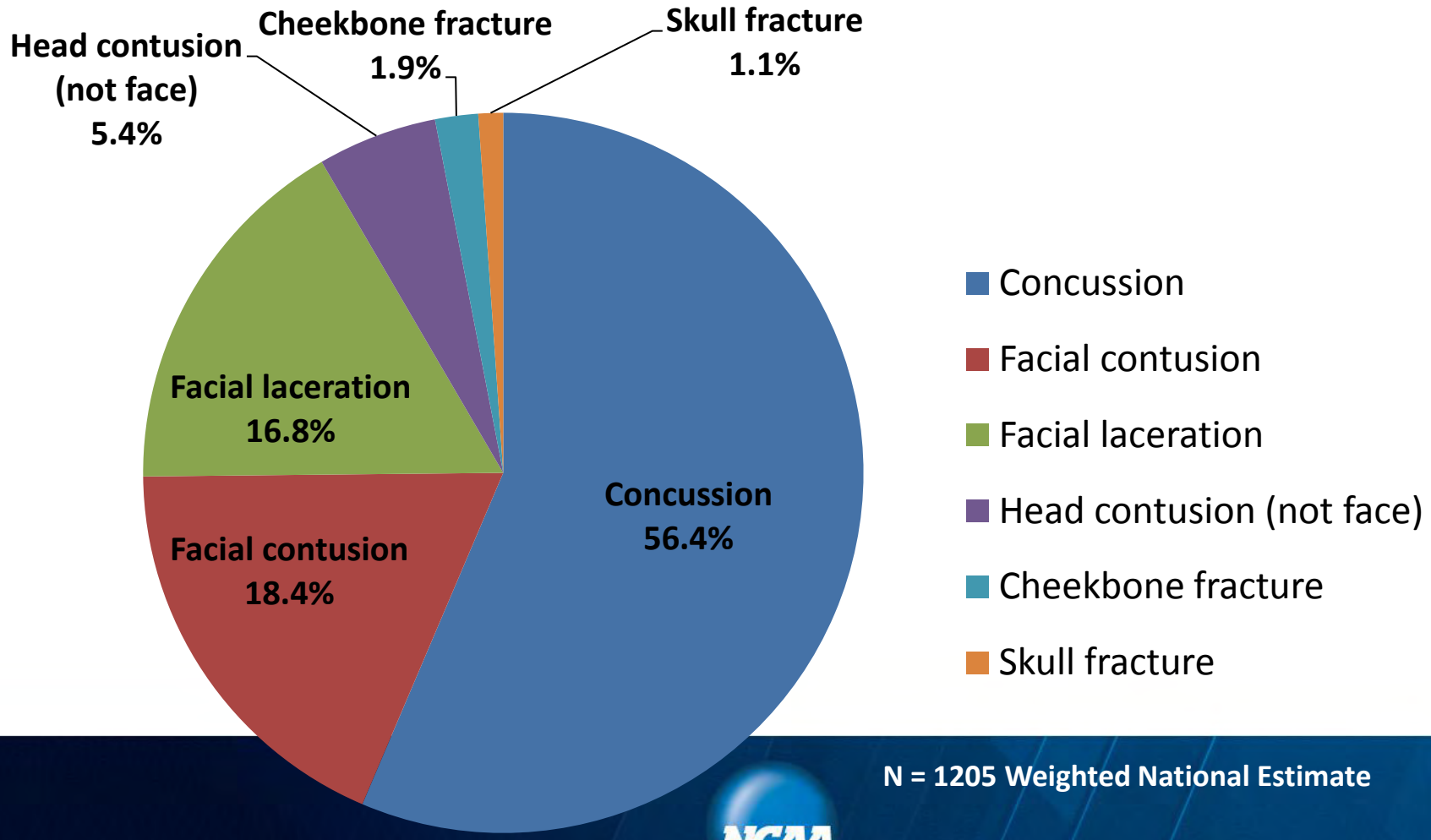


N = 1632 Weighted National Estimate

What are the Specific Types of Injuries that Occur to the Head and Face

Specific Head & Face Injuries

(Practice and Competition Combined)

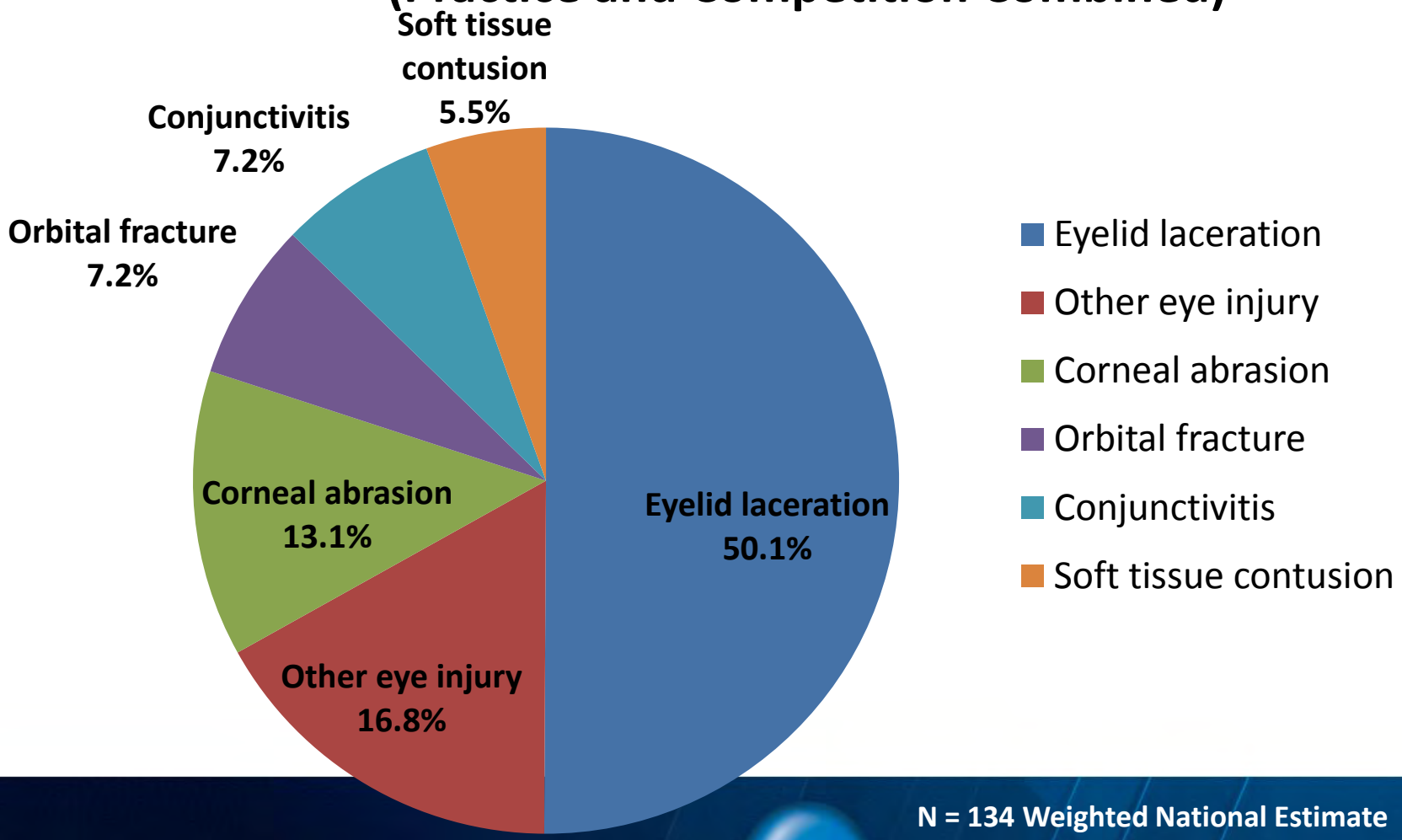


N = 1205 Weighted National Estimate

What types of Specific Injuries Occurred to the Eye

Specific Eye Injuries

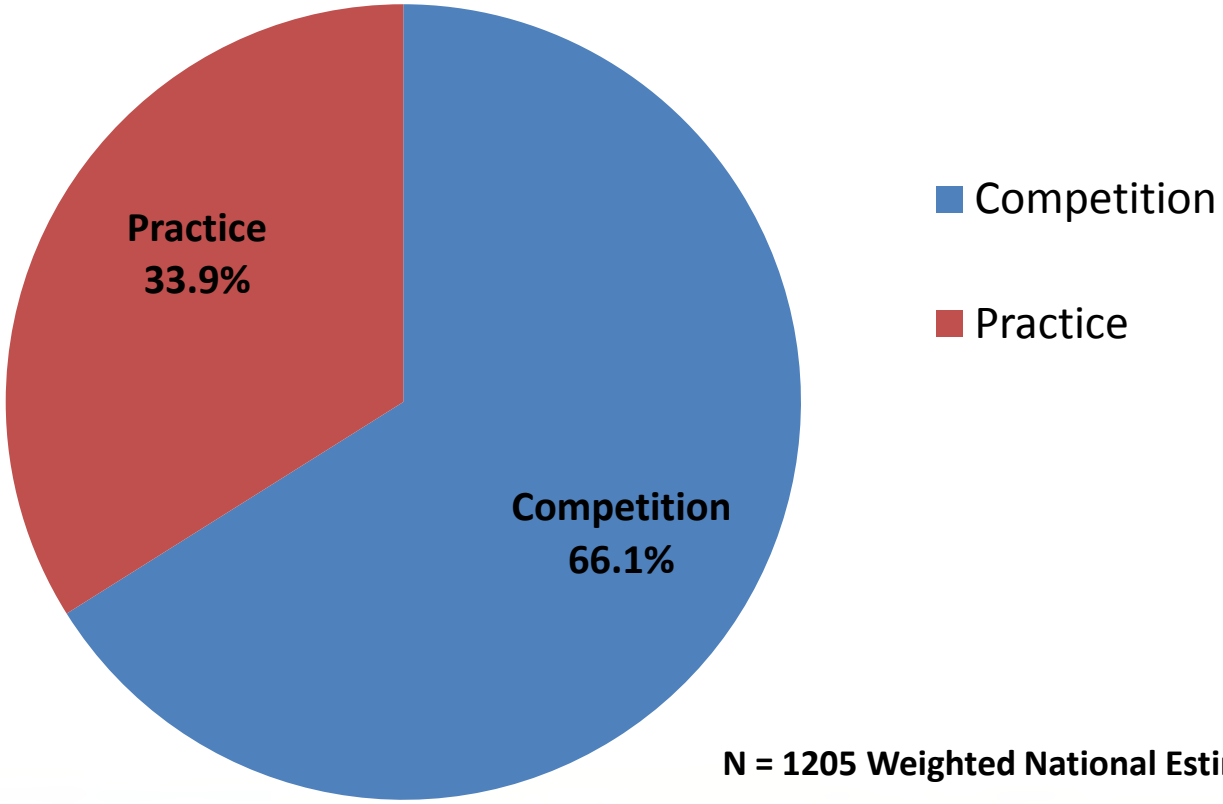
(Practice and Competition Combined)



N = 134 Weighted National Estimate

Did More Injuries Occur to Head in Game or Practice Situations

Head & Face Injuries by Event Type

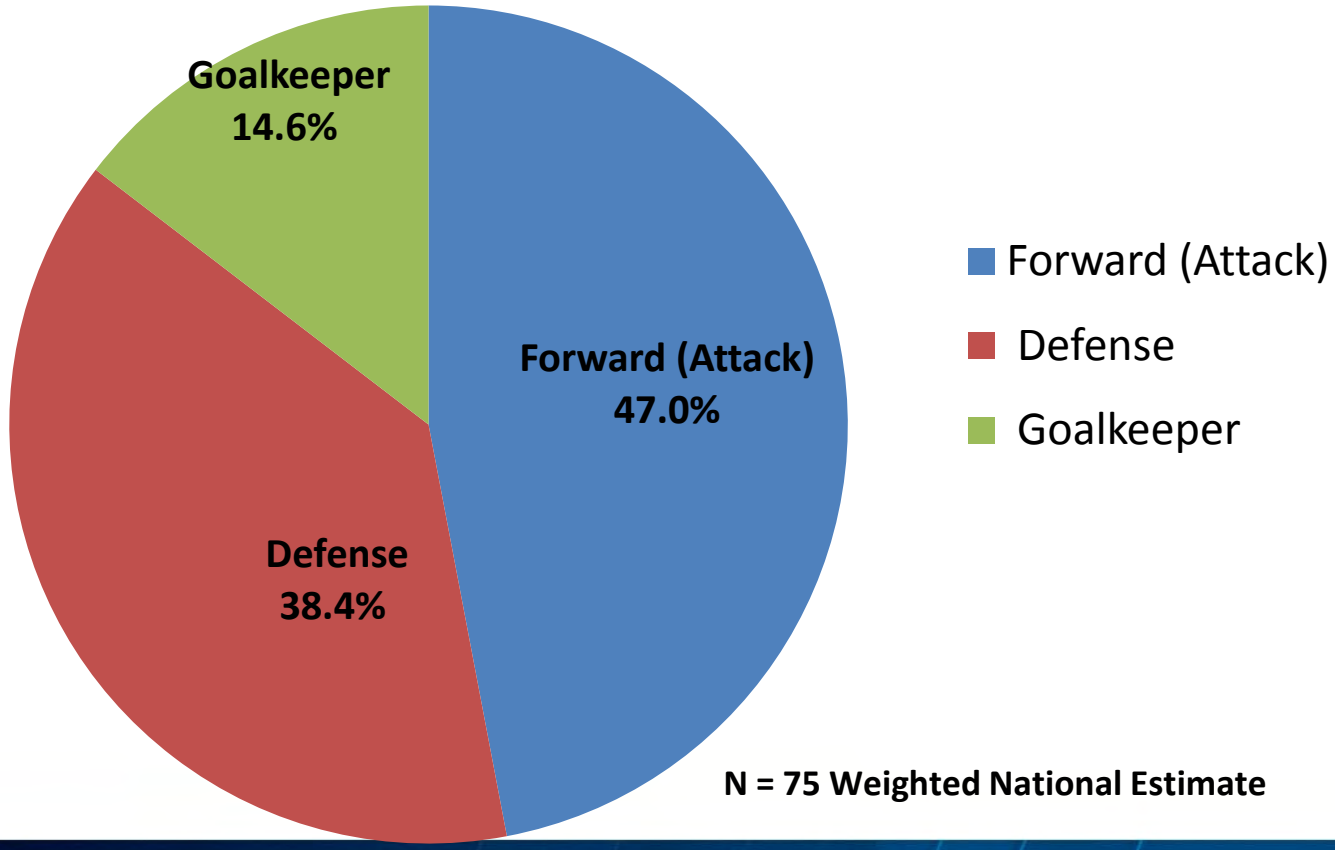


N = 1205 Weighted National Estimate

Did Eye Injuries Differ by Position

Eye Injuries by Position

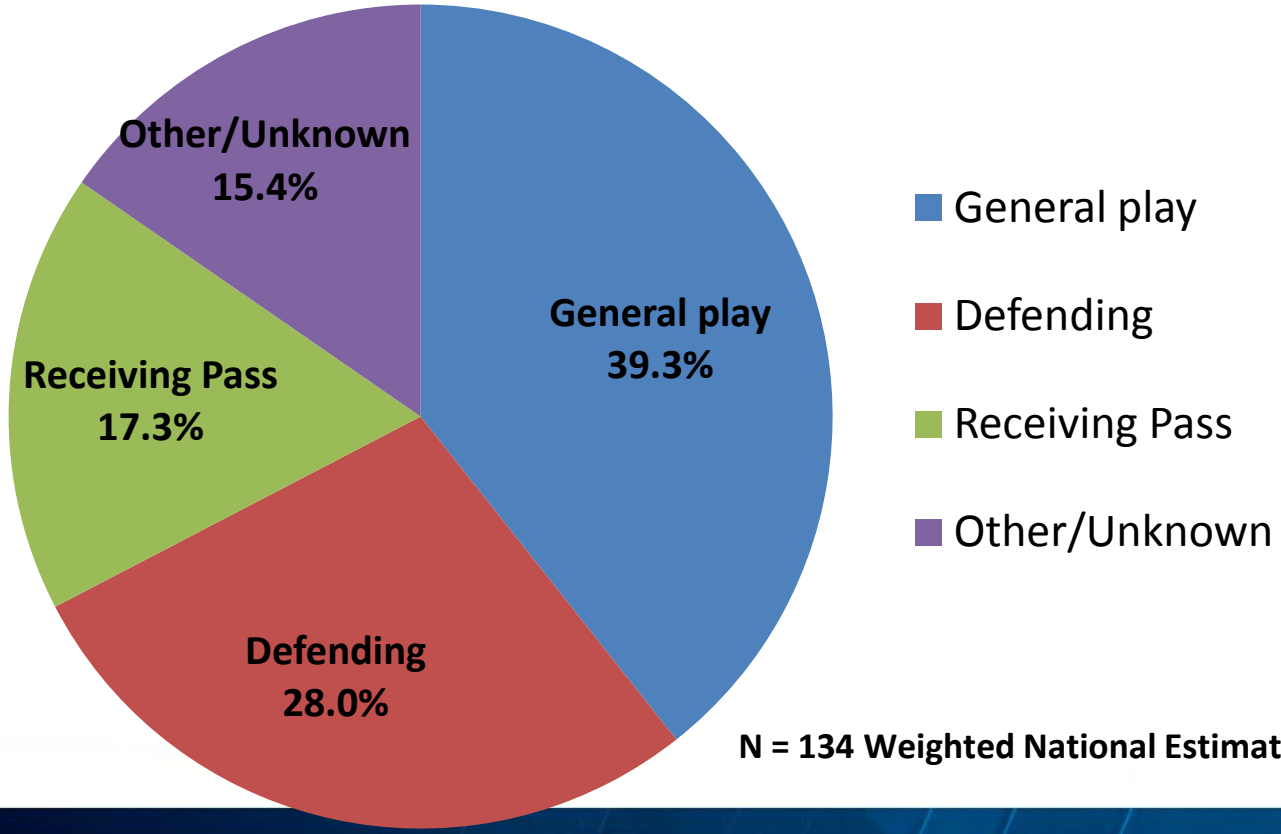
(Competition Only)



N = 75 Weighted National Estimate

Did Eye Injuries Differ by Activity at Time of Injury

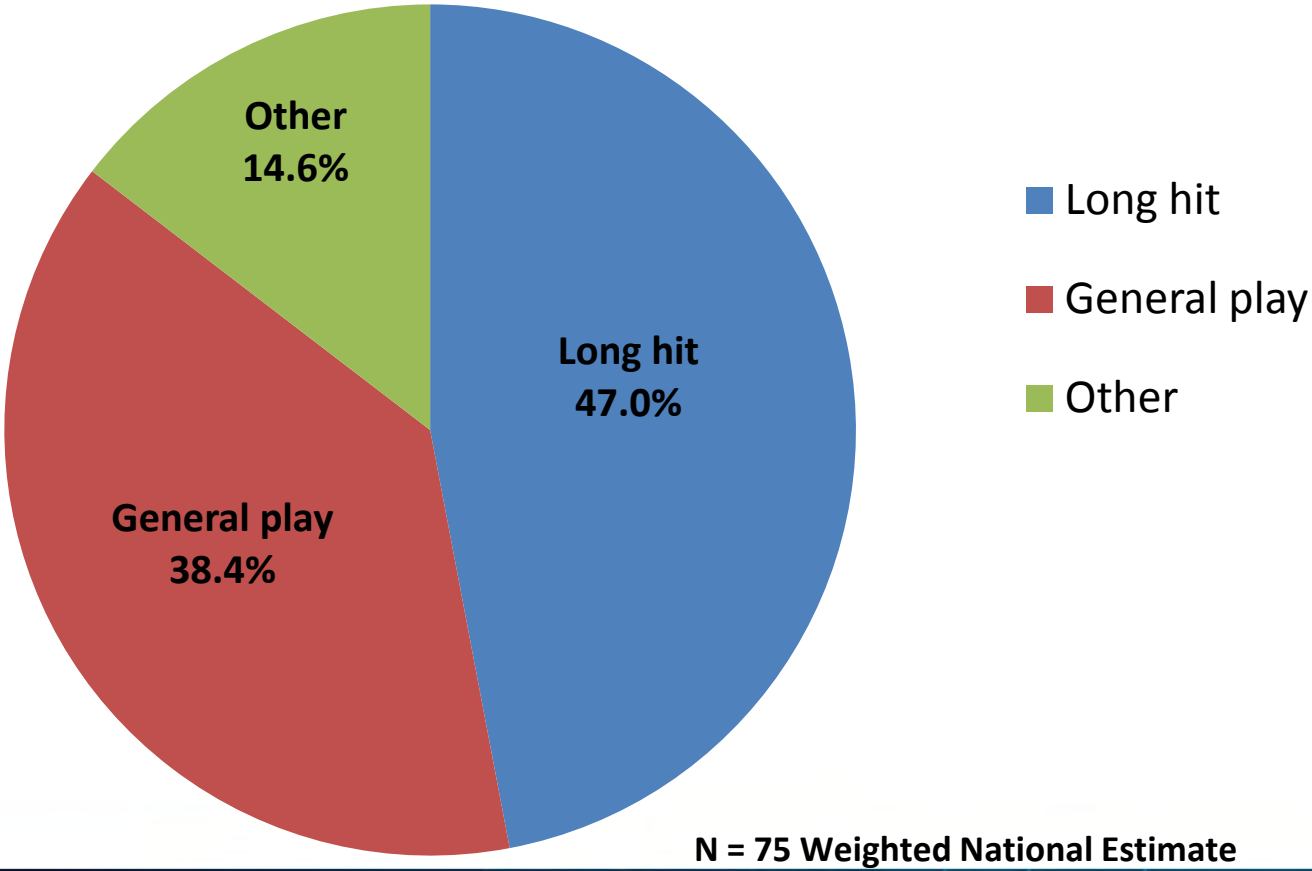
Eye Injuries by Activity
(Practice and Competition Combined)



N = 134 Weighted National Estimate

Did Eye Injuries Differ by Game Situation

Eye Injuries by Situation
(Competition Only)

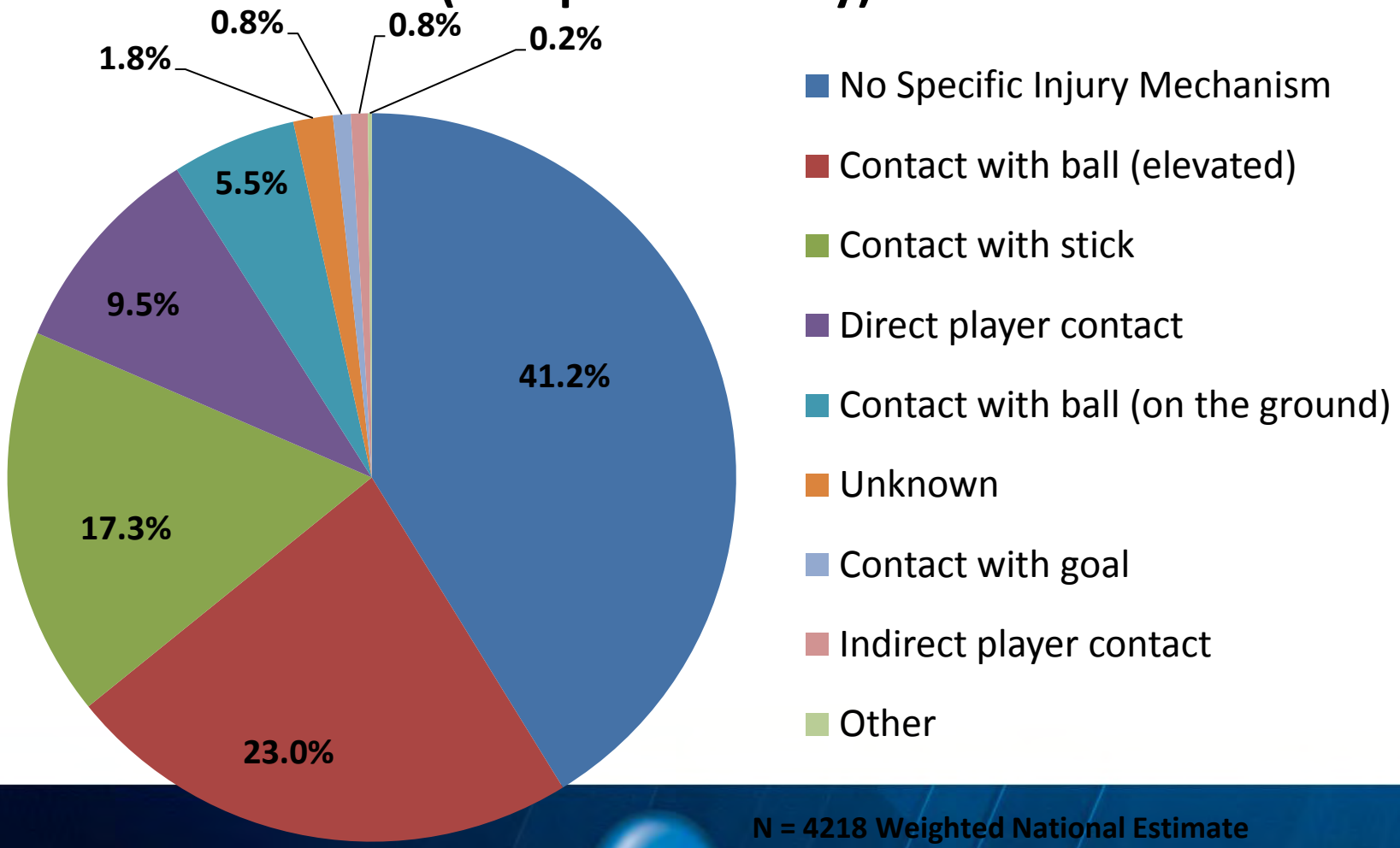


N = 75 Weighted National Estimate

Are the Differences Among Injury Mechanisms During Competition

All Injuries by Specific Mechanism

(Competition Only)

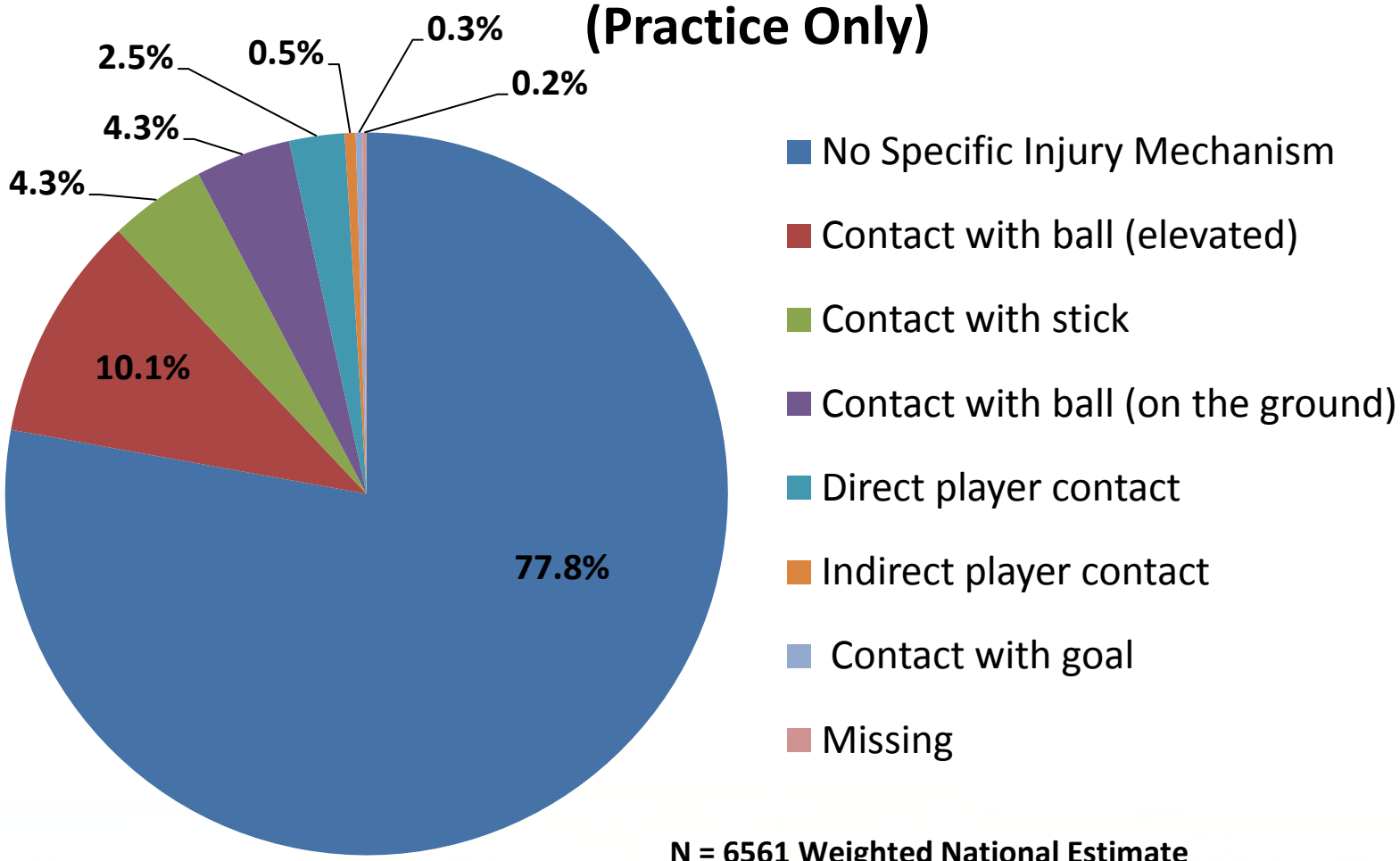


N = 4218 Weighted National Estimate

Are the Differences Among Injury Mechanisms During Practices

All Injuries by Specific Mechanism

(Practice Only)



N = 6561 Weighted National Estimate

One FH Program's History

- 2003 - Run of serious facial injuries
  - Another zygomatic fracture from ball to face and required surgical reduction/fixation.
  - Had two concussions
  - A facial laceration
  - chipped tooth and a bad lip laceration on opposing teams

- 2002 - Had a zygomatic/temporal bone fracture with a brain bleed
    - Tried to wear a protective eye mask like lacrosse players now mandated to wear, but was told in one game this year that she could not wear it by the official.
  - In addition to other laceration's/orbital contusions
  - A jaw fracture requiring surgical fixation
  - A bad eye injury with laceration surrounding

- Led to larger season long research study in 2004

BIG TEN CONFERENCE

2004 Field Hockey
Facial Injury Survey

- 6 Schools
- 127 Total Athletes

- 128 Games
  - ~ 11 Starters + 3 Substitutes
  - ~ 84 Athletes

10,752 Exposures

2004 Field Hockey
Facial Injury Survey

- **35 Incidences**

Contact with Ball	24	68%
Contact with Stick	8	23%
Contact with Player	3	9%
Contact with Goal	0	

2004 Field Hockey
Facial Injury Survey

37 Injuries

	#	Rate	%
Facial	17	1.6	46%
Head	10	0.9	27%
Eye	6	0.6	16%
Dental	4		11%

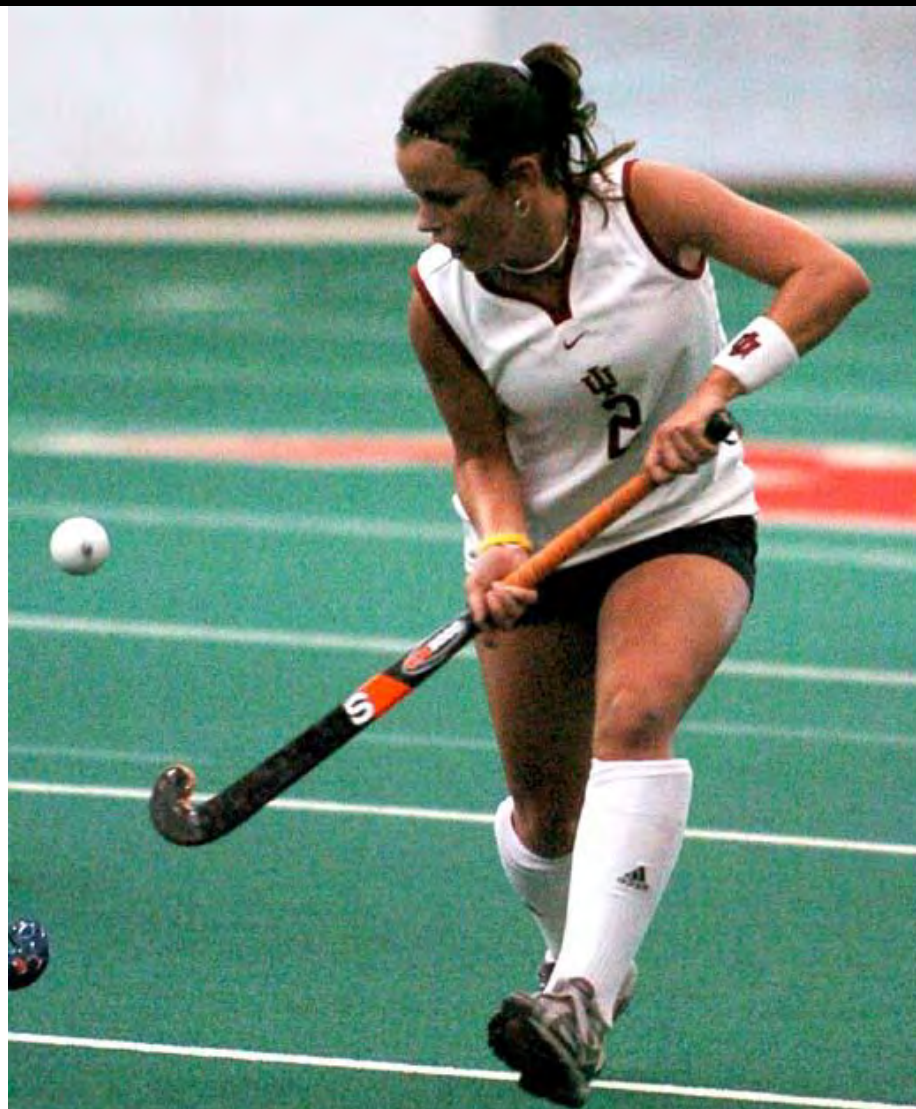
- **4 Surgeries**
- **9 Time Loss**
- **10 Sutures**

NCAA Rate: 0.3

2004 Field Hockey
Facial Injury Survey

37 Types of Injuries

Contusion	13	35%
Fracture	10	27%
Laceration	9	24%
Concussion	4	11%
Ear Drum Rupture	1	3%



2004 Field Hockey
Facial Injury Survey

6 Eye Injuries

Orbit Hematoma	1	
Orbit Fracture	1	
Laceration (lid, brow)	4	

Equipment

ASTM Standards for Field Hockey		
	New	Old*
ASTM designation	F-2713-09	F-803-03
Year Approved	2009	2003
Sports included in standard	Field Hockey	Field Hockey, Lacrosse, Baseball and Racquet sports
Projectile	Field Hockey ball	Lacrosse ball used for field hockey test
Projectile speed	55 MPH	60 MPH
Penetrator	Field Hockey stick toe (dimensions based on the rules of the game; they are identical to the old ASTM standard)	Field Hockey stick toe
Chairman of Subcommittee	Dr. Liz Ryan	Dr. Paul Vinger
Members of ASTM Subcommittee	Brine, Cascade, Halo,K2 (deBeer's parent company), Oakley,Reebok, STX, Warrior, consumer advocates, medical professionals and academics.	Cascade, consumer advocates, US Lacrosse, medical professionals and academics.
Approved by	Consensus vote**. Every negative vote is debated by the subcommittee, if a negative vote is found non persuasive by the subcommittee those arguments are sent to an independent nine member panel that must agree the negative vote is non persuasive. (unchanged from 2003)	Consensus vote**. Every negative vote is debated by the subcommittee, if a negative vote is found non persuasive by the subcommittee those arguments are sent to an independent nine member panel that must agree the negative vote is non persuasive.
	[illegible]	[illegible]

New Designs

Impact testing of some products Small sample, limited products

Conducted and provided by:
Dave Halstead, Director of Southern Impact Research Center

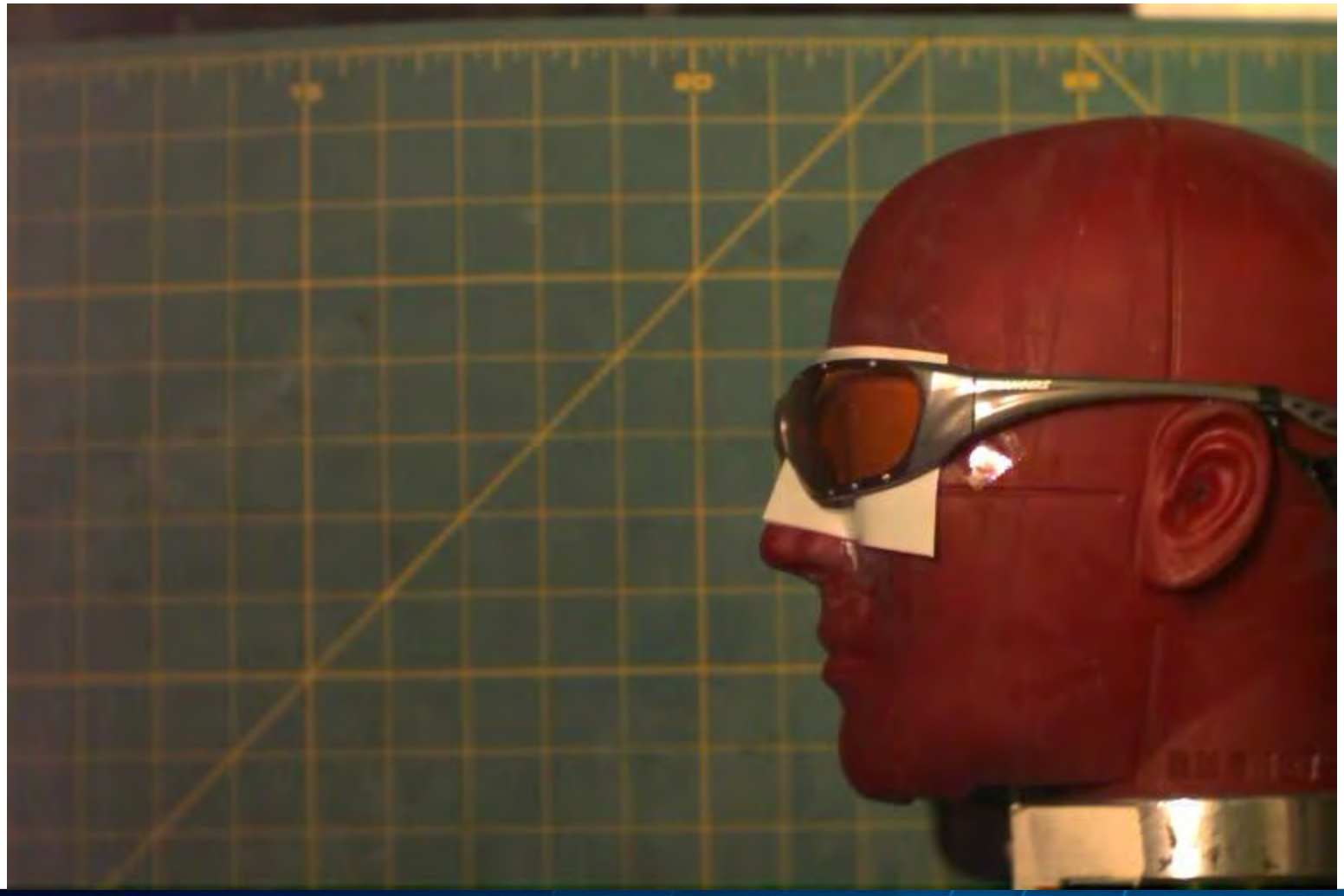
- NFSHS Ball
- NOCSAE HEAD FORM
- ASTM impact velocity – highest end 55 mph +/- 6 mph

Unprotected NOCSAE size 6 ⅝ Headform, 50% Female Head Size
Impact Towards Left Eye of Headform Facing Forward

Fuji Film Pressure Indicator - 725 to 1450 psi

- Longstreth Multi-Turf NFHS Field Hockey Ball Velocity = 63.30 MPH Resultant Peak g, Measured at the Headform CG = 445

Bangerz Eye Protector, ASTM F2713-09, ASTM F 803-03, PECC Impact to center of Left Lens Towards Eye of Headform Facing Forward



Fuji Film Pressure Indicator - 725 to 1450 psi

- Longstreth Multi-Turf NFHS Field Hockey Ball Velocity = 62.80 MPH
- Resultant Peak g, Measured at the Headform CG = 460

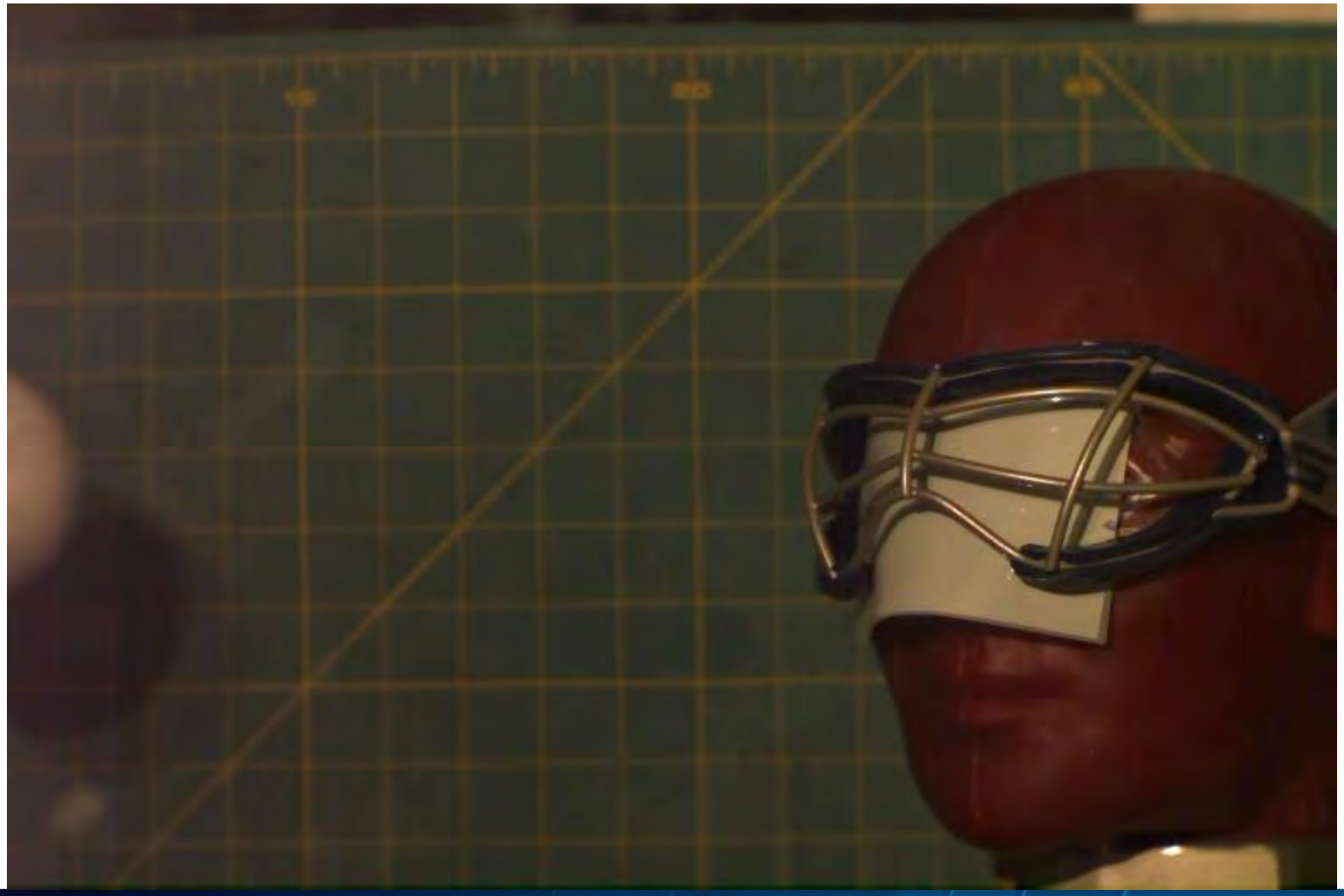
Cascade Iris Eye Protector, ASTM F2713-09, ASTM F 803-03, PECC Impact to Left Viewing Area Towards Eye of Headform Facing Forward

cascade

Fuji Film Pressure Indicator - 725 to 1450 psi

- Longstreth Multi-Turf NFHS Field Hockey Ball Velocity = 62.94 MPH
- Resultant Peak g, Measured at the Headform CG = 126

STX Eye Protector, ASTM F2713-09, ASTM F 803-03
Impact to Left Viewing Area Towards Eye of Headform Facing 45 degrees Right



Fuji Film Pressure Indicator - 725 to 1450 psi

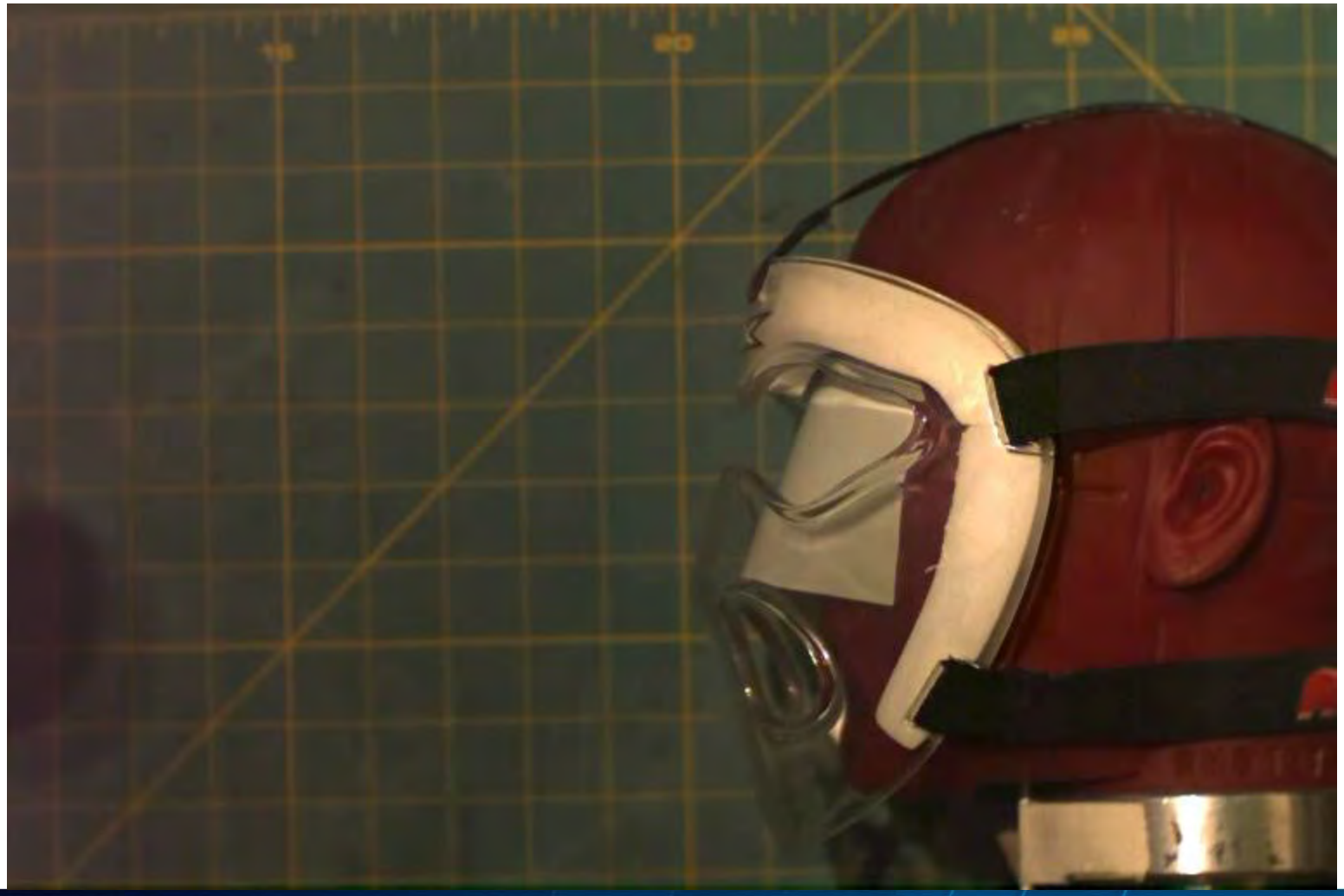
- Longstreth Multi-Turf NFHS Field Hockey Ball Velocity = 62.78 MPH
- Resultant Peak g, Measured at the Headform CG = 161

Face Masks

- NCAA allows for face protection of all field players at all times.
  - "Field players are permitted to wear throughout a game only a smooth preferably transparent or white but otherwise dark plain colored face mask which fits flush with the face, soft protective head covering or eye protection in the form of plastic goggles (i.e., goggles with a soft-covered frame and plastic lenses. A caged frame cannot be worn)."
- However, there is no known research on the appropriate design or effectiveness of the devices in use.

Face masks

TK Mask, designed specifically for Short Corner Phase of Field Hockey
Impact to Left Viewing Area Towards Eye of Headform Facing Forward



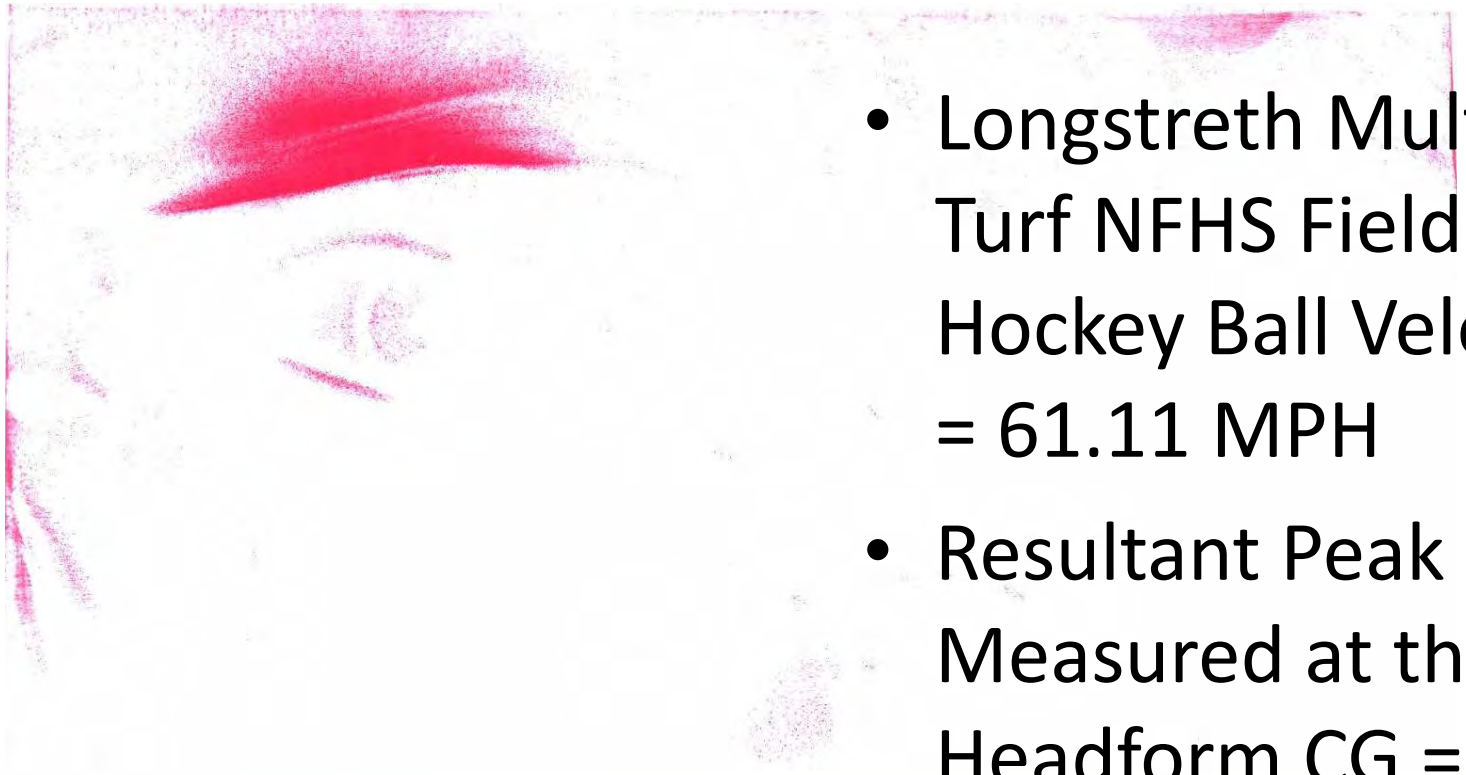
Fuji Film Pressure Indicator - 725 to 1450 psi

- Longstreth Multi-Turf NFHS Field Hockey Ball Velocity = 63.51 MPH
- Resultant Peak g, Measured at the Headform CG = 227

TK Mask, designed specifically for Short Corner Phase of Field Hockey
Impact to Right Viewing Area Towards Eye of Headform Facing 45 degrees Left

TK

Fuji Film Pressure Indicator - 725 to 1450 psi



- Longstreth Multi-Turf NFHS Field Hockey Ball Velocity = 61.11 MPH
- Resultant Peak g, Measured at the Headform CG = 264

OBO Face Off, designed specifically for Short Corner Phase of Field Hockey
Impact to Left Viewing Area Towards Eye of Headform Facing 45 degrees Right

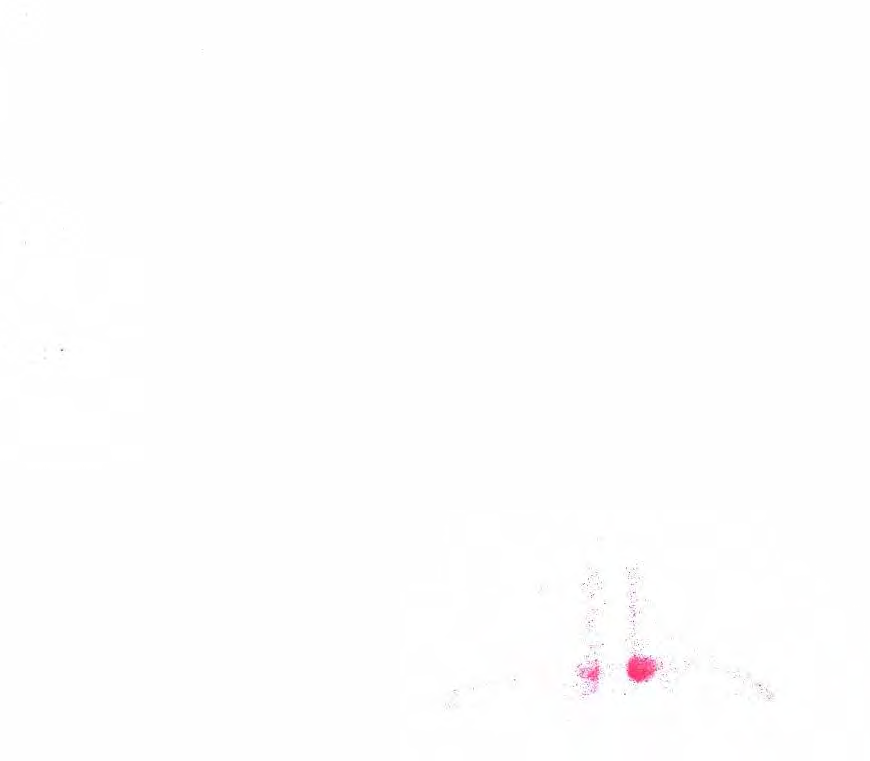
OBO

Fuji Film Pressure Indicator - 725 to 1450 psi

- Longstreth Multi-Turf NFHS Field Hockey Ball Velocity = 64.01 MPH
- Resultant Peak g, Measured at the Headform CG = 413

OBO PE Helmet Goal Keeping, CEN/TC-158
Impact to Left Viewing Area Towards Eye of Headform Facing Forward

Fuji Film Pressure Indicator - 725 to 1450 psi



- Longstreth Multi-Turf NFHS Field Hockey Ball Velocity = 62.15 MPH
- Resultant Peak g, Measured at the Headform CG = 41

2011 CSMAS Recommendation.

- The committee recommended NCAA field hockey players be required to wear protective ASTM-approved protective eyewear during play to minimize the risk of eye injury in the sport.

2011 CSMAS Recommendation

The committee set a hold on the implementation of the recommendation for field hockey eye protector until the equipment on the market can be certified to meet the demands for the collegiate sport.

(a) the need to confirm a standard ball velocity for the collegiate game;

(b) the need to confirm a field hockey ball standard that describes the mechanical properties and performance criteria of the ball so that it is consistent for both testing and playing purposes;

(c) the need to consider a refinement in the ball velocity requirements in the ASTM standard; and

(d) confirmation that the products on the market meet the demands for the collegiate sport.

Additional Future Actions

- Review recent rule changes believed to reduce injury in Field Hockey and compare with injury data
- Field of vision assessment
- Face masks assessments

- Interim: Allow permissive wire cage that meets ASTM standard

Men’s Ice Hockey

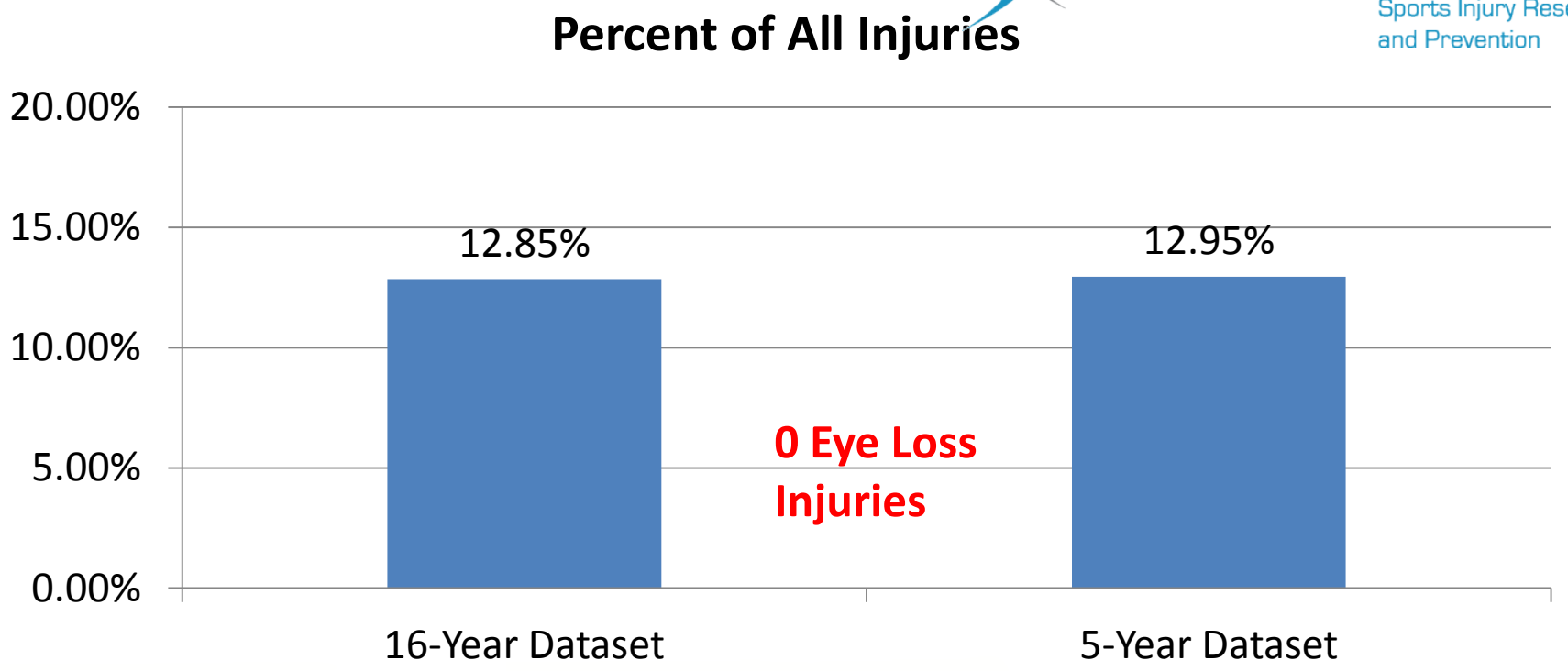
Full Face Masks in Ice Hockey

Ice Hockey

- NCAA Mandates Full Face Masks in Ice Hockey. Other organizations do not. Current NCAA rule is under discussion to allow lesser face protection by the coaching community.
- No Catastrophic Eye Injuries in NCAA IH since face masks in place.
- Full mask or cage provides the highest level of safety with dramatic reduction of facial fractures, lacerations, dental trauma and eye injury.
- There are no documented cases of blindness to a hockey player wearing full protection anywhere in the world.
- There are cases of blindness to a hockey player wearing a visor.

Injuries to the Head and Face

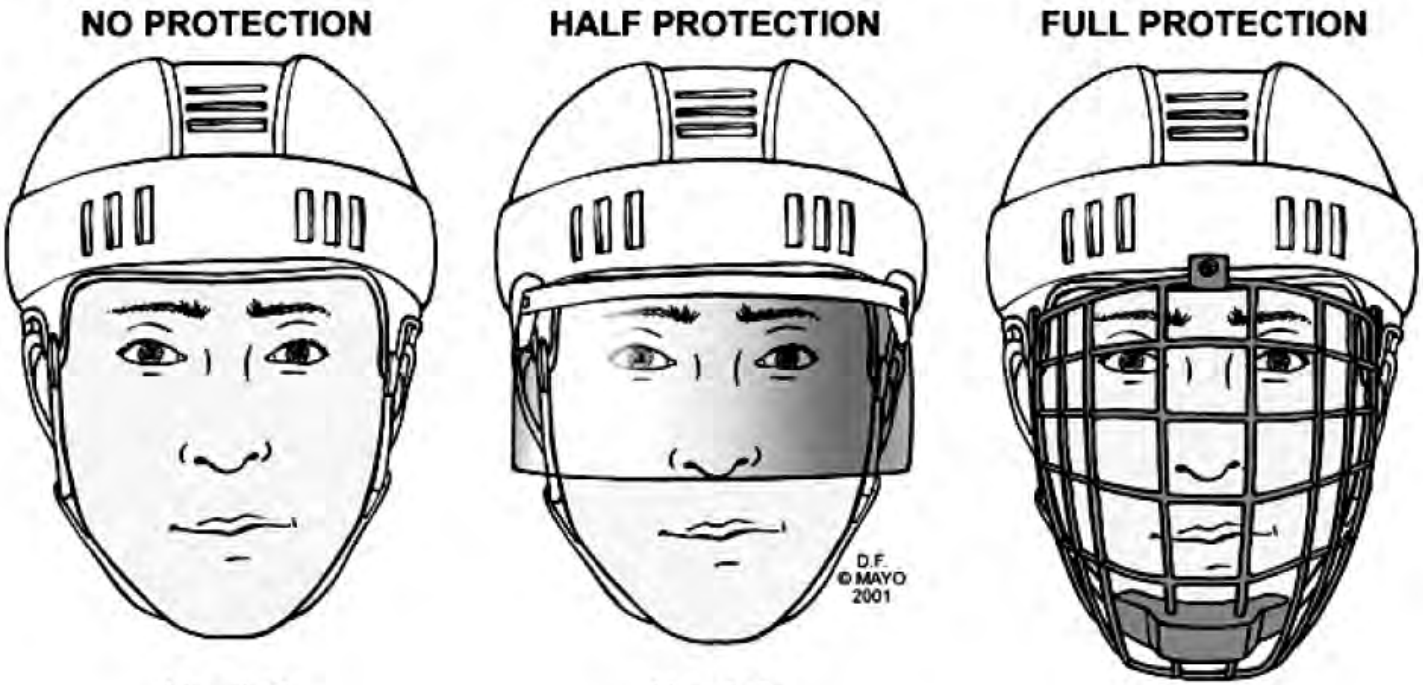
DATALYSCENTER
Sports Injury Research and Prevention



NCAA

Comparison to Historical Data

- Prior to widespread use of full face shields, injuries to the head and face were reported to be between 37%-64% of all injuries, compared to 13% in the NCAA since full face masks mandated (Vinger, 2008)



	NO PROTECTION	HALF PROTECTION	FULL PROTECTION
Head	7	6	2
Face	26	27	13
Eye	10	4	0
Mouth/Teeth	7	8	1
Neck	1	0	0
Ear	1	0	0

Stuart MJ, Smith AM, et al. "A comparison of facial protection and the incidence of head, neck, and facial injuries in Junior A hockey players. A function of individual playing time". *American Journal of Sports Medicine,* 30: 39-44, 2002.

NCAA Ice Hockey Face Protection

The following summary is the result of a review of current scientific data and communications with medical field experts relative to the use of half shields versus full face protection. Medical field experts beyond the NCAA CSMAS and Artie Poitras, ATC from the NCAA Ice Hockey playing rules committee included Michael J. Stuart MD, (USA Ice Hockey); Mark Aubry, MD (IIHF Medical Committee Chairman); and Alan Ashare, MD (Boston, Massachusetts).

The Datalys Center for Injury Prevention and Research which conducts the NCAA Injury Surveillance program has also provided consultation for this overview.

The current data clearly notes that a full mask or cage provides the highest level of safety with dramatic reduction of facial fractures, lacerations, dental trauma and eye injury. There are no documented cases of blindness to a hockey player wearing full protection anywhere in the world. There are many cases of blindness to a hockey player wearing a visor.

Fortunately, hockey players currently learn the sport while wearing full facial protection and thus transition well into the NCAA game environment for both men and women.

There literature does not support the theory that the use of a full visor or facemask increases the risk of a spinal injury. In addition, several studies have shown that facial protection is not associated with an increased risk of concussion. Several authors have concluded that the existing rules, if taught and enforced, should decrease the risk of catastrophic injury regardless of the type of facial protection.

Leading head first with the head down and unsportsmanlike conduct (checking from behind, head hit, boarding, fighting, etc.) are both dangerous, not directly related to facial protection and should be avoided.

The literature notes that half visors provide more protection than no visor at all, however full face protection provide optional protection from injuries. The data concludes that the half visor will lead to more facial injuries including fractures of the facial area (nose, mandible), facial lacerations, dental injuries and potentially eye injuries where the stick gets up under the visor or the visor is not worn properly. Current helmets with visors have a very poor attachment system. Tipping the helmet back (which lifts up the visor) is very common in Junior A and professional hockey. This scenario is the same as wearing no facial protection, which places the athlete at risk for a blinding eye injury.

Comparisons have been made to competition rules with Junior A hockey in the USA Hockey system and the International Ice Hockey Federation (IIHF). The IIHF has been collecting injury data on its Championships for the last 12 years. Because of the numerous facial injuries, the IIHF is currently examining the reintroduction of the full visor for the U20 Championships. Official Junior Playing Rules of USA Hockey specifically requires that all players in the Junior age classification wear HECC-approved helmets and recommends and encourages full facial protection for all games and practices. Any player, having reached the age of majority, is permitted to sign the proper waiver and wear a HECC-approved half facemask shield. Junior A hockey injuries document both facial and eye injuries with the use of half shields.

The importance of facial protection was documented by a study funded by USA Hockey. The investigators showed a 4.7 times greater risk with no protection compared to a visor (half-shield). No eye or neck injuries occurred to players wearing full protection.

Appendix 2: Leagues in Ice Hockey

Table 7: Hockey leagues and visor regulation.

Name	Short name	League type	Face protection
National Hockey League	NHL	Pro	Visors are non-mandatory
United Hockey League	UHL	Minor pro	Mandatory since 2004
American Hockey League	AHL	Minor pro	Mandatory since 2006
East Coast Hockey League	ECHL	Minor pro	Mandatory since 2003
Central Hockey League	CHL	Minor pro	Mandatory since 2004
Western hockey league	WHL	Major junior (CA)	Mandatory since 1976
Ontario hockey league	OHL	Major junior (CA)	Mandatory since 1976
Quebec Major junior hockey league	QMJHL	Major junior (CA)	Mandatory since 1976
British Columbia Junior Hockey League	BCJHL	Junior (CA)	Mandatory since 1981
Ontario Provincial Junior A Hockey League	OPJHL	Junior (CA)	Mandatory since 1981
British Columbia Hockey League	BCHL	Junior (CA)	Mandatory since 1981
Saskatchewan Junior Hockey League	SJHL	Junior (CA)	Mandatory since 1981
Atlantic Junior Hockey League	AJHL	Junior (CA)	Mandatory since 1981
Metropolitan Junior Hockey League	MetJHL	Junior (CA)	Mandatory since 1981
Ontario Junior Hockey League	OJHL	Junior (CA)	Mandatory since 1981
Canadian Junior Hockey League	CJAHL	Junior (CA)	Mandatory since 1981
United States Hockey League	USHL	Junior (U.S.)	Always been mandatory
North American Hockey League	NAHL	Junior (U.S.)	Always been mandatory
Western Collegiate Hockey Association	WCHA	College (NCAA)	Mandatory since 1980
Central Collegiate Hockey Association	CCHA	College (NCAA)	Mandatory since 1980
NCAA East Division	H-East	College (NCAA)	Mandatory since 1980
Eastern College Athletic Conference	ECAC	College (NCAA)	Mandatory since 1980
National Collegiate Athletic Association	NCAA	College (NCAA)	Mandatory since 1980
College Hockey Association	CHA	College (NCAA)	Mandatory since 1980
International Hockey League	IIHL	International	Mandatory since 1994
Sweden Elitserien	SEL	European elite	Mandatory since 1969
Finland SM-liiga	FNL	European elite	Mandatory since 1988
Russian Elite League	KHL	European elite	Mandatory since 1994
Switzerland National League A	Swiss-A	European elite	Mandatory by 2004
Deutsche Eishockey League	DEL	European elite	Mandatory since 1998

Notes: This table shows the different leagues used in our study, as well as their respective regulation regarding facial protection. In college leagues, players are required to use a full cage if they are under 18, and may choose between full cage or a visor if they are older. Mandatory visors were introduced in European and international leagues with a "grandfather clause" which exempted some players from using a visor. We take that into account when coding the variable M_l.

P.O. Box 6222
Indianapolis, Indiana 46206
Telephone: 317/917-6222

Shipping/Overnight Address:
1802 Alonzo Watford Sr. Drive
Indianapolis, Indiana 46202

www.ncaa.org

MEMORANDUM

October 26, 2011

TO: NCAA Men's and Women's Ice Hockey Coaches, Conference Commissioners and Supervisors of Officials.

FROM: Steve Piotrowski, secretary-rules editor
NCAA Men's and Women's Ice Hockey Rules Committee.

SUBJECT: Equipment Rules Guidance and Clarifications.

The rules committee has received several requests for clarification regarding the proper use and how equipment shall be worn in accordance with NCAA Ice Hockey Rules. It has been requested that the rules committee provide some guidance in this area.

As a reminder, it is the responsibility of each member institution coach, athletic trainer, and equipment manager to be knowledgeable and informed of the NCAA Ice Hockey Rules that govern all protective equipment. Student-athlete safety is of the utmost importance. Institutions must be diligent in properly equipping the student-athletes to be fully compliant with all NCAA rules and regulations.

Equipment shall not be modified from its original manufactured state and shall be worn in the manner the manufacturer intended it to be worn.

As a further reminder, it is the responsibility of each member institution coach, athletic trainer, and equipment manager to be knowledgeable and informed of the NCAA Ice Hockey Rules that govern all protective equipment. Institutions must be diligent in monitoring equipment used and be in compliance with all NCAA rules and regulations. Appropriate rules are listed below and full language is listed in the NCAA ice hockey rules book (available for download free of charge at www.ncaapublications.com):

Mouth Guard

RULE 3, SECTION 6. All players are required to wear an internal mouth guard that covers all the remaining teeth of one jaw. The mouth guard shall not be altered from original manufacturer specifications, except with the prescription of a medical authority.

PENALTY—For initial violation, offending player shall be immediately replaced on the ice, and the referee shall warn the offending team that subsequent violations by any player of that team shall result in the player being assessed a misconduct.

Note: It is recommended that mandatory protective mouth guards be made from a colored material for easy identification.

Uniforms

RULE 2, SECTION 1.b. Each player and each goalkeeper listed in the lineup of each team shall wear an individual identifying number at least 10 inches high on the back of the jersey and at least 4 inches high on the front or on both sleeves. The visiting team shall wear dark-colored jerseys and stockings; the home team shall wear light-colored jerseys and stockings. All players of each team shall dress uniformly in that day's game jersey.

Team Uniform—All players of each team shall be dressed uniformly with approved design and color of their helmets, sweaters, short pants, and stockings.

Adjustment to Clothing and Equipment

RULE 6, SECTION 2. a. Play shall not be stopped nor the game delayed for adjustments to clothing, equipment, skates or sticks.
PENALTY—Minor.

b. The responsibility of maintaining clothing and equipment in proper condition shall be upon the player. If adjustments are required, the player shall retire from the ice; play shall continue without interruption with a substitute.
c. Unnecessary delay shall not be permitted for the repair or adjustment of a goalkeeper's equipment. If major adjustments are required, the goalkeeper shall retire from the ice and be replaced by the substitute goalkeeper immediately. A referee may allow for minor delays.
PENALTY—Minor.

Helmet

RULE 3, SECTION 5. a. All players are required to wear ice hockey helmets with throat straps securely fastened. There shall only be enough room between the throat strap and the chin to insert two fingers.
PENALTY—Minor.

Note: An on-ice official shall check each starting goalkeeper's throat strap before the start of the game.

All players must wear a HECC-approved hockey helmet. It is recommended that goalkeepers wear a HECC-approved helmet.

Helmets brackets ("J-Clips")

The rules committee has received several requests for clarification regarding the brackets that hold the facemask in place on the helmet (commonly known as "J-Clips") and how this impacts the HECC Certification of this equipment. It has been requested that the rules committee provide some guidance in this area.

After researching this issue with HECC representatives, the committee wishes to remind the ice hockey community that all players must wear a HECC-approved hockey helmet and facemask. HECC requires all mounting hardware of the facemask to be in place, which includes the "J-Clip". This assembly must not be altered in anyway when mounting the facemask to the helmet, as these clips are designed to help keep the face mask properly in place. The removal of the "J-Clip" further increases the risk of potential injury to a player and, according to HECC representatives, may void the HECC Certification, which would make the facemask illegal under current NCAA Ice Hockey Rules.

The committee requests that this information be reviewed with all players and equipment personnel.

Thank you for your time and attention and good luck this season.

NCAA/10/26/11/TY:kg

2012 Ice Hockey Student-Athlete Equipment Survey

Last Modified: 04/13/2012

Introduction

NCAA Men's Ice Hockey mandated the use of a helmet with full face protection and mouth guards for the prevention in injuries in the 1970s. Historically, the full mask or cage provides the highest level of safety with a reduction of facial fractures, lacerations, dental trauma and eye injury. Moving to a visor with less coverage may lead to more facial injuries, but also could improve visibility and awareness.

A group of NCAA coaches, administrators, commissioners and medical experts met in December 2011 to review many facets of this issue. A main change is the technology available for visors today (especially the ¾ visor) and the number of similar level hockey leagues that either allow or mandate the use of a visor as opposed to a partial shield.

The group decided to launch a survey of current NCAA student-athletes in an effort to gain that perspective on the issue and determine if potential changes should be considered.

Survey Instrument/Process

Survey administration involved an online survey tool. A link to the survey instrument was sent to all NCAA head ice hockey coaches, they were asked to then forward the link to their student-athletes for completion. A rough estimate of the Division I Men's Hockey institutions suggests about 1,600 players and 1,038 responded to this survey.

1. What year in NCAA eligibility are you?

Answer		Response	%
Freshman		333	32%
Sophomore		333	32%
Junior		238	23%
Senior		134	13%
Total		1,038	100%

2. Before NCAA play, in what level did you participate?

Answer		Response	%
USHL		168	16%
High School		162	16%
Junior Program		642	62%
Other, please specify		67	6%
Total		1,039	100%

For this question, an "other" option could be selected that provided the opportunity to write in a response to question 2. The themes that emerged are the following:

Other, please specify	
Prep School	18
NAHL	15
BCHL	10
Sweden	7
AJHL	5
NTDP	4
Junior Hockey	4
EJHL	3
AAA	1
NAHL	1
High School	1
Club Hockey	1
Midget	1
SJHL	1
OPJHL	1
Westminster School	1

3. Have the NCAA's contact to the head/hitting from behind penalties changed the way you play the game?

Answer		Response	%
Yes, I'm more careful with how I check.		381	37%
I'm somewhat more careful with how I check.		336	32%
No, not at all.		317	31%
Total		1,034	100%

4. Have you played with a ¾ shield before? (Pictured below)

Answer		Response	%
Yes		535	51%
No		504	49%
Total		1,039	100%

5. How, if at all, do you feel that your play differs with a full shield versus a ¾ shield?

Answer		Response	%
No difference at all		274	52%
Less aggressive		136	26%
More aggressive		82	16%
No Opinion		34	6%
Total		526	100%

6. Do you feel safer wearing a full shield versus a ¾ shield?

Answer		Response	%
Yes		110	21%
No		338	64%
No Opinion		78	15%
Total		526	100%

7. Have you had an injury playing with a ¾ shield that you would not have had with a full shield?

Answer		Response	%
Yes		130	25%
No		397	75%
Total		527	100%

8. What was injury did you sustain when playing with a ¾ shield?

Answer		Response	%
Facial laceration		82	63%
Eye injury		1	1%
Dental		37	28%
Fracture		5	4%
Spinal Injury		0	0%
Other, please specify		5	4%
Total		130	100%

For this question, an "other" option could be selected that provided the opportunity to write in a response to question 8. The themes that emerged are the following:

Other, please specify	
Lacerations	2
Broken Jaw	2
Dental/lacerations	1

9. What would be the benefits of wearing a ¾ shield? (Mark all that apply)

Answer		Response	%
Improved vision		493	95%
Keeping sticks down		475	91%
Mutual respect between players		487	93%
Safer environment		365	70%
Other, please specify		118	23%

For this question, an "other" option could be selected that provided the opportunity to write in a response to question 9. The themes that emerged are the following:

Other, please specify	
Better Preparation for Professional Hockey/Looks More Professional	21
Less Hits to Head	15
Better Quality Hockey	14
Increased Awareness of Actions	14
Less Concussions	13
Less Injuries to because of Cage	7
Improved Breathing	5
Less Overall Fighting	5
Less Injuries	3
More Offense/Better Shots	3
Looks Better	2
Increased Vision	1

10. If given a choice, would you decide to use a ¾ shield as opposed to the current full shield?

Answer		Response	%
Yes		836	83%
No		111	11%
No Opinion		55	5%
Total		1,002	100%

11. If the NCAA allowed ¾ visors, should all players be required to wear the ¾ shield (with the exception of an injury)?

Answer		Response	%
Yes		774	77%
No		183	18%
No Opinion		43	4%
Total		1,000	100%

12. Please rate the level of aggressiveness in the following categories:

Answer	Min Value	Max Value	Average Value	Standard Deviation	Responses
Your aggressiveness on the ice	0.00	10.00	7.32	1.93	996
Your teammate's aggressiveness on the ice	0.00	10.00	7.05	1.62	997
Your opponents' aggressiveness on the ice	0.00	10.00	7.14	1.52	997

13. Do you feel the current helmet fits securely to your head so that it does not move upon impact?

Answer		Response	%
Yes		773	77%
No		188	19%
No Opinion		41	4%
Total		1,002	100%

14. If you choose to wear a ¾ shield, wearing a mouth guard would be strictly enforced. Would you have any issue with this?

Answer		Response	%
Yes		83	8%
No		888	89%
No Opinion		28	3%
Total		999	100%

15. If you play professional hockey, will you plan to wear a protective face shield?

Answer		Response	%
Yes, the minimum allowed		606	63%
Yes, a ¾ shield		231	24%
No, I will not wear a shield unless forced to.		118	12%
Total		955	100%

16. Please include any other thoughts you may have on this topic.

The following themes emerged:

Prefer Half Shields/Visors	67
Support the use of a 3/4 shield	14
Keep it the way it is because otherwise the number of head injuries will increase	7
Cages are problematic, cause concussions, are not wanted	5
Shields will help prepare for professonal hockey	3

If Shields are used, it needs to be consistent for everyone	2
Shields will make players more aware of surroundings	2
Shields should be optional	1

The following survey was sent to all 58 NCAA Division I Men's Ice Hockey athletic trainers:

1. Should NCAA Division I Men's Ice Hockey allow the visor to be allowable equipment?
   Yes No Don't Care

2. If the visor is approved: should only the visor be allowed or should the use of full facial protection continue to be allowed as allowable equipment?

   Visor only Both allowed

3. Number of catastrophic neck injuries at your institution since 1990 and their current status (paralysis, complete recovery)
4. Number of years as athletic trainer in college ice hockey:
5. Have you had experience working in a league that allowed the visor?

   Feel free to make any other comments/suggestions.

RESULTS:

44/58 responded	76%
HE	6/10
ECAC	10/12
CCHA	8/11
WCHA	10/12
AHA	10/12
IND	0/1

1. Yes 16 (37%) No 19 (43%) Don't Care 9 (20%)
2. Visor Only 24 (55%) Both 20 (45%)
3. 7 neck injuries total; 2 quadraplegic, 4 total recovery, 1 recovering
4. Avg. experience 11yrs. Range: 1-30yrs
5. 4/44 have experience with visor league (USHL)

- I do think it should be all or none (with the exception of injury - jaw etc.) otherwise the point will be lost. We need to ready for more facial and dental injuries, particularly during the "learning curve" time. Pucks to the face as well. I feel like I see a puck go off a cage at least once a game. The sticks zip the puck so quickly that there will be conditioning needed in this area as well.

- If removing protective equipment around the head/face, decreasing the influence that “hard shell” elbow pads and shoulder pads have on the head/face should occur at the same time.

- I have felt for years that college hockey should go to the visor, the athletes play with such a reckless abandon developed over the years from having every square inch of their body covered while they play. They give and receive checks with their hands and sticks up, punch guys in the cage after the whistle; a rule has been added just for grabbing and pulling on the cage. I also believe that the number of boarding and checking from behind penalties would decrease with the cage removed, kids might not go after a guy on the boards with such ferocity if their face might be the next one exposed to the boards. The players come to us from juniors wearing the visor and if they go on to the next level they will be wearing the visor so I think college hockey should move in that direction as well.

- Anyone who thinks taking away the cage is going to decrease injuries is delusional. If it decreases any injuries facial lacerations, dental injuries etc. then why would we want to change it? I have one athlete who showed up here this past Sept. after he took a cross check to the jaw (visor only - Tier II, jr. A) his jaw was dislocated and he has not fully recovered from a concussion he suffered at the same time in March of last year. We have sent him to several neuro-psychologists who have said he should never play hockey again. He has yet to practice and has finally excepted the fact that he is done playing hockey

- At a time when concussion management is the hottest topic in athletic medicine not providing our student athlete’s with the best protection available would be tragic.

- I do care about this though, and feel that a full shield is most likely the safer choice for athletes overall for obvious reasons. That being said, I also concede that a visor option may make the game more marketable and therefore be good for college hockey. If the NCAA is willing to accept the risk of more facial and teeth injuries with half shields then why not. As we’ve talked about, there needs to be a culture change in the game to cut down on elbows, high sticks and head hunting in all leagues – half shields by themselves won’t do this as proven by the major junior leagues. Moving to a visor will not decrease head injuries despite any argument – research supports this.

- I agree with many of my players that use of the visor will reduce some of the reckless play that currently takes place in NCAA hockey. I suspect the first year after implementation (if it occurs) will be a somewhat difficult year as players get used to the change.

- First, thanks for asking for our opinions. At first glance, I would say that I am on the fence about transitioning to only using half shields in college. I would say in some respects it may help to keep elbows down, etc. because they are more afraid of repercussions, but on the other hand I would suspect we have a huge increase in facial lacerations and dental injuries. So my major question to the NCAA would be if they will then require actual enforcement of the rule of mouth guards and/or will they require that schools purchase dental insurance for the DI players. Those are the questions I would have.

- Comments: In regards to the allowance of visors into college hockey, I think there is some benefit. It does create an atmosphere where a player must be attentive to the high sticking rule in the sport. In many cases, players are currently more erratic with their stick as they don't have the same level of caution that would be necessary with a visor. While, this does increase the risk of facial injury in the sport, the hope is that the level of talent that these collegiate players possess should be enough to avoid shooting high. I also think the learning curve would be quick and smooth in terms of adjusting to stick control.

- If the argument is that it will "make the game safer", than only the visor should be allowed! It would be hypocritical to use that argument, but still allow the ffp.

- I do not see the benefit to move to the visor. It does not decrease head/neck injuries and increases facial injuries.

- I also voted that if visors are approved- visors should only be available for use, unless injury prevents that. I do not think that you can have some wearing visors and some not- increases risk for injury to those wearing visors.

- Seems silly to remove protection when everyone agrees injuries will increase.
- I think this is a tough topic. It can be defended well for both sides. I think it will be difficult to remove protective masks but I believe it may prevent the hard, full speed hits delivered by someone with a mask. They are less likely to come in hard with the face forward to hit if they don’t have full protection on their own face. However, if the rules are not enforced for elbows and shoulders to the head we are going to continue to see concussions rise in frequency. Hard to prove this theory but, I believe, if we remove masks and enforce the rules with stiffer penalties, it will lead to less concussions and head injuries. Again, hard to prove.

- I still think, ultimately the mask will stay on for liability reasons. How do you justify removing a protective device….

- . I really think it would be a mistake to take the facemasks off of the college players. While I understand the argument for doing it, apparently the data doesn't support the theory and I think we would have some bad facial injuries as a result.

- No, because a number of facial, and dental injuries occur during practice and game. I also would like to mention physicians (dentists and plastic surgeons, etc) accessibility for games and practices during weekend. I am not sure dentist, plastic surgeons, etc. are available for every D-I hockey program. On the other hands, it has been discussed last few years. So I have been preparing / discussing with my superior / administration and other athletic trainers who have hockey experience. I am thinking using visor will be starting sooner or later.

  I am not sure about level of contact rule to head if wearing visor is allowed. I am hoping that they do not target opponent head as much when player start wearing visor. Again I am not sure. Allowing visor or full protection is not working well because of confusing their physicality during game. For example, we had exhibition games against Canadian teams last 3 seasons, they had 3-4 facial injuries (lacerations most) every year. Our players and official thought it was clean hit but Canadian player got facial injury.

- Comment: This proposal makes no sense as it will definitely cause an increase in injury rate. Why would players or coaches want athletes to miss more time off the ice potentially caring for an injury during a contest or missed practice opportunities?

- I agree with the last statement above, based on all the numbers I've seen. The rationale for the change is very unclear to me. The rationale that switching to the visor will change the way the game is played and therefore reduce the risk of injury is unfounded and rather baseless. The numbers certainly don't support it. So essentially proponents of half shields want to trade increased facial injuries and increased risk of eye injury for no change in any other type of injury? I've heard coaches try to explain that the half shield will change the hits from behind because it will usher in more "respect". Again this is an unfounded argument, especially since the enforcement in college hockey is rules and penalties versus fighting and "respect "in the pros. If another guy has on less protection why would my player care about his well-being? There's little consequence for hitting him.

- The real problem for the dangerous hits falls on the rules committee, the referees, and the conferences—the definitions of illegal hits are embarrassingly inconsistent, subjective, and not enforced. The players are not coached to hit properly. The referees don't call the hits properly. No one can agree on what's an illegal hit.

- I understand the idea that removing the full facemask may help the players play with a little more respect, reduce some of the "stick-work," and overall reduce serious injuries to the head and neck. However, I am not sure that the above is worth the potential for serious eye, dental, and facial injuries that would come with going to visors.

- I did not have many injuries in the USHL with the visor as opposed to the Full cage. Actually I had many impact lacerations from the chin cup and only two facial lacerations to players with a visor. Only 1 dental injury and I worked well over 120 games.

- I think though if we do go to visors we need to adopt the rule that the chin strap MUST have contact with the skin as they do in Canadian junior leagues and this must be enforced by the officials. I saw several players that would basically push their helmet back so the visor was not even over their face and this was causing the helmet to be worn improperly. This has to start with the officials on the ice!
- If the visor is allowed, have the NCAA make it **mandatory** for all schools participating in NCAA men's ice hockey to have a secondary insurance policy to cover all medical costs for the athlete while participating in an athletic event/practice.

If the visor is allowed, the use of mouth guards worn by athletes must be better enforced by officials.

If the visor is allowed, better enforcement of high hits by officials.

NCAA Men's Ice Hockey – Use of Face Shields
December 15-16, 2011 Meeting – Ann Arbor, Michigan

Issue overview: The NCAA Men's Ice Hockey community has requested a review of face shield protection by the NCAA Ice Hockey Rules Committee. The NCAA mandated the use of full face shields in 1980-81 in order to reduce facial injuries in the sport. Since that time, technology has improved and the concept of visor protection and warrants a review of available equipment and the potential application for NCAA men's ice hockey.

2011 NCAA Rules Survey Results: In the survey compiled for the 2011 Ice Hockey Rules Committee annual meeting, 96 percent of Division I men's coaches were in favor of pursuing the use of half shields as a legal piece of equipment; in other recent surveys, the response was 100 percent in favor.

Potential reasons to allow visors in NCAA play:

- Better vision allows student-athletes to better avoid and prepare for contact.
- Visor technology has improved dramatically and protects the eyes/nose/face much better than early versions.
- Self awareness would keep sticks down and lower the checking target.
- Adjustment from junior hockey to the NCAA level would not require a change in equipment from what student-athletes are used to.
- USHL data indicates that visors are appropriate for male players in this age group.

Potential issues with allowing visors in NCAA play:

- Data does not show evidence that playing with a visor is safer than full shields in protecting against catastrophic injury.
- Data indicates that more facial injuries may occur by using visors as opposed to full shields.
- There are some questions with regard to the legal ramifications – would student-athletes sign a waiver to be allowed to wear these shields?
- The rules committee has been clear that full shields would continue to be legal; would this create an issue when some players wear visors and others wear full shields?

Potential areas of research/additional communication:

- Student-athlete survey.
- Review of legal questions/ramifications.
- Support of further research (if needed) to close gaps in available data.

Intended outcomes of the meeting:

- Clear understanding of the NCAA rules process.
- Thoroughly review changes with equipment and style of play.
- Determine what information is needed or would be helpful for the Ice Hockey Rules Committee and Competitive Safeguards Committee to take a position on this issue.
- Finalize timeline for potential decision/implementation if approved.

EXPECTED ATTENDEES

NCAA Committees/Membership Representatives

Ed McLaughlin, NCAA Ice Hockey Rules Committee chair, Niagara University (meeting chair)
Tom Anastos, coach, Michigan State University
Red Berenson, coach, University of Michigan
Brant Berkstresser, Harvard University assistant AD/certified athletic trainer/CSMAS member
Frank Cole, NCAA national coordinator of officials
Dan Hendrickson, University of Michigan team physician
Jeff Jackson, coach, University of Notre Dame
Jack Parker, coach, Boston University
Steve Piotrowski, NCAA secretary-rules editor
Fred Pletch, commissioner, Central Collegiate Hockey Association

Invited guests

Dr. Paul Comper, neuropsychologist/researcher, National Hockey League advisor
Tom Dompier, Datalys (NCAA injury data collection)
Paul Kelly, College Hockey, Inc.
Artie Poidras, University of Massachusetts, Lowell – National Athletic Trainers Association
Skip Prince, commissioner, United States Hockey League
Dr. Michael Stuart, physician/researcher, USA Hockey advisor (may join via teleconference)

NCAA Staff

Ty Halpin, Championship and Alliances, Playing Rules and Officiating
David Klossner, Health and Safety
Naima Stevenson, Legal Affairs

AGENDA/SCHEDULE
*****Meetings will be held at the Marriott Ann Arbor/Ypsilanti in Salon 1 Room*****

December 14, 2011 (Attendee arrival on site – no meetings scheduled this day.)
Note: Please arrange transportation to the hotel and we will reimburse you for this expense.

December 15, 2011

7:30-8:30 a.m.	Breakfast available. (NCAA provides)
8:30-8:45 a.m.	Opening remarks/welcome. (Ed McLaughlin, NCAA rules committee chair)
8:45-9 a.m.	Overview of NCAA rules process. (Ty Halpin, NCAA/rules committee)
9-9:15 a.m.	Overview of Competitive Safeguards input. (David Klossner, NCAA/CSMAS)
9:15-9:30 a.m.	Brief overview of issue. (Paul Kelly, College Hockey, Inc.)
9:30-10:15 a.m.	Discussion of coaching viewpoint. (Jack Parker, Boston University; Jeff Jackson, Notre Dame; Tom Anastos, Michigan State; Red Berenson, Michigan)
10:15-10:45 a.m.	USHL experience. (Skip Prince, commissioner)
10:45 a.m.	Break.
11-11:45 a.m.	Further discussion.
11:45 a.m.- Noon.	Dr. Michael Stuart, Mayo Clinic/USA Hockey. *Call info: 866/590-5055, 1579483*
Noon-1 p.m.	Lunch. (NCAA provides)
1-1:30 p.m.	Overview of research data. (Dr. Paul Comper)
1:30-2 p.m.	NCAA Injury Data Review. (Tom Dompier, Datalys)
2-2:30 p.m.	Discussion of equipment and potential student-athlete survey. (All)
2:30-3 p.m.	Officiating and rules enforcement. (Steve Piotrowski/Frank Cole)
3-5 p.m.	Additional discussions/adjourn/dinner on own. (All)
6:30 p.m.	Shuttle departs hotel for Team USA-USHL game.
Postgame	Shuttle returns to hotel.

December 16, 2011

7:30-8:30 a.m.	Breakfast available. (NCAA provides)
8:30 – 10 a.m.	Follow-up items (if needed).

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Sports Injury Research and Prevention

NCAA Injury Surveillance Program

Report to the NCAA Ice Hockey Rules Committee

Thomas P. Dompier, PhD, ATC

December 15, 2011

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Sports Injury Research and Prevention

Overview

The Injury Surveillance System began in 1982, two years after the NCAA mandated full face masks in 1980. Surveillance of hockey began in 1988.

The NCAA currently maintains two datasets, 1988-2004 (16 years) and 2004-2009 (5 years). Data collection is ongoing.

There is no historical NCAA data to compare before and after the institution of full face masks, except that which was published in scientific journals and media reports.

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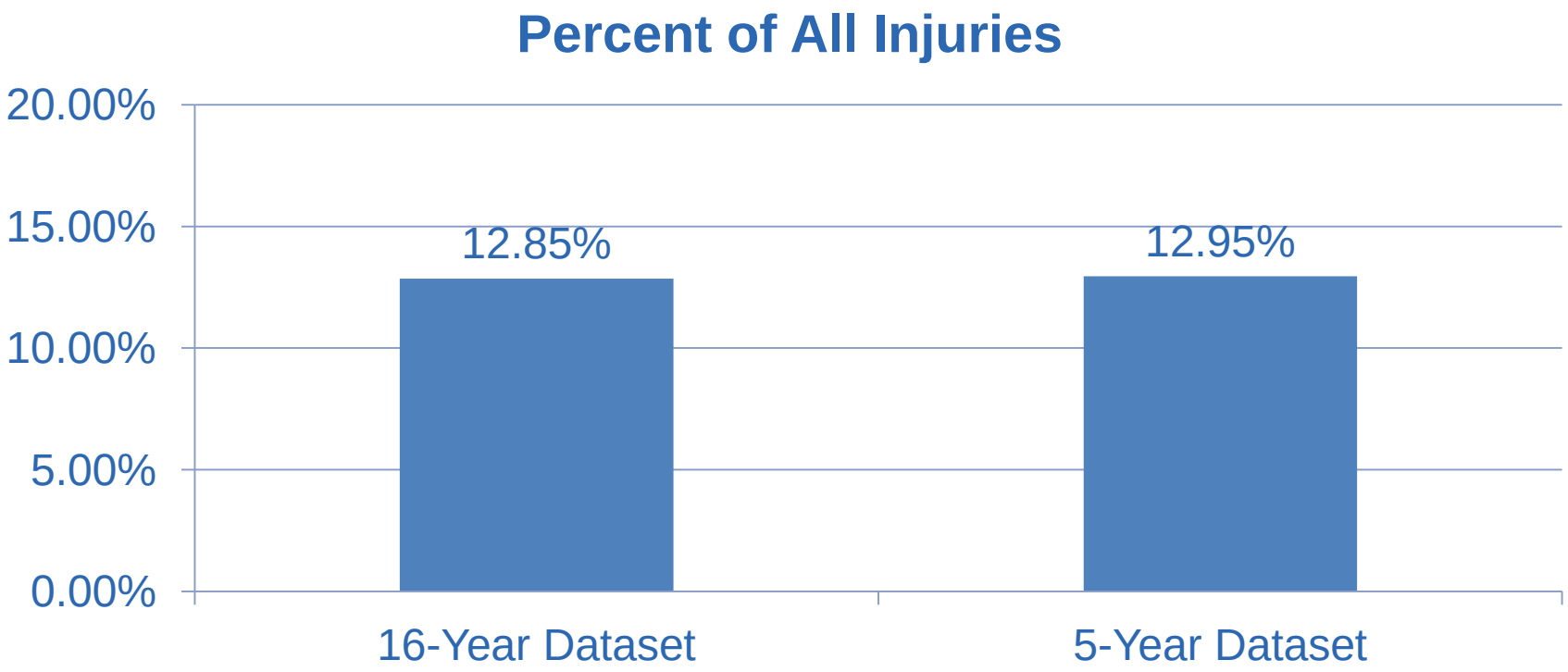
Sports Injury Research and Prevention

Injuries to the Head and Face



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Comparison to Historical Data

- Prior to widespread use of full face shields, injuries to the head and face were reported to be between 37%-64% of all injuries, compared to 13% in the NCAA since full face masks mandated (Vinger, 2008)

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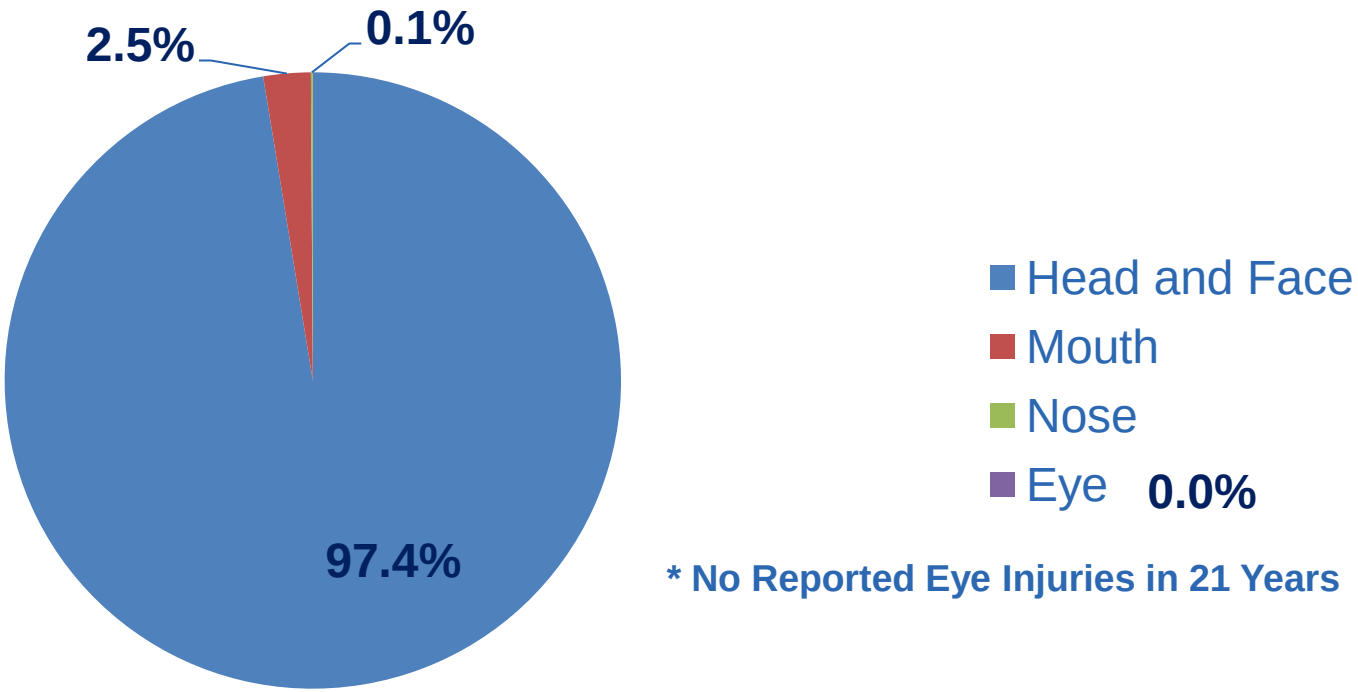
Sports Injury Research and Prevention

Location of Injuries to the Head



*** No Reported Eye Injuries in 21 Years**

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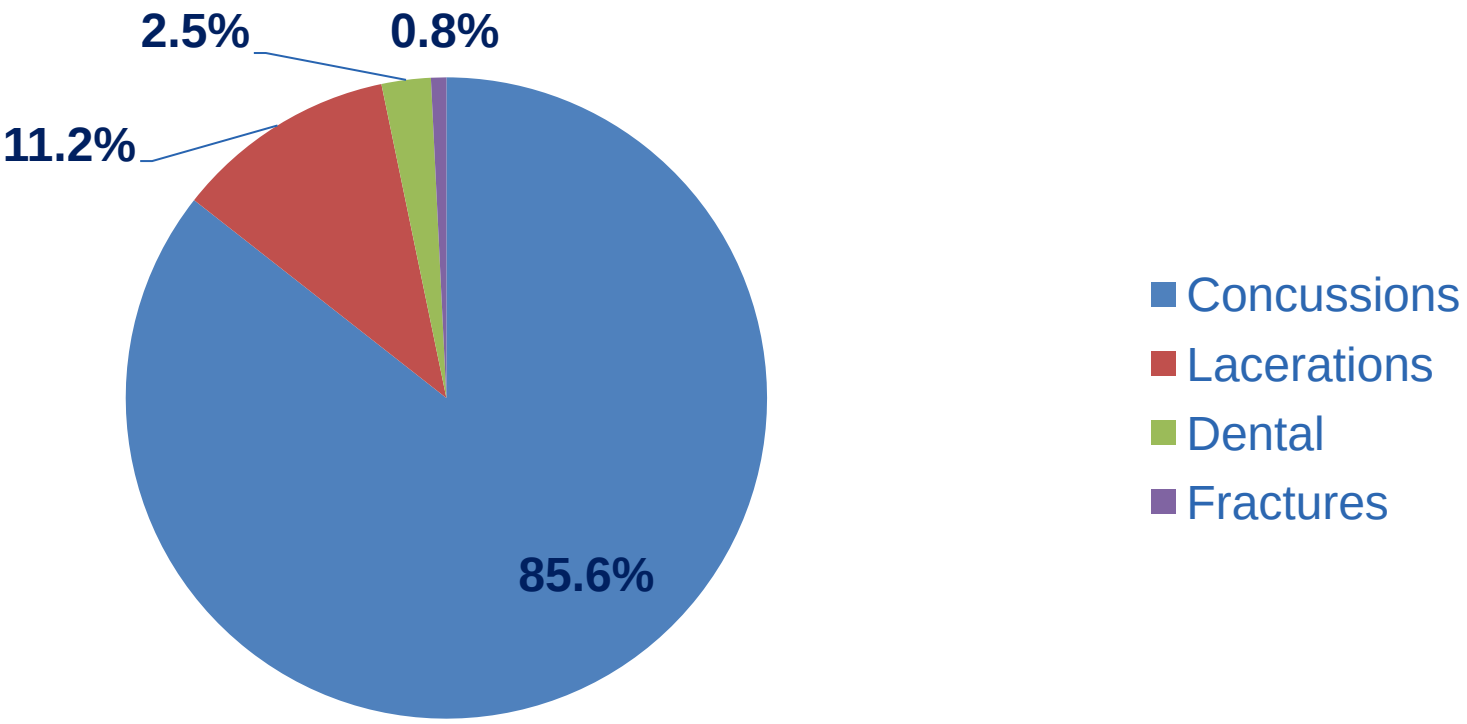
Specific Injuries to the Head & Face



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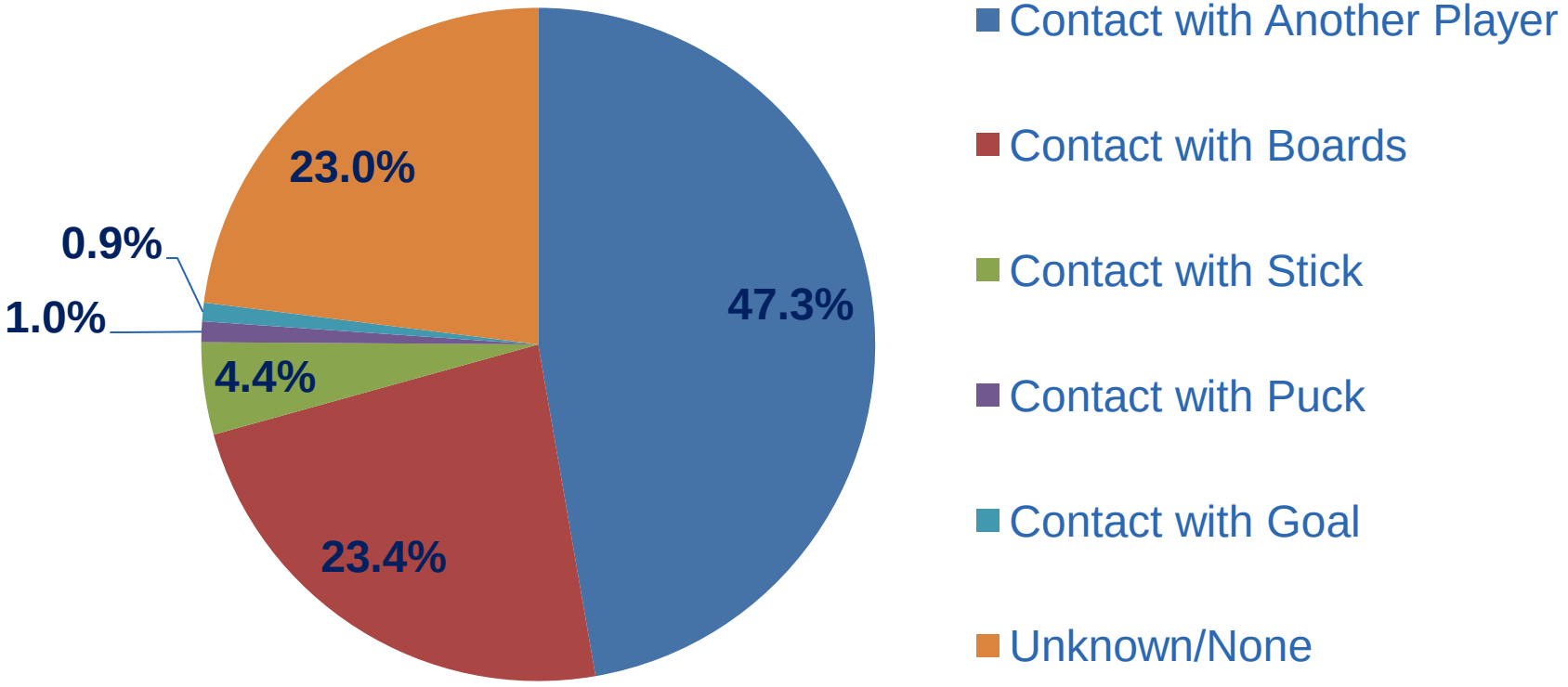
Mechanism of Injury to the Head



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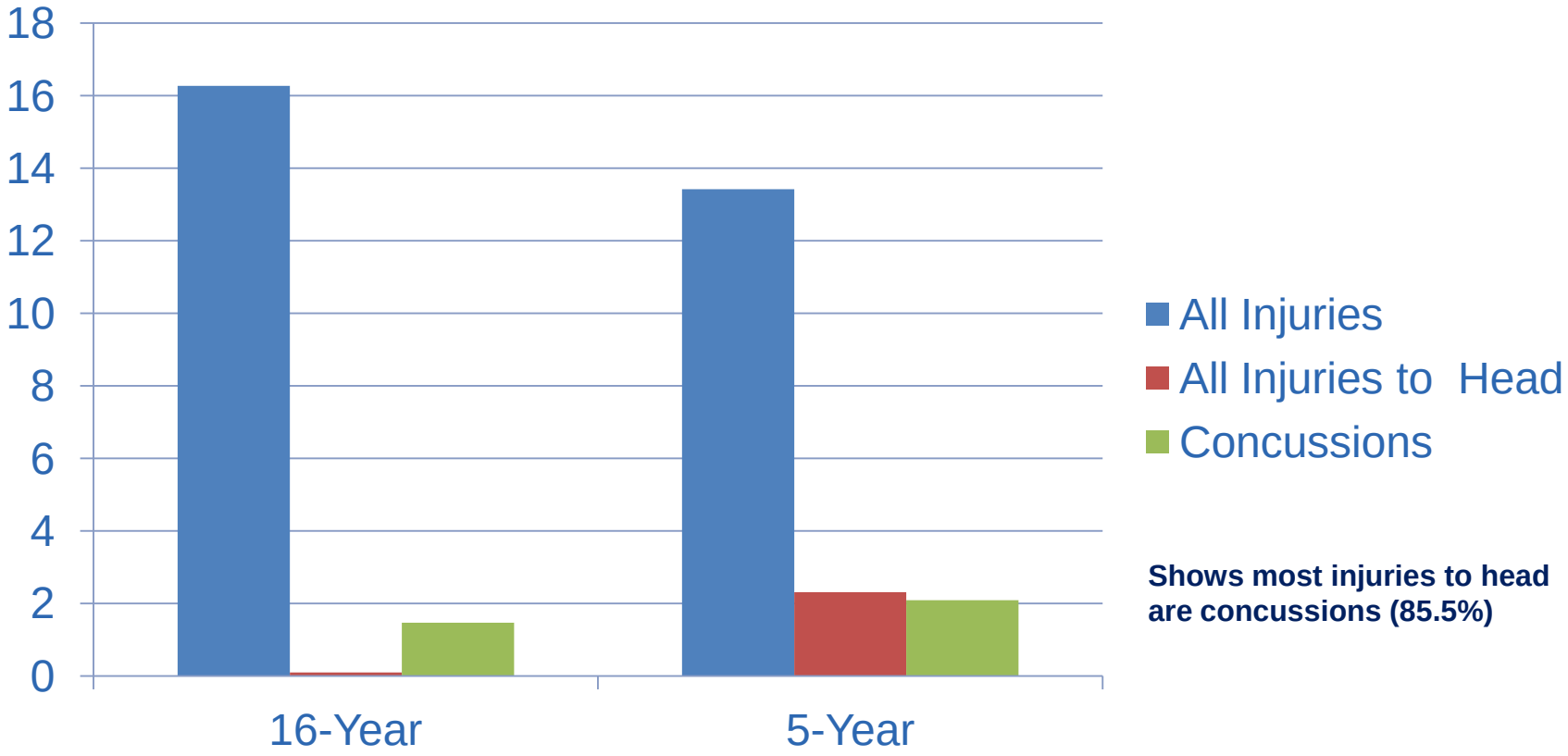
Injury Rates in Games

per 1000 athlete exposures



Shows most injuries to head are concussions (85.5%)

*** All head Rate Not Available**

NCAA® News Release

FOR IMMEDIATE RELEASE
Friday, June 11, 2010

CONTACT:
Ty Halpin
Associate Director for Playing Rules Administration
317/917-6136

NCAA ICE HOCKEY RULES COMMITTEE PROPOSES CHANGES TO STRENGTHEN CONTACT TO HEAD RULES

INDIANAPOLIS -- The NCAA Ice Hockey Rules Committee proposed changes to its rules to enhance student-athlete safety and to continue to encourage speed, skill and scoring chances in the game. The committee's proposals must be considered by the Playing Rules Oversight Panel in July before implementation.

The committee approved a more stringent rule and enforcement standard of its contact to the head rule. A violation will carry a minimum of a major penalty and a game misconduct or disqualification penalty. A major penalty calls for the offender to be ruled off of the ice for five minutes, during which time a substitute shall not be permitted.

"Player safety is critically important to the college hockey community," said Forrest Karr, chair of the committee and director of athletics at the University of Alaska Fairbanks. "Players will be penalized severely for contact that targets the head and neck area."

Student-athlete safety is of the utmost importance to the NCAA and the committee, as witnessed by the NCAA's contact to the head rule originally being passed in 2003. In addition, the committee believes the NCAA's strong stance on hitting from behind has altered player behavior, making the game safer.

The committee also approved changes to its icing rule to combine the current automatic procedure with the touch-up system used professionally. In the new model, the official determines which player will reach the puck first, using the faceoff dots as a reference point. If it is determined to be the attacking player, icing is waved off. If it is the defending player, icing is called. A tie goes to the defender.

In addition, the committee approved a rule to enforce icing throughout the game. Previously, shorthanded teams were allowed to ice the puck. This new rule has been used in USA Hockey Player Development Camps.

“In keeping with the committee’s philosophy to encourage skill and create scoring chances, this change will enhance power play opportunities,” Karr said. “After lengthy discussion, the committee concluded that the previous rule inappropriately provided relief for a team that committed an infraction.”

The committee members addressed a proposal to allow optional half shields as legal equipment for men’s ice hockey only. The committee identified the need for additional scientific data before a formal proposal can be forwarded to the Committee on Competitive Safeguards and Medical Aspects of Sport.

In other actions, the committee:

- Kept overtime largely unchanged, with the exception of having goalkeepers change ends.
- Removed the obtainable pass rule.
- Altered the delayed penalty rule to provide the non-offending team a power play, even if a goal is scored during the delay.
- Appointed Ed McLaughlin, Niagara University director of athletics, as chair.

College Hockey: Shielded From Harm

Candace Horgan
USCHO.com
January 31, 2008

Jan. 31 — Those who follow college hockey might have gotten a surprise if they watched the 2008 World Junior Championships. Whether the player was Wisconsin's Kyle Turris, Minnesota's Ryan Flynn, Michigan's Max Pacioretty, or New Hampshire's James vanRiemsdyk, almost every college hockey player participating in the World Junior Championships had ditched the ubiquitous full face mask of college hockey for the half shield seen so often in the NHL.

Ask the NCAA why it requires the full facemask in college hockey and the cited reason is injury risk.

"Basically, it stems from findings of a study done by Dr. Alan Ashare that shows full shields prevent more than 70,000 eye and face injuries per year," said NCAA spokeswoman Jennifer Kearns. "We've done some research through our injury surveillance systems and it has proven that these masks prevent a lot of eye and face injuries and that there haven't been any blinding eye injuries in any player who was wearing full face protection. So, the NCAA, our membership has decided that this is a good way to go to protect our student-athletes and their eyes and their vision. It's just a precaution."

The use of face shields in the college game is a relatively recent development. The NCAA started recommending the use of face shields at the start of the 1978-79 season. Eastern teams started wearing the face shields in 1979, and the NCAA required their use by all teams at the start of the 1980-81 season.

For a brief period, you could have two teams sporting different levels of protection, and sometimes problems arose. When the New Hampshire Wildcats played the Ohio State Buckeyes in November 1979, the Wildcats sported face shields while the Buckeyes did not. Several fights broke out, and the Wildcats ended up forfeiting two games to the Buckeyes.

This incident seems to bolster the opinion of Dr. Michael Stuart, the chief medical officer for USA Hockey, who feels that if the NCAA ever allows visors instead of full face shields, it has to be universal.

"Part of the problem as I see it is uniformity; my honest opinion is, and I'm not saying you should, but if the NCAA wants to make a change, that change should be all or none, meaning everybody wears a visor, or everybody wears full protection," said Stuart. "If some players decided to wear full facial protection and some decide to wear visors, then the argument that the sticks may come down only applies to part of the group, so those who do not have full facial protection are at increased risk."

Given that more and more college players go on to play in professional leagues, an argument could be made that players should start to get used to playing without the cage earlier. Some also believe that use of full cages has led to an increase in high-sticking and roughing penalties, and a decrease in respect between players.

Denver Pioneers sophomore forward Tyler Ruegsegger found that wearing the visor in the World Junior Championships did make him more aware of his stick. “It definitely makes you more aware of keeping your stick down on the ice when you get high-sticking penalties, things like that.”

“From a player’s standpoint, or at least from what they tell me, they are more respectful, at least with high-sticking, if you are not wearing a face mask and your opponent is not wearing a face mask,” said Stuart. “There’s a lot of interesting and I think valid reasons why you would want to go to a visor compared to a face mask.

“Some proponents in the NCAA would say you should take the mask off because a full face mask gives players a sense of protection so they become more reckless, especially with high-sticking, and they become more reckless with body-checking and using their head as a weapon, therefore increasing the risk of concussion or cervical spine injury, although that has never been proven. Numerous studies have not shown an increased risk of injury to the head or neck in players who wear full facial protection.”

If you ask the players, you get a variety of opinions, although the fact that many players abandoned the full shield at the World Junior Championships seems to indicate a lot would do the same in the NCAA if they had the option.

“I hate the cage,” said Wisconsin Badgers’ star freshman Kyle Turris. “It’s something that you have to play with, but I really don’t like it. It feels like it always gets in the way. It’s just not very comfortable. I feel like it obstructs my vision a little bit too, but that’s part of college. It’s just something that would be nice if the NCAA looked into. I would definitely wear a half shield if I had the opportunity.”

Ruegsegger has a more mixed opinion on the half shield, and is fine with wearing the cage in the college game.

“As far as the cage and the half shield, I like the cage,” said Ruegsegger. “I don’t know; it’s my preference personally. The divider sometimes gets fogged up or scratched, so I prefer the cage.”

Of course, when Ruegsegger has participated in camps with the Toronto Maple Leafs, he has worn the half shield.

“I’ll do what the Leafs say to do,” laughs Ruegsegger. “I mean, you get hit in the jaw here or there, stuff like that, but if the other players are playing smart, with their stick down, I think it’s all right.”

Despite the fact that many like Turris would probably go to a half shield if the NCAA allowed it, the NCAA hasn't looked at it at this point.

"The staff liaison who works with the NCAA rules committee says that this does come up now and then because of the professionals not using a full face protector, but there hasn't been anything proposed to change it," stated Kearns.
"Our member institutions, just to make sure our players are protected and safe, that's what our membership has decided is best for our student-athletes."

Pioneers' coach George Gwozdecky says that it has been looked at, but he doesn't see the face shields coming off.

"They've looked at it for years, and as soon as the medical people get involved, and the lawyers get involved, the discussion ends. There's no question that someone's health is without a doubt the top priority for our medical people, our trainers, the safety standards people, so as a result, the health of the student-athlete comes first."

While the use of visors does decrease the incidence of injury to the face or eye, it doesn't completely eliminate it. Players have suffered serious injuries even while wearing the visor. In his studies, Stuart found that 95 percent of players at the NHL level injure their face at some point in their career, including facial fractures, eye injuries, lost teeth, and lacerations.

A study by the American Medical Association found there was a 2.31 times greater risk of players sustaining a facial laceration or dental injury when wearing a half shield vs. a full shield.

Stuart believes there are valid arguments for both using a half shield and using a full shield, but also indicates that if the NCAA goes to the half shield, there would be more injuries.

"I agree with a lot of the arguments on both sides, but it's clear to me that if you remove full facial protection in the NCAA, you will have an increased incidence of injury to the face, and accompanying those injures to the face could be injuries to the eye, and those could be serious and permanent. Maybe it won't happen, but there certainly is that risk."

Risk Compensation

A "Side Effect" of Sport Injury Prevention?

Brent Hagel, PhD, and Willem Meeuwisse, MD, PhD†*

It is discouraging, to say the least, that countermeasures (e.g., protective equipment use) designed to lower the burden of injuries in sport and recreational activities do not warrant the same scientific scrutiny required in almost any other field of health research. New drug therapies require extensive research before they are adopted into medical practice. Yet there are countless examples of untested, unregulated countermeasures designed, albeit with good intentions, to lower the burden of injury in a particular sport or recreational setting. As William Haddon Jr. et al[1] so eloquently stated,

■ *One can, of course, argue that the introduction of essentially unevaluated accident prevention measures "can't do any harm," but two potential dangers in this approach need to be noted. First, the introduction and enforcement of insufficiently evaluated measures may lead to an inappropriate choice of emphasis and may, as a result, dissipate funds, time, and public concern that might be applied to more effective measures. Secondly, the public and its government may conclude that everything that can be done is being done.*

We would add that the possibility exists that the countermeasure in question may have other negative unintended consequences. These effects may include simply shifting the distribution of injury, a change in the behavior of participants resulting from a false sense of security, to reduced participation in the activity due to public discontent if the safety measure is imposed.

There are many examples in sport and recreation in which concern has been raised over the introduction of protective equipment. These include wrist guard use in snowboarding,[2] ski helmet use and risk of neck injury,[3,4] shoulder pad use in rugby,[5] and use of soft baseballs in Little League.[6,7] However, there are few compelling data to suggest the existence or absence of unintended effects.

RISK COMPENSATION

Some assert that protective equipment may prompt users to act more aggressively and thereby increase the potential for serious injury. This postulated phenomenon, referred to as *risk homeostasis* or *risk compensation*, hypothesizes that each person has a target level of risk they are willing to accept.[8] If a person perceives an intervention (e.g., helmet use) has lowered their level of risk, proponents of the theory argue users will change their behavior in a way that brings them back to their desired risk level (e.g., ski faster or more aggressively, on more difficult runs, through trees, or out of bounds).

Perhaps the greatest difficulty with the evidence for risk compensation is that much of it is ecologic; that is, there are few comprehensive examinations of individual risk-taking behavior before and after a safety measure is implemented. For example, increases in the rate of pedestrian injuries after the introduction of federal motor vehicle safety regulations

*Alberta Centre for Injury Control and Research, Department of Public Health Sciences, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Alberta, Canada, †Sport Medicine Centre, University of Calgary, Calgary, Alberta, Canada
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were noted by proponents of the risk compensation hypothesis, despite several methodologic issues with the analysis of their data.[9]

What is the evidence for this phenomenon in the sport and recreation literature? The following examples describe some of the salient research in different activities that bears on the issue, but are not meant to be a comprehensive literature review. Perhaps the best place to begin is bicycle helmet use because it is such a contentious area of injury research.

Bicycle Helmet Use and Risk Compensation

Two studies on bicycle helmet use offer evidence that is incompatible with the risk compensation theory.[10,11] Farris et al[10] report that helmeted bicyclists were significantly more likely than nonhelmeted bicyclists to make legal stops and use standard hand signals to indicate a turn or stop. A study by Spaite et al[11] compared the non–head-neck injury severity among helmeted and nonhelmeted bicyclists presenting to an emergency department for treatment. These investigators reported that nonhelmeted bicyclists were over 7 times as likely to sustain major trauma, defined as an Injury Severity Score greater than 15, compared with helmeted bicyclists. Together, these studies indicate that helmeted bicyclists may represent a more cautious subgroup of all bicyclists. But even if they were risk compensating, it suggests that helmet wearers were not doing so to a level that increased their risk above that ob bicyclists not using helmets.

American Football and Risk Compensation

The plastic shelled helmet was introduced toward the end of the 1940s in American football.[12] Players were taught that the initial point of contact when tackling should be the shoulder. In the early 1960s, however, there was a move to make the initial point of contact the head because of the protection afforded by the helmet. There was a noted increase in the number of tackling drill fatalities over the period 1955 to 1964 compared with 1945 to 1954, persisting to 1974. Mueller suggested, "Players were wearing full face masks and felt well protected when striking their head."[12] After a rule change that abrogated spearing (i.e., initial contact with the head) in 1976, and the adoption of standards for football helmets in 1978, the number of head and cervical spine fatalities declined even in the face of an estimated increase in sport participation. If one attributes the increase in head and neck injury fatalities to the introduction of the football helmet and subsequently more dangerous participation, this example demonstrates that accompanying rule changes can counteract any compensatory effect.

Baseball and Risk Compensation

Pasternack et al[7] noted that the rate of injury for players using a soft-core baseball was actually higher compared with players using standard baseballs. This led to speculation that when using a soft-core baseball, "Children may be more likely to take a greater risk in fielding the ball or not moving away from a wild pitch if they believe that there is a decreased risk of injury."[6] This is alarming considering that the biomechanical data suggest that soft-core baseballs do not lower the risk of fatal chest impact injury over standard baseballs.[6]

Ice Hockey and Risk Compensation

Evidence for the effectiveness of ice hockey helmets in preventing brain injuries has been available since 1968.[13] Concern over the number of deaths in ice hockey in the late 1960s and early 1970s led to the adoption of a Canadian Standards Association standard for helmets in 1973.[14] Two concerns about hockey helmets were raised. The first was that due to a biomechanical mechanism, they may increase the risk of a neck injury. A biomechanical study suggested that this was not likely.[15] However, there was speculation that the use of a helmet may alter the nature of the game, "giving players such a feeling of invincibility that they take excessive and unwarranted risks during games and practices."[15]

Benson et al found evidence to suggest that players wearing half-face shields were 2.3 and 9.9 times more likely to sustain facial and dental injuries, respectively, compared with players wearing full-face shields.[15a] In fact, these investigators found evidence to suggest greater injury severity and a greater proportion of illegal play-related injuries among those wearing half shields. Stuart et al[16] noted similar findings among elite hockey players under age 18 years when they compared the rate of head, neck, and facial injury in full, partial, and no facial protection groups. The results of both studies run counter to what risk compensation would predict.

Rugby and Risk Compensation

The relatively recent introduction of protective equipment such as helmets and shoulder pads in various forms of rugby provides another opportunity to examine risk compensation. Finch et al[17] studied the attitudes of 140 rugby union players between the ages of 14 and 16 years toward the use of protective headgear. Interestingly, players with no prior head or neck injury were more likely to report that they felt safer wearing protective headgear compared with those with a prior injury. Further, of the players wearing protective headgear, 67% reported that they played with more confidence when they wore the headgear. The authors concluded that "players report that they are more confident and able to tackle harder if they wear headgear, suggesting that a belief in its protective capabilities may influence behavior."[17] This is particularly troublesome given that recent attempts at gauging the effect of protective headgear indicate that it is not effective at preventing concussion injuries in rugby union players under the age of 15 years.[18]

After finding a sharp increase in injury rates in rugby union, Garraway et al[19] contended that it might be due to the adoption of protective equipment and suggested, "The Inter-

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national Rugby Board should place a moratorium on the use of protective equipment in competitive matches until its contribution to player morbidity has been fully assessed."

Skiing-Snowboarding and Risk Compensation

As long ago as 1962, Earle et al[20] reported on a series of ski injuries presenting to their Sun Valley medical clinic serving major ski hills in Idaho. The authors attempted to examine the effectiveness of safety bindings (multimode release bindings) and concluded that despite their use, injury rates remained unchanged through the 1950s—a time when they saw rapid increases in safety binding use. Although the authors acknowledged that the actual skiing time in 1960 to 1961 was likely greater than a decade earlier (increased skier exposure), they provide an interesting explanation why safety bindings did not lower the injury rate:

■ *. . .we wonder if wearing safety bindings may not lull the skier into a false sense of security. Thus, believing that he is protected, he skis with less caution and so deprives himself of whatever protection his release bindings may bestow. We believe that "safety binding" is an unfortunate term, which should be deleted from the skier's vocabulary.*[20]

In reports on skier fatalities, investigators generally state that the person died on a run at or below their ability level and that visibility was good.[21,22] This suggests skiers may modify their behavior in response to reduced environmental risk.

Findings of Shealy et al[23] that 35% of fatally injured skiers-snowboarders were wearing helmets support the theory of risk compensation. Helmet use estimates of uninjured populations are not typically close to 35%. Further, the circumstances surrounding the fatalities were consistent with extreme risk-taking behavior among those wearing helmets in 4 of the 6 cases in which the individual was wearing a helmet at the time of death. Whether the skiers increased their risk-taking behavior in response to wearing the helmet or they wore a helmet because they planned on taking greater risks is a subtle but important distinction with implications for the risk compensation theory.

Although far from convincing evidence, these mostly anecdotal reports and the speculation about a risk compensation effect cannot be ignored. Clear measures of this effect should be incorporated into any plan to evaluate protective equipment use in sport and recreational activities. Other possible consequences of mandating protective equipment should also be considered.

MANDATING USE

After 1 of the first rigorous studies of bicycle helmet effectiveness was published,[24] the debate began about the implementation of helmet laws. Arguments against mandatory use included the issues discussed here, but also involved societal level effects. Some investigators feared that mandatory helmet use would result in a net decrease in cyclists and the beneficial cardiovascular effects of cycling.[25] Although some evidence for this decline in ridership in adolescents after helmet laws was found in Australia, a recent Canadian study suggested no such effect.[26] The debate continues,[27,28] and all the reader can reasonably conclude is that more work is required.

SUMMARY

It was not our intention to offer an indictment of protective equipment in sport and recreation. Indeed, the evidence suggests that many of these devices do offer the individual a great deal of protection without deleterious effects. The issues presented here simply highlight the dire need for the early evaluation of protective equipment adopted in sport and recreation activities. Without examining the broad range of potential negative effects outlined here, it may indeed be the case that introducing well-intentioned countermeasures is more harmful than doing nothing. Only through well planned and executed studies can we evaluate the net benefit of our interventions and minimize the "side effects" of injury prevention strategies.

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Alberto Chong
George Washington University and IADB

Pascal Restrepo
Massachusetts Institute of Technology

August 4 2011

Institute for International Economic Policy
1957 E St. NW, Suite 502
Voice: (202) 994-5320
Fax: (202) 994-5477
Email: **iiep@gwu.edu**
Web: **www.gwu.edu/~iiep**

Peltzman on Ice: Evidence on Compensating Behavior Using a Natural Experiment from Ice Hockey.

Alberto Chong and Pascual Restrepo*

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Abstract

We provide evidence of the Peltzman effect by tracking the professional path of each hockey player that ended up in the National Hockey League from 2001 to 2006. We take advantage of the fact that visor use has not always been compulsory throughout a player's career, which allows us to compare the change in behavior of users and non-users of visors when they are forced to use them. We find that whereas the average penalty minutes per game is 0.8, visors cause a substantial increase of 0.2 penalty minutes per game. Players become more aggressive when forced to wear a visor, partially offsetting its protective effect and creating potential spillover effects to other players.

JEL: K32, K23, H40

Keywords: Peltzman Effect, Ice Hockey, Compensating Behavior

The effectiveness of safety and protective devices, such as seat belts, airbags, helmets, pads, safety caps, and the like, hinges on the critical assumption that the behavior of individuals remains constant regardless of the use of such equipment and devices. However, it is uncertain whether this is the actual case, or whether individuals will change or adapt their behavior in response to any extra protection in such a way that the intended effect of the protective device will end up diluted or even lost. Theoretically, protective equipment will reduce the price of risky behavior and people will respond rationally by demanding more risk. The possible existence of this compensating behavior was first proposed by Peltzman (1975) and is commonly referred to as the "Peltzman effect". This issue has long been the

*Chong: George Washington University, e-mail: aechong@gwu.edu. Restrepo: Massachusetts Institute of Technology, e-mail:pascual@mit.edu. We are grateful to David Karp, Ugo Panizza, and Tomas Sjorstom for useful comments and suggestions. Saul de Vicente provided excellent research assistance. Additional thanks to Scott Cullen and Maximilian Spiess for providing valuable data. All remaining errors are ours.

subject of vigorous debate in economics. In fact, the Peltzman effect has strong implications which are not only circumscribed to local applications, but rather are at the heart of the debate related to government intervention in the economy as this effect is, in essence, directly related to moral hazard issues.

While many researchers have long tried to show the existence, or lack thereof, of the Peltzman effect, the existing empirical evidence remains inconclusive. Initial attempts on providing evidence on compensating behavior focus on laws requiring the use of seat belts in the United States and elsewhere. Since "driving intensity" of the individual cannot be measured when seat belt use is required, researchers have studied fatalities among car occupants and fatalities among non-occupants, as the latter are expected to be affected only indirectly by the less careful driving hypothetically caused by seat belts. Peltzman (1975),Crandall and Graham (1984), Harvey and Durbin (1986), Asch, Levy, Shea and Bodenhorn (1991), Garbacz (1992), Risa (1994), Loeb (1995), among others use time series and cross-section data and find some evidence on seatbelt laws and compensating behavior. Recently, Cohen and Einav (2003) use an instrumental variable approach instrumenting seatbelt use with changes in states regulations, and find that seat belt use decreases occupants fatalities, but has no significant effect on non-occupant deaths. This last finding does not support the existence of compensating behavior. [1]

The evidence on compensating behavior using seat belt data presents many shortcomings. The most problematic issue is that most of the studies focus on aggregate, imprecise and indirect measures of behavior, so that a truly clean exercise cannot be observed as it is difficult to control for aggregate time-varying variables. Moreover, the available evidence is unable to identify which specific individuals are 'treated' and which ones are not. As a consequence, the estimates produced may only be interpreted as average effects, while compensating behavior only arises locally from those who were actually treated (i.e., those for whom the regulation was binding). Another issue is related to the fact that compliance with laws may not be perfect, whereas usage can be very difficult to measure. Furthermore, seat belt use may be endogenous. Studies that apply instrumental variable procedures to address this problem tend to use changes in regulation as an instrument, but these laws may be associated with additional enforcement or they may be accompanied by safety campaigns, which conceivably may reduce accidents and all type of deaths through different channels. If this is the case, such seat belt rules may not satisfy the exclusion restriction and may be invalid instruments.[2] Finally, empirical approaches that use time series analysis cannot

[1] On a related paper, Cohen and Dehejia (2004) find moral hazard costs in terms of traffic fatalities when focusing on compulsory insurance laws in the United States. Furthermore, they also find that reductions in accident liability produced by no-fault liability laws have led to an increase in traffic fatalities.

[2] Additionally, some types of enforcement may increase the cost of other infractions. For instance, in

control for national shocks; whereas estimates using cross sections of locations may be biased by unobserved state characteristics. Overall, all these issues make it difficult to provide credible evidence in favor or against the existence and extent of compensating behavior.

Given the limitations of the car seat belt literature, researchers have looked for other empirical scenarios in which to test for compensating behavior. Car racing has provided another fertile area to do so, as compliance is automatic and it does not require further enforcement, which helps avoid some of the issues that affect the seat belt literature. However, the resulting empirical evidence still remains unpersuasive. For instance, Pope and Tollison (2010) find that the introduction of a neck and head security device in NASCAR was associated with an increase in caution laps, their proxy of driving intensity. However, their exercise faces many empirical difficulties. When the device was introduced, all racers but one were already using it. This suggests that racers were already responding to increased risks of accidents. Also, their findings may be capturing upward trends in the number of accidents due to the existence of faster cars. In a related paper, Sobel and Nesbit (2007) find that the probability of injury is negatively related to accidents, measured again by caution laps. While they use past injury rates as a proxy, this variable is unconvincing as it may be endogenous since it may be related to changes in NASCAR that may affect accidents, in particular, increases in car speed. Furthermore, it might also be the case that after a few years with high injury rates, NASCAR might have decided to fine-tune the rules in order to decrease accidents for subsequent seasons. In short, it is not clear whether drivers may be responding to the change in the probability of injury caused by the exogenous introduction of some protective device, as suggested by the Peltzman effect.

In this paper we take a different route in order to test for the presence of the Peltzman effect or compensating behavior. We do so by employing data from professional ice hockey which allows us to overcome most, if not all, of the existing limitations of previous empirical studies. In particular, we follow the career path of each hockey player that ended up in the National Hockey League (NHL) from 2001 to 2006 and take advantage of the fact that visor use has not always been compulsory throughout a player's career. While currently the use of hockey visors is not compulsory in the NHL, the top league in the world, its use has been made mandatory in different feeder leagues around the world at different points in time. This allows us to compare the behavior of individual NHL players who use a visor (control group) and players who do not do so (treatment group) with respect to their behavior in other leagues in which they were forced to wear a visor. Crucially, we are able to follow the

locations with secondary enforcement, drivers may be pulled over for committing unrelated infractions, and receive an additional fine if they are found not wearing seat belts. This may increase the cost of committing an infraction for those not wearing one.

playing path of each individual player and thus we are able to control for player fixed effects, which typically may be a major source of bias. The required identification assumption is that the behavior of players that use visors in the NHL and those who do not will follow similar trends as they change leagues, ignoring the effect of visors. These institutional features of professional ice hockey allow us to set an empirical research design that is analogous to a difference-in-differences approach.

Unlike most of the existing empirical evidence produced to this day, we do find that there is significant compensating or offsetting behavior among hockey players when forced to wear visors. We estimate that whereas the average penalty minutes per game is 0.8, visors cause a substantial increase of 0.2 penalty minutes per game. In fact, we find that players that wear visors play more aggressively, partially offsetting its protective effect and creating potential spillover effects to other players. We also find that the use of a visor does not significantly affect performance, measured by goals and assists per game. We conduct different exercises to check the robustness of our results. In particular, we provide evidence suggesting that our estimates are not driven by differences in adaptation as players change leagues; or equivalently, suggesting that the equal trends assumption holds. These results are meaningful as they imply that, contrary to common belief, mandatory use of visors do not raise consciousness about safety. On the contrary, visors decrease the cost of unsafe skating, which increases risk-taking and aggressiveness on the ice. In fact, some hockey commentators have argued that skating behind a visor provides players with some sense of invincibility that may actually lead them to skate more aggressively and recklessly. Additionally, players may become reckless with their sticks if everyone else has protection (see Schwarz (2001) for an example of these arguments in Lacrosse).

This paper is organized as follows. In section 1 we explain in detail the natural experiment that ice hockey allows us to take advantage of, which in turn provides a good setting to test for compensating behavior. In section 2 we describe the data and our empirical strategy. In sections 3,4 and 5 we present and discuss our empirical results. Finally, in section 6 we summarize and conclude.

1 Visors in Ice Hockey as a Natural Experiment

An on-going controversy in the National Hockey League (NHL), the top professional ice hockey in the world, has to do with the fact that the use of visors in helmets is optional. Visors are strong transparent fiber shields designed to protect a players' eyes and face. During our period of study, from 2001 to 2006, only a third of hockey players chose to wear visors. This number appears to be exceedingly low if one takes into consideration the fact

that year after year there are horrifying and high profile cases of players that become gravely injured for the lack of use of a visor. There are two reasons commonly associated to this behavior. First hockey players believe that their performance may be compromised, as sweat and dirt in the visor may interfere with the player's vision. Second, ice hockey is commonly associated with a macho subculture in which it is important to send the signal that one is courageous enough so much so that wearing a visor may send a detrimental signal to other players. The extent to which the lack of visor use has to do with a perceived reduction in performance or to peer pressure is unclear. Interestingly, it may be argued that visor use can give a player a sense of additional security that, instead of reduce his performance, may actually help him play better as, for instance, wearing a visor may allow him to manage risk better.

Ice hockey provides an ideal natural experiment to test for compensating or offsetting behavior for two reasons. First, a particular feature that affects all players that end up in the NHL has to do with the fact that before, and sometimes after, playing in the NHL- where visor use is optional- players participate in North American or European feeder leagues which had mandatory visors by the time they were playing.[3] As a consequence, we are able to track individual playing careers for the different cohorts of NHL players during the 2001-2006 seasons and retrieve their play statistics for seasons played at leagues which mandated them to use a visor during their time they played there. Importantly, these changes in visor regulation give us an exogenous source of variation in visor use. Players must wear a visor for exogenous reasons unrelated to their characteristics, since conditional on playing in the NHL, every single player had at least one season in a feeder league that mandated him to wear a visor.

This feature of ice hockey allow us to set a research design similar to a difference-in-differences. We are able to observe which players stop using visors once they get into the NHL as well as which players use one regularly during the 2001-2006 seasons. Non-users are the treated players, since they are the ones affected by mandatory visor rules in other leagues. Regular users are the control group, since they always use some kind of face protection regardless of league-specific rules. The latter are not affected, or at least are less likely to be affected, by mandatory visor rules in other leagues. The 2001-2006 seasons in the NHL are those for which the treatment is not active. The seasons played in leagues with compulsory visor use are those for which the treatment is active. Moreover, we can control for player fixed effects as we are able to track the same set of players throughout their entire careers.

[3]Some of these feeder leagues changed their visor regulation during our period of study. Most notably, the AHL, CHL and EHL, some of the most important minor pro leagues mandated visors between 2003 and 2006. For our purposes, we are only interested in the seasons in which visors were already mandatory.

The comparison with players that are not affected by mandatory visor rules, but which are similar in other fixed characteristics allows us to control for league specific effects under the premise that similar players that only differ in their use of a visor in the NHL follow similar trends when playing in different leagues, ignoring the effect of visors. If this is the case, any difference in performance between both group of players in leagues with mandatory visors with respect to the NHL must be caused by the exogenous imposition of visors.

Another advantage of ice hockey is that it allows us to capture changes in behavior. We are able to measure individual aggressive or reckless behavior with precision by looking at the penalties committed in games. A penalty committed on the ice is a punishment for behavior that is deemed inappropriate. It is enforced by detaining the offending player within a so-called penalty box for a set number of minutes, during which, the player is not allowed to play. The offending team usually cannot replace the player on the ice, leaving them with one player short during the penalty period. The statistic used to track penalties is called "Penalty Infraction Minutes" (PIM) or "Penalties in Minutes" which represents the total amount of penalties measured in minutes accrued by a player during a period. This statistic is a very good proxy to measure potential compensating behavior of the player during a season, as this variable captures a very detailed and standardized set of behavior that is directly linked with recklessness, risky behavior, and aggressiveness of the player. Typical behavior that is penalized in the form of either a "minor" penalty or a "major" penalty which are assigned a specific and pre-defined time of penalty in minutes include charging, boarding, elbowing, kicking, head shots, attempt to injure other players, fighting, cross-checking, abuse to officials, and several others that are clearly spelled out in the NHL rules, as described in Appendix 1.[4]

2 Data and empirical strategy

2.1 Data

Our sample consists of data containing detailed characteristics of 763 professional hockey players who were active in the NHL between the 2001 and 2006 regular seasons. For each of

[4]Contrary to what one would expect, head injuries are not the most adequate variable to capture compensating or offsetting behavior. On the one hand, reckless or more aggressive behavior is not reflected in such injuries only, but in a more general pattern of behavior, precisely the one captured by penalties in minutes. On the other hand, because of this macho subculture that permeates ice hockey, injuries in general, and head injuries (e.g., concussions) in particular, tend to be concealed by players and thus, accurate statistics on actual head injuries, beyond the obvious ones (e.g., eye injuries), which may be biased, are difficult to obtain. Furthermore, a current controversy in the NHL is directly related to the fact that frequent times head shots have produced injuries that have not been penalized by referees as they were deemed legal.

these players we observe if they use a visor which, as mentioned above, is not mandatory in this league. We find that 250 of the players in our sample, nearly 30% of the total number of players, used a visor regularly while 513 players never did. For each player we collect a broad set of personal characteristics including date of birth, birthplace, weight, height, the year of his first season in the NHL, position, experience, and several others.

The key feature in this paper is that we are able to follow these players throughout their entire playing career prior to their arrival to the NHL and thus we can collect their corresponding playing statistics for the seasons in which they played in leagues that did require a mandatory use of visors, if this was the case. These leagues include European elite leagues, minor pro leagues, Canadian junior leagues, U.S. junior leagues, and college leagues. Appendix 2 shows the corresponding regulation regarding visor use for the different leagues included in this paper and the year at which mandatory visor use was first introduced.

In particular, for each of these players we collect a set of play statistics, including goals, assists, games played, and penalty in minutes for each of the following seasons, provided that the player took part in any of them: i) the 2001, 2002, 2003, 2005 and 2006 NHL seasons, which we identify by a dummy $M_l = 0$ and label "NHL seasons", and ii) any other season during the career of the player in a league other than the NHL that mandated him to use a visor, which we identify by a dummy $M_l = 1$ and label "other leagues' seasons".[5] It should be noted that we exclude seasons in leagues other than the NHL, for which either the player is not forced to wear a visor, or we do not have enough information to attribute visor use. In fact, while it may be the case that a league may have compulsory rules related to visor use, sometimes grandfather clauses exempting some players are applied when such regulation is first introduced. We take a conservative approach and exclude any data on players that belong to an exempted group for a particular season. Out of the 763 players in our sample, there were 716 who played at least one season in leagues other than the NHL in which they were required to wear a visor. We can safely assume that players used a visor during these seasons for exogenous reasons, given that compliance is automatic.

Our unit of observation is player-per-season. For the players who used visors in the NHL during 2001-2006, we are able to obtain data on 1012 player-per-seasons in the NHL, and 1139 player-per-seasons in the other leagues. On the other hand, for the players who did not use visors in the NHL, we observe 2315 player-per-seasons in the NHL, and 2620 player-per-seasons in other leagues. Table 1 summarizes the main variables of interest used in this study. The table is divided in two columns. The first column summarizes the data for those players who use a visor during their NHL career. The second column summarizes the data for players who did not. We show the statistics for the NHL 2001-2006 seasons

[5] We do not have data for 2004 as during this year there was a league lockout.

in one column, and the statistics for seasons in other leagues in the other. The table also summarizes the main characteristics of the players.

As shown in the table, players who used visors in non-NHL leagues scored more goals, gave more assists and were assessed less penalty minutes than players who never used visors. Since all players had to use a visor or worse, full face protection in these leagues, these differences cannot be attributed to the protective equipment, and suggest that players who use a visor in the NHL are systematically different from those who do not use one. Our data suggest that more aggressive players inherently do not use visors while high-performing players inherently do so. This simple observation implies that any cross sectional estimate of the effect of visors would be biased as users are a non random sample. The table also shows that players who use visors are more likely to come from Europe and be less tall and heavy, although there are no significant differences in their age or experience in the NHL.

As mentioned above, the key dependent variable used in this study is penalty in minutes per game. As mentioned above, this variable is defined as the total amount of time that a player was detained in a penalty box for rule transgressions on the ice rink, which are typically linked with aggressiveness and recklessness, as shown in Appendix 1. We normalize this measure by dividing it by the total number of games played. At the heart of our estimate lies the fact that, although users of visors are inherently less aggressive (1.11 penalty minutes as compared with 1.59 penalty minutes received by non-users in leagues with mandatory visors), they become more aggressive relative to non-users when these players remove their visors (penalty minutes decrease from 1.11 to 0.63 for users and from 1.59 to 0.96 for non-users in their NHL seasons). Thus, as long as the equal trends assumption holds, visor use must account for the 0.14 additional penalty minutes per game received by users in their NHL seasons. This simple calculation shows the importance of gathering statistics for feeder or European leagues as this effect would be impossible to find in cross sectional data.

2.2 Empirical Strategy

As the above discussion suggests, cross-sectional estimates of the effect of visors on other variables are likely to be biased, since players with particular characteristics that cannot be observed use visors. Exploiting the panel dimension of the NHL seasons cannot do the job, either. This is because only a limited number of players changed their visor status during this period, thus limiting variation. Moreover, these changes may occur for endogenous reasons. For instance, a player may realize that NHL hockey is more dangerous than he initially thought; he may choose to wear a visor during the subsequent season and he could choose to play more cautiously. Thus, variation in visor usage within the NHL may be endogenous, and estimates with panel data for the NHL seasons only may be biased.

In fact, one would like to compel players who are not using a visor to use one in order to compare their performance and statistics, relative to other players who did not change their visor status. This is exactly what occurs when players move from (or to) minor, junior, college or European leagues, where visors are mandatory, to the NHL, where such use is optional. This variation in league regulations regarding visor use is the key to this paper. It provides a large exogenous source of variation in visor use that allows us to overcome problems typically confronted in previous research and thus help us estimate a causal impact. It is exogenous because players are forced to use a visor, and this has nothing to do with their unobservable characteristics since all the players have to go through one or more of these leagues before playing in the NHL.

The basic equation we estimate is given by

$$y_{isl} = \alpha_i + \theta_l + \kappa_s + \beta NV_i \times M_l + \varepsilon_{isl}. \tag{1}$$

Here y_{isl} is any outcome for player i during season s at league l. NV_i is a dummy that takes the value 1 if the player never used a visor during the 2001-2006 NHL regular seasons, and 0 otherwise. [6] M_l is another dummy that takes the value 0 for the NHL seasons between 2001 and 2006 and 1 for seasons played in other leagues where the player was forced to use a visor at that time. Strictly speaking, our notation should include M with a player and a season subscript, as many leagues changed their visor rules during the years we observe and some leagues had grandfather clauses. However, we use M_l as a constant for a given league in our sample since, as explained above, we dropped the observations in non-NHL leagues in which the player was not forced to wear a visor. We estimate the model with a full set of player, league and season fixed effects (α_i, θ_l and κ_s). In some specifications we drop the fixed effects and replace them for observable player characteristics such as age, birth year, height, weight, birth place, other individual characteristics, and the dummy NV_i, which captures common characteristics of players not using a visor. Finally, ε_{isl} is the error term.

The OLS coefficient β of the interaction term $NV_i \times M_l$ consistently estimates the effect of using a visor on the outcome y, as long as players who use a visor and players who do not use a visor (but are similar in all other dimensions), follow similar trends when moving from (or to) non-NHL leagues to (or from) the NHL. At the heart of our identification assumption we have a typical difference-in-difference approach. The treated group consists of players who did not use a visor in the NHL, and hence are likely to be affected by a rule mandating it in other leagues, while the control group consists of players who used visors in the NHL and are less likely to be affected by such a rule. In this setting instead of pre-treatment and

[6] Our results are robust to different coding for this variable. In particular, we do not use changes in visor use for a particular player during his NHL seasons as this may be endogenous.

post-treatment periods, we have leagues in which the treatment is active that is, seasons in non-NHL leagues, as well as leagues in which the treatment is not active, namely the NHL.

Our identification assumption is apparent once one notices that β is given by (ignoring those players who changed their visor during 2001-2006, fixed effects and other controls)

$$\widehat{\beta} = \begin{array}{c} \left[\widehat{E}(y|M=1,NV=1,V=1) - \widehat{E}(y|M=0,NV=1,V=0)\right] - \\ \left[E(y|M=1,NV=0,V=1) - E(y|M=0,NV=0,V=1)\right]. \end{array} \tag{2}$$

Here V is a dummy equal to 1 if the player is using a visor and 0 otherwise. Thus, β estimates the local effect of a visor, on those who do not use one, $E(y|M=0,NV=1,V=1) - E(y|M=0,NV=1,V=0)$, as long as

$$\begin{array}{c} \widehat{E}(y|M=1,NV=1,V=1) - \widehat{E}(y|M=0,NV=1,V=1) = \\ \widehat{E}(y|M=1,NV=0,V=1) - \widehat{E}(y|M=0,NV=0,V=1), \end{array} \tag{3}$$

which is an equal trends assumption. Given the importance of this assumption, we will discuss it thoroughly and present some evidence showing that it holds.

While player fixed effects reduce the bias arising from the fact that users and non-users are inherently different in many fixed characteristics, they are not sufficient to guarantee identification. The interaction may be capturing heterogeneous effects of playing in the NHL, or differences in adaptation between users of visors and non users of visors. However, fixed effects make the equal trends assumption more plausible, since we just have to assume that similar players who are only different because one wears a visor at the NHL and the other does not, follow similar trends in non-NHL leagues relative to the NHL, ignoring the effect of visors. In the above equation, league effects capture common differences between groups as they move to play in different leagues, while season effects capture common trends in hockey affecting all players. We also allow for differential trends by league in some specifications. The last issue when estimating equation 1, is that the error term may be serially correlated. Thus we clustered errors by player, since league and season fixed effects will partially remove other types of serial correlation, and we do not have enough clusters at that aggregation level.

3 Results

Table 2 shows different estimates of model 1. The first row shows our estimates for the effect of a visor, which is the coefficient of the interaction term, β. In the first four columns we exclude player fixed effects and replace them with observable characteristics and a dummy for players who do not use visors, NV_i, which captures common characteristics among this group of players. In these columns, the coefficient of this dummy, reported in the row "No-visor

type", can be interpreted as a measure of self-selection bias arising from group differences. The last two columns include player fixed effects. We include season and league effects except for the first column; player characteristics including age, experience, birth year, birth region and others; and in column 4 and 6 we allow for different time trends in each major category of leagues (NHL, minor pro, junior U.S., junior Canada, major junior, college and European leagues). All models have standard errors clustered at the player level.

As shown in the table, we estimate that visors increase penalty in minutes per game by 0.2. This is a large effect, especially when taking into account that the average penalty in minutes per game in the NHL was about 0.8 during our period of study. An average player plays about 50 games in the regular season, so using a visor causes about 5 penalty minutes per season. This is a local effect that tells us how penalty minutes would change for a player who does not use a visor if he is suddenly forced to use one. Our estimates suggest that there is compensatory behavior as players feel more protected. The cost of aggressive skating falls and players respond by playing more recklessly and aggressively, as implied by the increase in penalty minutes. This effect is significant at the 1% level, and it is fairly stable across specifications.

When player fixed effects are not included, the dummy for players who do not use visors in the NHL is positive and statistically significant. This dummy shows that there is considerable selection bias, and players who do not use a visor in the NHL are less cautious to start with. This is precisely why it is so important to use minor league statistics as part of our natural experiment as pure cross-sectional estimates will be biased.[7]

As mentioned above, a concern is that players using visors in the NHL may adapt differently compared to players who do not use them, or performance and behavior may evolve in a different way for visor users and non-users as they move from other leagues to the NHL. It could also be the case that different penalization standards or league characteristics may have an heterogeneous effect on both groups of players, and thus the equal trends assumption may not hold. In order to provide some evidence in support of the equal trends assumption, which is the key identification assumption required for our results, we conduct some robustness exercises in Table 3.

The first two columns in Table 3 exploit the NHL lost season of 2004 due to contract issues. During this year, there was no regular season in the NHL and several players migrated to professional leagues in Europe or minor pro leagues in North America which already had instituted mandatory visor rules. This specification is helpful because players were already

[7]In fact, cross sectional estimates using the NHL seasons as a pooled sample show a negative relationship between using a visor and penalty minutes per game, but since players who use a visor are more cautious to start with, this result is due to pure selection bias.

used to NHL hockey and were forced to change leagues because of an exogenous event. Overall, these leagues are not very different from the NHL, especially given the fact that the intention of the players was to keep in shape. In these two columns we estimate model 1, but we redefine the dummy variable M_l. In this case it takes the value of 1 when it corresponds to a player in his 2004 season, if he played in a professional league with mandatory visors. On the other hand, it is set to 0 for the NHL regular seasons during 2001, 2002, 2003, 2005 and 2006. As shown in the table, these estimates suggest that using a visor increases penalty minutes per game in about 0.5, which is considerably larger than our baseline estimates. In this case, the identification assumption becomes more plausible because we only need players to follow equal trends as they moved to other professional leagues during the NHL lockout.

In the next two columns of Table 3 we exclude college, U.S. junior and European leagues and keep Canadian feeder leagues only, which are considered to be the closest to the NHL. Our estimates are not affected by restricting our sample and remain positive and statistically significant. This result is important because it shows that our estimates are not driven by differences in adaptation by group to Canadian hockey, or its rules, which may differ from the European ones or College rules. These regressions also show that the effect is not driven by college leagues, where full face protection is required.

The above table also rules out other potential confounding factors. For instance, it may be argued that leagues other than the NHL may apply different penalization standards that may impact more aggressive players differently, thus biasing our original estimate. However, European leagues tend to be regarded as less tolerant towards violence than the NHL; while Canadian feeder leagues are regarded as more tolerant towards violence than the NHL. Thus, the fact that we get a positive and significant effect of visor use on penalty minutes using both comparison groups, suggest that our main result is not driven by systematic differences in penalization standards across leagues. That is, our results hold either when we compare NHL seasons to seasons in leagues that tolerate less violence (European leagues in columns 1-2), or when we compare NHL seasons to seasons in leagues which tolerate more violence (Canadian feeder leagues in columns 3-4).[8]

In the last two columns of Table 3 we control for heterogeneous adaptation to different leagues using a parametric approach. We rank the different categories of leagues according to how many of their players end up becoming NHL players. This ranking starts with the NHL itself and suggests that minor pro leagues are more alike the NHL. We then have the European leagues followed by major junior and junior leagues, and finally college leagues.

[8] According to the conventional wisdom Canadian feeder leagues appear to tolerate more violence than the NHL. At the NHL level, players are careful not to take penalties because they do not wish to put their team shorthanded, and they are under pressure from coaches not to risk injury (i.e. the cost of a penalty is higher at the NHL level). At lower levels, there is much more fighting and thus more penalty minutes.

These two columns present estimates in which we control for an interaction term between the rank number of a league and a dummy that identifies players who never used visors in the NHL seasons. This approach is analogous to what is done in a typical difference-in-difference analysis when including group specific trends as a robustness check. We find that this variable is not statistically significant, but its negative sign suggests that players who did not use a visor in the NHL, and were inherently more aggressive, became more aggressive as they moved from lower ranking leagues to the NHL or other professional leagues, ignoring the effect of visors. This finding implies that, if anything, this differential trend would create a downward bias in our estimate, making our baseline estimate a lower bound. Consistent with this, our estimates are larger than in our base case scenario once we control for the difference in trends.

As shown in the descriptive statistics of our sample, visor use is less prominent among Europeans, heavier and taller players and is also correlated to a player position. Thus, it could be the case that players who differ in these characteristics may adapt in distinct ways to the NHL. This possibility would violate the equal trends assumption and bias our estimates. In order to rule this out, we include interactions of the "treatment leagues" dummy, M_l, with observable players' characteristics, which control for potential differences in adaptation to the NHL between visor users and non-users. The first column in Table 4 shows estimates controlling for different adaptation by birth and year of first NHL season; that is, a full set of interactions between M_l and years of birth and first NHL season. The second column in Table 4 shows estimates controlling for different adaptation by position; that is, a full set of interactions between M_l and position dummies. The third column in Table 4 shows estimates controlling for different adaptation by weight and height; that is, a full set of interactions between M_l and these variables. The fourth column in Table 4 shows estimates controlling for different adaptation by birthplace; that is, a full set of interactions between M_l and dummies for players born in Canada, the U.S. or Europe. In the last column we include all controls simultaneously. In all the specifications our estimates remain positive and significant, but the coefficients become smaller. These results suggest that our results are not driven by differential adaptation to the NHL among players with different observable characteristics related to visor use.

We also conduct a series of placebo tests in order to verify the equal trends assumption in a non-parametric way. Table 5, presents different placebos in which we drop the NHL seasons, and explore whether there are significant differences between visor users and non-users, as they move from-and-to leagues that require mandatory use of visors. If there are no differences in adaptation and the equal trends assumption holds, one would not expect any significant difference between both types of players, as visor regulation is the same in

these leagues where players had to use a visor or full face protection. To do this, we remove the NHL seasons and estimate the interaction of NV_i with dummies for different subsets of leagues. In the first two columns we use a dummy which takes the value 0 for seasons in minor pro leagues and 1 otherwise. The negative and not significant coefficient suggest that, consistent with the equal trends assumption, there are no differences between visor users and non-users as they move from European or junior leagues to the minor pro leagues. In the third and fourth column we estimate the same model but excluding European leagues, which are also professional leagues. The results are very similar and suggest that there is no significant difference between visor users and non-users as they move from junior or college leagues to minor pro leagues.

In the last two columns in Table 5, we perform a similar exercise but we use a dummy which takes the value of 0 for European leagues and 1 for junior and college leagues. Again, we do not find a significant difference between visor users and non-users behavior as they move from junior or college leagues to European leagues. Our evidence suggests that the equal trends assumption holds, at least for movements of players between these feeder and European leagues. The signs of the placebo tests suggest that, if anything, players who do not use visors and are more inherently aggressive, become even more so as they move to more competitive leagues, relative to players who use visors. Thus our baseline estimates are biased against our hypothesis and hence represent a lower bound.[9]

Our results also hold when including team effects in order to control for sorting by team, as Canadian-based teams may tend to be more aggressive than US-based teams. They also hold when excluding college seasons, and to different variable definition of the no-visor dummy, NV. These results are available upon request.

4 Do Visor Use Reduce Performance?

We are also able to test whether the use of visors has an impact on performance, measured by assists and goals per game. Table 6 shows our estimates when these variables are used as outcomes. We find no particularly robust or statistically significant effect of wearing a visor on performance. Player complaints about the use of visors reducing their performance because they get fogged or are uncomfortable appear to be somewhat exaggerated. As some

[9]In a related exercise not reported here, we exclude the NHL seasons and include a full set of interactions between dummies for each type of league and the dummy for players that do not use visors. Players who do not use a visor do not play any differently in any two of these leagues. That is, the coefficients of these interactions are not statistically different from one another. Results are available upon request. The placebo test also show that our strategy is not capturing differential effects of penalization standards, since we would get significant estimates if users and non-users were affected differently by league specific standards.

have argued, given that players skate with a visor during most of their careers, it should not be too difficult to get used to it in the NHL.[10] Interestingly, this result is reassuring for our identification strategy for two reasons: First, as it is consistent with the fact that players do not play more aggressively in order to compensate for any potential poorer performance resulting from visor use. Second, this result indirectly suggests there are no differences in adaptation by group to the NHL, at least in performance, which indirectly suggests that the equal trends assumption holds.

The corresponding coefficients on the dummy that accounts for players who do not use visors imply that these players would decrease performance independently of visor use. There is considerable self-selection bias in this dimension as well, with the better players using visors. Any simple OLS cross-section results that show that players who use visors perform better are not taking into consideration the endogeneity and upward bias implied by this result.

5 Discussion

The logical question which immediately follows from the results presented above is why do players skate without a visor in the NHL? We have shown that performance is not affected and that they can play more safely, while being able to take more risks and, perhaps, suffering less injuries. It seems that the decision to use a visor is not determined by a trade-off between safety and performance. Instead, our evidence suggests that using a visor may be related to a predominant culture in ice hockey by which players who wear a visor are considered weak or less manly whereas those who do not use visors are considered tough. In fact, according to the conventional wisdom, there is a negative stigma associated with using visors in the NHL that is responsible for the low rate of use. Our results appear to confirm such conventional wisdom.

Players who do not use visors in the NHL are inherently more aggressive or more reckless than those who do wear a visor. This suggests that skating without a visor may be acting as signal for toughness, while using one signals lack of it. Visors may be a credible signal because players that are not tough enough prefer protection, while tough players may be willing to sacrifice some of it. Not using a visor is a credible signal because only tough players can afford it. Moreover, it may be the case that players may benefit from others thinking that they are tough (e.g., either the crowd or other players). Examples of this sort of behavior can be also found in biology, where it is known as the "Handicap effect" and is

[10] However, it may be the case that while visors may reduce vision and performance, they may also allow players to take on additional risks, and both effects could cancel each other out.

also analogous to signaling theory in economics (see Zahavi (1975)).

Our data does not support an alternative explanation in which players do not use a visor because it reduces their relative performance, and as a consequence there are players skating without one because they lack a commitment technology to enforce its use. This insight is similar to the one presented by Schelling about helmet use in hockey (see Schelling (1978)). In fact, when players are polled by the players' union (NHLPA) about use of visors, they have repeatedly rejected the idea of making their use mandatory in the NHL.

Either in the Zahavi signaling model or the Schelling model, it may be Pareto improving to mandate visors. In the signaling case, mandating visors make their use uninformative about a players' type, or removes the stigma associated with it. Players would no longer use a visor because they are not tough enough, but because they have to. In the Schelling model it makes all players safer without altering their relative performance. There may be other situations in which an intervention is justified. For instance, this would be the case when government has better information than the agents, or if the agents systematically underestimate risk.

Many researchers have focused their attention on determining if compensatory behavior offsets all the gains from more protection on the number of injuries or fatalities. From a welfare perspective this is irrelevant. If an agent (e.g., a hockey player) can increase his safety, his utility will increase even if he exhibits compensatory behavior, a direct consequence of the envelope theorem. It may be the case that if visors became mandatory, players who did not use them because of the stigma they carried, would start playing more aggressively, as our findings suggest. As a consequence, the number of injuries to these players may increase, despite the greater protection, but the direct effect of the visor will always dominate the indirect effect of the offsetting behavior. Thus, players may become more frequently injured, but would also play more aggressively, skate faster, and perhaps move better in the ice. Their "utility" as players would increase.

However, the existence of compensatory behavior creates some additional complexities. First, if agents have complete information and rationally choose not to adopt a safety measure, in the absence of externalities, they would be worse-off if they are forced to do so. Second, there is a direct positive effect on agents (envelope theorem), but they may also be subject to other individuals' risky behavior. For instance, in the case of seat belt laws, pedestrians and other drivers suffer from the less cautious driving of others who are forced to wear seat belts. In ice hockey, players do not face the risk of their own behavior only, but also the risk created by other players who skate with visors. Thus, optimal policy should balance between the private gains from protection and the negative effect from externalities or spillovers created by others. The existence of spillovers means that mandating visors

or, for that matter, any safety measures is not necessarily optimal, even if non-users are signaling their type or there is a commitment problem.

6 Concluding remarks

In this paper we provide empirical evidence on the Peltzman effect, or compensating behavior by using data at the individual level from ice hockey at both minor and major leagues. We take advantage of the fact that during the full playing career of a typical professional player the use of visors in helmets has not always been voluntary. By exploiting this variation we are able to apply a difference-in-difference approach, and control for player fixed effects to sort out the specific impact of visor on increased aggressiveness, as measured by the penalty times accrued by the player during games. Whereas increased aggressiveness occurs, thus showing the presence of the Peltzman effect, visor use does not significantly affects performance.

The implications of our findings are not limited to ice hockey or, for that matter, sports. More generally, we show that behavior changes as a result of perceived protection by the individual. We provide causal evidence that the imposition of safety measures can have perverse effects that can go against the originally intended objective of such measure to the point that the overall effect may be detrimental to society. In fact, such economy-wide implications of our research raise the question of whether or not governments should attempt to save us from ourselves.

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Table 1: Summary Statistics.

	Visor in NHL (2001-2006)		No visor in NHL (2001-2006)	
	NHL	Other leagues	NHL	Other leagues
Games played	56.87	40.66	47.00	41.53
	(25.26)	(19.39)	(28.25)	(20.42)
Goals per game	0.18	0.33	0.11	0.28
	(0.14)	(0.26)	(0.12)	(0.24)
Assists per game	0.32	0.50	0.19	0.43
	(0.20)	(0.34)	(0.16)	(0.31)
Penalty minutes per game	0.63	1.11	0.96	1.59
	(0.49)	(0.94)	(0.89)	(1.34)
Number of players	250	228	513	488
	Visor in NHL (2001-2006)		No visor in NHL (2001-2006)	
Birth year	1976.32		1976.56	
	(4.18)		(4.08)	
Born in Canada	0.41		0.63	
	(0.49)		(0.48)	
Born in Europe	0.47		0.21	
	(0.50)		(0.41)	
Born in U.S.	0.12		0.16	
	(0.33)		(0.37)	
First season	1997.40		1997.92	
	(4.29)		(4.43)	
Weight	200.19		204.74	
	(13.19)		(15.80)	
Height	5.79		5.82	
	(0.39)		(0.38)	

Notes: This table shows summary statistics of the main variables used in the paper. Standard deviation is reported in parentheses below the mean. The columns labeled "NHL" include game statistics for the NHL regular seasons 2001,2002, 2003, 2005 and 2006, while the columns labeled "Other leagues" include game statistics for the same players when they were playing in leagues with mandatory visors either in Europe or in minor leagues in North America. There was no NHL regular season in 2004.

Table 2: Estimates of the impact of using a visor on penalty minutes per game.

	(1)	(2)	(3)	(4)	(5)	(6)
Effect of visor	0.165***	0.198***	0.198***	0.187***	0.200***	0.191***
	(0.059)	(0.066)	(0.062)	(0.062)	(0.057)	(0.056)
No-visor type	0.331***	0.250***	0.181***	0.181***		
	(0.041)	(0.043)	(0.044)	(0.045)		
R-squared	0.114	0.197	0.304	0.307	0.619	0.620
Observations	7347	7347	7334	7334	7347	7347
Season effects	N	Y	Y	Y	Y	Y
League effects	N	Y	Y	Y	Y	Y
Player attributes	N	N	Y	Y	Y	Y
Player effects	N	N	N	N	Y	Y
League trends	N	N	N	Y	N	Y

Notes: This table shows estimates of the impact of using a visor on penalty in minutes per game. In the first four columns we control for different player characteristics and the dummy NV_i. The coefficient on this dummy reported in the second row, captures common characteristics among non-users. The last column shows estimates including players fixed effects. Player attributes include age, experience, year of birth, draft year, weight and height. Robust standard errors clustered at the player level are shown in parentheses below each point estimate. For the reported coefficients, those with *** are significant at the 1% level; those with ** are significant at the 5% level; and those with * are significant at the 10% level.

Table 3: Robustness checks of the impact on penalty minutes.

	(1)	(2)	(3)	(4)	(5)	(6)
	Using only lockout years		Using only Canadian leagues		Including treated trends	
Effect of visor	0.403**	0.501***	0.229**	0.174**	0.314**	0.295**
	(0.170)	(0.181)	(0.091)	(0.083)	(0.133)	(0.122)
No-visor type	0.196***		0.189***		14.972	
	(0.038)		(0.043)		(18.824)	
R-squared	0.276	0.659	0.360	0.667	0.304	0.619
Observations	3630	3637	5353	5358	7334	7347
Player effects	N	Y	N	Y	N	Y

Notes: This table shows estimates of the impact of using a visor on penalty in minutes per game using alternative sub-samples. The first two columns show estimates in which we compare NHL performance to that of other leagues during 2004, when the NHL was in a lockout. The next two columns show estimates in which we do not use the data from European, college or U.S. junior leagues. Finally, the last two columns show estimates in which we control for treated players specific trends (both for time and league ranking). Odd columns show estimates without fixed effects controlling for common characteristics among non-users (NV_i). Even columns show estimates including players fixed effects. All the specifications include a full set of controls, including league effects, season effects and player characteristics. Robust standard errors clustered by player are shown in parentheses below each point estimate. For the reported coefficients, those with *** are significant at the 1% level; those with ** are significant at the 5% level; and those with * are significant at the 10% level.

Table 4: Robustness checks controlling for differences in adaptation.

	(1)	(2)	(3)	(4)	(5)
Effect of visor	0.207***	0.197***	0.155***	0.158***	0.153***
	(0.055)	(0.056)	(0.055)	(0.054)	(0.052)
R-squared	0.624	0.623	0.623	0.622	0.629
Observations	7347	7347	7334	7347	7334
By cohorts	Y	N	N	N	Y
By position	N	Y	N	N	Y
By weight and height	N	N	Y	N	Y
By birthplace	N	N	N	Y	Y

Notes: This table shows estimates of the impact of using a visor on penalty in minutes per game. All estimates include players fixed effects. We include interactions of a dummy that takes into account treatment leagues, M_l, with different characteristics of the players in order to control for potential differences in adaptation to the NHL. The latter include birthplace, year of first season, playing position, weight, height, and date of birth. Robust standard errors clustered at player level are shown in parentheses below each point estimate. For the reported coefficients, those with *** are significant at the 1% level; those with ** are significant at the 5% level; and those with * are significant at the 10% level.

Table 5: Placebo test of the impact of using a visor on penalty minutes per game.

	(1)	(2)	(3)	(4)	(5)	(6)
	Minor pro as "pre-treatment" I		Minor pro as "pre-treatment" II		European leagues as "pre-treatment"	
Effect of visor	-0.005	-0.006	-0.077	-0.202	0.001	-0.081
	(0.161)	(0.185)	(0.174)	(0.241)	(0.144)	(0.158)
No-visor type	0.370**		0.443**		0.390***	
	(0.162)		(0.177)		(0.111)	
R-squared	0.289	0.671	0.330	0.721	0.296	0.698
Observations	3929	3937	2544	2544	3462	3470
Player effects	N	Y	N	Y	N	Y

Notes: This table shows estimates of the differences in adaptation to leagues with similar visor regulation. The first two columns show estimates comparing visor users and non-users in minor pro leagues relative to that in feeder or European leagues. The next two columns show estimates comparing visor users and non-users in minor pro leagues relative to that in other feeder leagues, excluding European leagues. Finally, the last two columns show estimates comparing visor users and non-users in European leagues relative to that in junior and college leagues. All specifications include a full set of controls. Robust standard errors clustered by player are shown in parentheses below each point estimate. For the reported coefficients, those with *** are significant at the 1% level; those with ** are significant at the 5% level; and those with * are significant at the 10% level.

Table 6: Estimates of the impact of using a visor on performance.

	(1)	(2)	(3)	(4)	(5)	(6)
	Assists per game			Goals per game		
Effect of visor	0.013	-0.009	-0.010	0.001	-0.013	-0.005
	(0.015)	(0.015)	(0.032)	(0.012)	(0.013)	(0.027)
No-visor type	-0.090***			-0.041***		
	(0.012)			(0.008)		
R-squared	0.463	0.610	0.610	0.467	0.587	0.587
Observations	7334	7347	7347	7334	7347	7347
Player effects	N	Y	Y	N	Y	Y
Treated trends	N	N	Y	N	N	Y

Notes: This table shows the impact of using a visor on performance. The first three columns show estimates on assists per game, the last three columns show estimates on goals per game. All models include a full set of controls, including league effects, season effects, player characteristics and league specific trends. Robust standard errors clustered by player are shown in parentheses below each point estimate. For the reported coefficients, those with *** are significant at the 1% level; those with ** are significant at the 5% level; and those with * are significant at the 10% level.

Appendix 1: Infractions that result in Penalty in minutes

- Abuse of officials: Arguing with, insulting, using obscene gestures or language directed at or in reference to, or deliberately making violent contact with any on or off-ice official.
- Aggressor penalty: Assessed to the player involved in a fight who was the more aggressive during the fight. This is independent of the instigator penalty, but both are usually not assessed to the same player (in that case the player's penalty for fighting is usually escalated to deliberate injury of opponents, which carries a match penalty).
- Attempt to injure: Deliberately trying to harm an opponent.
- Boarding: Pushing an opponent violently into the boards while the player is facing the boards.
- Butt-ending: Jabbing an opponent with the end of the shaft of the stick. It carries an automatic major penalty and game misconduct.
- Charging: Taking more than three strides or jumping before hitting an opponent.
- Checking from behind: Hitting an opponent from behind. It carries an automatic minor penalty and misconduct, or a major penalty and game misconduct if it results in injury. Illegal check to the head: Lateral or blind side hit to an opponent, where the player's head is targeted and/or the principal point of contact
- Clipping: Delivering a check below the knees of an opponent. If injury results, a major penalty and a game misconduct will result.
- Cross-checking: Hitting an opponent with the stick when it is held with two hands and no part of the stick is on the ice. Delay of game: Stalling the game.
- Diving: Falling to the ice in an attempt to draw a penalty.
- Elbowing: Hitting an opponent with the elbow.
- Fighting: Engaging in a physical altercation with an opposing player, usually involving the throwing of punches with gloves removed or worse.
- Goaltender Interference: Physically impeding or checking the goalie.

- Head-butting: Hitting an opponent with the head. A match penalty is called for doing so.
- High-sticking: Touching an opponent with the stick above shoulder level. A minor penalty is assessed to the player. If blood is drawn, a double-minor is usually called. Referees may use their discretion to assess only a minor penalty even though blood was drawn. They may also assess a double-minor when blood is not drawn, but he believes that the player was sufficiently injured or that the offending player used excessively reckless action with his stick.
- Holding: Grabbing the body, equipment, or clothing of opponent with hands or stick.
- Holding the stick: Grabbing and holding an opponent's stick, also called when a player deliberately wrenches a stick from the hands of an opposing player or forces the opponent to drop it by any means that is not any other penalty such as Slashing.
- Hooking: Using a stick as a hook to slow an opponent, no contact is required.
- Instigator penalty: Being the obvious instigator in a fight. Called in addition to the five minute major for fighting.
- Interference: Impeding an opponent who does not have the puck, or impeding any player from the bench.
- Joining a fight: Also called the "3rd man in" rule, the first person who was not part of a fight when it broke out but participates in said fight once it has started for any reason (even to pull the players apart) is charged with an automatic game misconduct in addition to any other penalties they receive for fighting.
- Kicking: Kicking an opponent with the skate or skate blade. Kicking carries a match penalty if done with intent to injure, but otherwise carries a major penalty and a game misconduct.
- Kneeing: Hitting an opponent with the knee.
- Roughing: Pushing and shoving after the whistle has been blown or checking an opponent with the hands in his face.
- Slashing: Swinging a stick at an opponent, no contact is required.
- Slew Footing: Tripping an opponent by using your feet.
- Spearing: Stabbing an opponent with the stick blade.

- Starting the wrong lineup: When offending team fails to put the starting lineup on the ice at the beginning of each period.
- Substitution infraction: When a substitution or addition is attempted during a stoppage of play after the linesmen have signaled no more substitutions or if a team pulls its goalie and then attempts to have the goalie re-enter play at any time other than during a stoppage of play.
- Too many men on the ice: Having more than six players (including the goalie) on the ice involved in the play at any given time.
- Tripping: Using a stick or one's body to trip an opponent.
- Unsportsmanlike conduct Arguing with a referee; using slurs against an opponent or teammate; playing with illegal equipment; making obscene gestures or abusing an official.

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Facial protection and head injuries in ice hockey: a systematic review

C Asplund, S Bettcher, J Borchers

The Ohio State University Sports Medicine Center, Columbus, Ohio, USA

Correspondence to: Dr C Asplund, The Ohio State University Sports Medicine Center, 2050 Kenny Road, Suite 3100, Columbus, OH 43221, USA; chad.asplund@osumc.edu

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ABSTRACT

Objective: To summarise the best available evidence to determine if facial protection reduces head injury in ice hockey.

Data Sources: MEDLINE and Cochrane databases through January 2009.

Review Methods: Utilising terms: "head injuries," "craniocerebral trauma [MeSH]", "head injuries, closed [MeSH]", head injuries, penetrating [MeSH]", "face mask", "face shield", "visor" and "hockey", 24 articles were identified through our systematic literature search. Of these, six studies met the inclusion criteria. Three independent reviewers reviewed the articles. The study results and generated conclusions were extracted and agreed upon.

Results: Studies reviewed suggest that facial protection reduces overall head injuries in ice hockey. Facial protection showed a statistically significant ($p<0.05$) reduction in the number and type of facial injuries. In studies evaluating full facial protection (FFP) versus half facial protection (HFP), FFP offered a significantly higher level of protection against facial injuries and lacerations than HFP (relative risk (RR) 2.31, CI 1.53 to 3.48). There was no significant difference in the rate of concussion (RR 0.97, CI 0.61 to 1.54) or neck injury (CI 0.43 to 3.16) between full and partial protection. In those who sustained concussion players with FFP returned to practice or games sooner than players with partial facial protection (PFP) (1.7 sessions, CI 1.32 to 2.18).

Conclusions: There is good evidence that FFP reduces the number and risk of overall head and facial injuries in ice hockey compared with PFP and no facial protection. PFP, while not as protective as FFP, appears to offer more risk reduction than no protection.

Worldwide, ice hockey is a popular sport with a participation of over 1 million players in the USA and Canada alone.[1 2] It is anticipated that this number will continue to grow as the popularity of ice hockey increases. As participation increases, the number of injuries seen in ice hockey will also increase. The risk of injury in ice hockey is high, with players travelling up to 30 mph and a puck exceeding 100 mph.[3] Ice hockey provides an environment extremely conducive to the occurrence of head and facial injuries, with multiple opportunities for high speed collisions with other players, fast moving objects, sticks and unforgiving surfaces.

Early investigations performed before the mandatory facemask and helmet rules demonstrated a high incidence of facial injury and lacerations.[4 5] Stick-related head and facial injuries have been the cause of up to 14% of injuries in other studies, especially studies reporting injuries from European teams, whose players are often not required to wear facemask protection.[3 5] Concussion also remains a serious concern in this population and accounted for nearly one out five injuries in ice hockey.[6] Not surprisingly, head injuries constitute 10–40% of all hockey-related injuries, with traumatic brain injuries accounting for 10–15%.[7] The annual incidence of concussions seen in German ice hockey has been reported as 3.7 per 100 athletes.[8] In the 1970s and 1980s, full face masks became mandatory for secondary school and collegiate hockey players because of concerns for ocular and dental injuries.

With the increase in head injuries as hockey evolved, further protective headgear recommendations followed, and helmets are now required at all levels of play. The widespread use of helmets with face masks has essentially eliminated ocular, facial, dental and extracranial head injuries in amateur leagues, but brain injuries remain a serious concern. With the increased utilisation of protective gear, there appears to have been an associated increase in the level of aggressive play and a concomitant rise in the occurrence of concussions and spinal injuries.[7] Until recently, there was speculation that the added mass and area of a helmet and full face shield (FFS) may increase the rotational acceleration of a player's head following impact, producing greater neuronal shearing forces and increased severity of concussion.[9 10] It has also been hypothesised that increased facial protection may lead to more aggressive play and actually lead to more head and facial injuries in hockey.

While wearing a helmet is now required by the National Hockey League (NHL), facial protection remains optional. It is unknown to what degree face visors influence concussion, other head injury and eye injury rates in hockey. The purpose of this systematic review was to evaluate the best available evidence to determine if facial protection provides additional benefit in the prevention of head injuries in ice hockey.

MATERIALS AND METHODS

A literature review was completed using MEDLINE and Cochrane database searches to January 2009 to identify all English language or translated clinical papers that assessed the impact of the use of facial protective gear on the prevention of head injuries in ice hockey. Head injuries were defined as any injury to the head, face or teeth. Injury was defined as any event requiring attention from medical personnel. Several key search terms were used to narrow our literature search (fig 1). Initially, "head injuries" was used, with 105 963 articles identified. Re-entering "head injuries" as a MeSH term

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yielded no additional articles. Key terms used for protective headgear included "face shield", "face mask" and "visor." These terms were run through the databases independently and then collectively, generating 2910 articles. When combined with "head injuries", a total of 110 articles was identified. The term "hockey" was added to narrow the literature to 23 relevant articles.

A secondary search of references from relevant articles was also performed, yielding one additional article. Further criteria for inclusion in this review were then applied to the 24 articles. Articles had to be of level I or II evidence for prognostic studies (prospective study, systematic review of level I studies, retrospective study, study of untreated controls from a previous randomised controlled trial, or systematic review of level II studies).[11] Articles must also have included the use of facial protective equipment as an intervention and head injuries (facial lacerations, dental injuries, concussions, etc) as a clinical outcome. Applying these criteria, a total of six articles was identified as meeting our criteria for systematic review (fig 1). Of the 24 articles, 11 were excluded because they were not of level I or II evidence. Another five were excluded because head injury outcomes were not assessed and two were excluded because facial protective gear was not used as an intervention.

Three independent reviewers, using predetermined methodology, as described by Spindler *et al*,[11] reviewed and scored the six articles included in this systematic analysis and reached consensus through discussion. The study methods and demographic data were first identified with attention to the study type, objectives, outcome measures and examined cohorts. The study results and generated conclusions were extracted and agreed upon.

RESULTS

Results for the six studies meeting inclusion criteria for this systematic review are summarised in tables 1 and 2.

Studies

Amateur hockey

Stuart *et al*,[12] using a prospective study design, evaluated the effect of facial protection on head, neck and facial injuries in elite amateur ice hockey players. The results of this study showed that the use of full and half-face shields reduce the risk of eye and facial injuries compared with no facial protection (NFP). In addition, there was no increase in the incidence of concussion or spinal injuries in the players wearing facial protection. As in similar studies, facial, dental, eye and head injuries were reported regardless of time lost. In comparison to other studies, the principal difference in injury definition in this study was the inclusion of neck injuries that did not result in time lost from play.

Limitations of the study include restricted data collection for playing time and injuries, which were only recorded during home games. Therefore, injuries from away games were not included in the analysis and this limits the study results. Also, players under the age of 18 years had to wear mandatory full facial protection (FFP), whereas players above the age of 18 years could choose to wear a full shield, half shield, or no shield. It is unclear if the injury pattern for those under 18 years differed from those over 18 years. Varying styles of play, such as more aggressive or conservative play based on age, may have skewed the results. These age-related differences may bias the study results. It is also unknown if there are variables among players that influence the choice of FFS use or not. It is possible that these variables could confound the study results.

Collegiate hockey

In a study of 642 Canadian college hockey players, Benson and colleagues,[13 14] looked at the use of FFS versus half face shields (HFS) regarding head and neck injuries and the incidence of concussion.[13 14] A power analysis and sample size calculation was performed, and the study was appropriately powered for the primary outcome. The study revealed that the use of FFS significantly reduced the risk of facial and dental injuries compared with the use of HFS without an increase in the risk of neck injuries, concussions, or other hockey-related injuries. Concussion risk in this cohort was similar among players, regardless of the type of face shield use (FFS vs HFS). Interestingly, those players wearing a FFS returned to play sooner following a concussion than those players wearing a

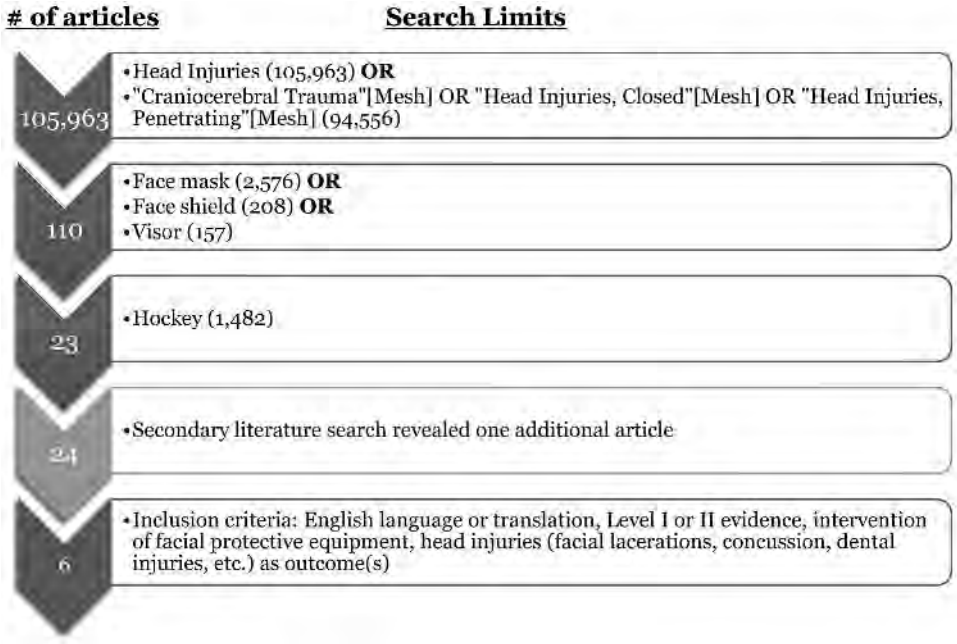

Figure 1 Diagrammatic illustration of systematic literature search. Primary search terms and results (blue), secondary search results (light grey), and applied inclusion criteria and results (dark grey) are shown. The number of articles identified by each search term is noted in parentheses.

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Table 1 Systematic review demographics and methods

Author(s) and journal	Objective	Study type	Population	Cohorts	Primary/secondary outcomes
LaPrade *et al*,[4] *Am J Sports Med*, 1995	To assess the effects of wearing a face mask on facial lacerations and head and neck injuries	Prospective cohort observational study	NCAA division 1 varsity ice hockey team, 798.5 practice hours and 163 games observed	Mandatory face shields versus historical NFS cohort	Primary: Facial laceration (lacerations requiring suturing) incidence rate Secondary: Head and neck injury (an event that caused the player to miss the next practice or game) incidence rate
Benson *et al*,[13] *JAMA*, 1999	To determine the risk of head and neck injuries in players with FFS versus HFS	Prospective cohort	Collegiate level male ice hockey players, 642 total athletes (mean age 22 years)	FFS versus HFS	Primary: Reportable injury incidence (any event requiring assessment or treatment by a team therapist or physician or any mild traumatic brain injury or brachial plexus stretch)
Benson *et al*,[14] *Br J Sports Med*, 2002	To identify specific risk factors associated with increased concussion severity	Prospective cohort	Collegiate level male ice hockey players, 642 total athletes (mean age 22 years)	FFS versus HFS	Primary: Concussion incidence (any traumatic brain injury that required assessment or treatment by a team therapist or physician regardless of playing time lost) Secondary: Playing time lost per concussion
Stuart *et al*,[3] *Am J Sports Med*, 2002	To examine the relationship between injury and type of facial protection and to characterise these injuries	Prospective cohort	Elite amateur male ice hockey players, 282 total athletes (16–21 years of age)	FFP versus PFP versus NFP	Primary: Injury incidence rate Secondary: Concussion incidence rate and injury specific variables (type, anatomical location, diagnosis, mechanism of injury, perceived intent to injure and penalties called)
Stevens *et al*,[10] *J Sci Med Sport*, 2006	To determine the impact of face visors on concussion, head injury and eye injury rates	Prospective cohort observational study	US Hockey League junior A (elite amateur) male ice hockey players, 282 total athletes (16–21 years of age)	FFP versus PFP versus NFP	Primary: Concussion incidence Secondary: Non-concussion head injury and eye injury rates (injury that resulted in at least one missed game and did not include minor facial injuries such as lacerations)
Bunn,[15] *Phys Sportsmed*, 2008	To identify the effectiveness of half-visors based on the severity, location and mechanism of upper-half facial injuries	Retrospective cohort	East Coast Hockey League male ice hockey players, 186 total athletes	HFS versus NFS	Primary: ISS for injuries to the upper half of the face Secondary: Mechanism and location of injury to the upper half of the face

FFP, full facial protection; FFS, full face shield; HFS, half face shield; ISS, injury severity score; NCAA, National Collegiate Athletic Association; NFP, no facial protection; NFS, no face shield; PFP, partial facial protection.

HFS. Moreover, the use of a mouth guard allowed for a faster return to play following concussion regardless of face shield type.

This study used an injury surveillance system to collect nearly 100% of injury data for this cohort while controlling for between-group variables. Despite the accuracy of data collection, the relative severity of injury between the groups (FFS vs HFS) was not determined. Injury severity was defined only by time lost due to injury and those injuries without time lost were not recorded. Also, there may have been differences between team therapists and physicians in their return to play protocol, which may have further skewed the injury severity data. Other limitations of this study include lack of generalisability to high-school age or younger skeletally immature players.

LaPrade *et al*[4] prospectively evaluated almost 800 practice hours and 163 games of US division 1 hockey players to assess the effects of wearing a face shield on facial lacerations and head and neck injuries in collegiate hockey. The results of this study showed that there was a dramatic decrease in head and neck injury and facial lacerations in both games and practices after the introduction of a FFS compared with the use of NFP. Injuries were only recorded if they caused a player to miss a game or practice. This definition could have excluded a significant number of less severe injuries such as facial lacerations. Data analyses included all players on the ice during practices and games, including the goalie. It is not clear if there was a difference in injury rates among goalies before and after mandated FFS use. This could have biased the results as the goalie was required to wear FFP throughout the entire study period.

This study differed from the study by Benson and colleagues[13 14] as it used a historical cohort of players with NFP as a control group for comparison. The use of this historical cohort instead of an actual control cohort is a relative limitation in study design. Sample size calculation and power analysis were not performed so it is unclear if this study was appropriately powered to show statistical significance for the primary outcome. Despite the difference in methodology and the study limitations, the findings support the results of the study by Benson and colleagues.[13 14]

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Table 2 Systematic review results and conclusions

Author(s) and Journal	Results	Conclusions
LaPrade *et al*,[4] *Am J Sports Med*, 1995	1. Lower incidence of facial lacerations per 1000 player-hours in the face mask group compared with the historical group (14.7 to 15.1 in games and 0.0 to 0.2 in practice vs 21.8 in games and 0.6 in practice, respectively; based on 95% CI)	Face masks decreased the incidence of facial lacerations when compared with a historical cohort wearing NFP. The study was unable to assess the effect of face mask use on head and neck injury incidence due to lack of a comparable historical cohort to analyse data.
Benson *et al*,[13] *JAMA*, 1999	1. Significantly fewer head and facial injuries in the FFS cohort ($p<0.001$, RR 2.52, CI 1.73 to 3.68)	There is a reduced risk of head injuries, specifically facial lacerations and dental injuries, in collegiate ice hockey players who wear FFS as opposed to HFS. FFS do not increase the risk of neck injuries or concussions.
	2. Risk of facial laceration was 2.31 times greater in HFS cohort compared with FFS cohort ($p<0.001$, CI 1.53 to 3.48)	
	3. Risk of dental injury was 9.90 times greater in HFS cohort compared with FFS cohort ($p<0.007$, CI 1.88 to 52.1)	
	4. Risk of neck injury was not significantly different between cohorts ($p = 0.78$, CI 0.43 to 3.16)	
	5. No significant difference in number of concussions between the cohorts ($p = 0.90$, RR 0.97, CI 0.61 to 1.54)	
Benson *et al*,[14] *Br J Sports Med*, 2002	1. No significant difference in overall risk of concussion between the HFS and FFS cohorts ($p = 0.90$, RR 0.97, CI 0.61 to 1.54)	There is no difference in the incidence of concussion in players wearing a FFS versus those wearing a HFS; however, in players who do sustain concussion, those wearing a FFS return to play sooner.
	2. In players who did sustain concussion, those in the FFS cohort returned to practice or games sooner (1.7 sessions, CI 1.32 to 2.18) than those in the HFS cohort (4.07 sessions, CI 3.48 to 4.74)	
	3. Less time lost after concussion in players wearing mouth guards	
	4. No difference in risk of sustaining a concussion based on position played or on level of play	
Stuart *et al*,[3] *Am J Sports Med*, 2002	1. Significantly decreased injury rate in the FFP and PFP cohorts compared with the NFP cohort ($p<0.001$, $p<0.001$, respectively)	FFP and PFP significantly reduce facial and eye injuries compared with NFP without an increase in neck injuries and concussions. No association between mouth guard protection use and rate of injuries.
	2. Players in the NFP cohort sustained injuries at a rate more than twice those in the PFP cohort and almost seven times more than those in the FFP cohort	
	3. No significant differences in the rate of concussions between the cohorts ($p = 0.58$, $p = 0.11$)	
	4. Similar frequency of mouth guard use in those injured and not injured	
Stevens *et al*,[10] *J Sci Med Sport*, 2006	1. Significant difference in rates of injuries (recorded in rate per 1000 player-hours) among the cohorts with the lowest rate in the FFP cohort and highest rate in the NFP cohort (FFP 23.2, CI 13.3 to 37.7; PFP 73.5, CI 53.6 to 98.3; NFP 158.9, CI 118.7 to 208.5; $p<0.001$)	FFP and PFP significantly reduce facial and eye injuries compared with NFP without increasing neck injuries and concussions; thus FFP is more protective of facial and eye injuries than PFP. Mouth guard use does not appear to affect the rate of injuries, although this was not specifically studied and further research would need to be done.
	2. Players with NFP sustained injuries at a rate more than twice that of players with PFP and almost seven times more than those with FFP	
	3. No significant differences in the rate of concussions per 1000 player-hours ($p = 0.58$)	
	4. No significant differences in the rate of neck injuries per 1000 player-hours ($p = 0.11$)	
Bunn,[15] *Phys Sportsmed*, 2008	1. Significantly higher ISS for forehead lacerations in the HFS cohort compared with NFS cohort (4 (SD 0.15), 2.75 (SD 0.49), respectively, $p<0.05$)	The HFS or visor does not reduce the severity of injuries to the upper half of the face compared with NFP. Visors may thus not adequately protect the athlete's upper half face and eye region. Study validity is questioned given methodological flaws.
	2. Significantly higher ISS for cheek lacerations in the HFS cohort compared with the NFS cohort (3.69 (SD 0.35), 2.30 (SD 0.49), respectively, $p<0.05$)	
	3. No significant difference in the number of periorbital injuries between the cohorts although trend towards more in the NFS cohort	
	4. High-sticking was the most common mechanism of injury	

FFP, full facial protection; FFS, full face shield; HFS, half face shield; ISS, injury severity score; NFP, no facial protection; NFS, no face shield; PFP, partial facial protection; RR, relative risk.

Professional hockey

Stevens *et al*[10] followed over 800 NHL players prospectively over the course of one complete season to evaluate the impact of face visors on concussions, other head injuries and eye injury rates at the professional level.[10] Results from this prospective cohort showed that the use of facial protection (FFS or HFS) significantly reduced the risk of facial and eye injuries in comparison with NFP. There was no increased incidence of neck injuries or concussions in the players wearing facial protection compared with those not using facial protection. In addition, FFS use reduced the risk of facial and eye injuries significantly more than the use of HFS.

Limitations of this study included the method of data collection. Injury data were recorded from a hockey register and not prospectively by an investigator. The type of facial protection wear was determined by a review of sports cards and NHL photographs, which may not be representative of the players' actual use during the entire season. This methodology of review for facial protection had been validated in a much smaller study but it remains unknown if it is accurate and truly

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Table 3 Impact of facial protection on head (non-concussive) and neck injuries

Author(s) and journal	Study findings
LaPrade *et al*,[4] *Am J Sports Med*, 1995	Lower rate of facial lacerations during games in the face shield cohort compared with the NFS cohort (14.7 to 15.1 injuries per 1000 player-hours and 21.8 injuries per 1000 player-hours, respectively)
	Lower rate of facial lacerations during practice in the face shield cohort compared with the NFS cohort (0.0 to 0.2 injuries per 1000 player-hours and 0.6 injuries per 1000 player-hours, respectively)
Benson *et al*,[13] *JAMA*, 1999	Significantly fewer head and facial injuries with FFS compared with HFS ($p<0.001$, RR 2.52, CI 1.73 to 3.68)
	No significant difference in risk of neck injury between the cohorts ($p = 0.78$, CI 0.43 to 3.16)
Stuart *et al*,[3] *Am J Sports Med*, 2002	Significantly decreased injury rate in the FFP and PFP cohorts compared with the NFP cohort ($p<0.001$)
	Players in the NFP cohort sustained injuries at a rate more than twice that of the PFP cohort and almost seven times more than those in the FFP cohort
Stevens *et al*,[10] *J Sci Med Sport*, 2006	Lowest rate of injury in the FFS cohort compared with the HFS and NFS cohorts (23.2, 73.5 and 158.9 per 1000 player-hours, respectively)
	No significant difference in rate of neck injuries between the cohorts

FFP, full facial protection; FFS, full face shield; HFS, half face shield; NFP, no facial protection; NFS, no face shield; PFP, partial facial protection; RR, relative risk.

representative of facial protection used by players. Finally, power analysis or sample size calculation was not performed.

Minor league hockey

Bunn[15] evaluated the use of HFS compared with NFP in reducing the severity of facial injuries and eye injuries over the upper half of the face in East Coast Hockey League players.[15] Injury data were collected in a prospective fashion but were evaluated retrospectively. Results of this study showed no difference in injury risk between players using HFS compared with those using NFP. Limitations of this study include the use of a non-validated injury severity score, which was weighted heavily towards eye injuries compared with other head injuries. The study was also influenced by selection bias as the study groups were limited and not all players with injuries were included for analysis. It is unclear whether the results of the study would be different if all players with injuries were included for analysis.

DISCUSSION

Overall head and neck injuries

It has previously been hypothesised that added facial protection (FFS or HFS) may increase the risk of head and neck injuries in ice hockey. One reason for this concern is that the use of facial protection in ice hockey may give players an increased sense of invincibility, leading players to take excessive and unwarranted risks. This hypothesis has been supported in previous studies. Gerberich *et al*[16] reported that 66% of surveyed high school hockey players from Minnesota felt that the requirement of the face mask allowed them to be more aggressive in their style of play. This perception of increased aggressive play with the use of facial protection leading to increased head and neck injury is not consistent with this systematic review (table 3).

Results from our systematic review support the protective effect of FFS use in reducing the severity of concussion and facial, eye and dental injuries, without increasing the risk of other head and neck injury. Results also show that the lowest overall rate of injury was seen in the FFS cohorts compared with the HFS and NFS cohorts. The use of half face shields did prove to be more protective than NFP. There was no significant difference in neck injury between the cohorts, suggesting that facial protection does not lead to an increase in the overall risk of head and neck injury compared with the use of NFP. The hypothesis that facial protection in ice hockey increases the potential for the overall risk of head and neck injury compared with NFP is inconsistent with the findings of this systematic review and should not be used as a reason to discourage the use of facial protection in ice hockey.

Concussions

Historically, helmet use alone has not been found to be protective against concussions in either football or hockey.[17] As with the overall risk of head and neck injury, there has been concern that the use of FFS or HFS in ice hockey would lead to an increased risk of concussion. The results of our systematic review suggest that there is no increased risk of concussion in ice hockey players using facial protection (table 4). Our systematic review further reveals that there is no difference in the risk of

Table 4 Impact of facial protection on concussion

Author(s) and journal	Study findings
Benson *et al*,[13] *JAMA*, 1999	No significant difference in the number of concussions between FFS and HFS cohorts ($p = 0.90$, RR 0.97, CI 0.61 to 1.54)
Benson *et al*,[14] *Br J Sports Med*, 2002	No significant difference in risk of concussion between FFS and HFS cohorts ($p = 0.90$, RR 0.97, CI 0.61 to 1.54)
	Players who did sustain a concussion in FFS cohort missed fewer sessions than those in the HFS group (1.7, CI 1.32 to 2.18 and 4.07, CI 3.48 to 4.74, respectively)
	No time lost following concussion in players with FFS wearing mouth guards compared to 1.80 missed sessions in players with FFS not wearing mouth guards (CI 1.38 to 2.34)
Stuart *et al*,[3] *Am J Sports Med*, 2002	No significant difference in concussion rate between FFP versus PFP versus NFP cohorts ($p = 0.58$, $p = 0.11$)

FFP, full facial protection; FFS, full face shield; HFS, half face shield; NFP, no facial protection; NFS, no face shield; PFP, partial facial protection; RR, relative risk.

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Table 5 Impact of facial protection on lacerations and dental injuries

Author(s) and journal	Study findings
LaPrade *et al*,[4] *Am J Sports Med*, 1995	Lower rate of facial lacerations (recorded in number per 1000 player-hours) during games and practice in the face mask cohort compared with the historical cohort (14.7 to 15.1 and 0.0 to 0.2 vs 21.8 and 0.6, respectively; based on 95% CI)
Benson *et al*,[13] *JAMA*, 1999	Risk of facial laceration was 2.31 times greater in the HFS cohort compared with the FFS cohort ($p<0.001$, CI 1.53 to 3.48)
Bunn,[15] *Phys Sportsmed*, 2008	Significantly higher ISS for forehead lacerations in the HFS cohort compared with the NFS cohort (4 (SD 0.15) and 2.75 (SD 0.49), respectively, $p<0.05$)
	Significantly higher ISS for cheek lacerations in the HFS cohort compared with the NFS cohort (3.69 (SD 0.35) and 2.30 (SD 0.49), respectively, $p<0.05$)
Benson *et al*,[13] *JAMA*, 1999	Risk of dental injury was 9.9 times greater in the HFS cohort compared with the FFS cohort ($p<0.007$, CI 1.88 to 5.21)

FFP, full facial protection; FFS, full face shield; HFS, half face shield; ISS, injury severity score; NFP, no facial protection; NFS, no face shield; PFP, partial facial protection; RR, relative risk.

concussion in ice hockey players regardless of facial protection used (FFS vs HFS vs NFS). There does appear to be a faster return to play from concussion in players wearing a FFS compared with those wearing a HFS, which suggests that the severity of concussion may be less in players wearing FFP. Overall, the results of our systematic review suggest that the use of facial protection in ice hockey does not lead to an increased risk of concussion, and that the use of a FFS may actually decrease the severity of concussion and allow for fewer missed practices and games with a faster return to play.

Although the benefits of mouth guard use in protecting athletes from dental injury is supported in the literature, controversy exists as to whether mouth guard use can reduce athletes' risk of concussion.[18] At this time, there is no clear association between mouth guard use in ice hockey and reduced concussion risk, but there may be less time lost following concussion in those players wearing a mouth guard and a FFS compared with those wearing a FFS alone. This potential benefit, along with the decreased risk of dental injury, supports the use of mouth guards in ice hockey.

Facial lacerations and dental injuries

There is good evidence that the use of a FFS in ice hockey reduces the number of facial lacerations compared with the use of a HFS or NFS (table 5).

Our systematic review illustrates that the mandatory use of FFS in ice hockey results in a reduction in the number of facial lacerations and dental injuries among ice hockey players. It appears that use of a HFS does not confer the same protection from facial lacerations as the use of a FFS. There is also a significant increased risk of dental injury with partial facial protection (PFP) compared with FFP. Moreover, the use of a HFS has been associated with more severe facial lacerations when compared with a NFS control. This result is influenced by the use of a non-validated injury-grading tool, which preferentially gave lacerations closer to the eye a higher injury score than other facial lacerations. Our systematic review found that the use of a FFS results in a decreased risk of facial lacerations and dental injuries compared with the use of a HFS or NFS. Whether there is a difference in the risk of facial laceration or dental injury in players wearing a HFS compared with NFS remains unclear.

Limitations

This systematic review does have several limitations. The pre-hoc search strategy and inclusion criteria employed for this review were limited to high level clinical outcomes studies and only produced six studies meeting criteria sufficient for review. There are studies of lesser levels of evidence that address this topic, but their results are limited by study methodology and a lack of control groups. Although we recognise that these studies may contribute to the overall body of literature on this topic, our purpose was to examine the best clinical outcomes evidence to arrive at our conclusions regarding facial protection in hockey.

A second limitation of this systematic review is the definition of head injury used for this review. Although we tried to be inclusive in our definition of head injury to include any injury to the head, face or teeth, all of the studies used various definitions of head injury. The lack of a uniform definition of head injury and the severity of injury among these studies is a limitation and makes generalisation of the results more difficult in this review.

The results of this review are limited to the populations represented in the studies; specifically men in amateur, collegiate or professional hockey. The results of this review do not consider women's or youth hockey and may not be generalisable to these populations. It should be noted that FFS are mandatory in youth and women's hockey and we would suggest that the benefits noted in this review from the use of a FFS are also present in these populations even if they are not specifically included in this review.

The final limitation of this review is related to the lack of uniformity among the reviewed studies with respect to data collection and the definition of injury severity. All of the studies reviewed used various forms of data collection and this is a potential limit to the conclusions of this review. Because most studies used time lost to play to estimate injury severity, it is possible that this could lead to misclassification of injuries and bias the overall recommendations of this review. It would be preferable to have a uniform method of data collection and a more objective definition of injury severity in individual studies to strengthen this review.

What is already known on this topic

Ice hockey is a fast, high impact sport with a high propensity for head and facial injuries.

What this study adds

- FFP reduces the number and risk of overall head and facial injuries in ice hockey.
- PFP, although not as protective as FFP, appears to offer more risk reduction than no protection at all.

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CONCLUSION

Based on the results of our systematic review, we strongly recommend FFS protection with a mouth guard to reduce the number of head injuries and time lost from practice or games across all levels of ice hockey. The lowest overall rate of injury was seen with FFP, whereas PFP had a lower rate of injury than NFP. FFS have been shown to decrease concussion severity and rates of facial, eye and dental injuries, without increasing the risk of other head and neck injury. Half face shields, although not as protective as FFS, have been shown to be more protective than NFS. Mouth guards, while not decreasing the number of concussions in ice hockey, have been shown to allow for a faster return to play following concussion, suggesting milder head injury in those wearing mouth guards. Despite these results, more prospective evidence is needed to evaluate further the role of protective equipment and injury reduction in ice hockey. The results of this systematic review should prompt administrators in hockey leagues at all levels to consider strongly mandatory FFS use.

Competing interests: None.

Provenance and peer review: Not commissioned; externally peer reviewed.

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COS Survey – Eye Injuries in Canadian Sports

(Blinded eyes statistics in brackets)

Sport	1972-1973	1974-1975	1976-1977	1977-1978	1978-1979	1979-1980	1980-1981	1981 - 1982	1982-1983	1983-1984	1984 - 1985	1985 1996	1986-1987	1987-1988	1988 - 1999
Hockey	287(20)	258(43)	90(12)	52(8)	43(13)	85(21)	68(20)	119(18)	115(13)	124(12)	121(18)	123(22)	93(18)	62(11)	37(8)
Racquet Sports			43(3)	12(1)	28(1)	58(1)	103(4)	100(3)	88(5)	115(6)	81(6)	83(1)	66(3)	45(4)	62(3)
Baseball			19(2)	2	2	10	15	41(5)	68(3)	56(3)	43(2)	32(3)	34	16	24(2)
Ball Hockey			24(3)	8	9(2)	27(2)	22(4)	10(2)	19(3)	25(2)	29(1)	28	18(1)	31	24(1)
Football			13(1)	2	3	1	8	4	27(1)	22(1)	15(1)	10	20(1)	12(1)	10(1)
War Games											26(14)	8(2)	9(1)	2	6(4)
Golf			5(1)		1	1		5(4)	7(2)	3(2)	4(1)	5(2)	4(1)	1	3
Basketball				1			2	4	3	5	1	2	7		2
Skiing			1(1)	1	3(2)			3(2)		2(1)	4	2(1)	6(1)	1	
Volleyball			3(3)			6	3	2	3	4(1)		2			3
Broomball			2(2)			2	2	3	3	2		1	4	3	1
Lacrosse			3	1	1				3	4	1	1			
Hunting & BB Guns							4(1)			1	5(2)	1			3(1)
Snowmobiling			2(1)					1 (1)		1	1	2(1)	1 (1)	1 (1)	
Other			6				3	6(1)	6(1)	9(2)	19(2)	7(1)	5(1)	9(3)	7(1)
Total	287(20)	258(43)	211(29)	79(9)	92(18)	197(24)	230(29)	298(36)	342(28)	373(30)	350(47)	307(33)	267(28)	183(20)	182(19)

Notes:

309 Blind Eyes – Canadian Hockey – 1972 – 2001

0 Blind Eyes of players wearing CSA approved full face protectors

8 Blind Eyes of players wearing CSA approved visors because of loose neck straps, chin cup & chin strap

(Blinded eyes statistics in brackets)

Sport	1989-1990	1990 - 1991	1991-1992	1992-1993	1993-1994	1994-1995	1995-1996	1996-1997	1997-1998	1997-1999	1999-2000	2000-2001	2001-2002	TOTAL
Hockey	33(6)	21(3)	28(7)	32(5)	16(7)	19(5)	22(7)	12(3)	23(4)	8(5)	13(1)	6(1)	4(2)	1914(311)
Racquet Sports	40(2)	35(1)	33	31(1)	27	14(1)	17	18(1)	12	5	11	7	1	1135(47)
Baseball	15(1)	14(1)	18(1)	17(1)	23(2)	17(2)	11(2)	13(1)	10	5(1)	4	4(1)		513(32)
Ball Hockey	14(1)	20(2)	12(2)	16(2)	11(3)	'7	4	13(1)	7	8(2)	5	6		397(35)
Football	8	6	6	3(1)	5(1)	4	2	8	2	5	1	1	4(1)	202(10)
War Games	4(2)	4	3(2)	1 (1)	3(3)	2(1)	3(1)	4(1)	1	1 (1)	1		2	80(33)
Golf	5(1)	4	4(1)	1 (1)	2	1	1	4(3)	3(1)	1				65(20)
Basketball	2	2	2	4(1)	5	1	2	3	3(1)			1		52(2)
Skiing	4	3(1)	1	2		3(1)	1	2(1)	1			1		40(11)
Volleyball		1						1		1				29(4)
Broomball	2													25(2)
Lacrosse	1	2		1			1			1				20
Hunting & BB Guns		1	1					3			1 (1)			20(5)
Snowmobiling	1								4(1)					7(6)
Other	6(2)	5	8(1)	2	3		2(1)	4	9	1		1		128(17)
Total	135(16)	118(8)	116(14)	110(13)	95(16)	68(10)	66(11)	85(11)	75(7)	36(9)		27(2)	11(3)	4638(535)

Notes:

309 Blind Eyes – Canadian Hockey – 1972 – 2001

0 Blind Eyes of players wearing CSA approved full face protectors

8 Blind Eyes of players wearing CSA approved visors because of loose neck straps, chin cup & chin strap

American Collegiate Men's Ice Hockey

An Analysis of Injuries

Kyle Flik, MD, Stephen Lyman, PhD, and Robert G. Marx,* MD, MSc, FRCSC
From the Hospital for Special Surgery, New York, New York

Background: Reported rates and types of ice hockey injuries have been variable. Ice hockey combines tremendous speeds with aggressive physical play and therefore has great inherent potential for injury.

Purpose: To identify rates and determinants of injury in American men's collegiate ice hockey.

Study Design: Prospective cohort study.

Methods: Data were collected from 8 teams in a Division I athletic conference for 1 season using an injury reporting form specific for ice hockey.

Results: There were a total of 113 injuries in 23 096 athlete exposures. Sixty-five percent of injuries occurred during games, although games accounted for only 23% of all exposures. The overall injury rate was 4.9 per 1000 athlete exposures (13.8 per 1000 game athlete exposures and 2.2 per 1000 practice athlete exposures). Collision with an opponent (32.8%) or the boards (18.6%) caused more than half of all injuries. Concussion (18.6%) was the most common injury, followed by knee medial collateral ligament sprains, acromioclavicular joint injuries, and ankle sprains.

Conclusions: The risk of injury in men's collegiate ice hockey is much greater during games than during practices. Concussions are a main cause for time lost and remain an area of major concern.

Keywords: ice hockey; athlete exposures; injuries; concussions

Considered one of the fastest and most aggressive team sports, men's ice hockey has great potential for injury. Players move on sharp skates at speeds of up to 30 mph on a solid ice surface that is confined by rigid boards along the rink's periphery. Sticks made of wood, carbon graphite, or aluminum are used to propel a piece of vulcanized rubber at speeds of up to 100 mph (161 km/h).[15] The goalposts, which are made of steel, add an additional hazard. In such a setting, injuries are to be expected.

Ice hockey is played in many countries throughout the world and has become a popular sport for both men and women in many regions of the United States. At the elite level, this unique team sport is played by highly conditioned athletes and requires a combination of strength, agility, balance, skill, and controlled aggression. Specialized equipment is needed to protect players from each other, the ice, boards, goalposts, skates, pucks, and sticks. With improved protection and required use of helmets with facemasks, certain injuries such as facial lacerations, eye injuries, and dental injuries are diminishing. Nonetheless, blunt trauma remains the most common cause of injury, followed by fatigue and overuse.[4,17]

The rate and types of ice hockey injuries at different levels of play vary throughout the world.[1-4,7,8,10-12,14,17,19] When comparing injury data from American elite hockey players with that from their European counterparts, one must understand the subtle differences in the North American and European games. In general, the North American style of play is considered more aggressive and physical than that in Europe, where many prior studies were performed. In addition, the surface area of American rinks (approximately 1560 m^2) is considerably smaller than European rinks (1800 m^2). For these reasons, it may be reasonable to expect injury patterns to be different between American and European amateur elite hockey leagues.

The objective in this study was to describe the injury patterns in collegiate men's ice hockey in the United States with respect to the distribution of injuries by body region, player position, type of exposure (game vs practice), timing of injuries, and the associated time lost from participation.

MATERIALS AND METHODS

All data were collected prospectively during the 2001-2002 season. The athletic trainers of all 12 Eastern College

*Address correspondence to Robert G. Marx, MD, MSc, FRCSC, Sports Medicine and Shoulder Service, Director, Foster Center for Clinical Outcome Research, Hospital for Special Surgery, 535 East 70th Street, New York, NY 10021 (e-mail: marxr@hss.edu).

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Athletic Association Division I Men's Ice Hockey programs were contacted and asked to participate. Eight schools agreed and were included. An anonymous ice hockey injury data capture form was developed to record detailed information regarding each injury (Appendix, available in the online version of the article at www.ajsm.org/cgi/content/33/2/183/dc1). Injury data were recorded by using this method for each qualifying injury. A pilot study was performed 2 years before this to evaluate the form and confirm its ease of use.

The certified athletic trainer on each team was responsible for entering injury data at the completion of each exposure. The injury form was mailed to the study center at the end of each month, providing the opportunity to change the initial diagnosis if further testing or review of the injury by a team physician led to a change in diagnosis. For each injury, the player's age, height, weight, and position were recorded. Included in the information gathered was whether the injury occurred during practice or a game, the period within the game, and the location on the ice. In addition, it was noted if the injured body part was protected by equipment or had been previously injured. The injured player and the team's trainer determined the direct cause of the injury. Time lost was calculated by adding all consecutive practices and games that were missed because of the injury. Additional documentation included whether the injury was due to illegal activity and whether this activity was penalized. Information was also collected on the diagnostic procedures required, the treatment received, and the final diagnosis. Ultimately, the trainers were asked the question, "Do you believe the injury could have been prevented by better equipment, conditioning, or refereeing?"

An athlete exposure (AE) consisted of a single player participating in a single game or practice. Exposure information was recorded based on the at-risk population determined by the average number of players at each position participating at practices and the number participating in each game. A specific daily attendance log was not kept. An injury was defined specifically as any injurious episode that led to loss of participation in the immediate subsequent AE, whether it was a practice or a game. The injury definition was validated on the injury form by recording specific time loss information.

Each team's athletic trainer was responsible for completing the injury forms and returning them to the study center at the end of the season. Each trainer was contacted bimonthly to ensure compliance. For each team, exposure information was calculated based on number of games and practices and number of players at each game or practice. All data were reviewed for accuracy and completeness by the researchers before statistical analysis. Any unclear or missing data were clarified with the responsible trainer and/or player at the end of the hockey season.

Descriptive statistics consisted of the calculation of frequencies and percentages. Injury rates were calculated as the number of injuries per 1000 AEs. Inferential analysis consisted of χ^2 tests comparing rates within subgroups. A P value < .05 was considered statistically significant. All analysis was performed using SPSS for Windows version 11.0 (SPSS Science Inc, Chicago, Ill).

RESULTS

There were a total of 113 injuries in 23 096 AEs for a total overall injury rate of 4.9 per 1000 AEs (Table 1). The game injury rate was 13.8 per 1000 AEs (74 injuries in 259 games), whereas the practice rate was 2.2 per 1000 AEs (39 injuries in 676 practices) for a risk ratio of 6.3. Therefore, game injuries were 6.3 times more common than practice injuries ($P < .001$). Forwards and defensemen had similar injury rates, whereas goalies had significantly lower injury rates during games ($P < .05$) (Figure 1).

The incidence of game injuries was slightly higher in the first half of the season (57%) than in the second half (43%). Of the 74 game injuries, 27 occurred in the first period, 27 in the second period, and 20 in the third period. Fifty seven percent of injuries occurred to a player who was on the visiting team, and 43% occurred to a home player.

A collision, either with an opponent (32.8%) or the boards (18.6%), was the cause for more than half of all injuries (Figure 2). Skates, sticks, or pucks were directly responsible for only 11.5% of all injuries. Eight percent of injuries were considered overuse injuries. For only 9 injuries did the team trainer feel that the injury was preventable by better equipment, conditioning, or refereeing.

Injuries during games were related to collisions in 69%, whereas practice injuries were related to collisions in only 38%. Nearly 40% of all injuries occurred along the boards. Concussion was the single most commonly sustained injury (18.6% overall) and was responsible for nearly one quarter of all game injuries. Of the 21 concussions recorded, only 4 (19%) occurred during practice. Six of the 17 game concussions were thought to be due to illegal activity, with no penalty called on the play. Elbowing was the most common illegal play. It was felt that the injury could not have been prevented in 8 cases and could have been prevented by better equipment in 3 cases. The average time loss for each concussion was 2.1 games and 6.9 practices (approximately 9 AEs total). Of the 21 concussions, forwards sustained 16 and defensemen suffered 5.

Knee medial collateral ligament (MCL) sprains were the second most frequent injury. Interestingly, these were all game related; no MCL sprains occurred during practices. The injury type that led to the longest average time lost was a syndesmotic ankle sprain ("high ankle sprain"). Five such injuries resulted in a mean of 5.4 games and 14.6 practices missed. Overall, injuries affected a wide variety of body parts. See Figure 3 for a complete breakdown of injury by anatomical location and Figure 4 for time loss by major injury type.

DISCUSSION

This prospective epidemiological study demonstrates unique injury patterns in men's collegiate ice hockey in the United States. Although the overall injury rate is high

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TABLE 1
Injury Rates for Game Versus Practice, Position, Home/Away, and by Period

Variable	N	%	AEs[a]	Rate	Rate Ratio (95% CI)[b]	P
All injuries	113	100.0	23 096	4.9		
Game	74	65.5	5377	13.8	6.3 (4.2-9.2)	<.01
Practice	39	34.5	17 719	2.2	Referent	
Position						
Goalie	7	6.2	2583	2.7	Referent	
Defense	37	32.7	7293	5.0	1.9 (0.8-4.2)	.16
Forward	69	61.1	13 220	5.1	1.9 (0.9-4.2)	.09
Game injuries						
Home	32	43.2	2688	11.9	Referent	
Away	42	56.8	2688	15.6	1.3 (0.8-2.1)	.24
First period	27	36.5	1792	15.1	1.5 (0.8-2.4)	.30
Second period	27	36.5	1792	15.1	1.5 (0.8-2.4)	.30
Third period	20	27.0	1792	11.2	Referent	

[a]AEs, athlete exposures.
[b]CI, confidence interval.

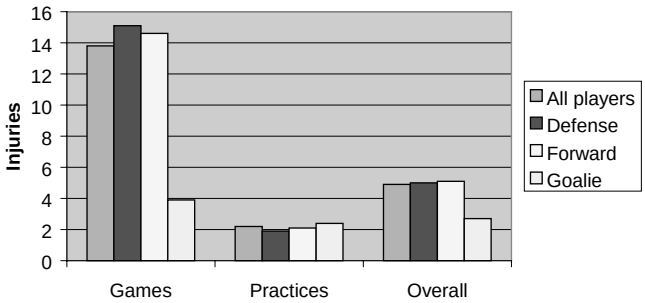

Figure 1. Injury rate by position per 1000 athlete exposures.

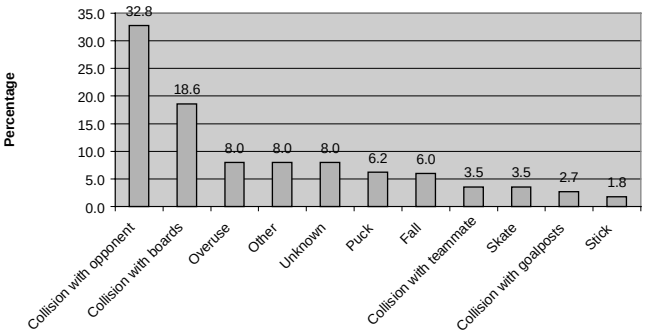

Figure 2. Cause of injuries.

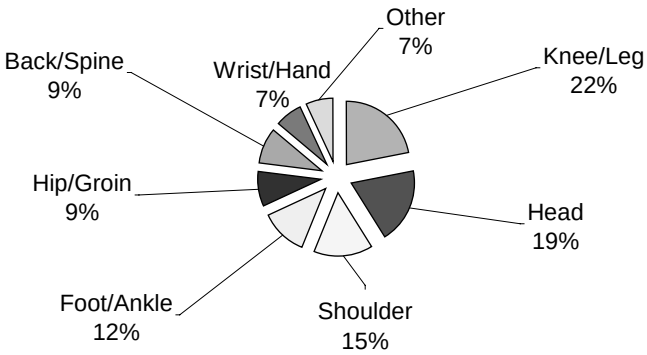

Figure 3. Injuries by body part.

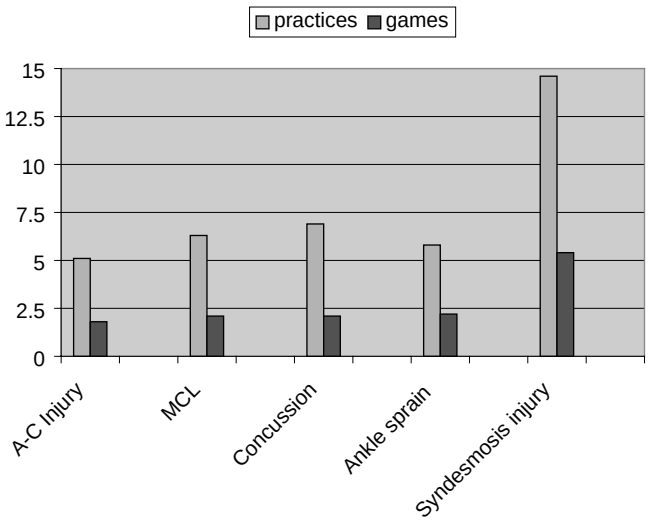

Figure 4. Mean time loss by major injury type.

when compared to other collegiate sports (National Collegiate Athletic Association [NCAA] Injury Surveillance System data), of interest is the relative and absolute low number of injuries that are sustained during practices compared to during games. Practice injury rates in men's ice hockey were the lowest of all winter sports included in the NCAA injury surveillance system data in 2001. This finding is possibly a result of the aggressive nature of the sport during competition, which would explain the higher incidence of collision-related injuries

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during games. The hockey player is well protected by equipment and therefore unlikely to be injured unless contact at high speed is involved (more common in a game scenario). Checking (purposeful body contact in which 1 player attempts to disrupt the progress of an opposing player) and other potentially injurious acts, such as slashing with the hockey stick, are less common during practices than during games.

Prior studies that have used a retrospective design were less accurate in identifying a true at-risk population. The prospective design in this study allows accurate calculations of specific incidence rates. Our overall injury rate of 4.9 per 1000 AEs is consistent with prior North American studies.[4,5] The practice injury rate in this study of 2.2 per 1000 AEs is similar to the recent review by Ferrara and Schurr, who examined intercollegiate ice hockey injuries via a causal analysis and reported a rate of 2.5 per 1000 AEs.[4] Our game-associated injury rate of 13.8 per 1000 AEs is also similar to 14.7 per 1000 AEs reported in their series. The NCAA Injury Surveillance System data from the same 2001-2002 season revealed a practice injury rate of 2.0 and a game rate of 19.7. The slightly higher game injury rates are likely attributed to the fact that the NCAA includes injuries that require sutures, even if there was no associated time loss from the injury.

Collisions are the main cause of injury in Division I Men's Ice Hockey. One third of all injuries in this study were caused by collision with an opponent, and nearly 20% of injuries were related to collision with the boards. Stuart and Smith[17] found collisions to be the cause of 51% of the injuries reported in their review of American Junior A level hockey.

Early investigations performed before the mandatory facemask and helmet rules demonstrated a high incidence of facial injury and lacerations.[9,13,17,18,21] Our extremely low rate of laceration injury is partly explained by the now mandatory use of helmets with facemasks in NCAA hockey but also by our injury definition. A laceration in our investigation was reportable only if it caused the player to miss the subsequent game or practice. Therefore, unless a laceration was major, it was unlikely to be included.

Stick-related injuries have been the cause of up to 14% of injuries in other studies,[10,16,17] especially studies reporting injuries from European teams, whose players often are not required to wear facemask protection. We found only a small number of injuries (1.8%) to be caused by the stick. Although this finding may be mostly related to diminished stick-related facial lacerations secondary to facemask use, it may also reflect the improved padding in today's equipment, which may diminish other stick-related injuries such as fractures and contusions.

Concussion remains a serious concern in this study population and accounted for nearly 1 in 5 injuries. As documented previously,[6] hockey has one of the highest rates of concussion injuries among contact sports. Consistent with other studies of hockey injuries, concussion occurred most often during games (81%) compared with practice (19%). This proportion is nearly identical to that reported in Swedish elite hockey by Tegner and Lorentzon.[20] Of the 6 concussions that were considered to be the result of illegal play, 3 were the result of elbowing. More severe penalties and/or suspensions should be considered for elbowing to the head to reduce these injuries.

The position of the player has been reported to be an important factor in concussion susceptibility. Of the 21 reported concussions, forwards suffered 16 (76%), defensemen 5 (24%), and goalies none. Therefore, higher concussion rates exist in forwards compared to their proportional representation on the ice (50%), whereas the rate in defensemen is more consistent with their proportional representation on the ice (33%). When adjusted for the number of players on the ice, forwards had 2.1 times as many concussions as the defensemen. Although these data are similar to those of a recent Canadian study of players of similar caliber,[6] a Finnish study recently reported that of 9 concussions suffered in the Finnish National League during a season, 5 were sustained by goaltenders.[11] This difference in concussion rates and susceptibility between American and Finnish players may be explained in part by the differences in the game. The American style is traditionally more physical than the European style and places the forwards at higher risk for contact injuries because of the common "dump and chase" offensive strategy. In addition, American ice hockey is played on a smaller ice surface. Nonetheless, we feel that referee vigilance and strict penalization for delivering direct blows to the head would help reduce the number of concussions suffered by ice hockey players in view of these findings.

Rule enforcement or rule changes have been effective in the past. Watson et al collected injury and penalty data in Canada to evaluate the effectiveness of the "checking from behind" rule and concluded that the rule change led to a safer environment in a Canadian university league.[22] They found a significant decrease in injury rates to the head, neck, and back after the institution of the rule, designed specifically to lower the rate of cervical spine injuries in hockey players. Despite a high incidence of concussion in our study, there were no cervical spine injuries. Rules against checking from behind are strictly enforced in college hockey in the United States.

One unique injury that appears to be relatively common in ice hockey is the syndesmosis injury or high ankle sprain. The elevation provided by the hockey skate blade combined with high speeds and rapid direction changes while skating place the ankle at higher potential for torque injury, often related to "catching a rut" in the ice with the skate blade. Also, the stiff hockey boot provides stability to the ankle but perhaps places the region directly proximal to the boot at higher risk. Future biomechanical testing could provide further insight into this problem and potentially lead to alteration in skate design.

Strengths of the study include its prospective design and the comprehensive hockey injury data capture form that was created to provide detailed information about each injury. Standardized forms that are used across a variety of sports provide little detail regarding sport-specific circumstances surrounding an injury. *Injury* was defined as an event that caused a player to miss the subsequent practice or game.[10] This standard injury definition was used because inconsistent injury definitions make comparisons

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of epidemiological descriptions of sports injuries difficult. Certainly, with this definition it is possible that some legitimate injuries (such as a deep laceration) were not counted because the player may not have missed the subsequent exposure.

An additional potential weakness of this study might be the reliance on reporting of injuries by the team trainer. In each case, the trainer was very interested in study participation and was in contact with 1 of the authors (K.F.) on a biweekly basis. Although concern over the accuracy of the diagnoses made by the team trainer is legitimate, it was not feasible to have a physician opinion on each injury. If an injury required an evaluation by an orthopaedic surgeon, the final diagnosis reported to us was that diagnosis given by the physician. It is certainly possible that the trainers misdiagnosed some injuries. Finally, a single-season injury study, even one that includes many teams, has the potential to not represent actual risk. For example, our 0% rate of concussions to goaltenders underestimates the true rate.

CONCLUSION

There was a significant difference in the injury rates suffered during games and practices. Contact among players is the major cause of injury, and this occurs mainly during game situations. Concussions and knee MCL injuries are common and are primarily incurred during games. The relatively high rate of syndesmosis injuries is unique to ice hockey and shows little preference for game or practice situations. Overall injury rates were similar for forwards and defensemen, but the rate of concussion injury was highest among forwards. Stricter enforcement of rules relating to illegal blows to the head may reduce the rate of this significant injury.

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ORIGINAL CONTRIBUTION

Head and Neck Injuries Among Ice Hockey Players Wearing Full Face Shields vs Half Face Shields

Brian W. Benson, MSc
Nicholas G. H. Mohtadi, MD, MSc
M. Sarah Rose, PhD
Willem H. Meeuwisse, MD, PhD

Context Speculation exists that use of a full face shield by ice hockey players may increase their risk of concussions and neck injuries, offsetting the benefits of protection from dental, facial, and ocular injuries, but, to our knowledge, no data exist regarding this possibility.

Objective To determine the risk of sustaining a head or neck injury among intercollegiate ice hockey players wearing full face shields compared with those wearing half shields.

Design, Setting, and Participants Prospective cohort study conducted during the 1997-1998 Canadian Inter-University Athletics Union hockey season of 642 male hockey players (mean age, 22 years) from 22 teams. Athletes from 11 teams wore full face shields and athletes from 11 teams wore half face shields during play.

Main Outcome Measure Reportable injury, defined as any event requiring assessment or treatment by a team therapist or physician or any mild traumatic brain injury or brachial plexus stretch, categorized by time lost from subsequent participation and compared by type of face shield.

Results Of 319 athletes who wore full face shields, 195 (61.6%) had at least 1 injury during the study season, whereas of 323 who wore half face shields, 204 (63.2%) were injured. The risk of sustaining a facial laceration and dental injury was 2.31 (95% confidence interval [CI], 1.53-3.48; $P<.001$) and 9.90 (95% CI, 1.88-52.1; $P = .007$) times greater, respectively, for players wearing half vs full face shields. No statistically significant risk differences were found for neck injuries, concussion, or other injuries, although time lost from participation because of concussion was significantly greater in the half shield group ($P<.001$), than in the group wearing full shields.

Conclusions These data provide evidence that the use of full face shields is associated with significantly reduced risk of sustaining facial and dental injuries without an increase in the risk of neck injuries, concussions, or other injuries.

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ICE HOCKEY HAS BEEN CHARACTERIZED as the fastest and most violent team sport in the world played with clubs (hockey sticks), a bullet (puck), and knives (skates).[1] The high intensity of the sport results in frequent collisions between players and forceful impacts with the side boards, goal posts, pucks, and hockey sticks. The head and neck are particularly vulnerable to hockey-related injury,[2-10] and the search for improved player safety to prevent these injuries has been an ongoing process for sports governing bodies and researchers.[11]

Ice hockey associations from Canada and the United States have introduced head and neck risk management strategies, the most significant being the mandatory use of full facial protection for athletes across many different age groups and levels of play. These rules, combined with advancements in face shield standards, have been effective in reducing the frequency of facial and eye injuries.[12-14] However, a trend of increasing catastrophic hockey-related injuries to the cervical spine has led to speculation that use of the full face shield may increase players' risk of sustaining a neck injury, possibly due to biomechanical alterations or changes in the style of play.[15-20] To our knowledge, no prospective cohort or experimental research has been conducted in the sport of ice hockey to address this issue.

The purpose of this study was to determine the risk of sustaining head or neck injury among intercollegiate ice hockey players wearing full face shields compared with those wearing half shields.

METHODS

This prospective cohort study was conducted during the 1997-1998 Canadian Inter-University Athletics Union (CIAU) hockey season. The CIAU is a national league consisting of 4 divisions with identical officiating and player eligibility rules,

Author Affiliations: Faculties of Medicine (Mr Benson and Dr Rose) and Kinesiology, Sport Medicine Centre (Drs Mohtadi and Meeuwisse), University of Calgary, Calgary, Alberta.
Corresponding Author and Reprints: Willem H. Meeuwisse, MD, PhD, University of Calgary Sport Medicine Centre, 2500 University Dr NW, Calgary, Alberta, Canada T2N 1N4 (e-mail: meeuwiss@ucalgary.ca).

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except that each division may mandate use of protective equipment. The Ontario Universities Athletic Association (OUAA) requires that all athletes under its jurisdiction wear full face shields for every practice and game throughout the varsity season. In contrast, the Canada West Universities Athletic Association (CWUAA) and Atlantic Universities Athletic Association (AUAA) require all athletes to wear, at a minimum, half shields (visors) for all practices and games. A half shield is a clear plastic visor that is attached to a helmet and extends down to the tip of a player's nose. A full face shield extends down to the bottom of a player's chin and covers the entire face. For this study, 2 cohorts were defined by their associations' facial protection mandate.

Subjects and Data Collection

The study population included 642 consenting male ice hockey players from 11 CIAU university teams competing in the OUAA, 7 teams in the CWUAA, and 4 teams in the AUAA. Thus, athletes from the 11 OUAA and 11 CWUAA/AUAA teams formed the cohorts wearing full and half face shields, respectively.

The 11 OUAA teams were selected based on geographic proximity to a preseason meeting in Toronto, Ontario, between the investigator and team therapists. The 7 CWUAA team therapists had been collecting data for the Canadian Intercollegiate Sport Injury Registry (CISIR), Calgary, Alberta, during the previous 3 seasons, and the 4 AUAA teams were selected based on geographic proximity to a preseason meeting in Halifax, Nova Scotia. The purpose of the meetings was to discuss the data collection protocol with team therapists so that they would have a thorough understanding of what was required of them in terms of data collection.

Approved by the University of Calgary's institutional review board, this study required team therapists to obtain informed verbal consent from all players prior to participation. Previous injury status was documented for each CWUAA player using a standardized medical form during a mandatory preseason medical examination with the team physician. Because no standardized form was uniformly used by team physicians of institutions participating in the OUAA and AUAA, athletes competing in these divisions completed an injury history questionnaire with their team therapist during a preseason meeting. The injury information recorded on these forms served as a baseline for the 1997-1998 hockey season. From the first practice of the season, team therapists used a standardized weekly exposure sheet[21] to record the level of individual participation (full, partial, or none) and the type of face shield worn for every practice and game throughout the season. If a player sustained an injury that met the reportable injury definition, team therapists, physicians, or both were required to complete an injury report form.[21] A subjective assessment of whether an injury was the result of an illegal action during a game and subsequently whether a penalty was called by a referee was included on the injury report form so that a descriptive comparison could be made with respect to style of play between facial protection divisions. The completed forms were sent to the CISIR, where they were checked, coded, and entered into a database using a dual entry system to minimize data entry errors.[21]

Team Therapists

Athletic therapists were important members of the investigative team because of their medical orientation and diagnostic skills; their relationships with athletes, coaches, and team physicians; and their daily presence at both practices and games. Canadian Athletic Therapist certification requires: (1) enrollment in a university undergraduate degree program in kinesiology or physiotherapy, (2) the completion of 1200 hours of practical experience (600 clinical and 600 field hours), and (3) successful completion of a national examination consisting of both written and practical components. During training, student athletic therapists are under the direct supervision of certified Canadian Athletic Therapists. Each participating coordinating athletic therapist received a small honorarium for overseeing data collection throughout the 1997-1998 season.

Outcome Variables and Statistical Analysis

The main outcome (dependent) variable measured in this study was an injury defined as "any event requiring assessment or treatment by a team therapist or physician" and "any mild traumatic brain injury or brachial plexus stretch (ie, burner/stinger)." Reportable injuries were then categorized by the amount of time lost from subsequent participation. To avoid potential injury reporting biases between institutions, the following injury definition was used for data analyses: (1) any injury received during an organized practice or game during the 1997-1998 CIAU hockey season that required assessment or treatment by a team therapist or physician and resulted in at least 1 missed participation, or (2) any facial laceration/fracture, dental injury, eye injury, traumatic brain injury, or brachial plexus stretch (regardless of playing-time lost).

The independent variable of interest was full face shield vs half facial shield. Individual athlete-participation (*exposure*), defined as 1 player participating in 1 practice or game in which the athlete was exposed to the possibility of injury, was measured to determine the amount of time under the different exposure conditions during which each individual was at risk of injury. Athlete-exposures were calculated by weighting a full session of participation as "1," a partially missed session as "0.5," and a completely missed session as "0." Goal tender exposure and injury information was not included in the analyses so that a true comparison could be made between players wearing full face shields vs half shields. For example, 20 athletes fully participating in 10 games would yield 200-game athlete exposures. In addition, several potential confounding or effect-modifying variables were assessed, including player posi-

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tion (forward vs defense), injury status (new vs recurrent), and injury setting (game vs practice).

The sample size chosen for this study was calculated based on injury data from the CISIR database during the previous CWUAA varsity season, which showed that neck injuries accounted for approximately 7% of the total reported player injuries wearing half shields. Using a 2-sided test ($\alpha = .05$, $\beta = .20$), it was estimated that 300 subjects in each group would be required to achieve 80% power to detect a relative risk of 2.0 or greater between the 2 study groups. The test-based method of Miettinen[22] was used to calculate 95% confidence intervals (CIs) for relative risks based on incidence density ratios[23] and *Epi Info 6* (Version 6.04a, Centers for Disease Control and Prevention, Atlanta, Ga) was used for all *P* value computations (2-tailed tests).

RESULTS

Athletes and Athlete-Exposures

All 642 varsity athletes from the 22 participating teams consented to have data collected and sent to the CISIR . In addition, athlete-exposure information was 100% complete throughout the 1997-1998 CIAU hockey season. Furthermore, the weekly exposure sheets were verified when they were received at the CISIR for any indication that a player had missed time due to an injury. In all cases but 1, an injury report form was sent to the CISIR (99.9% completion rate). It was not possible to determine the completion rate for injuries that did not result in time lost from participation.

The median age for athletes in both categories was 22 years (range, 17-29 years, full face shield division; range, 18-29 years, half face shield division). Of the 319 athletes wearing full face shields, 195 players (61.1%) were injured during the 1997-1998 season compared with 204 (63.2%) of the 323 athletes wearing half face shields. In addition, the 2 study groups, classified as rookies, had a similar level of playing experience, with 118 (41.9%) of the 281 players wearing full face shields, compared with 116 (39.5%) of 294 players wearing half shields. Furthermore, 280 (95.2%) of the 294 athletes competing in half shield divisions had 1 or more injuries prior to the study compared with 247 (87.9%) of 281 players competing in the full shield division. Of note, 38 athletes (11.9%) competing in the full shield and 29 athletes (9%) competing in half shield divisions did not have medical forms or injury history questionnaires returned to the CISIR. These individuals participated for a minimal amount of time as varsity players or joined a team later in the season.

Excluding goal tenders, players wearing full face shields accumulated 24 147.5 athlete-exposures during the 1997-1998 varsity season, while players wearing half shields amassed 26 823.0 athlete-exposures.

Injury Risk

Crude relative risk estimates of head and facial injuries, neck injuries, concussions, and other types of injuries sustained by players from each cohort are shown in TABLE 1. Although we found a significant difference in rates of head and facial injuries between the 2 groups ($P<.001$), there was no significant difference in risk of sustaining a concussion, neck, or other injury (overall) for athletes wearing half shields compared with those wearing full face shields. Determined by multivariate (stratified) analysis, these findings were not confounded or modified by injury setting (game vs practice), injury status (new vs recurrent), or position (forward vs defense), which were recorded on the injury report forms by the team therapists and physicians. The same differences between face shield types were found across all strata. The incidence rates of head injuries sustained for each cohort are shown in FIGURE 1. There were no significant differences in the number of concussions sustained between the 2 study groups ($P = .90$).

Facial Lacerations and Dental Injuries

The risk of sustaining a facial laceration was 2.31 times greater for players wearing half shields compared with those wearing full face shields (95% CI, 1.53-3.48; $P<.001$). Based on the absolute number of facial lacerations sustained during the 1997-1998 season (half shield, 77; full shield, 30), an average of 7 facial lacerations per team are expected during any given season for athletes wearing half face shields, compared with 2 to 3 injuries per team for athletes wearing full face shields.

The incidence rates for various types of facial lacerations sustained by athletes in the 2 study groups are presented in FIGURE 2. The lip and eyebrow are 2 areas of the face that receive the most lacerations among players wearing half shields. The majority of lacerations sustained by athletes wearing full face shields were to the chin. Although there was a higher incidence of chin lacerations among players wearing full face shields, the incidence was not significantly greater than for players wearing half shields ($P = .25$).

Table 1. Crude Relative Risk Estimates of Injury for Players Wearing Full Face Shields Compared With Half Shields

	Half Shield		Full Shield				
Variables	No. of Injuries	Injury Rate*	No. of Injuries	Injury Rate*	Relative Risk	95% Confidence Interval	*P* Value
Head and face injuries†	95	3.54	34	1.41	2.52	1.73-3.68	<.001
Concussions‡	41	1.53	38	1.57	0.97	0.61-1.54	.90
Neck injuries§	9	0.34	7	0.29	1.16	0.43-3.16	.77
Other injuries‖	202	7.53	150	6.21	1.21	0.98-1.49	.07

*Per 1000 athlete-exposures.
†Any facial laceration or fracture, dental, or eye injury (excluding concussions), regardless of time loss.
‡Any concussive head injury including all transient traumatic brain injuries, regardless of time loss.
§Injuries resulting in 1 or more lost participation and all transient brachial plexus injuries, regardless of time-loss.
‖Only injuries resulting in 1 or more lost participation.

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Stick contact to the face was the predominant mechanism of lacerations for athletes wearing half shields, whereas contact with a stick or an opponent via body check or collision caused the majority of lacerations among athletes wearing full face shields (TABLE 2). Six facial lacerations sustained by athletes wearing half shields were caused by a skate.

Eleven players wearing half face shields sustained dental injuries (ie, teeth fractures) during the 1997-1998 varsity season. Nine of the 11 dental fractures sustained by athletes wearing half shields were caused by contact with a hockey stick, 1 resulted from contact with a player's own visor, and the remaining mechanism was unknown. Seven of the 11 players injured in this cohort were wearing mouthguards at the time of injury. Only 1 athlete, who was wearing a full face shield but not a mouth guard, sustained a dental injury when he was punched in the face during an organized practice. The risk of sustaining a dental injury was 9.90 times greater for players wearing half shields than for those wearing full facial shields (95% CI, 1.88-52.1; $P = .007$). One eye injury (traumatic iritis) was sustained by a player wearing a half shield. This injury occurred when an opposing player's elbow struck the injured player's eye during an organized game. No eye injuries were reported for players wearing full face shields.

Concussions and Neck Injuries

We found no statistically significant risk differences in the number of concussions sustained by players in either study group ($P = .90$). However, the time lost from practices and games associated with these injuries was significantly greater for players wearing half shields than for those players wearing full face shields ($P < .001$). Forty-one players who sustained concussions while wearing half shields missed a total of 166.5 sessions, and 38 players who sustained concussions while wearing full face shields missed 64.5 sessions. In addition, 24 of the 41 players who experienced concussions while wearing half shields missed 1 or more complete sessions compared with 13 of the 38 players wearing full face shields. Using a time-lost definition, the injury rate is approximately double for players wearing half face shields. Concussive head injuries were randomly distributed between the participating teams; that is, there was no clustering of concussions on any 1 particular team within either cohort.

Only 5 neck injuries experienced by athletes wearing full face shields resulted in 1 or more completely missed games or practices, compared with 7 for athletes wearing half shields. The risk of neck injury was not significantly different between the 2 comparison groups ($P = .78$).

Reported Injury Factors

High sticking, slashing, and checking from behind, respectively, were the 3 most common types of illegal play causing injury reported by therapists of athletes competing in half shield divisions. In comparison, slashing, checking from behind, and cross-checking, respectively, were the top 3 reported types of illegal play causing injury in full face shield divisions.

Team therapists from the half shield divisions reported that 41.3% of the injuries sustained during games were caused by an illegal action, compared with 32.2% of the injuries sustained by players competing in the full face shield division. In addition, it was reported that a referee called a penalty after 10% of injuries sustained by players competing in half shield divisions vs 8.5% of injuries of players in the full face shield division.

An accurate assessment of the penalty information reported by team therapists was not possible because official game sheets were not collected for verification.

COMMENT

Public and professional concern about the number of head and neck injuries occurring in ice hockey is increasing. There is speculation that while full face shield use reduces facial, dental, and ocular injuries,[24] it may increase injury rates to other anatomic regions such as the neck, brain, and spinal cord, possibly due to biomechanical alter-

Figure 1. Incidence Density of Head and Facial Injury Outcome by Facial Shield Worn at Time of Injury

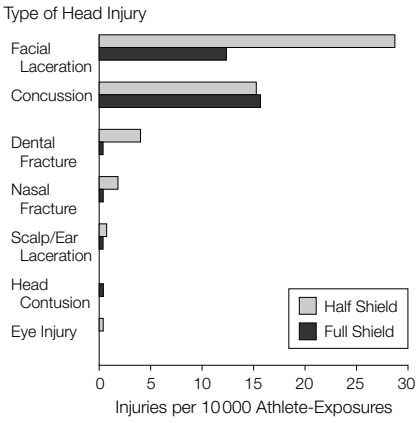

Figure 2. Incidence Density of Facial Lacerations by Area of Face and Facial Shield Worn at Time of Injury

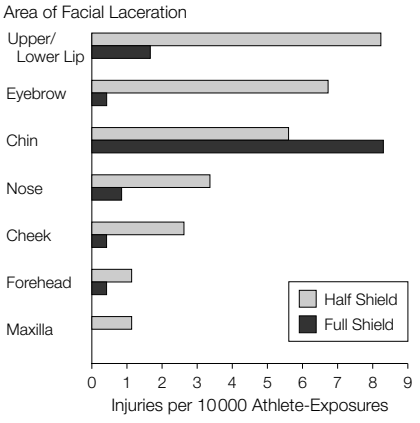

Table 2. Mechanism of Facial Lacerations by Type of Face Shield Worn at Time of Injury: Absolute Numbers

Mechanisms of Facial Laceration	Half Shield	Full Shield
Stick contact	45	12
Boards or glass contact	12	2
Player contact	8	11
Skate contact	6	0
Puck contact	5	1
Ice contact	3	3
Unknown	1	1

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ations[25,26] or changes in the style of play.[27] In this study, the risk of injury was prospectively evaluated in a natural experimental setting using 2 groups similar in every respect, apart from face shields use. There were no differences between sites, league divisions, or physicians and therapists in their threshold to decide to remove a player or limit a player's participation in a game or practice, and there was no differences in player skill levels or training. Furthermore, the data were collected using a previously validated system of injury surveillance,[21] and data collection was nearly 100% complete.

For intercollegiate ice hockey players wearing half shields compared with full face shields, we found that the risk of sustaining a head injury (excluding concussions), facial laceration, and dental injury was 2.52, 2.31, and 9.90 times greater, respectively. We found no evidence in this study to support the speculation that full face shield use increases players' risk of sustaining a neck injury or concussion. Furthermore, the overall risk of injury to an anatomic region other than the head or neck was not significantly different between the 2 comparison groups.

Several other findings warrant comment. First, a significant number of facial lacerations in the half shield group were to periorbital regions, which carries a risk of catastrophic eye injury. This concern is compounded when the evidence shows that stick and skate related facial injuries are far higher in this group.

Second, the finding that 7 of the 11 athletes who sustained dental injuries in the half shield group were wearing mouth guards at the time of injury suggests that the use of such protective equipment in combination with half shields is not enough to offer protection from these injuries. The use of full face shields significantly reduces players' risk of sustaining a dental injury.

Third, concussions sustained by players wearing half shields resulted in significantly greater time lost from competition than for players wearing full face shields ($P<.001$). Possible explanations for this finding may be that many players wearing half shields place their helmets in a manner such that the visors are raised above the level of their nose in order to get a clearer view of the ice and surrounding area. Since the helmet is placed further back on the head, the protective effect of the padding is minimized on the forehead area. In addition, a loose chin strap may allow a properly fitted helmet and half shield unit to shift from its original position during impact with an opposing player, thereby decreasing its protective effect. Furthermore, a direct blow to the exposed jaw of players wearing half shields may increase concussion severity compared with the full facial shield, which because of its chin piece may cushion the jaw. The chin piece of the full face shield also helps hold the helmet in place during impact, thereby maintaining maximum player-protection from brain injury. However, data or detailed evaluations of concussions, such as grade or associated or persistent neurologic deficits, were not collected, so the relative severity of injury between the groups could not be determined.

Finally, these findings are based on data from collegiate hockey players and may not be generalizable to high school and younger, skeletally immature players.

In this study, use of full face shields was associated with significant reductions in the risk of facial and dental injuries without increasing the risk of neck injuries, mild traumatic brain injuries, or other injury rates (overall). Sports governing bodies at the intercollegiate level of competition should seriously consider mandating full facial protective equipment for all participants under their jurisdiction.

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REVIEW

The avoidability of head and neck injuries in ice hockey: an historical review

N Biasca, S Wirth, Y Tegner

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The number of minor traumatic brain injury (mTBI), cerebral concussions, is increasing and cannot be eliminated by any kind of equipment. Prevention strategies, such as the introduction of "checking from behind" rules have become effective in decreasing the number of severe spinal injuries. A new "head checking" rule should reduce mTBI in the same way in the following years. Mouthguards should be mandatory as an effective device for the prevention of dental and orofacial injuries, as well as reducing the incidence and severity of mTBI. A new internet database system, the International Sports Injury System (ISIS) should improve epidemiological analysis of head, face, and spinal injuries worldwide. ISIS should provide an internationally compatible system for continuous monitoring of risk factors, protective effects of equipment, and protective effects of equipment and effects of changes in rules through the years.

See end of article for authors' affiliations

Dr Biasca, Head of Orthopaedic Surgery, Co-Head of Trauma Surgery, Spital Oberengadin, CH-7503 Samedan, Switzerland

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Ice hockey is a fast sport with a risk of injury as an integral part of the game. To reduce the risk of injuries, various types of protective equipment have been developed. However, this equipment should neither be a source of new injuries nor compromise the nature of game. In attempting to eliminate one risk, other risks may be created and the nature of the game may be influenced. The injury pattern of head and neck injuries has changed since the introduction of helmets and full facemasks. To meet the needs of an expanding ice hockey population, equipment manufacturers began to produce more encompassing body protection. Shoulder pads became large, covering the shoulder, chest, and half of the back with a hard shell. Elbow pads became sizeable, and their hard shell became a new weapon in the player's arsenal. Parallel to the better design and resilience of elbow and shoulder pads, the increased aggressiveness and the feeling of invincibility of the players paradoxically contributed to increase the risk of head and neck injuries. Catastrophic injuries, such as subdural haematoma and blinding, were reduced in the period 1963–1991 as a result of the compulsory wearing of helmets and full facemasks.[1–6] Despite this, the incidence of head injuries is now increasing.[7–20] Head and face currently represent the most common location of ice hockey injuries.[7–10] World wide, there is also an increasing incidence of minor traumatic brain injuries (mTBI), even called cerebral concussion.[7–20] Parallel to this, in the period 1982–1996, there was world wide a persistent number of 17 major spinal injuries a year.[21–26] These epidemiological data are very worrying and need to be analysed. The development of protective equipment requires a sound understanding of the injury mechanism, location, and type as well as the tissue response to stress, fatigue, and displacement velocity or acceleration. There is also the necessity to continuously analyse the epidemiological data with respect to the evolution of protective devices and ice hockey rules. The use of a uniform computerised www.ISIS (International Sports Injury System) based database is one way of detecting changes in the epidemiological pattern early on.[27]

HISTORICAL REVIEW

The beginnings of North American ice hockey have been traced back to 1783 when British troops used the natural arena of frozen ponds and rivers to ease the bleakness of winter.[2] In the 1920s, padding was minimal and worn as much to keep warm as to diffuse the blows of sticks and pucks. Players started to wear pants and soccer shin guards for leg protection. The large open areas of ice used for outdoor games facilitated a more open style of play which may account for the lack of concern about injury.

In 1929 the first facemask with leather was introduced for use by a goaltender to cover a broken nose, but, as vision was impaired, it was not used.[2] Jacques Plante, goaltender of the Montreal Canadians, started wearing a mask during games after he was hit on the nose by a slap shot from Ranger Andy Bathgate.[2] Head protection for goaltenders was met with mixed reviews, and it was not common in the National Hockey League until the North Star Bill Masterson was struck on the head by a puck and died.[2]

In the late 1930s shoulder protections with a leather cap added began to appear.[2] Elbow pads were fashioned from leather tubes filled with horsehair.[2]

In the late 1940s, hard plastic shoulder pads for football were covered with foam rubber and sometimes used in ice hockey.[2] Hard caps with foam covering also served as elbow pads.[2] Hockey gauntlets with leather palms and a cloth covered wrist guard made of bamboo strips formed the wrist-hand protection.[2] The first head protection appeared in the 1950s. It had squares of leather with thin felt backing for comfort and was worn on the forehead and back of the head[2] (fig 1).

When the game moved indoors, after the second world war, the play was more constricted

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Figure 1 Helmet of the late 1950s.[2]

Figure 2 Adjustable helmet with a liner of the cradle type, certified in 1963.[3]

and the extra clothing and padding were hot and cumbersome.[2] The first artificial ice surface for indoor hockey opened 5 January 1912 in Vancouver with a series of rinks and leagues operating that year both in the West and East. The introduction of the slap shot in the 1950s created new challenges for the goaltender and changed the game.[2] Up to this point, raising the stick above the shoulder drew a two minute penalty. Players started to heat the stick blade in hot water forming the first curved blade.[2] Whipping the puck rather then shooting it made the puck dip and drop unexpectedly, which was a new challenge for the defence. Although written injury information is not available, it appears that when hockey was played outdoors on the large ice sheets with limited boundary materials, injuries were not a great concern.[2] As the game moved to the enclosed indoor arena, play was more interactive between players and the environment. High flying pucks and sticks resulted in a high risk of above the shoulder injuries.[2]

The first catastrophic injury—the loss of an eye—occurred in 1929.[2] After this, the NHL rules did not permit players to play with only one eye.[2] The same rule was applied in the Canadian Amateur Hockey Association (CAHA) at the end of 1920s, when a player from Ontario, named Trushinski, suffered an injury to one eye and later to his other eye, leaving him legally blind.[4] One eyed players were no longer permitted to play in the CAHA. The list of players who were blinded, ending promising careers, is extensive.[4 28] The incidence of above the shoulder injuries was high not only in the professional leagues but also in the amateur ones.[29] Eyes were permanently damaged, teeth were knocked out, and many players had scarred faces from lacerations.[2] Missing teeth and facial scars were the mark of a hockey player.[2]

The history of helmets and face protectors represents the best example of the evolution of hockey protective equipment.

HELMETS AND FACE PROTECTORS

Helmets

The use of hockey helmets began in Sweden in the middle of the 1950s. An insurance survey in 1961 found over 100 closed head injuries, with one death, 22 mTBIs, and three facial fractures; it also found that the number of serious head injuries in ice hockey was increasing.[3] This survey led to the development of hockey helmets, the use of which became mandatory in Sweden in 1963.[3] All players taking part in matches in Sweden were obliged to wear a helmet that had been tested and certified by the Swedish Hockey Association (fig 2). The first rules were formulated for testing and certifying helmets.[3]

In 1965 the CAHA made helmet wearing mandatory for all non-adults.[2 4] Subsequently the Amateur Hockey Association of the United States (AHAUS) established a new rule requiring players to wear other head protection as well.[2 4] In 1968 Fekete *et al*[30] reported on two teenage ice hockey players in Canada who died after closed head injuries while wearing helmets. This led to a request by the CAHA to form a technical committee for approving helmets. A Canadian Standards Association (CSA) technical committee was charged with this in 1969.[4] In 1975 all CAHA players were required to wear these CSA approved helmets.[4]

Face protectors

The first face protectors developed in 1972 were an excellent start toward ensuring adequate eye protection for hockey players, including goaltenders. Many goaltenders wore moulded plastic face protectors, and other players wore wire grid face protectors.[2] Unfortunately the blade of a hockey stick could still penetrate many of the face protectors and could result in a serious eye injury.[4] In 1973, forwards and defencemen wore no eye protection, although some goaltenders were wearing moulded masks introduced by Jacques Plante. In a retrospective questionnaire study involving 600 Canadian ophthalmologists, 287 eye injuries were found, including 20 blinded eyes.[4] Three quarters of these injuries were stick induced. The consequence of this survey was that in 1975 the CAHA introduced high sticking penalties, which prohibited the carrying of sticks above shoulder level.[4] In 1974 Pashby[4] began a prospective study on this topic. In the season 1974–1975, there were 43 blinded eyes, and the puck caused 38% of these injuries. It was evident that some type of eye protection was necessary. The eye and face protectors available were unsuitable because the hockey stick blade could penetrate them immediately over the eye area. The CSA technical committee was charged with investigating standards for ice hockey visor and face protectors. In 1976 the AHAUS required all amateur players under the age of 20 to wear a full facemask.[4] In 1978, the CAHA required all hockey players playing under their jurisdiction to wear a CSA certified face protector attached to a CSA certified helmet.[4] This rule was not compulsory for senior and junior players. To ensure that only CSA certified protectors were available in Canada, the Canadian government ruled that only certified protectors could be imported into or sold in Canada. At the same time, Dr Paul Vinger of Boston was asking the American authorities to make eye protectors mandatory equipment in US hockey also.[4] In the 1977–1978 season, the National Collegiate Athletic Association (NCAA) required full facemasks to be worn in all college hockey games in the United States.[28] In Sweden, there was a similar development, and since the 1987–1988 playing season it has been mandatory for senior players (born after 1966–1967) to wear visors (fig 3).[2]

Ice hockey eye injuries gained further international prominence in 1978 when the first international ice hockey doctor's

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Figure 3 Player wearing a standardised helmet with a visor.

conference of the International Ice Hockey Federation (IIHF) took place in Prague, Czechoslovakia, under the chairmanship of Dr Tintera during the world ice hockey championships 1978.[4] The European and American doctors were most impressed, and the use of eye protectors for hockey players became common in all hockey playing countries. Wearing of a full facemask was then mandatory for juniors in European countries from 1978.[4] Helmets were not required in the professional leagues until 1981.[28]

Over the years, the Canadian subcommittee on head and face protectors for ice hockey goaltenders have developed a standard for goaltenders, which was completed in 1996.[31]

Figure 4 Bauer hockey helmet HH5000: an example of a standardised helmet (CSA, HECC, and CE certified) with outstanding comfort and look, protection, optimum fitting, and lightweight performance.

Figure 5 An example of a standardised hockey visor (CSA, HECC, and CE certified) with professional look and protection.

Continued helmet, full facemask, and visor improvements have been the result of testing by researchers, collaborative efforts between manufacturers and both hockey and standards organisations, and the use of new materials.

International standards for ice hockey helmets and face protectors

The creation of the International Organization for Standardisation (ISO), whose mandate has been to facilitate the international cooperation and unification of industrial standards, officially began its operations on 23 February 1947.[5] In 1979, at the annual AHAUS meeting in Lake Placid, it became apparent that an ISO for hockey helmets and hockey face protectors was needed.[5] The inaugural meeting of ISO/TC83/SC5 was conducted in March 1987 in Ottawa, Canada, and it developed the first ISO draft standards on hockey helmets and face protectors. Since 1987, the subcommittee SC5 has met annually.[5] As a result of the Treaty of Rome, the EU decided to harmonise the requirements throughout Europe to remove obstacles to the free movement of goods.[32] After the enforcement of the EU directives in 1989, the European Commission and the European Free Trade Association (EFTA) mandated the technical committee (CEN/TC158) of the CEN (European Committee for Standardisation) to prepare European standards. In October 1996, the ISO published the first international standards (ISO 10.256 and 10.257), which addressed head and face protection of ice hockey players.[33] The Committee Européen de Normalisation (CEN) published at the same time a European standard (EN 967), which also addressed the subject of head and face protection for ice hockey players (fig 4).[33]

After the meeting of the SNV (Swiss Association for Standardisation) in October 1996 in Zurich, Switzerland, the members of the two standard association committees, ISO and CEN, realised that they would have to combine their standards in order to develop a single international approved standard.[33] A European committee (CEN TC 158) was founded to develop a single international standard for head and face protection of ice hockey players and was sent to all ISO members (figs 5 and 6).

Today there are four official standards for ice hockey head and face protection (CEN, ISO, CSA, and ASTM) (table 1).

If the committee members of the ISO and CEN approve the proposed single international standard, by the end of 2002 there will be a single international standard for head and face protection (ISO/EN 10256).[33]

RESULTS OF RULES AND PLAY CHANGES

Head injuries

Before the introduction of full facemasks and helmets, head injuries, including face, scalp, and brain injuries, accounted for at least 50% of all serious injuries in ice hockey.[4] Before the use of any type of hockey helmets and before the mandatory use of standardised helmets, there were deaths from head injuries in Sweden, Canada, and United States.[4] However, these data go back to the 1960s. Since 1963 there have been no deaths from head injury reported in Sweden.[3]

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Figure 6 An example of a standardised hockey full facemask (CSA, HECC, and CE certified) with increased protection for the ear and chin.

The problem of head protection in ice hockey is unique because of the demands of the player to wear small and lightweight helmets to be used to protect against high sticks, strikes against the board, ice surface, or glass and collisions, which often result in severe head injuries.

To analyse the effectiveness of hockey helmets, it is necessary to classify head injuries as described by Patrick Bishop. He classified them into two types: focal head injury, in which the trauma is localised to a specific region of the head; diffuse head injury, involving widespread neural disruptions of the brain.[34]

Focal head injuries

Focal head injuries are caused by the force of contact and the head acceleration of direct blunt trauma, such as being struck with a hockey stick or a puck, or falling on the ice surface and striking the head. This type of focal injury generally includes skull fractures and intracranial haematoma (epidural or subdural haematoma, intracranial haemorrhage) (figs 7 and 8). It is estimated that about 50% of hospital admissions for head injury and nearly 66% of head injury deaths are related to focal injury. Wearing an internationally approved standardised helmet can reduce these injuries.[34–36]

Diffuse head injuries

Diffuse head injuries, on the other hand, are caused by the inertial effect of the mechanical blow to the head. These injuries may involve axonal damage from excessive angular acceleration—for example, after an abrupt body check—caused by either direct or indirect inertial loading of the head. They represent a continuous spectrum of the same pathology: a progressive widespread but heterogeneous shearing of axons, throughout the white matter of the cerebral and cerebellar hemispheres up to the brainstem. They are characterised by a more widespread or global disruption of neurological function. The proportion and total number of axons damaged, as well as their anatomical location, determines the severity and reversibility of the clinical syndromes and the neurological deficits. The exact subcellular events and their time course in the injured axons are not 100% clear. The degree of severity of traumatic axonal damage can range from transient disturbance of ionic homoeostasis to swelling and impairment of axoplasmic transport with a secondary (delayed) axotomy, which finally leads to structural irreversible lesions.[37 38]

Sequelae of these injuries can range from recovery for a "concussion" to either coma and/or death. These injuries account for only about 25% of head injury deaths, but they are considered more serious because of permanent neurology disability in survivors. Furthermore they are characterised by a cumulative effect, which may end an athlete's career.[9–11 17 39 40–42]

An mTBI, even called concussion, may represent the mildest form of a diffuse brain injury.[37 38] The persistently high incidence of mTBI, concussions, over the last few years is alarming. In the Canadian Hockey League (CHL) the incidence of mTBIs rose from 4% in the period 1991–1996 to 8.5% in the season 1997–98.[7] The National Collegiate Athletic Association (NCAA) injury surveillance system recorded a comparable development in North America.[8] Similarly in Sweden, according to Tegner and Back,[18] one Swedish ice hockey team showed an increasing incidence of mTBI in the last 15 years from 2% of all injuries in the season 1986–1987 to 18% in the season 2000–2001.[18] John Powell reported a distinct increase in the incidence of minor traumatic brain injury in the professional ice hockey league (NHL), from 2% in the season 1989–1990, to 4.9% in 1995–1996, to 8% each in the last two seasons 1999–2001.[43] mTBI represented one fifth of all head injuries in the Swiss National Ice Hockey leagues A and B during the seasons 1996–1998 (fig 9).[9] According to national and international statistics the proportion of mTBI in ice hockey to the overall number of injuries fluctuates between 2% and 20%.[7–10 14 15 17 19 20 37 40 44 45] However, the diagnosis of mTBI is subject to widespread misunderstanding in definition and severity, which have contributed to a paucity of reliable epidemiological data.

Often the mechanism of a head injury is complex involving a focal and a diffuse component. Direct head contact can induce both rotational acceleration of the head necessary to cause a diffuse injury and the conditions necessary to cause a focal injury. Hockey helmets were originally developed to protect against brain injury death and/or intracranial haematoma and to reduce the risk of serious head injuries precipitated by direct blunt traumas.[2–5 15 17] Helmets provide such protection by means of a firm outershell, intended to distribute the load over a wider area, and by means of an energy absorbing liner system.[34–36] All hockey helmets are evaluated and tested using a collision simulation model that accounts for direct loading of the head by blunt trauma.[32 34–36]

In cooperation with Professor Bishop, we analysed the videotapes of forty professional ice hockey players, who sustained a an mTBI in the NHL. We proposed three different possible mechanisms responsible for causing mTBI in ice hockey: (*a*) a direct eccentric blow to the head; (*b*) a direct blow to the face or jaw, and (*c*) a blow directed to the chin.

(*a*) **A direct blow to the head** may cause many forces to the brain tissue. These forces may be simplified to a force F_t, with translatory components (with transversal and axial forces), and a force F_r, with rotatory components in the frontal, sagittal, and transversal (horizontal) planes (fig 10).

(*b*) **A direct blow to the face or jaw** may cause a force F_t with translatory and a force F_r with rotatory components as well. In ice hockey there is a lot of contact with the jaw and face, a similar mechanism to the knockout punch in boxing, and the result is often significant rotational acceleration of the head and brain. However, there is no protective mechanism that can reduce the effects of this force (fig 11).

(*c*) **A blow directed to the chin** may cause a translatory force F_t, which is transmitted from the chin through the lower jaw, through the temporomandibular joint at the base of the skull, and then to the brain. These forces may also cause wave propagation through the brain and may cause concussion as well (fig 12).

Improvements in the padding materials used for hockey helmets have been made recently.[35 36] Such materials are usually made from urethane foams with a thickness of about 16 mm. Over the past few years, polypropylene, a lighter and more effective energy dissipating material than urethane foam, has been introduced in some helmet models. Although the padding materials in helmets have been improved to absorb more energy, the crucial factor in mTBI appears to be rotational acceleration of the brain.[11 15 17 20 34 36–38 40 44]

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Table 1 Official standards for ice hockey head and face protection (CEN, ISO, CSA, ASTM)

CEN European Standard
- EN 967:96 for "Head protectors for ice hockey players"
(Includes helmets, visors and full facemask for players, goaltenders, and functionaries)

ISO International Standards
- ISO 10256:96 for "Protective helmets for ice hockey players"
(For players, goaltenders, and functionaries)
- ISO 10257:96 for "Face protectors and visor for ice hockey players"
(For players, goaltenders, and functionaries)

Canadian Standards
- CAN/CSA Standard Z262.1-M90 for "Ice hockey helmets"
- CAN/CSA Standard Z262.2-M90 for "Face protectors and visor for ice hockey players"
- CAN/CSA Z262.3-M96 for "Head and face protectors for ice hockey goaltenders"

American Standards
- ASTM Standard F1045-90: "Standard performance specification for ice hockey helmets"
- ASTM Standard F513-89: "Standard performance specification for ice hockey full facemask"
- ASTM Standard F1587-96: "Standard performance specification for helmet and facemask for ice hockey goaltender"
- ASTM Standard F737-86: "Standard performance for skate blades"

The two most critical properties of helmets are impact energy attenuation and load distribution. These properties reduce the magnitude of the forces applied to the head reducing the stress and strain in the skull and brain.[46 47] Through this mechanism, more serious head injury may be prevented and the severity of an mTBI may be reduced.[46] The magnitude of the acceleration required to produce an acute brain injury has been shown to vary with the duration and direction of the acceleration.[47] Results from these experiments led to the publication of tolerance curves and severity indices to distinguish impact with high likelihood of producing injury from those with a low likelihood.[48 49] Two such indices, the Gadd severity index and the HIC score, are currently used to determine the likelihood of a serious brain injury.[48 49] A Gadd score of 1500, a HIC score of 1000, or a peak acceleration of 200g are considered threshold values for a single impact to be likely to cause a severe brain injury. Moreover, engineers and manufacturers have used these indices to better determine the injury potential of impact associated with lower acceleration but a more prolonged duration.[47] Previous studies have shown that repeated impacts at subconcussive threshold levels may have a cumulative deleterious effect both histopathologically and on cognition, even if no single event led to neurological signs or symptoms.[37 47] The critical acceleration threshold that would be likely to lead to cumulative neurological dysfunction is currently not known.

The current standards require a drop test with a pass/fail criterion of 275–300 g headform acceleration. However, current research shows that a minor traumatic brain injury, a concussion, is probable at a head acceleration in the 150–200 g range.[46] Therefore, the assessment criterion may not drive helmet improvement and a greater potential reduction in mTBI.[46] Further examination of the impact characteristic in sports—for example, impact energy, location, and frequency—and their relation to mTBI are necessary.[46 47] Further research with a match video and the use of an internet based database system is an invaluable adjunct.

Naunheim *et al*[47] found that peak acceleration measured at the surface of the head was 160–180% greater from heading a soccer ball than from a non-injurious impact during hockey or American football.[47]

The ability of hockey helmets to reduce the effect of forces delivered through the jaw or face, or to reduce brain rotation, is limited.[34 36 44] If the helmet's padding material is able to reduce the total force acting on the head, then the tangential component causing head rotation will also be reduced.[34–36] However, for blows delivered to the region of the jaw or face, or blows on the chest or back, the hockey helmet is not effective in reducing rotational effects.[34 36 44 45]

Therefore sports organisations should consider alternative strategies to reduce the risk of mTBI. Firstly, they should

Figure 7 A high stick injury to one of the most dangerous areas of the head with the possibility of causing an epidural haematoma.[1]

Figure 8 Acute epidural haematoma after a stick injury to the temporal region of the skull. The bone in this region of the skull is very thin. The 12 year old player died four hours later.[1]

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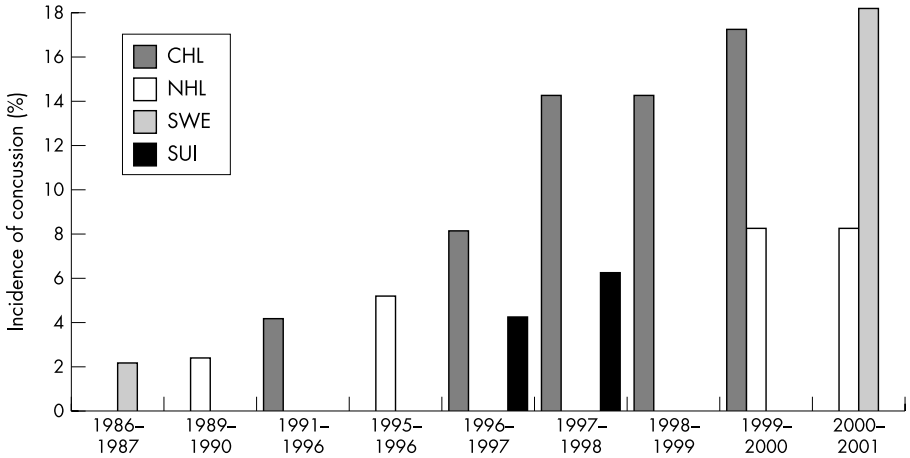

Figure 9 Incidence of concussion (mTBI) from 1986 to 2001 in Sweden, Canada, North America, and Switzerland.[7 9 18 43] CHL, Canadian Hockey League, 1991–200; NHL, National Hockey League, 1989–2001; SWE, Swedish Ice Hockey League, 1986–2001; SUI, Swiss Ice Hockey League, 1996–1998.

eliminate head checking with any part of the body—for instance the elbow or shoulder (fig 13).

For this reason, the rules committee of the International Ice Hockey Federation IIHF proposed in 2001 a new rule to eliminate every "clean" check to the head. This new rule will be mandatory worldwide from July 2002 by the national associations under the regulation of the IIHF (New Rule 540 IIHF: Checking to the Head and Neck Area). This means that every contact to the head with the body, elbow, shoulder, knee, or stick of an opposing player will be penalised more severely (Minor Penalty/Major Penalty plus automatic Game Misconduct or Match Penalty) at the discretion of the referee in the same progression of the penalty as laid out in the checking from behind rule (Rule 523 IIHF).[50]

Secondly, sports organisations have to make mandatory the use of an internal mouthguard, which may not only prevent dental and orofacial injuries, but also play a role in the reduction of incidence and severity of mTBI.[51]

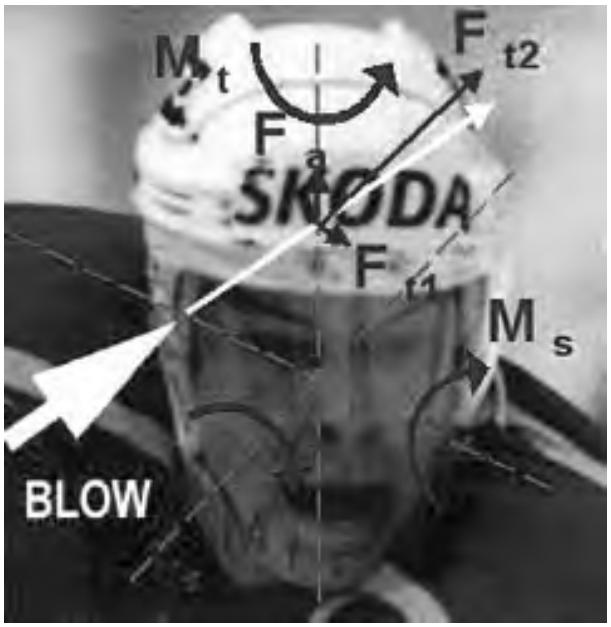

Figure 10 Direct eccentric blow to the head. Protective headgear, with its padding, helps to reduce the effect of the final force F and therefore may reduce the risk of a focal head injury. M_t, moving force in the transversal plane; M_f, moving force in the frontal plane; F_t, force in the transversal plane; F_{t2}, force 2 in the transversal plane; F_a, force in the axial plane.

Eye injuries

Since the introduction of the mandatory use of a full facemask in 1978, the pattern of injury has changed. Most ice hockey injuries before the use of a full facemask were found above the shoulder, most of them being lacerations.[2] The reduction in ocular and facial trauma has certainly been impressive. The consequences of these changes show that blinding injuries are virtually non-existent when approved full facemasks are used.[4 28] Soft tissue injuries around the face and mouth, primarily lacerations and contusions, also decreased significantly from 38% of players to about 9% a year from 1975 to 1988.[2] It has also been reported by Pashby,[28] that properly fitted visors are as effective as full facemasks in the prevention of severe ocular injury, and they also decreased laceration by about 58%.[28] However, the problem of eye injury persists. Eye injuries are still being documented in all players, primarily older ones, but always those not wearing eye protection. From 1972 to 2002, the Canadian Ophthalmological Society (COS) registered 1914 eye injuries in ice hockey, with 311 cases of blindness (fig 14).[28]

Figure 11 Direct blow to the face or jaw. No protective equipment, such as helmets and mouthguards, is effective in reducing rotatory components. M_t, moving force in the transversal plane; M_f, moving force in the frontal plane; F_t, force in the transversal plane.

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Figure 12 Blow directed to the chin. A mouthguard may have the ability to absorb the impact loading from the chin and to distribute the remaining energy, through the resilience of the material combined with its design, over a much larger surface area, thereby reducing the final force on the brain. F_t, force in the transversal plane.

The major decrease in the number of eye injuries was first noticed after the introduction of the new rule in 1975.[52] However, soon after the mandatory use of certified full facemasks for juniors in 1978, the number of blinding injuries started to increase. This may be because these injuries occurred in older players who were not wearing face protection. None of the blinding injuries were suffered by players wearing a certified full facemask.[28] Over the last few years, nine players (eight in Canada and one in Sweden) have received blinding injuries when wearing a visor.[28] Tegner and Pashby analysed these injuries using video recordings. All these players were not wearing the visor correctly; these injuries would not have occurred if they had been (fig 15).[28]

The controversy over the use of a full facemask and a visor was first analysed by Clayton in a study of the Quebec Major Junior Hockey League during the seasons 1991–1992.[53] This study compared the number of neck, head, and face injuries incurred while wearing a full facemask versus visor. The results showed that the eye injury rate was triple that when a visor was worn instead of a full facemask.[53] On the other hand, head injury rate fell dramatically when this change was implemented, and neck injury rate as well as face and jaw injury rates were halved (fig 16).[53]

In contrast with these findings were the results of Benson *et al*,[54] who conducted a prospective study during the 1997–1998 Canadian Inter-University Athletics Union (CIAU) hockey season and compared the risk of sustaining a head or neck injury by the wearing of full facemasks or visors.[54] The Ontario Universities Athletic Association required that all athletes under its jurisdiction wore full facemasks for every practice and game. In contrast, the Canada West Universities Athletic Association required all athletes to wear, as a minimum, visors, for every practice and game. The results of this study showed that the risk of sustaining a facial laceration was 2.3 times greater for players wearing a visor than for those wearing a full facemask. Mostly lips and eyebrows were lacerated. These findings may be explained by incorrecting wearing of the visors and helmets. In fact, too many players wear visors incorrectly, in order to obtain a clearer view, and wear helmets with a loosened chinstrap. These two possible risk factors of wearing the protective equipment incorrectly were not analysed by Benson *et al*.[54] In a prospective study of the Swiss Ice Hockey Leagues A and B 1996–1998, we reviewed the causes of head and face injuries and we analysed the correctness of wearing visors and helmets.[9 55] Similarly to other studies, we found that lacerations, at 55%, were the most common head and face injuries,[9 55–65] followed by fractures (23%), mTBI (17%), and eye injuries (8%).[9 55] Facial lacerations were recorded mainly in the forehead region.[9 55 56 59] In three quarters of the cases, the players were not wearing a visor or a full facemask at all, and in the remainder the players were wearing the visors incorrectly. All eye injuries occurred in players not wearing a visor or full facemask at all.[9 55] None of these injuries would have happened if the players had been wearing at least visors, and if the visors and helmets had been worn correctly.[55]

Benson *et al*[53] further reported that stick contact with the face was the predominant mechanism of the lacerations. The incidence of chin lacerations was higher among players wearing a full facemask than those wearing a visor.[53] Contact with an opponent or stick by body checks or collisions caused the majority of lacerations in athletes wearing a full facemask.[53] These findings may also be explained by the incorrect wearing of helmets with loosened chinstraps.[55] In fact, when a force is directed to the face, the helmet can ride back on the forehead allowing the stick to slide under the visor or full facemask and lacerate the eyes or face, even if the equipment is worn correctly (fig 17).[55]

The rules of the IIHF allow wearing of helmets only with a properly fastened chinstrap.[52] Incorrect fitting or misuse of helmets with single straps could result in increased movement of the helmet during collisions.[56] LaPrade and Broxterman[56] asserted that, even when properly worn, the single strap helmet fixation system does not secure the helmet efficiently to the head. Pashby[28] came to the same conclusions. They both recommended the use of a double chinstrap to prevent the helmet from riding back on the head during a collision. Unfortunately, double chinstraps are not so acceptable to the players, and even the ice hockey organisations do not mandate their use. Further studies are necessary.

There is a tendency in modern ice hockey for a more aggressive playing style. The players today are bigger, faster, and stronger, which increases the energy involved in collisions thereby increasing the likely rate of injury. We showed in a prospective study of the Swiss Ice Hockey leagues A and B a direct correlation between players violating the rules and head and facial injuries. Head and facial injuries were caused by illegal activity in most cases.[9] Benson *et al*[53] found that over 40% of facial injuries in players wearing a visor were caused by an illegal action. Stick contact with the face was also a predominant mechanism. Pettersson and Lorentzon[57] found that 57% of facial lacerations were caused by an opponent's stick and that most of these stick injuries were caused by illegal play. Many other studies confirm these observations.[1 9 10 15 18 25 26 45 55 58–60 64 66 67]

In contrast with the opinion of referees, the reporting doctors considered that most head and facial injuries were caused

Figure 13 A head check against the glass can generate a sudden acceleration-deceleration of the brain, which can result in a mTBI.[35–38 44]

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Figure 15 Incorrect way to wear a visor. The loosened chinstrap allows the helmet to ride back on the head when a force is directed to the frontal region and allows the stick to slide under the facemask and lacerate the eye or forehead region.

by illegal activities. It is alarming that the referees in most of these instances of potentially serious face and head injuries did not call a penalty. Our prospective study on the Swiss Ice Hockey leagues A and B showed that 56% of head injuries and 46% of facial injuries were caused by illegal activity with the stick as judged by the reporting doctors.[9] Of these illegal activities, only 29% were called a penalty by the referee.[9] There are many studies world wide showing that most of these injuries occurred during on-ice events that were not penalised at all.[26 54 55 63 64 67] Besides stricter enforcement of the rules, changes in the attitudes of hockey officials are needed. When rules are not enforced regularly, they are seldom obeyed, and the formal control system—that is, the referee—becomes merely symbolic.[68]

The increased protection of the face through the compulsory wearing of helmets with full face protection, which was introduced in 1975, appears to have led to a more aggressive playing style perhaps because it is believed that the head, face, and throat are now at less risk. Many believe that, after the mandatory use of helmets with a full facemask, players developed a false sense of security and invincibility leading to excessive risk taking behaviour with a resultant increase in illegal and injurious activity.[2 9 15 17 22 23 26 35 40 44 45 63 69 70] It is also interesting that increased high stick violation and the use of the full facemask as a weapon were noted in 1980 by Reynen and Clancy[26] after mandatory use of the full facemask. In 1988, the American College Hockey Coaches Association meeting came to an anonymous agreement that full facemasks promoted rough and violent play.[70] They called for approval of a visor type of mask to decrease the violent play. In 1988 and 1992, the US Olympic team used a visor for over nine months, playing a 60 and 80 game schedule against college and NHL teams, and they documented no eye injuries.[70] Other authors have even speculated that the mandatory use of helmets with full facemasks has increased the risk of neck injuries and mTBI.[54 62 67 68] Benson *et al*[54] found no statistically significant risk differences for neck injuries and mTBI for players wearing visors versus full facemasks. Stuart *et al*[67] recently investigated, in a prospective cohort observational study of the United States Hockey League Junior A, the relation between injuries and type of facial protection, and determined whether full or partial facial protection was associated with an increased incidence of mTBI, eye injuries, and neck injuries. This is the only epidemiological ice hockey study to accurately control for individual exposure time and examine vulnerability to injury based solely on the facial protection afforded. Junior A hockey players were between 16 and 21 years of age. Full facemasks were mandatory for all players younger than 18, and players 18 years and older were free to choose no facial protection, visor, or full facemask. No significant differences in the risk of neck injuries or mTBI based on the type of facial protection worn were found. It was also found that the risk of an eye injury was 4.7 times greater for players wearing no

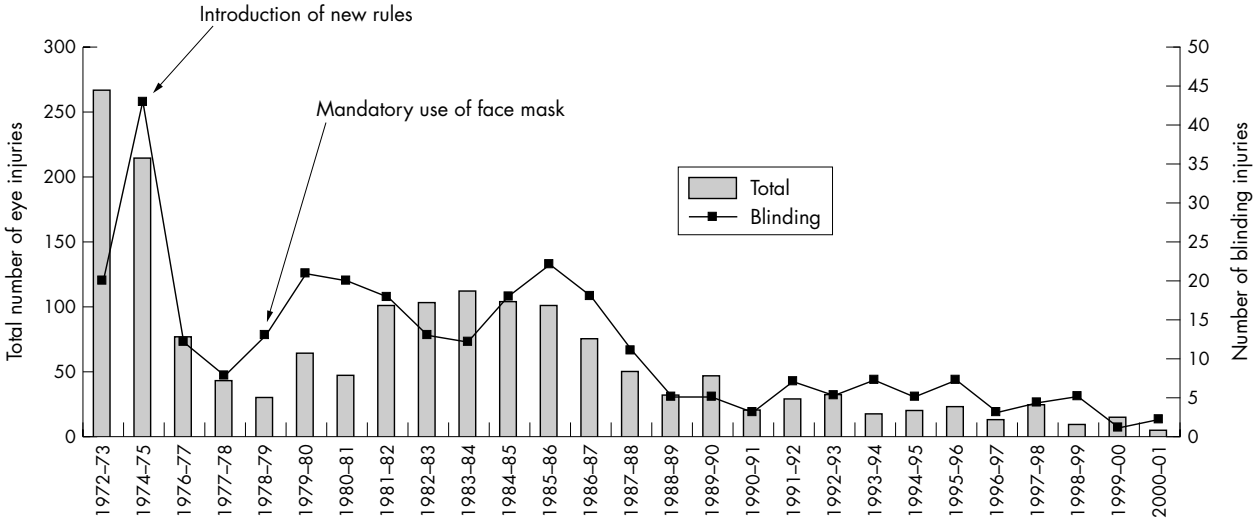

Figure 14 Canadian Ophthalmological Society survey of eye injuries from 1972 to 2002. The introduction of high sticking and hooking penalties in 1975 and the mandatory use of certified full facemasks in 1978 is indicated.[4 28]

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Figure 17 The visor is correctly fixed to the helmet, but the helmet is attached with a loosened chinstrap. This allows the stick or puck to slide directly under the visor. There is also no longer any padding effect of the helmet, which may help to reduce the forces to brain and maybe the risk and severity of an mTBI.[55] The same mechanism can occur if a full facemask is worn with a loosened chinstrap.

facial protection than for those wearing partial protection. No eye injuries occurred in players wearing full protection.

As a full facemask may tend to encourage aggressive play and a feeling of invincibility in young players, and as senior and professional players never play with full facemasks, compromises have to be found. For these reasons, we have proposed the use of helmets with visors, as approved by the standard organisations, and enforcement of the correct way to wear them. It may therefore be expected that the number of severe head injuries, primarily the avoidable eye injuries and the periorbital skin lacerations, will be reduced through the correct use of helmets and visor. One cannot expect all head and face injuries to cease. A further decrease in all kinds of severe head and face injuries can only be achieved by better education of the players and strict enforcement of the rules. It would appear that the penalties that most leagues administer for high sticks and slashing, which make contact with the opponent, have not been enough to deter violent stick work. Many authors recommend that a high sticking or violent slashing penalty should be the same as for checking from behind: removal from the present game and suspension from the next game.[68] This is a critical point, because, if the ice hockey organisations make rules that are too strong, the referees will not enforce them. We therefore need to devise rules that referees are willing to endorse, otherwise they will not enhance the safety of the players.

Spinal injuries

After head injuries, neck injuries are considered the most catastrophic. American football has been considered the most dangerous sport for causing such spinal injuries. Approximately 819 deaths were directly caused by football from 1931 to 1986, with a peak incidence of 3.4 deaths per 100 000 players in 1968.[71] The National Football Head and Neck Injury Registry System was then developed to register and review these data.[71] American football is a typical example of a sport, in which the consequences of the introduction of new rules and their strict endorsement had dramatic effects on the incidence of spinal cord injuries. After the introduction of new rules in 1976—that is, prohibition of head impact in blocking, tackling, and head butting—and helmet standards, the incidence of death fell to approximately 0.5 per 100 000 players in 1978, and the number of spinal cord injuries rapidly decreased as well.[71] Rule changes have played an important role in reducing fatalities. Using Torg's data from 1976 to 1987 and Mueller's data from 1988 to 1993, a summary of the incidence of cervical quadriplegia in high school and college football is shown in fig 18.[71 72]

There is no doubt that this 1976 rule change has made a major contribution to the reduction in deaths from head and cervical spine injuries. Data collection has also played an important role in the prevention of football injuries. The American Football Coaches Association has encouraged the tracking of football fatalities annually since 1931. There is no question that the beneficial changes are the result of reliable data collection and the publication of the results in the athletic and medical literature. Persistent surveillance of sports injury data is vital if progress is to continue in the prevention of fatalities. To help offset the trend of increasing head and neck injuries and fatalities in American football, the National Operating Committee for Safety in Athletic Equipment (NOCSAF) was founded in 1969 to establish safety standards for athletic equipment. In football also, initial efforts were directed to head protection. A safety standard for football helmets was achieved in 1973, and the first helmets were tested on the NOCSAE Standard in 1974. It was then made mandatory for all students in college (in 1978) and high school (in 1980) to wear NOCSAE certified helmets.[22] The education of coaches and correct tackling techniques being taught by coaches have also played an important role in decreasing the number of head and cervical spine fatalities.[22] Improved protective equipment has also been purchased and more time

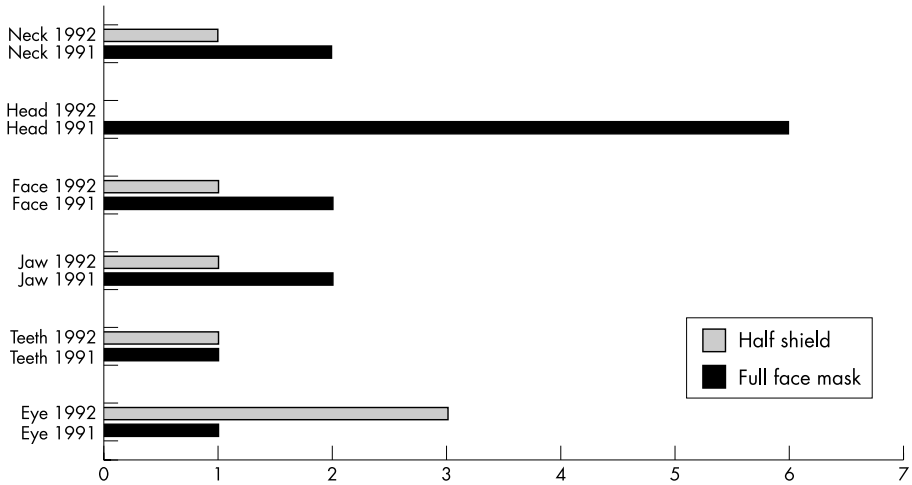

Figure 16 Relation of head and facial injuries by the wearing full facemasks (season 1991) or visors (season 1992) in the Quebec Major Junior Hockey League.[53]

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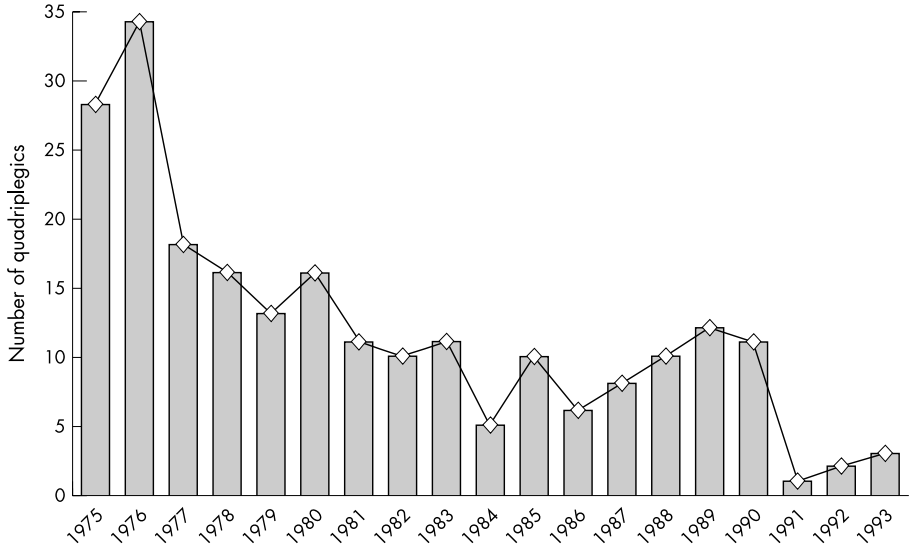

Figure 18 Implementation of American football rule changes in 1976 prohibiting the use of the head as the initial point of contact resulted in a steady decline in quadriplegia at the high school and college level.[71 72]

spent in fitting the equipment. Helmet manufacturers have also stressed the importance of a proper fit to help to reduce head injuries.[22] Because of the large number of participants in American football, this sport still has the greatest number of catastrophic injuries a year.

However, football is not the only sport that causes spinal injuries. Charles Tator and SportSmart Canada began in the early 1980s to draw attention to cervical spine injuries in ice hockey. In the English literature, there were no cervical spinal cord injuries attributed to ice hockey reported.[22–25 71 73] A review of the records of two major hospitals in Toronto found no ice hockey spinal injuries between the years 1948 and 1973. Between 1974 and 1981, six cases were seen at these hospitals. In 1981 this led to the formation of a committee on the prevention of spinal cord injuries in ice hockey.[22–25] Questionnaires were sent to specialists throughout Canada, including neurosurgeons, orthopaedic surgeons, and physical medicine and rehabilitation specialists.[22–25] Major spinal injuries (injury with neurological deficits due to spinal injury and any fracture or dislocation of the spine with or without permanent neurological deficits) were rare in ice hockey in the 1960s and 1970s.[22–25 73] The SportSmart data showed that there were 286 spinal injuries from 1982 to 1996, including many cases from the United States and Europe, but beginning in 1980, the annual incidence increased considerably. From 1981 to 1996 there was an average of 17 cases a year, with a maximum of 26 injuries in 1995.[22–25 73] The incidence of severe spinal injury with paralysis in 1994 was about three times greater in Canadian hockey than in American football.[71] Between 1994 and 1996, there were 53 spinal injuries.

At the beginning of this survey, the players were injured during illegal play, in particular pushing or checking from behind (59.8% in 1966–1987).[22–73] As a result of the data made available by this survey, the CAHA introduced in 1985 specific rules against pushing or checking from behind.[73] These rules were then also introduced, through the support of Dr Pietro Segantini, a past member of the rules committee of the International Ice Hockey Federation (IIHF), in 1994 into the IIHF rules books (Rule 606 "Charging and Checking from behind").[74] A few years later, an international network of doctors realised that it was necessary to register all known spinal injuries in the world. An International Committee of Spinal Injuries in Ice Hockey was then formed in 1997 under the chairmanship of Professor Tator with members from Sweden, Finland, Switzerland, United States, and Canada. During the Eighth International Symposium of the IIHF on Medicine and Science in Ice Hockey in May 1998 at the University Hospital Zurich-Balgrist, Switzerland, an endorsement of the IIHF was obtained in order to secure doctor contacts in each of its 56 member nations.[23]

Charles Tator presented the recent results of the SportSmart registry during the 2001 meeting of NHL doctors in Toronto. For the years 1966 to 1999, the SportSmart registered world wide 401 major spinal injuries (fig 19).[25] Eight players were known to have died as a result of their injuries[25]

More than 300 major spinal injuries were adequately documented.[22–25] The average age of the players was 18 years (46% were 16–20 years). A spinal cord injury was documented in 53% of the cases, and there were 22% spinal injuries with quadriplegia. This recent data suggest that preventive strategies and the introduction of a specific rule against hitting from behind have been effective in decreasing the number of severe spinal injuries.[74] Checking from behind now causes "only" 25% of the spinal injuries.[25]

Several causative factors have been discussed, such as increased aggressiveness and willingness to take risks, a feeling of invincibility, and the lack of awareness of the possibility of a spinal cord injury in ice hockey.[73] Many coaches have also not instructed their players about the risk of a spinal injury and the methods of protecting themselves.[24]

Biomechanical research by Bishop *et al*[75 76] showed that the greatest axial forces were applied to the cervical spine in slight flexion. This position of injuries was consistent with actual injury situations in hockey and football. Approximately two thirds of players with cervical spine injuries wore full facemasks and all wore helmets.[22–25] Further research by Bishop and Wells[77] found that the padding in the helmet adequately decelerated the head on impact; however, the neck itself had to decelerate the torso. They found that impact velocities as low as 1.8 m/s provided compressive loads from C3 through C5 to 75% of their failure loads in axial compression.[77] Sim and Chao[78] have shown skating speeds of 9.4–12.2 m/s and the speed of a sliding skater on ice is approximately 6.7 m/s.[78] Both situations are certainly well above the 75% loads applied by Bishop and Wells.[77 78] Another investigation by Bishop *et al*[76] dealt with the mechanical effect of the helmet and full facemask. There were speculations that the full facemask and helmet may shift the centre of the head mask anteriorly enough to result in flexion of the head on impact. Their studies showed that there did indeed seem to be a slight increase in the centre of the mask anteriorly and increased inertia of the head form, but it was believed to be quite small and thought not to contribute to injury.[76]

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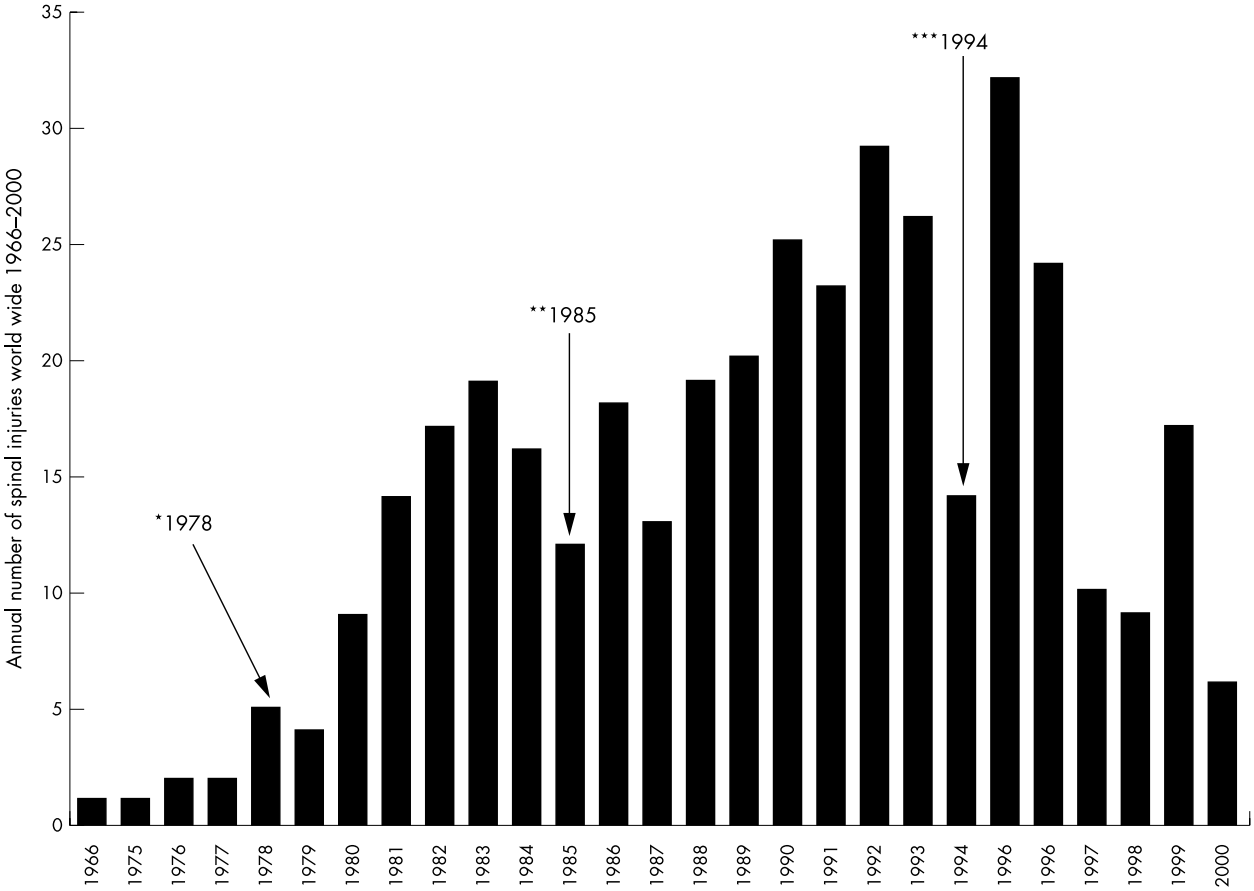

Figure 19 Total number of major spinal injuries world wide from 1966 to 1999 in ice hockey. *1978, Mandatory wearing of full facemask by junior players; **1985, new checking rules CAHA; ***1994, new checking from behind rules, IIHF.[22–25 74]

Dental injuries and use of a mouthguard

This review of the epidemiology of orofacial sports related injuries presents and assesses only scientific databases within the past 10 years. To our knowledge, the Eighth International Symposium of the IIHF on Medicine and Science in Ice Hockey in May 1998 was the first to deal with dental problems in ice hockey.

Piccininni[79] suggested categorising dental injuries in ice hockey in the following way: A, soft tissue injuries; B, dental injuries; C, bony injuries; D, other injuries.

(A) Soft tissue injuries: these would include contusions or lacerations to the lips, tongue, or gingiva, as well as dislocations or trauma to the temporomandibular joint, bruising to the facial muscles, nerves, fascia, and blood vessels.

(B) Dental injuries: chips or minor fractures in the outer edentulous layer, moderate fractures that involve the underlying dentin layer, and severe fractures that result in exposure of the pulp or nerve of the tooth. This group include situations where a tooth is luxated (moved out of its normal position), intruded (repositioned inward into the alveolus), or avulsed (knocked completely out of the socket). A tooth concussion, where the tooth is traumatised but not moved out of position, can also be a serious injury. Trauma to the periodontal ligaments supporting the tooth in the alveolar socket can be placed in this category.

(C) Bony injuries: minor fractures to the alveolar bone surrounding a particular tooth or group of teeth as well as fractures to the facial bones. These are especially important in the maxilla or mandible, including fractures in the ramus or condyle of the mandible.

(D) Other injuries: these may include aspiration of teeth or other dental prostheses, and cerebral concussion resulting from an upward blow to the jaw that caused the teeth to meet violently or the condyle of the mandible to be driven into the fossa of the temporal bone.

To date, only a few epidemiological studies on dental injuries in ice hockey have been published. In a six year study in Finland, Sane[80] reported, in 1988, 11.5% of all ice hockey injuries as maxillofacial and dental injuries, which represented up to 38% of the costs of all injuries. Morrow *et al*[81] conducted in 1991 a survey in 626 high schools with college and university athletes and noticed that the highest rates of orofacial injury were in basketball (10%) followed by ice hockey in 9.8%. Of the traditional sports, 64.1% of the injuries to the teeth occurred during participation in basketball, baseball, and ice hockey. Kujaya[82] suggested in 1995 that dental injuries comprised over 32% of all ice hockey injuries in a four year study of Finnish sports, and a survey in 2000 reported that the sports receiving the most orofacial injuries were baseball and biking, followed by ice hockey and basketball.[83] Piccininni[79] showed that most dental injuries were caused by the stick (75%).

However, most of the literature on dental injuries and mouth protection comes from contact sports such as American football, rugby, baseball, and field hockey. Very early studies indicated that mouthguards may help to decrease the number of mouth injuries in competitive sport such as American football. Stenger *et al*[84] reported that initially dental and mouth injuries were almost 50% of all football injuries, and that with the introduction of full facemasks this number was reduced to almost half, and with the addition of mouthguards the number of dental injuries decreased to 1.4%. Stenger *et al* also claimed that mouthguards reduced mTBI and neck injuries during a five year study of Notre Dame University football

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team. They showed that, with a mouthguard in place, there was an altered mandibular position on lateral skull radiographs so that the condyles were distracted from their fossae. They postulated that forces from mandibular impact applied to the head and neck would be attenuated resulting in fewer injuries. Their views are, however, speculative.

In 1967, Hickey *et al*[85] determined the effect of mouth protectors on pressure changes and bone deformation within the skull in a cadaver model. Using an intracranial pressure transducer, they showed that mouthguards reduced both intracranial pressure and bone deformation by about 50% after a blow to the chin.

Chapman[86] described two basic possible impact directions on the mandible that generate distinct acceleration forces on the skull: a blow on the inferior and anterior aspect of the mandible. A blow received on the inferior aspect of the mandible is transmitted through the midfacial skeleton, forcing the skull to rotate backwards about an axis passing roughly through the mastoid processes. He proposed that a mouthguard could act by absorbing some of this impact, when the mandibular teeth close on its occlusal surface. Therefore the rotation effect of the impact on the skull would be considerable lessened. In the case of an impact delivered to the anterior aspect of the mandible, the head and neck would be displaced posteriorly along the line of the force causing linear acceleration of the brain. He showed, on radiographs of the skull, that a bimaxillary mouthguard can separate the elements of the temporomandibular joint and therefore reduce transmission to the base of the skull from the impact on the chin.

Porter and O'Brien[87] suggested a third potential effect of the bimaxillary mouthguard when the force is directed to the lateral side of the mandible causing the head to rotate about an axis passing vertically through the odontoid process. They suggested that the resilience of the material from which the bimaxillary mouthguard is made, combined with its design, could absorb some of this force by deformation of the mouthguard. They also suggested that the ability of the mouthguard to attenuate any impact depends on the absorption of some of the energy by the material at the impact side and that the remaining energy is distributed throughout the mouthguard over a much larger surface area. All this is, however, speculative.

However, already in the early 1970s, when further studies confirmed the earlier findings on the high incidence of injury to the oral region, the AHAUS began to promote and mandate the use of either external or internal mouthguards for younger players.[44 79] This mouthguard mandate was adopted by a number of leagues, including the NCAA, but there appeared to be wide variation in both enforcement and type of guard chosen. Sweden has also required the mandatory use of extraoral teeth protectors for junior and boy's team since 1970. Since the compulsory use of face protectors for junior and boy's team from 1978, extraoral teeth protectors are no longer required.

Enforcement of the mouthguard rule in US College hockey was found to be difficult. The lack of knowledge about mouthguards by players, coaches, and team trainers was largely responsible for this. Players believed that uncomfortable, ill fitting protective equipment seriously compromised their ability to perform. Commercial mouthguards were not only uncomfortable because of poor fit, but there was also evidence that they interfered with normal ventilation. Athletes often did not appreciate the importance of mouthguards, and less than 50% believed that mouthguards prevent injuries.[44] Coaches were also not totally supportive of mouthguard use, as, in a survey of college football coaches, only 74% would tell players about a violation.[44]

Athletic mouthguards were first designed to protect the lips and intraoral tissues from bruising and laceration, to protect the teeth from crown fractures, root fractures, luxations, and avulsions, to protect the jaw from fractures, and dislocations, and to provide support for edentulous space (fig 20).[51 88]

Figure 20 Dental trauma of a junior ice hockey player wearing no face protection and no mouthguard: the opposing player directed an apparently intentional blow with the result of fractures and loss of many teeth.[51]

Although there are no official mouthguard standards, several organisations—for example, ASTM, NCAA, the American Dental Association (ADA), and AHAUS—have offered information based on empirical evidence.[88] The ASTM (ASTM F697–80) and the CEN (CEN/TC162/WG11 N148) have developed working groups for mouthguard standards to further improve them.

Because many sports related traumatic dental injuries are preventable, the risk to benefit ratio can be increased by the use of appropriate properly fitted protective athletic equipment. At present, mouthguards are recommended in some sports (amateur boxing, football, ice hockey, men's lacrosse, women's field hockey) to reduce both the likelihood and severity of such injuries.[88] The only professional sport that requires a mouthguard to be worn is boxing. Many enlightened amateur and professional athletes voluntarily wear mouthguards for protection against injuries. More athletes need to recognise the importance of mouthguards for their own personal safety, and coaches, team doctors, and athletic trainers need to be aware of the benefits of mouthguards for the players under their guidance.[88]

Castaldi,[44] reviewing the history of mouthguards from the early 1930s when they were first used by professional boxers, described how mouthguards protect against injury to teeth, broken jaws, and mTBI. He suggested that custom made mouthguards may not only protect against injuries to the anterior teeth, but may also cushion the force of impact, thus offering protection against jaw fracture and mTBI. Wearing a custom made mouthguard over the lower teeth creates a small gap at the temporomandibular joint and reduces the forces transmitted to the brain from a blow to the jaw or face (fig 21).

However, evidence of injury protection by the use of mouthguards is mostly based on case series and retrospective injury surveys. McCrory[89] stated that the ability of mouthguards to protect against head and spinal injuries in sports falls into the realms of "neuromythology" rather than hard science. He even suggested that there is no convincing evidence to support a protective effect against any type of sporting injuries, because studies with sufficient power have not yet been performed.

Piccininni[51] suggested that mouthguards alone or in conjunction with additional face protection have been shown to be an effective means of preventing or reducing dental and orofacial injuries. Numerous anecdotal reports have also indicated possible correlation between mouthguards and mTBI. Piccininni focused on two primary mechanisms to explain this correlation. The first was dissipation of forces delivered to the maxilla, skull, and temporomandibular joint complex when the mandible received a blow. The second was stabilisation of

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Figure 21 The force of a blow is transmitted from the chin (A) through the lower jaw (B), through the temporomandibular joint at the base of the skull (C), and then to the brain, causing mTBI (D). The thickness gauge reading reveals that the skull in the temple area (E) can be as little as 2–3 mm thick. Similar types of injury occur in the upper jaw but less commonly.[44]

the skull through increased neck muscle activity when clenching, which may be enhanced with the presence of a mouthguard (fig 22).

Piccininni stated that, for this reason, mouthguards should be promoted as effective devices for prevention of dental and orofacial injuries and that properly designed mouthguards may also play a role in the reduction of incidence or severity of mTBI Barth. *et al*[90] came to the same conclusions. He suggested that the use of properly fitted mouthguards may increase the time and distance involved in acceleration when the mechanism of injury is an upward blow to the head through the mandible.

We think that the minimal cost of properly fitted mouthguards would reap numerous benefits in terms of reducing medical, financial, cognitive, psychological, and social consequences of dental injuries and mTBI. For this reason, the rules committee of the IIHF has proposed that all players participating in the under 20-year category and not wearing a full facemask must wear a custom made mouthguard.[50] This new rule will be mandatory under the IIHF regulations from July 2002 (New Rule 227 IIHF: Mouthguard). This is the first step in this regard. It is important that this mandate also applies to players wearing a full facemask, because a full facemask cannot reduce the forces transmitted to the brain from a blow to the jaw or chin (fig 23).

This hypothesis seems tenable because properly fitted mouthguards produce a separation between the head of the condyle and the base of the skull. This space supplies the increased distance necessary to reduce acceleration, reducing the force of impact during a blow and thus reducing the impact of those forces on the brain and reducing trauma.[90]

Piccininni further reviewed the types of mouthguard currently being used, which has been proposed by Heintz and completed by Dorney. Today mouthguards can be classified into four types[79 91].

- **Type I, stock mouthguards:** this type offers the least protection and is not acceptable to most athletes (fig 24).
- **Type II, mouth formed mouthguards:** this type comes in two varieties: shell lined and boil and bite. The latter types come in a wide variety of styles and colours with or without strap attachments. Most of these types are too balky at the periphery or too thin over the critical occlusal table, and are often uncomfortable. They lose up to 99% of their thickness when fitted (fig 25).
- **Type III, custom fabricated mouthguard:** this type is made professionally over the dental cast of the athlete's maxillary arch. They are fabricated from a plaster model of teeth using a single layer of material vacuum moulded by a dental office or commercial laboratory. This type has an inconsistent and often minimal thickness across the occlusal surface because of the pressures of the vacuum forming machine. Although this type can be made with or without a strap attachment, a strap may be contraindicated. This type has been popular for several decades. Ethylene vinyl acetate (EVA) material is available in single or double laminated sheets. One product line includes an incisal brace between the two EVA layers. The incisal brace is purported to prevent lingual displacement of the incisors from a traumatic force directed labiolingually (fig 26).
- **Type IV, heat and pressure laminated mouthguards:** this type has gained popularity in Europe and Australia. The material is better adapted for a more precise fit and retains structural integrity for longer periods of time, compared with all other mouthguards and fabrication techniques. The heat/pressure laminating machine offers the added capability of fusing multiple layers of EVA material to the desired thickness. These multilaminated guards require both upper and lower plaster models to be taken of the athlete. The predicability of material thickness in the critical area is believed to enhance protection and allows articulation of the arches to enhance mandibular stability during impact (fig 27).

These mouthguards can be customised to suite the requirements of different sports. At the moment there are four types of heat and pressure laminated mouthguards[91].

Figure 22 A mouthguard can dissipate forces from blows to the inferior and anterior aspect of the mandible.

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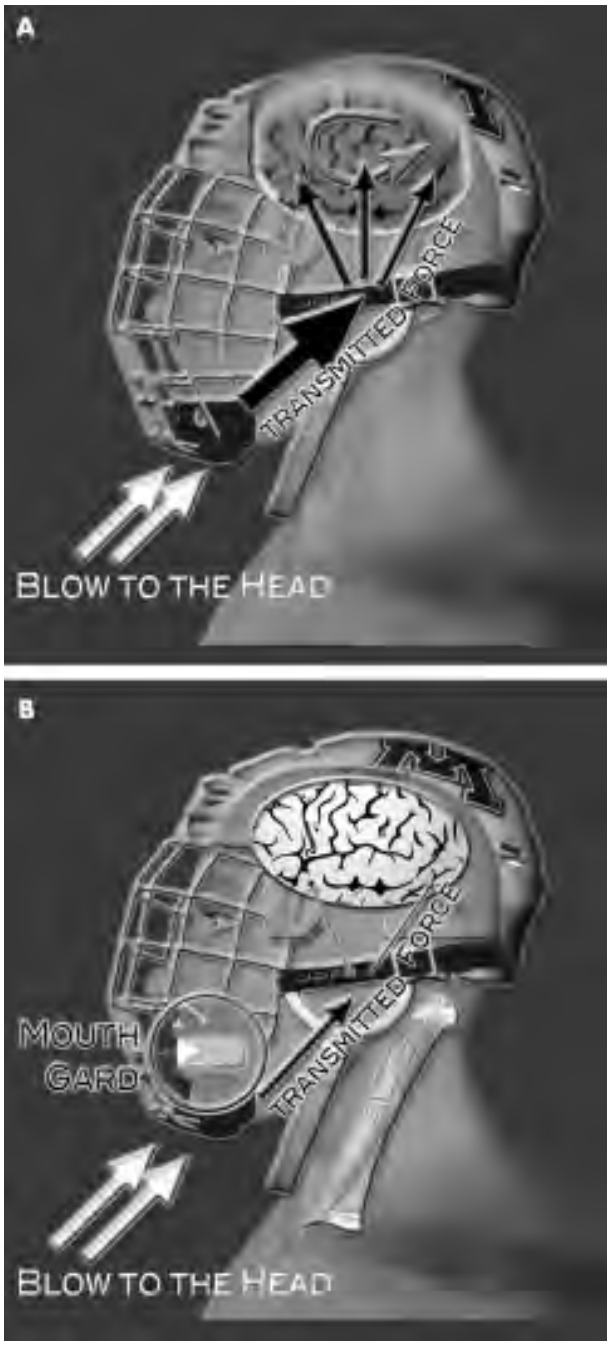

Figure 23 (A) A blow to the head (yellow arrows), such as might be received on the frontal part of a full facemask, may cause mTBI by delivering all the force (large red arrow) from the facemask to the chin, through the lower jaw, through the temporomandibular joint, and then to the brain (three small red arrows) causing shock wave propagation. (B) A custom made mouthguard may absorb all forces delivered to the full facemask (yellow arrows), through the chin, through the lower jaw, through the temporomandibular joint, and then to the brain (small red arrows). A mouthguard may also stabilise the skull through increased activity of the neck muscles induced by clenching to keep the mouthguard in place. Both mechanisms may reduce the risk and severity not only of dental and orofacial injuries, but also of mTBI.

- Type 1, light: with two layers of laminated EVA material with a thickness of 3 mm. This type is designed for wrestling, volleyball, mountain biking, and motocross.
- Type 2, medium: with two layers of laminated EVA material with a thickness of 5 mm. This type is designed for soccer, rugby, basketball, softball, rollerblading, skating, and skateboarding.
- Type 3, heavy: with two layers of laminated EVA material with three unique power dispersion bands and 5 mm thickness. This type is specially designed for baseball, football, racquetball, martial arts, and boxing.
- Type 4, heavy pro: with three layers of laminated EVA material (one hardened layer) with a 5 mm thickness. This type is specially designed for ice hockey, field hockey, street hockey, and kickboxing.

Figure 24 Stock mouthguards.[79 91]

Figure 25 Mouth formed mouthguards.[79 91]

There is a need to determine scientifically which mouthguard material is best, what material thickness is most effective for injury prevention, and which fabrication technique produces the best results for an individual athlete.

CONCLUSIONS

The incidence and severity of ice hockey injuries appear to be increasing. Also the costs of treatment of sports related injuries are increasing. The incidence of head and facial injuries is still too high. Even now such injuries are the most common in many countries. It has been shown statistically that there is a correlation between violation of the rules and head and face injuries. A less significant correlation has been registered between facial injuries and accidental events. It is certain that careless performance combined with illegal use of the stick, lack of protective equipment, and the incorrect wearing of helmets, full facemasks, and visors play a decisive role.

Following our analysis, Philippe Lacarrière, Chairman of the IIHF Rules Committee, endorsed in 1998 new rules that make it compulsory to wear helmets and full facemasks (including

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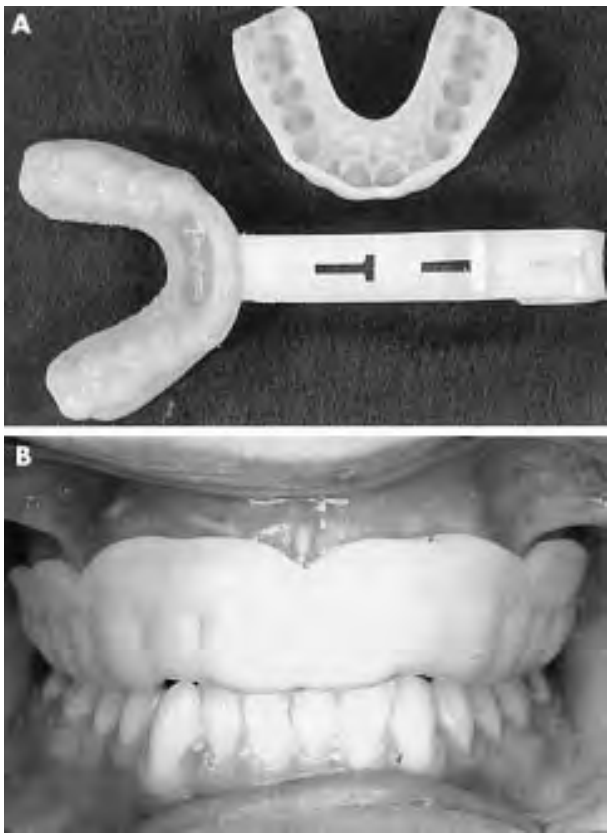

Figure 26 Custom fabricated mouthguard with and without strap attachment. (A) Note the incisal brace. (B) Mouthguard in place.[79 91]

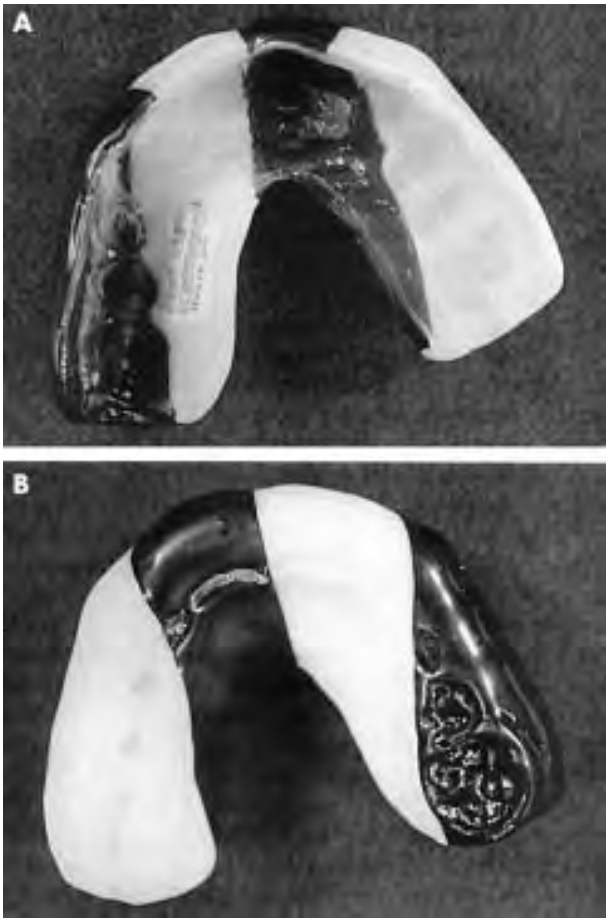

Figure 27 Heat and pressure laminated mouthguards: internal (A) and external (B) aspects. Note indentations of mandibular teeth for stability during contact.[79 91]

visors), which are in accordance with internationally approved standards organisations (CEN, ISO, ASTM, CSA) (IIHF Rules 223 and 224).[50] At the moment there are four standards for head and facial protections, but by the end of 2002 there should be a single international standard (ISO EN 10256).[33] This should represent a big improvement in safety for players, because it will finally be possible to make mandatory the introduction of labels for head and face protection in ice hockey world wide. In Switzerland, these rules have already been introduced (in the 1997/98 season) on a compulsory basis through the SIHA medical committee.

Unfortunately many players wear their helmets with the chinstrap very loosely buckled. Just wearing new protective equipment is not enough; it has to be used and worn properly. For these reasons, the IIHF rules committee has also made mandatory the correct way to wear helmet, visor, and full facemask (IIHF rules 223 and 224) (fig 28)[50]:

- **Correct way to wear a helmet (IIHF rule 223):** "a helmet shall be worn so that the lower edge of the helmet is not more than one finger-width above the eyebrows, and there should only be enough room between the strap and the chin to insert one finger."
- **Correct way to wear a visor (IIHF rule 224):** "the visor shall extend down to cover the lower edge of the nose.".

Unfortunately there are still too many players wearing the helmet and facial protection incorrectly. According to the *Laws of the game* of the Fédération International de Football Association" (FIFA), every football referee must check that the player's equipment is correct before the game starts and before allowing him to re-enter the field (law 4).[92] The rules committee of the IIHF similarly made it mandatory in 1998 for referees to issue warnings to the team for first violations concerning equipment and they should assess penalties for subsequent violations (rules 223–224, IIHF).[50]

Prevention strategies and the introduction of a specific rule against hitting from behind (IIHF Rule 523: "Checking from behind") have been effective in decreasing the number of severe spinal injuries. For these reasons, the IIHF has introduced a new "Head Checking" rule from July 2002, which does not allow any contact to the head. These rules should have the same effect in reducing head injuries, primarily mTBI, which are caused by an acceleration-deceleration blow to the head.

Properly designed mouthguards should be mandatory as devices for the prevention of dental and orofacial injuries and to reduce the incidence and severity of mTBI.

It may therefore be expected that the number of head injuries, primarily mTBI, and avoidable eye and forehead facial injuries will be reduced by eliminating contact with the face and head, through the correct use of helmets, the proper wearing of the visor and full facemask, and the mandatory wearing of properly fitted mouthguards. However, one cannot expect all head and face injuries to disappear.

A further decrease in all kinds of ice hockey injuries can only be achieved by better education of players, coaches and trainers, managers, referees, equipment manufacturers, standards organisations, national sports associations, and doctors.

(1) Players should learn to respect their opponents, to be aware of the hazards of their actions, and to learn the correct techniques of carrying out and receiving body checks and the correct way to wear properly fitted equipment.

(2) Coaches and trainers should teach players about the correct use of the stick, the way to give and take body checks,

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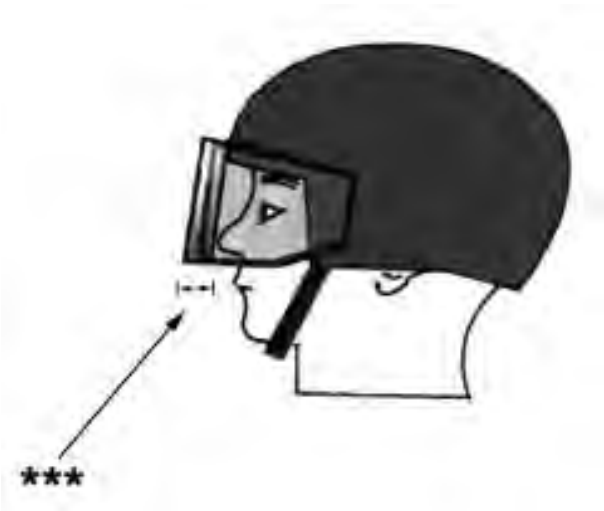

Figure 28 The correct way to wear a visor: the helmet should be worn so that the lower edge of the helmet is not more than one finger width above the eyebrows; there should be room for the insertion of only one finger between the chinstrap and skin; the visor should extend to cover the lower edge of the nose; the distance between the nose and the inside of the visor should not be more than 15 mm or one finger width (***); the visor should always cover the eyes and the nose in the frontal and lateral projections.

and the safe way to hit the boards and fall on the ice. They should further support the correct way to wear protective equipment (see *Safer hockey, smarter hockey, better hockey*).[93]

(3) All hockey personnel, especially players and coaches, should be educated about the potential for catastrophic injury, the risk, consequences, and prevention of head, eye, teeth, and neck injuries, and the limitation of protective equipment.[94]

(4) Managers should provide proper equipment and control the proper use of all equipment and the correct way to wear it. The cost of a missed game is much greater than the cost of new equipment.

(5) Referees should enforce the hockey rules and punish more severely head checks, high sticks, and checks from behind. They should also supervise the correct use of all equipment.

(6) Equipment manufacturers should further improve the design, material, and impact performance of the equipment in order to reduce the severity of injuries and to ameliorate the acceptability to the players.

(7) The work of the standards organisations should not end with the publication of standards currently under development. It is possible that standards on the skate blade, sticks, ice rink, and puck will be introduced. The research data supporting the current thinking about head, face, and body protection is constantly being reviewed, revised, and amended. As this occurs, the standards should also be reviewed, revised, and amended.

(8) It is the responsibility of the national associations and all sports organisations to insist on the use of suitable protective equipment. It will be the responsibility of the national authorities to guarantee this high testing standard by monitoring the mark.

(9) Doctors should continue to systematically evaluate and analyse the injury data in order to recognise the potential risk and identify the causal relations.

For these reasons, we have developed an International Sports Injury System (ISIS), which is an expanded computerised version of the Injury Report System (IRS).[27] ISIS is an internet based database system, which should improve the epidemiological analysis of head, face, and spinal injuries world wide.

Take home message

Prevention strategies against head checking, properly designed mouthguards, and continuous epidemiological research on an internet based database system (ISIS) are all necessary to reduce the number and severity of ice hockey injuries world wide, as well as to create the basis for further research to improve equipment, rules, and training schedules.

ISIS should also make available an internationally compatible system for the continuous monitoring of the risk factors, the protective effects of the equipment, and the effects of changes in rules through the years. Epidemiological research contributes to an increasing understanding of the incidence and mechanism of injury and has the ultimate goal of reducing risk. All these steps are necessary to create the basis of further research to improve equipment, rules, and training schedules.

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Authors' affiliations

N Biasca, Head of Orthopaedic Surgery and Co-Head of Trauma Surgery, Spital Oberengadin, CH-7503 Samedan (St Moritz), Switzerland
S Wirth, Orthopaedic Clinic, Spital Oberengadin
Y Tegner, Winternet, Institution of Health Sciences, Luleå University of Technology, Hedenbrovägen, Boden and The Ermine Clinic, Timmermansgatan 60, SE-972 33 Luleå, Sweden

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Pole Vault Summary

At its annual meeting in June, the Men's and Women's Track and Field Committee approved the following three proposals for immediate implementation:

Pole vault landing pad: Require that suitable padding be placed around the base of the standards and cover any hard or unyielding surface in a reasonable manner.
Rationale: *To provide as much padding as possible around fixed and solid objects to absorb a possible impact.*

Vaulting box: If the landing pad does not extend to the area immediately around the vaulting box, a collar of 5 to 10 centimeters of uniform thickness shall cover the area behind and to the sides of the vaulting box that are not protected by the landing pad and may extend into the box.
Rationale: *To provide as much padding as possible around fixed and solid objects to absorb possible impact.*

Helmets: A helmet used for the pole vault shall be specifically designed for such purpose and be manufactured to comply with the ASTM standards for such product. It shall bear a marking to indicate that it complies with this standard.
Rationale: *Mandates that if a helmet is worn, it must meet the ASTM standard.*

PROP reviewed in July the three pole vault proposals passed by the Track and Field Committee and took the following action:

- Approved for immediate implementation the proposal regarding helmets (***if*** a helmet is worn it must meet the ASTM standards);
- Sent back the two proposals regarding additional padding around the landing pad and the box collar to the pole vault subcommittee for further clarification and review

As a result, the pole vault subcommittee, the rules subcommittee and the Men's and Women's Track and Field Committee took the following action:

1. Vaulting box. The following proposal was approved for **immediate implementation**:

   A collar of 5 to 10 centimeters of uniform thickness shall cover the area behind and to the sides of the vaulting box that are not protected by the landing pad and shall extend into the box. Such collar shall be immovable and shall not affect the bend of the pole.

2. Pole vault landing pad. It was determined that more data and research should be obtained before implementing a rule requiring additional padding around the landing pad and standards. Specifically, the pole vault subcommittee will work with experts in the field to determine the required area to be covered, type of suitable/acceptable material and required thickness. Once these parameters have been established, the pole vault subcommittee will submit a recommendation to the rules subcommittee for consideration next June. If approved by the Men's and Women's Track and Field Committee and PROP, the new rule and requirements would go into effect for the 2012-13 academic year.

In September, the vaulting box proposal was rejected by PROP. The Panel expressed concern about the immediacy of the proposal, the associated cost, the limited availability of equipment, and the language of the current proposal. PROP communicated that they would like the pole vault subcommittee to continue studying the proposal and bring back a recommendation to the rules subcommittee next summer.

NFHS / NCAA / USATF
Pole Vault Education Video

Learning Objectives

1. Understand basic equipment and facility safety guidelines for pole vault.
2. Understand the basic physics involved with the pole vault.
3. Understand the basic pole mechanics and jump techniques required for the beginner vaulter.
4. Understand the basic movements of the beginner vaulter.
5. Understand the basic components of the approach and vault.

Menu

1. **Introduction (0:55)**
2. **Facility/equipment (4 min)**
   a. Pit, base pads, box, box collar, perimeter area of pit, runway
   b. Poles (lengths, labels, test weights, engraving, butt plugs, tape)
   c. Standards, horizontal movement, zero line, crossbar
3. **Basic laws of physics governing the vault (3 min)**
   a. Pole is a lever, rotates around fixed spot, the longer the lever the more work it takes to move it
   b. Vaulters body is a second lever rotating around fixed points on the pole
   c. Vaulter must convert horizontal energy into vertical energy (force vector)
4. **How to start a beginner (9 min)**
   a. How to grip pole, width of hands on pole, carry position of pole
   b. How to establish standing grip height
   c. Movement of pole, hands and body in a walking drill into sand
   d. Increasing grip height, discuss implications of grip, increase speed into movement
   e. Incorporate concept of jumping
   f. Incorporate concept of horizontal movement
   g. Incorporate concept of vertical movement
   h. Incorporate concept of turning
5. **Developing pole carry, run and pole drop (7 min)**
   a. Discuss proper run mechanics, talk about tempo, posture
   b. Develop a comfortable pole carry
   c. Develop concept of pole drop
6. **Develop short run straight pole vault (3 min)**
   a. Focus on developing a short approach (40’ from takeoff) so athlete can grasp the concepts of the overall jump in which the pole is not bent
7. **Develop short run bent pole vault (3 min)**
   a. Focus on developing a short approach vault that follows the same concepts of straight pole vaulting but now incorporates the bending action of a fiberglass pole.
8. **Test Questions/answers**
9. **Conclusions. (0:40)**
10. **Resources.**

A G E N D A

National Collegiate Athletic Association

Pole Vault Meeting

NCAA Hall of Champions April 26, 2011

1. Welcome and introductions. (Debra Runkle)

2. Playing rules process.

    a. USATF. (Duffy Mahoney)

    b. NFHS. (Becky Oakes)

    c. NCAA. (Rachel Seewald)

3. Injury review. (David Klossner, Jan Johnson)

4. Key issues discussion. (Greg Hull)

    a. Pole Vault Education.

        (1) Athlete Responsibility.

        (2) Facility Checklist.

        (3) Rules Modifications.

        (4) Coach Education.

            a. Tiered approach.

            b. NFHS model. (Tim Flannery)

            c. Other programs.

            d. Webinar.

b. Equipment.

(1) Helmets.

(2) Pits and padding.

(3) Poles.

5. Future timeline.

6. Other business.

7. Adjournment.

Pole Vault Meeting Summary
Indianapolis, Indiana
April 26, 2011

Participants:

Lou Andreadis, Grand Valley State University
Will Freeman, Grinnell College
Chris Graham, Commissioner of the Midwest Conference
Jeff Guy, Desert Vista High School
Greg Hull, USATF Pole Vault High Performance/Development
Jan Johnson, Sky Jumpers Vault Club
Peter McGinnis, State University New York College at Cortland
Dave Nielsen, Idaho State University
Debra Runkle, University of Dubuque
Beth Alford-Sullivan, Penn State University
Becky Oakes, NFHS
Dan Schuster, NFHS
Katie Holmes, NCAA
David Klossner, NCAA
Rachel Seewald, NCAA
Latrice Sales, NCAA
Holly Sheilley, NCAA
Liz Suscha, NCAA
Duffy Mahoney, USATF
Shayna Sangster, USATF
Sam Seemes, USTFCCCA

Participants that were invited but unable to attend include: Dave Halstead; Southern Impact Laboratory; Fred Mueller, National Center for Catastrophic Sport Injuries; Toby Stevenson, Stanford University; and Brian Yokoyama, Mt. San Antonio College.
[NOTE: This report provides a summary of comments and opinions of the coaching community for the convenience of future reference. The meeting was not a sanctioned committee meeting of the NCAA, NFHS, or USATF and therefore voting was not conducted nor actions on rules modifications taken.]

Meeting Overview
A pole vault meeting was held on April 26, 2011 at the National Collegiate Athletic Association (NCAA) headquarters in Indianapolis including representatives from the NCAA, National Federation of State High School Associations (NFHS), USA Track and Field (USATF) and expert coaches in the sport.

The NCAA, NFHS, and USATF invited the participants in an effort to focus on the development of pole vault continuing education modules that would focus upon safety and risk minimization. The group discussed the various sport organizations rules making processes, injury data, and potential solutions to help minimize risk within the sport.

In 2002 the pit for pole vault was expanded by a new ASTM standard, reducing, but not eliminating injury. A review of data is included in this summary report. A higher percentage of prior injury incidences were related to missing the pit both off the back and on the sides. Since 2002, these incidents have declined however there have been three deaths and five catastrophic injuries sustained by collegiate pole vaulters, 3 with full recovery according to statistics from the National Center for Catastrophic Sport Injury Research.

Greg Hull gave an update on the discussions from the pole vault knowledge team organized by USATF. The knowledge team identified four key issues to addressing pole vault safety: athlete responsibility, creation of a facility checklist, two key rules modifications and coach education.

From the discussion among the pole vault coaching group, two main themes became apparent as keys to enhancing risk minimization by evaluating solutions that would: (1) encourage athletes to land in the middle of the landing pad, (2) protect vaulters from the result of errant jumps that can lead to bounce outs or landing within the plant box or standards.

The following items were suggestions discussed during the meeting that could encourage athletes to land in the middle of the pole vault landing pad:

1. Create a national pole vault registry for schools that sponsor pole vault, coaches and athletes. The registry would track coaches and athletes that have accomplished the education, facility checklist and assumption of responsibility.

    - **Coach and Athlete Education.** The group supported developing a multi-tiered approach to coach education that could that involve a web-based program, a hands on clinical session and an in-depth mentorship program. The NFHS online education system was identified as a robust system that essentially creates a registry of coaches who have completed their training courses. The online course not only reaches coaches, but also administrators, athletic directors, teachers and parents. This group will move forward with the development of this concept.

    - **NCAA volunteer pole vault coaches education**. The group suggested evaluating legislative relief options to allow institutions to pay for voluntary coach certification and continuing education within the sport.

    - **Establish a Facility Checklist.** In an effort to increase the awareness of facility safety, the group suggested creating a simple checklist the coach, athlete and/or official could use to review and ensure compliance of their facility. This information can be kept on file by a state governing body [conference office and/or national governing body] so that travelling schools/institutions will know the facilities they will be competing in meet commonly accepted safety standards.

- **Athlete Responsibility and Assumption of Risks.** The group established that the athlete has the responsibility to ensure their own safety. Athletes should be required to demonstrate proficiency both in a written exam (covering, rules, facilities, equipment and basic vaulting knowledge) as well as a practical demonstration of basic physical vaulting skills that could lessen the chance of pilot error during jumping.

2. **Coaches meeting.** The group suggested modifying rules to make it permissible for a coach to meet with their athlete based on officiating standards. This would allow a coach to intervene with corrections when a vaulter is competing in an unsafe manner. Two options were discussed: (1) identity a safe zone and/or warning zone within the landing pad and/or (2) use a flag system that identifies landing in the zones.

   - Example 1: Possibly create a "safe zone" and/or "warning zone" that if the vaulter lands outside of the warning zone they may be disqualified. If a vaulter lands outside of the safe zone multiple times, they may be disqualified. Outline this zone in a diagramed card for the officials, coach and athletes to ensure consistency.

   - Example 2: Possibly have a flag system (yellow, red) if the vaulter lands outside of the warning zone. The official would raise the respective flag at the end of each jump. For example, if the vaulter lands in the middle of the pit, the official will raise a white flag; if the vaulter is outside of the safe zone a yellow flag is raised; and if the vaulter lands outside of the warning zone a red flag is raised. Multiple yellow flags or a red flag would signify an immediate coaches conference rather than a disqualification.

   - Example 3: Possibly require a countable jump to include landing in the safety zone within the pit. An athlete's head and at least one shoulder would need to land within the designated area or the jump would be classified as a missed jump. This would enhance awareness regarding proper landing sites and prompt necessary corrections. The rectangular box could measure 10 feet wide by 8 feet deep, centered on the pit with the line closest to the runway 3 feet, 6 inches back from the center of the stop board of the planting box.

3. **Allow the pole to touch the cross bar.** The group noted that clearance begins when the pole leaves the athletes hand and that when many of the world's best vaulter's jump their pole continues under the bar and lands in the pit. Penalizing an athlete for the pole being longer than the height of the bar seemed inconsistent with judging quality jumping mechanics. In addition, athletes may worry more about pushing the pole away from the bar rather than clearing it and landing in the middle of the pad. This modification could also help multi-event athletes.

- Example: If the athlete clearly makes the bar and their pole follows them into the pit and dislodges the crossbar it shall be considered a make. The athlete must have released their hands from the pole for this to count.

4. **Limit run up attempts to prevent fatigue.** The group suggested that any attempt by the athlete to initiate a takeoff in the vault should be considered a miss, preventing an athlete from then maximally running back down the runway and begin a second approach run because they did not "break the plane" within a 2 minute time frame.

5. **Runway markings**. The group suggested placing permanent standard incremental runway markings for athletes and coaches to encourage facility consistency.

6. **Weather modifications.** The group suggested clear examples with the NCAA case book for officials to determine when to move inside for the vault during inclement weather conditions.

7. **Establish cross bar standards settings.** The group noted that pole vaulters seem to use the standards to make adjustments versus their jump technique. Limiting the range (e.g., 60 – 80 centimeters) available to make the standards/equipment adjustments would help teach, educate, and train athletes to jump correctly rather than adjust the standards.

The following items were suggestions discussed during the meeting that could protect against the results of errant jumps, even among the most skilled vaulters, to minimize risk from bounce outs and landing within the box or standards:

1. **Establish a Facility Checklist.** In an effort to increase the awareness of facility safety, the group suggested creating a simple checklist the coach, athlete and/or official could use to review and ensure compliance of their facility. This information can be kept on file by a state governing body [conference office and/or national governing body] so that traveling schools/institutions will know the facilities they will be competing in meet commonly accepted safety standards.

2. **Weather modifications.** The group suggested clear examples within the NCAA case book for officials to determine when to move inside for the vault during inclement weather conditions.

3. **Exterior padding around the pole vault landing pads and the plant box area.** The group discussed that additional padding may help attenuate forces while falling into the plant box area or bouncing out of the landing pad area, however noted that a limitation to this solution is the lack of currently defined standards for such materials. The NFHS identifies at least 2 inches of padding while NCAA schools have chosen other options. Even playground

standards were identified as an option for facilities. Additional padding in the area of the plant box such as a box collar could help attenuate forces if an athlete stalls and falls down the poll or rolls back toward the runway.

4. **Helmets as permissible equipment:** An ASTM standard for pole vault helmets has been developed for use in pole vaulting as six states mandated the use of a helmet during high school events and a standard did not exist. As many helmets are designed to prevent catastrophic injuries, pole vault helmets are designed to prevent injuries such as skull fractures from a fall of 2 meters or less (e.g. bounce-outs or falls while exiting the pit). Use of a pole vault helmet will not reduce all risks and more testing is needed to ensure current models meet the ASTM standard. A common question that has been raised in the past was whether the helmet increased risk of injury. In a prospective study of 140 high school pole vaulters that were required to wear helmets, there were no head or neck injuries reported by the vaulters over two track seasons. This study is included with this summary for reference.

   - **Waivers.** Big Ten Conference pole vaulters are required to document in writing that they have reviewed with their institution's coaches the pros and cons of wearing a helmet for the pole vault event, acknowledging they understand the risks involved and are willing at accept them prior to jumping without a helmet. Grinnell College is beginning to require the use of helmets while Penn State University requires a multi-week trial period of helmet use followed by voluntary use as determined by the student-athlete. It is currently permissive to wear a helmet under sport rules.

Here is the first version of the Box collar document I referred to yesterday. It's still a little rough around the edges.

I know you all have meetings coming soon for the NCAA and USATF rules so I will put my 2 cents in now regarding some of these PV safety changes. Here are my proposals:

1. Immediately, mandate box collars as outlined in the attached document- for the 2012 outdoor season beginning in March. This will pad 70% of the poorly or unpadded surfaces in and around the plant box area. These collars will work with any or all box and landing pad set ups for years to come.
The cost is low and it is very easy to do. If you choose to do this let me know ASAP, Gill and their subsidiary manufactures will need to ramp up production a little bit. My assumption is that only 4 or 500 units will be necessary to cover all college programs.

2. Mandate for the 2013 season: padded pole slides for all PV boxes.
Although it would be nice to have total box padding right away, time is short and it is not feasible for the 2012 season. Depending on the option each school chooses the costs and installation time will vary.

3. Recommend the removal or padding of all hard surfaces along the sides and back of pole vault landing systems by the 2013 season. These costs could be low or extremely high depending upon the location. We have had very few serious accidents relative to box injuries. But they need to be addressed.

Thanks,

Jan Johnson

ASTM WK XXXX

Work Item

Standard Specification for Pole Vault Box Collars

This specification covers minimum requirements of size and physical characteristics of materials for pole vault box collars and padding inside and around the plant box. It is intended for uses attempting heights up to 6.40 m. Surveys by Boden (2001), and others have shown that falls into the pole vault box have caused significant injuries to some pole vaulting participants. Leg and arm fractures are the most common forms of injuries in plant box landings. But severe head injuries, paralysis, and even death are also related to falls into the pole vault box. Currently there are no standard specifications for the pole vault box. The rules makers for track and field only specify the dimensions of the pole vault box. Use of appropriate impact-attenuating surfacing materials in and around the plant box area may help reduce the risk of fall related injuries and deaths.

Introduction

This specification defines impact attenuation requirements for Pole Vault Box Collars and provides a means for determining impact attenuation performance using a test method that simulates the impact of a pole vaulters head when impacting the areas around the perimeter and along the inner walls of a pole vault plant box. This method quantifies the impact in terms of Gmax and HIC scores. The standard includes procedures allowing surfacing materials to be performance rated before installation and also be tested after installation for compliance with the specification.

The purpose of this specification is to reduce the frequency and severity of fall related head injuries to pole vaulters by establishing a uniform and reliable means of comparing and specifying the impact attenuation of pole vault planting boxes. Its use will give designers, manufacturers, installers, prospective purchasers, owners and operators of pole vault facilities a means of objectivity in assessing performance of surfacing materials and padding under and around pole vault landing facilities, and hence evaluating the associated injury risk

1. Scope

1.1 This specification establishes minimum performance requirements for the impact attenuation of pole vault box collars which cover the areas around and on the inner side walls of pole vault planting boxes.

1.1 This specification also establishes the minimum requirements of size, physical characteristics of materials, standard testing procedures, labeling and identification of pole vault box collars.

1.2 This specification establishes impact attenuation performance values as stated Gmax and Hic units are to be regarded as the minimum specifications.

1.3 The critical fall height (CFH) of pole vaulting materials and surfaces under laboratory conditions may not account for important factors that have the potential to influence the actual performance of installed materials. These factors may include but are not limited to weather, age, moisture, temperature extremes, and sunlight**.**

1.4 The following safety hazards caveat pertains only to the test methods portion, Section 8, of this specification: This standard does not purport to address all of the safety concerns, if any, associated with its use. It is the responsibility of the user of this standard to establish appropriate safety and health practices and determine the applicability of regulatory limitations prior to use.

2. Terminology

2.1.0 *Pole Vault Box Collar* – a devise used to offer fall protection to pole vaulters in and around a pole vault planting box.

2.1. 1 P*ole Vault Planting Box* —a device used to capture the end of a pole vaulting pole and thus give it a point of rotation for pole vaulting.

2.1.1 *Stop Board* – Also know as the back of the box. Is the 8" metal strike plate against which the pole strikes as the vaulter begins his ascent.

2.1.2 *Pole Slide* – The area down which the tip of the vaulting pole slides to the stop board.

2.1.3 *Side Walls* – The inner walls which enclose the lateral sides of the planting area from the runway material.

2.1.4 *Front opening* - The opening at the front of the plant box nearest the approach runway.

2.1.5 *Bottom Pan* – The area underneath the pole side which contains shock absorption materials.

2.1.6 *Rotation Ledge* – The bottom pan area nearest the strike plate upon which the tip of the vaulting pole rotates and carries the full weight of the vaulter and the pole.

2.1.7 *Pole Vault Use Zone* – The area beneath and surrounding a pole vault landing pit that is designed to decelerate a pole vaulters falling body

2.1.8 *Surfacing Materials* - Materials used to cover pole vault zone play areas.

2.1.9 *Loose Fill* – a compliant top layer of small, independently moveable components; for example wood fiber, bark, pea gravel, shredded foam, mulch, shredded rubber, and sand.

2.2.0 *Aggregate Surface* – A loose fill surface in which the compliant top layer is made of particulate materials (for example) sand, gravel crushed marble, pea gravel, slag, or cinders.

2.2.1 *Unitary Surface* – A complaint top layer of one or more material components bound together to form a contiguous surface: for instance molded foam, gymnastic mats, urethane, and rubber composites.

2.3.1 *Unobstructed Perimeter Surfaces* – Areas around the sides of a landing system without hard surfaces or equipment which could present a problem for the users.

2.3.2 *Front Buns* – The sectional padding between the standard up right which hold the cross car in place and the planting box.

2.3.3 *Box Collar Wings* – That portion of the box collar which extends down the inner side walls of a pole vault plant box. The wings help to hold the box collar in place and provide important protection.

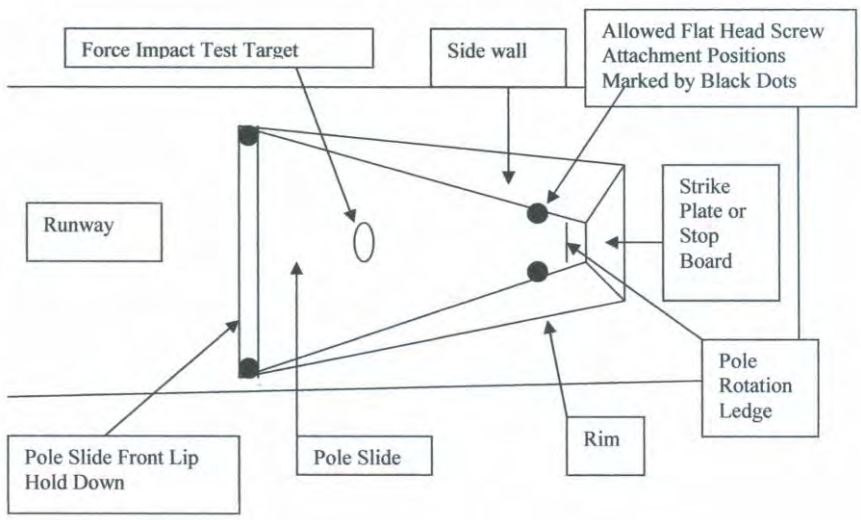
2.3.4 *Box Collar Body* - That Portion of the collar which pads the horizontal surfaces around the rim of the box and under the front buns.

2.3.5 *Pole Slide Opening* – The area at the front of the collar which fits around where the runway meets the pole slide .

2.3.6 *Pole Bend – Rotation Cavity* **-** The opening in the padded area immediately in front of the top of the strike plate which allows the pole to bend and rotate to vertical.

Pole Vault Planting Box

Figure1



Pole Vault Planting Box Profile

Figure 1

3. Definitions of Force Impact Terms

3.1 *Impact* – Contact caused by a moving object striking another object and during which one of the objects is subject to high accelerations.

3.1.1 *Critical Fall Height* (CFH) – A measure of the impact attenuation performance of a surface of surfacing materials. Defined as the highest theoretical drop height from which a surface meets the impact attenuation performance criterion specified by this specification. The critical fall height approximates the maximum fall height from which a life threatening head injury would not be expected to occur. .

3.1.2 *Impact attenuation* – The property of a surface or landing pad that through localized deformation, absorbs the energy of an impact in a way that reduces the magnitude of peak impact force and peak acceleration.

3.1.3 *Impact test* – A procedure in which the impact attenuation of a pole vault landing mat component or surface area is determined by measuring the acceleration of a missile dropped on to the surface.

3.1.4 *Impact test results* – One or more measured or calculated values from one or more tests used to define the impact attenuation of a surface or pole vault landing system component.

3.1.5 *g*- The acceleration due to earth's gravity at sea level, having a standard value od 9.80665m s-. The standard value may be approximated as 32.174 ft/s2. Accelerations may be expressed in units of g = the acceleration due to gravity.

3.1.6 *Impact test Location* – The point on the surface that is selected as the target of an impact test.

3.1.7 *g-max* – The maximum acceleration of a missile during an impact, expressed in g-units

3.1.8 *Head Injury Criteria (HIC*) – A specific integral of the acceleration-time history of an impact, used to determine relative risk of head injury.

4. Physical Characteristics of Materials and Performance Specifications

4.1.1 The box collar shall provide not more than 3" (7.62cm) of padding so long as it leaves a 22" opening at the front of the pole slide, and sufficient space as the plant box narrows to allow the bottom of the vaulting pole to pass undisturbed. The front of the collar nearest the strike plate shall allow the pole to bend and rotate to the sides as it naturally does. This padding shall meet the ASTM 1162 minimum specification for shock attenuation from a drop height of 12'6 (3.80m) impact peak deceleration shall not exceed 200g / 1700h.

5. Dimensions

5.1.1 Over all minimum dimensions of a Pole Vault box Collar are 48" wide x 54" long x 3" thick.

5.1.2. To allow the pole to bend undisturbed , the padding above the strike plate and above the side walls adjacent to it shall be tapered 105 degrees to match the angle of the strike plate.

5.1.3. The Body of the box collar must be wide enough to go under landing pads far enough to improve PV pit padding deficiencies around the front bun sections. (Force impact testing indicates that these areas are very poorly padded on all landing pads currently in existence).

5.1.2. The wings of the box padding must be limited to 3” thickness on the inner upper side walls only. Additionally, this padding must allow room for a pole to slide freely down the interior edges of the plant box where the side walls meet the pole slide.

5.1.3. The front opening nearest the runway must minimum unobstructed opening across the front of the Plant box no less than 22” wide

5.1.4 The wings must allow improperly planted poles to slide forward and downward into the bottom of the plant box. The wings should conform to the inner walls of the plant box so as to provide protection and yet guide a poorly plant pole into to the strike plate at the bottom-rear of the box.

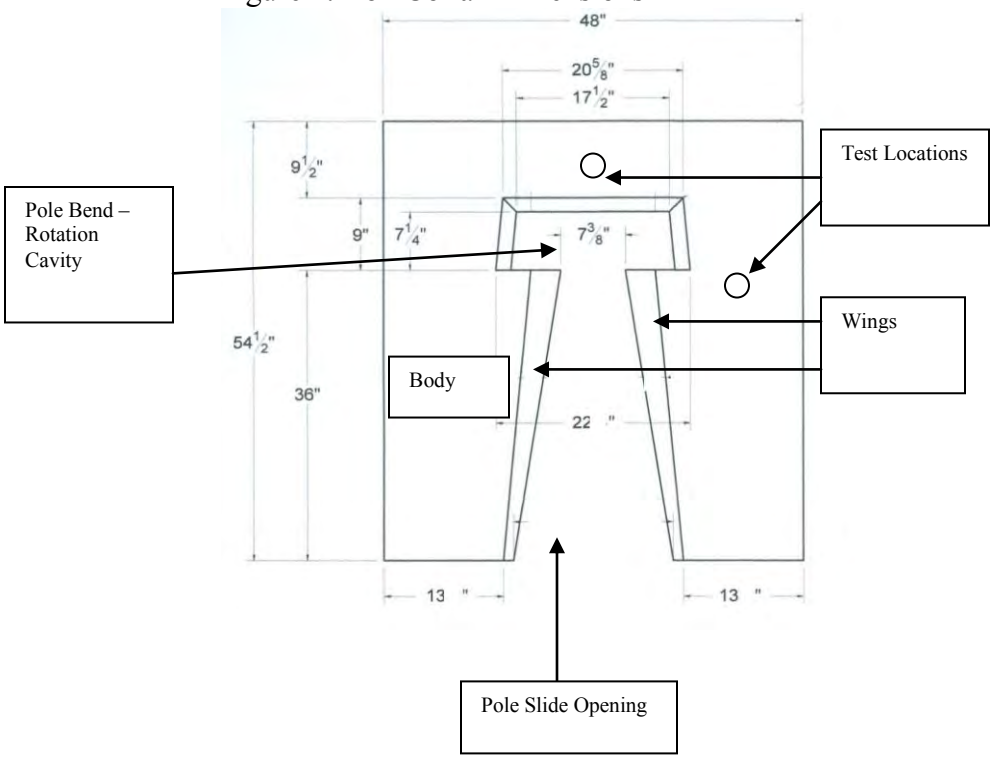
5.1.5. The padding must be no thicker than 3” on the inner walls so as to allow an opening near the bending cavity for the pole to pass as it rotates upward and forward without touching the interior padding.

5.1.6 The bend cavity must allow pole to bend into the corners formed by the side walls and the stop board, and beyond. Some poles bend in a circular rotation back toward the runway.

5.1.7. The collar should be fixed to the ground or the inner walls during use.

5.1.8 Pole Vault Box Collar must form fit the outside dimensions of the rim of a pole vault plant box, so that no hard surfaces are exposed.

Figure 2. Box Collar Dimensions



6. Significance and Use

6.1 The dynamic data obtained with the procedures given in this specification measure the cushioning properties of the box collars tested.

6.2 The size and the shape of the Pole Vault Box Collar is based upon the dynamics of the pole vaulting pole as it enters and bends against the strike plate of the plant box.

6.3 The potential shock attenuation capabilities of a Box Collar are dependant upon the physical characteristics of materials used and the spaces available in and around the plant box area.

7. Testing Apparatus

7.1 Testing Machine -Any type of dynamic testing apparatus that impacts the specimen on a solid, rigid surface (typical of service conditions) with the prescribed missile at the prescribed impact velocity and monitors and records impact deceleration-time histories.

7.2.1 Top disk with cross-sectional area 387.5 in.^{2} (0.25 m^2) outer radius 11 $^1/_8$ in. (0.282 m) height 1 $^7/_{16}$ in. (0.0371-m).

7.2.2 Inverted frustum of cone with major radius 11 $^1/_8$ in. (0.282 m), minor radius 8 $^3/_4$ in. (0.222 m), height 4 $^9/_{16}$ in. (0.1158 m).

7.2.3 Spherical segment with radius of curvature 9 $^{13}/_{16}$ in. (0.25 m), enclosed arc 124.8_, height 5 $^{5}/_{16}$ in. (0.1342 m).

7.2.4 The acceleration transducer shall be mounted within 5_ of the vertical center of the impactor (see Fig. 1).

7.2.5 The total mass of the impactor with its associated mounting hardware shall be 29.5 ± 1.1 lb (13.6 ± 0.45 kg).

7.3 Deceleration Time—The selection of specific deceleration-time recording instrumentation, including transducers and recorders is discretionary. None-the-less, the recording system shall exhibit frequency response adequate for measurement of peak deceleration to an accuracy of 65% of the true value. The total system, including transducer and recording instrumentation, shall be capable of measuring deceleration pulses with amplitude at least equal to 200 g and frequencies ranging between 0 and 1000 Hz to an accuracy of 65%.

7.4 Impact and Rebound Velocities—The dynamic testing equipment must be capable of recording Gmax and HIC with in a reasonable degree of tolerance.

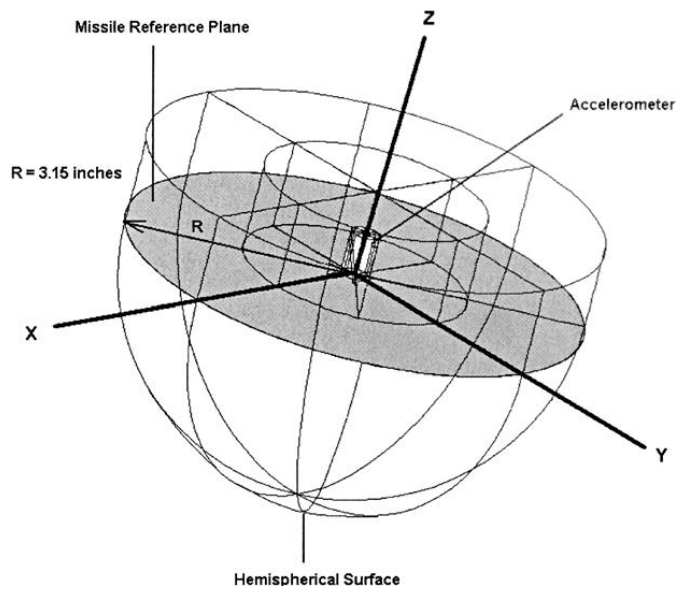


Figure 2. Force Impact Missile

8. Conditioning

8.1 Pole Vault Box Collars shall be tested under ambient conditions that match those of intended use.

9. Field Impact Testing Procedure

9.1 Summary of Procedures —Test specimens are impacted at a specified velocity by an impacting missile of prescribed mass and geometry. A transducer is mounted in the missile so that the deceleration-time history of impacts can be recorded. The procedure described in this test method employs a missile with geometry similar to that of the human head.

9.2 Pre-warm the recording instrumentation as recommended by the manufacturer. Calibrate deceleration-time and displacement-time instrumentation in accordance with procedures recommended by instrumentation manufacturers.

9.3 Place the Box Collar under the missile or orient the dynamic test equipment over the landing surface.

9.4 Raise the missile to 3.80m 12’ 51/2” above the center of the body of the box collar.

9.5 Release the missile and record the test results in accordance with the recommended procedures of the instrumentation manufacturers.

9.6 Make three successive impacts on the same area of the Box Collar.

9.7 The minimum ASTM force impact specification for the body box collar is 200g / 1700h.

10. Report

10.1 Report the following information:

10.2 Complete identification of material tested, including type, source, manufacturer’s lot number (if appropriate), thickness (if measurable), and any other pertinent information relating to sample identification.

10.3 Conditions of test including temperatures, humidity, and any other pertinent data.

10.4 Date of test.

10.5 Impact velocity.

10.6 Average values of impact parameters for the last two of three impacts or as specified.

10.7 Peak deceleration Gmax.

10.8 Head Injury Criteria -HIC

11. Instructions and Labeling

11.1 Each pole vault box collar shall be provided with instructions for proper installation.

11.2 Each landing pit shall be permanently labeled with the following items:

10.2.1 Identification of manufacturer,

10.2.2 Model designation,

10.2.3 Specific warning on installation, and

10.2.4 A warning label limiting the intended use.

12. Referenced Documents

12.1 ASTM Standards:

F1162 Standard For Pole Vault Landing Systems
F2440 Standard Specification for Indoor Wall/Feature Padding
F1292 Standard Specification for Impact Attenuation of Surfacing Materials within the Use Zone of Playground Equipment.
F355 - Test Method for Shock –absorbing properties of Playing Surfaces and Materials
F1487 - Consumer Safety Performance Specifications for Playground Equipment for Public Use.
F2075 - Specification for Engineered Wood Fiber for use as a Playground Safety Surface.
F2650 - Terminology Relating to Impact Testing of Sports Surfaces and Equipment
F2651 - Terminology Relating to Soil and Turf grass Characteristics of Natural Playing
F1936 - Specification for Impact Attenuation of Turf Playing Systems as Measured in the Field
F1487 - Consumer Safety Performance Specifications for Playground Equipment for Public Use.
F2075 - Specification for Engineered Wood Fiber for use as a Playground Safety Surface.

12.2 Federal Documents:

U.S. Consumer Product Safety Commission, Publication #325, Handbook for Public Playground Safety
U.S. Consumer Product Safety Commission, Special Study: Injuries and Deaths Associated with Children's Playground Equipment. April 2002.

Catastrophic Injuries in Pole-Vaulters: A Prospective 10-Year Follow-up Study

Barry P. Boden, MD*, Matthew G. Boden*, Rebecca G. Peter, Peter M. McGinnis**, Fred O. Mueller, PhD***, and Jan Johnson MS. ****

From *The Orthopaedic Center, Rockville, Maryland, **State University of New York Cortland, New York, the ***Department of Physical Education, Exercise and Sport Science, the University of North Carolina at Chapel Hill, North Carolina, and the ****National Pole Vault Safety Committee, Atascadero, California

Counts: words (max 6000), references (max 60)
Running Head: Catastrophic Injuries in Pole Vaulters

[§]Address all correspondence and reprint requests to: Barry P. Boden, MD, The Orthopaedic Center 9420 Key West Ave. #300 Rockville, MD 20850 (301-424-1287; fax 301-424-5266; bboden@starpower.net).

Catastrophic Injuries in Pole-Vaulters: A Follow-up Study

ABSTRACT (words; max, 300)
Background: A prior review of catastrophic pole-vaulting injuries revealed that 69% were due to athletes landing off the sides or the back of the landing pad. In response the NCAA and NFHS mandated several rules changes in 2003 including enlarging the minimum dimensions of the landing pad.
Hypothesis: We hypothesized that the new, larger landing pads would reduce the number of catastrophic pole-vaulting injuries.
Study Design: Prospective, case series.
Methods: We prospectively reviewed all catastrophic pole-vaulting injuries over a 10-year span from the 2002 through the 2011 calendar years in the United States. In addition, a survey was performed on 3335 pole-vaulters to determine the frequency of landing in the vault box.
Results: There were 25 total catastrophic injuries for the 10 year period, with 19 occurring after the 2003 rule changes. After the 2003 rule changes 14 (74%) injuries occurred when a pole-vaulter landed in or around the vault box, 4 (21%) were the result of athletes landing off the sides or back of the landing pad, and one (5%) injury was the result of a pole breaking. Catastrophic injuries for the 10 year period included 14 severe head injuries, 3 resulting in fatalities; 8 spine or pelvic fractures of which 3 were associated with intra-abdominal injuries; 2 brainstem injuries resulting in deaths; and one thoracic injury (rib fractures and pneumothorax). The vault box survey revealed that 77.12% (n=2572) landed in the vault box one to three times during their career, 6.12% (n=204) four to six times, 0.84% (n=28) seven or greater times, and 15.92% (n=531) never landed in the vault box.
Conclusion: The 2003 rule changes that mandated a larger landing pad have significantly reduced the number of catastrophic injuries from pole-vaulters missing the back or sides of the landing pads; however, the problem of pole-vaulters being injured from landing in the vault box remains a significant problem.

By a large margin, pole-vaulting is associated with the highest risk of direct catastrophic injuries in track and field. Prior to 2003, pole-vaulting had one of the highest rates of direct, non-cardiac catastrophic injuries per 100,000 participants for all sports monitored by the National Center for Catastrophic Sports Injury Research (NCSSIR) (ref). In a previous report on catastrophic pole-vaulting injuries, 32 incidents over a 16-year span between the 1982 and 1998 academic years (Boden) were documented. Three common mechanisms of injury were described: 17 (53%) athletes landed with part of their body on the landing pad but their head struck the surrounding hard surface, 8 (25%) landed in the vault box after being stranded at the height of the jump, 5 (16%) completely missed the landing pad, and 2 were unknown (ref). Thirty-one (97%) of the 32 accidents resulted in a serious head injury and one (3%) in a thoracic spine fracture with paraplegia (3%) (ref). Of the 32 individuals who sustained a head injury, 16 (52%) died as a result of the trauma and neurologic recovery was incomplete for 6 (19%) (ref).

After publication of the prior manuscript, the authors decided to perform a 10 year prospective study on catastrophic pole-vaulting injuries starting in 2002. During the 2002 calendar year there were 6 catastrophic pole-vaulting injuries including 3 deaths. The increased awareness of catastrophic injuries in pole-vaulting based on the prior report from 1982 to 1998 as well as the high number of catastrophic injuries in 2002 prompted the National Collegiate Athletic Association (NCAA), the National Federation of State High Schools Associations (NFHS), and USA Track and Field (USATF) to mandate rule changes to promote safety in the sport of pole-vaulting (ref). The rule change with the most potential to reduce injuries was an increase in the minimum dimensions of the landing pad behind the vault box from 16’ X 12’ to 19’8” X 16’5” (NCAA, NFHS) (Figure 1). In addition to increasing the dimensions of the landing pad, the NFHS also mandated that all hard surfaces surrounding the sides and back of the landing pads should be removed or padded with a minimum of 2 inches of dense foam or other suitable material (s) (ref). The NFHS also recommended that any excess material such as asphalt or concrete that extends out from beneath the landing pad be removed. The NCAA did not mandate any regulations concerning the surfaces surrounding the sides and back of the landing pad. Both the NCAA and the NFHS mandate 2 inches or 2 to 4 inches respectively of dense foam or other suitable material in the box collar (space between the vault box and landing pads). In an attempt to reduce the number of vault box injuries the NCAA() and NFHS() also mandated that the crossbar be a minimum of 45 and 40 cm. respectively beyond the vertical plane of the back of the planting box with a goal of encouraging pole-vaulters to land closer to the center of the landing pads. The 2003 NFHS rules also allowed the runway to be marked so athletes can better gauge their takeoff and the NFHS recommended a preferred landing zone be painted or sewn in the middle of the landing pad to encourage landing in the center of the landing pad. The 2003 NCAA rules prohibited tapping during warm-ups and competition. The 2004 NCAA rules allowed the runway to be permanently marked in the takeoff area.

The purpose of this study was to prospectively review all catastrophic pole-vaulting injuries in the United States over a 10 year span from 2002 through 2011. Particular attention was focused on whether the rule changes in 2003 have been effective in reducing the number of catastrophic injuries. In addition a survey was performed to determine the frequency of pole-vaulters landing in the vault box.

MATERIALS AND METHODS

Injury Definitions

For the 2002 through the 2011 calendar years, we prospectively collected data on catastrophic pole-vaulting injuries in the United States. Direct catastrophic injuries in this study were defined as any severe injury such as a brain hemorrhage or skull fracture, spine or pelvic fracture, or significant pulmonary or intra-abdominal injury resulting from the participation in pole-vaulting. Indirect injuries resulting from systemic failure due to exertion were not included in this study. Concussions and extremity injuries were not considered catastrophic injuries.

Injury Reports

Data were collected from the NCCSIR as well as internet searches and communication with the National Pole Vault Safety Committee (NPVSC) and the Pole Vault Safety Certification Board (PVSCB). The NCCSIR contacts high school and college coaches and athletic directors annually, as well as executive offices of state and national athletic organizations, athletic trainers, and a national newspaper clipping service and requests a report on any catastrophic injuries. The PVSCB also documents catastrophic injuries and meets annually with the American Society for Testing and Materials (ASTM) to discuss equipment, rule changes, injuries, and safety measures in the sport of pole-vaulting. When information was received from either organization concerning a possible catastrophic sports injury, contact was made by telephone, personal letter, or questionnaire to obtain preliminary data including athlete's name, date of injury, age, diagnosis, and school, as well as a brief description of the mechanism.

Injury Surveillance

On an annual basis any injuries identified through the NCCSIR, NPVSC, PVSCB, and internet searches were recorded and additional detailed information was obtained through contact with the athlete, family member, coach, and/or witness, through internet searches and Facebook, and/or internet videos of the injuries. A detailed questionnaire was employed to collect data on patient characteristics (age, gender), date of injury, level (high school, college, other), and highest jump previously attained. Information concerning the mechanism of injury (missed landing pads completely or incompletely, vault box, other) were elicited with specific attention to whether the equipment was in compliance with the 2003 rule changes. Additional information was obtained on whether the injury occurred during practice or competition, the height attempting to jump at the time of injury, whether the athlete was wearing a helmet, weather conditions, and equipment employed. Information was also elicited concerning the medical diagnosis, treatment, outcome, prevention, and whether there was litigation.

Survey

In addition to reviewing the catastrophic pole-vaulting injuries a survey was performed at all pole vault camps and clinics provided by one of the authors (AU5) from 2007 through 2011. The purpose of the survey was to determine the frequency of pole-vault athletes landing in the vault box. All participants were queried on age, gender, number of seasons participating in the sport of pole-vaulting, personal best vault attained, and frequency of landing in the vault box. Participants that had landed in the vault box were asked whether the injury required medical attention and if so what portion of their body they injured.

RESULTS

A total of 25 direct catastrophic pole-vaulting injuries were identified from 2002 through 2011. Of the 23 contacts who were located for completion of the detailed questionnaire, information was obtained via phone call or email correspondence with the athlete in 15 cases, the coach in 4, a parent in 3 accidents, and a teammate that witnessed the injury in 1 case. Information from internet articles only was obtained for 2 athletes, but a parent was interviewed in each article. A videotape of the injury was reviewed for 2 athletes.

Epidemiology and Demographics Patterns

Twenty-two injuries occurred in males and three in females in this study. The level of participation for the athletes was college in 13 (52%), high school in 10 (40%), middle school in 1 (4%), and professional in 1 (4%) (Fig. 2). The mean age at the time of injury was 19 years (range, 15 to 26). The mean highest previous vault successful completed by the athletes was 13 feet, 7 inches (range, 9 ft. to 19ft, 8 in.). For the 10-year period the overall annual rate of injuries was 2.5 per year (2.2 per year for males and 0.3 per year for females) and 2.0 per year (1.78 per year for males and 0.33 per year for females) after the 2003 rule changes.

The NFHS and the NCAA record the number of track and field participants each year; however, they do not record the number of pole-vaulters within the sport of track and field. We estimated the total number of high school pole-vaulters in the 2008 and 2010 years to be 80,000 (pvp). This number was estimated by determining the exact number of pole-vaulters in the states of Washington and Oregon and then calculating the number of pole-vaulters per total track and field athletes in these states. This ratio was then multiplied by the total number of track and field athletes in the United States to determine the total number of pole-vaulters in the United States at the high school level. Since only athletes that competed in track meets and not those that may have practiced with the teams without competing were included, we estimated that the total number of pole-vaulters at the high school level each year is 90,000 (50,000 males and 40,000 females). At the college level the website mile-split estimated 3700 female and 4000 male competing pole-vaulters per year (ref). This may underestimate the total number of college pole-vaulters since many teams may not register with mile split and only pole-vaulters that are competing and not those that practice with the team are recorded. If 10,000 college pole-vaulters per year (5556 males and 4444 females) is the correct estimate, this equates to approximately 100,000 total pole-vaulters for high school and college each year.

Based on these estimates the total high school and college direct catastrophic injury rate for the 10-year period was 2.5 per 100,000 pole-vaulters (3.96 per 100,000 males and 0.68 per 100,000 females) and 2.0 per 100,000 (3.20 per 100,000 male athletes and 0.75 per 100,000 females) after the 2003 rule changes. The total incidence during the 10-year period for college pole-vaulters was 13 per 100,000 (19.8 per 100,000 males and 4.5 per 100,000 females) and 1.1 per 100,000 (1.8 per 100,000 males and 0.25 per 100,000 females) for high school athletes. After the 2003 rule changes the incidence was 12.2 per 100,000 college pole-vaulters (18.0 per 100,000 males and 5.0 per 100,000 females) and 0.07 per 100,000 high school pole-vaulters (1.11 per 100,000 males and 0.28 per 100,000 females).

Timing of Injury

Eleven of the accidents occurred during a competition, 8 during practice, 5 while the athlete was warming up for a competition, and one was unknown. In 13 of the accidents the average height attempted at the time of injury was 13 feet, 10 inches (range, 9 ft. to 18 ft. 6 in.). The remaining injuries occurred in practice without a bar (n = 6) or the height was unknown (n =6). None of the athletes were wearing helmets at the time of the injury. Weather conditions were not considered to be a causative factor in any of the injuries.

Mechanisms

The most common mechanism of injury (17, 68%; 14, 74% post 2003) occurred when a pole-vaulter released the pole prematurely or did not have enough momentum and landed in the vault or planting box (Fig. 3B). There was padding in the box collar around the vault box in 4 cases, no padding in 4 cases, and unknown in 6 cases. There were a variety of dangerous circumstances and landing patterns for the vault box injuries. Many reported landing with part of

their body on the landing pads or front buns and their head in the vault box or vice versa. Several landed completely in the vault box or on an asphalt or metal box edges, or the runway surface material in the space between the vault box and the landing pads (Fig. 4). In one case the athlete's pole tip caught on the front lip of the vault box which was warped and above the surface of the runway which disrupted the planting position of the pole. Other risk factors for landing in the vault box were releasing the pole early or changing to a stiffer pole or higher grip before the catastrophic jump in an attempt to clear a higher vault. Often the pole-vaulter had a late plant, poor approach run, or did not have enough speed and/or power to allow the stiffer pole to carry them into the landing pad. Several pole-vaulters reported that their hands slipped off the pole before completing the jump. Another risky behavior reported was the practice of tapping or having an assistant(s) give the pole-vaulter a forward and upward push at takeoff to help clear the bar. With appropriate supervision this practice may be beneficial during training but can lead to a false sense of security during competition when tapping is not allowed.

The second most common mechanism occurred when (7, 28%; 4, 21% post 2003) athletes landed on a hard surrounding surface to the sides or back of the landing pad. Of the 6 injuries that occurred in 2002 before the 2003 rule changes 3 were from athletes missing the sides or back of the landing pad and 3 were from athletes landing in the vault box. Therefore, after the rule changes there were only 4 athletes that were injured from missing the sides or the back of the landing pad. All 4 athletes were participating at the college level. In all 4 cases (Burnett, Keegan and Yin and Kear) there were no soft surfaces around the landing pads and the athletes landed on concrete in 3 cases, and a rubberized track in 1 case. In 2 cases the facilities did not comply with the 2003 rules either due to a landing pad that didn't meet the minimum size requirements or multiple high jump pads being used as a pole vault landing pad. In addition to multiple high jump pads (Lutz) not being allowed, the total dimensions of the high jump pads were less than the minimum landing pad size requirements. {Find out in Oct. if landing pads on Kear were small.}

One college athlete sustained a skull fracture and severe head injury with seizures when the pole broke and hit him in the head while attempting to vault 14 feet. After being struck by the pole he landed in the middle of the landing pad. The pole was reported to meet regulation standards and had no known defects.

Injury Profile

The events resulted in a major head injury in 14 (56%) athletes, a spinal column fracture in 5 (25%) cases, a pelvic fracture in 3 (12%) athletes, a brainstem injury in 2 (8%) athletes, and a pneumothorax and rib fractures in 1 (4%) athlete (Table 1). At least 6 of the head injuries had a concomitant skull fracture. In addition to these more serious injuries, temporary quadriparesis, a nondisplaced C7 transverse process fracture, a lumbar herniated disc, and a clavicle fracture each occurred in one athlete. Two of the spinal injuries resulted in paraplegia. The athletes that sustained a pelvic fracture also were diagnosed concomitantly with a pneumothorax in 2 cases, a splenic rupture and/or intraabdominal bleed in 2 cases, rib fractures in 2 athletes, a hip fracture in 1 case, and a clavicle fracture in 1 case.

There were 5 fatalities (20%, 0.5 per year) for the 10 year span and 2 (0.22 per year) after the 2003 rule change. Three deaths were from athletes partially or completely missing the sides or back of the landing pads (2 prior to 2003) and 2 from athletes landing in the vault box (1 prior to 2003). Three deaths resulted from trauma to the head and 2 to the brain stem. One athlete died instantaneously and the remaining 4 died within 4 days.

At least 12 athletes underwent at least one surgical procedure: 8 required a cranial decompression, 3 required a spinal procedure, and 1 a spleenectomy and clavicle open reduction and internal fixation.

Outcome

The recovery for the 20 athletes who survived was complete for 12 and partial for 8. Permanent disabilities included cognitive impairment in three, paraplegia in two, back pain, paresthesias, and depression in one, headaches and vertigo in one, and an unspecified disability in one patient. Of the 12 athletes who made a full recovery 7 returned to competition in pole vaulting, but several eventually quit due to psychological scars from the catastrophic injury. In addition to the devastating health and emotional impact, at least 6 of the accidents resulted in a lawsuit.

Vault box survey

3335 pole-vaulters participated in the vault box survey (Table). The age range of the survey participants ranged from 12 to 18 years old with 85% being 15 to 17 years old (mean, 15.63 years old). 55.29% (n=1844) of the survey participants were males and 44.71% (n=1491) were females. The mean number of seasons participating in the sport of pole vaulting was 2.15 with less than 10% of the participants having greater than 3 seasons of participation. The mean personal best height vaulted by the participants was 10.11 feet (range, 6 to 15 feet). The personal best vaulted was unknown for 7 participants.

Of the 3335 participants in the study 77.12% (n=2572) landed in the vault box one to three times, 6.12% (n=204) four to six times, 0.84% (n=28) 7 or more times, and 15.92% (n=531) never landed in the vault box. Of the 2,804 participants who landed in the vault box at least once, only 3.03% (n=101) required medical attention. There were 130 injured body parts (Table) with the most frequently injured body part being the ankle (n=32, 24.62%), the heel (n=25, 19.23%), the lower back (n=16, 12.31%), and the knee (n=11, 8.46%).

DISCUSSION

There is little information on pole-vaulting injuries in the literature (Gainor, Rebella, Wallgren) with only one report on catastrophic injuries (Boden). Boden reported that there were 32 catastrophic injuries between the 1982 and 1998 academic years. The majority (69%) of injuries occurred when an athlete landed off the sides or the back of the landing pad followed by accidents in the vault box (25%)( ). Thirty-one of the accidents resulted in a serious head injury and one in a thoracic spine fracture causing paraplegia. Of the athletes who sustained a head injury, 16 died as a result of their injury and 6 had a permanent disability. In an effort to reduce the number of injuries in pole-vaulting several rule changes were instituted by the NCAA and NFHS in 2003 with the most significant being an increase in the minimum pole vault landing pad size behind the vault box from 16’ X 12’ to 19’8” X 16’5” (NCAA, NFHS).

Since the 2003 rule changes, the number of catastrophic injuries per year (2 per year) was identical to the rate of catastrophic injuries in the prior 16 year report ( ). However, the rate of fatalities was markedly diminished at 0.22 per year versus 1.0 per year in the prior study ( ). The reduction in fatalities may be secondary to a higher percentage of vault box landings in the new report compared to athletes landing off the sides or back of the landing pad in the prior report. Due to the lower number of injuries in this report identified by the NCCSIR and the higher number located through internet searches and other avenues, it is possible that the new report has more injuries that were “less catastrophic.” If more advanced internet searches had been possible at the time of publication of the prior study, it is likely that a larger number of nonfatal catastrophic injuries would have been identified than were reported in that study.

Pole-vaulting ranks as one of the most dangerous sports (Thomas, Mueller). In one report that analyzed the US National Registry of Sudden Death in Young Athletes for a 30-year period from 1980 to 2009, pole vaulting had the second highest number of direct, traumatic fatalities after football (Thomas). For the 28-year period from the fall of 1982 through the spring of 2010 the NCCSIR reported 1284 direct catastrophic injuries in high school and college sports (NCCSIR, 28th report). During the same time period the direct catastrophic injury rate per 100,000 athletes for all sports combined was 0.62 and 2.54 for high school and college respectively (28th report). After the 2003 rule changes the direct catastrophic rate of 0.7 (1.1 in males and 0.28 in females) per 100,000 high school pole-vaulters is comparable to the overall catastrophic incidence for all high school sports. However, the rate of direct catastrophic injuries in college pole-vaulters after the 2003 rule changes (12.2 overall, 18.0 for males and 5.0 for females) was significantly higher than the combined rate of 2.54 for all college sports and indicates that more emphasis needs to be placed on preventing pole vaulting injuries at the college level. For male college athletes, gymnastics (25.05) has the highest rate of catastrophic injuries per 100,000 participants, followed by pole-vaulting (18.0) ice hockey (10.22), football (9.5) and lacrosse (5.28) (NCCSIR, 28th report). For female college athletes, skiing (12.46) has the highest rate of catastrophic injuries per 100,000 participants, followed by equestrianism (10.85), pole-vaulting (5.0), gymnastics (4.8) and ice hockey (4.71) (NCCSIR, 28th).
In contrast to the level of participation (78% high school and 9% college) at the time of the injury in the prior study on catastrophic injuries, this study identified a higher percentage of injuries in college athletes (52%) compared to high school (40%) pole-vaulters (ref). If participation numbers are accounted for, the incidence per 100,000 pole-vaulters was over 12 fold higher in college versus high school pole-vaulters. Similarly the average age of the injured athletes in this study was 19 years old versus 17.5 years in the prior study (ref). The older age of the injured athletes in the current study may be due to the larger landing pad preventing injuries in high school athletes, whereas older college athletes are attempting higher vaults, gripping the pole higher, and may be stranded over the vault box. Unlike the prior study which reported no injuries in female athletes, this study identified 3 injured female athletes. This is likely due to the increased participation by females in pole-vaulting over the past decade.

According to the National Pole Vault Safety Committee there have been 8 catastrophic injuries in the 350 male athletes in the United States who have pole vaulted over 17’6 ¾” since 1971 for an incidence of 2,286/100,000 or 2.3 per 100 pole-vaulters. There was one catastrophic injury in the 55 US women pole-vaulters who have cleared 14’ for an incidence of 1818/100,000 or 1.8 per 100 pole-vaulters. Many of these athletes were injured while vaulting in competitions governed by U.S.A. Track and Field (USATF) rules or International Association of Athletics Federation (IAAF) rules. Neither the USATF nor the IAAF requires the use of a box collar or the padding of hard surfaces surrounding the landing pad. The higher catastrophic injury rate for these elite or semi-elite pole-vaulters is primarily related to the higher heights from which the pole-vaulters are falling. However, the high injury rate may also be related to the less stringent USATF and IAAF rules regarding padding of hard surfaces and the use of a box collar, at least for those injuries occurring after 2002.

Landing Pads

The new rule in 2003 enlarging the minimum dimensions of the landing pads from 16’ X 14’ to 19’8” X 16’5” behind the plant box at both the college and high school levels has been extremely effective in reducing the number of catastrophic injuries due to pole-vaulters missing the sides or back of the landing pad. In a previous report on catastrophic pole-vaulting injuries, there were

22 injuries during a 16 year span (1.4 per year) from athletes missing the sides or back of the landing pad (Boden). After the 2003 rule changes there were 4 athletes (0.4 per year) that landed partially or completely off the sides or rear of the landing pads for a greater than 3-fold reduction. Since there were no participation numbers for the initial study, the incidence of injuries over the 2 time periods cannot be compared. The disadvantage of the larger landing pad is the additional cost, often between $7000 and $11,000, causing some schools to drop the pole vault event or to practice at a centralized location. Nonetheless, the added cost appears to be beneficial based on the reduction of catastrophic injuries. It is also probable that for every catastrophic injury that is prevented by the larger landing pads, there are numerous non-catastrophic injuries that are also prevented. In addition to the larger landing pad being responsible for the reduction of injuries, it is possible that the coaches box, soft surrounding surfaces at the high school level, and increased awareness and emphasis on safety for both coaches and athletes has played a role.

All 4 of the athletes that missed the sides or rear of the landing pads after the 2003 rule changes were participating at the college level and landed on hard surfaces, concrete in 2 cases, a rubberized track in 1 case, and a hardwood indoor floor in 1 case. Two of these cases were in noncompliance with the 2003 rule changes using a landing pad that didn't meet the minimum size requirements or substituting multiple high jump pads for a pole vault landing pad. In 2003 the NFHS also mandated that all hard surfaces surrounding the sides and back of the landing pad should be eliminated or covered with a minimum of 2 inch thick unspecified soft padding (ref). The NCAA has no similar rule. If the NCAA mandated a similar rule requiring soft surrounding surfaces and all schools were in compliance there may have been no catastrophic injuries from athletes missing the sides or back of the landing pad after 2003. None of the catastrophic injuries in the current or prior study occurred due to an athlete landing on a surrounding surface of grass, dirt, sand, wood chips, or 2" of padding ( ). We recommend strict adherence to the minimum landing pad dimensions and that the type of padding on the surfaces that surround the sides and back of landing pad be specified and at least 2 inches of padding be used.

Vault box injuries

While the number of catastrophic injuries from athletes missing the sides and back of the landing pads decreased compared to the prior study, the number of injuries due to athletes landing in the vault box more than tripled from 0.5 per year in the earlier study (ref) to 1.7 per year in the new study (1.55 after the 2003 rule changes) and is cause for concern. The vault box survey also indicated that a large percentage of pole-vaulters landed in the vault box during their career. In fact 84% of those surveyed landed in the vault box at least once and 7% landed in the vault box at least 4 times. Most of these pole-vaulters were relatively inexperienced, amateur pole-vaulters with an average personal best of 10 feet and 2 years of vaulting experience. It is difficult to determine if these numbers plateau, increase, or decrease with more years of vaulting. The number of vaults would likely increase the chances of landing in the vault box, but the additional experience may reduce the incidence of box landings. It is likely that elite pole-vaulters land less frequently in the vault box, but have more catastrophic results when they do land in the vault box from the higher heights attempted. It is also possible that the elite and college pole-vaulters use a higher grip on the pole and therefore have a greater likelihood of a vault box landing since the execution of their takeoff speed and technique are so critical to getting the pole to rise to vertical. Despite the survey revealing a high percentage, 84%, of at least one vault box landing in inexperienced pole-vaulters, there were only a small percentage of injuries requiring medical attention. Medical attention was required in only 3% of the 2,804 participants that landed in the

vault box at least once. This may be explained by the fact that most jumps being attempted at the time of injury in the survey were less than 10 feet so the athletes impacted the box or ground with less force on landing. The majority of injuries occurred to the ankle, heel, lower back, and knee. Although this report focuses on catastrophic pole-vaulting injuries it is clear from this survey that numerous non-catastrophic injuries occur each year from pole-vaulters landing in the vault box. Based on the number of participants in the survey and their average number of years pole-vaulting, it can be extrapolated that there may be over 1000 non-catastrophic injuries requiring medical attention each year from pole-vaulters landing in the vault box.

There are several possible explanations for the increased rate of catastrophic injuries from athletes landing in the vault box. In 1995 the NFHS rules committee mandated that "the vaulter's weight shall be at or below the manufacturer's pole rating." (ref 1994 NFHS). This rule was instituted to encourage athletes to use a lower hand hold which should reduce the bend of the pole and lower the risk of control problems such as over-shooting the landing pad, landing off the sides, landing in the vault box, and/or the pole breading. However, the stiffer poles are more resistant to rotating to vertical. If the resistance of the pole is too great for the amount of take-off energy the pole-vaulter produces, the pole-vaulter become stranded and may land in the vault box. In many cases the athlete reported changing to a stiffer pole prior to the catastrophic jump in an attempt to clear a higher vault. The stiffness, length, and weight of the pole as well as the grip height in relation to the athlete's height, weight and attempted vault height requires further research to determine safe standards.

The high school and college rules state that the front edge of the vault box shall not extend above the grade of the runway ( ). Nonetheless, one athlete reported a warped front lip that disrupted the planting position of the pole and contributed to the injury. The front lip of the vault box needs to be carefully checked before attempting any vaults. Another situation that may place pole-vaulters at risk for injury is the practice of tapping or having an assistant give the pole-vaulter an upward push at takeoff to help clear the bar. With appropriate supervision this practice may be beneficial during training but can lead to a false sense of security during competition when tapping is not allowed. Pole-vaulting is a complex sport and coordination of speed and power of the athlete as well as timing of pole release, in particular avoiding early release, are skills that requires significant practice, expertise, and coaching.

In 2003 the new high school and college pole-vaulting rules also mandated that the crossbar be moved away from the vault box and further over the landing pad in an attempt to reduce vault box injuries. It is unclear why this rule change was not effective, but perhaps pole-vaulters are having a harder time reaching the cross-bar with a higher risk of being stranded over the vault box. The ideal position of the crossbar requires further research.

Box collar

Many pole vault landing pads do not adequately cover the area immediately surrounding the sides and back edges of the vault box. The design of these landing pads allows the pole to bend without contacting the landing pad. After 2003, the NHFS required that these exposed surfaces be covered with "a minimum of 2-inch dense foam padding (box collar)" (ref) and the NCAA required that these surfaces be covered with "a collar of 2 to 4 inches of padding of uniform thickness" (ref). Many of the vault box injuries in this study occurred when the athletes landed on the exposed surfaces surrounding the sides or back edges of the vault box or a combination of these surfaces and the vault box. In spite of the NCAA and NFHS rules requiring padding in the box collar, many facilities were in noncompliance at the time of the catastrophic vault box injury in this study. One of the authors (JJ) has also noted a high rate of noncompliance with this rule at

track and field meets. Therefore, strict enforcement of this rule is important, and specific standards regarding the shock attenuation capabilities of the padding used in box collars is needed.

There are several preventive strategies for reducing the number of vault box injuries. Enforcing the existing rules requiring the box collar use is a critical first step and writing standard specifications for the box collar and its shock absorbing capabilities is next. The use of newer materials that allow greater force absorption than the standard 2 inches of unspecified dense foam should be investigated. In addition padding the sides of the vault box as well as the bottom of the vault box may be beneficial. A one piece box collar that also extends down the side walls of the vault box would not only pad the side walls of the vault box but would also hold the box collar padding in place better. The current vault box dimensions require a flare on the sides and rear to allow space for the fiberglass pole, introduced in the 1960's to bend and rotate to vertical. However, the flare on the sides of the vault box significantly increases the area of exposed hard surface in the vault box and may not be necessary to allow the pole to bend like the rear flare in the box. Eliminating the flare on the sides of the vault box or padding the interior side walls would reduce the hard surfaces and potentially diminish the risk of injury. Another change that may reduce or eliminate vault box injuries is making the vault box narrower. Requiring spotters near the vault box has been proposed but is controversial, as they may be at risk of injury from the pole or from impact with a descending athlete.

Helmets

Helmet use is optional in most states according to NCAA and NFHS rules and may be helpful for reducing head injuries. A standard for pole vault helmets, F2400-06 Standard Specification for Helmets used in Pole Vaulting, was published by ASTM in 2006. This standard is the first and only existing standard for pole vaulting helmets. In 2003, before a standard for pole vaulting helmets existed, the state high school associations in four states (Maine, Minnesota, North Dakota, and Wisconsin) began requiring their high school pole vaulters to wear helmets. High school pole vaulters in Maine, Minnesota, and North Dakota were still required to wear helmets in 2011, but beginning in late April 2010, Wisconsin high school pole vaulters were no longer required to wear helmets. The rules requiring helmet use in these states do not indicate the type of helmet to be used or require that the helmet meet any standard or specification.

The majority of the catastrophic injuries to pole vaulters reported in this study and in the previous study by Boden, et al. (2001) were severe head injuries, so the use of helmets by pole vaulters would seem warranted. However, a fall from a typical pole vaulting height of 3 meters or higher onto a hard surface would easily exceed the protection capabilities of a helmet. The highest drop test height specified in the ASTM pole vault helmet standard, F2400-06, is only 2 meters. The introduction to F2400 Standard Specification for Helmets for Pole Vaulting includes the following statement, "A helmet, however, is not likely to prevent serious injury or death if a vaulter lands outside of the pole vault pit and strikes his or her head after a vault." (ASTM, p.1). A helmet may provide some protection but it is unlikely to fully protect a vaulter from a severe head injury if the vaulter falls from a height higher than 2 meters and strikes his or her head on a hard surface outside the landing pad. Two to four inches of proper padding is far superior to a pole vaulting helmet at reducing impact forces.

Pole breaking

One athlete sustained a catastrophic head injury when a regulation size pole with no known defects broke and hit him in the head while attempting to vault 14 feet. After being struck by the pole he landed in the middle of the landing pad. This type of injury is extremely rare. Of 71

known catastrophic pole-vaulting injuries since 1971, there was only one other reported case of a pole breaking resulting in severe injury. The energy possessed by the pieces of the pole after it breaks is very large. The vaulter as well as the officials, spectators and other vaulters or persons close to the landing pad are at risk of injury if they are struck by a piece of a broken pole.

Education and coaching experience

Although none of the witnesses to these accidents reported inadequate experience of the coaches as a contributing factor, pole-vaulting is a complex event requiring specialized training. Education of pole vault coaches and athletes is necessary. We recommend a coaching certification for all pole-vault coaches. The current estimate of certified coaches is less than 30%. In addition the authors recommend that every school have an administrator verify and sign a form that the pole vault facility is in compliance with the rules.

CONCLUSIONS

The 2003 rule changes that mandated a larger landing pad have significantly reduced the number of catastrophic injuries from pole-vaulters landing off the back or sides of the landing pads. However, the annual rate of catastrophic injuries from pole-vaulters landing in the vault box has tripled over the last decade and remains a significant problem. Potential preventive strategies that require further research include developing standard specifications for box collars and requiring the use of box collars that meet these standards, improving box collar shock absorption materials, padding the sides of the vault box as well as the bottom of the plant box, and making the vault box narrower. The dimensions of the pole, especially the stiffness and proper grip heights, as well as the ideal position for the crossbar also require further study.

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Figures

Figure 1: Dimensions of the new landing pad.

Figure 2: Number of catastrophic injuries in pole vaulting at the high school and college levels from 2002 through 2011.

Figure 3: A, Location of landing for athletes in the 1982 to 1998 study of catastrophic pole-vaulting injuries. Arrow head represents the head of the pole-vaulter and the line the torso and feet of the pole-vaulter. B, Location of landing for athletes in the current 2002 to 2011 study.

Figure 4: Vault box and box collar

Pole Vault Caution Zone Proposal

By Jan Johnson

Purpose:

To improve pole vault safety by bringing greater attention to landing in or near the middle of the landing system by penalizing vaulters who land too close to the edges or in front of the zero line. This rule effectively minimizes most of the classic dangerous jumps as outlined below.

Benefits:

*This rule would help to reduce wild and out of control jumps by penalizing the participant.
*Simple and easy to enforce.
*Low cost
*The markings we recommend will allow coaches and vaulters to better judge the position of the landing system in relation to the plant box, and also allows a general view of the position of the standards relative to the zero line.

Costs:

Any landing system may be marked with a tape measure and a can of spray paint in about 10 minutes time. (see example below)

Pole Vault Caution Zone Rule wording:

The vaulter must land so that he or she does not touch, or go beyond a 3" wide caution stripe painted in a contrasting color on the top of the landing system. The "Caution Box" shall be 16′ wide x 14.5′ deep beginning at the zero line (top of the back of the box). Maximum and minimum standard settings hash marks 12" long shall also be clearly visible on the top of the system. Additionally, the zero line shall also be marked across the entire landing system including the standard base padding and the sloped sections of the front buns which surround the plant box.

Below: Suggested Caution Zone markings (red outside lines) 16′ wide and 14.5′ deep starting at zero. In fact the caution box is the same size as the old ASTM landing system specification pre 2003.

Enforcement

During the course of competition, In the completion of a pole vault jump - the vaulter must land so that his entire body is within a 16’ wide x 14.5” caution box beginning at the zero line defined by the top of the strike plate. The vaulter may not touch the caution line with any portion of his or her body.

Vaulters who do not land within this area will be awarded a yellow caution flag, three caution flags during the course of a contest result in elimination with the vaulters best height cleared as his final mark.

Three consecutive misses remain as the other way the vaulter may be eliminated. (So now we will have two methods for elimination.)

Examples of dangerous vaults which would result in caution flags…

* Any vaulter landing on or in front of the zero line shall receive a caution flag.

* Any vaulter landing or touching the side lines or the back line of the caution box shall receive a caution flag.

* All vaulters landing in the plant box shall receive a caution flag.

* All foot landings resulting in a back or side roll where the vaulter contacts a side or rear caution line shall receive a caution flag.

* All vaulter landings upon the standard base padding shall receive a caution flag.

* All hand slips resulting in landings in or around the plant box area, where the vaulter makes contact in front of, or on the zero line should receive a caution flag.

Draft Proposal

National Pole Vault Safety and Education Foundation

"License to Vault"

By Jan Johnson
Sky Jumpers Vertical Sports Club/ Cuesta College/ Atascadero HS

Mission Statement

A comprehensive, systematic program of pole vault education and testing for the purpose of improving the general state of pole vault knowledge among athletic directors, coaches, athletes, risk management personnel, parents and officials. Additionally, a teaching system designed to improve the methodology of reduce at risk vaulters and coaches who subscribe to the over gripping and over bending the pole mentality.

Program Overview

All domestic pole vaulters and coaches at all levels must have a license to pole vault, or to coach pole vaulting. The cost to the participants will be only $10 per year. The program will include 6 levels of training and testing, and 3 additional levels of membership as outlined below.

The foundation will potentially employ several people whom are in charge of the key areas of administration including: curriculum, promotions, news letter, and web site development.

A board of trustees will oversee the program which will entirely self fund from licensing registrations and donations. The trustees will represent each of the ruling bodies, the manufactures, the ASTM pole vault sub committee, and the USATF officials association.

The most important aspect of the program will be a national e-news and information letter which will be free of charge to anyone who wishes to register for it. The information in this letter will also be posted on the foundation's web site and sorted into categorical areas for study. Those who wish to obtain their license to vault or coach will then be tested from a combination e-news materials and a standard curriculum pertinent to each of the 6 tested levels. To advance to the next level of licensing; each year vaulters and coaches must pass the test associated with their new level. In this way we are assured that our vaulters are studying and showing intellectual progress each year as they develop.

It is estimated that between HS and College coaches, athletic directors, and facilities risk management personnel; approximately 80,000 people will get the early versions of this e-letter. This number will of course grow rapidly as the licensing registration process takes hold. It is estimated that the potential reach is in the neighborhood of 500,000 people by the time you include parents, vaulters and interested parties who could register.

Each level of testing will require approximately an hour of actual test taking time, assuming the participant has studied the e-letter materials, or the web site through out the year.

All licensed vaulters and coaches must also agree to, and sign a pole vault participation waiver saying that they understand the sport of pole vaulting is dangerous but that they agree to use safe methods and judgment in their participation ect..…..

Upon completion of his or her licensing the participant shall receive an emailed license verification for the year in which he or she registered. Additionally, a complete directory of all licensed pole vault coaches and athletes will be available to the public at the foundation web site. This list will be searchable by name, state, school/ affiliation, level of certification and first three digits of registration zip code.

Purposes

1. To grow the sport of pole vaulting in the USA
2. Reduce liability to the sponsors of pole vaulting. (schools, coaches and other facilities owners)
3. Improve safety for pole vault participants
4. Provide funding for the research of pole vault projects
5. Provide funding for selected domestic competitions
6. Improve officiating
7. Improve competition rules
8. Influence all US pole vaulters and PV coaches regarding the proper skill sets and methodology (especially reading pole vault jumps and making adjustments for safety and performance)
9. Influence all head coaches, athletic directors, and school safety personnel regarding pole vault use areas.
10. Influence all PV officials regarding good officiating methods, as well as proper enforcement of the rules for safety and efficiency.
11. Increase number of USA medalists at the World Championships and the Olympic games by increasing the size of our talent base and improving our at large knowledge base.

Funding

The project can be entirely self funding if we have the cooperation of all three ruling bodies and some of the state HS associations. It appears possible that sufficient funds can be developed from registrations and donations as outlined below to fund such an aggressive project.

1. Income sources: On line registration and testing: $10 per year per participant.
2. Donations to our not for profit web site: (estimated $5,000 first year).

Note: There are currently approximately 10,000 PV coaches, 80,000 HS vaulters, 6,000 college vaulters and 500 open vaulters in the United States each year.

First year Budget - $200,000
Second year Budget - $400,000
Third year Budget - $800,000
Potential Annual Budget - $3,000,000

Expenses Year one (based upon income)
Salaries
Travel
Web
Bulk Email
Testing
Event Sponsorships
Awards

Strategic Plan
All pole vaulters and pole vault coaches in order to compete in USATF, HS or NCAA meets must have a license to coach or vault. All USATF Pole Vault Clubs, High Schools, and Colleges, must have a certified person conducting practice or coaching at meets. Additionally, all pole vault participants each year must show progress in the key pole vault related topics prior to first competition each season. It is suggested that high school and college beginners be allowed a 30 grace period beginning from the time of first allowable competition respectively .
The materials for testing will be developed by the foundation curriculum board with the over sight of the board of trustees and the executive director. These materials will be a combination of web site information, and e-news letters arranged into categories relevant to each level of testing. These shall include a combo of written materials, sequence photos and video clips with instructional comments.
Those wishing to obtain a current license will have to pass an on-line self grading multiple choice exams over each portion of the curriculum. Once the participant has passed all six levels he or she becomes a lifetime member.

Level 1 License - Beginners Curriculum Pt 1 (yr 1)
* 2 weeks until the first meet progression
* Technique systems necessary for clearing a bar 12” under your grip
* Basic concepts of safety in the pole vault
* Pole Speed and its relationship to success.
* Staying behind the pole for safety and skill
* Finding steps and proper hand holds
* Drillz for skillz requiring no equipment
* Using the PLZ for skill development
* The “Beginning Drill Sequence” with emphasis on the learning of basic technique and advancement to higher hand holds and longer runs.
* Using the take off mark

* Care of pole vaulting equipment
* Things pole vaulters should bring to bring to a track meet

Level 2 License - Beginners Curriculum Pt 2 (yr 2)
* Technique necessary for clearing your grip
* Pole vault safety as it relates to reading pole speed
* The relative stiffness between different sizes of poles
* Applying and using the pole vault safety zone
* Basic PV mechanics for sound technique
* Hand grips to cross bar relationships- clearing more over your grip
* Pole vault history pt 1
* Feet to hands
* Key skill building imitative exercises and drills requiring little or no equipment
* Pole Vault for dummies

Level 3 License - Intermediate Curriculum Pt 1 (yr 3)
* Technique necessary for clearing a bar 6" over your grip
* Pole vault energy equation dynamics explained
* Pole vault safety and as it relates to pole and grip selection
* Using the mid mark for safety and efficiency
* Pole Vault History pt 2
* How to conduct a PV contest
* setting standards for maximum efficiency
* Over bending and its potential consequences
* Feet to hands and shoot the turn

Level 4 License - Intermediate Curriculum Pt 2 (yr 4)
* Technique necessary for clearing bars 12" above your hand hold
* Pole vault History Pt 3
* The importance of consistent mid-marks and take-offs
* Developing an efficient run and planting system
* Weight Training methods
* Essential Gymnastics
* Developing a jump up at take off
* Rhythm builders

Level 5 License - Advanced Curriculum Pt 1 (yr 5)
* Technique necessary for clearing bars 18" above your handhold
* PV history pt 4
* Elite training concepts, Basic Gymnastics of stunts and methodology
* Strength for body weight Indexing
* Speed training mechanics and drills
* Jumping higher with less jumping and more training
* Importance of short run vaulting for technique

Level 6 License - Advanced Curriculum Pt 2 (yr 6)
* Technique necessary for clearing bars greater than 24" above your hand hold.
* Metabolic systems
* Advanced Gymnastics
* Drugs
* Restoration/regenerative methods
* Building Facilities / ASTM specifications
* Technique examples of Elite jumps
* Crashes
* Cause and effects
* Training systems
* PV history pt 5

Level 7 License - Lifetime members
Qualification**:**
Have passed all 6 levels and have six years of participation in the sport.

Level 8 License- Honorary members (Donors)

Level 9 License- HOF members, Grandfathering?

National PV Safety and Education E-News Letter (40- 50 issuers per year) potential exposure 500,000 people
Topics:
History
Safety
Records
Facilities
Training
Technique
Special Equipment
Care Of Equipment
Rules
Lists
Traditions
Team-mates ship
Coaching tips
Mysteries
All previous E Newsletters are also posted at the National Web site and relate to test questions

Organization and Administration

Board of Directors: (4 yr terms)
1 NCAA representative
1 USATF representative

1 NFHS representative
1 ASTM representative
1 USATF official representative
1 vaulting pole manufacture representatives
1 landing pad mfg representative

Executive Director (2 year renewable)
Deputy Administrator
Web Content administrator
Customer Service

Content Development Committee (4 year renewable terms)

1 HS coach
1 HS coach
1 HS coach
1 College coach
1 College coach
1 Club coach

Sponsorships:
Speakers Bureau for Clinic presentations
Domestic Event Sponsorship
Prize Money
Research and Development
Travel
Annual Awards
Endowments

Special Projects:

ASTM International Standard for Weight Value assignments and maximum hand-holds on vaulting poles - based on user body weight and ability levels. .
ASTM Box Collar Specifications
ASTM Pole Vault Use Area Specification (will include specifications for runways, runway markings, landing systems perimeters, walls, fences, curbs, concrete, sand pea gravel, wood chips, under-layments, perimeters PLZ, side line or runways flags, plant boxes, shock attenuation values).
ASTM Plant Box Specifications ~ The size, positioning, and shock attenuation values for the bottom of the plant box.
ASTM Landing System Specifications ~ Size, shock attenuation values for the various parts landing system parts, PLZ, Caution Zone size, standard base padding,

Potential Rules Changes Which Influence Safety and Costs (most important in italics)

Rules Standardization ~ the officiating of pole vaulting is made more difficult because we have three sets of rules and much variation on some important issues, the IAAF is no friend to USA pole vaulting in this regard.

Rules changes requiring runway calibration ~ all runways must be marked in min 5', beginning 5 feet from zero and extending to 130 minimally. All runways must have mid-mark and take of distances, all individual step markers should be written in playground chalk. No marks are allowed more than 4" from the outside edges from the runway. All mid-marks must be visible from the coach's area.

Rules changes regarding gloves and taping the pole, and hands. Let the vaulter choose his/her own method of securing the best grip on the pole. Continue to ask for uniform tape thickness and no knobs on the pole.

Rules changes regarding dangerous jumps ~ employ the 16' x 14.5' pole vault caution zone specification a system of warning flags as a portion of pole vault judging.

Rules changes regarding plant box padding (Reduce Force Impact by over 90%)

Return the peg length back to 3" ~ Lobby the IAAF to do the same.

Abandon HS Weigh-ins, and pole check-ins, and simply have coaches and athletes sign a doc that says they are using appropriate poles for their body weights and handholds. Allow multi weight poles that meet the ASTM specification for weight assignments and hand holds.

Rules changes regarding handholds and weight assignments will help our school systems reduce the costs of pole vaulting by allowing more users per pole. Maximum hand hold on specific poles will be determined the users body weight. For those manufactures who wish to participate and follow the proposed ASTM specification ordering poles will become simpler and more effective

Standardized Rules regarding the taping of the pole and gloves

Standardized Rules regarding warm up limitations after several passed heights

Standardized Rules regarding dangerous conditions rules for contests

Risk and Cost Analysis Regarding Plant Box Safety

By Jan Johnson

Thanks to the following for your timely help and support with this project:

Jason Shimko - Get Vertical PV Club, Milwaukee, Wisconsin, Peter Mcginnis, Ph.D. - SUNY Cortland, Eddie Seese - Former Chair ASTM PV Sub Committee, David Spease -NPSI certified Play Ground Inspector, Will Freeman - Head Track Coach Grinnell College, Kevin Ferguson – PV Coach Henderson Ky. HS, Dr Russ VerSteeg - New England School of Law, Sky Jumpers Connecticut PV Club, David Nielson Idaho State University, Mike Yurcho – Cal Track and Engineering. Dr Barry Boden, The Orthopedic Center, Rockville, Maryland, and David Hodge - Gill Athletics

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P1 Introduction- Head Injury Criteria of Padded and Unpadded Box Surface Comparisons
P2 Mechanism and Result Serious and Catastrophic Injuries
P2 Landing Pad Schematic
P Head Injury Risk Probability Curves
P5-8 Catastrophic Injuries in Pole-Vaulters: A Prospective 10-Year Follow-up Study
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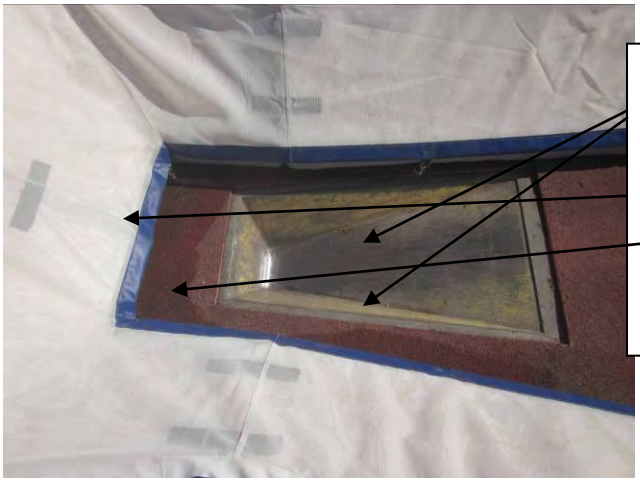
Introduction:

Although major injuries in falls off the side of landing pads, and off the rear are now far less common than in the past. Box landings resulting in Major injuries are still very common. The purpose of this project is to better protect the pole vault plant box area and thus improve the safety of Pole Vault Participants in the future. Unfortunately over the past ten years we have had a large number of plant box landings result in serious injury.

This document shows how much protection can be increased and what the expected costs and benefits will be. Several methods and combination of methods are currently available for this purpose.

Below: This college facility has approximately 1728 sq inches of poorly or unprotected surfaces. I have labeled the approximate HIC values from our testing for each location. Keep in mind that in the probability chart at the bottom of this document tells us that fatal head injuries begin at approximately 1000 HIC, 3000 Hic is almost always fatal. See Chart at the end of this document to better understand HIC and its relationship to head injuries. .



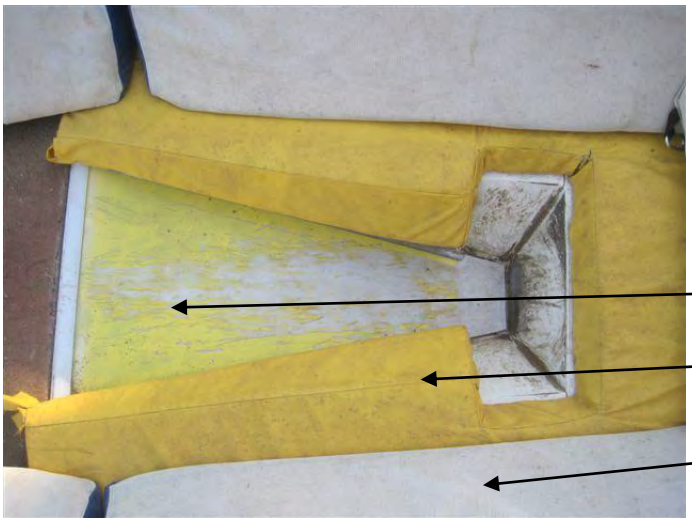
Potential Force Impacts:

10,000 HIC Pole Slide and Inner Walls

4500 HIC Interior Edges of Front Buns

8500 HIC Runway Surface behind box

Below: with box collar and Soft Pan only 112 sq inches of unpadded surfaces remain, 94% are padded well beyond the 1000 HIC threshold where research shows serious injuries begin. (See HIC probability chart below for more information.) Many ways exist to improve the protection in this area. Total costs to retrofit the plant box as show below is less than $1000. More expensive methods also exist.



Potential Force Impacts:
Soft Pan and Collar together Protect 94% of the poorly protected or unprotected surfaces shown in the top photo.

314 HIC Pole Slide

750 HIC Interior Walls

500 HIC inside Edges of Front

Estimated Methods Costs and Benefits:

Box Collar with Interior Side Wall Padding: $500 parts, Labor $0 Install works with any plant box to increase protection, can be carried from facility to facility. (pads 65% of currently unprotected surfaces, works in any box, when combined with GreyBack or SoftBox padding will exceed ASTM specification by 50%) Available immediately, could be the first of two part upgrade over the next couple of years.

GreyBack Box:. Not yet on the market. Projected cost $1400 parts, labor $500 works best with collar as show above, still leaves a hard rim, put softens inner side walls more, fits into the same sized hole as the old box. Pads 98% of unprotected surfaces.

New SoftPan Box: $1400 parts plus labor $500, (pads bottom pan or 30% of unpadded surfaces could be second of two part upgrade, fits directly into the same sized hole the old style box come out of)

New Box: $2500 parts plus Labor $2000, (Pads 100% of all hard surfaces, can't work in many facilities, may have deformation problems)

Retrofit existing Plant Box #1 Method: Parts $150 Labor $200, Replaces bottom of box with desired padding and plastic in a simple way. Any program with a maintenance dept can install in 2 or 3 hours.

Retrofit existing Plant Box #2 Method: Parts $200, Labor $200, Replaces bottom of box with desired padding in a simple way. Any program with a school maintenance dept can install in 2 or 3 hours.

Polyurethane Box: Parts (estimated) $500, Labor (estimated) $1000**,** currently is in the early phase of testing but showing some promise. Force impact capabilities unknown**.**

Foam Core Box: Costs unknown, currently in design phase, holds great promise, Force impact capabilities showing great potential.

Pole Vault Helmets: $60 each**, not very** protective, approximately 4x less protective than our *proposed* ASTM specification for box padding. 10x less protective than pole slide testing. Long term costs look high when you consider number of participants per school and replacements each year.

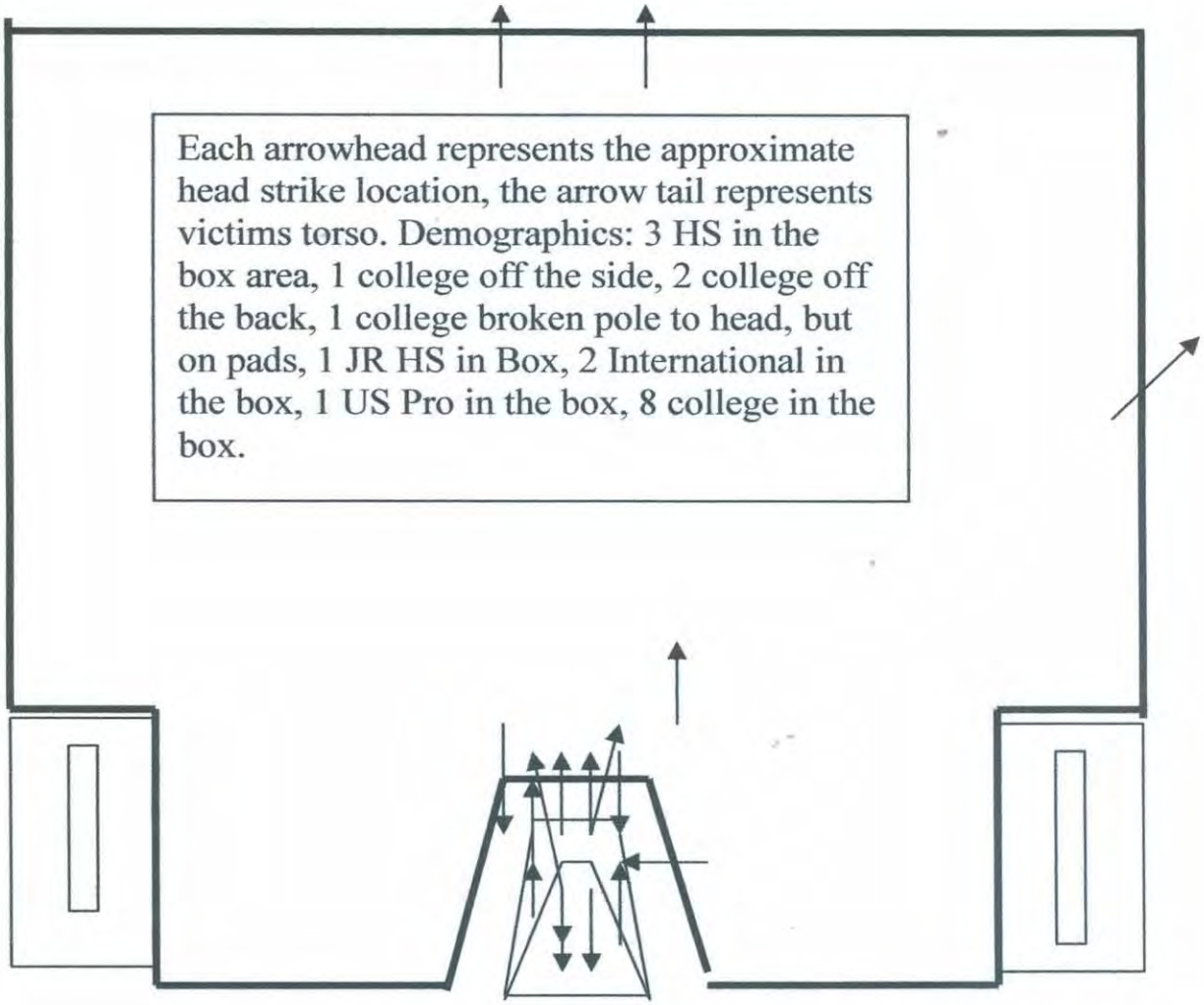
*** Note that we have instructional videos available for all installs and retrofits.

Protec PV Helmet
6830 HIC, tested 9x less protective than SoftPan prototypes.

Mechanism and Result of Serious and Catastrophic Plant Box Landings

Box Area Accidents Only 1971-2011	
Mechanism	
Hand slip off pole and head hit in box	**8**
Stall and land short in the box	**25**
Total	**33**
Skull Fractures	**22**
Temporary paralysis	**1**
Permanent paralysis	**3**
Fractured vertebrae	**7**
Severed Brain Stem	**3**
Broken or separated shoulder	**4**
Fractured Tailbone	**4**
Punctured lung	**5**
Broken Ribs	**5**
Broken hip	**2**
Broken Pelvis	**1**
Death	**7**
Box Area Accidents 2003-2011	
Mechanism	
Hand slip off pole and head hit in box	**5**
Stall and land short in the box	**9**
Total	**14**
Skull Fractures	**5**
Temporary paralysis	**1**
Permanent paralysis	**1**
Fractured vertebrae	**5**
Severed Brain Stem	**0**
Broken or separated shoulder	**2**
Fractured Tailbone	**3**
Punctured lung	**3**
Broken Ribs	**3**
Broken hip	**1**
Broken pelvis	**1**
Death	**1**

Fall Locations Resulting in Serious or Catastrophic Injuries 2003-11



X1. INJURY RISK CURVES

X1.1 Most of what is known about the relationship between impact magnitude and head injury risk comes from experiments using cadavers and human volunteers subject to high accelerations and impacts under laboratory conditions. The data from these experiments form the basis of automotive and aircraft impact protection standards. There has been no research directly relating the magnitude of an impact from a playground fall to the severity of the injuries sustained. We, therefore, rely on data from automotive industry experiments to provide insights into injury risk.

X1.2 Fig. X1.1 shows the probability of different degrees of injury occurring as a result of impacts with a given HIC score. These "Expanded Prasad/Mertz Curves" are based on data from cadaver experiments in which the relationship between

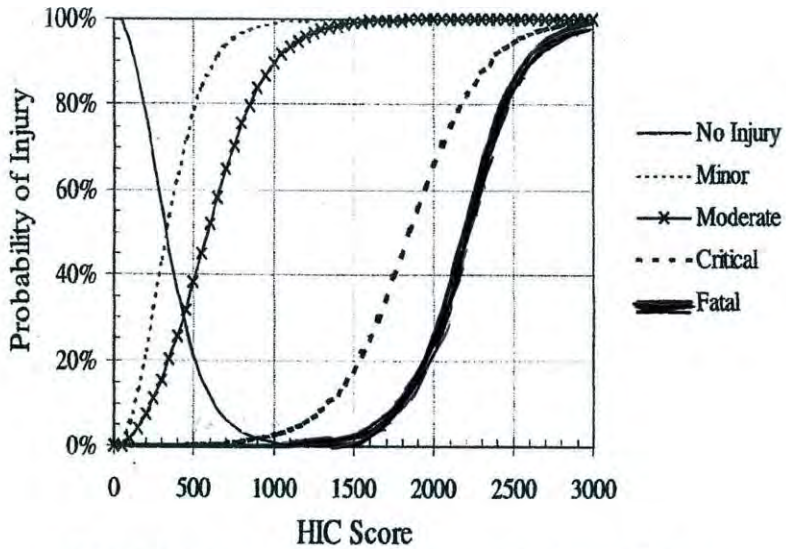


FIG. X1.1 Probability of Specific Head Injury Level for a Given HIC Score

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HIC scores, skull fracture, and brain damage were observed.[6,7] The two solid curves in this figure show the probabilities of no injury and of fatal head injury. Broken lines show the probability of minor, moderate, and critical head injuries, defined as follows:

X1.2.1 *Minor Head Injury*—A skull trauma without loss of consciousness; fracture of nose or teeth; superficial face injuries.

X1.2.2 *Moderate Head Injury*—Skull trauma with or without dislocated skull fracture and brief loss of consciousness. Fracture of facial bones without dislocation; deep wound(s).

X1.2.3 *Critical Head Injury*—Cerebral contusion, loss of consciousness for more than 12 h with intracranial hemorrhaging and other neurological signs; recovery uncertain.

X1.3 As an example of how Fig. X1.1 is interpreted; if a person experiences a head impact equivalent to a HIC score of 500, there is a 79 % chance that they will suffer a minor injury. At 38 %, the risk of a moderate injury at this HIC level is also significant. The risk of this impact producing a severe or fatal head injury is very low, however. It is also notable that the chance of experiencing a 500 HIC impact without suffering an injury of any kind is only 21 %.

X1.4 *Discussion*—HIC injury risk curves should be interpreted cautiously in the context of injuries resulting from playground falls. The data on which the Prasad/Mertz Curves are based are from adult cadavers subjected to frontal impact. The extent to which this data is valid for children experiencing non-frontal impacts to the head is not known. Also, a rigid missile such as that specified by this specification produces HIC scores that are somewhat higher than those generated by a cadaver or a headform with lifelike properties.[8] HIC scores determined in accordance with this specification will overestimate the probability and severity of head injury if they are interpreted using Fig. X1.2, will tend to be overestimated. Consequently, the criteria established by this specification are more conservative than if a lifelike headform were used. The more conservative criteria are warranted by the absence of specific data for the head injury tolerance of children falling from playground equipment and by the fact that the limiting HIC score of 1000 is set at the threshold of fatal injury risk. As the Prazad-Mertz curves show, a 1000 HIC criterion limits the probability of a fatal injury, but still infers a significant risk of severe, non-fatal injury. The probability of experiencing a 1000 HIC impact with no injury is very low (less than 1 %).

[6] National Highway Traffic Safety Administration (NHTSA), Department of Transportation., 1997, FMVSS201, Head Impact Protection, 49 CFR 571.201.

[7] Prasad, P. and Mertz, H. J., "The Position of the United States Delegation to the ISO Working Group on the Use of HIC in the Automotive Environment," *SAE Paper No. 851246*, Society of Automotive Engineers, Warrendale PA, 1985.

[8] Saczalski, K.J., States, J.D., Wagar, I.J, Richardson, E.Q., A Critical Assessment of the Use of Non-Human Responding Surrogates for Safety System Evaluation. SAE Paper # 760805, 1976, Society of Automotive Engineers, Warrendale PA.

Catastrophic Injuries in Pole-Vaulters: A Prospective 10-Year Follow-up Study

SUPPLEMENT NO. 66

(Digested Version of Findings)

Barry P. Boden, MD*, Matthew G. Boden*, Rebecca G. Peter, Peter M. McGinnis**, Fred O. Mueller, PhD***, and Jan Johnson MS. ****

From *The Orthopaedic Center, Rockville, Maryland, **State University of New York Cortland, New York, the ***Department of Physical Education, Exercise and Sport Science, the University of North Carolina at Chapel Hill, North Carolina, and the ****National Pole Vault Safety Committee, Atascadero, California

Counts: words (max 6000), references (max 60)

[§]Address all correspondence and reprint requests to: Barry P. Boden, MD, The Orthopaedic Center 9420 Key West Ave. #300 Rockville, MD 20850 (301-424-1287; fax 301-424-5266; bboden@starpower.net).

Risk Comparisons

Pole vaulting ranks as one of the most dangerous activities according to the NCCSIR (Thomas, Mueller). In one report that analyzed the US National Registry of Sudden Death in Young Athletes for a 30-year period from 1980 to 2009, pole vaulting had the second highest number of direct, traumatic fatalities after football (Thomas). For the 28-year period from the fall of 1982 through the spring of 2010 the NCCSIR reported 1284 direct catastrophic injuries in high school and college sports (NCCSIR, 28th report). During the same time period the direct catastrophic injury rate per 100,000 athletes for all sports combined was 0.62 and 2.54 for high school and college respectively (28th report). After the 2003 rule changes the direct catastrophic rate of 0.7 (1.1 in males and 0.28 in females) per 100,000 high school pole vaulters is comparable to the overall catastrophic incidence for all high school sports. However, the rate of direct catastrophic injuries in college pole vaulters after the 2003 rule changes (28.4 overall, 42.3 for males and 11.8 for females) was significantly higher than the combined rate of 2.54 for all college sports and indicates that more emphasis needs to be placed on preventing pole vaulting injuries at the college level. For male college athletes, pole vaulting (42.3) has the highest rate of catastrophic injuries per 100,000 participants, followed by gymnastics (25.05), ice hockey (10.22), football (9.5) and lacrosse (5.28) (NCCSIR, 28th report). For female college athletes, skiing (12.46) has the highest rate of catastrophic injuries per 100,000 participants, followed by pole vaulting (11.8), equestrianism (10.85), gymnastics (4.8) and ice hockey (4.71) (NCCSIR, 28th).

Accidents off the Side and Off the Rear

All four of the athletes that missed the sides or rear of the landing pads after the 2003 rule changes were participating at the college level and landed on hard surfaces, FieldTurf in two cases, concrete with a thin layer of rubber in one case, and a rubberized gym floor in one case. Two of these cases were in noncompliance with the 2003 rule changes by using a landing pad that didn't meet the minimum size requirements or substituting multiple high jump pads for a pole vault landing pad. In 2003 the NFHS also mandated that all hard surfaces surrounding the sides and back of the landing pad should be eliminated or covered with a minimum of 2 inch thick unspecified soft padding (ref). The NCAA has no similar rule. If the NCAA mandated a similar rule requiring soft surrounding surfaces and all schools were in compliance there may have been no catastrophic injuries from athletes missing the sides or back of the landing pad after 2003. None of the catastrophic injuries in the current or prior study occurred due to an athlete landing on a surrounding surface of grass, dirt, sand, wood chips, or 2" of padding ( ). We recommend strict adherence to the minimum landing pad dimensions and that the type of padding on the surfaces that surround the sides and back of landing pad be specified and at least two inches of padding be used.

Vault Box injuries

While the number of catastrophic injuries from athletes missing the sides and back of the landing pads decreased compared to the prior study, the number of injuries due to athletes landing in the vault box more than tripled from 0.5 per year in the earlier study (ref) to 1.7 per year in the new study (1.55 after the 2003 rule changes) and is cause for concern. The vault box survey also indicated that a large percentage of pole vaulters landed in the vault box during their career. In fact 84% of those surveyed landed in the vault box at least once and 7% landed in the vault box at least four times. Most of these pole vaulters were relatively inexperienced, amateur pole vaulters with an average personal best of ten feet and two years of vaulting experience. It is difficult to determine if these numbers plateau, increase, or decrease with more years of vaulting. The number of vaults would likely increase the chances of landing in the vault box, but the additional experience may reduce the incidence of box landings. It is likely that elite pole vaulters land less frequently in the vault box, but have more catastrophic results when they do land in the vault box ~~from the higher heights attempted~~ because they are falling from a

higher height. It is also possible that the elite and college pole vaulters use a higher grip on the pole and therefore have a greater likelihood of a vault box landing since the execution of their takeoff speed and technique are so critical to getting the pole to rise to vertical.

Despite the survey revealing a high percentage (84%) of at least one vault box landing in inexperienced pole vaulters, there were only a small percentage of injuries requiring medical attention. Medical attention was required in only 3% of the 2,804 participants that landed in the vault box at least once. This may be explained by the fact that most jumps being attempted at the time of injury in the survey were less than ten feet so the athletes impacted the box or ground with less force on landing. The majority of injuries occurred to the ankle, heel, lower back, and knee. Although this report focuses on catastrophic pole vaulting injuries it is clear from this survey that numerous non-catastrophic injuries occur each year from pole vaulters landing in the vault box. Based on the number of participants in the survey and their average number of years pole vaulting, it can be extrapolated that there may be over a thousand non-catastrophic injuries requiring medical attention each year from pole vaulters landing in the vault box.

High School Weight Rule

There are several possible explanations for the increased rate of catastrophic injuries from athletes landing in the vault box. In 1995 the NFHS rules committee mandated that "the vaulter's weight shall be at or below the manufacturer's pole rating." (ref 1994 NFHS). This rule was instituted to encourage athletes to use a lower hand hold which should reduce the bend of the pole and lower the risk of control problems such as over-shooting the landing pad, landing off the sides, landing in the vault box, and/or the pole breading. However, the stiffer poles are more resistant to rotating to vertical. If the resistance of the pole is too great for the amount of take-off energy the pole vaulter produces, the pole vaulter become stranded and may land in the vault box. In many cases the athlete reported changing to a stiffer pole prior to the catastrophic jump in an attempt to clear a higher vault. The stiffness, length, and weight of the pole as well as the grip height in relation to the athlete's height, weight and attempted vault height requires further research to determine safe standards.

The high school and college rules state that the front edge of the vault box shall not extend above the grade of the runway. Nonetheless, one athlete reported a warped front lip that disrupted the planting position of the pole and contributed to the injury. The front lip of the vault box needs to be carefully checked before attempting any vaults. Another situation that may place pole vaulters at risk for injury is the practice of tapping or having an assistant give the pole vaulter an upward push at takeoff to help clear the bar. With appropriate supervision this practice may be beneficial during training but can lead to a false sense of security during competition when tapping is not allowed. Pole vaulting is a complex sport and coordination of speed and power of the athlete as well as timing of pole release, in particular avoiding early release, are skills that requires significant practice, expertise, and coaching.

Effects of Deeper Standard Settings Rules

In 2003 the new high school and college pole vaulting rules also mandated that the crossbar be moved away from the vault box and further over the landing pad in an attempt to reduce vault box injuries. It is unclear why this rule change was not effective, but perhaps pole vaulters are having a harder time reaching the crossbar with a higher risk of being stranded over the vault box. The ideal position of the crossbar requires further research.

Box collar

Many pole vault landing pads do not adequately cover the area immediately surrounding the sides and back edges of the vault box. The design of these landing pads allows the pole to bend without contacting the landing pad. After 2003, the NHFS required that these exposed surfaces be covered with "a minimum of 2-inch dense foam padding (box collar)" (ref) and the NCAA required that these surfaces be covered with "a collar of 2 to 4 inches of padding of uniform thickness" (ref). Many of the vault box injuries in this study occurred when the athletes landed on the exposed surfaces surrounding the sides or back edges of the vault box or a combination of these surfaces and the vault box. In spite of the NCAA and NFHS rules requiring padding in the box collar, many facilities were in noncompliance at the time of the catastrophic vault box injury in this study. One of the authors (JJ) has also noted a high rate of noncompliance with this rule at track and field meets. Therefore, strict enforcement of this rule is important, and specific standards regarding the shock attenuation capabilities of the padding used in box collars is needed.

Preventative Strategies

There are several preventive strategies for reducing the number of vault box injuries. Enforcing the existing rules requiring the box collar use is a critical first step and writing standard specifications for the box collar and its shock

absorbing capabilities is next. The use of newer materials that allow greater force absorption than the standard 2 inches of unspecified dense foam should be investigated. In addition padding the sides of the vault box as well as the bottom of the vault box may be beneficial. A one piece box collar that also extends down the side walls of the vault box would not only pad the side walls of the vault box but would also hold the box collar padding in place better. The current vault box dimensions require a flare on the sides and rear to allow space for the fiberglass pole, introduced in the 1960s to bend and rotate to vertical. However, the flare on the sides of the vault box significantly increases the area of exposed hard surface in the vault box and may not be necessary to allow the pole to bend like the rear flare in the box. Eliminating the flare on the sides of the vault box or padding the interior side walls would reduce the hard surfaces and potentially diminish the risk of injury. Another change that may reduce or eliminate vault box injuries is making the vault box narrower. Requiring spotters near the vault box has been proposed but is controversial, as they may be at risk of injury from the pole or from impact with a descending athlete.

Participation Levels Indicate Higher Risk in College than HS

In contrast to the level of participation (78% high school and 9% college) at the time of the injury in the prior study on catastrophic injuries, this study identified a higher percentage of injuries in college athletes (52%) compared to high school (40%) pole vaulters (ref). If participation numbers are accounted for, the incidence per 100,000 pole vaulters was over 12 fold higher in college versus high school pole vaulters. Similarly the average age of the injured athletes in this study was 19 years old versus 17.5 years in the prior study (ref). The older age of the injured athletes in the current study may be due to the larger landing pad preventing injuries in high school athletes, whereas older college athletes are attempting higher vaults, gripping the pole higher, and may be stranded over the vault box. Unlike the prior study which reported no injuries in female athletes, this study identified 3 injured female athletes. This is likely due to the increased participation by females in pole vaulting over the past decade.

Elite Vaulters Carry the most Risk

According to the National Pole Vault Safety Committee there have been eight catastrophic injuries in the 350 male athletes in the United States who have pole vaulted over 17’6 ¾” (5.35m)since 1971 for an incidence of 2,286/100,000 or 2.3 per 100 pole vaulters. There was one catastrophic injury in the 55 US women pole vaulters who have cleared 14’ (4.25m) for an incidence of 1818/100,000 or 1.8 per 100 pole vaulters. Many of these athletes were injured while vaulting in competitions governed by USA Track and Field (USATF) rules or International Association of Athletics Federation (IAAF) rules. Neither the USATF nor the IAAF requires the use of a box collar or the padding of hard surfaces surrounding the landing pad. The higher catastrophic injury rate for these elite or semi-elite pole vaulters is primarily related to the higher heights from which the pole vaulters are falling. However, the high injury rate may also be related to the less stringent USATF and IAAF rules regarding padding of hard surfaces and the use of a box collar, at least for those injuries occurring after 2002.

CONCLUSIONS

The 2003 rule changes that mandated a larger landing pad have significantly reduced the number of catastrophic injuries from pole vaulters landing off the back or sides of the landing pads. However, the annual rate of catastrophic injuries from pole vaulters landing in the vault box has tripled over the last decade and remains a significant problem. Potential preventive strategies that require further research include developing standard specifications for box collars and requiring the use of box collars that meet these standards, improving box collar shock absorption materials, padding the sides of the vault box as well as the bottom of the plant box, and making the vault box narrower. The dimensions of the pole, especially the stiffness and proper grip heights, as well as the ideal position for the crossbar also require further study

Examples and Prototypes

Since 2003 we have studied the problem of padding the pole vault plating box for the purpose of increased safety.

Our project goal was to totally eliminate all serious and catastrophic falls into the in Plant Box.

Soon the ASTM will no doubt finalize minimum specifications for both Plant Box and Plant Box Perimeter Padding. These specifications will in fact, not mandate any one kind of padding or brand, but rather they will simply describe the *Voluntary*

Consensus Industry Standards for dimensions and shock attenuation capabilities of pole vaulting equipment. They will also describe the test methods necessary to validate their capabilities.

Above Left: Traditional Plant box pole slide outlined on SoftPan retro fitted box with card board insert.
Above right: What passes for legal since 2003 is in fact poor.

Above Left and Center: Is their any question that the one on the left is safer? Above Right: Atascadero Retro fit Soft Pan

Above: Typical box landing position directly on to top edge of the box. This position usually results in fractured ribs or pelvis and or punctured lung.

Above: Padding 3” thick also reduces impact greatly along to edges of the pole bend area.

Above: This type of “short landing” often results in fractured tail bone, pelvis or spine. Reducing the potential impact by 93% would obviously have a huge effect on the outcome.

Above: Head and shoulder protection is also a priority.

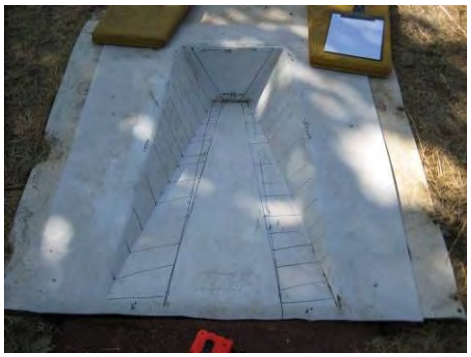
Above: Force impact is greatly reduced since the wings into the box help reduce the amount of space to fall into. The narrower it can be made the safer it is.

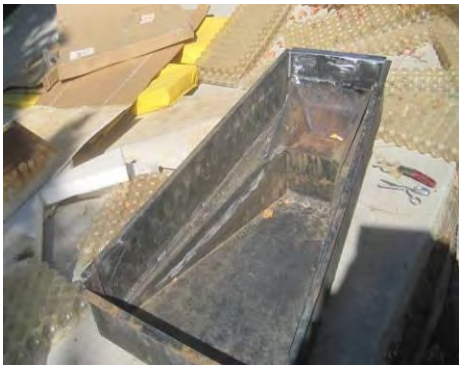
Above Left: GreyBack proto-type with padded bottom and sides. Not on Market yet Anticipated cost of parts $1200.
Note: All metal Strike Plate reduces chances of pole slipping out of the top on “late tip type plants” so common among beginners.
Above Center: Gill Soft Box showing wear and strike board deformation.
Above right: Soft Box Padding Cavity.

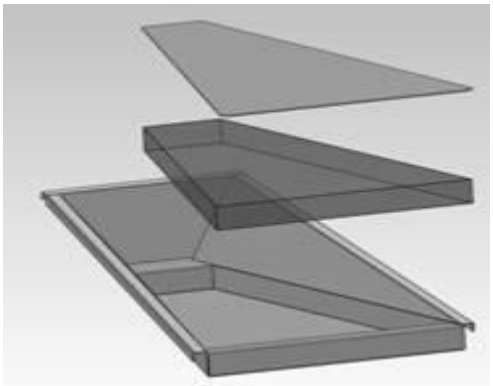
Above: Early SoftPan proto-type box with metal coping to prevent lip on front edge of box. On any retro fit this is a must.

In this version we simply glued the bottom of the pole slide to the ledge where the pole rotates. Two yrs and over 150,000 jumps later it’s still going strong. However, since this development we have found three other ways to retro existing boxes with out screws or glue. These will represent a huge savings to our schools.

Below: A few more glimpses of some of our Prototypes and experiments.







Jan Johnson
Sky Jumpers Vertical Sports Club
6505 Santa Cruz Rd
Atascadero, Ca 93422
Janjohnson18@charter.net
1805 423-2363

Draft Script for 2011-12 NCAA Wrestling Rules Presentation

This segment of the NCAA Wrestling Rules DVD is to educate and remind the coach, student-athlete, and athletic training and medical staffs about the rules for evaluation and participation status of student athletes diagnosed with injuries and/or skin infections. It will address the basics of skin infections and the rules that govern the disposition of athletes diagnosed with skin problems most commonly associated with the sport of wrestling. It is not intended to be an all inclusive instruction on the identification of skin infections, as this should be left to trained medical personnel. We are hopeful that this information will help all parties involved in the sport realize equal responsibility in the prevention, detection, timely referral and treatment of these problems.

The NCAA Wrestling Rules Committee feels it necessary to address some issues that are specific to the relationship between Athletic Trainers and coaches at NCAA institutions. We recognize that most coaches understand that the athletic trainer is responsible for the health and safety of student athletes and respect the education and skill sets specific to the modern athletic trainer. However, it has come to our attention that sometimes the line of who is responsible for the ~~health care~~health assessment and return to play of student athletes with a possible injury during competitive events gets crossed and decisions for the participation status are being made by individuals other than the athletic medical staff. Coaches and administrators should not place their medical staffs in compromising situations by having them provide inequitable treatment in violation of their medical codes of ethics or state medical practice acts. During injury time outs, it is essential that medical staff be provided ample space and latitude to properly assess the "on mat" condition of an injured wrestler in the short time frame allowed. A member of the

institution's sports medicine staff and or the event host institutions medical staff should be empowered to have the unchallengeable authority to cancel or modify the participation of the student-athlete as he or she deems appropriate.

Comment [d1]: Slight modification from page 11 of SMH.

~~Decisions regarding the evaluation, diagnosis or participation status by coaches, administrators or others in situations other than the activation of an emergency plan or providing first aid when no AT or Physician is innapropriate and is specifically a danger to the health and safety of the student athlete. Athletic Trainers and Physicians are the only individuals that can or should make a diagnosis and subsequent decision about participation status. Coaches or administrators that interfere with the process of evaluation and diagnosis are affecting the qualitiy of health care that can be provided by the athletic medical staff. They may also be placing the student athlete at risk for permanent danger by doing or suggesting proceedures that are inappropriate and in violation of the rules of the sport. Timely diagnosis, or at the very least the evaluation to identify symptomology of injuries is the goal of the athletic medical staff. Coaches and others asking questions of the athlete or athletic trainer during the evaluation process slows the process down and increases the potential for harm to the student athlete. Coaches and administrators should pay attention specific to their job descriptions until invited by the athletic medical staff to assist in the management of an injury.~~ We believe this model will increase the potential wellbeing and health of student athletes at NCAA institutions.

Title: Point of Emphasis: Dehydration Risks

~~It has also been reported that some wrestlers and programs have reverted back to using~~The use of rubber or plastic suits, saunas, steam rooms ~~etc.~~ to dehydrate for making weight is against the

rules regardless of the justification~~. Some programs have justified using these practices because they are used in some countries as a therapeutic modality. To our knowledge, we know of no Athletic Training, Physical Therapy or medical school that teaches saunas, or any other proceedure to stimulate dehydration as part of their cirriculum. Aside from this fact, using these ways to dehydrate to make weight or for any other reason is against the rules of the sport. It is against the rules regardless of the justification~~. Policing and adhereing to the rules of the sport of wrestling is ultimately the responsibility of the student-athlete, coaches and athletic medical staff assigned to the wrestling program. If you, as a student-athlete, coach, athletic trainer or doctor, become aware that an individual wrestler or team members are engaging in unsafe health and safety practices, including cutting weight outside of the designated weight-management protocol, which may lead to poor performance, dehydration, heat illness~~stress~~ and catastrophic outcomes, it is your responsibility to report any such violation to the NCAA National Office. ~~Jim Thornton, NCAA Wrestling Committee Certified Athletic Trainer Liaison, Clarion University of Pennsylvania, 814/393-2456; jthornton@clarion.edu.~~ Penalties may be imposed by the NCAA Wrestling Rules Committee including, but not limited to, public or private reprimand, financial penalty, disqualification of individual contestants, disassociation of the institution's staff member from team activities for one or more competitions, and other penalties as deemed appropriate. Rule 9.3.1 specifically states that "...artificial rehydration (that is intervenous rehydration) are also prohibited, and to allwo participation and use of such is a violation of weight management rules. Violaters of these rules shall be suspended for the competition(s) for which the weigh-in is intended. A second violation would result in suspension for the remainder of the season."

Comment [d2]: Either Teresa or David. Since it is a rule violation it would be best of Teresa to receive the violation. This should be a staff person rather than someone in the membership.

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New section: Title: Skin Infections

Comment [d4]: Combine with other skin infection section

Skin infections and the rules that govern the disposition of student-athletes with these medical conditions ~~issues are~~ can be confusing but are important to ensuring student-athlete health. ~~subjects for athletic training and medical staffs, coaches, student-athletes, rules committees and referees, as well as the parents of the student-athlete~~. The key is education and understanding the nature of these infections, their contagiousness, and the options for prevention.

Report all skin infections to your athletic trainer, team physician or coach. Recognize the signs and symptoms of an infection such as skin sores and lesions that have redness, pain swelling or pus. When in doubt, check it out. Don't treat yourself.

~~The key is education and understanding the nature of these infections, their contagiousness, and the options for prevention.~~

This presentation will address what types of infections are most common, their nature, perspective as to the seriousness of the infection, the rules that surround the disposition of the student-athlete, as well as some basic prevention and possible treatment strategies. For more information about skin infections, refer to the NCAA Sports Medicine Handbook. It should be noted that prevention strategies are helpful but not a fail safe for all occurrences in the outbreak of these types of infections. One of the most important and helpful strategies for identification and treatment of these problems is a team approach in the development of a relationship with a reputable dermatologist or physician that is familiar with the sport. It may be necessary to ~~personally educate~~ inform the physician you have access to ~~regarding~~ about the sport and the rules surrounding disposition of the student-athlete. This can be done by provide the guideline out of the NCAA Sports Medicine Handbook along with the form used for documenting the skin lesion. Of particular concern is the disposition of student-athletes that have been found to have an infection that has not been treated or diagnosed within the rules of the sport when they show

Comment [d5]: Show educational poster.

Comment [d6]: Show graphic of each on the screen

up for competition. If the onsite ~~sports~~ medical ~~team~~ staff identifies an infection during the skin check at weigh-ins, ~~it is imperative that the rules for disposition are followed~~the wrestler must provide a complete Skin Evaluation and Participation Status Form from the treating physician. There cannot be any exception to the rules regardless of the infection. For example, there is only one skin problem that can be covered to allow ~~completion~~competition, and that is ringworm. If a bacterial or viral infection is diagnosed, even if it is in a coverable area on the body, it cannot be covered to allow competition. It is ~~also~~ the responsibility of the host ~~sports medicine~~medical staff to be familiar with the rules and carry them out regardless of the situation surrounding an individual wrestler. These rules are in place for the protection of both the wrestler with the infection as well as the other competitors. The decision made by a physician and/or certified athletic trainer onsite should be considered "FINAL".

A copy of the NCAA Wrestling Rules and Interpretations Book, available online at www.NCAApublications.com (select Rules Books), should be familiar and accessible to coaches and athletic trainers. ~~I encourage athletic trainers to meet with the m~~Medical staff, ~~and~~ coaching staff and~~, as well as the~~ student-athletes should meet together~~,~~ to discuss the rules governing skin infections. This learning process will assist everyone involved when it comes time to make decisions about identification, referral, treatment and disposition of the student athlete.

New Section: Title: Medical Checks.

We felt it is necessary to outline and comment on the rules concerning medical checks, as well as who should conduct them. Recently is has come to our attention that in some cases, medical checks have either not been conducted hours before competition or have been conducted days in advance. Rule 9 in the Wrestling Rules and Interpretations Book covers medical examinations

and states that medical or skin checks shall be conducted on all contestants by a qualified examiner (certified athletic trainer or physician) less than two hours before all tournaments and less than one hour before all meets. It is also important to note that at conference championships and at the NCAA national tournaments, having a dermatologist present is a requirement and is outlined in the championships handbook. The section regarding medical checks gives information on the appropriate student athlete attire, the specific logistics for~~s of~~ medical checks at tournaments, as well as the decision making process for participation status of a student-athlete found to have a skin disease "or any other condition that, in the opinion of the examining physician or certified athletic trainer, makes the participation of that individual inadvisable." Another extremely important section describes the responsibility of the ~~athletic training or~~ medical staff to provide written documentation using the NCAA Skin Evaluation and Participation Status Form. INSERT DIAGRAM OF SKIN FORM

Specifically it states: "If a student-athlete has been diagnosed as having such a condition and is currently being treated by a physician (ideally a dermatologist) who has determined that it is safe for that individual to compete without jeopardizing the health of the opponent, the student-athlete may compete. However, the student-athlete or his or her coach or athletic trainer shall at provide current written documentation from the treating physician to the medical professional at the examination with the approved NCAA Skin Evaluation and Participation Status Form describing (1) the diagnosed skin disease or condition; (2) the prescribed treatment and the time necessary for it to take effect (essentially this means time under treatment as outlined in Appendix B and is specifically different for the different infections); and (3) that the skin disease or condition would not be communicable or harmful to the opponent at the time of competition. Such documentation shall be furnished at the medical examination."

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Comment [d8]: I don't think we govern these with the handbooks

~~Skin infections are in most cases an unfortunate or unpleasant side effect in the sport of wrestling. In most cases the implementation of appropriate topical or systemic treatments will make it safe for activity.~~ These conditions are common in sports where there is close physical contact with other individuals, pads, mats or equipment. In most cases proper medical treatment by a physician will make it safe for activity. Return to participation depends largely on the referral access to an experienced physician for timely identification and treatment. Once again it is critical that a relationship is established with a physician who knows the sport, ~~or is willing to be educated about the sport and the~~ common skin conditions and the parameters for restriction of participation. When this happens, timely referral and treatment will be more likely.

New Section; title: Skin Infections

The section of the rules book that specifically outlines the skin infections most common in the sport is Appendix B. It is important to note that ~~10~~ 19 percent of time loss injuries in the sport of wrestling are associated with skin disease. It is also becoming more common for entire programs to be shut down for periods of time, and in extreme cases entire conferences or state athletic associations, because of an outbreak of skin infection. In this presentation we are going to discuss the three most common types of skin infections to the sport. It is important to be aware that these skin infections are different in nature and therefore need to be treated differently with regard to medication and disposition. We will outline the basics and the rules that surround bacterial, viral and fungal infections. They are very different in nature but in some cases **look** very similar depending on the stage of infection. For example ringworm, which is a fungal infection, may look very similar to an outbreak of Impetigo, which is a bacterial INSERT PICTURES OF RINGWORM AND IMPETIGO infection, depending on the age or stage of infection. A herpetic lesion, which is viral in nature, may look very much like folliculitis, which

Comment [d9]: Why do we have 2 sections on skin infections.

Comment [d10]: Show graphic of names of conditions and percent from Sports Medicine Handbook

Comment [d11]: We will need approvals for use of the pictures unless we took them ourselves.

is bacterial. INSERT PICTURES OF BOTH TYPES OF FOLLICULITIS ~~One problem is that it is common for~~ S~~s~~tudent-athletes or coaches should never try to self treat ~~to self treat and in fact may be treating a bacterial infection with an antifungal medication or herpes with an antibiotic~~. When you notice any lesion, check it out with medical staff. Early detection and accurate diagnosis by appropriate medical personnel, and appropriate treatment are critical for return to participation.

Does this section encourage s-a and coaches to self diagnose? We typically conduct medical staff education separate from s-a and coach education.

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First on the list for discussion are bacterial infections. These are puss producing infections of the skin most commonly associated with Staphylococus or streptococcus bacteria. These infections are contagious and should be considered so when puss, exudates or fluid is present. It is when there is an overpopulation of this bacteria coupled with abrasive activity that we may see an infection. INSERT PICTURES OF IMPETIGO, FURUNCLES AND CARBUNCLES These infections would include Furuncles, Carbuncles, Folliculitis, Impetigo, Cellulitis, Erysipelas, or Staphylococcal disease. In addition to these common problems, we are seeing an increasing occurrence of an antibiotic resistant strain of Staph called MRSA. MRSA stands for Methicillin-resistant Staphylococcus aureus. This is an extremely dangerous infection that in extreme cases can even cause death. Bacterial infections are systemic in nature. They must be treated by either oral or a combination of oral and topical antibiotics. The disposition for a student athlete found to have a bacterial infection is outlined in the rule book as follows:

1. The wrestler must have been without any new skin lesion for 48 hours before the meet or tournament.

2. The wrestler must ~~have completed 72 hours of antibiotic therapy and~~ have no moist, exudative or draining lesions at meet or tournament time.
3. Gram stain of exudates from questionable lesions (if available).
4. Active bacterial infections shall not be covered to allow participation.

Prevention for these types of infections is cleanliness. There is a common theme when discussing keeping the mats, locker room, equipment and clothing clean. Wrestlers ~~must~~ should shower after every work out and should never wear workout gear unless they are dry and clean. We recommend using an antibacterial soap with a plastic body scrubbing device. INSERT PICTURE OF SCRUBBIE They can be purchased at a local store in most cases for less than a buck. The student athlete should use it, rinse it and hang it to dry. They should also be replaced every 30 days. Showering and using appropriate cleaning products on mats, equipment and in locker rooms are the most effective methods to combat skin infections in general and specifically dangerous bacterial infections. INSERT PICTURE OF HERPES SIMPLEX Another infection that is becoming more and more common is Herpes Simplex. Herpes simlex accounts for 10% of injuries that keep wrestlers off the mat. There is no cure for herpes. Once a student-athlete has contracted herpes, they will have the potential for an outbreak the rest of their life. Herpes is extremely contagious and especially if it is a primary lesion. This means that a first time lesion will be more difficult to resolve and will most likely cause a slightly longer time loss. Individuals can have an outbreak of herpes at any time. Common triggers for outbreak are fatigue, stress, flu, colds, fever, or sunlight. It is not unusual to see outbreaks at the beginning of the season when positions on the team are being decided by challenge matches and then again at the end of the season just before conference and national championships. Herpetic lesions commonly produce exudates and drainage. Because of these open sores, ~~the double whammy of~~

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Comment [d13]: Who is "We" I don't think this procedure recommendation is in the guideline.

Comment [d14]: On screen

a secondary bacterial infection is always a possibility. ~~The athletic training and medical staff may want to consider treating these problems with antiviral as well as antibiotic medications to prevent secondary infections~~. The disposition of a student-athlete with Herpes Simplex is as follows:

For a primary infection

1. The wrestler must be free of systemic symptoms of viral infection (fever, malaise, etc.).
2. The wrestler must have developed no new blisters for 72 hours before examination (before competition).
3. The wrestler must have no moist lesions; all lesions must be dried and surmounted by a firm adherent crust. (It is important to note that viral particles can exist even in this crust and are especially prevalent in primary lesions or infections.)
4. The wrestler must have been on appropriate dosage of systemic antiviral therapy for at least 120 hours before and at the time of the meet or tournament. Documentation of this information is critical and if not produced at the time of medical check, is reason for restriction from participation.
5. **Active herpetic infections shall not be covered to allow participation.**

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For recurrent infections:

1. Blisters must be completely dry and covered by a firm adherent crust at the time of competition or the wrestler shall not participate.
2. The wrestler must have been on appropriate dosage of systemic antiviral therapy for at least 120 hours before and at the time of the meet or tournament. Once again documentation is a requirement
3. **Active herpetic infections shall not be covered to allow participation.**

It is common for a student-athlete to have multiple outbreaks or to be prone to outbreaks throughout the season. Wrestlers with a history of recurrent outbreaks should be considered for season long prophylaxis~~ with Zovirax (acyclovir) or Valtrex~~. ~~Dosage and the implementation of prophylaxis~~This decision should be made after consultation with the team physician or dermatologist. INSERT PICTURES OF RINGWORM Finally the last infection we will discuss is tinea infection or commonly known as ringworm. Tinea can be found on different parts of the body including the head, body, groin, and feet. Proper identification is critical because ringworm can look like other problems including impetigo, exema, or dry patches. ~~Unfortunately this infection is the one that historically has received the most press and is quite frankly the least dangerous of the ones we have discussed here.~~ Ringworm is a fungal infection. Fungus thrives in a warm, moist, unsanitary, dark environment. This environment is prevalent in locker rooms where used workout gear and wet towels exist. Mats, locker rooms, clothing and equipment must be kept clean, cool, lighted, and dry. Student-athletes should never hang a wet towel over the door of a vented locker. They should never store dirty or used workout gear in a locker. Equipment bags should be kept clean and dry. Tinea infections that go untreated may also sustain a secondary bacterial infection, but is less likely than the possibility with herpes. Tinea or ring worm is the only infection that may be covered to allow participation following treatment. The disposition, treatment and covering of a tinea infection are as follows:

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1. A minimum of 72 hours of topical therapy is required for tinea lesions. ~~The cidal topic antifungals terbinifine or naftifine (lamisil or naftin) are suggested for treatment. There are other antifungals but they are fungistats instead of fungicides. Fungistats will retard the growth of the infection where fungicides will kill the infection much quicker~~.
2. A minimum of two weeks of systemic antifungal therapy is required for scalp lesions.

3. Wrestlers with extensive and active lesions will be disqualified. Activity of treated lesions can be judged by KOH prep or a review of therapeutic regimen. Wrestlers with solitary, or closely clustered, localized lesions will be disqualified if lesions are in a body location that cannot be "adequately covered." Medical staff should consider a c~~C~~overing routine ~~should~~ that includes selenium sulfide washing of the lesion, followed by application of antifungal cream, then a gas permeable dressing followed by pre-wrap and stretch tape. Coverings should be changed after every match so the lesion can air dry.

Comment [d17]: Treading on too prescriptive and not in the SMH.

4. The disposition of tinea cases will be decided on an individual basis as determined by the examining physician and or certified athletic trainer.

In closing we believe the most important message is education and cooperation. Student-athletes, coaches, ~~athletic trainers~~ and medical staffs need to work together to make sure early detection, referral and appropriate participation status is implemented to protect ~~other~~ student-athletes. Skin infections are frustrating because they do not appear as serious as other injuries and illnesses that ~~restrict~~ impede participation. Often student-athletes will try to conceal infections so that their participation is not restricted. We strongly encourage student-athletes, coaches and athletic training staffs to be leaders in prevention, referral, detection and treatment, as it will help individual programs stay active. Thank You!!

REPORT OF THE
NCAA WRESTLING RULES SUBCOMMITTEE
APRIL 9-11, 2012, MEETING

KEY ITEMS.

- **Returning to Wrestler's Certified Weight.** The Wrestling Committee has strong concerned for the wrestler who weighs-in two or more weight classes above their original certified weight and then is prevented by playing rule from returning to compete at that original certified weight class. Currently Rule 8.3.5.3 says when a wrestler weighs-in two or more weight classes above their original certified weight, their new class becomes one weight class below that higher class at which they weighed-in.

ACTION ITEMS.

1. Legislative Items.

- **None.**

2. Nonlegislative Items.

- **Returning to Wrestler's Certified Weight.** In the late 1990's, the playing rule regarding a wrestler's ascending/descending weight was instituted as part of the NCAA Weight Management Program. Its goal was to help control a wrestler from using weight gain and weight loss inappropriately. At that time the weight certification process was figured on a calculator and noted on a form that was mailed to the national office. The system had little oversight and limited verification. The online Optimal Performance Calculator (OPC) system of today streamlines the weight management process, and the Wrestling Committee has been diligent to close loopholes whether they were being used intentionally or not.

  The NCAA Wrestling Committee requests the NCAA Committee on Competitive Safeguards and Medical Aspects of Sports to consider allowing a change to the rule below to allow the wrestler to descend to the original certified weight after the wrestler weighs-in two or more weight classes above it. The individual would accomplished this by following their weight-loss plan, which means they would not lose more than 1.5 percent of their weight per week.

  > **Rule 8.3.5.3.** A wrestler weighing in two weight classes above an original certified weight class shall forfeit his right to return to the original wrestled weight class. This includes preseason competition while a wrestler is making the descent toward his lowest allowable certified weight. For example, if a wrestler's lowest allowable weight class is 149 and the wrestler weighs in at 165, the student athlete is ineligible to return to the 149-pound weight class. The lowest weight class at which this wrestler may compete is 157.

(1) Recommendation. Allow the student-athlete to descend to their original certified weight by following their individual weight-loss plan after weighing-in two or more weight classes above their original certified weight.

(2) Effective Date. This change would be effective for the 2013-14 season.

(3) Rationale. It is a coaching decision to have a student-athlete wrestle up in a higher weight class as a strategy to fill each of the 10 weight classes. The student-athlete is getting by not being allow to compete at a weight class more than two classes above their lowest allowable weight while they are following their weight loss plan to descend to the lower weight. The OPC system tracks each wrestler's descent plan with every weigh-in, ensuring their weight loss does not exceed more than 1.5 percent body weight per week, making the rule is obsolete.

(4) Estimated Budget Impact. None.

(5) Student-Athlete Impact. This would have a favorable impact for the student-athlete who would be allowed to descend to their original lowest allowable weight.

INFORMATIONAL ITEMS.

1. Assessor's Role. The assessor (a member of the institution's athletic medical staff) is the only individual permitted to perform any of the mandated weight-assessment protocols. Once the assessor enters the student-athletes' data in the OPC system, the information is stored there for 48 hours. With-in the 48 hour window, the head coach and assessor discuss and review the data. The coach shall discuss and review with each student-athlete their assessment data before confirming the assessment as official.

2. Tournament Procedures. The NCAA Wrestling Committee is emphasizing to all tournament directors, institutions and organizations that host an NCAA registered tournament that the NCAA Wrestling Rules and Interpretations book, including the wrestling administrative rules found in Part II of the book (Rules 8 and 9), shall be followed. Failure to follow required procedures shall result in the institution or organization being banned from hosting an NCAA registered tournament for a minimum of one year.

a. The official NCAA Weigh-In Form generated by the OPC system shall be provided by each individual at registration.

b. All competing teams shall be present before weigh-ins occur. Anyone aware that weigh-ins begin before all teams are present have an obligation to report it to Ron Beaschler, secretary-rules editor, or Jim Thornton, certified athletic trainer liaison.

c. Weigh-ins at a tournament shall occur not more than two hours before the first match begins. Anyone aware that weigh-ins begin sooner than two hours before the first match have an obligation to report it to Ron Beaschler, secretary-rules editor, or Jim Thornton, certified athletic trainer liaison.

d. Match results shall be submitted to the OPS system within 24 hours of completion of the tournament.

3. Weigh-Ins at Dual, Triangular and Quadrangular Meets. Weigh-ins at dual, triangular and quadrangular meets shall occur not more than one hour before the first match begins. Anyone aware of weigh-ins beginning at the wrong time have an obligation to report it to Ron Beaschler, secretary-rules editor, or Jim Thornton, certified athletic trainer liaison.

4. Unattached Wrestler in a Open Tournament. The coach of a wrestler whose name appears on the team roster has specific responsibilities.

a. An institution having a wrestler (name appears on the team roster) who competes unattached in an open tournament shall list that tournament on their schedule in the OPC system. The competition schedule listed in the OPC system is NOT used to calculate the institution's dates of competition. If only one individual from a team participates in an open event, that event shall be listed on the OPC schedule. This allows the results from the tournament to be downloaded into the wrestler's Individual Season Record Form.

b. An institution having a wrestler (name appears on the team roster) who competes unattached in an open tournament shall provide for the wrestler their official NCAA Weigh-In Form. The NCAA Weigh-In Form identifies the wrestler's lowest certified weight class.

Committee Chair: *Dave Martin, Oklahoma State University, Big 12 Conference*
Staff Liaison(s): *Jeff Jarnecke, Championships and Alliances – Tournament Operations*
Mary Berdo, Championships and Alliances – Tournament Operations
Teresa Smith; Championships and Alliances – Playing Rules and Officiating

April 9-11, 2012	
Attendees	Absentees
Jason Borelli, Stanford University	
Brad Bruhn, SUNY Cortland	
Dax Charles, Colorado State University, Pueblo	
Gary Franke, Washington and Lee University	
Tom Gioglio, East Stroudsburg University of Pennsylvania	
Mike Kirk, University of Central Oklahoma	
Tom Minkel, Michigan State University	
Andy Noel, Cornell University	
Doug Peters, Minnesota State University	
Deborah Polca, Old Dominion University	
Dick Simmons, Cornell College	
Jeff Swenson, Augsburg College	
Gary Taylor, Rider University	

Other Participants:
John Baldwin, NCAA; Ron Beaschler, Ohio Northern University (secretary-rules editor and weight management program liaison); Bob Colgate, National Federation of State High School Associations; Anthony Holman, NCAA; David Klossner, NCAA; Pat McCormick, Hampton University (national coordinator of officials); Mike Moyer, National Wrestling Coaches Association (NWCA); Archie Randall, Oklahoma City University (NAIA representative); Jim Thornton, Clarion University of Pennsylvania (athletic trainers' liaison); and Pat Tocci, NWCA.

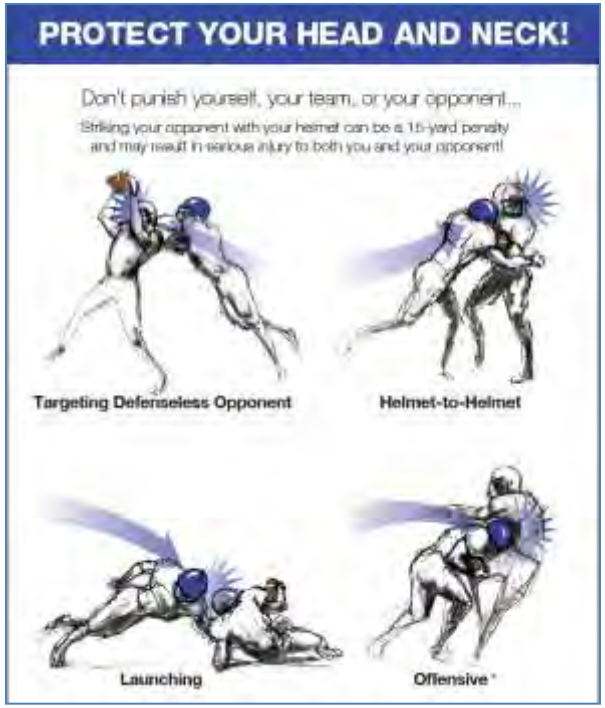
Head Injury

Loss of Helmet During Play.

- If a player intentionally takes off his helmet during the play it is unsportsmanlike conduct which carries a 15-yard penalty.
- It is a personal foul if the player whose helmet comes off continues to participate in the play beyond the immediate action in which he is engaged.
- If the helmet comes off other than as the direct result of an opponent's foul, the player must also leave the game and is not allowed to participate for the next play.

Kick offs

- Injuries from Kick offs are more severe than the injuries from other phases of play, in terms of the type of injury and time lost.
- Teams will kick off at the 35-yard line instead of the 30.
- Players on the kicking team can't line up for the play behind the 30-yard line - limits the running start.
  - NFL reported a 40% reduction in concussions following their rule change.
- Touchbacks on free kicks will be moved to the 25-yard line instead of the 20 to encourage more touchbacks.



NCAA
Football Rules / Safety Education Video

Learning Objectives
Understand 2012 NCAA football rules changes.
Understand proper helmet fitting and basic maintenance.
Understand basic techniques to protect the defenseless player.
Understand basic techniques for proper tackling.
Concussion awareness.

Audience:
Primary: Student-athletes.
Secondary: Coaches.

Menu

1. **Introduction (0 – 2 min)**
    a. The Football Code.
    b. Risk.
    c. Safer play, avoid catastrophic injury.

Creative Note:
Highlight tackling, proper technique in each area.
Use text on screen to highlight rules/penalties associated with each area.

2. **Tackling (3 - 5 min)**
    a. Discuss types of tackling techniques, proper mechanics and how athletes can safely break up a play across the middle.
    b. Proper wrapping technique, drills
    c. Blocking Below the Waist.
    d. Horse collar tackle
    e. Spearing
    f. Scenarios, plays, flagrant fouls, conference review.
        i. Show positive plays drills.
        ii. Show one illegal hit followed by three highlighting proper tackling technique.

Resources/References:
NCAA rules video
NCAA Spearing education poster
NCAA Rules book (defenseless player)

NCAA injuries data

3. **Football Helmet: Protecting the Head and Neck (3 – 5 min)**
    a. The football helmet: fit and maintenance.
        i. The helmet is intended to protect the player from head injuries. It must therefore be fitted properly so that it does not come off through play.
            1. Reduction of skull fractures and death.
            2. Show chart of catastrophic fatalities.
        ii. Chin strap and mouth guards.
    b. NCAA football rules change: Loss of Helmet during Play.
    c. Targeting/Initiating Contact with the Crown of the Helmet (Rule 9-1-3) and the Defenseless Player.
        i. Resource: NATA Heads up video.
    d. Helmet and face mask fouls.
    e. Concussion awareness.
        i. Resource: NCAA concussion education video.
    f. Scenarios, plays, penalties, conference review.

    Resources/References:
    NCAA helmet fitting poster
    AMSSM injury and rules panel presentation
    NCAA concussion video
    NCAA Rules book
    NCAA injuries data

4. **Kicking Game**
    a. NCAA football rules change: Kickoff and Touchback Starting Lines Moved
    b. Shield Blocking Scheme on Punting Plays.
    c. NCAA football rules change: Additional Protection to Kick Returner.
    d. Reemphasize proper tackling technique.
    e. Scenarios, plays, flagrant fouls.

    Resources/References:
    Spearing
    NCAA Injury data (cervical, head down contact)

5. **Summary (0:60 – 90 s)**

6.

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
INTRO	Section 1: Introduction	1. To introduce the football code.	The Code	Logos: NCAA Voice Over with text on-screen (est. time 00:20) B Roll: • Football through the years • Enter Time Start: Stop	Presenter (NCAA staff or football playing rules committee member): [Sample intro] "The NCAA Football Rules Committee has extraordinary pride in the Football Code, which was introduced in 1916 and has been updated several times. These guidelines form a harmony of agreement among coaches, players, game officials and administrators that places each contest in an environment of fairness and sportsmanship. It is noted that the Code emphasizes the following unethical practices: "Using the helmet as a weapon. The helmet is for protection of the player..." and "players and coaches should emphasize the elimination of targeting and initiating contact against a defenseless opponent and/or with the crown of the helmet." Every participant in the collegiate football scene shares a responsibility for ethical conduct that enhances the future of this American tradition."

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
INTRO	Section 2: Introduction	Teach proper technique Provide a safe environment for practice and competition of in football.	Presenter name plate. Teach proper technique of football. Create the conditions necessary for student-athletes to play the game safely. Successful student-athletes and coaches: are well trained in the tactics and techniques of their sport. Recognize that safety is important	Logos: NCAA Voice Over with text on-screen (est. time 00:30)	Presenter: (recent graduate, current NCAA student-athlete or prominent NCAA coach) "We know the game of football can be tough …" … Make the game a little bit safer and reduce the risk of catastrophic/season-ending/life-changing injuries…" The National Collegiate Athletic Association is proud to bring you ... This video is designed to help teach you the techniques you need to make the game safer.

Resources:

1. National Center for Catastrophic Sport Injury Research.

2.

Reference List:

1. 2011 and 2012 NCAA® Football Rules And Interpretations.
2.

Transition to Unit 2: Tackling.

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
TACKLING	Section 1:	1. Understand how improper contact can lead to head and neck injuries 2. Recognize proper tackling techniques and how to safely break up a play across the middle. 3. Recognize proper wrapping technique.		Logos: NCAA Voice Over with text on-screen (est. time 00:15)	Presenter:

Resources:
1.
2.

Reference List:
1.
2.

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
TACKLING	Section 2: NCAA rules and rules changes.	1. Understand NCAA football rules changes related to blocking below the waist. 2. Understand rule 9-1-4: Targeting the defenseless player. 3. Horse collar tackle, spearing.	Rule 9-1-4. No player shall target and initiate contact to the head or neck area of a defenseless opponent with the helmet, forearm, elbow or shoulder.	Logos: Voice Over with text on-screen (est. time 00:15)	Presenter:

Resources:

1. 2011 NCAA CFO Video
2. Student-Athlete Poster - *Protect Your Head and Neck!*
3. NCAA Sports Medicine Handbook Guideline 3d. Use of the head as a weapon in football and other contact sports.
4.

Reference List:

1. NCAA Football Rules Committee 2012 Changes Approved Memo
2. Blocking Below the Waist – Interp Memo
3.
4.

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
TACKLING	Section 3: Scenarios, plays, flagrant fouls, review.	1. Review various tackling techniques, plays, scenarios and fouls.		Logos: NCAA Voice Over with text on-screen (est. time 00:00)	Presenter:

Resources:
1. 2011 NCAA CFO Video
2.

Reference List:
1. 2011 and 2012 NCAA® Football Rules And Interpretations.
2.

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
TACKLING	Section 4: Summary	1. Summarize the importance of proper tackling in protecting the health and safety of football student-athletes.		Logos: NCAA Voice Over with text on-screen (est. time 00:00)	Presenter:

Resources:
1.
2.

Reference List:

1.
2.

Transition to Unit 3: The Football Helmet

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
The Football Helmet	Section 1:	1. Understand the importance of proper fit and maintenance of the football helmet. 2. Understand how to properly wear the football helmet, including chin strap and mouth guard. 3. Understand basic maintenance of the football helmet.		Logos: NCAA Chart of catastrophic fatalities. NCAA Helmet fit poster.	Presenter: "The helmet is intended to protect the player from head injuries. It must therefore be fitted properly so that it does not come off through play…" "Reduce skull fractures and death…"

Resources:
1. NATA Heads Up Video
2. NCAA Football Safety Rules
3. NCAA Helmet Fit Poster

Reference List:
1. 2011 and 2012 NCAA® Football Rules And Interpretations.
2. NCAA Football Injury Data
3. Catastrophic fatalities (AMSSM presentation slide)

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
The Football	Section 2:	1. Understand	Loss of Helmet During Play.	Logos: NCAA	Presenter:

Helmet		the NCAA football rules change: Loss of helmet during play.		Video/Still: loss of helmet during play	"If a player's helmet comes off during play, he must not continue to participate in the play to prevent injury. If the helmet comes off other than as the direct result of an opponent's foul, the player must also leave the game and is not allowed to participate for the next play."

Resources:
1. Helmets Flying In College Football. USA Today Images
2.

Reference List:
1. 2011 and 2012 NCAA® Football Rules And Interpretations.
2.

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
The Football Helmet	Section 3:	1. Understand how improper contact can lead to head and neck injuries.	Targeting/Initiating contact with the crown of the helmet. Keep your head up. Lead with your shoulder	Logos: NCAA Graphic: Axial Loading Position NCAA spearing poster Graphic: Catastrophic cervical spine injuries.	Presenter: "…reduce the risk of injury" "...Steps to protect yourself" "the person at greatest risk …" "Overcome the natural tendency to lead with your helmet …" "every incident of head down contact puts the player at risk of a serious head and neck injury" "Proper contact techniques can reduce that risk …"
		2. Understand rule 9-1-3.	No player shall target and initiate contact against an opponent with the crown (top) of his helmet.		

Resources:
1. NATA Heads Up Video
2.

Reference List:
1. 2011 and 2012 NCAA® Football Rules And Interpretations.
2.

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
The Football Helmet	Section 4: Review	1. Review various plays, scenarios and fouls.		Logos: NCAA Voice Over with text on-screen (est. time 00:15)	Presenter:

Resources:
1.
2.

Reference List:
1. 2011 and 2012 NCAA® Football Rules And Interpretations.
2.

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
The Football Helmet	Section 5: Concussion Awareness	1. Understand the signs and symptoms of a concussion.	Concussion symptoms Don't hide it. Report it. Take time to recover. When in doubt, get checked out.	Logos: NCAA Graphic: Concussion symptoms. Graphics: video of how the brain responds to contact.	Presenter: "a concussion is a serious injury …"

Resources:
1. NCAA Concussion Education materials.
2. NCAA Concussion education video.

Reference List:
1. NCAA Sports Medicine Handbook Guideline 2i: Concussion or Mild Traumatic Brain Injury in the Athlete.

Transition to Unit 4: The Kicking Game

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
The Kicking Game	Section 1:	1. Understand NCAA football rules change to move the kickoff to the 35-yard line. 2. Understand NCAA football rules change to move the touchback distance on free kicks to the 25-yard line.	Kickoff and Touchback Starting Lines Moved. 35-yard line 25-yard line	Logos: NCAA	Presenter: Committee voted to move the kickoff to the 35-yard line (currently set at the 30-yard line), and to require that kicking team players must be no further than five yards from the 35 at the kick, which is intended to limit the running start kicking teams have during the play "Moved touchback distance on free kicks to the 25-yard line instead of the 20-yard line to encourage more touchbacks…" "NCAA data indicates injuries during kickoffs occur more often than in other phases of the game…" "injuries from head-down contact …"

Resources:
1. B-roll, NCAA football game coverage.
2. NATA Heads Up Video
3. Student-Athlete Poster - *Protect Your Head and Neck!*

Reference List:
1. NCAA Football Injury data
2. 2011 and 2012 NCAA® Football Rules And Interpretations.
3. NCAA Football Rules Committee 2012 Changes Approved Memo
4.

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
The Kicking Game	Section 2:	1. Shield Blocking Scheme on Punting Plays. 2. Understand 2012 NCAA football rules change adding additional protection to the kick returner.		Graphics: video of shield blocking plays. Highlight one incorrect and at least two correct plays Graphic:	Presenter: "… not allow the receiving team to jump over blockers, unless the player jumps straight up or between two players…" "…provide a kick returner additional protection to complete a catch before allowing contact by the kicking team …"

Resources:
1. B-roll, NCAA football game coverage.
2. NATA Heads Up Video

Reference List:
1. NCAA Football Injury data
2. 2011 and 2012 NCAA® Football Rules And Interpretations.
3. NCAA Football Rules Committee 2012 Changes Approved Memo
4.

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
The Kicking Game	Section 3: Review	1. Understand how improper contact can lead to head and neck injuries. 2. Review various plays, scenarios and fouls.			Presenter: "Proper contact techniques can reduce that risk …"

Resources:
1. B-roll, NCAA football game coverage.
2. NATA Heads Up Video

Reference List:
1. NCAA Football Injury data.
2.
3.

Transition to Unit 5: Summary / Conclusions.

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
EXAMPLE	1.		Enter minimal size standards per NFHS, NCAA, and USATF rules (chart format) Overlay graphics for key set up points.	Voice Over with text on-screen (est. time: 4 min) Voice Over B Roll:	Presenter:

	• Clip Name • Enter Time Start:Stop	
	Voice Over B Roll: • Clip Name Enter Time Start:Stop	Presenter:
		Presenter:

Resources:
1. **Check lists.**
2. **Additional Clips**
3. **Where to find more information on this topic.**

Reference List:
1. **Position stands, data, literature, etc. used in this section.**
2. **.**

Unit	Section	Learning Obj.	Text / Data Displayed	Graphics: Video / Pictures	Audio
Basic laws of physics governing the vault	Sect 1: Pole is a lever, rotates around fixed spot, the longer the lever the more work it takes to move it	1.	Overlay graphics for key set up points. Highlight correct positions	Voice Over with text on-screen (est. time: 3 min) Voice Over B Roll: • Clip Name • Enter Time Start:Stop	Presenter:
	Sect 2. Vaulters body is a second lever rotating around fixed points on the pole	1.		Voice Over B Roll: • Clip Name Enter Time Start:Stop Slow motion at:	Presenter:
	Sect 3. Vaulter must convert horizontal energy into vertical energy (force vector)	1.			Presenter:

Resources:
1. **Additional Clips**
2. **Text book, papers, camps, etc.**

Reference List:
1. **papers, books used in this section.**
2.

Background: The NFL bounty scandal stems from the New Orleans Saints involvement in a system that offered financial incentives for hits intended to injure opposing offensive players between 2009 and 2011.

Recognizing that student-athletes are prone to mimicking the behavior of professional athletes, the NCAA is taking proactive steps to discourage similar behavior in NCAA football.

Overall Goals: In anticipation of questions regarding bounty programs and player safety in college football, the major focus of this campaign is to educate NCAA athletics professionals and administrators, football coaches and student-athletes on existing conduct and sportsmanship rules via membership engagement, resource development and other methods.

Target Audiences:

- NCAA football coaches and administrators
- NCAA student-athletes
- NCAA member institutions

Key Messages:

- Targeting of opponents with intent to injure is strictly prohibited under NCAA rules and will not be tolerated.
- There is no indication that bounty programs are a problem in college football. The NCAA will remain proactive and vigilant in protecting student-athlete safety.
- Any football programs proven to be in violation of these rules will be held accountable according to existing NCAA rules.

Tactics/Next Steps:

- NCAA will partner with the American Football Coaches Association (AFCA) to emphasize existing rules on this issue in preseason correspondence. (June)
- The AFCA will put this issue on the agenda for discussion at its annual conference. (January 2013)
- NCAA members and student-athletes will be encouraged, via ongoing membership updates, to utilize the anonymous tip line (a resource staffed by AMA and Enforcement personnel) to report bounty programs. Through this anonymous tip line, the NCAA will launch a new initiative to track the number of calls related to allegations of bounty systems. (June)
- NCAA communication staff will draw attention to the issue by placing a feature story on NCAA.org on this issue. (June)
- The NCAA Committee on Competitive Safeguards and Medical Aspects of Sports (CSMAS) which is responsible for overseeing NCAA health and safety rules, and the NCAA Committee on Sportsmanship and Ethical Conduct (CSEC) will issue a joint

INTERNAL DRAFT

statement to emphasize the NCAA position, and to encourage member institutions to be vocal against this behavior and to immediately address any indication of this occurring in their programs. (June)

- Additionally, CSMAS and CSEC will work together to determine if a clarification of the current rules is appropriate. (June)
- The NCAA plans a video production that will focus on contact that is above the shoulders, reminding players of the risks and the penalties associated with such contact. This video will be produced in conjunction with several high-level NCAA coaches and conference commissioners and will be in a format intended to be viewed with teams during fall practice. (June)
- The NCAA will continue to assist in the review of fouls that, by rule, require a conference to consider suspension and suggest penalties when appropriate. (ongoing during football season)
- President Emmert will also discuss the issue at the next Division I Board of Directors meeting. (August)

INTERNAL DRAFT

Relevant Bylaws to prevent bounty or reward-type behaviors

Institutional Control and Responsibility

Within the NCAA Constitution, Article 2 places the responsibility on each member institution to monitor and control its athletics program in compliance with the rules and regulations of the Association. (NCAA Constitution 2.1.1, 2.8.1). The scope of this responsibility includes actions of its staff members, which would include both coaches and administration. (NCAA Constitution 2.1.2). The NCAA mandates member institutions establish policies for sportsmanship and ethical conduct in intercollegiate athletics consistent with the mission and goals of the NCAA. (NCAA Constitution 2.4(a)). In accordance with the NCAA's mission of sportsmanship and ethical conduct, member institutions are to ensure their athletics programs are conducted in a manner designed to protect and enhance the physical well-being of student-athletes by providing a safe environment for each participating student-athlete. (NCAA Constitution 2.2, 2.2.3). In addition, each member institution has the duty to report compliance violations to the Association and cooperate fully with any investigation or imposition of corrective actions. (NCAA Constitution 2.8).

The Constitution places the final authority and ultimate responsibility for the conduct of member athletic departments with the president or chancellor of the institution. (NCAA Constitution 6.1). However, all individuals employed in connection with members' athletics programs have a duty to act with honor and sportsmanship at all times. (NCAA Bylaws 11.1.1). In order to promote the dignity of fair play in intercollegiate athletics the Bylaws place an additional duty on head coaches to promote an atmosphere of compliance and to monitor compliance activities by those who report to him or her. (NCAA Bylaws 11.1.2.1).

Ethical Conduct

Article 10 of the NCAA Bylaws provides greater detail on the NCAA's mission of ethical conduct stating that individuals employed or associated with a member institution to administer, conduct, or coach, along with all student-athletes, shall act with honestly and sportsmanship at all times and shall represent the honor and dignity of fair play. (NCAA Bylaw 10.01.1). The Bylaws state it is unethical to offer or provide a student-athlete an improper inducement or extra benefit or improper financial aid. (NCAA Bylaw 10.1).

As specifically enumerated in NCAA Bylaw 10.3, participation in any sports wagering activities are considered a violation of ethical conduct and are prohibited to student-athletes and all athletic department staff members. The scope of this prohibition extends to wagers on any institutional practice or competition in which the Association conducts a championship, as well as to bowl subdivision football. (NCAA Bylaw 10.3.1).

Extra Benefits and Pay

In accordance with the NCAA's principle of amateurism, student-athletes are prohibited from receiving a benefit not expressly authorized by NCAA legislation or generally available to the institution's student body. (NCAA Constitution 2.9, NCAA Bylaw 16.02.3). In addition, student-athletes are prohibited from using his or her athletic skill in their sport for payment in order to maintain their eligibility and status as an amateur athlete. (NCAA Bylaw 12.1.2). Violations of these regulations subject student-athletes to the enforcement procedure outlined in the Bylaws (NCAA Bylaws 19, 32).

Penalties

The NCAA Bylaws generally outline the enforcement process and procedure, including available penalties the Committee on Infractions may impose on a member institution found to be in violation of Association rules. (NCAA Bylaws 19, 32). These penalties range from a public reprimand to a reduction in scholarships or recruiting activities to probation for up to five years or a show cause order. (NCAA Bylaw 19.5). A few violations have automatic penalties associated with a finding of noncompliance written into the Bylaws.

In accordance with the NCAA's mission to promote the dignity of fair play, student-athletes found to have knowingly participated in sports wagering activities involving their institution permanently lose all remaining eligibility. (NCAA Bylaw 10.3.2(a)). The Bylaws also dictate that a student-athlete found in violation of the unethical conduct regulations will be ineligible for further competition, but may appeal such a finding to the Committee on Student-Athlete Reinstatement for the restoration of eligibility. (NCAA Bylaw 10.4).

NCAA Football Rules Committee

Historical review of rules intended to prevent/reduce head injury

1906 – As the culmination of six meetings called by President Teddy Roosevelt to address the number of catastrophic injuries in college football, a joint committee approves nearly 30 changes to the playing rules, with most intended to remove brutality and to change the character of the game in a positive way.

1916 – First NCAA Football Rules Code developed and published.

1939 – All players required to wear helmets.

1962 – Recommended that properly fitted mouth protectors be worn.

1964 – No player may deliberately and maliciously use his helmet or head to butt or ram an opponent.

1970 – Spearing is defined as "the deliberate and malicious use of the head and helmet in an attempt to punish a runner after his momentum has stopped."

1973 – All players required to wear mouth protectors.

1973 – All players must have a helmet "with a secured chin strap."

1975 – Recommended that helmets meet NOCSAE test standards, and announced that it would be required starting with the 1978 season.

1976 – All players must have a helmet with a four-point chin strap fastened to participate in play.

1976 – Spearing redefined as "the deliberate use of the helmet in an attempt to punish the opponent."

1979 – Striking a runner with the crown or top of the helmet added as a foul.

1994 – Fighting defined and penalties established.

1996 – If the ball carrier's helmet comes off, the play is blown dead immediately. Also, the snapper is protected and may not be contacted for one second after snapping the ball.

2002 – Wording added to define a "defenseless player" and a point of officiating emphasis added to protect these players.

2006 – Eye shields must be completely clear to allow for quick medical diagnoses of student-athletes.

2005 – Modified rules regarding spearing and head-down contact, removing any reference to "intent." A significant educational focus is placed on the prevention of head and neck injuries with the membership and encourages appropriate groups (e.g., American Football Coaches Association) to teach proper tackling techniques.

2008 – The NCAA makes the horse-collar tackle illegal, revamps illegal contact of an opponent and simplifies the chop-block rule. More emphasis is placed on eliminating hits on defenseless players and blows to the head. No player is permitted to initiate contact and target an opponent with the crown of his helmet, and no player is permitted to initiate contact and target a defenseless opponent above the shoulders.

2009 – It becomes mandatory for the conference to review any flagrant personal fouls for targeting defenseless players or using the crown of the helmet. *(Note: Following the rules change in 2009 requiring conferences to further examine these fouls after games, 73 plays were reviewed, resulting in four suspensions from games. In addition, some conferences sent letters of reprimand to coaches and student-athletes following post-game reviews.)*

2010 – Strengthened existing rules regarding targeting and initiating contact and mandated that any injured player be removed from play and cleared by medical personnel before returning to play. Limited blocking schemes on kick plays to no more than two players shoulder to shoulder.

2011 – Moved the kickoff location to the 35-yard line from the 30-yard line to encourage more touchbacks and limited kicking team players to be no more than five yards behind the kickoff line. Moved the touchback spot on free kicks to the 25-yard line to further encourage touchbacks. Also protected the receiving team during onside kick plays to allow for a reasonable opportunity to catch the ball. Further clarified and limited blocking below the waist.

Also in 2011, the committee approved a proposal to treat the helmet becoming dislodged (except if by a facemask or foul by the opponent) to be treated like an injury. The player that loses his helmet must be removed from play for at least one play to have the helmet checked and refitted by the team's equipment staff. By rule, a player that loses his helmet must not continue to participate and that player may not be contacted by the opposition.

Another safety rule was put in place to reduce potentially dangerous actions by the team receiving a punt. Shield block formations in use by kicking teams have created efforts to block the punt by jumping over the blockers, causing some receiving team players to land on their head/neck if contacted in the air. This action is now illegal and the receiving team is not allowed to leap over a blocker.

Note: Research for portions of this report comes from former NCAA Secretary-Rules Editor David Nelson's book, The Anatomy of a Game, published in 1994.

Compiled October 18, 2011 *(Updated April 11, 2012)*

APPLE

Promoting Student-Athlete Wellness and Substance Abuse Prevention

SPRING 2012 REPORT

Gordie Center for Substance Abuse Prevention
University of Virginia
170 Rugby Rd, Lower Level
Charlottesville, VA 22903

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EXECUTIVE SUMMARY

The 2012 APPLE Conferences were held in Charlottesville, Virginia on January 20-22, 2012 and Huntington Beach, CA on January 27-29, 2012. We are pleased to report that 68 institutions were represented at the conferences this year. Of the 385 participants at the two conferences, 224 (58%) were student-athletes, 151 (39%) were athletics or university administrators, 5 (1.5%) were student body representatives and 5 (1.5%) were graduate assistants or interns within an athletic department.

A total of 1,214 teams from 519 individual campuses (48.1% of all NCAA member institutions) have attended the conferences since 1992. One strong indication of the continued impact of the conferences is that 99% of all attendees agreed or strongly agreed that they "would recommend the APPLE Conference to others."

As you will see throughout this report, the APPLE Conferences continue to be an opportunity for student-athletes and administrators to work collaboratively towards the common goal of promoting student-athlete wellness and substance abuse prevention on their respective campuses. This report includes details of participants' pre- and post-test evaluations as well as campus progress in implementing their action plans. Additionally, we have included summaries of the overall evaluation and breakout sessions.

Planned enhancements to the 2013 conference experience are detailed in the "Future Directions" section and are aligned with the five APPLE Conference recommendation categories suggested by the NCAA.

PARTICIPATING INSTITUTIONS

Sixty-eight colleges and universities participated in 2012. Of these campuses, 40 (59%) were NCAA Division I institutions, 13 (19%) were Division II institutions, and 15 (22%) were Division III institutions. Twelve institutions (18%) had not attended any previous APPLE conference (as indicated by *). Three additional institutions were registered for the conference but were unable to attend at the last moment. Two cancelled because they were unable to assemble a team and the other due to travel issues. Eight schools cancelled their registration prior to January 2012 and 13 schools were left on the waitlists. In total, 92 institutions registered for the conference.

Division I

Boston College
Canisius College
Cal State Northridge
Cal State Longbeach
Duke University
Fresno State
High Point University
Iowa State University
Kent State University
Liberty University
Loyola Marymount University
Northwestern University
The Ohio State University
Radford University*
Saint Francis University
Saint Louis University*
Seattle University*
Siena College*
St. John's University
Texas A&M Corpus Christi
Towson University
Tulane University
University of Akron
University of California at Berkeley
University of California, Irvine
University of California, Los Angeles
University of California, Riverside
University of Central Florida
University of Colorado
University of Delaware
University of Maryland, College Park
University of Minnesota - Twin Cities
University of North Carolina, Chapel Hill
University of Oklahoma *
University of Southern California
University of Virginia
Utah State University
University of Virginia
Utah State University
Virginia Commonwealth University
Winthrop University
Xavier University

Division II

Caldwell College*
Davis and Elkins College
Glenville State College
Limestone College
Lincoln Memorial University
Maryville University
Mount Olive College
Rollins College
Shaw University
University of California, San Diego
University of Central Oklahoma*
University of Findlay
University of New Haven*

Division III

Beliot College*
Cabrini College
Cedar Crest College*
Chapman University
Elizabethtown College
Elms College
Fontbonne University *
Keystone College
Lesley University*
Muhlenberg College
Randolph-Macon College
Salisbury University
Transylvania University
University of Mary Washington
Wilson College

Schools on the waitlist

Division I

George Mason University*
Jackson State University
Monmouth University
Temple University
University of Cincinnati
University of Maine
University of North Carolina, Greensboro

Division II

Converse College
Nazareth College
Lenoir Rhyne University
Livingstone College

Division III

Hiram College

Schools that registered but cancelled

Division I

Howard University
James Madison University
Marquette University
University at Buffalo*
University of California, Santa Barbara
University of Maryland, Eastern Shore

Division II

East Stroudsburg University
Mercyhurst College
Tuskegee University*

Division III

California Lutheran University
Emory and Henry College*

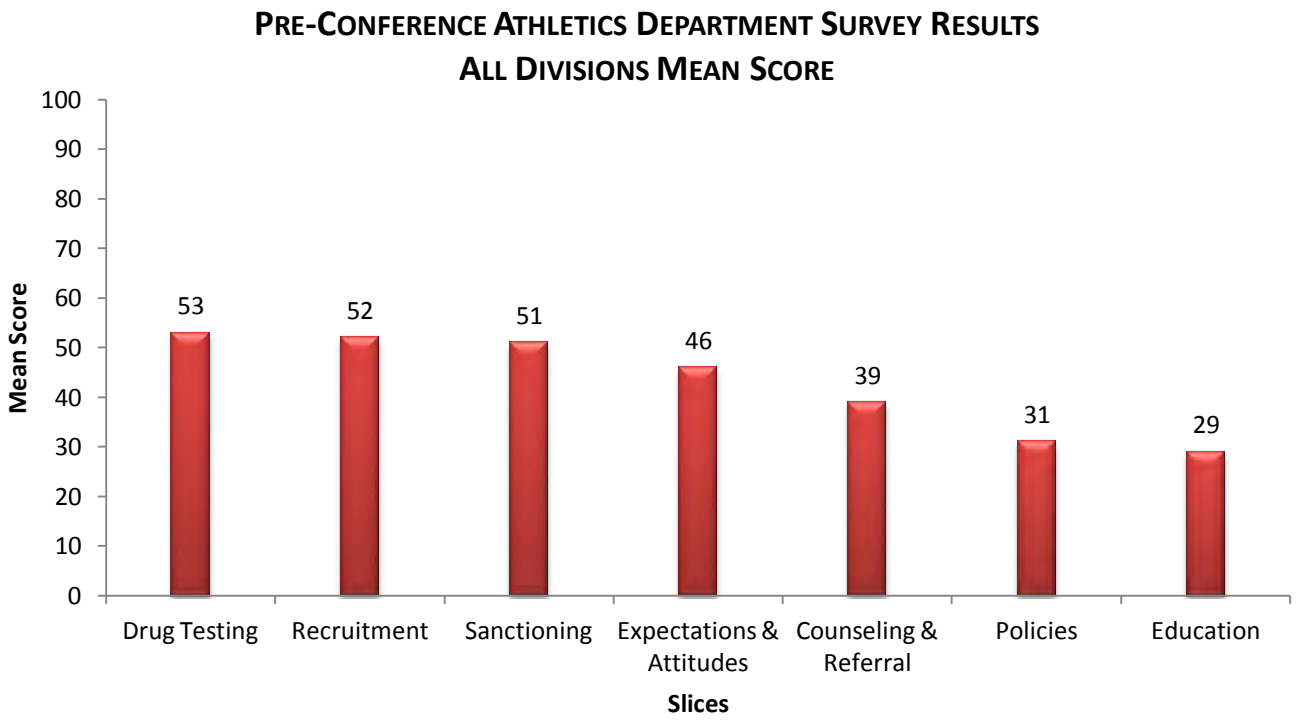
MARKETING STRATEGIES

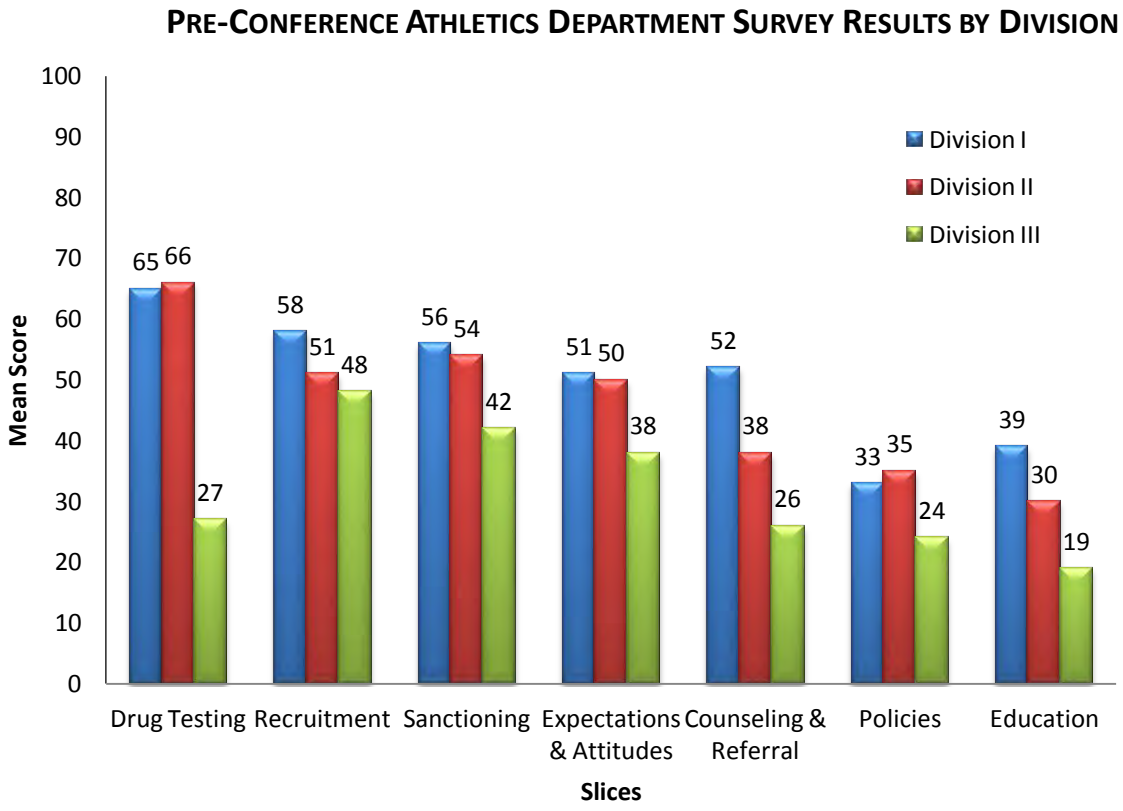
Marketing efforts for the 2012 conference consisted of electronic correspondence/announcements and phone calls. All schools on the 2011 waitlist and attendees were sent a "save the date" email in mid-May. An email announcing the conference dates and locations was sent out through NCAA list-serves. Additional outreach was made to schools within a 5 hour drive of the Huntington Beach, CA location. The former APPLE Conference Coordinator contacted athletics directors, head athletic trainers, health educators, and Lifeskills coordinators at those institutions via email and phone calls. Less outreach was required for the Charlottesville, VA conference since it filled in less than a month.

PRE-CONFERENCE SURVEY RESULTS

Understanding the strengths and weaknesses of alcohol, tobacco and other drug (ATOD) prevention efforts within an athletics department is a critical step in developing an action plan to improve programs and services. Prior to attending the APPLE conference, each institution is asked to complete a survey which asks about policies, practices, and specific criteria for each of the seven "slices" of the APPLE model; recruitment, expectations and attitudes, education, policies, drug testing, sanctioning, and counseling and referral. A total of 68 team contacts (100%) completed the online pre-conference survey this year.

At the conference, each team reviewed the confidential scores for their campus and discussed the unique needs of its department in order to determine which 'slice' would become the focus of the team's action plan for improvement. The chart below shows the mean scores of all schools that attended the APPLE conference in 2012. An additional chart on the following page breaks down the scores by NCAA division. The maximum number of points a school can receive for each slice is 100.




PRE-CONFERENCE ATHLETICS DEPARTMENT SURVEY RESULTS BY DIVISION
Mean Score
100
90
80
70
60
50
40
30
20
10
0
Division I
Division II
Division III
65 66 27
58 51 48
56 54 42
51 50 38
52 38 26
33 35 24
39 30 19
Drug Testing
Recruitment
Sanctioning
Expectations & Attitudes
Counseling & Referral
Policies
Education
Slices

PRE- & POST- CONFERENCE PARTICIPANT EVALUATION

The eight learning outcomes for APPLE conference participants are to:

- improve relationships between administrators and student-athletes
- increase understanding of the APPLE Model
- increase ability to impact substance abuse problems
- increase student ability to confront teammates
- increase student leadership skills
- increase knowledge of alcohol, tobacco and other drug (ATOD) issues for student-athletes
- increase knowledge of effective ATOD prevention programs
- create a clearly defined, measurable action plan

In order to assess individual participants' progress in these areas, all conference attendees complete pre- and post-conference evaluation questionnaires.

The three areas that show the greatest improvement for conference participants are:

- I have the knowledge to develop effective substance abuse prevention programs for student-athletes.
- I can identify my athletic department's weaknesses regarding substance abuse prevention.
- I can identify my athletic department's strengths regarding substance abuse prevention.

The results for the 2012 APPLE conference participants show statistically significant gains in participant progress towards all but two APPLE learning outcomes ("I am confident in my ability to impact substance abuse problems" and "I believe I can be a positive role model for student-athletes at my school") using an independent-samples t-test for differences in means ($p \leq .001$). Appendix B provides a summary of the mean scores for all participants before and after the conference

OVERALL CONFERENCE EVALUATION

All conference participants are asked to complete extensive evaluations regarding numerous aspects of the conference. They are asked to rate the various areas on a scale of 1 (very poor) to 5 (excellent) and provide additional comments. The conferences received ratings at or above 4.5 (out of 5) indicating high overall satisfaction. The table below breaks down the scores by conference and attendee with a selection of the additional comments. A summary of all other components evaluated (hotel, food, speakers, materials, etc.) is located in Appendix C.

	Student-Athlete	Administrator	AVG
Charlottesville, VA	4.72	4.50	4.62
Huntington Beach, CA	4.90	4.70	4.80

General Comments

- The Apple conference inspired me to become more involved and be a leader – my voice is more effective than I thought.
- Very first Apple conference – overall an excellent experience. Learned some fantastic, useful information, met some wonderful people, will definitely try to be back.
- This is my 3rd year and I feel like it was the most informative and I'm excited to go back.
- I would highly recommend this conference to others and learn new information every year.
- We get a great deal from this conference – students – fellowship/share ideas with other schools – bring back ideas for our administrators – students input on policies, alcohol awareness week.
- This conference I feel has been one of the best ones I've been to. I think having everyone on the staff so personally involved creates a great atmosphere and will make people want to keep their own schools more! So I thank you for this awesome learning experience and excellent job!
- We missed last year, we were on the waitlist. That really slowed down our progress from years before. An email being sent out as to when to register would be great. It would be good to hold 3 weekend, east, central and west.
- This conference was very informative and will help our campus become more educated and hopefully help to drop the number of alcohol/drug/marijuana use on our campus of both students and student athletes. Excellent conference.
- It is truly an honor to attend the APPLE conference. It is the best conference for both student-athletes and administrators. This conference not only improves campuses and athletic departments, but it changes lives.
- It was great to have the opportunity to sit and take in all this information and then turn around and apply it to our own athletic department. It was great to get input and ideas of our student athletes. They have some great insight and ideas. Thank you for allowing us this amazing opportunity!
- Many conferences I have attended for education included spiral bound notebooks with power point notes. The only improvement I would think of… Tremendous job!
- Overall, I thought this was an excellent conference. As an administrator, I saw my student-athletes in a different light, and for the first time, I really was able to see how their minds work. Normally, I only see the athletic side. Rarely do I get to see the student side. I like the emphasis actually implementing what we've honed, discussed. Thank you for the opportunity to be here, for your mission, and your commitment to our student-athletes.

SPEAKER EVALUATION

In addition to the overall evaluation, each participant is asked to complete an evaluation of both the keynote and breakout speakers. The evaluation includes four quantitative questions and two open-ended questions. Below are the quantitative results of the evaluations. All sessions received a rating of 3.7 (out of 5) or higher. Appendix D provides a summary of each session with scores by both student-athlete and administrators along with a summary of their comments.

	HAZE	Sexual Assault	Keynote	Sexual Assault	Hazing	APPLE Success	Drug Testing	Mental Health
	Susie Bruce	One Student	Linda Hancock	No Woman Left Behind	Betsi Burns	Wilson & UCF	K. Turpin M.Bockelman	Bob Corb
1	4.75	4.65	4.55	4.45	4.55	4.45	4.3	4.3
2	4.83	4.9	4.8	4.6	4.35	4.4	4.35	4.3
3	4.88	4.8	4.55	4.6	4.3	4.3	4.2	4.4
4	4.88	4.9	4.8	4.6	4.6	4.35	4.5	4.3
Overall	**4.84**	**4.81**	**4.68**	**4.56**	**4.45**	**4.38**	**4.33**	**4.33**

	SAM	Step Up!	Supplements	Policy	Keynote	Mental Health	Facilitat-ion	Hazing
	Doug Everhart	Doug Everhart	Eric Patterson	Craig Littlepage	Chris Kilmartin	Jim Bauman	K. Willet C. Klecka	Rick Farnham
1	4.4	4.3	3.95	4.1	3.9	3.75	3.75	3.75
2	4.1	4.2	4.2	3.9	4.2	3.9	3.85	3.55
3	4.3	4.1	4.1	4.2	4.0	3.7	3.7	3.65
4	4.2	4.3	4.1	4.2	4.3	3.9	3.75	3.75
Over all	**4.25**	**4.23**	**4.1**	**4.1**	**4.1**	**3.8**	**3.75**	**3.7**

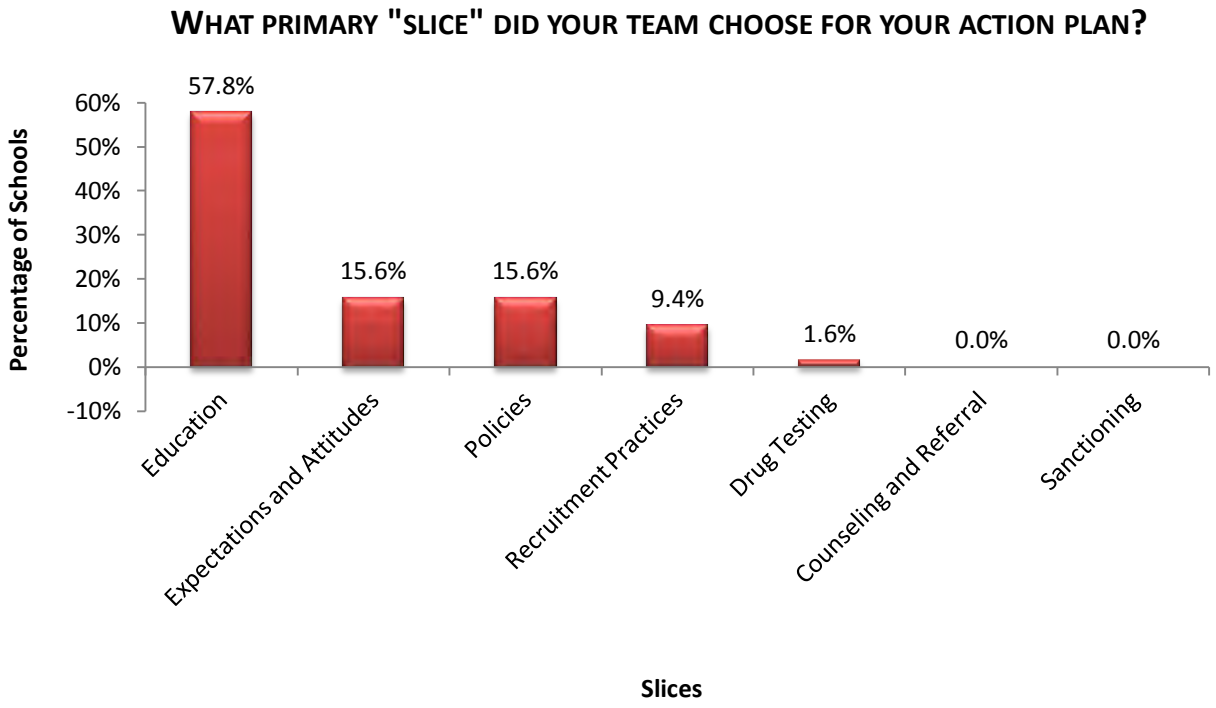
Number	Question				
1	The session gave me ideas/information for prevention programs on my campus.				
2	The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.				
3	This session motivated me to make a positive change in my personal behavior or on my campus.				
4	I would recommend this session to others.				
Scale	1 = Strongly Disagree	2 = Disagree	3 = Neutral	4 = Agree	5 = Strongly Agree

SPRING FOLLOW-UP EVALUATION

Team contacts were surveyed midway through the spring semester and asked to complete an online follow-up survey to assess their progress toward implementing their action plan and to revisit the impact of the conference. 63 out of 68 institutions responded (93% of schools).

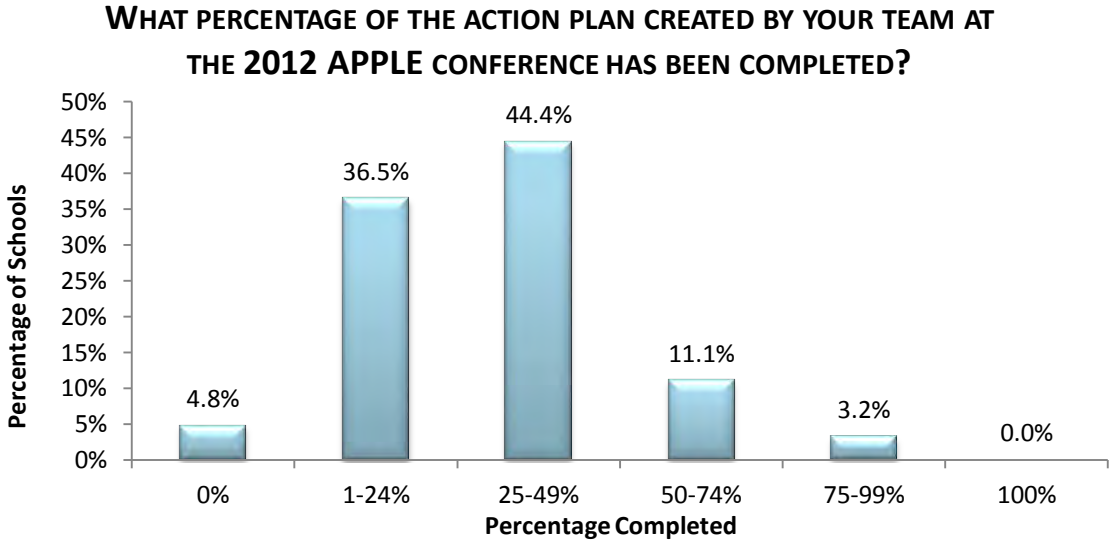
Prevention areas selected

The chart below shows which prevention areas campuses selected for their action plans. The greatest number of schools (57.8%) focused their action plans on the education slice, which is also the area that had the lowest average scores in the pre-conference campus assessment. An equal number of schools (15.6%) focused on policies and the expectations and attitudes slice and 9.4% selected the recruitment slice. Only one school selected the drug testing slice as their area of emphasis.



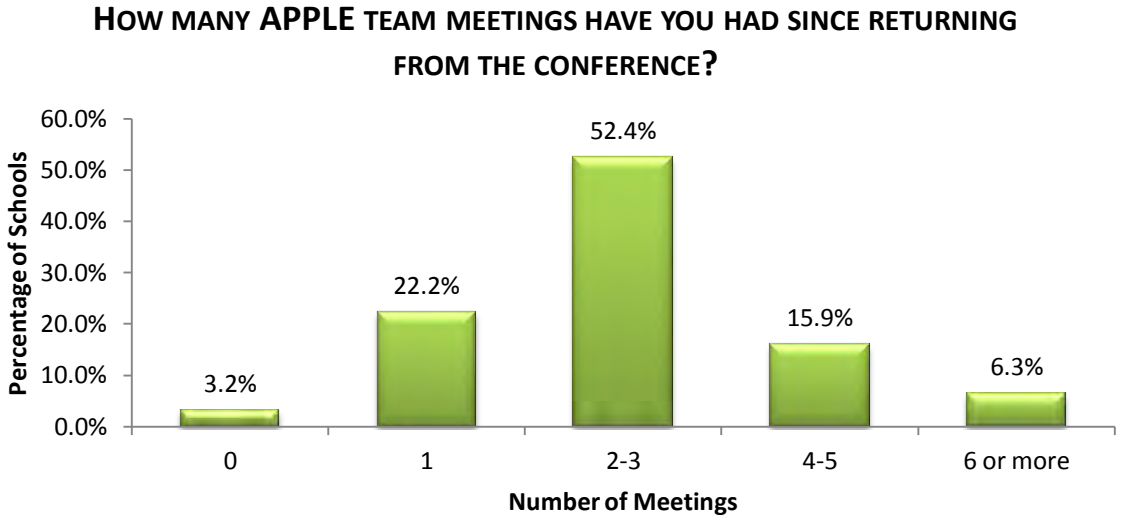
Progress toward goals

95% of schools (60 of 63) made at least some progress in their action plans including 28 (44%) that have completed more than 25% of their plan and 9 schools (14%) that reported that their goals are more than halfway complete. These actions demonstrate a commitment to improving the climate of substance abuse prevention within their athletics departments.



APPLE team meetings

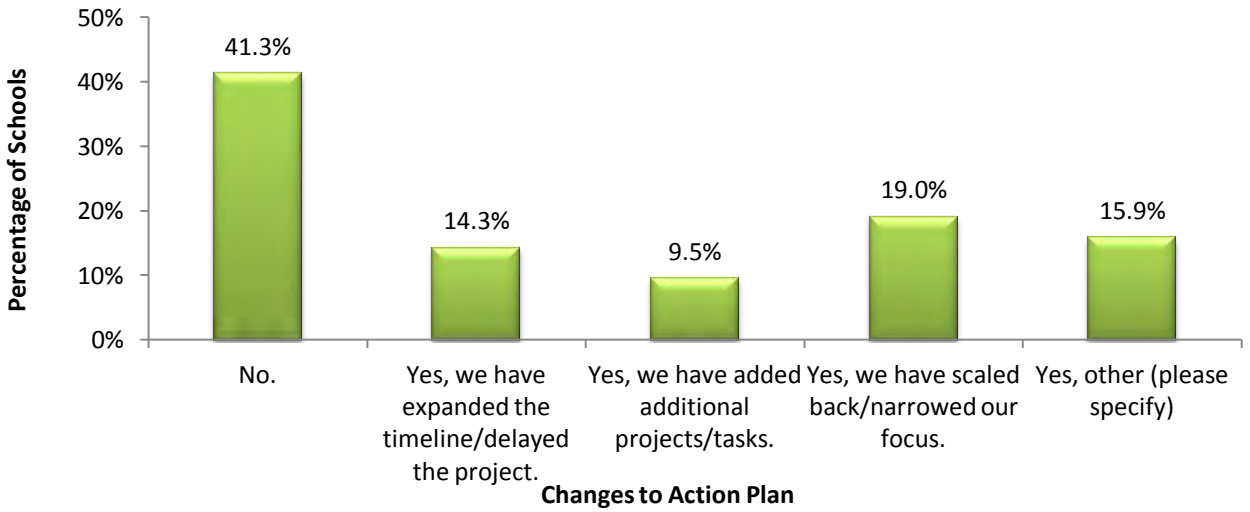
The majority of teams (97%) had at least one APPLE team meeting since the conference, including 14 schools (22%) that had four or more meetings. There is a highly significant ($p<.001$) positive correlation between the number of team meetings and percent goal completion, indicating that those schools which meet more often are more likely to have made progress.



Action plan revisions

Most schools – 58.7% or 37 schools – revised their goals. The primary revisions were to narrow the focus (19% or 12 schools) or expand the timeline/delay the project (14.3% or 9 schools). A similar number of institutions (15.9% or 10 schools) that answered 'Other' indicated that they experienced staffing/team changes. These data are consistent with previous years.

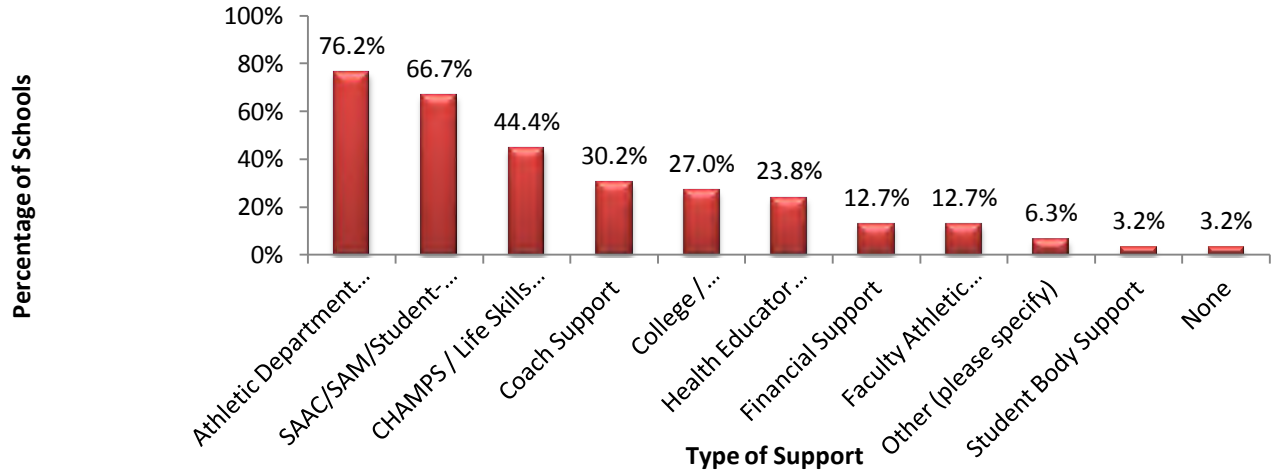
HAVE YOUR TEAM GOALS BEEN REVISED SINCE ATTENDING THE CONFERENCE?



Campus support

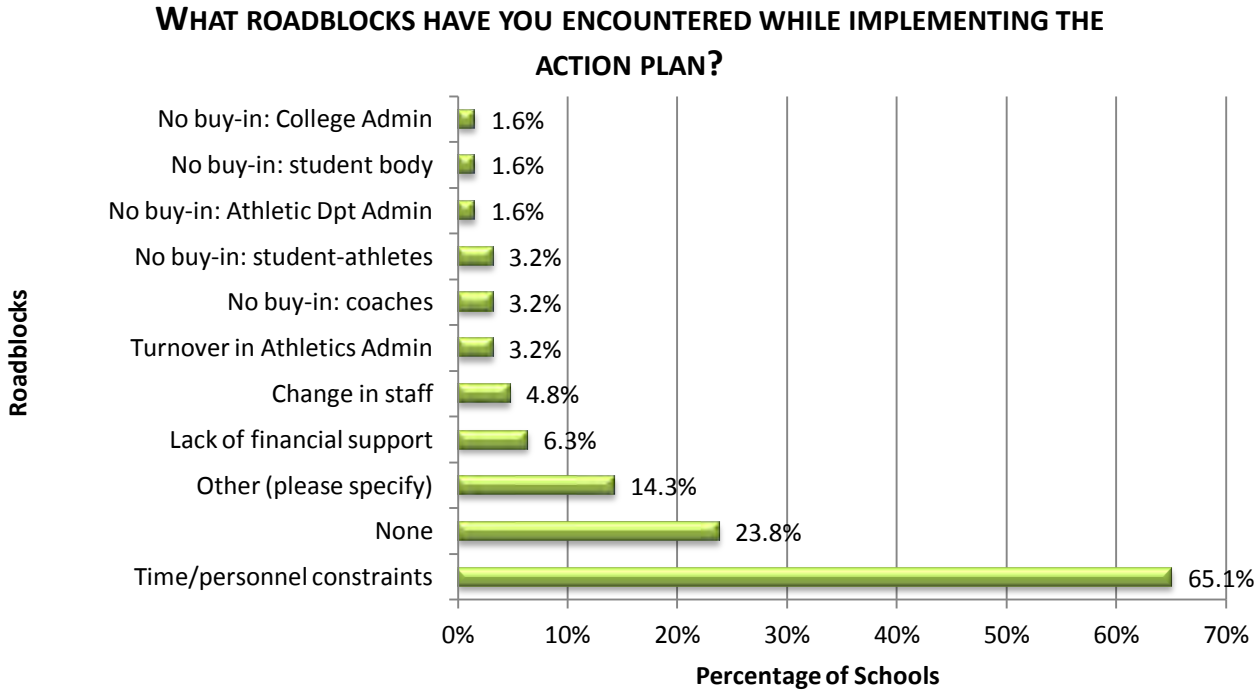
Every team (100%) discussed their action plans with at least one representative from their school that did not attend the APPLE conference. Three-quarters of team contacts (76.2%) indicated that they had received support for their action plans from their athletics departments. Many schools reported receiving support from SAAC, SAM, or other student-athletes (66.7% of schools) and CHAMPS / Life Skills (44.4% of schools). Only one team reported not receiving any support.

WHAT KIND OF SUPPORT HAS YOUR TEAM RECEIVED FOR YOUR ACTION PLAN?



Roadblocks to success

By a large margin, time and personnel constraints were rated as the most frequent roadblocks to implementing action plans and many contacts citied over-extended student-athletes as a particular problem. Similar to 2011, approximately a quarter of teams did not experience any roadblocks. Fourteen percent of schools reported "Other" and included reasons such as scheduling issues, facility space, and struggles to determine next-steps in the action planning process.



"It was great to have the opportunity to sit and take in all this information and then turn around and apply it tour own athletic department. It was great to get input and ideas of our student athletes. They have some great insight and ideas. Thank you for allowing us this amazing opportunity!"

FUTURE DIRECTIONS & NEW STRATEGIES

Based on assessment results, the 2012 APPLE conferences were successful and we look forward to implementing new ideas to enhance future conferences. We have begun to plan the 2013 conferences, which will be held in Charlottesville, Virginia on January 18-20 and in Indianapolis, Indiana on January 25-27. Planned enhancements to the 2013 conference experience are detailed below and are aligned with the five APPLE conference recommendation categories suggested by the NCAA.

1. Align with the priorities of the NCAA membership specific to student-athlete wellness and substance abuse education
    - Presentations at the 2013 APPLE conference are planned to address the following topics: alcohol, marijuana, sexual health, drug testing, supplements, hazing, peer education and bystander intervention.
    - The NCAA will share member issues and concerns at the May 2012 APPLE meeting. Topics for the 2013 conference will be finalized at the May meeting.
2. Design differentiated curriculum for first-time and repeat participants.
    - In order to orient new participants to the APPLE model and expectations, the following enhancements are planned:
        - Host 60-minute webinar in June or early July to provide a general conference orientation for those considering registration.
            - Webinar will then be posted to the APPLE website
        - Host 60-minute webinar in November to provide a conference overview for team contacts and other campus team members
            - Webinar will then be posted to the APPLE website
    - Create themed breakout blocks to enhance flow of conference and promote sharing of returning schools' experiences.
        - Blocks 1 & 2: content information on current issues (ATOD, sexual health, etc.)
        - Block 3: Successful action plans (returning school presentations)
        - Block 4: Implementation strategies (returning school presentations and APPLE conference staff)
3. Strengthen the support for the facilitator role within the participant team.
    - Host 60-minute webinar in November for team contacts and other administrators on effective team facilitation skills specific to the APPLE model.
            - Webinar will then be posted to the APPLE website
    - Create additional facilitator guides for use at and following the conference. The guides will be sent electronically to team contacts to allow them to customize them for their action plans.
4. Extend the participant pool and reach institutions who have not yet participated in the conference.
    - Identify Athletic Director, athletic trainer, CHAMPS/Lifeskills coordinator and health educator contacts at schools within a six hour drive of Indianapolis and Charlottesville
    - Send electronic marketing materials in late June/early July to:

        - All Choices grantees, schools within driving distance of the conference locations, NCAA listserves of CHAMPS/Lifeskills coordinators, athletics directors, athletic trainers, and others
        - BACCHUS listserv and Area Consultants closest to Indiana
        - NASPA listserve and ATOD Knowledge Community
        - Higher Ed Center
        - Network members
        - Others identified at May 2012 meeting
    - Partner with NCAA to develop other methods/strategies for conference promotion
    - Open registration in late August so schools have time to get approval to attend
    - Save 20% of team registration spots (n= 14) for schools that have not attended APPLE before
5. Follow-up on the actual action plans implemented at schools to create a repository of best practices in different kinds of schools.
    - New APPLE website is created and is being populated. The anticipated launch data is July 2012 (Appendix A)
        - A clearinghouse of model SAM programs has been compiled and will be included in the new website on a "SAM" page.
        - In summer 2012, will follow up with teams from the 2011 and 2012 conference to identify model programs for each slice. Highlights will be included on the website with representation from the three NCAA divisions as available.
    - Returning schools will be invited to share their past APPLE action plan successes through poster displays in the resource room, breakout session presentations, and program highlights shown on scrolling PowerPoint slides during lunch.

APPENDIX A: APPLE WEBSITE SCREEN SHOT

APPENDIX B: PRE-POST TEST DATA

		Mean	Change
I am comfortable working with the student-athletes on my APPLE Team.*	Pre test Post test	6.6540 6.8503	+ .1963
I am comfortable working with the administrators/coaches on my APPLE Team.*	Pre test Post test	6.6460 6.7861	+ .1401
I am confident in my ability to impact substance abuse problems at my school.	Pre test Post test	5.9501 6.1617	+ .2116
I am confident in my ability to confront a student-athlete about unhealthy choices.*	Pre test Post test	6.1235 6.2814	+ .1579
I have the knowledge to develop effective substance abuse prevention programs for student-athletes.*	Pre test Post test	5.3079 6.1662	+ .8583
I am a positive role model for student-athletes at my school.	Pre test Post test	6.4076 6.5000	+ .0924
I believe that peer education can positively affect other students' views about alcohol.*	Pre test Post test	6.2405 6.5631	+ .2956
I can identify my athletic department's strengths regarding substance abuse prevention.*	Pre test Post test	5.6129 6.1006	+ .4877
I can identify my athletic department's weaknesses regarding substance abuse prevention.*	Pre test Post test	5.3676 6.1841	+ .8165
Scale	1 = Strongly Disagree	4 = Uncertain	7 = Strongly Agree

**Statistically Significant*

APPENDIX C: OVERALL CONFERENCE EVALUATION RESULTS

	Hotel Check-In			Hotel Facilities			Food			APPLE Check-In		
	SA	AD	AVG	SA	AD	AVG	SA	AD	AVG	SA	AD	AVG
Charlottesville	4.80	4.89	**4.85**	4.84	4.56	**4.70**	4.67	4.51	**4.59**	4.84	4.85	4.85
Huntington Beach	4.92	4.85	**4.89**	4.95	4.96	**4.96**	4.85	4.81	**4.83**	4.93	4.84	4.89

General Comments

Hotel Check-In:

- Went smoothly, just bottlenecked with quite a few people – understandable.
- Smooth and quick.
- They were so friendly and excited to help us in any way possible.
- Very surprised that I was expected to room with someone from another school. We crammed into one room (3 of us) instead.
- Didn't know we needed a credit card for incidentals.

Hotel Facilities:

- The hotel was very nice and very accommodating to our needs.
- Hotel staff was wonderful.
- Need more space for activities.
- Cold shower and key card access didn't always work the first time
- Amazing!! Loved the beach and 24hr gym.
- Excellent all round from the room to the pool, the buildings and the location in relation to the beach and shopping plaza and eating places.

Food:

- Hotel staff was very accommodating with vegetarian meals
- The food was great. Lots of variety.
- Didn't care for the Asian style food that much.
- Great job of catering to dietary needs – gluten-free!
- Great! Perhaps have soy milk options for the lactose-intolerant.
- I was completely spoiled. Every meal raised the bar higher and higher. Very impressed.
- Excellent food all around, breakfast suggestion maybe pancakes.

APPLE Check-In:

- Great – flow + clear of what to do + apples!
- Everyone was friendly and organized! Everything in my opinion was well thought! Cool gifts.
- Where are the pens with Apples on the end?
- Green apples too!
- Very smooth and simple process.
- Friendly, welcoming, lots of enthusiasm which heightened my excitement for the conference.

	Introduction to Model			Campus Implementation			Taking APPLE Back Home		
	SA	AD	AVG	SA	AD	AVG	SA	AD	AVG
Charlottesville	4.47	4.22	4.35	4.42	4.26	4.34	4.47	4.41	4.44
Huntington Beach	4.70	4.49	4.60	4.65	4.33	4.49	4.75	4.31	4.53

General Comments

Introduction to Model:

- Very informative -> learned some valuable information to bring back to present to student-athletes at home. Certain facts shed new light on the issue of educating our student athletes.
- Great information. Could use some interaction to keep SA interested.
- Helpful to provide hard copies of campus survey results for each team member; time consuming and ineffective trying to share around the table.
- Opened new perspectives on what it means to have a healthy department through various means such as social events to bring people closer together.
- I liked the Turning Point technology used in the presentation to collect.
- Conference did an excellent job of providing info and energizing participants. I am excited to return and get started.

Campus Implementation:

- Useful information on how to select our 'slice' as well as making sure we don't take too big a bite out of what we want to change.
- More interactive
- Good length – opened for good conversation with the group. Like the format of quick talk and a lot of discussion time.
- Liked the worksheet you provided us!
- Good information but a bit lengthy/boring.

Taking APPLE Back Home:

- Quick and enjoyable.
- Excellent video!
- Encouraging, motivating – provides a lot of resources.
- Great closer to great weekend!
- Nothing was particularly helpful, but it was motivational.

	Value of Team Meetings			Conference Format/Structure			Session Topics			Support Materials		
	SA	AD	AVG	SA	AD	AVG	SA	AD	AVG	SA	AD	AVG
Charlottesville	4.77	4.75	4.76	4.62	4.51	4.57	4.40	4.31	4.36	4.41	4.18	4.30
Huntington Beach	4.89	4.66	4.78	4.68	4.51	4.60	4.65	4.24	4.45	4.70	4.36	4.53

General Comments

Value of Team Meetings:

- We talked a lot and really have this action plan that is realistic and I'm really excited about putting it into action.
- Wonderful to hear the student voices and hear their passion for change and education of their fellow athletes.
- Very good! Being able to check in with your team throughout the conference was very helpful.
- For our first APPLE conference it was a little overwhelming to take all the information and develop our action plan but we had some great discussion and came up with some awesome ideas.
- This was a great opportunity to discuss implementing an alcohol policy within our athletic department. Prior to this conference, we had no idea we did not have a uniform policy.
- Talks prepared/sparked many good ideas that resulted in long term goals and a beginning action plan.
- Based on presentations and speakers we were able to communicate the ideas in our meetings and then apply that information to our school through problem-solution method of analysis.
- I liked the team meetings during the conference, Made the distribution and collection of information from various speakers easier.

Conference Format:

- Like the structure -> speakers are long enough to gain valuable information, but not too long that people get restless. Breaks are well times as were meals, snacks, meetings, etc.
- 15 minute breaks really helped to keep me focused for presenters.
- Too much sitting throughout day – move an activity during Saturday.
- Very smooth, professional, and organized.
- Everything was scheduled and it flowed well.
- Loved the combination of breakout sessions and speakers. Small focused environment allowed communication and participation/interaction from student-athletes.
- The breaks were timed perfectly and it was great to be able to enjoy the location.
- I appreciate how you let us have "beach" breaks.
- I liked having breakfast start at 8 vs. earlier. It allowed people time to work out. Frequent team meetings were helpful.
- One of the better conferences to show practical application and specific plans upon learning to take back.

Session Topics:

- Keynotes were great. Everyone in our group loved the breakout sessions but some were elementary like "how to have the difficult conversation"
- I loved being able to hear from other schools SAM programs. Helped see what works and what doesn't.

- I liked student-led sections showing their previous actions and plans – good to know roadblocks/other sessions very informative – good blend of slices.
- Need to have session on teaching out SA to be better facilitators and peer educators so they can take skills back home.
- Maybe suggest to speakers to incorporate more active/interactive component to the sessions.
- Helped connect and apply what we learned.
- Funny, informational, interactive, and inspired me as a student-athlete to want to implement change in my school and my athletic department.
- Some sessions needed clearer focused topics. Ex: the supplement talk didn't need to talk about alcohol. The hazing talk was not very beneficial – no ways to make changes on campus.
- A keynote speaker about nutrition would be interesting – as well as a speaker with more info on the effects of alcohol on an athlete's body and performance
- Very diverse and interesting topics covered!
- Liked all of my breakouts, but could probably combine the Drug Free Sport presentations to one or two rather than three.
- A workshop on social networking and how it affects alcohol and drug abuse would be relevant for our society and SA's.

Supporting Materials:

- Felt like I could really pay attention because I knew all power points would be online
- Resource room was empty the first evening. Need more supplies.
- Supply more posters that we can use around campus.
- Very helpful to have info and resources to bring back.

	Friday Night Activity			Saturday Night Activity		
	SA	AD	AVG	SA	AD	AVG
Charlottesville	4.30	4.09	4.20	4.26	4.20	4.23
Huntington Beach	4.52	3.57	4.05	4.28	3.88	4.08

General Comments

Friday Night Activity:

- I would really enjoy a true mixer for administrators – no agenda just meeting others and learning more about what they do.
- Gave me the opportunity to interact with other student athletes in a very engaging manner.
- Awesome! The staff at the beginning made a huge difference. They got us pumped up!
- Even though I was extremely tired I still met a ton of people and had a great time.
- It was very relaxing and fun to interact with everyone on a more personal, but silly level. Had a great time.
- I don't enjoy ice breakers, but it was actually a lot of fun. It was just a little too long.

Saturday Night Activity:

- Liked t-shirt swap, but was too tired because of time change to stay much later.
- Great team-builders to bring back to campus and the students seemed to have a blast! Always love the t-shirt swap too!
- I thought too much time was set aside for the night activity.
- I wanted free time to hang out with the people I met.
- It was fun, maybe could have had a more social aspect, maybe a dance competition, a non-alcohol dance party possibly.
- It was great to meet new people and I will be able to bring these activities back to campus.
- I would make the session a little bit shorter to keep everyone's attention.
- I liked the activities from previous (like the golf course) where build things and get to go around and try other things, etc.

APPENDIX D: SPEAKER EVALUATION SUMMARIES

Keynote Speakers

Presenter: Linda Hancock
Session Title: Molecules that Monkey with your Mind
Location(s): Charlottesville, VA & Huntington Beach, CA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
	CV/HB	CV/HB
The session gave me ideas/information for prevention programs on my campus.	4.5/4.6	4.6/4.5
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.7/4.8	4.9/4.8
This session motivated me to make a positive change in my personal behavior or on my campus.	4.5/4.7	4.6/4.4
I would recommend this session to others.	4.8/4.9	4.8/4.7

How can you apply what you learned in this session when you return to campus?

- Use the power point and add our own data.
- Love the message. Let's educate about making choices instead of repeating or negative reinforcement.
- Snowball ideas or questions when I don't have enough clickers. I plan to use the pour an ounce and genetic snowflakes to SAAC members to increase their knowledge.
- Promote the idea to other student athletes that "Each of us is a seed of divinely inspired responsibility"
- I can take the shot glass and show my friends the proper way to pour 1 oz.
- Rather than telling them no, make them realize they can be smarter.
- I hope to become a voice of advocacy for alcohol awareness. I plan to relay information I have gathered with my university and help to plan new events.
- Encourages me to use "less formal" methods to introduce this material
- Always get questions about genetics and drug categories from students, more confidence having this conversation
- To prevent littering of cigarettes, I like the idea of engaging middle/high school students with VCU's "Keep Your Butts Off the Ground"

How could this session be enhanced?

- More time for presentations.
- Include sex, nutrition, and sleep.
- Break sex and drugs apart.

Presenter: Chris Kilmartin
Session Title: Guy Fi: The Fictions that Rule Men's Lives
Location(s): Charlottesville, VA & Huntington Beach, CA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
	CV/HB	CV/HB
The session gave me ideas/information for prevention programs on my campus.	3.7/4.1	3.8/4.0
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.2/4.6	3.7/4.3
This session motivated me to make a positive change in my personal behavior or on my campus.	3.9/4.3	3.8/4.1
I would recommend this session to others.	4.2/4.6	4.1/4.2

How can you apply what you learned in this session when you return to campus?

- Discuss gender identity during BASICS appointments – explore impact on drinking choices. Also, femininity and alcohol.
- Be aware of the social stereotypes and educate people on campus about what their actions/words represent. Example: you throw like a girl.
- Look into exploring masculinity discussions on campus.
- Campaigns on men and sexism/gender biases, etc.
- Didn't even think of connection between gender and alcohol. Incorporate this topic in discussions with and programs for students.
- I would have never thought about including gender in our education.
- Creating programs to educate men on masculine behavior.
- Add gender education/awareness to our educational programs.
- Noticing and changing my own perceptions of men and women.

How could this session be enhanced?

- More interaction with crowd.
- Some additional interaction during presentation would be helpful, but overall it was great.
- Break up lecture format with video clips of ads, products, etc. that support points. Power Point slide could be visually enhanced.
- Demonstrate application for those attending women's college
- More on how women's/men's athletic teams are disrespectful to each other.
- I think more concrete examples of how to engage coaches/admin. In this conversation would have been helpful.
- Smoothness of transitions.

Hazing

Presenter:	Betsi Burns
Session Title:	Turning Purple Haze into Positive Purple Pride
Location(s):	Charlottesville, VA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
The session gave me ideas/information for prevention programs on my campus.	4.3	4.8
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.4	4.3
This session motivated me to make a positive change in my personal behavior or on my campus.	4.2	4.4
I would recommend this session to others.	4.5	4.7

How can you apply what you learned in this session when you return to campus?

- New ideas for programming/team building
- Show Haze at our campus
- Meet with AD to suggest ways to welcome first-years without exclusion
- Create lists of activities that teams can do that do not promote hazing
- Establishment of social networking policy
- More positive team building activities
- Talk to teammates about boundaries, each others' backgrounds
- Awareness of activities that are actually hazing

How could this session be enhanced?

- Handouts
- More specific examples of hazing and prevention

Presenter:	Rick Farnham
Session Title:	Hazing
Location(s):	Huntington Beach, CA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
The session gave me ideas/information for prevention programs on my campus.	4.0	3.5
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	3.8	3.3
This session motivated me to make a positive change in my personal behavior or on my campus.	4.0	3.3
I would recommend this session to others.	4.0	3.5

How can you apply what you learned in this session when you return to campus?

- Relay this information to my teammates and other athletic teams on campus.
- I can talk with my team and suggest better ways of team bonding.
- Talk to students about potential hazing issues on campus

How could this session be enhanced?

- Increased engagement with the audience
- Get audience involved. Ask us questions. Let us tell our hazing stories.
- More specifics about action plans- what's in the contract? What did the team meetings look like?

Presenter: Susie Bruce
Session Title: HAZE: A Feature Documentary
Location(s): Huntington Beach, CA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
The session gave me ideas/information for prevention programs on my campus.	4.7	4.8
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.96	4.7
This session motivated me to make a positive change in my personal behavior or on my campus.	4.96	4.8
I would recommend this session to others.	4.96	4.8

How can you apply what you learned in this session when you return to campus?

- Look up info on cost of ambulance and publicize, show the video to students.
- Having our teams watch this as part of orientation.
- Send a clear message – Get help!
- Making an impact on as many as I can, making them realize that binge drinking can take you to a nightmare from which you never wake up.
- Remind people to stay in the buddy system and tell friends to help someone when you're not sure.
- Tell people about what happened to keep them from drinking heavily.
- Know when to make a call to help someone and prevent deaths.

How could this session be enhanced?

- More interactive/full-version.
- Small group (lead) discussions.
- Ideas for change/prevention. Yes, here's the problem but how do I fix it.

Mental Health

Presenter: Jim Bauman
Session Title: Sports Psychology – An Evolving Resource for Student-Athletes
Location(s): Charlottesville, VA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
The session gave me ideas/information for prevention programs on my campus.	3.8	3.7
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.0	3.8
This session motivated me to make a positive change in my personal behavior or on my campus.	3.8	3.6
I would recommend this session to others.	4.0	3.75

How can you apply what you learned in this session when you return to campus?

- Great analogies, wonderful speaker, very engaging
- Plan to take the handouts back to use as educational tools
- Better understand when to refer students to counseling
- Training your "software" mind to perform on demand
- Create a help line

How could this session be enhanced?

- Have more information about successful ways to utilize counseling services on campus when it's not possible to hire specifically for athletics
- Needed to bring it back to what were are here for—disconnected from APPLE slices
- Talk more in depth about what sports psychology is and what problems it deals with rather than the history of the profession

Presenter: Bob Corb
Session Title: Strategies for Effectively Addressing the Behavioral/Mental Health Needs of Student-Athletes at Your Institution
Location: Huntington Beach, CA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
The session gave me ideas/information for prevention programs on my campus.	4.3	4.3
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.2	4.4
This session motivated me to make a positive change in my personal behavior or on my campus.	4.3	4.5
I would recommend this session to others.	4.1	4.5

How can you apply what you learned in this session when you return to campus?

- Identify allies and collaborate with student affairs professionals on campus
- Normalize psychological services; collaborate with our "experts."
- Passive programming around stigma of mental health.
- Adjust/implement self-disclosure. Seek sport specific health care providers.
- I plan to reach out to faculty and staff, and find those interested in supporting athletics. Promote advocacy for oneself.

How could this session be enhanced?

- Links to example policies and health care provider trees – personal profile.
- Include how administrators/ staff can help identify and refer SA's
- Give specific projects of how to implement change

Sexual Assault

Presenter: Kelly Addington & Becca Teider
Session Title: Let's Talk About "IT"
Location(s): Charlottesville, VA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
The session gave me ideas/information for prevention programs on my campus.	4.6	4.7
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.8	5.0
This session motivated me to make a positive change in my personal behavior or on my campus.	4.8	4.8
I would recommend this session to others.	4.9	4.9

How can you apply what you learned in this session when you return to campus?

- Present what we learned here to SAAC and continue to spread the word of empowerment.
- Educate on what is definitions, different ways to help and example on how to prevent, discuss and handle.
- I will always make sure that there is a sober friend.
- Educate my campus on how to empower themselves and avoid sexual violence.
- I can be that friend to a victim of sexual assault. I now know how to properly react positively and supportively to a friend or acquaintance.

How could this session be enhanced?

- More ways to communicate; how to address those on our campus.

Presenter: April Grolle, Lauren Bryeans & Sabrina Sadler
Session Title: No Woman Left Behind
Location(s): Huntington Beach, CA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
The session gave me ideas/information for prevention programs on my campus.	4.4	4.5
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.6	4.6
This session motivated me to make a positive change in my personal behavior or on my campus.	4.6	4.6
I would recommend this session to others.	4.6	4.6

How can you apply what you learned in this session when you return to campus?

- Get the word out. Work on bystander intervention.
- Discuss with other administrators to show the video to all student athletes. There are numerous ways to intervene.
- Implementing Step up! and NWLB foundations into student training.
- I plan to spread this message to my roommates and teammates and share the story.
- Make sure everyone has a plan when going to party.

How could this session be enhanced?

- More focus on applicable transferring skills.

Drug Testing

Presenter: Mark Bockelman
Session Title: Developing Drug Testing Policies
Location(s): Charlottesville, VA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
The session gave me ideas/information for prevention programs on my campus.	4.3	4.0
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.3	4.3
This session motivated me to make a positive change in my personal behavior or on my campus.	4.1	4.0
I would recommend this session to others.	4.4	4.3

How can you apply what you learned in this session when you return to campus?

- Re-evaluate our policy
- Review alcohol policy on campus
- Help to begin discussion of sanctions for either drug testing and also alcohol policy

How could this session be enhanced?

- Allow more time for small group discussion to gather ideas from other students/administrators
- Handouts

Presenter: Kathy Turpin
Session Title: Developing Drug Testing Policies
Location(s): Huntington Beach, CA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
The session gave me ideas/information for prevention programs on my campus.	4.5	4.5
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.5	4.3
This session motivated me to make a positive change in my personal behavior or on my campus.	4.4	4.2
I would recommend this session to others.	4.6	4.6

How can you apply what you learned in this session when you return to campus?

- Start a drug testing program.
- Help our AD get a drug policy test under way and make it unpredictable.
- My school is working on making a policy. Now we have a starting point.
- How deterrence can be applied as leverage.
- Speak with other administrators to devise a plan.
- Random testing is a must. Policy is needed and written.
- Reviewing drug testing policy every year.

How could this session be enhanced?

- More examples of how to test.
- More discussion time.

Supplements

Presenter:	Eric Patterson
Session Title:	Dietary Supplements: Worth the Risk**?**
Location(s):	Charlottesville, VA & Huntington Beach, CA

Evaluation Questions	**Average Score** 5 = Strongly Agree	
	Student Athletes	**Administrator**
	CV/HB	CV/HB
The session gave me ideas/information for prevention programs on my campus.	3.9/4.0	4.1/3.8
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.2/4.2	4.6/3.9
This session motivated me to make a positive change in my personal behavior or on my campus.	4.3/4.3	4.1/3.9
I would recommend this session to others.	4.1/4.0	4.4/3.8

How can you apply what you learned in this session when you return to campus?

- Educate students more to be sure their supplements do not contain a banned substance.
- Utilize information and activity in SAM programs on campus.
- I can use the information I have learned to make programs to educate on dietary supplement use.

How could this session be enhanced?

- Stay on a smaller, more focused area within nutritional/dietary supplements.
- Practical ideas/suggestions on ways to implement programs on campus.
- Focus more on why supplements are not effective.

Policy

Presenter: Craig Littlepage
Session Title: Creating & Improving Athletics Department ATOD Policies
Location(s): Charlottesville, VA & Huntington Beach, CA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
	CV/HB	CV/HB
The session gave me ideas/information for prevention programs on my campus.	3.9/4.3	4.2/4.1
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	3.6/4.4	3.7/4.4
This session motivated me to make a positive change in my personal behavior or on my campus.	4.0/4.4	4.0/4.1
I would recommend this session to others.	3.8/4.4	4.1/4.3

How can you apply what you learned in this session when you return to campus?

- Talk to athletic director to push for a more consistent policy in department
- Use the info in order to get our AD, coaches, student-athletes, etc. on the same page with our policies
- Educated me to approach the policy change to our department students and administrators one in the same, starting with basic evaluations
- Present to AD-importance of continuity across the board, unique to our school, very good discussion
- Gain input from a variety of groups, use the questions presented in helping evaluate and revise policies
- All student-athletes need to know and understand policies
- In reviewing current policies, make sure to get other (legal, SA's, college standards personnel) look at them to have all work together (policies in agreement, you don't have to reinvent wheel).
- I loved the exercise of thinking like an AD as far as how I would develop a policy. This is out of my typical job duties, but an interest of mine in the future.

How could this session be enhanced?

- A breakdown on how to actually structure or change the policy, examples of actions or broken down steps/process
- Would have liked to have seen their policy as an example to see the structure and foundation
- More interactions, examples of actual plans used at UVA
- Improved visual aids, more recent info (1990 policy info is outdated)

Past APPLE Teams Success Stories

Presenter: Wilson College & University of South Florida
Session Title: Successful APPLE Action Plans
Location(s): Charlottesville, VA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
The session gave me ideas/information for prevention programs on my campus.	4.5	4.4
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.4	4.4
This session motivated me to make a positive change in my personal behavior or on my campus.	4.3	4.3
I would recommend this session to others.	4.4	4.3

How can you apply what you learned in this session when you return to campus?

- Provided resources that can be applied when doing our action plan
- It helped that this presentation came from actual student-athletes
- Good ideas for getting students involved in the education process
- Great job by the students! I was impressed by their initiative

How could this session be enhanced?

- Add a third presenter because the two shared great ideas
- Give more time and larger group collaboration
- Would love to see more universities presenting, often info is much more useful than general sessions—suggest adding 2-3 more university break outs

Facilitation

Presenter: Katie Willet & Cari Klecka
Session Title: Keys to Effective Facilitation
Location(s): Charlottesville, VA & Huntington Beach, CA

Evaluation Questions	Average Score 5 = Strongly Agree	
	CV	HB
The session gave me ideas/information for prevention programs on my campus.	3.9	3.6
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.0	3.7
This session motivated me to make a positive change in my personal behavior or on my campus.	3.8	3.6
I would recommend this session to others.	3.9	3.6

How can you apply what you learned in this session when you return to campus?

- Guidelines, rules, and experiments can be used to have better meetings
- Use the model when developing an agenda or meeting
- Allows to educate how to facilitate, participate and run meetings with fellow co-workers as well as students
- Reinforced some things I had been thinking about for facilitation, distinguishing process/content was helpful
- I went to facilitator conference so it was a good review.

How could this session be enhanced?

- More lively, at a different time during the conference, more interaction
- More interaction—maybe give some concrete examples
- Presentation was a bit elementary for this crowd, would have preferred more interaction/group discussion—topic more appropriate for students—they are new to leadership positions
- Facilitate more than lecture, it's been a long day of traveling so people are tired; keep in mind that many of us have been facilitating for a while, most of use aren't beginners

Bystander Intervention

Presenter: Doug Everhart
Session Title: "I Care About You": How to have the Difficult Conversation
Location(s): Charlottesville, VA & Huntington Beach, CA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
The session gave me ideas/information for prevention programs on my campus.	4.2/4.6	4.3/4.1
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.1/4.7	4.3/3.8
This session motivated me to make a positive change in my personal behavior or on my campus.	4.2/4.4	4.1/3.7
I would recommend this session to others.	4.1/4.7	4.2/4.1

How can you apply what you learned in this session when you return to campus?

- Going to research implementing Bystander Intervention/StepUp programs.
- Creating a student mentor program – want group to go through step-up training.
- Having the ability to communicate with others who are participating in risky behavior will be extremely useful on my campus.
- I can apply the skills from this session to approach situations I've noticed on campus.

How could this session be enhanced?

- I wish it had been noted on the agenda that this session is really intended and targeted to students. I had hoped to hear more about theories/research associated with StepUp as well as how some campuses are creatively incorporating/training students in StepUp
- Time for examples/practice situations.

Student-Athlete Mentor Programs

Presenter: Doug Everhart
Session Title: Student-Athletes, Peer Education and Building a SAM Program
Location(s): Charlottesville, VA & Huntington Beach, CA

Evaluation Questions	Average Score 5 = Strongly Agree	
	Student Athletes	Administrator
The session gave me ideas/information for prevention programs on my campus.	4.4/4.1	4.7/4.5
The way the session was delivered (format, presenter style, use of AV, handouts) assisted me in the learning process.	4.2/4	4.6/3.5
This session motivated me to make a positive change in my personal behavior or on my campus.	4.4/4.3	4.4/4
I would recommend this session to others.	4.3/4.2	4.5/3.8

How can you apply what you learned in this session when you return to campus?

- How to try to keep interest in SAM growing through recruitment
- I am thinking of creating a database for UCF student-athlete alumni
- Start and implement planning for SAM and running the program
- Sparked ideas to develop fresh orientation programs
- Loved the 3 different perspectives on the program, love panel style

How could this session be enhanced?

- Need representation for institutions that do not have a wellness coordination or resource person on staff (advisor) for SA i.e. Division III programs where we wear many hats and are not experts in this area
- Maybe have copies/handouts of the programs each student in the panel has talked about
- Time for more questions, answers were informative but long, talked a lot about campus resources but not all schools have these resources
- Provide specific direction from Doug on his direction for implementing program.

2012-15 *CHOICES* GRANT RECIPIENTS

Institution	Year 1	Year 2	Year 3
Agnes Scott College *DIVISION III* Elizabeth Douglass - Grant Program Administrator 141 East College Avenue Decatur, GA 30030 404/471-5238 edouglass@agnessott.edu	$14,955	$9,800	$5,000
University of Alabama – Huntsville *DIVISION II* Larry Cantor – Dir. of Counseling Ctr., Disab. Supt. Svcs. 301 Sparkman Drive Madison Hall 136 Huntsville, AL 35899 256/824-6203 Larry.cantor@uah.edu	$14,641	$9,910	$5,000
Bridgewater State University *DIVISION III* Anne Doyle – Coordinator – Outreach Ed 131 Summer Street Bridgewater, MA 02325 508/531-2274 adoyle@bridgew.edu	$15,000	$10,000	$4,970
University of California – Irvine *DIVISION I* Doug Everhart –Interim Dir. & Alc. Programs Mngr. Student Center G-319 Irvine, CA 92697 949/824-2296 everhart@uci.edu	$15,000	$10,000	$5,000

Chestnut Hill College *DIVISION II* Krista Bailey Murphy – Dean of Student Life 9601 Germantown Avenue Philadelphia, PA 19118 215/248-7142 murphyk@chc.edu	$15,000	$10,000	$5,000
Colorado State University – Pueblo *DIVISION II* David Arnold – Coord., ALC & Other Drug Prev. 2200 Bonforte Blvd. Pueblo, CO 81001 719/549-2121 David.arnold@colostate-pueblo.edu	$14,650	$9,000	$5,000
Grinnell College *DIVISION III* Jennifer Jacobsen – Wellness Coordinator 1201 10th Avenue Grinnell, IA 50112 641/269-3704 Jacobsen@grinnell.edu	$13,864	$6,075	$4,675
Immaculata University *DIVISION III* Scott Jones – Head Athletic Trainer 1145 King Road Immaculata, PA 19345 610/647-4400 sjones@immaculata.edu	$15,000	$10,000	$5,000
Johnson State College *DIVISION III* Camille Campanile –Coord. Of Student Ath. Dev. 337 College Hill Road Johnson, VT 05656 802/635-1468 Camille.campanile@jsc.edu	$15,000	$10,000	$5,000
University of Missouri – Kansas City *DIVISION I* Ursula Gurney – Associate Athletic Director 5100 Rockhill Road Kansas City, MO 64110 816/235-5093 gurneyu@umkc.edu	$14,957	$9,627	$5,080

The University of North Carolina @ Charlotte *DIVISION I* Angela Allen – Student Health Center 9210 University City Boulevard Charlotte, NC 28223 704/687/7366 Angela.allen@uncc.edu	$15,000	$10,000	$5,000
Pacific Lutheran University *DIVISION III* Jennifer Thomas – Asst. Athletic Dir., Compliance 1010 122nd Street S Athletics Dept. Tacoma, WA 98447 253/535-8109 thomasjl@plu.edu	$15,000	$10,000	$5,000
Salisbury University *DIVISION III* Dawn Chamberlin – Coach, Women's Field Hockey 1101 Camden Avenue Salisbury, MD 21801 410/548-2588 drchamberlin@salisbury.edu	$15,000	$10,000	$5,000
Texas Christian University *DIVISION I* Sparkle Greenhaw – Dir., Alc and Drug Ed Ctr. TCU Box 297740 Fort Worth, TX 76129 812/257-7100 k.greenhaw@tcu.edu	$14,820	$9,750	$4,700
Yale *DIVISION I* Andy Dunn – Asst., Athletic Dir., Varsity Sports Admin 20 Tower Parkway New Haven, CT 06511 203/432-1436 Andrew.dunn@yale.edu	$14,970	$10,000	$5,000

TO BE DISTRIBUTED

Health and Safety Webinars

Timeline

I. Develop overall teaching and learning objectives.

- Align with educational programming learning objectives.
- Create outcome metrics for each topic.

II. Develop framework for webinars.

- Standard opening and/or closing that is included in all webinars.
- Include boiler plate language to include in all webinars.

III. Develop general evaluation.

- Include framework to evaluate topic-specific learning objectives.
- Develop outcome metrics.
- Include polling and post-webinar evaluations.

IV. Develop topic-specific webinars

- Research scientific references to develop content.

Health and Safety Webinars

The health and safety principal of the National Collegiate Athletic Association's constitution provides that it is the responsibility of each member institution to protect the health of, and provide a safe environment for, each of its participating student-athletes.

Overall Teaching Objectives

To provide:

- Guidance in accomplishing this objective and to assist member schools in developing a safe intercollegiate athletics program.
- Guidance on health and safety topics relevant to intercollegiate athletics, applicable to a large population of student-athletes, and not accessible in another easily obtainable source.
- Guidance in developing health and safety policies appropriate for its intercollegiate athletics program.
- Quality education based on a diversified lens of athletics, academics, and research.
- Education that incorporates research, technology and best teaching practices.
- Promotion and maintenance of a healthy and safe environment for student-athletes participating in intercollegiate athletics.
- Engaging segmented content targeting a student-athlete and coach; an administrator; and medical personnel.

Outcome Metrics:

- All participants will be required to register in order to track numbers and general demographics (division, current position)
- Competence will be determined through a free CEU/CME post test for medical participants voluntarily.

Concussion: Impact on Academics

Learning Objectives

To:

- Understand prevention methods to lower the risk of concussion.
- Understand the progression of return to participation following clearance by medical staff.
- Understand the impact of concussion on student-athlete academic performance.

- Develop a campus policy for academic accommodations.

Sickle Cell Trait

Learning Objectives

To:

- Learn and understand in general how sickle cell trait can interact with exercise.
- Understand the available processes for confirming sickle cell trait status.
- Understand the importance for the student-athlete to take care of oneself and report symptoms of acute illness.
- Understand under which circumstances a student-athlete's activity may need to be modified .
- Understand how to recognize and respond to a sickling crisis.
- Develop a willingness to interrupt instances of extreme exertion that may put student-athletes at risk.
- Understand the components of appropriate counseling for individual with SCT.

Emergency Action Plan (EAP)

Learning Objectives

To:

- Learn the top 10 causes of sudden death in sports.
- Understand the key elements of a comprehensive Emergency Action Plan.
- Understand staff roles and responsibilities within in an Emergency Action Plan.
- Understand campus models for placement and planned access to an AED
- Understand the importance of practicing the EAP with all athletics staff at least yearly.
- Understand the role of a post event catastrophic incident guideline.

Lightning Safety

Learning Objectives

To:

- What you Need to Know: Learn the Facts and Risks of lightning strikes.
- You and Your Team:
    - When Thunder Roars, Go Indoors.
    - Understand the flash-to-bang method for determining when to seek shelter and/or resume athletics activity.
    - Identify the characteristics of a safe shelter and location of safe shelter sites of common athletics venues.
    - Understand the criteria for Return to Activity following a lightning delay.
    - Understand the proper response if someone is struck by lightning.
- Safety Plans.
    - Personal safety/team practices.
    - Large Venue.
    - Use of electronic monitoring systems.

Exertional Rhabdomyolysis

Learning Objectives

To:

- Understand the general physiological process of rhabdomyolysis.
- Understand the signs and symptoms of rhabdomyolysis.
- Understand how to prevent this condition from occurring.
- Understand the risk factors and need for gradual implementation of new training programs.
- Develop a willingness to interrupt instances of extreme exertion that may put student-athletes at risk.

Heat Illness

Learning Objectives

To:

- Understand the signs and symptoms of heat stress, including heat exhaustion and exertional heat stroke.

- Understand the Importance of identifying heat stress and immediate referral to a medical professional.
- Understand potential risk factors for heat illness.
- Understand heat illness prevention strategies.
- Develop a willingness to interrupt instances of extreme conditions that may put student-athletes at risk.

Acknowledgements

Each webinar will be hosted by the NCAA and sponsored by representative medical, health, or other expert group organization. Webinars will be co-branded as appropriate.

Resources

All webinars will have an extensive resource list available post event as well as the webinar itself.

A scientific reference used to develop the content will also be posted.

All webinars will require a written script 2 weeks prior to the scheduled date.

TO BE DISTRIBUTED

NCAA Drug- Education and Testing Video Segments:

Intro

In order to assure a level playing field and to protect student-athlete health and safety, the NCAA supports a comprehensive approach to drug- use deterrence, and emphasizes this is a shared responsibility, among all of its members-- the national office, conferences and member schools, administrator, coaches and student-athletes. NCAA drug testing, along with clear policies and effective educational programs, contributes to a campus environment that supports healthy choices and respect for the game. And because MOST Student-Athletes do not use and abuse drugs, NCAA programs support their efforts to play and stay drug free.

Play Clean – Banned Drugs

The World Anti-doping effort seeks to preserve what is intrinsically valuable about sport. This is often referred to as the spirit of sport; it is how we play true. The NCAA contributes to this

effort by banning the use of performance-enhancing and street drugs, to protect the health of its student-athletes, and to ensure fair play. Sometimes student-athletes feel pressure to use performance-enhancing substances, and just like any other college student, athletes might be tempted to use illegal drugs. NCAA drug testing provides one more good reason to stay drug free, because a positive drug test will result in loss of eligibility and suspension from sport.

NCAA banned drug classes.

There are eight banned drug classes that may cause a student-athlete to test positive during NCAA drug testing: anabolic agents like testosterone and steroids; stimulants like amphetamine, cocaine and ephedrine; peptide hormones like EPO and Growth Hormone'; diuretics, like water pills, and other urine manipulators or masking agents sold to try to beat a drug test; anti-estrogens like tamoxifen and clomiphene, used to modify unwanted effects of steroid use; beta-2 agonists like

formoterol; alcohol and beta blockers banned only in rifle; and street drugs which include marijuana, heroin and synthetic cannabinoids. It is not possible to name all substances that would fall under any banned drug class. This is because chemists continue to create new synthetic substances that are harder to detect, and they use various and confusing names to identify these newly created banned drugs, which makes it hard to include a complete list of banned drugs. **It's important to you as an NCAA student-athlete to understand that all drugs chemically belonging to the class of banned drugs are banned, whether or not they are listed as an example.**

Also, some medications and supplements may contain NCAA banned substances. Prescribed or not, check out all medications with your athletic trainer or team physician.

If your own physician prescribes a medication, you still need to check it with your school's athletic trainer or team physician before you take it.

All medications you take should be listed with your sports medicine staff. This will allow you to request an exception if you test positive for the use of banned medications. For health and safety reasons, your athletic trainer must know what medications you are taking. And using someone else's prescription medication is not only illegal, but could result in a positive drug test penalty. **Even legal, over-the- counter products may contain NCAA banned ingredients.** As an NCAA student-athlete, YOU are responsible for anything you ingest.

Just so you know: the NCAA bans any tobacco use during practice and competition. This ban also applies to coaches, athletics personnel, and game officials. Everyone is responsible for ensuring that tobacco products are not used at practices or competitions.

Possible outtake

Let's take a closer look at these banned drug classes:

Anabolic Agents/Steroids

Anabolic steroids and other anabolic agents mimic testosterone in the body and are banned by all sports governing bodies as they provide an unfair advantage in sports; these hormones also can cause negative side effects such as injuries to tendons, ligaments and muscles as well as hormonal changes, which can change sex characteristics in both men and women. There is also a risk to mental health from steroid use, such as uncontrolled aggression, and depression related to steroid withdrawal.

Some over the counter dietary supplements might contain steroids that can cause you to test positive, and ordering steroid products over the internet without a legitimate prescription is a federal offense.

Testosterone boosters that contain steroids or other banned substances are also banned. Be cautious about using any supplement or medication. Check with your athletic trainer before using any of these products.

Peptide Hormones

These are hormones that promote normal growth and development, but when used inappropriately have the potential to enhance athletic performance and also can cause serious health effects, such as blood clots, tumors, and even death. These hormones may also be prescribed for medical issues, but are banned by all sports governing bodies when used outside of medical necessity.

Stimulants

Stimulants may be promoted to help students to stay awake, study, lose weight, or heighten performance. The use of stimulants during strenuous training and competition in hot weather can trigger heart and heat stress problems which can be fatal. Stimulants ar also prescribed for attention deficit disorder. Unless these substances are legitimately prescribed and medically necessary, they are banned for use in sport.

If you are prescribed a stimulant medication, such as Adderall or Ritalin, you must report that to your athletic trainer and provide medical documentation to receive approval to use these banned medications.

Diuretics and Other Urine Manipulators or Masking Agents

These products are often marketed as "water pills", and promoted for for weight loss or to beat a drug test. Diuretics lead to dehydration, which negatively affects your performance. Use of diuretics to beat a drug test does not work, because the NCAA tests for diuretics too.

Anti-Estrogens

These drugs may be prescribed for people undergoing fertility or cancer treatments, but also may be used to mask the side effects of anabolic steroid use. Use of anti-estrogens without documentation of a legitimate medical need can result in the loss of your eligibility.

Beta 2 Agonists

These drugs are used to treat asthma, but can also be used to unfairly enhance performance. The NCAA allows the use of inhalers for asthma, but otherwise bans the use of these substances.

Alcohol and Beta Blockers *The NCAA bans the non-medical use of beta blockers, medications that slow heart rate, and alcohol, currently only in rifle competition. You school may have other rules about alcohol use. Consuming alcohol can dehydrate student-athletes and generally detracts from athletic performance.*

Play Smart—Nutritional/Dietary Supplements

NCAA student-athletes need to understand the risks involved in the use of dietary supplements. Weight loss/weight gain, sexual performance enhancement and sports performance supplements are big business. But they **are not**

required to be tested for purity, effectiveness and safety prior to going to market. Have you ever seen an advertisement for a supplement that claims it can help you lose weight quickly, or increase your energy? Those supplement products can go on the shelves without substantiating those claims, and can carry labels that may not accurately reflect what's in the bottle. [POSSIBLY INSERT EXAMPLE FROM COURT CLAIMS TO REINFORCE] **USADA VIDEO**

Supplement products may contain NCAA banned substances that will cause a positive drug test, including anabolic agents, diuretics and stimulants. Many supplement products including energy products and vitamin packs, may contain multiple sources and high levels of caffeine. NCAA student-athletes can test positive if they consume too much caffeine; , how much caffeine varies by individual size and metabolism. The 15 mc/mL threshold for a positive test may occur by consuming about 500 mg of caffeine within a few hours of drug testing.

Student-athletes should avoid using over-the-counter medications, weight loss aids and energy products that contain caffeine. The NCAA warns student-athletes against use of these poorly regulated products, and not to rely on just anyone's advice. NCAA student-athletes should consult with the appropriate athletics department representative prior to using any supplement or medication. In the end, student-athletes assume the risk that supplement products may be mislabeled or contaminated with an undisclosed banned substance. **Ultimately, athletes are responsible for any substance they put in their bodies. Athletes, trainers and coaches should work together to avoid substances that can trigger a positive test.**

Check with the designated athletics staff before consuming any product other than food. Athletics staff and student-athletes can contact the Resource Exchange Center, or REC, to ask if any product ingredients are banned. The REC is supported by the NCAA as a credible and

confidential source of information about supplements, medications and NCAA banned drugs. ***REC INFO here (link)***

Play by the Rules – Drug Testing

NCAA drug testing protects student-athletes who play clean by supporting their choices and removing student-athletes from competition who test positive for using banned substances. In order to compete, at the start of each school year, all eligible student-athletes sign a consent form that details what to expect in NCAA drug testing and provides for consent to be tested. Student-athletes also sign the student-athlete statement, which confirms eligibility to compete and compliance with NCAA regulations related to drug testing policy and other eligibility requirements.

Most student-athletes don't use drugs and will not have to face a positive test. If a student-athlete has tested positive with any other

athletics organization, such as USA Track and Field, that must be reported on the student-athlete statement. A student-athlete under a suspension by another athletics organization may not be allowed to participate in NCAA competition. Student-athletes should check with theirschool's compliance director to assure forms are complete.

Possible outtake

The NCAA drug-testing program involves urine collection at post-season events and on college campuses throughout the year. You might get selected to be tested because you won an NCAA championship event or you may be randomly selected.

After you are officially notified that you've been chosen for a drug test, you must report to the drug-testing site at the designated time. You may bring along a witness to observe the drug testing protocol or just to lend support. During championship testing, you will be given time to

get your award, talk to reporters, or attend to other responsibilities after the championship before you come to the testing area. It is very important that you arrive on time or you may be penalized for failing to show up, which is the same penalty as a positive drug test.

Once you get into the testing area, you need to stay until you provide an adequate specimen. This means there has to be enough urine at the right concentration. If you need additional fluids, there are approved drinks for you to have while you are in the testing area. These drinks will not have caffeine, alcohol, or anything else that can show up on a drug test.

You have as much time as you need to provide an adequate urine specimen. While you're at the drug-testing site, ask the crew any questions you have about the collection procedure or about banned drugs in general.

The crewmembers are experienced in sports drug-testing and can answer any questions you might have.

When you think you're ready to give your specimen, you'll wash your hands and then select a beaker. Someone of your own gender from the drug-testing crew will keep you under constant observation, which.

means the crewmember will watch you give your urine specimen to prevent any tampering. This is an important step in the procedure because it ensures that the urine specimen is genuine and belongs to you.

Once you finish, you'll take your specimen to the processing table where you'll be able to assist the crewmember as he or she prepares your specimen for the laboratory. The crewmember will measure the concentration of your urine and your sample's pH. These

screens are done to make sure your specimen is appropriate to be sent to the laboratory.

The crewmember will then split your sample into the A & B bottles, making sure they are tightly sealed, and pack them for shipment to the laboratory.

Before you leave the site, you will confirm that the NCAA drug-testing protocol was followed.

The samples are transported from the testing site to the NCAA testing laboratory in accordance with the NCAA chain of custody protocol. Upon arrival at the NCAA laboratory, all safety seals are checked. Each sample container and bottle is checked and stored securely.

Because all samples are number coded, the laboratory staff does not know your name or the name of your school.

Well-trained technicians with years of experience in drug screening perform the actual analysis of the specimen. They use state of the art procedures and equipment.

Drug-testing results are reported to your director of athletics, who will contact you if necessary. If you test positive, you may request to be present or have a witness present when the B bottle is tested at the NCAA laboratory.

[back to core video]

The NCAA tests for steroids and masking agents in its year round program and adds street drugs and stimulants to its championship testing program. Many of your schools also test for these substances. Street drugs are not only harmful to your health and athletic performance; they are also illegal.

Any product that contains THC, the active ingredient in marijuana, is banned. And synthetic cannabinoids – sometimes referred to

as synthetic marijuana, are now illegal and banned for use by NCAA student-athletes.

Testing positive will affect your eligibility, which means you will not be able to compete in NCAA sports. If you have a positive test, your institution can appeal the findings on your behalf.

And if even one athlete tests positive at a championship, it could affect the entire team. Drug Testing provides protection to all student-athletes by deterring dangerous and unfair drug use from your athletics experience

Play Healthy – Health and Performance

The NCAA knows that most student-athletes don't use banned drugs, And drug-education programs support student-athletes to continue to make good choices. The best way to achieve peak energy and muscle building is to rely on

real food, appropriate training and conditioning, rest and recovery, and avoiding substance use. Student-athletes expend calories and fluids during their training and competition. Performance is enhanced by anticipating energy expenditures and providing fuel –FOOD – and hydrating beverages prior to activity and immediately afterwards. Untested supplement products are likely more performance detracting than performance enhancing, especially when they are used in place of food. **SCAN VIDEO.**

Alcohol use causes dehydration, and interferes with sleep and the body's natural testosterone and growth hormone production. Alcohol use increases fatigue and risk of injure, and decreases reaction time, coordination, balance strength and endurance – a major performance detractor.

If you want to learn more about how drugs and supplements can affect your athletic performance, go to the NCAA drug-education Web site at:

www.ncaa.org/health-safety

The NCAA hopes to foster a healthy and productive experience for student-athletes.

Play smart. Play by the rules.

Stay healthy and drug free!

April 2012

COACHES ASSIST

to deter alcohol and drug abuse.

Coaches Believe

- *Every student-athlete matters*
- *Most student-athletes will respond to clear expectations about alcohol and drug use*
- *We must step up and intervene when needed*
- *My behavior matters*

How To Recognize a Student-Athlete Needs Help (including emergencies)

How to Voice Your Concerns

How to Access Campus and Community Resources

COACHES ASSIST

Steps to Helping a Student-Athlete

- Notice problems
- Take problems seriously
- Feel responsibility for acting
- Check on protocol for your department/ school
- Initiate a conversation
- Make an appropriate referral
- Follow up as needed

How to Recognize a Student-Athlete Needs Help: Responding to an Emergency

- **If you are worried about imminent harm,** stay with the student and **call 911** ***immediately*** and then follow up with your campus protocol (typically a call to the Dean of Students).

- Police are well-trained in psychiatric emergencies and can take students to the Emergency Room for a full psychiatric consultation immediately.

- Use **"PUBS"** as a guide to the symptoms of alcohol overdose
  - **P**uking while passed out
  - **U**nresponsive to stimulation (pinch or shaking)
  - **B**reathing (slow, shallow or no breathing)
  - **S**kin (blue, cold or clammy)

If you see even one sign of alcohol overdose – Call 911

How to Recognize a Student-Athlete Needs Help: General Signs and Symptoms

Coaches are not expected to take on a counseling role, but the information below can help you identify student-athletes in distress and provide appropriate assistance. Here are some of the areas where students may display concerning behaviors:

Drop in grades/academic performance

Teammates express concerns

Sports performance changes

- Late for meetings/practice
- Increase in sport-related errors
- Disorganized or erratic performance

Self-destructive behaviors

- Alcohol/drug misuse
- Self-inflicted injuries

Changes in physical appearance

- Worsening personal hygiene
- Unkempt appearance
- Excessive fatigue
- Visible changes in weight

Changes in mood or interactions

- Disproportionate anger
- Negative statements
- Loss of interest in activities
- Social withdrawal
- Statements of hopelessness
- Marked changes in eye contact

How to Recognize a Student-Athlete Needs Help: Substance Abuse Problems

Student-Athletes may share experiences with staff that indicate a substance abuse problem. **Pay attention to learn more about problem severity** if you hear a student-athlete talk about any of these experiences, especially related to alcohol or other drug use:

- Had a hangover
- In trouble with police or other college authorities
- Had a memory loss (blackout)
- Missed class
- Damaged property, pulled fire alarm, etc.
- Got into an argument/fight
- Was hurt or injured
- Was nauseated or vomited
- Expressed sadness for two weeks or longer

How to Recognize a Student-Athlete Needs Help: Do You Need to Intervene?

- **Trust your instincts and take signs seriously.**
- If you experience unease about a student, it is important to pay attention to those inner signals and not disregard what you've observed.

EARLY identification and intervention increases successful outcomes.

How to Recognize a Student-Athlete Needs Help: When to Intervene

Diagnosis needs to be conducted by a trained professional. But if you're trying to determine if a referral is needed, here are some questions you can ask your student-athlete:

Use "CAGE" as a guide to determine if the problem is related to alcohol abuse.

- Have you ever felt you should **Cut down** on your drinking?
- Have people **Annoyed you by criticizing** your drinking?
- Have you ever felt bad or **Guilty about your drinking**?
- Have you ever had a drink first thing when you wake up to steady your nerves or get rid of a hangover (also known as an **Eye opener**)?

One "yes" answer suggests a *possible* alcohol problem.
More than one "yes" means that it is *highly likely* that a problem exists.

How to Voice Your Concerns: How to Prepare

- **Drum up your will power!** The conversation you will have is not easy, but it IS important.
- **Be aware of difficult times:** Holidays, birthdays, anniversary dates, etc. can increase unhealthy behaviors.
- **Get sobriety on your side:** When people are under the influence of alcohol or other drugs, they will be more resistant to your efforts to confront their behaviors.
- **Get sound advice** from professional colleagues to provide you with helpful phrases and approaches (see referral section).
- **Be prepared for opportunities.** If student-athlete approaches you with a concern, this indicates he/she needs to talk.

How to Voice Your Concerns: Confidentiality

When talking with the student, **avoid making sweeping promises of confidentiality.**

- Students who are suicidal need swift professional intervention and pledges of absolute confidentiality may make this more difficult.

How to Voice Your Concerns: While Talking to Your Student-Athlete

- **Indicate you want to help.**
- **Give your total attention** - look at the person.
- **Assure confidentiality** (within limits).
- **Keep it focused** - only address what you've personally noticed in the student's behavior – avoid hearsay .
- **Don't pile on criticism** - frame the conversation as a concern for well-being.
- **LISTEN.**
- **Ask questions only for clarification** without judgment.
- **Expect denial** (*see separate section for tips on how to handle this*).
- **Ask** if there is anything he/she needs.
- **Follow through–** help the student-athlete take next steps and be persistent.

How to Voice Your Concerns:
How to Share Your Concerns

- Talking about a problem does NOT make it worse. This is the first step toward getting help.
- Specifically point out signs you've observed and ask what is wrong.
- Keep the discussion focused on facts you have personally observed ("I saw you do [this]…. I heard you say [that]") and on your emotions concerning the student-athlete's behavior ("I feel scared for you when you….").
- **For example, you could say:**
  - "I wanted to talk to you because I notice you've been late to practice recently and you seem troubled. I'm concerned about you. Would you like to talk about it?"

How to Voice Your Concerns: Careful Listening

- Many students will have trouble articulating the source of their difficulties.
- Be available.
- Show interest and offer support.
- Try not to get upset or communicate your own personal judgments.
- Be calm, receptive and serious, conveying that you can tolerate hearing about their painful feelings.
- Sometimes what a student does ***NOT*** say is as important as what the student ***does*** say. Ask additional questions to help understand the root problem.

How to Voice Your Concerns: Handling Resistance

- It's normal for someone with a substance abuse problem to deny there is a problem. When this happens, direct confrontation is not helpful.
- Instead, try one of the following approaches:
  - **Reflect** – "I know it's not easy to hear what I have to say."
  - **Reframe** what you hear – "You're right, a lot of people on the team drink and I need your help to get them to realize how it hurts them."
  - **Emphasize the right to disagree** – "You don't have to agree with me, I'm just asking you to consider this before you continue."
  - **Emphasize personal choice and control**– People are likely to feel overwhelmed if they believe that their right to personal choice is being challenged. "I'm just offering information. You have the right to decide what to do."

How to Voice Your Concerns: Making the Referral

- **Encourage the student to see a professional counselor.** You may need to address the student's concerns about counseling.
  - "All sessions are confidential and free of charge."
  - "If you like, we can call the campus office now to set up an appointment."
  - "Being willing to get help for a difficult situation is a sign of strength and intelligence."

How To Access Resources: Take the Team Approach

Engage appropriate athletic staff

- Team Physician
- Athletic Trainers
- Life Skill Coordinator

Build relationships with campus resources who may include:

- Clinical Sports Psychologist
- Health and Counseling Center Staff
- Alcohol/Other Drug Educator
- Dean of Students

Identify and follow existing intervention and referral protocol with athletics department and/or your counseling center.

If you plan to make a referral, communicate plan with appropriate athletics staff (sports medicine and senior administrator) and **respect the confidentiality of the student-athlete.**

How To Access Resources: Respect Confidentiality

- **Do not discuss the student's name or problem in public areas** OR with anyone who does not have a direct need to know this information.
- You **can** contact campus and community resources for general information on how to get the student-athlete help **without** revealing the student's name or specific problem details.

How to Access Resources
Community Resources

Use your on-campus relationships/partners to connect you with the appropriate off-campus resources.

Where to start:

- Local emergency health clinical or treatment centers
- Local mental health association
- Hospital mental health/substance abuse center

Two free national resources to find local assistance:

Substance Abuse Treatment Facility Locator

Sponsored by the Substance Abuse and Mental Health Services Administration (SAMHSA) www.findtreatment.samhsa.gov/

Hazelden Treatment Hotline – 866-819-1927

Fee confidential 24-hour service. Can also identify a provider in your area.

Coaches Matter!

- Addressing alcohol & other drug issues is important for performance, health and life balance.
- Treatment works!
- Coaches and administrators can provide encouragement and support.
- By addressing substance abuse, coaches and administrators show they truly care for the well-being of their student-athletes.
- Link to It Matters video: NEED URL

Coming Soon….

- Check this site later in the spring of 2012 for a new section on:

  "How coaches can provide effective alcohol and other drug abuse prevention"

- For now, see the resources on the following slide

NCAA and Other Resources

NCAA Mental Health Handbook
www.ncaa.org/health-safety

Step Up! Bystander Intervention
www.stepupprogram.org

External Resources

BACCHUS Network
www.bacchusnetwork.org www.peoplepreventsuicide.org

Screening for Mental Health
www.mentalhealthscreening.org

Question, Persuade, Refer (QPR) Institute
www.qprinstitute.com

Jed Foundation
www.jedfoundation.org www.halfofus.org

Coaches Assist Resource

- Tell us what you think of the Coaches Assist resource and how we can make it better!
- Click on the link below to complete an anonymous 3-question survey.

- https://www.surveymonkey.com/s/GLFT3FW

- **THANK YOU!**

Governance Leadership Scorecard

Key Points

- The NCAA national office provides guidance and counsel to the membership regarding the creation, adaptation and change of NCAA rule, policies and procedures. We do this, in part, by providing service and leadership to and for the governance bodies and creating an environment for their success.

- To measure our progress, we developed the Governance Leadership Scorecard to assess whether governance body members believed they had what they needed from the NCAA staff to make informed decisions.

- This scorecard replaces the existing committee feedback mechanism which did not address many of the service deliverables necessary for informed governance and decision-making, nor did it seek input from individuals other than the committee chairperson.

- Given that all members of a committee vote and the NCAA staff is charged with creating an environment for success of the entire committee, it was necessary to create a new survey/scorecard instrument and process to include a fuller complement of services and a complete population of participants.

- We identified 10 performance measures that combined, will create the environment for success. We will survey each committee member on dual levels, first to rank the staff performance but also to rank the level of importance attached to that element on a scale of 1-10 with 10 being the highest ranking.

- It's important to note that these metrics are not intended to serve as a performance review of individual staff members instead to measure the staff interaction as a whole. The 10 performance measures are

    - Provide direction, guidance and leadership
    - Encourage, enable and facilitate collaboration and discussion.
    - Develop substantive and relevant agendas
    - Track, research and synthesize pertinent/relevant and high-priority topics and issues.
    - Develop insights and strategies and recommend solutions.
    - Provide appropriate data and information to support decision-making.
    - Effectively and appropriately influence outcomes in the best interest of intercollegiate athletics.
    - Demonstrate good communication skills including listening.
    - Coordinate logistics including meetings, conference calls, presentation equipment, and meeting materials.
    - Produce informative reports and communications in a timely manner.

- In order to understand whether we are providing service and leadership successfully, we will establish a baseline satisfaction rating based on all committee members' responses to questions in the scorecard.

- The survey will be administered annually during the month of June

- Data will be synthesized during the month of July and feedback provided to the MSAA Leadership team in August.

- Committee liaison questions should be directed to Delise O'Meally in the MSAA staff (domeally@ncaa.org)

December 2012

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
						1
2	3	4	5	6	7	8
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SUPPLEMENT NO. 80

June 2013

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
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