Journal of Athletic Training

VOLUME 35 • NUMBER 4 • OCTOBER–DECEMBER 2000

NATA

Official Publication of The National Athletic Trainers' Association

JOURNAL OF ATHLETIC TRAINING

www.journalofathletictraining.org

Official Publication of the National Athletic Trainers' Association, Inc **Volume 35, Number 4, October-December 2000**

INDEXES: Currently indexed in Focus on Sports Science & Medicine (ISI: Institute for Scientific Information), Research Alert® (ISI: Institute for Scientific Information), Physical Education Index, SPORT Discus (SIRC: Sport Information Resource Centre, CANADA), CINAHL (Cumulative Index to Nursing & Allied Health Literature), AMED (Allied and Alternative Medicine Database).

The *Journal of Athletic Training* (ISSN 1062–6050) is published quarterly ($32 for one-year subscription, $40 foreign) by the National Athletic Trainers' Association, Inc, 2952 Stemmons Freeway, Dallas, TX 75247. Periodicals postage paid at Dallas, TX, and at additional mailing offices.

POSTMASTER: Send address changes to: *Journal of Athletic Training* c/o NATA, 2952 Stemmons Freeway, Dallas TX 75247. CHANGE OF ADDRESS: Request for address change must be received 30 days prior to date of issue with which it is to take effect. Duplicate copies cannot be sent to replace those undelivered as a result of failure to send advance notice. ADVERTISING: Although advertising is screened, acceptance of the advertisement does not imply NATA endorsement of the product or the views expressed. Rates available upon request. The views and opinions in the *Journal of Athletic Training* are those of the authors and are not necessarily of the National Athletic Trainers' Association, Inc.

Journal of Athletic Training

Official Publication of the National Athletic Trainers' Association, Inc **Volume 35, Number 4, October-December 2000**

Original Research

Case Reports

Communications

Position Statement

Contents Continued

Departments


©2000 S-VC, Inc.
About the only time
your athletes
should be using water
over Gatorade.
Attention Athletic Trainers: If you want your athletes to be better hydrated, make sure they're drinking Gatorade.®
Gatorade is proven to hydrate better than water, because it's scientifically formulated to encourage greater consumption
and superior replenishment. So – if you're looking to combat dehydration, think of it as 32 ounces of prevention.
is *it* in you?™
www.gssiweb.com
Gatorade
thirst quencher

CEU Quiz

The CEU quiz for the December 2000 issue (Volume 35, Number 4) of the *Journal of Athletic Training* will be located in the January 2001 *NATA News*.

23rd Annual Student Writing Contest

Entries must be received at the following address by March 1, 2001:

NATA Student Writing Contest
Deloss Brubaker, EdD, ATC
Life College
1269 Barclay Circle
Marietta, GA 30060

For a detailed description of the contest rules, please refer to the September 1999 issue of the *Journal of Athletic Training*.

Statement of Ownership, Management, and Circulation

1. Publication Title	2. Publication Number	3. Filing Date
JOURNAL OF ATHLETIC TRAINING	1062-6050	9/28/00
4. Issue Frequency	**5. Number of Issues Published Annually**	**6. Annual Subscription Price**
Quarterly	4	$32.00

7. Complete Mailing Address of Known Office of Publication *(Not printer) (Street, city, county, state, and ZIP+4)*
National Athletic Trainers' Association
2952 Stemmons Freeway, Suite 200
Dallas, TX 75247-6196

Contact Person: Leah Stovall Jones
Telephone: 214-637-6282

8. Complete Mailing Address of Headquarters or General Business Office of Publisher *(Not printer)*
Same as #7

9. Full Names and Complete Mailing Addresses of Publisher, Editor, and Managing Editor *(Do not leave blank)*

Publisher *(Name and complete mailing address)*
Same as #7

Editor *(Name and complete mailing address)*
David Perrin, PhD
209 Memorial Gymnasium, University of Virginia, Charlottesville, VA 22903

Managing Editor *(Name and complete mailing address)*
Leslie Neistadt
P O Box 9517, Columbus, GA 31908-9517

10. Owner *(Do not leave blank. If the publication is owned by a corporation, give the name and address of the corporation immediately followed by the names and addresses of all stockholders owning or holding 1 percent or more of the total amount of stock. If not owned by a corporation, give the names and addresses of the individual owners. If owned by a partnership or other unincorporated firm, give its name and address as well as those of each individual owner. If the publication is published by a nonprofit organization, give its name and address.)*

Full Name	Complete Mailing Address
NATIONAL ATHLETIC TRAINERS' ASSOCIATION	2952 STEMMONS FRWY., STE. 200 DALLAS, TX., 75247-6196

11. Known Bondholders, Mortgagees, and Other Security Holders Owning or Holding 1 Percent or More of Total Amount of Bonds, Mortgages, or Other Securities. If none, check box → ☒ None

Full Name	Complete Mailing Address

12. Tax Status *(For completion by nonprofit organizations authorized to mail at nonprofit rates) (Check one)*
The purpose, function, and nonprofit status of this organization and the exempt status for federal income tax purposes:
☒ Has Not Changed During Preceding 12 Months
☐ Has Changed During Preceding 12 Months *(Publisher must submit explanation of change with this statement)*

PS Form 3526, September 1998 *(See Instructions on Reverse)*

13. Publication Title: JOURNAL OF ATHLETIC TRAINING
14. Issue Date for Circulation Data Below: SEPTEMBER 2000

15. Extent and Nature of Circulation	Average No. Copies Each Issue During Preceding 12 Months	No. Copies of Single Issue Published Nearest to Filing Date
a. Total Number of Copies *(Net press run)*	27,433	27,893
b. Paid and/or Requested Circulation (1) Paid/Requested Outside-County Mail Subscriptions Stated on Form 3541. *(Include advertiser's proof and exchange copies)*	26,398	26,652
(2) Paid In-County Subscriptions *(Include advertiser's proof and exchange copies)*	0	0
(3) Sales Through Dealers and Carriers, Street Vendors, Counter Sales, and Other Non-USPS Paid Distribution	154	116
(4) Other Classes Mailed Through the USPS	254	0
c. Total Paid and/or Requested Circulation *[Sum of 15b. (1), (2),(3),and (4)]*	26,806	26,768
d. Free Distribution by Mail *(Samples, compliment ary, and other free)* (1) Outside-County as Stated on Form 3541	0	0
(2) In-County as Stated on Form 3541	0	0
(3) Other Classes Mailed Through the USPS	0	0
e. Free Distribution Outside the Mail *(Carriers or other means)*	200	100
f. Total Free Distribution *(Sum of 15d. and 15e.)*	200	100
g. Total Distribution *(Sum of 15c. and 15f)*	27,006	26,868
h. Copies not Distributed	427	1,025
i. Total *(Sum of 15g. and h.)*	27,433	27,893
j. Percent Paid and/or Requested Circulation *(15c. divided by 15g. times 100)*	99%	100%

16. Publication of Statement of Ownership
☒ Publication required. Will be printed in the December issue of this publication. ☐ Publication not required.

17. Signature and Title of Editor, Publisher, Business Manager, or Owner: [signature] Date: 9/29/00

I certify that all information furnished on this form is true and complete. I understand that anyone who furnishes false or misleading information on this form or who omits material or information requested on the form may be subject to criminal sanctions (including fines and imprisonment) and/or civil sanctions (including civil penalties).

Instructions to Publishers

1. Complete and file one copy of this form with your postmaster annually on or before October 1. Keep a copy of the completed form for your records.
2. In cases where the stockholder or security holder is a trustee, include in items 10 and 11 the name of the person or corporation for whom the trustee is acting. Also include the names and addresses of individuals who are stockholders who own or hold 1 percent or more of the total amount of bonds, mortgages, or other securities of the publishing corporation. In item 11, if none, check the box. Use blank sheets if more space is required.
3. Be sure to furnish all circulation information called for in item 15. Free circulation must be shown in items 15d, e, and f.
4. Item 15h., Copies not Distributed, must include (1) newsstand copies originally stated on Form 3541, and returned to the publisher, (2) estimated returns from news agents, and (3), copies for office use, leftovers, spoiled, and all other copies not distributed.
5. If the publication had Periodicals authorization as a general or requester publication, this Statement of Ownership, Management, and Circulation must be published; it must be printed in any issue in October or, if the publication is not published during October, the first issue printed after October.
6. In item 16, indicate the date of the issue in which this Statement of Ownership will be published.
7. Item 17 must be signed.

Failure to file or publish a statement of ownership may lead to suspension of Periodocals authorization.

PS Form 3526, September 1998 *(Reverse)*

2001 NATA Continuing Education Committee Schedule of Events

District Lecture Series
(One-hour presentation during each district meeting)

"The Hand and Wrist in Sport"

districts 1 & 2	January 8, Providence, RI
district 3	May 20, Greenville, SC
district 4	March 9, Milwaukee, WI
district 5	March 17, Topeka, KS
district 6	TBD, Arlington, TX
district 7	March 23, Tucson, AZ
district 8	April 21, San Diego, CA
district 9	March 24, Atlanta, GA
district 10	March 24, Boise, ID

One-Day Workshop
(worth 7 CEU credits)

"Sacroiliac Joint: Evaluation and Functional Rehabilitation"

district 3	May 18, Greenville, SC
district 4	August 11, Naperville, IL
district 6	July 17, Arlington, TX
district 8	April 19, San Diego, CA
district 10	March 25, Boise, ID

Clinical Instructor Educator Seminar
(worth 6.8 CEU credits)

districts 1 & 2	January 7, Providence, RI
district 4	March 10, Milwaukee, WI
district 6	January 19, Fort Worth, TX (sold out)
district 8	June 18, Los Angeles (registration begins in March)
district 9	March 22, Atlanta, GA

Visit the Continuing Education Committee website at www.cewl.com and click on "Continuing Education" for more details, or call (800) 879-6282, ext. 140, or email lorim@nata.org.

Journal of Athletic Training 2000;35(4):403–406

www.journalofathletictraining.org

Metrecom Measurement of Navicular Drop in Subjects with Anterior Cruciate Ligament Injury

Mary K. Allen, MS; Ward M. Glasoe, MA, PT, ATC

Physiotherapy Associates, Cedar Rapids, IA

Objective: Research suggests that excessive pronation of the foot contributes to the incidence of anterior cruciate ligament (ACL) tears by increasing internal tibial rotation. Studies have documented greater navicular drop values in individuals with a history of an ACL tear using methods that may not accurately follow the motion of underlying bone. The purpose of our investigation was to compare the navicular drop of subjects with a history of ACL tears with healthy controls when measured by a Metrecom.

Subjects: Eighteen subjects previously diagnosed with a torn ACL were matched by age, sex, and limb to noninjured control subjects.

Design and Setting: Static group comparisons of navicular drop in subjects with an injured ACL and subjects having no history of ACL injury.

Measurements: A single investigator performed the measure of navicular drop. The position of the navicular tuberosity was digitized while the subject stood barefoot on a flat surface in subtalar joint neutral and in relaxed stance. Intrarater reliability was assessed using intraclass correlation coefficient and standard error of the measurement. An independent *t* test assessed the difference between the amount of navicular drop in the ACL group and the controls.

Results: Analysis of repeated measures, intraclass correlation coefficient (2,1), demonstrated intrarater reliability for the measure of navicular drop to be 0.90; the standard error of measurement was 1.19 mm. The independent *t* test showed a statistically greater amount of navicular drop in the ACL group.

Conclusions: Excellent intrarater reliability was demonstrated when using the Metrecom to measure navicular drop. Excessive subtalar joint pronation, measured as navicular drop, was identified as 1 factor that may contribute to ACL injury.

Key Words: pronation, internal tibial rotation, navicular bone

The contribution of abnormal biomechanics in the foot to the development of knee pathology is clinically important in the prevention and treatment of injury.[1–5] During weightbearing, the foot and knee act as interactive segments, with pronation of the foot and internal rotation of the tibia occurring simultaneously.[6] One mechanical function of the anterior cruciate ligament (ACL) in the knee is to limit the internal rotation of the tibia.[7] Studies suggest a contributing mechanism to ACL injury is excessive tibial rotation due to hyperpronation of the subtalar joint, with the resulting strain on the ACL increasing the risk of a tear.[8–10]

Navicular drop is a clinical measure of foot pronation,[11] defined as the change in height of the navicular bone when the foot moves from subtalar neutral to a relaxed weightbearing stance.[12] We identified 4 studies that investigated the measure of navicular drop in subjects with a history of an ACL tear.[8–10,13] Three studies documented significantly greater navicular drop in patients with ACL injuries when compared with matched controls.[8–10] Smith et al,[13] however, reported no difference between groups. Three of the studies[8,9,13] measured navicular drop by recording the vertical change in the position of a pen mark on the skin overlying the navicular tuberosity, a method previously described by Brody.[12] The height of the navicular tuberosity was first marked on a file card or measured by ruler with the subject in subtalar neutral. The subject was then allowed to resume normal weightbearing stance, and the height of the pen mark was again recorded. Loudon et al[10] measured the change in the most distal point of the navicular tuberosity, which although not specified, would indicate direct palpation was used to identify the measurement site.

Testing for reliability was reported in only 2 of these 4 studies. Smith et al[13] repeated measures on the noninjured control group (intraclass correlation coefficients [ICC] values were 0.72 for the left foot and 0.82 for the right). Loudon et al[10] reported excellent intratester reliability for their method (κ value of 0.87).

Three aspects of these previous studies are problematic. First, the reported intratester reliability of the navicular drop measure[10,13–15] varies greatly, depending on the skill of the tester and the level of control exerted over repeated foot placement. Not testing or reporting reliability severely limits the usefulness of the data. Second, measurement of a pen mark is subject to skin movement.[16] Third, tracking the change in height of a pen mark or bony landmark attempts to measure the vertical component of displacement; however, navicular drop includes movement components in the medial and anterior directions as well.[17]

The purpose of our investigation was 2-fold. First, we wanted to assess the reliability of the measure of navicular drop when using a Metrecom (FARO Medical Technologies Inc, Lake Mary, FL). The second purpose was to compare the Metrecom-measured navicular drop of subjects with a history of ACL tears with that of noninjured matched controls. The

Address correspondence to Mary K. Allen, MS, Physiotherapy Associates, 600 7th Street SE, Cedar Rapids, IA 52401. E-mail address: MaryKayAllen@aol.com

null hypothesis was that navicular drop would not differ between groups.

METHODS

This study received approval from the Institutional Review Board of Physiotherapy Associates, which also approved the informed consent form signed by each subject. Thirty-six subjects volunteered for the study. Eighteen subjects (12 men, 6 women) had a history of an ACL tear diagnosed by a physician. Sixteen subjects had tears confirmed by magnetic resonance imaging or arthroscopy; 2 tears were diagnosed clinically, with the diagnosis supported by a KT-1000 test. ACL-injured subjects were matched with control subjects by age, sex, and limb. Sixteen subjects in the ACL group had undergone reconstructive surgery, and 2 had been treated conservatively. The mean age of the ACL group was 29.9 ± 9.5 years; range, 18 to 49 years. The control group (mean age = 29.9 ± 8.6 years) had no reported history of ACL injury. No subjects had a history of foot or ankle trauma during the 6 months before testing.

A single examiner measured navicular drop using the Metrecom. The Metrecom is an electromechanical, 3-dimensional digitizer (Figure 1).[17] The Metrecom measures the 2 positional points in 3-dimensional space and calculates the change in distance for the investigator, who is blinded from the results during the test. Linear accuracy, repeatability, and linearity of the system have been demonstrated.[18] A mean accuracy of 0.9 mm was reported when the Metrecom was used to digitize a calibration device.[18]

Subjects stood on an elevated platform with their feet a comfortable distance apart. The probe of the Metrecom was placed directly under the tuberosity of the navicular (Figure 2). The subject inverted the foot while the examiner palpated the congruency of the talar head in the mortise joint for subtalar neutral position. Once positioned, the location of the tuberosity was digitized. The probe of the Metrecom remained positioned under the tuberosity as the subject then resumed a normal, relaxed stance, and the second point was digitized. To test for reliability, the measure was repeated on all subjects. The average of the 2 trials was computed as the measure of navicular drop. Both feet were tested in all subjects. The limb with the ACL injury was considered the test limb (10 right and 8 left limbs) and matched with the same limb from the control group.

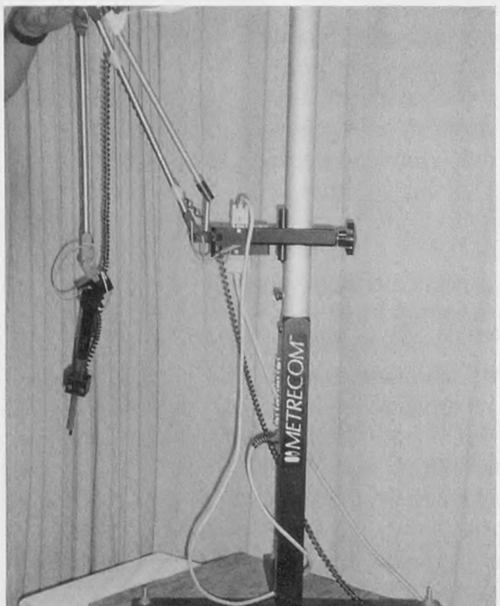


Figure 1. The Metrecom digitizing unit.

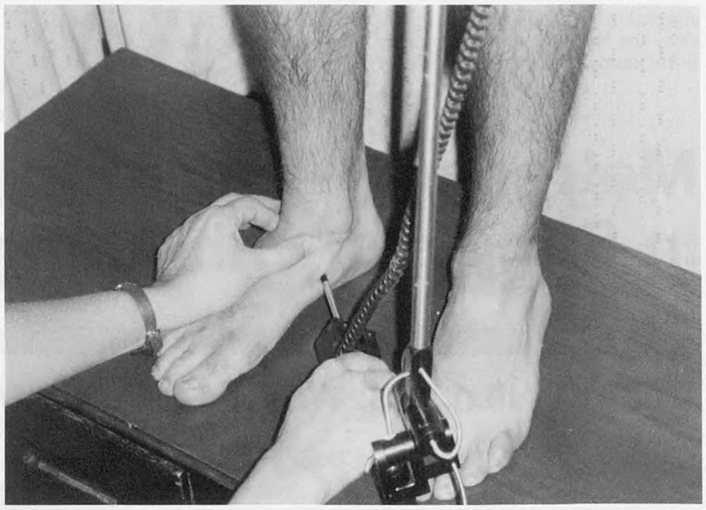


Figure 2. Metrecom measurement of navicular drop.

Intratester reliability of the navicular drop measure was determined using ICC (2,1),[19] and the standard error of measurement was calculated. We used an independent t test to assess the difference in navicular drop between groups.

RESULTS

Mean, standard deviations, and range of values for navicular drop are summarized in Table 1. ICC intrarater reliability was 0.90, with a standard error of measurement of 1.19 mm. The independent t test showed a statistically ($P < .05$) larger navicular drop in the ACL group. The noninjured limb of the ACL group also had a statistically ($P < .05$) larger navicular drop than the ipsilateral limb in the control group (Table 2). Values for navicular drop measured in ACL-injured subjects by sex are provided in Table 3. There was no significant difference between male and female values in the ACL-injured group.

DISCUSSION

Measurement Reliability

Navicular drop has been reported to be between 6 and 9 mm of movement in healthy, normal subjects.[8,17,20] The control group in our study consisted of a varied population of healthy, active individuals, and their mean navicular drop value (8.1 mm) fell within this range. In studies comparing ACL-

Table 1. Navicular Drop Values (mm)

Group	Mean	SD	Range
Control	8.1	2.8	3.5–13.5
ACL injured	10.5*	4.0	6.0–20.0

*Statistically significant difference from controls ($P < .05$).

Table 2. Navicular Drop Values of Noninjured Limbs (mm)

Group	Mean	SD	Range
Control	8.1	3.0	3.5–14
ACL injured	10.1*	3.6	5.5–16

*Statistically significant difference from controls ($P < .05$).

Table 3. Navicular Drop Values of ACL-Injured Group by Sex (mm)

Sex	n	Mean	SD	Range
Females	6	10.2	3.5	5.5–15.5
Males	12	10.7	4.3	6.5–20

injured with normal subjects, the mean navicular drop measurements have varied, depending on the subject population (Table 4).[8–10,13] One study's authors did not report mean values of the drop measure but grouped subjects according to low (< 6 mm), normal (6 to 9 mm), or high (> 9 mm) amounts of movement.[10] The ACL-injured groups in 3 studies[8,9,13] had significantly greater navicular drop values than healthy, normal subjects. The results of our study support these findings.

Smith et al[13] reported finding no significant difference in navicular drop between noncontact ACL-injured subjects and controls. It is not clear why their results contrast with other published data. They cite sex differences as a possible explanation, suggesting that women have smaller drop values. Thus, with an equal number of ACL-injured women[7] and men included in their study, the mean value for the injured group was diminished. We found, however, that the ACL-injured women in our study had a mean navicular drop value comparable with that of the men (Table 3). Both men and women in the ACL-injured group demonstrated greater navicular drop values than the control subjects. Other possible explanations offered by Smith et al[13] for the lack of difference between groups included sample size and the use of noncontact ACL-injured subjects. The other reported studies (Table 4) that did show significantly larger navicular drop values in ACL-injured subjects also included small sample sizes, similar subject populations, noncontact ACL-injured subjects, or a combination of these factors.[8–10]

Positioning of the subjects in our study followed the procedure described by Brody.[12] Previous studies[8–10,13] examining the relationship between ACL injuries and navicular drop followed Brody's method of manual measurement, but unlike Brody, varied the weightbearing position of the subjects between conditions. The subjects were seated (rather than standing) during measurement of the navicular position with the foot in subtalar neutral. The subjects then stood during the second measure. A study by Joyce et al[21] demonstrated that the variation in loading does have an effect on the overall movement of the navicular, with seated subtalar neutral to standing subtalar relaxed positioning resulting in larger navicular drop values.

Measuring the change of position of a pen mark on the skin is inexpensive and easy to perform in a clinical setting; however, navicular drop is a relatively small measure of displacement. Due to skin movement, the mark may not reflect the same relative position on the tuberosity as the navicular drops from subtalar neutral into pronation.[16] When measuring millimeters of bony displacement, movement of the skin can have a large impact on the measurement total. Added to the potential error of determining the subtalar neutral position,[15] the value of the measurement can be significantly affected; thus, issues of reliability and accuracy become critical when reporting navicular drop data.

We demonstrated excellent intratester reliability for the measurement of navicular drop. The probe of the Metrecom was positioned under the navicular tuberosity, and contact was maintained throughout the procedure. The probe's position was unaffected by skin movement. The examiner's hand never left the palpation points on the head of the talus; thus, a shift from subtalar neutral by the subject was easily detected and corrected. Controlling these factors of error in the measure of navicular drop provides data that are closer to reflecting the true motion that occurred.

Relationship of Navicular Drop to ACL Injury

McClay and Manal,[5] using 3-dimensional kinematic gait analysis, demonstrated greater internal rotation of the tibia during running in subjects with excessive foot pronation (11.1 ± 3.5° versus 8.9 ± 2.5° for the controls). Statistically significant higher peak velocities of foot eversion and knee flexion, as well as greater knee-flexion angles, were also seen in the pronation group when compared with normal subjects.[22] Excessive rearfoot pronation has been linked to overuse injuries of the knee.[23–24] Navicular drop studies suggest a link between excessive subtalar pronation and ACL injuries.[8–10] In our study, the similarity in navicular drop values between the injured (10.5 mm) and noninjured limb (10.1 mm) of the ACL group support the suggestion that the higher values were not due to the injury or surgical repair but inherent in the individuals with ACL injury. A bilateral comparison of the control group's navicular drop

Table 4. Reported Navicular Drop Values in ACL-Injured versus Healthy Subjects

Investigator	Group	Age (years)	n	Mean (mm)	Injury Mechanism
Woodford-Rogers et al[9]	ACL	19.1 ± 6.0	14 m*	8.4 ± 4.2	Mixed
	Control	18.1 ± 1.6	14 m	5.9 ± 2.4	
	ACL	19.5 ± 1.7	8 f*	5.0 ± 2.5	Noncontact
	Control	19.0 ± 1.2	8 f	3.0 ± 1.1	
Beckett et al[8]	ACL	22.9 ± 7.6	11 f, 39 m	13.0 ± 4.4	Mixed
	Control	21.8 ± 9.4	11 f, 39 m	6.9 ± 3.2	
Loudon et al[10]	ACL	26.5 ± 7.6	20 f	†	Noncontact
	Control	26.2 ± 7.8	20 f	†	
Smith et al[13]	ACL	21.1 ± 0.8	7 f, 7 m	6.3 ± 3.1	Noncontact
	Control	21.1 ± 2.0	7 f, 7 m	6.2 ± 2.6	

*m, males; f, females.
†Mean values not reported. Fifteen ACL subjects had > 9 mm of drop, 14 control subjects had < 9 mm of drop.

values also demonstrated similarity (8.1 mm for both limbs). These values were significantly lower than those of the ACL group, supporting the concept that excessive pronation may be a factor in ACL injury.

Further study is needed to determine if this is a causal relationship or related to generalized tissue laxity. The complexity of the anatomical relationships in the lower extremity,[25–27] coupled with the variance in subject population and data collection procedures, only allow us to suggest an association. ACL tears are a common injury. If excess pronation and tibial rotation contribute to the incidence of injury, screening and possible prevention with orthotic management of the foot could benefit many.

CONCLUSIONS

Our study is the first to report values for ACL-injured subjects using the Metrecom as the measurement tool. Excellent intrarater reliability for this method was demonstrated. The results of this study support previous work, which indicates that excessive pronation of the foot is a factor that may be associated with ACL injury.

REFERENCES

1. Busseuil C, Freychat P, Guedj E, Lacour JR. Rearfoot-forefoot orientation and traumatic risk for runners. *Foot Ankle Int.* 1998;19:32–37.
2. Nawoczenski DA, Cook TM, Saltzman CL. The effect of foot orthotics on three-dimensional kinematics of the leg and rearfoot during running. *J Orthop Sports Phys Ther.* 1995;21:317–327.
3. D'Ambrosia R. Orthotic devices in running injuries. *Clin Sports Med.* 1985;4:611–618.
4. Gross ML, Davlin LB, Evanski PM. Effectiveness of orthotic shoe inserts in the long-distance runner. *Am J Sports Med.* 1991;19:409–412.
5. McClay I, Manal K. Coupling parameters in runners with normal and excessive pronation. *J Appl Biomech.* 1997;13:109–124.
6. Donatelli RA. Normal anatomy and biomechanics. In: Donatelli RA, ed. *The Biomechanics of the Foot and Ankle.* 2nd ed. Philadelphia, PA: FA Davis; 1996:3–31.
7. Cabaud HE. Biomechanics of the anterior cruciate ligament. *Clin Orthop.* 1983;172:26–31.
8. Beckett M, Massie D, Bowers K, Stoll D. Incidence of hyperpronation in the ACL injured knee: a clinical perspective. *J Athl Train.* 1992;27:58–60.
9. Woodford-Rogers B, Cyphert L, Denegar C. Risk factors for anterior cruciate ligament injury in high school and college athletes. *J Athl Train.* 1994;29:343–346.
10. Loudon JK, Jenkins W, Loudon KL. The relationship between static posture and ACL injury in female athletes. *J Orthop Sports Phys Ther.* 1996;24:91–97.
11. Menz H. Alternative techniques for the clinical assessment of foot pronation. *J Am Podiatr Med Assoc.* 1998;88:119–129.
12. Brody DM. Technique in the evaluation and treatment of the injured runner. *Orthop Clin North Am.* 1982;13:541–558.
13. Smith J, Szczerba JE, Arnold BL, Martin DE, Perrin DH. Role of hyperpronation as a possible risk factor for anterior cruciate ligament injuries. *J Athl Train.* 1997;32:25–28.
14. Picciano AM, Rowlands MS, Worrell T. Reliability of open and closed kinetic chain subtalar joint neutral positions and navicular drop test. *J Orthop Sports Phys Ther.* 1993;18:553–558.
15. Sell KE, Verity TM, Worrell TW, Pease BJ, Wigglesworth J. Two measurement techniques for assessing subtalar joint position: a reliability study. *J Orthop Sports Phys Ther.* 1994;19:162–167.
16. Maslen B, Ackland T. Radiographic study of skin displacement errors in the foot and ankle during standing. *Clin Biomech.* 1994;9:291–296.
17. Mueller MJ, Host JV, Norton BJ. Navicular drop as a composite measure of excessive pronation. *J Am Podiatr Med Assoc.* 1993;83:198–202.
18. Smidt GL, McQuade KJ, Wei S. Evaluation of the Metrecom and its use in identifying skeletal landmark locations. *J Orthop Sports Phys Ther.* 1992;16:182–188.
19. Bartko J. On various intraclass correlation reliability coefficients. *Psychol Bull.* 1976;83:762–765.
20. Moul J. Differences in selected predictors of anterior cruciate ligament tears between male and female NCAA Division I collegiate basketball players. *J Athl Train.* 1998;33:118–121.
21. Joyce CJ, Arnold BL, Gansneder BM. Differences between navicular drop measures. *J Athl Train.* 1999;34:S71.
22. McClay I, Manal K. A comparison of three-dimensional lower extremity kinematics during running between excessive pronators and normals. *Clin Biomech.* 1998;13:195–302.
23. Brody DM. Running injuries: prevention and management. *Clin Symp.* 1982;39:1–36.
24. Messier SP, Pittala KA. Etiologic factors associated with selected running injuries. *Med Sci Sports Exerc.* 1988;20:501–505.
25. Saltzman CL, Nawoczenski DA. Complexities of foot architecture as a base of support. *J Orthop Sports Phys Ther.* 1995;21:354–360.
26. Tiberio D. Pathomechanics of structural foot deformities. *Phys Ther.* 1988;68:1840–1849.
27. Donatelli RA. Abnormal biomechanics. In: Donatelli RA, ed. *The Biomechanics of the Foot and Ankle.* 2nd ed. Philadelphia, PA: FA Davis; 1996:34–72.

Journal of Athletic Training 2000;35(4):407–411

www.journalofathletictraining.org

Long-Term Ankle Brace Use Does Not Affect Peroneus Longus Muscle Latency During Sudden Inversion in Normal Subjects

Mitchell L. Cordova, PhD, ATC; Chris V. Cardona, MS, ATC; Christopher D. Ingersoll, PhD, ATC; Michelle A. Sandrey, PhD, ATC

Sports Injury Research Laboratory, Athletic Training Department, Indiana State University, Terre Haute, IN

Objective: External ankle supports are widely used in sports medicine. However, ankle bracing in a healthy ankle over a sustained period has been scrutinized due to possible neuromuscular adaptations resulting in diminished dynamic support offered by the peroneus longus muscle. Although this claim is anecdotal in nature, we sought to investigate the effects of long-term ankle bracing using 2 commonly available appliances on peroneus longus latency in normal subjects. Our second purpose was to evaluate the effects of ankle bracing on peroneus longus latency before a period of extended use.

Design and Setting: A 3 × 3 × 2 design with repeated measures on the first and third factors was used in this study. All data were collected in the Sports Injury Research Laboratory.

Subjects: Twenty (12 men and 8 women) physically active college students (age = 23.6 ± 1.7 years; height = 168.7 ± 8.4 cm; weight = 69.9 ± 12.0 kg) free of ankle or lower extremity injury in the 12 months before the study and not involved in a strength-training or conditioning program in the 6 months before the study.

Measurements: We evaluated peroneus longus latency by studying the electromyogram of the muscle after sudden foot inversion.

Results: Application of a lace-up or semirigid brace did not affect peroneus longus latency. Additionally, 8 weeks of long-term ankle appliance use had no effect on peroneus longus latency.

Conclusions: The duration of the peroneus longus stretch reflex (latency) is neither facilitated nor inhibited with extended use of an external ankle support. Proprioceptive input provided by the muscle spindles within the peroneus longus does not appear to be compromised with the long-term use of ankle braces.

Key Words: peroneus longus reaction time, stretch reflex, ankle bracing, electromyography

Trauma involving the ankle and foot complex remains among the most common injuries in sport[1–3]; of these injuries, approximately 86% are sprains.[2] Furthermore, it has been estimated that nearly 1 million people in the United States suffer from acute ankle injuries annually.[4] Over the years, health care professionals have tried to prevent acute ankle sprains and chronic reinjuries by using various prophylactic measures, such as adhesive taping and commercially available ankle stabilizers. In an effort to combat this epidemiologic problem, many manufacturers have developed protective braces to support the ankle. Of these prophylactic devices, 2 basic types exist: lace-up and semirigid braces.[5] Lace-up braces are generally constructed of a soft canvas or nylon material, whereas semirigid braces contain a stirrup consisting of a thermoplastic material.[5]

Independent of any protective device, the musculature controlling the ankle and foot acts to provide a dynamic restraint against external forces. Specifically, the peroneus longus muscle acts as the primary defense mechanism against an inversion moment applied to the foot.[6] Because the peroneus longus plays a critical role in the dynamic support of the ankle-foot complex, its neuromuscular response during quasi-static[6–12] and dynamic inversion stress[13] has been well studied. The use of external ankle supports has been scrutinized due to testimony suggesting that supporting a healthy ankle can lead to the development of weakness in the surrounding muscles. Clinicians have surmised that long-term application of an ankle brace may cause the ankle's supporting structures to weaken and remodel so that they become dependent on this support. With the extended use of an ankle brace, the leg musculature's ability to respond to an external stimulus or perturbation may be delayed, thereby diminishing neuromuscular function and potentially placing the ankle-foot complex at risk for injury. To our knowledge, the effects of long-term ankle bracing on peroneus longus neuromuscular function in the healthy and chronically unstable ankle have not been addressed. Therefore, our primary purpose was to evaluate the reaction time, or latency, of the peroneus longus after long-term application of 2 selected ankle braces. Second, we were interested in assessing if ankle bracing affected peroneus longus latency before a period of extended use.

Address correspondence to Mitchell L. Cordova, PhD, ATC, Athletic Training Department, Indiana State University, Terre Haute, IN 47809. E-mail address: m-cordova@indstate.edu

METHODS

A 3 × 3 × 2 factorial design was used to determine if peroneus longus latency differed with 3 ankle brace applications, 3 ankle brace treatments, and before and after 8 weeks of extended use. The first independent variable (within-subjects factor) was test condition, with 3 levels: control (no brace), Active Ankle Training brace (Active Ankle Systems, Inc, Louisville, KY), and McDavid 199 (McDavid Knee Guard, Chicago, IL). The second independent variable (between-subjects factor) was treatment, with 3 levels: control (no brace), Active Ankle Training brace, and McDavid 199. The third independent variable (within-subjects factor) was time, with 2 levels: pretest and posttest. The dependent variable was peroneus longus reaction time, or latency.

Subjects

Twenty (12 men and 8 women) physically active, college-aged subjects (age = 23.6 ± 1.7 yrs; height = 168.7 ± 8.4 cm; weight = 69.9 ± 12.0 kg) volunteered for this study. Subjects had incurred no known ankle or lower extremity injuries in the 12 months before the study. Furthermore, all subjects were screened with a preparticipation survey to ensure that they had not been involved in a strength-training or conditioning program that would have altered the physiologic function of the peroneus longus in the 6 months before the study. Each subject read and signed an informed consent form approved by the School of Health & Human Performance Human Subjects Review Committee, which also approved the study. Each subject was required to report to the research laboratory on 2 separate occasions.

Instrumentation

A 4-channel, telemetered, biological signal-acquisition system (MP 100, BIOPAC Systems Inc, Santa Barbara, CA) recorded the electric activity of the peroneus longus and an analog signal derived from a switch positioned on the trap door. Disposable 10-mm Ag-AgCl surface electrodes (Ver Med, Bellows Falls, VT) arranged in a bipolar configuration were used to detect the electric activity and reaction time of the peroneus longus during sudden foot inversion. The raw electromyogram signal was digitally converted at 1000 Hz, amplified (gain set at 1000), and interfaced to a controlling desktop computer. The analog signal arising from the trap door was simultaneously sampled and time matched to the collected electromyogram signal. This analog signal identified the start of the inversion movement and allowed for assessment of peroneus longus latency. A custom-made inversion platform was used to produce the inversion movement. This device was constructed similarly to an inversion platform used in previous studies evaluating peroneus longus response.[14,15] The subjects stood on 2 separate, flat surfaces. At random, the platform was abruptly tilted to 35° of foot inversion by removing the primary support.

Testing Procedures

Subjects were introduced to the instrumentation and had the testing procedures explained before the pretest. The dominant lower extremity of each subject was first tested under each of the 3 ankle support conditions: control (no brace), Active Ankle Training brace, and McDavid 199 in a counterbalanced fashion. The dominant extremity was defined as the preferred extremity the subject would use to kick a soccer ball. Additionally, subjects performed this testing while wearing a cross-training shoe. The skin over the muscle belly of the peroneus longus was prepared for electrode placement by shaving any hair and cleansing with an alcohol pad to reduce skin impedance. Disposable, self-adhesive Ag-AgCl electrodes were placed over the muscle belly of the peroneus longus of the dominant extremity, as previously described.[16] The reference electrode was placed over the lateral malleolus of the same extremity.

Each subject was instructed to stand on both legs with the weight evenly distributed on the platform. We assumed that the weight distribution for all subjects was maintained throughout testing. The subject's elbows were flexed, with the hands on the hips. Once the subject was balanced, the platform under the subject's dominant extremity (tested ankle) was randomly dropped to a 35° angle. Dropping of the platform was random to eliminate premotor activity of the peroneus longus, as well as to prevent the subject from anticipating the release. Baseline activity of the peroneus longus was carefully evaluated to ensure that no heightened amplitude existed before the trap door was released, which would indicate premotor response. For safety purposes, 1 spotter was placed on each side in case the subject lost his or her balance. The pretest consisted of having subjects perform 5 trials of sudden foot inversion in which peroneus longus latency was measured. To assess peroneus longus latency accurately, the release of the trapdoor was indicated by an analog signal, which was synchronized with the peroneus longus electromyographic activity. Peroneus longus latency was defined as the time between the initiation of trapdoor release and the initial firing of the peroneus longus muscle.[6,11] Specifically, we measured the duration between the release of the trapdoor and the electromyographic amplitude associated with the second component (M2) of the stretch reflex.[17] The 5 scores for each testing condition were totaled, averaged, and recorded as the mean pretest score for each subject.

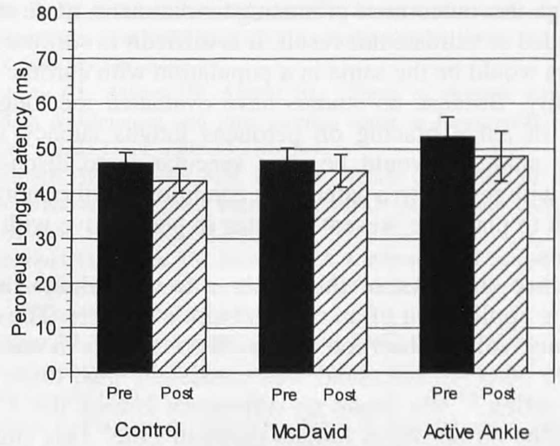
After the pretest, each subject was randomly assigned to 1 of the 3 treatment conditions (control [n = 7], Active Ankle [n = 6], or McDavid [n = 7]), to evaluate the potential long-term effect of each condition. For each brace condition, the subject was required to wear the brace on the dominant extremity for a minimum of 8 h/d, 5 d/wk, for an 8-week period. The brace was worn during an 8-hour time period in which the subject was active on his or her feet, and subjects checked in with the investigators regularly. Because subjects were not readily available on campus during the weekends, it was difficult to ensure compliance with the treatment protocol. Thus, subjects were instructed to wear the braces Monday through Friday, which allowed for better treatment compliance. Although we did not quantify the actual time the subjects wore the braces, regular interaction occurred throughout the treatment period to ensure that the subjects followed the protocol. Subjects were instructed not to wear the braces while sleeping. During the control condition, subjects were instructed to participate in their normal activities of daily living without emphasizing any particular activities. Immediately after the 8-week treatment period, peroneus longus latency was measured under the same pretest conditions described above. This posttest measurement allowed for assessment of the treatment condition (between-subjects factor) after 8 weeks. The average of the 5 trials for each condition obtained during the pretest and posttest was used for statistical analysis.

Statistical Analysis

We used a 3-way, repeated-measures analysis of variance to determine if peroneus longus reaction time differed across levels of brace condition, treatment condition, and time. Simple main-effects testing and the Tukey multiple-comparisons procedure were used to identify group differences. The level of significance was established a priori at $P < .05$.

RESULTS

The means and standard deviations for peroneus longus latency by testing condition, treatment condition, and time are presented in the Table. No significant 3-way ($F_{4,32} = 0.731$, $P = .53$) interaction was observed among the independent variables. Similarly, no significant 2-way interactions were observed between test condition and treatment condition ($F_{2,32} = 0.57$, $P = .69$), time and test condition ($F_{2,32} = 0.142$, $P = .89$), or time and treatment condition ($F_{2,32} = 0.170$, $P = .84$). As for each main effect, no difference was found for test condition ($F_{2,32} = 0.427$, $P = .56$), treatment condition ($F_{2,16} = 1.51$, $P = .184$), or time ($F_{1,16} = 4.24$, $P = 0.06$) on peroneus longus latency (Figure).



Peroneus longus latency across time and treatment conditions. No differences existed between treatment conditions and time ($P > .05$).

DISCUSSION

An important component in establishing and maintaining functional joint stability is the ability to improve and facilitate proprioception.[18] With respect to the ankle-foot complex, Freeman and colleagues[19,20] postulated that chronic ankle injury is due to mechanical instability and decreased afference from joint mechanoreceptors after injury. This theory has also been supported by the work of Lentell et al.[21] The effects of ankle bracing on talocrural and subtalar joint ranges of motion have been studied extensively and recently statistically summarized using meta-analysis procedures.[22] Various forms of ankle supports (tape and braces) are effective in providing mechanical stability while restricting joint range of motion.[23–28] While external ankle supports are effective in providing mechanical stability, their effect on joint proprioception is less understood. Improvement in proprioception has been shown to occur not only through the use of exercise and rehabilitation[29–31] but also through stimulation of cutaneous mechanoreceptors near and around the ankle through the application of various types of ankle support.[32,33]

In our study, we attempted to investigate the effects of long-term ankle brace use on the duration of the peroneus longus stretch reflex. Our main objective was to assess the influence of long-term ankle brace application on peroneus longus latency. Peroneus longus neuromuscular function is critical in dynamically protecting the ankle-foot complex from inversion injuries. As a result, peroneus longus reaction time, or latency, during a simulated ankle sprain has generally been studied in unbraced normal and chronically unstable ankles[6,8–12]; therefore, the effects of ankle supports on peroneus longus function have not been elucidated.[34,35] In all of these studies, the duration of the peroneus longus stretch reflex was being quantified. The stretch reflex involves activation of the group Ia afferent fibers of the muscle spindle, which results in an efferent motor response and contraction of the same muscle.[36] We observed no changes in latency in subjects who were assigned to the lace-up and semirigid brace conditions when compared with controls. We hypothesized that with extended ankle brace use, peroneus longus latency would increase during a sudden inversion movement. Our underlying assumption was that neuromuscular remodeling of the peroneus longus would occur as a result of the dependence on the external support. Such neuromuscular changes were thought to manifest in delayed activation of the peroneus longus with inversion stress. Because our subjects were braced 8 h/d for 5 d/wk over an 8-week period, we speculate that changes in peroneus longus latency probably do not exist, especially during the shorter durations of use common in athletes. Perhaps other changes in neuromuscular function (eg, amplitude of stretch reflex) exist; however, more research is needed in this area.

The lack of difference in peroneus longus latency between groups assigned to bracing may be attributed to the amount of restriction offered by the braces. Without the dynamic stabilization provided by the muscles, the ankle support may be insufficient to protect against external forces applied to the ankle-foot complex. In other words, normal peroneus longus activation may exist despite the mechanical support offered by an external appliance. The main implication of this result is that athletes with healthy ankles who wish to wear external ankle supports prophylactically throughout the season do not appear at risk for compromising the peroneus longus response to sudden inversion. The subjects tested in this study did not represent an athletic population, although they were physically active. This can be viewed as a limitation of our study. Nevertheless, we are confident that these results can be generalized to healthy collegiate athletes (men and women).

Mean (±SD) Peroneus Longus Latency* by Test Condition and Treatment Condition

	Test Condition		
Treatment Condition	Control	Active Ankle	McDavid
Control			
Pretest	46.4 ± 1.9	48.1 ± 2.4	45.9 ± 1.7
Posttest	42.2 ± 2.0	45.3 ± 2.9	41.0 ± 4.3
Active Ankle			
Pretest	56.0 ± 4.3	47.5 ± 3.9	54.6 ± 3.3
Posttest	48.2 ± 4.8	49.3 ± 5.7	49.0 ± 4.3
McDavid			
Pretest	48.3 ± 4.4	47.8 ± 2.9	45.7 ± 1.3
Posttest	48.1 ± 3.0	43.3 ± 3.5	43.9 ± 3.6

*Milliseconds.

Although this outcome is promising to clinicians, more studies are needed to validate this result. It is difficult to surmise if the findings would be the same in a population with chronic ankle instability. Because no studies have evaluated the long-term effects of ankle bracing on peroneus longus latency in the healthy ankle, it would be pure speculation to discuss our results with respect to a pathologic condition. Furthermore, it is difficult to place the present findings in perspective with other literature.

Another objective of this study was to evaluate latency after the application of an external ankle support. The range of latency values observed across all conditions in our study (41.0 to 56.0 milliseconds) was consistent with those for a spinal reflex.[37] We found no difference among the 3 brace conditions on peroneus longus reaction time. This suggests that application of an external ankle support (lace-up or semirigid brace) does not affect the duration of the reflex circuitry of the muscle spindles within the peroneus longus during sudden inversion. Our result is in agreement with Nishikawa and Grabiner,[37] who found no change in peroneus longus H-reflex latency after application of a semirigid ankle brace. Although they electrically stimulated the peroneus longus group Ia afferent nerve fibers percutaneously and not through deformation of joint mechanoreceptors (simulated ankle sprain), similar conclusions can be drawn because the H-reflex latency represents an artificially evoked response of the muscle after a given stimulus using the same reflex circuitry. However, when evaluating the effects of external support on peroneus longus reaction time during sudden inversion, Karlsson and Andreasson[35] found increased peroneus longus reaction time. The increase in reaction time with adhesive tape was observed in patients who suffered from chronic ankle instability.

Whether neuromotor changes with ankle bracing are influenced by ankle injury has been questioned.[37] In other words, does long-term application of an external ankle support facilitate the stretch reflex (ie, shorten the duration) in patients who suffer from chronic ankle instability? Similarly, does long-term ankle support enhance the amplitude of the stretch reflex? Our results cannot address whether long-term ankle brace use affects the neuromuscular response of the peroneus longus in the chronically unstable ankle. However, studies implementing methods similar to ours, using subjects with chronically unstable ankles, would provide greater insight into these questions. The fact that we saw no changes in peroneus longus latency can be viewed in a positive manner. Although external ankle supports provided no heightened response, neither did they induce an inhibitory effect. More studies are needed to characterize these possible relationships.

CONCLUSIONS

This study was undertaken to evaluate a commonly asked question: does long-term ankle bracing affect the neuromotor response of the peroneus longus? Although limited to healthy subjects, our study demonstrated that peroneus longus latency in response to sudden inversion after the extended use of ankle bracing remained unaffected. Furthermore, we also observed that peroneus longus reaction time did not differ between ankle braces, independent of the 8-week treatment. These findings suggest that the extended use of external ankle supports did not induce neuromuscular changes within the primary musculature that dynamically stabilizes against lateral ankle sprain. These results are encouraging for clinicians who advocate the use of prophylactic ankle support for extended periods of time, perhaps over the course of a sport season. Although these results are favorable, more studies are needed to understand the neurophysiologic characteristics (ie, latency and amplitude) of the peroneus longus stretch reflex in normal subjects and in subjects who suffer from chronic ankle instability.

REFERENCES

1. Garrick JG, Requa RK. The epidemiology of foot and ankle injuries in sports. *Clin Sports Med.* 1988;7:29–36.
2. Garrick JG. The frequency of injury, mechanism of injury, and epidemiology of ankle sprains. *Am J Sports Med.* 1977;5:241–242.
3. Glick JM, Gordon RB, Nishimoto D. The prevention and treatment of ankle injuries. *Am J Sports Med.* 1976;4:136–141.
4. Miller EA, Hergenroeder AC. Prophylactic ankle bracing. *Pediatr Clin North Am.* 1990;37:1175–1185.
5. Sitler MR, Horodyski M. Effectiveness of prophylactic ankle stabilisers for prevention of ankle injuries. *Sports Med.* 1995;20:53–57.
6. Konradsen L, Voigt M, Hojsgaard C. Ankle inversion injuries: the role of the dynamic defense mechanism. *Am J Sports Med.* 1997;25:54–58.
7. Beckman SM, Buchanan TS. Ankle inversion injury and hypermobility: effect on hip and ankle muscle electromyography onset latency. *Arch Phys Med Rehabil.* 1995;76:1138–1143.
8. Ebig M, Lephart SM, Burdett RG, Miller MC, Pincivero DM. The effect of sudden inversion stress on EMG activity of the peroneal and tibialis anterior muscles in the chronically unstable ankle. *J Orthop Sports Phys Ther.* 1997;26:73–77.
9. Isakov E, Mizrahi J, Solzi P. Response of the peroneal muscles to sudden inversion of the ankle during standing. *Int J Sport Biomech.* 1986;2:100–109.
10. Johnson MB, Johnson CL. Electromyographic response of peroneal muscles in surgical and nonsurgical injured ankles during sudden inversion. *J Orthop Sports Phys Ther.* 1993;18:497–501.
11. Konradsen L, Ravn JB. Prolonged reaction time in ankle instability. *Int J Sports Med.* 1991;12:290–292.
12. Lofvenberg R, Karrholm J, Sundelin G, Ahlgren O. Prolonged reaction time in patients with chronic lateral instability of the ankle. *Am J Sports Med.* 1995;23:414–417.
13. Cordova ML, Armstrong CW, Rankin JM, Yeasting RA. Ground reaction forces and EMG activity with ankle bracing during inversion stress. *Med Sci Sports Exerc.* 1998;30:1363–1370.
14. Nawoczenski DA, Owen MG, Ecker ML, Altman B, Epler M. Objective evaluation of peroneal response to sudden inversion stress. *J Orthop Sports Phys Ther.* 1985;7:107–109.
15. Kimura IF, Nawoczenski DA, Epler M, Owen MG. Effect of the air stirrup in controlling ankle inversion stress. *J Orthop Sports Phys Ther.* 1987;9:190–193.
16. Delagi EF, Perroto A. *Anatomical Guide for the Electromyographer: The Limbs.* 2nd ed. Springfield, IL: Charles C. Thomas; 1981.
17. Matthews PB. The human stretch reflex and the motor cortex. *Trends Neurosci.* 1991;14:87–91.
18. Lephart SM, Pincivero DM, Rozzi SL. Proprioception of the ankle and knee. *Sports Med.* 1998;25:149–155.
19. Freeman MA. Treatment of ruptures of the lateral ligament of the ankle. *J Bone Joint Surg Br.* 1965;47:661–668.
20. Freeman MA, Dean MR, Hanham IW. The etiology and prevention of functional instability of the foot. *J Bone Joint Surg Br.* 1965;47:678–685.
21. Lentell G, Baas B, Lopez D, McGuire L, Sarrels M, Snyder P. The contributions of proprioceptive deficits, muscle function, and anatomic laxity to functional instability of the ankle. *J Orthop Sports Phys Ther.* 1995;21:206–215.
22. Cordova ML, Ingersoll CD, LeBlanc MJ. Influence of ankle support on joint range of motion before and after exercise: a meta-analysis. *J Orthop Sports Phys Ther.* 2000;30:170–182.

23. Greene TA, Wight CR. A comparative support evaluation of three ankle orthoses before, during, and after exercise. *J Orthop Sports Phys Ther.* 1990;11:453–466.
24. Greene TA, Hillman SK. Comparison of support provided by a semirigid orthosis and adhesive ankle taping before, during, and after exercise. *Am J Sports Med.* 1990;18:498–506.
25. Gross MT, Bradshaw MK, Ventry LC, Weller KH. Comparion of support provided by ankle taping and semirigid orthosis. *J Orthop Sports Phys Ther.* 1987;9:33–39.
26. Gross MT, Lapp AK, Davis JM. Comparison of Swede-O universal ankle support and aircast sport-stirrup orthoses and ankle tape in restricting eversion-inversion before and after exercise. *J Orthop Sports Phys Ther.* 1991;13:11–19.
27. Gross MT, Ballard CL, Mears HG, Watkins EJ. Comparison of DonJoy ankle ligament protector and Aircast sport-stirrup orthoses in restricting foot and ankle motion before and after exercise. *J Orthop Sports Phys Ther.* 1992;16:60–67.
28. Gross MT, Batten AM, Lamm AL, et al. Comparison of DonJoy ankle ligament protector and subtalar sling ankle taping in restricting foot and ankle motion before and after exercise. *J Orthop Sports Phys Ther.* 1994;19:33–41.
29. Goldie PA, Evans OM, Bach TM. Postural control following inversion injuries of the ankle. *Arch Phys Med Rehabil.* 1994;75:969–975.
30. Sheth P, Yu B, Laskowski ER, An KN. Ankle disk training influences reaction times of selected muscles in a simulated ankle sprain. *Am J Sports Med.* 1997;25:538–543.
31. Docherty CL, Moore JH, Arnold BL. Effects of strength training on strength development and joint position sense in functionally unstable ankles. *J Athl Train.* 1998;33:310–314.
32. Feuerbach JW, Grabiner MD, Koh TJ, Weiker GG. Effect of an ankle orthosis and ankle ligament anesthesia on ankle joint proprioception. *Am J Sports Med.* 1994;22:223–229.
33. Simoneau GG, Degner RM, Kramper CA, Kittleson KH. Changes in ankle joint proprioception resulting from strips of athletic tape applied over the skin. *J Athl Train.* 1997;32:141–147.
34. Sprigings EJ, Pelton JD, Brandell BR. An EMG analysis of the effectiveness of external ankle support during sudden ankle inversion. *Can J Appl Sport Sci.* 1981;6:72–75.
35. Karlsson J, Andreasson GO. The effect of external ankle support in chronic lateral ankle joint instability: an electromyographic study. *Am J Sports Med.* 1992;20:257–261.
36. Leonard CT. *The Neuroscience of Human Movement.* St. Louis, MO: Mosby; 1998.
37. Nishikawa T, Grabiner MD. Peroneal motoneuron excitability increases immediately following application of a semirigid ankle brace. *J Orthop Sports Phys Ther.* 1999;29:168–173.

Journal of Athletic Training 2000;35(4):412–416

www.journalofathletictraining.org

Exercise After Cryotherapy Greatly Enhances Intramuscular Rewarming

J. William Myrer, PhD; Gary J. Measom, APRN, PhD; Gilbert W. Fellingham, PhD

Brigham Young University, Provo, UT

Objective: To determine the effect of moderate-intensity walking on rewarming of the triceps surae muscle group after a 20-minute application of a crushed-ice pack.

Design and Setting: Subjects were randomly assigned to either the ice-rest or the ice-exercise group. All subjects were treated on the left calf for 20 minutes with a 1.8-kg ice pack. The ice-exercise group walked on a treadmill at 5.63 km/h for 10 minutes and then assumed a prone position on an examining table for 20 minutes. The ice-rest group assumed a prone position on an examining table for 30 minutes after the cryotherapy treatment.

Subjects: Twenty-eight (19 men and 9 women) college-student volunteers.

Measurements: Intramuscular temperature was recorded at 10-second intervals for 50 minutes at 1 cm below the subcutaneous fat with a thermocouple implanted via a 21-gauge sterile hypodermic needle. Differences were analyzed within and between groups at pretreatment (T_0), the end of the ice treatment (T_{20}), 11 minutes after the end of ice treatment (T_{31}), and 30 minutes posttreatment (T_{50}).

Results: We found no differences at T_0 and T_{20} but significant differences at T_{31} and T_{50}. At T_{31}, the ice-exercise group was only 0.61°C colder than at pretreatment levels, while the ice-rest group was 8.05°C colder. By T_{50}, the temperatures were 0.93°C and 6.95°C colder, respectively, than at pretreatment levels.

Conclusion: Moderate walking significantly enhanced rewarming of the triceps surae.

Key Words: cryokinetics, rehabilitation, treatment

Cryotherapy has become the standard therapy in the immediate management and rehabilitation of acute sport injuries.[1–8] Most important in the immediate management of musculoskeletal trauma is cryotherapy's effect on metabolism. Cold decreases cellular metabolism,[2,3,5,7,9,10] thereby reducing secondary hypoxic injury.[2] This results in quicker healing, rehabilitation, and return to play.[2,11] The most effective use of cold during rehabilitation is via cryokinetics; cryotherapy is used to decrease pain and muscle spasm, thus allowing therapeutic exercise to begin earlier than would otherwise be possible.[1–5,12–15] The analgesic effect of cold is used to allow the recovering athlete to perform graded, active exercise.

Little is known of intramuscular rewarming after the application of ice-pack cryotherapy. What we do know is the result of research on nonexercising prone[12,16] or supine[7] subjects. Intramuscular temperature continues to decline for several minutes after the ice pack is removed and may not return to pretreatment temperatures for several hours after treatment has ceased.[7,12,16] Johnson et al[17] assumed "muscle temperature would increase at a greater rate if muscle contraction occurred during the posttreatment period, such as might occur during treatment using exercise." In 1979, Johnson et al[17] went on to say, "the effect of such treatment [exercise] on the increase of intramuscular temperature needs to be determined to identify the time period during which cold treatment would be most effective." The widespread use of cryokinetics in sports medicine and the practice of returning athletes to play after cryotherapy has continued over the last 2 decades, but the query by Johnson et al has remained unanswered. Because of the importance of the question, we designed this experiment to determine the effect of moderate exercise (walking) on intramuscular rewarming after a standard 20-minute crushed-ice-pack treatment.

Address correspondence to J. William Myrer, PhD, 120-A RB, Brigham Young University, Provo, UT 84602. E-mail address: Bill_Myrer@byu.edu

METHODS

Subjects

Twenty-eight (19 men and 9 women) college students (age = 23.9 ± 1.9 years; height = 176.6 ± 10.0 cm; weight = 74.2 ± 12.7 kg; calf skinfold = 16.6 ± 7.4 mm) volunteered and signed a university institutional review board informed consent to participate in this study (which was also approved by the board). We verbally screened subjects for a history of peripheral vascular disease or allergy to cephalexin hydrochloride. Subjects were randomly assigned to 1 of 2 experimental groups, either the ice-rest group (9 men and 4 women) (age = 24.2 ± 1.8 years; height = 179.1 ± 9.0 cm; weight = 77.8 ± 14.0 kg; calf skinfold = 17.7 ± 8.1 mm) or the ice-exercise group (10 men and 5 women) (age = 23.5 ± 2.0 years; height = 174.6 ± 10.7 cm; weight = 71.1 ± 11.0 kg; calf skinfold = 15.8 ± 6.8 mm). To minimize the risk of infection, each subject took a 500-mg dose of cephalexin hydrochloride immediately before the experiment and 3 similar doses at 6-hour intervals after the conclusion of the experiment.

Procedures

We measured the skinfold of the posterior left leg over each subject's gastrocnemius using a Lange skinfold caliper (Cambridge Scientific Industries, Ltd, Cambridge, MD) with the subject standing and bearing weight on the right leg. The measurement was taken at the visually estimated level of greatest girth and was divided by 2 to determine the depth of subcutaneous fat. Subjects assumed a prone position on a standard examining table. We cleansed a 4-cm × 4-cm area of skin over the midportion of the muscle belly of the left calf, first with a 10% povidone-iodine swab and then with a 70% isopropyl alcohol preparation. Before the study and after each use, the tissue-implantable thermocouples (diameter = 0.41 mm) (Columbus Instruments, Model TX-23–21, Columbus, OH), were washed with soap and water and then disinfected in Cidex-Plus (Johnson & Johnson, New Brunswick, NJ) for at least 20 minutes. We then washed the Cidex-Plus from the thermocouple with sterile water. An advanced practice registered nurse (G.J.M.) inserted the thermocouple into the left calf using sterile technique. The thermocouple was inserted perpendicular to the skin surface to a depth of 1 cm below the subcutaneous fat via a 21-gauge sterile hypodermic needle (Figure 1).[7] Insertion depth was controlled by marking a spot on the lead of the implantable thermocouples 1 cm above the sum of the subjects' subcutaneous fat plus 1 cm (the depth into the muscle). The thermocouple was inserted a few millimeters deeper than needed, the hypodermic needle was then removed, and the thermocouple was withdrawn until the mark on the lead was 1 cm above the skin surface. Transpore (3M Health Care, St. Paul, MN) clear tape (2.54 cm × 3.75 cm) was used over the thermocouple insertion site to secure it. The thermocouple was then connected to an electronic thermometer (Iso-Thermex 16-channel, Columbus Instruments) and, after 3 minutes, the baseline pretreatment intramuscular temperature was recorded.

Each member of both experimental groups had a 1.8-kg crushed-ice pack (approximately 25 cm × 30 cm × 5 cm) placed directly over the thermocouple's insertion into the triceps surae muscle group for 20 minutes. At the end of the treatment, the ice-rest group continued to lie prone on the examining table for 30 minutes. After the cryotherapy treatment, the ice-exercise group walked on a treadmill for 10 minutes at a pace of 5.63 km/h and then assumed a prone position on the examining table for 20 minutes. The thermocouple remained in the subject's calf while he or she walked pain free on the treadmill (Figure 2). For both groups, we recorded intramuscular temperature to the nearest 0.01°C every 10 seconds over the entire 20-minute ice treatment and for 30 minutes posttreatment. Then we removed the thermocouple, dried the limb, and swabbed the area with 70% isopropyl alcohol. Throughout the experiment, room temperature was monitored using a thermocouple (Model TX-31, Columbus Instruments) interfaced with the Iso-Thermex.

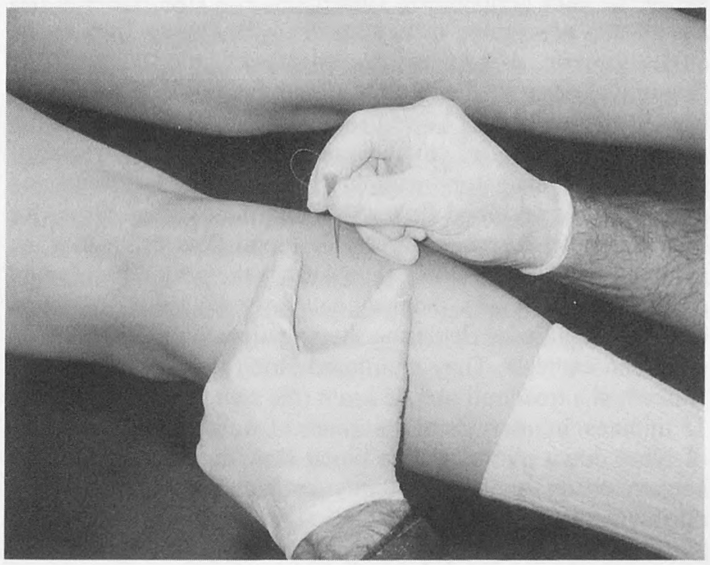


Figure 1. Insertion of the tissue-implantable thermocouple 1 cm deep into the muscle of the medial calf below the subject's subcutaneous fat.

Data Analysis

Our independent variables were experimental group and time. Our dependent variable was temperature. Although temperature measurements were available every 10 seconds, the measurements of primary interest were taken at pretreatment levels (T_0), end of ice treatment (T_{20}), 11 minutes after the end of ice treatment (T_{31}), and 30 minutes posttreatment (T_{50}). Because of the variability in temperature recordings during exercise and the time required for the subjects to move from the treadmill back to the examining table, we chose to analyze the data at 11 minutes posttreatment (T_{31}). As a preliminary test to identify the existence of overall temperature differences between groups, we performed a multiple analysis of variance on the 4 time points of primary interest gathered for each subject. We calculated temperature change from pretreatment to the end of the ice treatment (T_{20}–T_0), from pretreatment to 11 minutes after the end of ice treatment (T_{31}–T_0), and from pretreatment to 30 minutes posttreatment (T_{50}–T_0). We also calculated temperature change from the end of ice treatment to 11 minutes after the end of ice treatment (T_{31}–T_{20}) and from the end of ice treatment to 30 minutes posttreatment (T_{50}–T_{20}). Differences between groups were analyzed with *t*

Figure 2. Subject walking on the treadmill for 10 minutes at a pace of 5.63 km/h with the thermocouple in the leg.

Table 1. Intramuscular Temperature Change (°C ± SD) for Analyzed Time Points Between the Ice-Rest and Ice-Exercise Groups from Pretreatment (T_0) to End of Posttreatment (T_{50})

Time Points	Ice-Rest Temperature Change (°C ± SD)	Ice-Exercise Temperature Change (°C ± SD)	*t* value	*P value
T_0	35.86 ± 0.74	35.53 ± 0.65	−1.27	> .05
T_{20}–T_0	−6.19 ± 2.88	−7.36 ± 4.45	−0.81	> .05
T_{31}–T_0	−8.05 ± 2.47	−0.61 ± 1.46	9.85	< .05
T_{50}–T_0	−6.95 ± 1.53	−0.93 ± 1.07	12.22	< .05
T_{31}–T_{20}	−1.86 ± 1.00	6.75 ± 4.87	6.69	< .05
T_{50}–T_{20}	−0.76 ± 1.90	6.44 ± 4.83	5.32	< .05

*Bonferroni corrected for multiple tests.

tests for T_0, T_{20}–T_0, T_{31}–T_0, T_{50}–T_0, T_{31}–T_{20}, and T_{50}–T_{20}. Differences within groups were analyzed using paired *t* tests for T_{20}–T_0, T_{31}–T_0, and T_{50}–T_0. An α level of $P < .05$ was used for all statistical analyses. To account for the multiple *t* tests, a Bonferroni correction factor was used.

Two subjects were dropped from our statistical analysis because of malfunctions of the thermocouples. We analyzed 15 subjects from the ice-exercise group and 13 from the ice-rest group.

RESULTS

The multiple analysis of variance revealed significant differences in temperature between experimental groups ($F_{4, 23} = 36.44$, $P < .0001$). Figure 3 shows mean intramuscular temperatures throughout the treatment and posttreatment for both experimental groups. Table 1 presents the intramuscular temperature change between experimental groups across the analyzed time points. Only at pretreatment (T_0) and immediately after treatment (T_{20}) were the 2 groups not significantly different. By T_{31}, the ice-exercise group had rewarmed to be only 0.61°C colder than the pretreatment temperature, while the ice-rest group was 8.05°C colder than the pretreatment temperature ($P < .05$). By T_{50}, the temperatures were 0.93°C and 6.95°C colder, respectively, than at T_0 ($P < .05$). Table 2 presents the intramuscular temperature change within experimental groups across the analyzed time points. After moderate exercise (T_{31}), we found no significant difference in the ice-exercise group compared with its pretreatment temperature.

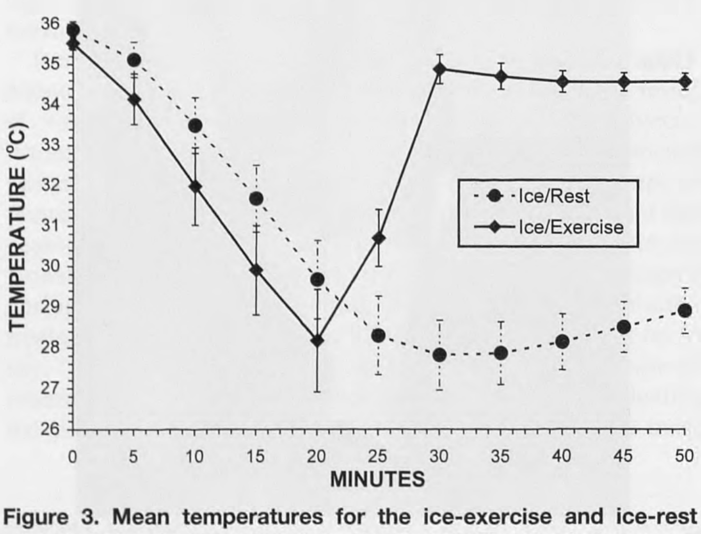


Figure 3. Mean temperatures for the ice-exercise and ice-rest groups during treatment and posttreatment. The rise in intramuscular temperature during exercise was significant.

The temperature of the laboratory during the time we collected data was 24.93° ± 0.69°C.

DISCUSSION

The scientific literature is in agreement that cryotherapy has a depressive effect on many physiologic functions[2,4–6,16,18,19] and that intramuscular temperature continues to decline even after the treatment ends.[1,4,7,12,16,17,20] What has remained unclear is the effect of exercise on intramuscular rewarming after cryotherapy treatment. Our study was the first designed specifically to examine the effect of exercise on intramuscular rewarming after a standard crushed-ice-pack treatment.

Rewarming has been defined in 2 ways: 1) return of the body part to the temperature before cold application, and 2) return to the temperature of the contralateral body part.[2] We operationally defined rewarming according to the first criteria. Knight[2] contended that the factors determining the rate at which a body part rewarms after a cryotherapy treatment are "heat conduction from the atmosphere, surrounding tissue, deep tissue, and circulating blood."

The amount of activity a body part is engaged in has a direct bearing on the temperature of the body part. If a body part is inactive for an extended period of time, metabolism and heat production decrease and intramuscular temperature decreases gradually.[17,21] However, if the body part is exercised, intramuscular temperature increases. Most rewarming studies have measured skin temperature change.[11,22–24] This research has shown that rewarming occurs faster in the fingers than in the ankle, forearm, and knee joint. Increased blood flow to the fingers has been attributed to bringing an increased amount of heat to the area.[11,22–24] Even mild activity, such as changing clothes or walking on crutches, increases the rate of rewarming.[11] One investigator[25] examined the effect of hot packs and exercise on local blood flow. Although the exercise lasted for only 1 minute, it resulted in a peak blood flow of 9 times the resting flow. This was more than 4 times the peak flow brought about by the hot packs. Others[21] have investigated the effect on blood flow in the ankle during therapeutic applications of heat, cold, and exercise. They monitored blood flow while subjects walked on a treadmill at 5.63 km/h (the same rate we used) for 15 minutes, in intervals of 3 minutes of walking and 3 minutes of lying down. Although the blood flow during exercise was conservatively estimated, they concluded that blood flow during moderate exercise was significantly greater than both control and heat-pack conditions.[21] Further research has established that for muscles to receive increased blood flow during exercise, they must be "active."[26] A very interesting series of experiments[26] was designed to examine blood flow

Table 2. Intramuscular Temperature Change (°C ± SD) for Analyzed Time Points Within the Ice-Rest and Ice-Exercise Groups from Pretreatment (T_0) to End of Posttreatment (T_{50})

	Temperature									
Group	T_0	T_{20}–T_0*	*t*	*P*†	T_{31}–T_0*	*t*	*P*†	T_{50}–T_0*	*t*	*P*†
Ice-exercise	35.53 ± 0.65	−7.36 ± 4.45	−6.41	< .05	−0.61 ± 1.46	−1.62	> .05	−0.93 ± 1.07	−3.35	> .05
Ice-rest	35.86 ± 0.74	−6.19 ± 2.88	−7.75	< .05	−8.05 ± 2.47	−11.74	< .05	−6.95 ± 1.53	−16.42	< .05

*Pretreatment temperature.
†Bonferroni corrected for multiple tests.

and temperature change in the forearm during sustained hand-grip contractions. The first experiment demonstrated increased blood flow during contractions at all tensions from 30% to 70% of maximum voluntary contraction. A follow-up experiment used indwelling electromyography and thermocouples to determine which muscles were the recipients of the extra blood flow. The researchers first cooled the forearms in water baths of 18° or 26°C and then monitored the muscles of the forearm during and after contractions. They then observed that, "Large rises in temperature were observed in the active muscles, while little or no change was observed in inactive muscle." The results of a recent study[27] looking at temperature changes in deep muscles during upper and lower extremity exercise concurred with this conclusion. These authors suggested that, "one must actively exercise the muscle in order to cause a significant temperature increase in that muscle."[27]

Our results involving intramuscular temperature change after ice-pack therapy agree with previous ice-pack research, which indicated that after the removal of the ice pack, intramuscular temperature continues to decline.[7,12,16] Our ice-rest group continued to get colder for approximately 10 minutes after the removal of the ice before the intramuscular temperature slowly began to rewarm. By 11 minutes posttreatment (T_{31}), our ice-rest group, without the intervention of exercise, was an additional 1.86° ± 1.00°C colder than at the time the ice pack was removed. They remained significantly below pretreatment level throughout the entire 30-minute posttreatment.

The intramuscular temperature changes of our ice-exercise group indicated a powerful effect of moderate exercise on the rewarming of active muscle following cryotherapy (Figure 3 and Table 1). This concurs with previous skin temperature research.[11,22,23] The effect of exercise on rewarming was immediate, and over the 11-minute time period from the end of the ice-pack treatment to the time we analyzed temperature change (T_{31}) (including 10 minutes during which the subjects were exercising on the treadmill), the ice-exercise group rewarmed 6.75° ± 4.87°C, to be only 0.61°C below their pretreatment level. We did note a slight decrease (0.32°C) in the intramuscular temperature during the time frame T_{31}–T_{50} in our ice-exercise group. This was probably due to the inactivity of lying motionless on the examining table.[17,21]

CONCLUSION

Moderate walking significantly enhanced the rewarming of the triceps surae after a standard ice-pack treatment. The clinical significance of our study is that exercise of the treated musculature significantly decreases the duration of the intramuscular temperature reduction brought about by cryotherapy. This would presumably also reduce the duration of the physiologic responses produced by the cold application. Practically, this would imply that if the prolonged physiologic responses caused by the application of cold are desirable in the treated structure, then that body part should not be exercised immediately after the removal of the cold treatment, ie, the immediate management of a contusion. However, if one wishes to use cryokinetics during rehabilitation, then our results add support to the contention that exercise is the critical component to cryokinetics.[2,13–15,21,22] The cold reduces the athlete's pain, so early mobilization through exercise can be achieved much sooner than would normally be possible.[2,11] Exercise increases intramuscular temperature, primarily due to increased blood flow, which enhances the removal of cellular debris from the injury site and increases the delivery of nutrients necessary for the healing process.[2,11]

REFERENCES

1. Halvorson GA. Therapeutic heat and cold for athletic injuries. *Phys Sportsmed.* 1990;18(5):87–94.
2. Knight KL. *Cryotherapy in Sport Injury Management.* Champaign, IL: Human Kinetics; 1995:71–230.
3. Kaul MP, Herring SA. Superficial heat and cold: how to maximize the benefits. *Phys Sportsmed.* 1994;22(12):65–74.
4. Meeusen R, Lievens P. The use of cryotherapy in sports injuries. *Sports Med.* 1986;3:398–414.
5. Swenson C, Sward L, Karlsson J. Cryotherapy in sports medicine. *Scand J Med Sci Sports.* 1996;6:193–200.
6. Lehmann JF. *Therapeutic Heat and Cold.* 4th ed. Baltimore, MD: Williams & Wilkins; 1990:590–632.
7. Merrick MA, Knight KL, Ingersoll CD, Potteiger, JA. The effects of ice and compression wraps on intramuscular temperatures at various depths. *J Athl Train.* 1993;28:236–245.
8. Kowal MA. Review of physiological effects of cryotherapy. *J Orthop Sports Phys Ther.* 1983;5:66–73.
9. Ho SSW, Coel MN, Kagawa R, Richardson AB. The effects of ice on blood flow and bone metabolism in knees. *Am J Sports Med.* 1994;22:537–540.
10. Ho SSW, Illgen RL, Meyer RW, Torok PJ, Cooper MD, Reider B. Comparison of various icing times in decreasing bone metabolism and blood flow in the knee. *Am J Sports Med.* 1995;23:74–76.
11. Palmer JE, Knight KL. Ankle and thigh skin surface temperature changes with repeated ice pack application. *J Athl Train.* 1996;31:319–323.
12. Myrer JW, Measom G, Fellingham GW. Temperature changes in the human leg during and after two methods of cryotherapy. *J Athl Train.* 1998;33:25–29.
13. Barnes L. Cryotherapy: putting injury on ice. *Phys Sportsmed.* 1979;7(6):130–136.
14. Grant AE. Massage with ice (cryokinetics) in treatment of painful conditions of the musculoskeletal system. *Arch Phys Med Rehabil.* 1964;45:233–238.
15. Hayden CA. Cryokinetics in an early treatment program. *J Am Phys Ther Assoc.* 1964;44:990–993.

16. Hartviksen K. Ice therapy in spasticity. *Acta Neurol Scand.* 1962;38(Suppl 3):79–84.
17. Johnson DJ, Moore S, Moore J, Oliver RA. Effect of cold submersion on intramuscular temperature of the gastrocnemius muscle. *Phys Ther.* 1979;59:1238–1242.
18. Cross KM, Wilson RW, Perrin DH. Functional performance following an ice immersion to the lower extremity. *J Athl Train.* 1996;31:113–116.
19. Evans TA, Ingersoll C, Knight KL, Worrell T. Agility following the application of cold therapy. *J Athl Train.* 1995;30:231–234.
20. Oliver RA, Johnson DJ. The effects of cold water baths on posttreatment leg strength. *Phys Sportsmed.* 1976;4(11):67–69.
21. Knight KL, Londeree BR. Comparison of blood flow in the ankle of uninjured subjects during therapeutic applications of heat, cold, and exercise. *Med Sci Sports Exerc* 1980;12:76–80.
22. Knight KL, Aquino J, Johannes SM, Urban CD. A reexamination of Lewis' cold-induced vasodilation in the finger and the ankle. *Athl Train.* 1980;15:248–250.
23. Knight KL, Elam JF. Rewarming of the ankle, finger, and forearm after cryotherapy: further investigation of Lewis' cold-induced vasodilation. *J Can Athl Ther Assoc.* 1981;8:17–18.
24. Knight KL, Bryan KS, Halvorsen JM. Circulatory changes in the forearm in 1, 5, 10, and 15°C water. *Int J Sports Med.* 1981;4:281.
25. Greenberg RS. The effects of hot packs and exercise on local blood flow. *Phys Ther.* 1972;52:273–278.
26. Humphrey PW, Lind AR. Blood flow through active muscle of the forearm during sustained handgrip contraction. *J Physiol.* 1962;163; 18P.
27. Wirth VJ, Van Lunen BL, Mistry D, Saliba E, McCue FC III. Temperature changes in deep muscles of humans during upper and lower extremity exercise. *J Athl Train.* 1998;33:211–215.

Journal of Athletic Training 2000;35(4):417–421

www.journalofathletictraining.org

Effect of Coupling Medium Temperature on Rate of Intramuscular Temperature Rise Using Continuous Ultrasound

Corey A. Oshikoya, MEd, ATC*; Sandra J. Shultz, PhD, ATC†; Danny Mistry, MD, ATC†; David H. Perrin, PhD, ATC†; Brent L. Arnold, PhD, ATC†; Bruce M. Gansneder, PhD†

*Denver Broncos Football Club, Englewood, CO; †University of Virginia, Charlottesville, VA

Objective: We determined the effects of coupling medium temperature on the rate of intramuscular temperature rise (RTR) during continuous ultrasound.

Design and Setting: Ultrasound was applied in a continuous mode at a frequency of 1 MHz and intensity of 1.5 W/cm^2. Each subject received 3 treatments, using water-based coupling gel at temperatures of 18°C, 25°C, and 39°C. All treatments were performed in an athletic training room under controlled environmental conditions.

Subjects: Eighteen healthy male subjects (mean age = 23.6 ± 3.5 years; height = 177.8 ± 6.9 cm; weight = 76.6 ± 8.2 kg; calf size = 37.6 ± 2.4 cm) participated in this study.

Measurements: A thermistor was inserted into the left medial triceps surae at a depth of 5 cm, and baseline tissue temperatures were recorded before treatment. Intramuscular temperature was recorded every 30 seconds until the temperature rose 4°C above baseline or until discomfort was felt. RTR was calculated by dividing the absolute temperature change by treatment time.

Results: A 1-way, repeated-measures analysis of variance revealed a significant difference in RTR among gel temperatures. RTR was significantly faster using the 25°C gel compared with the 18°C and 39°C gels. There was no difference between the 18°C and 39°C gel treatments.

Conclusions: These results suggest that the use of a cooled or heated gel may be counterproductive when maximal thermal effects are desired within a given time frame.

Key Words: modalities, thermal, water-based gel

Therapeutic ultrasound is a commonly used modality in the treatment of physical injuries. Many factors have been found to play a part in the effective transmission of ultrasound to the target tissues. Treatment-area size, treatment duration,[1] frequency and intensity parameters,[2] soundhead pressure,[3] and angle of application[4] have all been found to influence treatment efficacy.

A major factor influencing the transmission of sound waves during ultrasound is the coupling medium applied.[5,6] Because ultrasound cannot be transmitted through air, a dense coupling medium is needed between the transducer and the skin.[7,8] Previous studies[6,9–12] have investigated the effectiveness of coupling mediums by measuring temperature increases intramuscularly[13] and the transmission of ultrasonic waves. Their results indicate that a water-based gel provides the highest percentage of acoustic energy transmission compared with other mediums tested.[5,13–15]

In addition to the coupling medium used, clinicians have sought to enhance the transmission of ultrasound by combining it with other therapeutic agents.[16–20] These agents have been thermal in nature and are often administered before treatment to produce superficial tissue temperature and density changes in order to enhance effectiveness. Lehmann et al[18] evaluated the effects of an 8-minute hot-pack application before ultrasound and found that the hot pack produced no adverse effects to the ultrasonic treatment. Whether any positive benefits resulted from the addition of the hot pack was not explored. However, Draper et al[16] reported an additive effect to overall temperature increase when a hot pack was applied for 15 minutes before an ultrasound treatment.

The effects of cold application before continuous ultrasound have also been explored.[17,19] Rimington et al[19] found that an ice bag applied for 15 minutes before ultrasound decreased tissue temperature to the point that even baseline levels were not reached during the treatment. Similarly, Draper et al[17] demonstrated that a 5-minute ice-bag application resulted in only a 1°C increase in tissue temperature rise after ultrasound application. Baker and Bell[20] evaluated the effect of cold on blood flow rather than rate of temperature rise and found ultrasound, alone and when preceded by an ice massage, was effective in increasing blood flow. Interestingly, they found no significant increase in blood flow with the application of moist hot packs before ultrasound treatment.

Considering the effect of superficial thermal agents on intramuscular temperature rise, little research to date has evaluated the effects of varying the coupling medium temperature. Lehmann et al[21] sought to determine which coupling medium and temperature resulted in the greatest peak tissue temperature with ultrasound administered at 1 MHz and 1 W/cm^2. Using mineral oil and degassed water as the coupling

Address correspondence to Sandra J. Shultz, PhD, ATC, 203 Memorial Gymnasium, University of Virginia, Charlottesville, VA 22903. E-mail address: sjs3n@virginia.edu

mediums at both 21°C and 24°C, they reported that the 21°C mineral oil produced the highest peak temperatures in the deep tissues near the bone and the 24°C oil produced the highest peak temperatures in the superficial tissues. However, from the graphic data presented in the article, peak temperatures in the deep tissue appeared quite similar for both the 21°C and 24°C oil samples.[21] Therefore, it seems that coupling medium temperature may have had little effect on peak deep tissue temperatures. Although this was not reported by Lehmann et al,[21] it does appear from their data that the time rate to achieve peak temperature differed, with the 24°C oil being faster than the 21°C oil in the deep tissues.

Collectively, these researchers have attempted to evaluate the influence of superficial temperature changes on the thermal effects of ultrasound. While some studies indicate superficial heating enhances, or has no adverse effect on, intramuscular temperature during sonation,[16,18,20] others have shown no change or a decrease in intramuscular temperature rise with the application of heat and cold.[1,19,20] However, these studies differed considerably in their methods (eg, duration of heat or cold application, type of coupling medium used), which makes comparison across studies difficult. Furthermore, with the exception of Lehmann et al,[21] thermal agents were always applied before, rather than during, the ultrasound treatment. We were unable to find any research that specifically addressed whether varying the temperature of a water-based coupling medium would influence the rate of intramuscular temperature rise (RTR) during sonation. Therefore, our purpose was to compare the effect of cold, room temperature, and heated coupling mediums on the RTR in the human gastrocnemius muscle during continuous ultrasound.

METHODS

Subjects

Eighteen college-aged men (age = 23.6 ± 3.5 years; height = 177.8 ± 6.9 cm; weight = 76.6 ± 8.2 kg; calf circumference = 37.6 ± 2.4 cm) volunteered to participate in this study. All subjects were asymptomatic at the onset of the study and free of injury, infection, and swelling in the left leg for the past 6 months. All subjects read and signed an informed consent that explained all potential risks before participating in the study. The study received approval from the University's Human Investigation Review Board.

Instruments

We used the Omnisound 3000 (Accelerated Care, Inc, Topeka, KS) ultrasound unit, equipped with a lead zirconate titanate crystal and 1-MHz frequency sound head. The transducer size was 5 cm^2, with an effective radiating area of 4.1 cm^2 and a beam nonuniformity ratio of 4:1. The unit was calibrated 1 month before the study and was dedicated to the research project through the duration of the study. For the coupling medium, we used Aquasonic 100 (Parker Laboratories, Inc, Newark, NJ) transmission gel at standardized temperatures of 18°, 25°, and 39°C. We heated the 39°C gel using the model TM-1 Gel Warmer (Chattanooga Group, Inc, Hixson, TN).

To record intramuscular temperatures, we used a 23-gauge thermistor needle (Phystek MT-23/5, Physitemp Instruments, Clifton, NJ) attached to a monitor (Bailey Instrument BAT-10, Physitemp Instruments) to provide continuous digital temperature readings in degrees Celsius (°C). According to the manufacturer, temperature accuracy is within ±0.1°C.

Procedure

The subjects remained prone for all ultrasound treatments. All treatments were performed in the University's athletic treatment facility, with the room temperature controlled at 22.77°C. We performed all treatments during the same session, and gel samples were counterbalanced to control for order effect.

We controlled and monitored each gel sample temperature individually using a mercury thermometer before and during the treatment sessions. The 18°C gel sample was placed in the refrigerator before treatment, and the temperature was maintained during the treatment session using an ice bath. The 25°C gel sample was maintained at room temperature, and we maintained the 39°C gel temperature with the commercial gel warmer. Although we did not monitor the temperature of the gel once it was applied to the skin's surface, we maintained the respective gel temperatures throughout the ultrasound treatment by adding a new gel sample approximately every 2 minutes. The primary investigator (C.A.O.) administered all ultrasound treatments perpendicular to the thermistor in a continuous mode at intensity of 1.5 W/cm^2. In order to limit and standardize the treatment area, we cut a template to precisely 2 times the size of the effective radiating area of the ultrasound applicator (8.2 cm^2).[14] We moved the ultrasound head within the template at a rate of approximately 4 $cm \cdot s^{-1}$.

We applied all ultrasound treatments to a 10-cm diameter area on the left medial triceps surae muscle. We positioned the subject prone and measured the circumference of the lower leg to determine the cross-sectional area with the greatest muscle girth. We shaved and cleansed the area thoroughly with a 10% povidine-iodine scrub, followed by a 70% isopropyl alcohol swab. We used a caliper to determine the site of thermistor insertion (5 cm deep), and a physician injected 1 mL of 1% lidocaine subcutaneously to anesthetize the area before the thermistor was inserted. Once the area was anesthetized, the physician inserted the thermistor into the left medial triceps surae muscle belly at a tissue depth of 5.0 cm, using a level to keep the thermistor parallel to the frontal plane. We then connected the thermistor to the monitor, and tissue temperature was allowed to stabilize for 5 minutes. After this procedure, we recorded the baseline temperature for each subject.

Once the baseline temperature was established, we initiated the ultrasound treatment and recorded intramuscular temperatures at time 0 and every 30 seconds thereafter until intramuscular tissue temperature increased 4°C above baseline[22] or the subject began to feel discomfort. At the end of each treatment, the tissue temperature was allowed to return to baseline levels and stabilize for 5 minutes before we initiated the next treatment condition. On completion of the testing, we removed the thermistor, cleansed the area with the povidine-iodine solution, and applied an antibiotic ointment and bandage over the injection site. Before releasing the subject, we placed an ice pack over the area for 10 minutes to help reduce hematoma formation. After each test session, we sterilized the thermistor using ethylene oxide gas in the Central Sterile Supply area at the University's Medical Center.

Statistical Analysis

RTR was calculated by dividing the absolute temperature change by the total treatment time for each subject. We analyzed the data using a 1-way, repeated-measures analysis of variance with 1 within variable (rate of temperature rise) measured at 3 temperature levels (18°, 25°, and 39°C). We used the Tukey HSD method to determine which specific gel temperatures differed significantly. The α level for all analyses was set a priori at $P < .05$.

RESULTS

The mean baseline tissue temperature across all subjects was 35.47°C ± 0.74°C. Of the total 54 ultrasound treatments performed (3 treatments per subject), 44.6% of the treatments (44.6% cold, 55.8% room, and 33.5% hot) achieved the 4°C target increase in temperature, with the remaining treatments being terminated due to subject discomfort. The mean temperature increases obtained in treatments terminated by discomfort were 3.13° ± 0.71°C (cold), 3.33° ± 0.64°C (room), and 3.10° ± 0.75°C (hot).

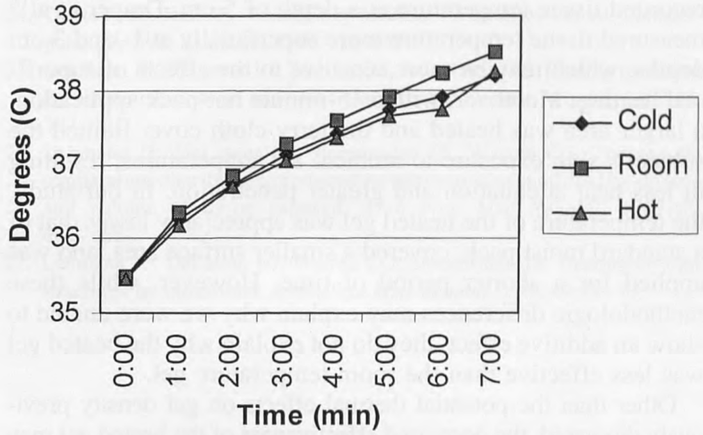
Means and standard deviations for final temperature, total temperature change, time to reach final temperature, and RTR for each gel condition are listed in the Table. The Figure plots the change in RTR across time for each treatment condition. We found a significant difference in RTR among the 3 gel samples ($F_{2,34} = 6.487$, $P = .004$). Observed power was 0.879. The Tukey post hoc analysis revealed that the RTR was significantly faster using the 25°C gel, compared with both the 18°C and 39°C gel treatments. There was no significant difference between the 18°C and 39°C gel treatments.

DISCUSSION

Our primary finding was that the room-temperature coupling medium was more efficient than either the cooled or heated gel in achieving maximal thermal effects at a 5-cm depth intramuscularly. Many clinicians attempt to enhance the efficacy of continuous ultrasound by applying superficial thermal agents, such as moist heat and ice packs, to the skin before sonation.[16–19] The superficial temperature change brought about by these agents is thought to influence ultrasonic wave propagation to deeper tissues by altering blood flow[20,22] and tissue density[8,16,17,19,21] in the superficial tissue layers. While some have tested the theory that the application of cold before ultrasound increases tissue density and, thus, improves wave propagation,[17,19] others used moist heat in an attempt to produce an additive heating effect.[16] Based on these therapeutic rationales, we sought to investigate whether varying the superficial temperature of the water-based coupling medium could similarly enhance the thermal effects of ultrasound.

Mean ± Standard Deviations for Final Intramuscular Tissue Temperature (FT), Total Temperature Change (TTC), Time to Reach Final Temperature (TFT), and Calculated Rate of Temperature Rise (RTR) for Each Gel Temperature

Gel Temperature	FT (°C)	TTC (°C)	TFT (min)	RTR (°C/min)
Cold (18°C)	38.92 ± .78	3.59 ± .57	12.96 ± 2.15	.31 ± .10
Room (25°C)	39.08 ± .94	3.66 ± .51	10.63 ± 1.60	.39 ± .11
Warm (39°C)	38.82 ± .95	3.50 ± .56	11.12 ± 1.93	.33 ± .09



Rate of temperature rise for each gel temperature over time.

Gel Temperature and Density

As previously stated, in order for wave propagation to occur, the ultrasound head must be in contact with a dense coupling medium. Various mediums have been tested, with a water-based gel producing the highest percentage of acoustic energy transmission. It would, therefore, be plausible that if the density of a water-based gel was altered, ultrasound transmission and, thereby, RTR could be affected. While previous authors have tested the theoretical model of increasing tissue density through cold application,[17,19] we are unaware of any studies that have directly evaluated the effects of thermal changes on the water-based gel density and ultrasound transmission. Theoretically, one might expect cooling to increase and heating to decrease the density of the coupling medium, thus affecting ultrasound transmission and RTR. While our findings, in part, indirectly support this theory with a smaller RTR for the heated gel compared with room-temperature gel, we found a smaller RTR rather than a greater RTR with the cooled gel. Lehmann et al[21] represent the only other researchers to evaluate the effects of a cooled coupling medium when applied during an ultrasound treatment. They concluded that a mineral oil coupling medium at temperatures of 21°C or less was more effective for deeper tissue heating than 24°C. However, on careful review of their data, while the 24°C oil resulted in slightly lower peak temperatures compared with the 21°C oil, it appears to have produced a faster RTR in deeper tissues. They did not evaluated heating effectiveness using a heated mineral oil.

Based on these findings, we believe that the density of the gel as a result of temperature change had negligible effects on tissue temperature or at least cannot alone explain our findings. It is likely that other factors, such as the cooling or heating effect of the gels on superficial tissue temperatures, also influence RTR.

Gel Temperature and Additive Thermal Effects

With regard to superficial heating, our results contrast with those of Draper et al,[16] who demonstrated an additive thermal effect when a hot pack was applied to the calf for 15 minutes before ultrasound. In fact, Draper et al[16] found such a profound heating effect that less energy was required by the subsequent ultrasound treatment to produce maximal heating effects. We believe the contrast in these findings can be explained by the difference in heating intensity, method of application, and the depth at which the temperatures were recorded. While we recorded tissue temperature at a depth of 5 cm, Draper et al[16] measured tissue temperature more superficially at 1- and 3-cm depths, which may be more sensitive to the effects of superficial heating. Moreover, with a 15-minute hot-pack application, a larger area was heated and the terry cloth cover limited the subject's skin exposure to ambient air temperatures, resulting in less heat attenuation and greater penetration. In our study, the temperature of the heated gel was appreciably lower that of a standard moist pack, covered a smaller surface area, and was applied for a shorter period of time. However, while these methodologic differences may explain why we were unable to show an additive effect, they do not explain why the heated gel was less effective than the room-temperature gel.

Other than the potential thermal effects on gel density previously discussed, the decreased effectiveness of the heated gel may also be explained by the body's physiologic reactions to thermal agents. When heat is applied to the skin, feedback from thermoreceptors initiates a sympathetic reflex circulatory response to increased blood flow to the area in an effort to regulate and maintain peripheral temperatures.[23,24] Hence, it is likely that the application of the 39°C coupling medium to the skin initiated this vasodilatory response, effectively dissipating heat in the surrounding tissues and potentially explaining the slower RTR in the deeper tissues. Therefore, it appears from these contrasting studies that the magnitude of superficial heating may dictate whether maximal thermal effects are enhanced or diminished. While profound heating may overwhelm the thermoregulatory response and result in an additive thermal effect, moderate heating may actually be counterproductive.

With regard to superficial cooling, we found the 18°C gel was also counterproductive to achieving maximal thermal effects. While absolute temperature increases were similar to those for the 25°C and 39°C gel treatments, the RTR was significantly reduced compared with room temperature. Therefore, it appears that using an 18°C gel does not produce any additive physiologic effect sufficient to overcome the gel's cooling effect, thereby enhancing the transmission of ultrasonic energy to deeper tissue layers. These findings are consistent with Draper et al[17] and Rimington et al,[19] who found the superficial application of an ice bag (5 and 15 minutes, respectively) before ultrasound reduced heating effectiveness in comparison with ultrasound alone. While our cooling may have been less intense than the ice-pack applications used in these studies, even moderate cooling is sufficient to limit the RTR in deeper tissue.

Clinical Implications

The clinical implication of these findings is that there are no apparent additive benefits when using a cooled or heated gel during a standard ultrasound treatment. The room-temperature (25°C) gel produced the fastest RTR, thus providing the most effective and time-efficient treatment to achieve maximal thermal effects. However, these findings were limited to temperature changes in muscle tissue at a 5-cm depth. As previous research has indicated, temperature increase and tolerance may vary considerably depending on the type, depth, and thickness of the target tissue, as well as its distance from the bone.[25–27]

Our findings also reinforce the need for clinicians to carefully consider the total treatment time required to achieve maximal thermal effects. As busy clinicians, treatment time is always a concern when treating injuries. However, in order for maximal thermal benefits to be achieved during continuous ultrasound, treatment duration must be sufficient to allow vigorous heating of the tissues.[22] Based on our RTR data (Table), we determined that the time required to reach vigorous heating (~4°C or maximal temperature tolerated) at a 5-cm depth was 13.0, 10.6, and 11.1 minutes for the 18°C, 25°C, and 39°C gel treatments, respectively. These time durations are considerably longer than the traditional 5-minute ultrasound treatment that is commonly administered to patients. Therefore, clinicians should consider a minimum treatment duration of 10 minutes if maximal thermal effects are warranted. Furthermore, when other thermal agents are used in conjunction with ultrasound, total treatment time may need to be adjusted further. Although the heated gel was found to be less effective in increasing tissue temperature compared with room-temperature gel, it still can be used effectively to provide patient comfort if a longer treatment time is incorporated.

REFERENCES

1. Draper DO, Castel JC, Castel D. Rate of temperature increase in human muscle during 1 MHz and 3 MHz continuous ultrasound. *J Orthop Sports Phys Ther.* 1995;22:142–150.
2. Draper DO. Ten mistakes commonly made with ultrasound use: current research sheds light on myths. *Athl Train Sports Health Care Perspect.* 1996;2:95–107.
3. Klucinec B. Denegar CR, Mahmood R. The transducer pressure variable: its influence on acoustic energy transmission. *J Sport Rehabil.* 1997;6: 47–53.
4. Kimura IF, Gulick DT, Shelly J, Ziskin MC. Effects of two ultrasound devices and angles of application on the temperature of tissue phantom. *J Orthop Sports Phys Ther.* 1998;27:27–31.
5. Reid DC, Cummings GE. Factors in selecting the dosage of ultrasound: with particular reference to the use of various coupling agents. *Physiother Can.* 1973;25:1:5–9.
6. Warren CG, Koblanski JN, Sigelmann RA. Ultrasound coupling media: their relative transmissivity. *Arch Phys Med Rehabil.* 1976;57:218–222.
7. Starkey, C. *Therapeutic Modalities for Athletic Trainers.* Philadelphia, PA: FA Davis; 1993:173–193.
8. Michlovitz SL. *Thermal Agents in Rehabilitation.* 3rd ed. Philadelphia, PA: FA Davis; 1996:168–207.
9. Cameron MH, Monroe LG. Relative transmission of ultrasound by media customarily used for phonophoresis. *Phys Ther.* 1992;72:142–148.
10. Griffin JE. Transmissiveness of ultrasound through tap water, glycerin, and mineral oil. *Phys Ther.* 1980;60:1010–1016.
11. Brueton RN, Campbell B. The use of Geliperm as a sterile coupling agent for therapeutic ultrasound. *Physiotherapy.* 1987;73:653–654.
12. Benson HAE, McElnay JC. Transmission of ultrasound energy through topical pharmaceutical products. *Physiotherapy.* 1988;74:587–689.
13. Ashton DF, Draper DO, Myrer JM. Temperature rise in human muscle during treatment using Flex-all as a coupling agent. *J Athl Train.* 1998;33:136–140.
14. Draper DO, Sunderland S, Kirkendall DT, Ricard M. A comparison of temperature rise in human calf muscles following applications of underwater and topical gel ultrasound. *J Orthop Sports Phys Ther.* 1993;17: 247–251.
15. Ciccone CD, Leggin BG, Callamaro JJ. Effects of ultrasound and trolamine salicylate phonophoresis on delayed-onset muscle soreness. *Phys Ther.* 1991;71:666–675.
16. Draper DO, Harris ST, Schulthies S, Durrant E, Knight KL, Ricard M. Hot-pack and 1-MHz ultrasound treatments have an additive effect on muscle temperature increase. *J Athl Train.* 1998;33:21–24.
17. Draper DO, Schulthies S, Sorvisto P, Hautala AM. Temperature changes in deep muscles of humans during ice and ultrasound therapies: an in vivo study. *J Orthop Sports Phys Ther.* 1995;21:153–157.
18. Lehmann JF, Stonebridge JB, deLateur BJ, Warren CG, Halar E. Temperature in human thighs after hot pack treatment followed by ultrasound. *Arch Phys Med Rehabil.* 1978;59:472–475.

19. Rimington SJ, Draper DO, Durrant E, Fellingham G. Temperature changes during therapeutic ultrasound in the precooled human gastrocnemius muscle. *J Athl Train.* 1994;29:325–327.
20. Baker RJ, Bell GW. The effects of therapeutic ultrasound on blood flow in the human calf. *J Orthop Sports Phys Ther.* 1991;13:23–27.
21. Lehmann JF, DeLateur BJ, Silverman DR. Selective heating effects of ultrasound in human beings. *Arch Phys Med Rehabil.* 1966;46:331–339.
22. Lehmann JF. *Therapeutic Heat and Cold.* 4th ed. Baltimore, MD: Williams & Wilkins; 1990:437–443.
23. Sherwood L. *Fundamentals of Physiology: A Human Perspective.* 2nd ed. St. Paul/Minneapolis, MN: West Publishing; 1995:468–476.
24. Guyton AC, Hall JE. *Textbook of Medical Physiology.* 9th ed. Philadelphia, PA: WB Saunders; 1996:918–919.
25. Lehmann JF, DeLateur BJ, Warren CG, Stonebridge JS. Heating produced by ultrasound in bone and soft tissue. *Arch Phys Med Rehabil.* 1967;48: 397–401.
26. Lehmann JF, DeLateur BJ, Stonebridge JB, Warren CG. Therapeutic temperature distribution produced by ultrasound as modified by dosage and volume of tissue exposed. *Arch Phys Med Rehabil.* 1967;48: 662–666.
27. Lehmann JF, DeLateur BJ, Warren CG, Stonebridge JB. Heating of joint structures by ultrasound. *Arch Phys Med Rehabil.* 1968;49:28–30.

Journal of Athletic Training 2000;35(4):422–426

www.journalofathletictraining.org

Interval Cryotherapy Decreases Fatigue During Repeated Weight Lifting

Frank M. Verducci, EdD

San Francisco State University, San Francisco, CA

Objective: To investigate the effect of icing the arm and shoulder between weight-pulling sets on work, velocity, and power.

Design and Setting: I used a 1 × 2 factorial, random, counterbalanced design in which each participant pulled 75% of his 1-repetition maximum on 2 separate days. The individuals pulled the weight 22 times for each set as fast as possible, then either iced (cryotherapy) or placed towels over their arms and shoulders for 3 minutes, and then rested 4.5 minutes at room temperature. The sets continued until the participants could not complete 22 pulls without stopping.

Subjects: Ten male members of a private athletic club, who weight lift on a regular basis, volunteered to participate in the study.

Measurements: Work was determined by the number of arm-pull sets completed before fatigue, velocity was measured by the time to complete each set, and power was determined by dividing work by velocity. Velocity and power were analyzed in 3 ways: first to fourth sets (88 pulls), matched sets (167.2 pulls), and all sets (191.4 cryotherapy and 167.2 towel pulls), using analysis of covariance with the base set as the covariate.

Results: Cryotherapy between sets resulted in a significantly greater number of total joules and arm pulls when compared with the towel treatment. Velocity was significantly faster for the first to fourth sets, matched sets, and all sets when subjects received intermittent cryotherapy. Power also was significantly higher for the first to fourth sets and matched sets. The all-sets comparison consisted of 14.5% more cryotherapy arm pulls.

Conclusions: Interval cryotherapy between weight-pulling sets is associated with increased work, velocity, and power.

Key Words: cold, ice, strength, work, velocity, power

Athletes consistently work to delay the onset of fatigue and improve motor performance. Skill development, weight training, cardiorespiratory exercises, and stretching are just a few of the many activities used to enhance achievement. Of these, almost all athletes use weight training. One possible way to increase the weight lifted is to apply cryotherapy (ice or cold therapy) to the skin surface before exercise.[1–20] Typically, cryotherapy is used in rehabilitation, but consideration can also be given to other possible uses, particularly in athletics.

Short-duration, local applications of cryotherapy lower muscle temperature and delay the onset of work fatigue.[1–10] Several researchers[1–5] indicated that the longest duration of work occurred when the average muscle temperature was near 27°C. Both higher and lower muscle temperatures resulted in a decrease in the duration of the contractions. Higher temperatures resulted in a significantly faster onset of work fatigue,[3,6,7] while mildly cold applications resulted in significantly longer work periods.[8,9] Extremely cold applications produced less work.[1,3,4,7] One group of investigators[10] obtained better performance using an increased temperature.

Later researchers[11–20] began to study the effects of cooling and warming on velocity and power. Applying cryotherapy reduced velocity and power.[11–15] Some investigators[11,14,16–18] reported enhanced performance as a result of warming muscles. Others[13,19,20] demonstrated that increasing temperature had little or no effect upon subsequent power and that cooling reduced performance. Most of the investigators who studied the relationship between velocity and warm applications used only 1 temperature above normal. In general, previous authors have not used interval cryotherapy between sets of exercises.

In 1 study,[21] 3-minute interval cryotherapy used by baseball pitchers between innings resulted in a significantly higher number of innings pitched and increased velocity with no difference in accuracy. The main purpose of the current study was to replicate the baseball study to determine if similar results would be obtained by increasing the weight from a baseball (142–149 mg) to 75% of the 1-repetition maximum (1RM) arm pull. The number of pulls for each set, cryotherapy time, and rest periods were similar to those used in the interval cryotherapy baseball study.

This procedure may be an innovative approach to delaying the onset of work fatigue while increasing velocity and power. A second area of interest was limiting cryotherapy to 3-minute time periods. Most investigations used a minimum of 20 minutes of cryotherapy immediately before motor performances.

METHODS

I used a 1 × 2 factorial, random, counterbalanced design. Participants received interval cryotherapy on 1 day and towels covering the arm and shoulder on the other day. The dependent variables were total work, velocity, and power.

Subjects

Ten male volunteers (age = 29.0 ± 2.8 years; height = 174.8 ± 7.1 cm; weight = 72.0 ± 2.0 kg; weight-training

Address correspondence to Frank M. Verducci, EdD, Department of Kinesiology, San Francisco State University, 1600 Holloway Avenue, San Francisco, CA 94132. E-mail address: verducci@sfsu.edu

experience = 5.5 ± 1.1 years; number of different sport experiences = 3.0 ± 0.3) who were athletic club members participated in the study. They indicated that they did not have any health, physical, arm, or shoulder problems that could influence the results of their performance before each treatment day.

Treatments

Interval cryotherapy treatment consisted of applying 5 plastic bags (28 × 46 cm) one-third filled with ice cubes (1.9 × 2.5 × 1 cm each) for 3 minutes: 3 on the shoulder and arm and 2 covering the arm and elbow. New ice bags were used after each 3 sets. The towel treatment consisted of covering the shoulder and elbow with 1 towel each for 3 minutes. This may be considered a mild warming of the arm and shoulder similar to baseball pitchers' wearing jackets between innings to keep the arm and shoulder warm. The participants did not lift weights for 1 week before testing, and the treatments were administered 7 days apart.

Work Load

A weight-training device (Figure 1) was used to fatigue the arm and shoulder muscles with a pulling motion. To fatigue the muscles, 74% ± 1.4% of a participant's 1RM was used for resistance. The pull started from a standing position, facing away from the weights, with the subject's upper body rotated to the dominant side, arm fully extended behind the shoulder, and holding the weight-lifting grip in his hand, palm up, with arm parallel to the ground. He positioned himself so that the weights were just off their support. For the right-handed subject, the right foot was at a 45° angle to the pull, while the left foot was parallel to the pull, with the knees slightly bent. On the command "start," the subject pulled the hand grip straight forward by flexing the elbow and shoulder, rotating the shoulder and hips, and finishing the pull with the hand grip in front of the shoulder and the arm fully extended. The pulling motion tended to be in a straight line parallel to the floor. The subject then returned the arm to the original starting position. This constituted 1 pull. The participant pulled the weight 22 times (1 set) as quickly as possible and rested 8 minutes. He repeated pulls and rest periods until muscle fatigue prevented him from completing the 22 pulls without stopping.

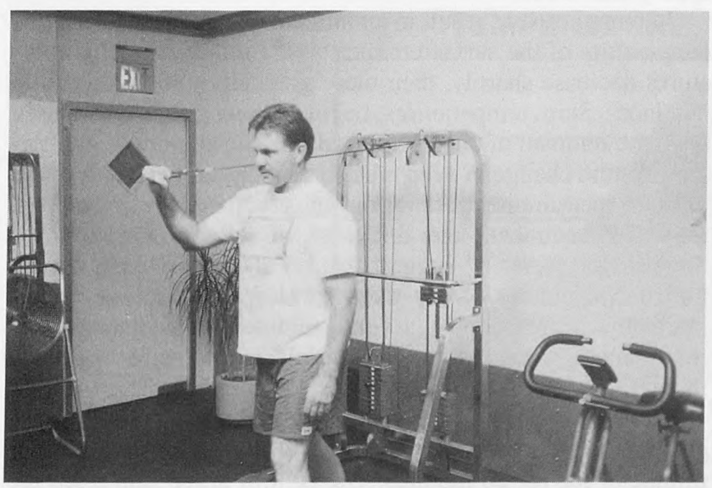


Figure 1. Arm-pull weight-lifting device.

Procedures

During the habituation session, the participants were informed of the purpose and procedures of the study, signed an informed consent form approved by San Francisco State University Committee for the Protection of Human Subjects (which also approved the study), and were randomly assigned to a group for testing order. They warmed up for 3 minutes, observed a demonstration of the movement, and performed 10 pulls with a weight of 6.8 kg (15 lb). The subject's maximum weight for 1 pull was determined by starting with a weight of 18.14 kg (40 lb), followed by the addition of 2.27-kg (5-lb) increments until he could not pull the weight through the full range of motion. Each participant was then tested to determined arm-pull length and pulled approximately 75% of 1RM weight 22 times as fast as possible. (Because the weights were in 2.27-kg increments, I could not obtain exactly 75% of a person's 1RM.) The subject was then scheduled for the 2 testing sessions.

On testing days, participants were asked if they had any soreness or injury to their shoulder or elbow. They then warmed up with the following: 1) running in place for 2 minutes, 2) stretching the arm and shoulder for 1 minute, 3) observing a demonstration of 6 pulls, 4) pulling approximately 75% of maximum weight lifted 11 times at a slow pace within 1 minute, 5) resting 1 minute, 6) pulling the weights 11 times as fast as possible, and 7) resting 3 minutes. The session's remaining procedures were then explained.

Subjects then completed 1 set of 22 pulls as quickly as possible for a base time. The procedures for the next 8 minutes consisted of a 30-second transition from the exercise to the treatments, 3 minutes of cryotherapy or towel treatment, 4 minutes of rest, and another 30-second transition from the rest to the next set of pulls. The number of pulls, treatment times, and rest periods were similar to those used in the previous interval cryotherapy baseball study.[21] This procedure continued until the subject could not complete the 22 pulls without stopping. Ice packs were applied to the shoulder, arm, and elbow for 10 minutes after completion of each testing day. The day after the second testing session was completed, the participants were asked to compare the interval cryotherapy and towel treatments as they related to next-day arm and shoulder soreness.

Length of the arm pull, weight lifted, number of arm-pull sets completed before fatigue, and time to complete repetitions (work rate) were measured; total work (joule = 1 $kg{\cdot}m^2{\cdot}s^{-2}$ = 0.738 ft·lb) and power (watt = $joule{\cdot}s^{-1}$) were calculated. Work was obtained by multiplying the weight lifted by the length of arm pulled, and power was determined by dividing work by time.

Statistical Analysis

The first set did not have a treatment before the 22 pulls. Because this set was not influenced by the cryotherapy or towels, it was used as a covariate in the analysis of covariance. The treatments occurred after this set. The dependent variables of velocity and power were analyzed in 3 different ways: means for first through fourth sets, means for matched sets, and means for all sets. The first through fourth sets include information for all participants. Data are missing from the remaining sets due to fatigue. The scores from sets 5 through 15 were included as part of means analyzed in matched and all sets. Matched scores included those sets with both interval cryotherapy and towel scores for the same set. If a participant had an interval cryotherapy score for the 5th set but not a towel score for the same set, the interval cryotherapy score for the set was eliminated from the analysis. This standardized the amount of work completed in each treatment. All sets included every participant's interval cryotherapy and towel scores. The subjects' interval cryotherapy sets included 14.1% more total work or 14.5% more pulls. Work consisted of the pulled total joules completed before fatigue, excluding the base set.

A 1 × 2 factorial, random, counterbalanced design was used. An analysis of variance was used to analyze work at the 0.05 level of significance. An analysis of covariance, with the base set as the covariate, was used for velocity and power.

RESULTS

Room temperature during testing ranged from 21.1° to 22.2°C. The mean arm-pull length was 1.44 ± 0.045 m, weight lifted was 21.89 ± 1.321 kg, and 1 RM was 29.94 ± 2.079 kg. The Table presents the means, standard errors, and percentage differences for work, velocity, and power.

Work

Participants performed 14.1% more total work ($F_{1,9}$ = 19.02, P = .002) and 14.5% more arm pulls ($F_{1,9}$ = 22.22, P = .001) during the interval-cryotherapy sets. Eight of the 10 participants performed more sets during the cryotherapy condition. The other 2 performed the same number of sets.

Velocities

Velocities for the first to fourth sets ($F_{1,8}$ = 7.45, P = .03), matched sets ($F_{1,8}$ = 12.35, P = .008), and all sets ($F_{1,8}$ = 6.25, P = .04) were higher with cryotherapy. No significant difference in the base sets was seen ($F_{1,9}$ = 0.07, P = .79). Velocity increased for interval cryotherapy between the base and the first to fourth sets, while it decreased during the towel condition. During both interval-cryotherapy and towel conditions, velocities showed a slight decline with each succeeding set, which is typical for fatigue.

Descriptive Statistics for Work, Velocity, and Power

Dependent Variable	Cryotherapy Mean ± SE	Towels Mean ± SE	Difference from Placebo (%)
Work			
Total joules	60233 ± 8223	52795 ± 7424	14.1%
Arm pulls	191.4 ± 23.7	167.2 ± 22.1	14.5%
Velocity* (seconds)			
Base set	38.9 ± 2.2	38.6 ± 1.8	.8%
1st to 4th sets	38.2 ± 1.8	39.7 ± 2.2	−3.7%
1st set	37.9 ± 2.0	38.4 ± 2.3	−1.4%
2nd set	37.5 ± 1.9	39.1 ± 2.1	−4.1%
3rd set	38.0 ± 1.7	40.5 ± 2.5	−6.1%
4th set	39.4 ± 1.7	40.7 ± 2.3	−3.2%
Matched sets	39.1 ± 2.0	41.4 ± 2.6	−5.5%
All sets	39.7 ± 2.1	41.4 ± 2.6	−4.2%
Power (watts)			
Base set	184.2 ± 18.2	182.8 ± 17.5	.8%
1st to 4th sets	185.8 ± 17.7	179.7 ± 17.2	3.4%
1st set	188.3 ± 18.6	185.8 ± 18.0	1.4%
2nd set	189.9 ± 18.7	181.0 ± 16.7	4.9%
3rd set	186.0 ± 17.4	176.6 ± 17.4	5.3%
4th set	178.8 ± 16.6	175.3 ± 16.9	2.0%
Matched sets	182.0 ± 17.7	174.2 ± 17.4	4.5%
All sets	180.1 ± 17.4	174.2 ± 17.4	3.4%

*Lower numerical values indicate faster times.

Power

Participants performed higher power values for the first to fourth sets ($F_{1,8}$ = 6.73, P = .03) and matched sets ($F_{1,8}$ = 8.41, P = .02) when treated with cryotherapy. However, there were no differences for all sets ($F_{1,8}$ = 3.23, P = .11) or base sets ($F_{1,9}$ = 0.09, P = .77). Power increased for interval cryotherapy between the base and the first to fourth sets, while the towel treatment power decreased. Both interval-cryotherapy and towel power values reflected a slight decline with each succeeding set.

Soreness

Six of 10 participants reported having less soreness on the day after lifting when treated with interval cryotherapy. Three could not distinguish a difference between the 2 conditions, and 1 felt more soreness after cryotherapy.

DISCUSSION

The rationale for this study was that muscle temperature remains in a relatively narrow range during resting conditions[22] but increases slowly in a warm environment[23] and rapidly when exercising.[24] Webb[23] reported that in a comfortable environmental condition of 27°C, temperatures were lowest at the skin surface and gradually increased to the highest at a 4-cm muscle site. When sweating starts, comfortable temperature differences tend to disappear. Heat production during exercise may cause muscle temperature to rise as high as 45°C.[25] When muscle temperature increases, the body must adjust rapidly by increasing heat loss in order to return to a normal balance (homeostasis).[26] The 3 major routes for heat dissipation are radiation, convection, and evaporation, with the last being the primary mechanism by which muscle heat is released during exercise.[27] Heat loss by conduction under normal conditions is very small. However, for people sitting or lying on very cold surfaces, conductive heat losses may be a considerable portion of their total heat loss. Cryotherapy results in conductive heat loss[28,29] and may assist the other body mechanisms in reducing muscle temperature to a more appropriate level.

Cold applications result in an immediate and rapid decline in temperature of the surface tissues.[28–32] Initially, these temperatures decrease sharply, then more gradually until a plateau is obtained. Skin temperatures begin to increase immediately upon the removal of the ice application. The amount of muscle-temperature change in deep muscle depends on the depth of the muscle measurement, duration of cryotherapy application, mode of treatment, and thickness of adipose tissue.[29,33,34] Some researchers[29,34] suggest that no effect is evident during the first 3 minutes of cryotherapy. Deep-muscle temperature gradually decreases for several minutes immediately after removing cryotherapy,[34] then muscle temperature begins to increase.

Researchers[35] measured temperature change in leg muscles at 1 cm during contrast therapy of 4 minutes of heat and 1 minute of ice for 20 minutes. The control group immersed the leg in a hot whirlpool (40.6°C) for 20 minutes. The contrast group had a significantly smaller increase (0.39°C) in muscle temperature than the control group (2.83°C). Alternating heat and cold treatment resulted in very little muscle-temperature change at 1 cm. Perhaps similar results occurred in this study. The heat production during the weight-pulling exercise may resemble the hot-whirlpool treatment in increasing muscle temperature. This, followed by the 3 minutes of icing and 4.5 minutes of rewarming, could have produced results comparable with the contrast treatment.

I applied 3-minute interval cryotherapy between innings on university baseball pitchers.[21] The participants threw 26% more pitches, which is greater than the 14% obtained with the heavier resistance in the current study. Interval cryotherapy appears to be effective for both light (142–149 mg) and heavy (21.9 kg) resistance-type work. Other studies[1–9,30–32] suggest that the most effective localized muscle temperature for accomplishing the most work is lower than normal temperature. As one moves away from this effective temperature, less and less work is accomplished: extremely high or low temperatures produced the least amount of work. Researchers[1] using water-bath temperatures of 2°C, 10°C, 14°C, 18°C, 26°C, 34°C, and 42°C reported the maximum duration of sustained contraction was at 18°C. A graph of the investigators' results[1–9,21] was similar to a normal probability curve, with its center being slightly below normal temperature (Figure 2). The 3-minute icing with 4.5 minutes of rewarming may have brought the temperature closer to the optimal work temperature. In general, more work is accomplished when the temperature is lower than normal.[1–9,21]

Lowering tissue temperatures results in decreased velocities,[12–14,19] but the literature is conflicting on the effect of motor performance at normal and higher-than-normal muscle temperatures. Several investigators[11,16–18] reported greater velocities at increased temperatures than at normal conditions, while others[13,19,20] indicated that increasing temperature above normal had little or no effect on subsequent velocity. Interestingly, 2 of these studies[19,20] used water baths of 40°C and 46°C, respectively, which suggests that extremely high temperatures may be no different than normal temperatures. Perhaps the velocity performances' distribution could also be similar to a normal probability curve[11–20] with its center somewhere between normal and high temperatures (Figure 2).

The velocity results were similar to those obtained in the baseball investigation.[21] The baseball interval-cryotherapy velocities were significantly faster by 3.7% for second to fourth, 3.7% for matched, and 2.9% for all innings. These percentage changes tended to be either equal to or lower than the respective weight-lifting sets of 3.7%, 5.5%, and 4.2%.

Participants' cryotherapy power values were higher for the first to fourth sets and matched sets but not for all sets. The lack of significant difference in all sets may be associated with the 14.1% more work completed in the interval-cryotherapy treatment.

In the baseball investigation,[21] 5 of 6 pitchers indicated they had less arm soreness the next day with interval cryotherapy. In my study, 6 of 10 participants reported less soreness. The participants in both studies performed more work.

One factor that may have influenced the results is the Hawthorne[36] effect, by which participants performed better because they believed they were receiving special attention and treatment. Because the interval-cryotherapy treatment was counter to all previous practices in weight training, the participants may have believed they were receiving special attention. Although towels were used in this study, the Hawthorne effect may have influenced performance.

The direct application of interval cryotherapy to weight training still needs to be demonstrated. Strength development usually involves 6 to 12 repetitions per set at a low speed, with 3 minutes' rest between sets. The 22 repetitions at a high velocity with 8 minutes of rest between sets may not be transferable to strength training. Further research must be conducted to support the use of interval cryotherapy in weight training.

The exact nature and cause of the complex phenomenon of muscle fatigue remains obscure. Performances affected by different localized environmental temperatures may be concurrently affected by many other factors, such as muscle temperature, the beneficial effects of cryotherapy on pain reduction, the functioning of the arm and shoulder joints, the depletion of substances needed for muscle contraction, byproducts of muscle contractions, accumulation of waste products in the muscles, reduction of secondary hypoxic injuries, the functioning of the central nervous system, rapid change in local environmental temperature, psychological considerations, or a combination of these. Researchers developing models dealing with fatigue should consider including the variable of muscle temperature.

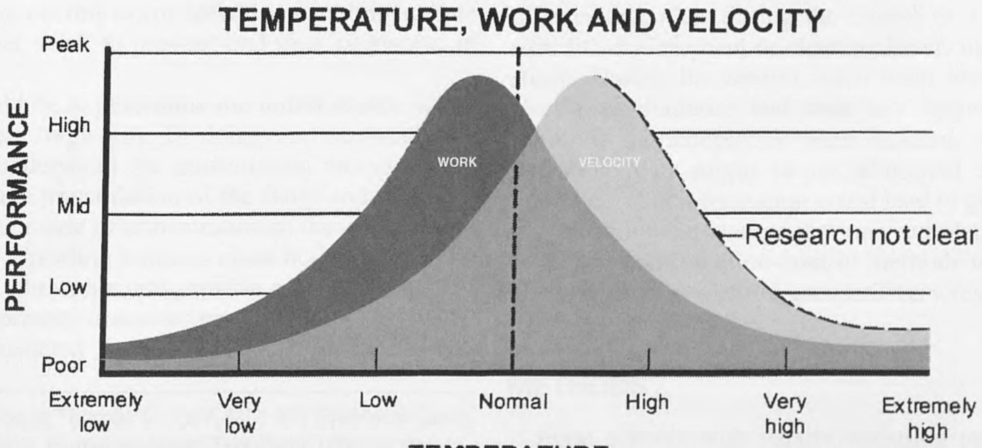


Figure 2. Model for temperature, work, and velocity as related to efficient motor performance.[1–9,11–21]

REFERENCES

1. Clarke RS, Hellon RF, Lind AR. The duration of sustained contraction of the human forearm at different muscle temperatures. *J Physiol.* 1958;220: 454–473.
2. Galloway SDR, Maughan RJ. Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. *Med Sci Sports Exerc.* 1997;29:1240–1249.
3. Grose JE. Depression of muscle fatigue curves by heat and cold. *Res Q.* 1958;29:19–23.
4. Lind AR. Muscle fatigue and recovery from fatigue induced by sustained contraction. *J Physiol.* 1959;147:162–177.
5. Olschewski H, Bruck K. Thermoregulatory, cardiovascular, and muscular factors related to exercise after precooling. *J Appl Physiol.* 1988;64: 803–811.
6. Chastain PB. The effect of deep heat on isometric strength. *Phys Ther.* 1978;58:543–546.
7. Edwards RH, Harris RC, Hultman E, Kaijser L, Koh D, Nordesjo LO. Effects of temperature on muscle energy metabolism and endurance during successive isometric contractions, sustained to fatigue, of the quadriceps muscle in man. *J Physiol.* 1952;220:335–352.
8. McGowan HL. The effects of cold application on maximal isometric contraction. *Phys Ther.* 1967;47:185–192.
9. van Beek EJ. *Effects of Reducing Intramuscular Temperature on Delaying the Onset of Fatigue: An Electromyographical Analysis* [master's thesis]. London, Ontario, Canada: University of Western Ontario; 1975.
10. King PC, Mendryk S, Reid DC, Kelly R. The effect of actively increased muscle temperature on grip strength. *Med Sci Sports Exerc.* 1970;2: 172–175.
11. Berg U, Ekblom B. Influence of muscle temperature on maximal muscle strength and power output in human skeletal muscles. *Acta Physiol Scand.* 1979;96:33–37.
12. Carlos J. *The Effects of Short-Term and Long-Term Ice Pack Applications on Peak Power and Mean Power* [dissertation].Tallahassee, FL: Florida State University; 1991.
13. Howard RL, Kraemer WJ, Stanley DC, Armstrong LE, Maresh CM. The effects of cold immersion on muscle strength. *J Strength Cond Res.* 1994;8:129–133.
14. Mattacola C, Perrin DH. Effects of cold water application on isokinetic strength of the plantar flexors. *Isokinet Exerc Sci.* 1993;3:152–154.
15. Oksa J, Rintamaki H, Rissanen S. Muscle performance and electromyogram activity of the lower leg muscles with different levels of cold exposure. *Eur J Appl Physiol Occup Physiol.* 1997;75:484–490.
16. Davies CTM, Mecrow IK, White MI. Contractile properties of the human triceps surae with some observations on the effects of temperature and exercise. *Eur J Appl Physiol.* 1982;49:255–269.
17. Davies CTM, Young K. Effect of temperature on the contractile properties and muscle power of triceps surae in humans. *J Appl Physiol.* 1983;55(1 Pt 1):191–195.
18. Sargeant AJ. Effect of muscle temperature on leg extension force and short-term power output in humans. *Eur J Appl Physiol Occup Physiol.* 1987;56:693–698.
19. Clarke DH, Royce J. Rate of muscle tension development and release under extreme temperatures. *Int Z Angew Physiol Einschl Arbeitsphysiol.* 1962;19:330–336.
20. Cornwall MW. Effect of temperature on muscle force and rate of muscle force production in men and women. *J Orthop Sports Phys Ther.* 1994;20:74–80.
21. Verducci FM. Interval cryotherapy and fatigue in university baseball pitchers. In: Fourth International Olympic Committee World Congress on Sports Sciences: Congress Proceedings; October 22–25, 1997; Monte Carlo; p 107. Abstract.
22. Hardy JD. The physiology of temperature regulation. *Physiol Rev.* 1961;41:521–601.
23. Webb P. Temperatures of skin, subcutaneous tissue, muscle and core in resting men in cold, comfortable and hot conditions. *Eur J Appl Physiol Occup Physiol.* 1992;64:471–476.
24. Nielsen B, Nielsen M. Body temperature during work. *Skan Arch Physiol.* 1962;56:120–129.
25. Salo DC, Donovan CM, Davies KJ. HSP70 and other possible heat shock or oxidative stress proteins are induced in skeletal muscle, heart, and liver during exercise. *Free Radic Biol Med.* 1991;11:239–246.
26. Bazett HC. The regulation of body temperature. In: Newburgh LH, ed. *Physiology of Heat Regulation and Science of Clothing*. Philadelphia, PA: WB Saunders; 1949:109–122.
27. Stitt JT. Central regulation of body temperature. In: Gisolfi CV, Lamb DR, Nadel ER, eds. *Perspectives in Exercise Science and Sports Medicine: Exercise, Heat and Thermoregulation.* Vol 6. Dubuque, IA: William C Brown & Benchmark; 1993;1–39.
28. Knight KL. *Cryotherapy: Theory, Technique and Physiology*. Chattanooga, TN: Chattanooga Corp; 1985:73–82.
29. Knight KL. *Cryotherapy in Sport Injury Management*. Champaign, IL: Human Kinetics; 1995:64–73.
30. Myrer JW, Measom G, Durrant E, Fellingham GW. Cold- and hot-pack contrast therapy: subcutaneous and intramuscular temperature change. *J Athl Train.* 1997;32:238–241.
31. Myrer JW, Measom G, Fellingham GW. Temperature changes in the human leg during and after two methods of cryotherapy. *J Athl Train.* 1998;33:25–29.
32. Ray CA, Hume KM, Gracey KH, Mahoney ET. Muscle cooling delays activation of the muscle metaboreflex in humans. *Am J Physiol.* 1997; 273(5 Pt 2):2436–2441.
33. Lowden BJ, Moore RJ. Determinants and nature of intramuscular temperature changes during cold therapy. *Am J Phys Med.* 1975;54:223–233.
34. Merrick MA, Knight KL, Ingersoll CD, Potteiger JA. The effects of ice and compression on intramuscular temperatures at various depths. *J Athl Train.* 1993;28:236–245.
35. Myrer JW, Draper DO, Durrant E. Contrast therapy and intramuscular temperature in the human leg. *J Athl Train.* 1994;29:318–322.
36. Brown JAC. *The Social Psychology of Industry*. Middlesex, England: Penguin; 1954.

Journal of Athletic Training 2000;35(4):427–430

www.journalofathletictraining.org

Wrestling Mats: Are They a Source of Ringworm Infections?

Thomas D. Kohl, MD; David C. Martin, MD; Richard Nemeth, MD; Deborah L. Evans, SM (AAM); Berks County Scholastic Athletic Trainers' Association*

Objective: To determine if the fungal molds (dermatophytes) responsible for causing ringworm could be isolated from a section of wrestling mat during the 1998–1999 season.

Design and Setting: A 2-part study was conducted. The first phase involved a culture evaluation of material taken from wrestling mats at 8 local high schools. The second phase was a bench laboratory study to determine if the fungus and molds could be grown from a wrestling mat in an optimal setting.

Subjects: We obtained material from areas of the practice mats of 8 high school wrestling teams at monthly intervals during the wrestling season. A 0.61-m (2-ft) × 0.31-m (1-ft) area of mat from 1 of the schools was used for the laboratory phase of the study.

Measurements: We cultured samples taken from each school's wrestling mats for growth of dermatophytes and used a questionnaire to determine the mat-washing habits and policies of each school. Also, wrestlers from the 8 schools were screened weekly by the designated team physician and certified athletic trainer. Any suspicious lesions were cultured for fungi.

Results: No dermatophytes were grown from the swab specimens taken at the 8 schools, and no dermatophytes were isolated from a section of mat evaluated in optimal laboratory conditions.

Conclusions: It is unlikely that wrestling mats are a source of ringworm infections in wrestlers.

Key Words: tinea gladiatorum, wrestling, tinea corporis, infection control

Dermatologic infections are a fairly common disease entity in contact sports. Wrestling in particular provides a competitive environment in which herpesviruses, *Staphylococcus* bacteria, and dermatophytes thrive. A small number of outbreaks of ringworm infections in wrestlers in the United States have been reported,[1–5] despite the widely held belief that these epidemics are quite commonplace in the wrestling world.[6] Prevention strategies such as hygienic practices, including showering and laundering clothing, in addition to educating officials, coaches, athletic trainers, parents, and athletes have been advocated in the literature.[7] We have found these measures to be inadequate in primary prevention.[8] As health care providers for these athletes, we must try to prevent these outbreaks by defining the disease entity better. With a better understanding of ringworm infections in this unique setting, we can better work to prevent and treat ringworm in wrestlers.

One strategy would be to determine the initial source of the organisms that cause ringworm. In doing so, we would be better equipped to understand the transmission and construct methods to prevent the transmission of the fungi and molds. It is most likely that the mode of transmission of these infections is person to person. Wrestling requires close body contact and often results in skin abrasions that provide a perfect opportunity for person-to-person transmission. However, dermatophytes have been isolated from several inanimate objects, including hairbrushes, combs, pillowcases, other bedding material, and dormitory floors.[9] Inanimate objects, or fomites, may be responsible for prolonged transmission of ringworm infections.[10] The competitive wrestling environment includes many fomites as possible sources of contagion. In a survey of 229 certified athletic trainers, athletic directors, and wrestling coaches in Pennsylvania, respondents identified towels, practice clothing, headgear, and wrestling mats as theorized sources of ringworm in high school wrestlers. Three fourths of those responding believed that ringworm could be contracted from wrestling mats (T.D. Kohl, unpublished data, 1999). To our knowledge, no one has proved that dermatophytes can exist on wrestling mats. We designed this 2-phase study to determine if dermatophytes could be found on samples of wrestling mats during the course of a season. We did not alter the environment or cleaning habits in any way during this study. During the season, each team involved was followed closely to diagnose and treat any ringworm infections that arose. If dermatophytes were isolated, we could infer that wrestling mats might be an additional source of ringworm infection. That information could lead to guidelines concerning mat cleaning and storage that may supplement evidence-based treatment and infection-control methods to lessen the number of cases of ringworm in competitive wrestlers.

METHODS

Eight schools with varsity wrestling programs in the same county and same league were involved in this study. We obtained consent from athletic directors and school boards

Address correspondence to Thomas D. Kohl, MD, 901 Prestwick Lane, Newport News, VA 23602. E-mail address: Tkohl34841@aol.com

*A complete list of the members of the Berks County Scholastic Athletic Trainers' Association appears at the end of this article.

before the season. Demographics of each school were obtained from the athletic directors of each school.

The first phase was a field study to evaluate cultured material from wrestling mats at each school. Culture samples were obtained from a 0.31-m × 0.31-m (1 square foot) area at the center of each school's practice mats. The surface area of each mat was swabbed before the regular season (early November) and monthly during the season (December, January, and February). The first sample was taken before any formal wrestling practices were conducted and after the mats had been sterilized. The subsequent monthly samplings were taken immediately after practice on the predetermined date each month, before any mat cleansing was done, to improve the chance of finding organisms. A sterile cotton swab was moistened with sterile water before collecting the specimen. Each specimen was acquired by rigorously rubbing the swab in multiple directions over the designated area at the center of the practice mats in the wrestling rooms of each school. This method of screening for dermatophyte growth on fomites has been shown to be effective in isolating the organisms.[11,12] The swab was returned to the culturette transport medium and transported to the microbiology laboratory within 24 hours. The reliability of using a cotton swab technique to isolate dermatophytes has been shown elsewhere.[13]

Each specimen was swabbed onto a Mycosel agar (Becton Dickinson, Sparks, MD). Mycosel is a Sabouraud-dextrose base agar that contains chloramphenicol, a broad-spectrum antibiotic to inhibit bacterial growth, and cycloheximide, which inhibits saprophytic fungi growth. Agar containing chloramphenicol and cycloheximide is the standard medium for growing dermatophytes.[14] Therefore, Mycosel would allow growth and recovery of dermatophytes responsible for ringworm infections while preventing the growth of contaminating organisms. We examined the agar plates weekly for growth. Colonies of mold suspected of possibly being a dermatophyte were reisolated to Sabouraud-dextrose plates for further identification.

We used a questionnaire to determine the mat-washing habits and policies of each school, including the frequency of washing, the substance used to clean the mats, and the person(s) responsible for cleaning the mats. The athletic trainers or wrestling coaches, or both, at each school provided the information.

The wrestlers from these 8 schools underwent weekly screening examinations by the designated team physician and certified athletic trainer. If any lesions were found during a screening examination or were self-reported by the wrestler, an epidermal scraping was obtained from the suspicious lesion for fungal culture. The epidermal cells were collected into agar tubes containing Dermatophyte Test Medium (DTM [Acuderm, Fort Lauderdale, FL]). The culture tubes were incubated at room temperature for 4 weeks. A positive culture was defined as growth on DTM with characteristic color change from yellow to red indicating alkalinization. Positive DTM cultures were subcultured on a fresh Sabourad-dextrose plate for 72 hours. The isolates were then examined microscopically after staining with lactophenol blue dye. A small sample of the mold was then inoculated onto Trichophyton agars 1–4 and Christensen's urea agar (Becton Dickinson, Sparks, MD). These tubes were examined daily for 10 days to determine the growth patterns. Six of the positive DTM cultures were not further identified because of inability to recover the organism secondary to delay in transport to the microbiology laboratory.

The second phase of the study was a bench laboratory experiment to determine if dermatophytes could be isolated from a sample of wrestling mat in ideal conditions. A 0.61-m × 0.61-m (2-ft × 1-ft) piece of a practice wrestling mat from 1 of the participating schools was removed from an existing practice mat and transported to the microbiology laboratory for evaluation. Touch preparations of the mat onto a fungal medium plate were performed to determine if the mat could be a source of dermatophyte growth. If any dormant fungal or dermatophyte spores were present on the mat, they would sporulate and grow once they had optimal conditions for growth. All agars were incubated at 28°C to 30°C in a Thelco model #4 incubator (Thelco, Englewood, CO). Two areas of the mat were chosen for the touch preparations, 1 dirty area and 1 clean area. The dirty area contained debris and dust. The clean area was relatively free of debris and dust to the naked eye. The sample of mat had not been washed or disinfected in 23 hours.

RESULTS

The 8 schools included 1 urban, 5 suburban, and 2 rural schools. The average number of wrestlers per school was 20.3 (range, 14 to 29). Coaches at all schools reported an average of 15 hours of practice time per week. Only 1 school in this study was free of ringworm during the season. The number of infections, number of wrestlers, and mat-washing habits from each school are provided in the Table. Each school used a commercial mat cleaner (Mint Quat [3M, Minneapolis, MN], Brute [Hadco-Denver Chemical Co, Denver, CO], or Vionex [Viro Research International, Toledo, OH]) to clean its mats. All cleaners contained ammonium chloride as the primary active ingredient.

A total of 32 mat samples were analyzed from the 8 schools. None of the mat specimen cultures collected at any time before, during, or after the season produced any dermatophyte growth. A few suspicious molds were isolated from 7 cultures from 4 different schools. These mold colonies were positively identified as *Penicillium* and *Fusarium*, which are common environmental molds that do not cause clinical disease. One culture grew *Aspergillus*, which could produce infection in an immunocompromised host. Two samples produced a large amount of yeast, which again were most likely environmental in nature. Finally, bacteria were found in 4 cultures (from 3 different schools) during evaluation. The bacteria were not further identified.

Characteristics of the 8 Wrestling Programs for the 1998–1999 Season

School	Wrestlers (n)	Ringworm Cases*	Frequency of Mat Cleaning
B	16	1 (6.25)	Daily
G	22	6 (27.2)	Daily
H	14	0 (0)	Daily
R	19	3 (15.7)	Monthly
S	29	1 (3.4)	Daily
WE	17	3 (17.6)	Daily
WI	25	5 (20)	Daily
WY	21	3 (14.2)	Daily

*Number of cases during the regular wrestling season (team incidence in percent for the entire season).

The 2 touch preparations that were processed in the laboratory did not reveal any dermatophyte growth. Each preparation, 1 from a dirty area and 1 from a clean area of mat sample, produced yeast and environmental molds. No bacteria were isolated from the touch preparations.

DISCUSSION

Tinea gladiatorum represents a significant nuisance to the wrestling world and to the health care professionals who care for wrestlers. Based on review of what little is in the literature and personal experience, outbreaks of ringworm infections seem to be the most common presentation. The best way to combat this problem is to determine the origin of the first dermatophyte. Prevention is the key to keeping wrestlers on the mats. Several possibilities may explain how these miniepidemics are started, but no single answer is likely to explain all the epidemics. If the dermatophytes responsible for ringworm infections have been isolated on inanimate objects before, it is logical to believe that they may exist on inanimate objects to which the wrestlers are exposed. We sought to prove or disprove 1 part of that theory with this study.

The mats that were used for practice at the 8 schools in Berks County did not harbor the organisms responsible for ringworm at the times and in the areas that we sampled. The mat-cleaning habits of the schools and the hygiene of the wrestlers were not controlled or altered in any way. We studied the mats in the state in which the participating schools kept them. We diagnosed infections in all but 1 of the schools. *Trichophyton tonsurans* was isolated from 17 of 23 positive DTM-screening cultures. The other positive DTM cultures could not be further identified. The predominance of *Trichophyton tonsurans* is consistent with other reports of ringworm infections in this population.[1–3,5,8,15] We showed in previous work[8] that it often takes more than 1 to 2 weeks to eradicate this dermatophyte in the wrestling population. Even if the mat was not the original source of the infections, there may have been exposure to the dermatophytes responsible for the infections when the infected wrestlers were practicing. Evidence has supported the existence of *Trichophyton tonsurans* outside a host for a prolonged period in an artificial setting.[16] If the dermatophyte or its spores were able to survive or perpetuate on wrestling mats, they should have been isolated.

Two explanations can illustrate why we were unable to isolate dermatophytes from the monthly mat cultures at the 8 schools. First, there may not have been enough dermatophytes present to spur growth. If this were the case, then there would not be enough organisms to produce or induce a clinical infection. Seven of the 8 schools washed or disinfected their mats daily. The disinfectant used by each school contains an active fungicidal ingredient. This would certainly limit the load of dermatophytes or any other organisms on the mats. Our results may make a strong argument in favor of washing the mats daily. By looking for the dermatophytes without altering the normal regimen of mat cleaning, we can infer that it is unlikely that the mats were the cause of the ringworm infections in these wrestlers because the mats were cleaned on a regular basis. Second, perhaps dermatophytes were there, but we missed them by only sampling a small area. We did sample the same area each month, and the area sampled was in the center of the mat, where much action presumably takes place. We may have also missed the opportunity to find any dermatophytes by only sampling monthly. We did increase our chance by sampling immediately after practice times. It is also difficult to determine if the wrestlers had sufficient contact with the mat at that particular area. A larger study with more random sampling may reveal dermatophytes on the mat.

Because we were unable to isolate dermatophytes from the mats and because we were limited in our ability to sample larger areas of the mats, the laboratory experiment phase was initiated. Under ideal growth conditions, the mat sample did not produce evidence of dermatophyte contamination. If dermatophytes could not be isolated under ideal conditions, it is difficult to believe that they can survive on mats in sufficient concentrations to produce clinical infections. The same limitations hold true of this part of the study. We only sampled 1 piece of mat from 1 school; this limited our ability to isolate any dermatophytes. The lack of growth under laboratory conditions does add some evidence to conclude that it is unlikely that dermatophytes can persist on wrestling mats in sufficient quantity or for sufficient duration to cause clinical infections.

The inability to isolate dermatophytes in either phase of this study lends more credence to the theory that person-to-person contact is the most likely mode of transmission. Others have reached a similar conclusion. Beller and Gessner[1] provided several reasons to believe the transmission of tinea gladiatorum is by direct skin-to-skin contact. Extensive cleaning of the mats with a disinfectant capable of killing dermatophytes failed in their study and in our study to prevent tinea infections. Beller and Gessner[1] also noted that there should have been more lower extremity infections if the mat played a role in the outbreak they studied. Direct contact with the fungus, in combination with other factors, including concomitant skin trauma and host susceptibility, is the most important source of infection.[17] Nosocomial outbreaks of *Trichophyton tonsurans* have affirmed direct personal contact as the prominent source of transmission.[11,18] The presence of a dermatophyte on a fomite or as part of a carrier state does not affirm it as the definitive source. The principles of infectious disease require a viable organism, a susceptible host, and an appropriate environment for clinical infection to occur. We need to study all aspects of this infection in this population in order to develop strategies to deal with it. We suggest focusing our efforts on studying the person-to-person transmission, studying when return to competition is safe, and looking at efficacy of treatment and prevention techniques such as the use of skin barriers[19] and pharmacologic prophylaxis.[20] We would suggest continuation of common-sense hygiene measures, including showering after every encounter, washing practice clothes daily, and disinfecting mats daily.[21] Until we have more definitive answers about ringworm in wrestlers, it is impossible to have sufficient infection control and prevention plans.

ACKNOWLEDGMENTS

We extend our gratitude to the following members of the Berks County (PA) Athletic Trainers' Association for their role in data collection: Daniel Giesen, ATC; John Moyer, ATC; Todd Bartley MS, ATC; Glenn Thompson, MEd, ATC; Terry Ventresca, ATC; Jennifer Ganter, MEd, ATC; Jennifer Motze, ATC; and Matt Blimline, ATC. We also thank the administration, athletic directors, and wrestling coaches in the following school districts for their cooperation: Wyomissing Area School District; Wilson School District; Reading School District; Governor Mifflin School District; Conrad Weiser

School District; Hamburg Area School District; Brandywine Heights School District; and Schuylkill Valley School District.

REFERENCES

1. Beller M, Gessner BD. An outbreak of tinea corporis gladiatorum on a high school wrestling team. *J Am Acad Dermatol.* 1994;31(2 Pt 1):197–201.
2. Cohen BA, Schmidt C. Tinea gladiatorum [letter]. *N Engl J Med.* 1992;327:820.
3. Hradil E, Hersle K, Nordin P, Faergemann J. An epidemic of tinea corporis caused by Trichophyton tonsurans among wrestlers in Sweden. *Acta Dermatol Venereol.* 1995;75:305–306.
4. Werninghaus K. Tinea corporis in wrestlers [letter]. *J Am Acad Dermatol.* 1993;28:1022–1023.
5. Stiller MJ, Klein WP, Dorman RI, Rosenthal S. Tinea corporis gladiatorum: an epidemic of Trichophyton tonsurans in student wrestlers. *J Am Acad Dermatol.* 1992;27:632–633.
6. Dienst WL, Dightman L, Dworkin MS, et al. Pinning down skin infections. *Physician Sportsmed.* 1997;25(12):45–56.
7. Mast EE, Goodman RA. Prevention of infectious disease transmission in sports. *Sports Med.* 1997;24:1–7.
8. Kohl TD, Martin D, Berger MS. Comparison of topical and oral treatments for tinea gladiatorum. *Clin J Sport Med.* 1999;9:161–166.
9. Rippon JW. *Medical Mycology.* 2nd ed. Philadelphia, PA: WB Saunders; 1982:154–248.
10. Kemna ME, Elewski BE. A US epidemiologic survey of superficial fungal diseases. *J Am Acad Dermatol.* 1996;35:539–542.
11. Arnow PM, Houchins SG, Pugliese G. An outbreak of tinea corporis in hospital personnel caused by a patient with Trichophyton tonsurans infection. *Pediatr Infect Dis J.* 1991;10:355–359.
12. Kane J, Leavitt E, Summerbell RC, Krajden S, Kasatiya SS. An outbreak of Trichophyton tonsurans dermatophytosis in a chronic care institution for the elderly. *Eur J Epidemiol.* 1988;4:144–149.
13. Head ES, Henry JC, Macdonald EM. The cotton swab technique for the culture of dermatophyte infections: its efficacy and merit. *J Am Acad Dermatol.* 1984;11(5 Pt 1):797–801.
14. Aly R. Culture media for growing dermatophytes. *J Am Acad Dermatol.* 1994;31(3 Pt 2):S107–108.
15. Rosenthal S, Sangueza OP, Klein WP, et al. Brote epidemico de tinea corporis producido por Trichophyton tonsurans en estudiantes universitarios practicantes de lucha greco-romana. *Piel.* 1992;7:483–485.
16. Hebert AA, Head ES, Macdonald EM. Tinea capitis caused by Trichophyton tonsurans. *Pediatr Dermatol.* 1985;2:219–223.
17. Baxter DL. Superficial and deep mycotic infections. In: Moschella SL, Pillsbury DM, Hurley HJ, eds. *Dermatology.* Philadelphia, PA: WB Saunders; 1975:621–707.
18. Lewis SM, Lewis BG. Nosocomial transmission of Trichophyton tonsurans tinea corporis in a rehabilitation hospital. *Infect Control Hosp Epidemiol.* 1997;18:322–325.
19. Hand JW, Wroble RR. Prevention of tinea corporis in collegiate wrestlers. *J Athl Train.* 1999;34:350–352.
20. Hazen PG, Weil ML. Itraconazole in the prevention and management of dermatophytosis in competitive wrestlers. *J Am Acad Dermatol.* 1997;36(3 Pt 1):481–482.
21. Nelson M. Stopping the spread of herpes simplex: a focus on wrestlers. *Physician Sportsmed.* 1992;20(10):116–127.

Journal of Athletic Training 2000;35(4):431–440

www.journalofathletictraining.org

Evaluation of a Screening Test for Female College Athletes with Eating Disorders and Disordered Eating

Deborah L. Nagel, PhD, RD, EdS*; David R. Black, PhD, MPH, CHES*; Larry J. Leverenz, PhD, ATC*; Daniel C. Coster, PhD†

*Purdue University, West Lafayette, IN; †Utah State University, Logan, UT

Objective: To develop a screening test to detect female college athletes with eating disorders/disordered eating (ED/DE). No validated eating disorder screening tests specifically for athletes have been available.

Design and Setting: In this cross-sectional study, subjects from a large midwestern university completed 3 objective tests and a structured diagnostic interview.

Measurements: A new test, developed and pilot tested by the researchers (Athletic Milieu Direct Questionnaire, AMDQ), and 2 tests normed for the general population (Eating Disorder Inventory-2, Bulimia Test-Revised) were used to identify ED/DE athletes. A structured, validated, diagnostic interview (Eating Disorder Examination, version 12.0D) was used to determine which test was most effective in screening female college athletes.

Subjects: Subjects included 149 female athletes, ages 18 to 25 years, from 11 Division I and select club sports.

Results: ED/DE subjects (35%) were found in almost every sport. Of the ED/DE subjects, 65% exhibited disordered eating, 25% were bulimic, 8% were classified as eating disordered not otherwise specified (NOS), and 2% were anorexic. The AMDQ more accurately identified ED/DE than any test or combination of items. The AMDQ produced superior results on 7 of 9 epidemiologic analyses; sensitivity was 80% and specificity was 77%, meaning that it correctly classified approximately 4 of every 5 persons who were truly exhibiting an eating disorder or disordered eating.

Conclusions: We recommend that the AMDQ subsets, which met statistical criteria, be used to screen for ED/DE to enable early identification of athletes at the disordered eating or NOS stage and to initiate interventions before the disorder progresses.

Key Words: anorexia, bulimia, dieting behaviors, Eating Disorder Examination

Disordered eating is one of 3 components of a serious syndrome called the female athlete triad. Its interrelated components include disordered eating, amenorrhea, and osteoporosis.[1] Research has shown that sports emphasizing low body weight pressure female athletes to achieve and maintain extremely unrealistically low body weights and body fat percentages.[1] Athletes are 2 to 3 times more likely than nonathletes (ie, general population and college students) to manifest characteristics of eating disorders.[2] In 1 study of 22 colleges and universities (n = 695 athletes), approximately 3% of the athletes met the medical criteria for anorexia nervosa, and 21% met the criteria for bulimia nervosa.[2] A substantial number of athletes (as high as 62%) practice pathogenic weight-control behaviors.[3–6] Despite serious medical complications (eg, bradycardia, electrolyte abnormalities, dehydration, dental erosion, hypotension)[7] associated with eating disorders and disordered eating, no screening test has been developed specifically for an athlete population. Current screening tests such as the Eating Disorders Inventory-2 (EDI-2)[8,9] (Psychological Assessment Resources, Inc, Odessa, FL) and the Bulimia Test-Revised (BULIT-R)[10,11] (M.H. Thelen, Columbia, MO) have not been validated with athletes, and the sensitivity and specificity of these tests in athletes are questionable. Wilmore,[12] for example, described 1 study that used the EDI to assess 14 female distance runners. The EDI identified only 3 of these athletes as having possible problems but not clear eating disorders. Seven runners, however, were subsequently diagnosed as having an eating disorder that required inpatient or outpatient treatment, or both. Wilmore[12] also noted similar conclusions in another study in which the Eating Attitudes Test (EAT) was administered to 110 elite female athletes. Based on the EAT, none of the athletes scored in the eating-disordered range, yet 18 (16.4%) received either inpatient or outpatient treatment for eating disorders in the subsequent 2-year period. O'Connor et al[13] also concluded that the EDI-2 can be easily faked and that response bias should be accounted for when using the EDI-2. These studies illustrate the diagnostic problems associated with the use of current, commercial, validated screening tests for eating disorders for the general population when they are used in female athletes.

The purpose of our study was to develop a screening test for eating disorders/disordered eating (ED/DE) specifically for female college athletes. The study was conducted as part of an initiative to develop a screening test for widescale distribution. The following research questions were addressed in this study. (1) Because conventional diagnostic and screening tools are

Address correspondence to David R. Black, PhD, MPH, CHES, Department of Health, Kinesiology, and Leisure Studies, Purdue University, 1362 Lambert Building, West Lafayette, IN 47907-1362. E-mail address: blackran@gte.net

not normed for athletes, are they the most effective tools to use in an athlete population? (2) Will specific items from the Athletic Milieu Direct Questionnaire (AMDQ), which we developed, or the 2 commercial screening tests not normed for athletes be useful in the development of a screening test specifically designed for female college athletes? We hypothesized that the AMDQ would more accurately assess the presence of ED/DE among female college athletes than the EDI-2 or BULIT-R, as verified by a systematic, psychometrically validated, diagnostic interview. Alternatively, we hypothesized that some combination of items from the AMDQ, EDI-2, and BULIT-R would more accurately assess the presence of ED/DE than any of these tests independently. Our extended purpose was to develop a new ED/DE screening test for female college athletes using items from the test pool (ie, the AMDQ, EDI-2, and BULIT-R) that best discriminated athletes with ED/DE from athletes without these disorders. The end product, therefore, would be a shorter test, specific for female college athletes, which could be used to screen for ED/DE.

METHODS

Subjects

The subjects' (n = 149) mean age was 20 years. Self-reported and observed weights were 61.24 kg (135 lb) and 62.14 kg (137 lb), respectively. Mean self-reported and observed heights were both 167.64 cm (66 in), body mass index (BMI, kg/m^2) was 22, and body fat percentage was 18. Subjects were athletes from a large midwestern Division I university and were recruited from all sports (n = 11) involving female athletes (ie, basketball, cheerleading, dance company, modern dance, golf, gymnastics, softball, swimming, tennis, track and cross-country, and volleyball). A census-selection procedure was used because the total number of participants was manageable in terms of the human and economic resources available to conduct the study. The institutional office of the Committee on the Use of Human Research Subjects approved the study and the procedures used for data collection.

Procedures

Test Administration. Data were collected in 2 sessions. In session 1, all subjects completed 3 tests in the order AMDQ, EDI-2, and BULIT-R. The time of completion of all 3 tests at 1 sitting was approximately 1 hour. The AMDQ was constructed because published eating disorder screening tests do not focus on athletes or the effect of the athletic environment on the athlete. Items for the AMDQ were developed based on Black[14]; Brownell et al[15]; Holliman[16]; Thompson and Sherman[17]; the research literature; and the diagnostic criteria for an eating disorder found in the *Diagnostic and Statistical Manual of Mental Disorders,* 3rd edition (*DSM-III*),[18] and the *Diagnostic and Statistical Manual of Mental Disorders,* 4th edition (*DSM-IV*).[19] Additional items were included pertaining to the effect of the athletic milieu on weight management and eating behaviors. Several demographic questions also were included. No attempt was made to disguise the purpose or intention of the objective test, and the response format was multiple choice. Extensive pilot testing of the AMDQ (described in the next column) was completed before this study to establish its psychometric properties (ie, readability, response bias, content validity, test-retest reliability, and criterion validity).

Pilot testing of the AMDQ was conducted in 4 stages over a 2-year period using another 175 female collegiate athletes not included in our study sample. In stage 1, we assessed the readability of the AMDQ (5.27 grade level) using the Gunning Fog Formula.[20] Subjects' written ratings and feedback about the clarity and wording of items and the adequacy of response options also were evaluated. In stage 2, we examined response bias by asking subjects whether or not each item would result in an honest answer or an answer that conformed to socially acceptable norms. The AMDQ was then revised based on stage 1 and 2 results. In stage 3, we assessed content validity by soliciting feedback from 3 experts in the areas of eating disorders and athletics using a structured evaluation form. Based on the stage 3 results, further revisions were made. Stage 4 involved evaluation of test-retest reliability and criterion validity. Test-retest reliability was determined by administering the tests to the same group of female athletes on 2 separate occasions, 2 to 4 weeks apart. Criterion validity was estimated by comparing 3 self-report items on the test (ie, height, weight, body fat percentage) with observed data measured by the researchers. The results for each stage of the pilot test clearly met psychometric standards for test construction, and detailed results can be obtained by contacting the authors.

A second session was scheduled to interview subjects to confirm written test results and to obtain physiologic data. We used Fairburn and Wilson's[21] interview questions and procedures (Eating Disorder Examination [EDE], Diagnostic Version, Edition 12.0D) to diagnose an eating disorder. The EDE is a psychometrically valid, systematic, structured interview. Interviewers (n = 2) were trained to conduct the EDE interviews by a licensed clinical psychologist who has extensive experience with eating disorders and athletes. Any athletes who met diagnostic criteria or were experiencing psychological or physical problems related to nutrition or weight management were referred to an appropriate health care provider (eg, registered dietitian, psychologist, physician, or a combination of these). Coaches were excluded from the recruitment and testing sessions to avoid response bias (ie, influencing athlete participation or responses). Many experts working with athletes with eating disorders[17,22,23] have recommended excluding coaches.

Physiologic Measurements. For purposes of subject classification with the EDE, we measured each subject's height, weight, and body fat percentage. Height was measured to the nearest 0.64 cm (0.25 in) and weight to the nearest 0.23 kg (0.5 lb) using a Detecto balance-beam scale (Detecto Scale Co, Webb City, MO). Body composition was calculated using Jackson and Pollock's formula[24] and measured using Harpenden (Burgess Hill, West Sussex, England) skinfold calipers. The body sites selected for assessment were the triceps, ilium, abdomen, and thigh. The same experienced laboratory technician took 3 skinfold measurements at each site. BMI was computed from height and weight measurements as another indicator of body composition.

Subject Classification. Subjects were classified according to *DSM-III-R*[18] and *DSM-IV*[19] criteria as having anorexia nervosa, bulimia nervosa, or an eating disorder not otherwise specified (NOS) based on responses to key EDE questions (Table 1). For anorexia nervosa, subjects had to meet 5 criteria. To satisfy criteria 1 and 2 for anorexia (fear of weight gain and feelings of fatness), they had to score 4, 5, or 6 on these EDE

Table 1. Subject Classification According to *DSM*[a]*-III-R*[13] and *DSM-IV*[14] Diagnostic Criteria for Eating Disorders

	Required Rating					
Criteria	Anorexia Nervosa	Bulimia Nervosa	NOS-Anorexic[b]	NOS-Bulimic[c]	Disordered Eating Major Criteria[d]	Disordered Eating Minor Criteria[e]
Fear of Weight Gain	4, 5, or 6[f] (for 3 mo)		3 (for 3 mo)			3 (for 3 mo)
Feelings of Fatness	4, 5, or 6 (for 3 mo)		3 (for 3 mo)			3 (for 3 mo)
Maintained Low Weight	1[g] (for 3 mo)		1 (for 3 mo)			2 (for 3 mo)
Menstruation (absence of)	0 or 7[h] (for 12 mo)		1–8 (for 12 mo)		9 (for 12 mo)	
Body Composition[i]						
1. Body Mass Index (BMI)	<18		18.0–19.9		18.0–19.9	
2. Body fat %	<14		14–17		14–17	
Purging Methods[j]						
1. Vomiting (self-induced)		>2×/wk (for 3 mo)		<1×/wk (for 3 mo)	<1×/wk (for 3 mo)	
2. Laxative misuse		>2×/wk (for 3 mo)		<1×/wk (for 3 mo)	<1×/wk (for 3 mo)	
3. Diuretic misuse		>2×/wk (for 3 mo)		<1×/wk (for 3 mo)	<1×/wk (for 3 mo)	
4. Intense exercise[k]		>3×/wk (for 3 mo)		1–2×/wk (for 3 mo)	1–2×/wk (for 3 mo)	
Objective Bulimic Episodes[l]		>2×/wk (for 3 mo)		<2×/wk (for 3 mo)	<2×/wk (for 3 mo)	
Subjective Bulimic Episodes[m]				<2×/wk (for 3 mo)	<2×/wk (for 3 mo)	
Importance of Shape/Weight		4, 5, or 6 (for 3 mo)		3 (for 3 mo)		3 (for 3 mo)

[a]*DSM, Diagnostic and Statistical Manual of Mental Disorders.*
[b]NOS-anorexic (not otherwise specified) subjects meet any 4 of the 5 criteria for anorexia but with lower scores.
[c]NOS-bulimic subjects meet all 3 criteria for bulimia but to a lesser degree or severity. Subjects classified as NOS-both meet NOS criteria for anorexia and bulimia.
[d]Disordered eating subjects meet 2 of the 4 major criteria: absence of menstruation (amenorrhea), body composition, purging methods, or bulimic episodes.
[e]Disordered eating subjects meet 2 of the 4 minor criteria.
[f]EDE scores can be between 0–6. Scores of 0–2 indicate more "normal" responses (for most items), a score of 3 indicate disordered eating, and scores of 4–6 indicate greater eating disorder symptomatology.
[g]1 = Attempts to lose weight or avoid weight gain because of weight or shape; 2 = attempts to lose weight or avoid weight gain for other reasons.
[h]0 = Absence of menses for 12 months. 7 = subject is taking birth control pills.
[i]Anorexic, NOS-anorexic, and disordered eating subjects satisfy 1 of the 2 criteria (BMI or body fat percentage) for body composition.
[j]Only 1 of the 4 purging methods listed must be used by the subject to meet classification criteria.
[k]Defined as ≥3×/wk for 30 minutes over and above scheduled team practices or competitions.
[l]Defined by the consumption of a large amount of food (eg, 1000 calories or more) in a very short time, with the subject experiencing a loss of control over eating. Bulimic subjects exhibit objective episodes. NOS-bulimic or disordered eating subjects experience either objective or subjective episodes and with less frequency.
[m]Defined as episodes in which the subject "feels" he or she ate too much but really ate a "normal" amount of food. Loss of control over eating is exhibited.

questions. EDE scores can be from 0 to 6; scores of 0–2 indicated more "normal" responses (for most items), while a score of 3 indicated disordered eating, and scores of 4 to 6 indicated greater eating disorder symptoms. For criterion 3, dieting behavior (ie, tried to "maintain low weight"), a score of 1 was required (meaning the subject was trying to lose or maintain weight because of shape or weight concerns). To satisfy criterion 4, amenorrhea, subjects had to score a 0 or 7, meaning they had no menstrual periods in 12 months or that they were on birth control pills (score of 7). To satisfy criterion 5, body composition, subjects had to be less than 14% body fat or have a BMI less than 18.

For bulimia nervosa, subjects had to meet 3 criteria (Table 1). To satisfy criterion 1, subjects had to report use of 1 of 4 purging methods (ie, self-induced vomiting, laxative misuse, diuretic misuse, or intense exercising that was defined as more than 3 times per week for 30 minutes over and above scheduled team practices or competitions). Purging methods had to be used at least twice per week for the past 3 months. To satisfy criterion 2, subjects had to report objective bulimic episodes twice per week for the past 3 months. According to Fairburn and Wilson,[21] objective bulimic episodes (commonly called "binging") are defined by the consumption of a large amount of food (eg, 1000 calories or more) in a very short time, with the subject experiencing a loss of control over eating. Subjective bulimic episodes are those in which subjects "feel" they ate too much, but in reality the amount of food was "normal," and loss of control also was exhibited. Finally, to satisfy criterion 3, subjects had to score 4 to 6 on "importance of shape or weight" (ie, how important any change in shape or weight would be in influencing how they felt about themselves as people).

NOS-anorexic subjects had to meet any 4 of the 5 anorexia criteria, but lower scores were required (Table 1). For criteria 1 and 2, subjects had to score 3 on questions dealing with fear of weight gain and feelings of fatness. For criterion 3, dieting behavior, a score of 1 was required. To satisfy criterion 4, amenorrhea, subjects had to score 1 through 8 (meaning they had at least 1 menstrual period, but no more than 8 during the last 12 months). To satisfy criterion 5, body composition, subjects had to have 14% to 17% body fat or a BMI of 18 to 19.9.

NOS-bulimic subjects had to meet all 3 criteria for bulimia but to a lesser degree of severity or frequency. To satisfy criterion 1 for NOS-bulimia, purging could occur less frequently than twice per week for the last 3 months. To satisfy criterion 2, objective or subjective bulimic episodes could occur less often than twice per week for the last 3 months. To satisfy criterion 3, subjects had to score 3 on the importance of shape or weight. Some subjects were classified as NOS-both, meaning they met the NOS criteria for both anorexia and bulimia.

The American College of Sports Medicine[1] has defined disordered eating as "a wide spectrum of harmful and often ineffective eating behaviors used in attempts to lose weight or achieve a lean appearance. The spectrum of behaviors ranges in severity from restricting food intake to binging and purging, to the *DSM-IV*–defined disorders of anorexia nervosa and bulimia nervosa." Criteria used in classification of subjects as disordered eating in this study were based on the *DSM-III-R*[18] and *DSM-IV*[19] diagnostic criteria for an eating disorder. Disordered eating subjects had to meet 2 of the 4 major criteria. First, subjects had to display signs of amenorrhea, meaning they had to score 9 of 12 on item 36 of the EDE (ie, they had to have had 9 menstrual periods in the last 12 months). Second, body composition scores had to show an abnormally low percentage of body fat (14% to 17%) or a BMI of 18.0 to 19.9. Third, subjects had to use purging methods (ie, self-induced vomiting, laxative or diuretic misuse) on a regular basis (ie, less than once per week for 3 months). Finally, bulimic episodes (subjective or objective) had to occur on a regular basis (ie, less than once per week for 3 months). Additionally, to be classified as disordered eating, subjects had to respond with 3 on at least 2 of the following minor criteria: importance of shape or weight, fear of weight gain, feelings of fatness, and maintaining a low weight.

Data Analyses

Item Identification. We statistically reduced a large item pool to increase the probability of identifying highly discriminant items.[25,26] The original item pool was composed of items from 3 tests: the AMDQ (119 items), EDI-2 (91 items), and BULIT-R (36 items). Because the EDI-2 uses only 3 of the 11 subscales (bulimia, drive for thinness, body dissatisfaction) for diagnoses of anorexia or bulimia, we only included items from these subscales (23 total) in the data analyses. Logistic regression, along with item analysis techniques, were used to determine the overall sensitivity and specificity of each screening test and the value of each individual item and to compare tests (and individual items from different tests) in terms of their ability to identify athletes with ED/DE. Logistic regression was selected because confounding variables can be controlled statistically and because it is appropriate for analyses of items on different scales of measurement (eg, 4- or 5-level Likert questions and responses on a continuous scale, such as body weight and age). Subjects were classified as ED/DE or OK (not ED/DE) based on EDE interview data. A nominal response variable based on the EDE (0 = no ED/DE, 1 = ED/DE) was used to compare results of the screening tests.

The tests or the best combination of items also were evaluated for their ability to identify each of the 4 types of ED/DE (anorexia, bulimia, NOS, disordered eating), resulting in a multinomial response. Analyses were undertaken using generalized logits to predict group membership. All test items were analyzed for internal consistency with the Cronbach coefficient α.

Item Selection. Items that were selected for analyses had to meet all of the following 5 statistical criteria. The first was mean separation: the mean score for ED/DE subjects on an item had to be significantly ($P < .01$) greater than the mean score for OK (not ED/DE) subjects. This value was used to reduce the number of items (although many other items had significant mean separation, with a P value between 1% and 5%). The second criterion was logistic regression: the item had to be a significant predictor of ED/DE versus OK subjects in a logistic regression model with α set at 1%. The third was correlation with total: the correlation of each retained item with the total score had to be at least 0.4. The fourth criterion was Cronbach α: each item's individual Cronbach α had to be at least 0.85. The fifth was sensitivity and specificity: the sensitivity and specificity had to be "satisfactory," meaning a sensitivity of 80% or greater and specificity of approximately 75 to 80% (criterion 5 above is essentially [1] and [2] of the epidemiologic analyses presented below).

Epidemiologic Analyses. We conducted epidemiologic analyses to evaluate the effectiveness of each test. These calculations included 9 interrelated analyses: (1) sensitivity, (2) specificity, (3) percentage of false-positives, (4) percentage of false-negatives, (5) positive predictive value, (6) negative predictive value, (7) yield, (8) accuracy, and (9) validity.[27–29] Sensitivity is the ability of the test to correctly classify those with the disorder (ie, ED/DE subjects). Specificity is the ability of the test to correctly classify those without the disorder (ie, OK subjects). False-positives are the percentage of subjects without the disorder who test positive. False-negatives are the percentage of subjects with the disorder who test negative. Positive predictive value is the probability that a person who tests positive does have the disorder. Negative predictive value is the probability that a person who tests negative does not have the disorder. Yield is the number of true positives correctly identified (ie, the proportion of true positives divided by the total number of subjects screened). Accuracy is the degree of agreement between the screening test and the gold standard (ie, the EDE) for identifying true-positives and true-negatives. Validity is the ability of a test to give a true measure: how well it measures what it is supposed to measure. Further indications of the validity of the 3 tests include sensitivity, specificity, positive predictive value, and negative predictive value.

RESULTS

Epidemiologic Analyses

The response rate was 85.5%. Results for each of the 9 epidemiologic values described above for the AMDQ, EDI-2, and BULIT-R and a combination of 26 items from all 3 tests called the AEBSC (AMDQ, EDI-2, and BULIT-R subsets combined) are presented in Table 2. Three different versions of the AMDQ and the BULIT-R also are reported in Table 2, for a total of 8 tests. The BULIT-R1 is the 28-item version using the scoring guidelines (cutoffs of 84 and 112) of Thelen et al.[11]

Table 2. Epidemiologic Evaluation (%) of Eating Disorder Assessment Tools

	AMDQ*				BULIT†			
	1	2	3	EDI-2‡	-R1†	-R2	-R3	AEBSC§
Sensitivity	80.00	80.00	82.00	64.00	26.92	69.23	69.23	70.59
Specificity	77.17	75.27	79.57	74.23	98.94	78.72	77.66	73.68
False-Positives	22.83	24.73	20.43	25.77	1.06	21.28	22.34	26.32
False-Negatives	20.00	20.00	18.00	36.00	73.08	30.77	30.77	29.41
Positive Predictive Value	65.57	63.49	68.33	56.14	93.33	64.29	63.16	59.02
Negative Predictive Value	87.65	87.50	89.16	80.00	70.99	82.22	82.02	82.35
Yield	28.17	27.97	28.67	21.77	9.59	24.66	24.66	24.66
Accuracy	78.17	76.92	80.42	70.75	73.29	75.34	74.66	72.60
Validity	57.17	55.27	61.57	38.23	25.86	47.95	46.89	44.27

*AMDQ, Athletic Milieu Direct Questionnaire. Three different versions of the AMDQ are presented. Each version of the AMDQ has a different number of items, with some items common to all 3 tests. The AMDQ 1 has 35 items; AMDQ 2 has 19 items; and AMDQ 3 has 9 items.
†BULIT-R, Bulimia Test-Revised. The BULIT-R1 is the 28-item version using the scoring guidelines (cutoffs of 84 and 112) of Thelen et al.[11] The BULIT-R2 is the 36-item version with a cutoff of 60, and the BULIT-R3 is the 28-item version with cutoff of 60. The cutoffs for the BULIT-R2 and R3 were chosen by a discriminant analysis to improve sensitivity and specificity.
‡EDI-2, Eating Disorder Inventory, 2nd edition.
§AEBSC = AMDQ, EDI-2, and BULIT-R subsets combined. The AEBSC has 26 items.

The BULIT-R2 is the 36-item version with a cutoff of 60, and the BULIT-R3 is the 28-item version with cutoff of 60. The cutoffs for the BULIT-R2 and R3 were chosen by a discriminant analysis to maximize sensitivity and specificity.

Sensitivity (criterion 5) was highest for the 3 AMDQ subsets (80% to 82%), lower for the EDI-2 (64%), and lowest for the BULIT-R1 (27%). Only the 3 AMDQ tests met criterion 5 for sensitivity (> 80%). In contrast, the AEBSC and BULIT-R2 and R3 have values that are moderately high but below the criterion (70%), while the EDI-2 and BULIT-R1 were far below the criterion (64% and 27%, respectively). More specifically, when AMDQ subset 2 was used, 80% (n = 40) of ED/DE subjects were correctly classified, and 10 ED/DE subjects were misclassified as OK (false-negatives) (Table 3). Of the 10 ED/DE subjects who were misclassified, 1 was bulimic, 1 was NOS, and 8 exhibited disordered eating. In contrast, when the BULIT-R1 was used, only 27% (n = 14) of ED/DE subjects were correctly classified, and 38 ED/DE subjects were misclassified as OK. Of the 38 misclassified subjects, 1 was anorexic, 7 were bulimic, 4 were NOS, and 26 exhibited disordered eating.

Specificity values (criterion 5) for all 8 tests were high (74% to 99%) and 5 tests met criterion 5 for specificity (approximately 75% to 80%). These tests included the 3 AMDQ tests, the BULIT-R2, and R3. The values for the EDI-2 and AEBSC (74%) were just under the criterion. The BULIT-R1 (99%) exceeded the criterion; however, it was accompanied by a high false-negative value (73%) and a low false-positive value (1%) (Table 1).

The false-positive value was highest for the EDI-2, AEBSC, and AMDQ subset 2 (25% to 26%) and lowest for the BULIT-R1 (1%). The other tests varied between 20% (AMDQ subset 3) and 23% (AMDQ subset 1). False-positive values for all 8 tests were acceptable; however, a false-positive value of 1% for the BULIT-R1 is not likely to be accurate and is probably due to the extremely high specificity (99%) of this test.

False-negative values were highest for the BULIT-R1 (73%), with moderately high values for the EDI-2 (36%), BULIT-R2 (31%), BULIT-R3 (31%), and AEBSC (29%). The lowest false-negative values were for the 3 AMDQ subsets (18% to 20%). Because of the seriousness of eating disorders,

Table 3. Sensitivity of Screening Tests by ED/DE Classification

	Frequency (%)					
	Anorexia Nervosa	Bulimia Nervosa	Not Otherwise Specified	Eating Disordered	Disordered Eating	Sensitivity
AMDQ 1	1 (100)	12 (92)	3 (75)	16 (89)	24* (75)	40 (80)
AMDQ 2	1 (100)	12 (92)	3 (75)	16 (89)	24* (75)	40 (80)
AMDQ 3	1 (100)	12 (92)	3 (75)	15 (83)	25* (78)	41 (82)
EDI-2	1 (100)	10 (77)	3 (75)	14 (78)	18* (56)	32 (64)
BULIT-R1	0 (0)	6 (46)	0 (0)	6 (33)	8 (23)	14 (27‡)
BULIT-R2	1 (100)	12 (92)	3 (75)	16 (89)	20 (59)	36 (69‡)
BULIT-R3	1 (100)	12 (92)	3 (75)	16 (89)	20 (59)	36 (69‡)
AEBSC	1 (100)	10 (77)	3 (75)	14 (78)	22† (67)	36 (71§)
Total	1	13	4	18	34	50

Note: AMDQ, Athletic Milieu Direct Questionnaire; EDI-2, Eating Disorder Inventory, 2nd edition; BULIT-R, Bulimia Test-Revised; AEBSC, AMDQ, EDI-2, and BULIT-R subsets combined resulted in 3 different versions of the AMDQ. Each version of the AMDQ has a different number of items, with some items common to all 3 tests. The AMDQ 1 has 35 items; AMDQ 2 has 19 items; and AMDQ 3 has 9 items. The BULIT-R1 is the 28-item version using Thelen et al (BULIT-R author) scoring guidelines (cutoffs of 84 and 112). The BULIT-R2 is the 36-item version with a cutoff of 60, and the BULIT-R3 is the 28-item version with cutoff of 60. The cutoffs for the BULIT-R2 and R3 were chosen by a discriminant analysis to improve sensitivity and specificity. The AEBSC is the AMDQ, EDI-2, and BULIT-R subsets combined (26 items total).
*Frequency missing = 2; †Frequency missing = 1; ‡Total number of items = 52; §Total number of items = 51.

a lower false-negative value is desirable in a screening test. The 3 AMDQ tests had the lowest false-negative values (18% to 20%). The BULIT-R3 and AEBSC values (31% and 29%, respectively) are questionable in terms of acceptability, and the EDI-2 value (36%) and BULIT-R1 value (73%) are unacceptable.

Positive predictive value was highest for the BULIT-R1 (93%) and lowest for the EDI-2 (56%). Positive predictive values for the other tests, including the AMDQ, were comparable (63% to 68%). Positive predictive values for all tests were acceptable, except for the EDI-2. However, the high positive predictive value of the BULIT-R1 is misleading and not necessarily desirable because this test also had the lowest negative predictive value (71%), which is of greater concern when screening for eating disorders.

Negative predictive value was highest for the 3 AMDQ subsets (88% to 89%) and lowest for the BULIT-R1 (71%). The EDI-2, BULIT-R2, BULIT-R3, and AEBSC tests all had similar values (80% to 82%). When screening for eating disorders, higher negative predictive values are desirable. Therefore, negative predictive values for the 3 AMDQ tests were preferable to the values for the other 5 tests.

Yield was highest for the 3 AMDQ subsets (28% to 29%) and lowest for the BULIT-R1 (10%). The EDI-2, BULIT-R2, BULIT-R3, and AEBSC had comparable values (22% to 25%). Yields for the 3 AMDQ tests were preferable to yields for the other 5 tests. The highest yield possible for ED/DE subjects (ie, the maximum ED/DE yield) is 34.90% (52/149; 52 ED/DE or true-positive subjects were identified by the EDE of 149 total subjects). Percentage of total maximum yield for the AMDQ tests was 80.14 to 82.15 (AMDQ yield/maximum ED/DE yield; 27.97/34.90 = 80.14; 28.67/34.90 = 82.15), meaning the AMDQ correctly identified approximately 4 of every 5 persons who were truly ED/DE.

Accuracy in identification of true-positives and true-negatives was highest for the 3 AMDQ subsets (77% to 80%) and lowest for the EDI-2, BULIT-R1, and AEBSC (71% to 73%). Accuracy for the 3 AMDQ tests was preferable to that of the other 5 tests.

Validity was highest for the 3 AMDQ subsets (55% to 62%) and lowest for the BULIT-R1 (26%) and EDI-2 (38%). The AEBSC, BULIT-R2, and BULIT-R3 had comparable values (44% to 48%). Validity for the 3 AMDQ tests was preferable to that of the other 5 tests.

AMDQ Subset Analyses

Initial Analyses. A total of 51 items were significant (P = .01) on both the mean separation t tests and the logistic regression analyses (criteria 1 and 2). Another 23 items were significant (P = .05) on both mean separation and logistic regression. These 23 items were not considered in the latter 3 analyses.

Using the 51 AMDQ items that were significant for criteria 1 and 2, several equally good subsets of AMDQ items potentially meet all 5 criteria. Two examples of such subsets and the corresponding analyses associated with each subset follow. Also included is a third subset, AMDQ subset 3, which did exceptionally well on criterion 5 but does not meet criteria 3 and 4, largely due to its small item set (only 9 items).

AMDQ Subset 1 (35 Items). Correlations with total and Cronbach α (criteria 3 and 4) were both used to demonstrate internal consistency of test items. Most of these items (32/35) met criterion 3, having a correlation with the total of 0.40 or above; 3 items were slightly below. Cronbach α values were all above 0.85 (criterion 4), and the average Cronbach α for raw and standardized variables was 0.9401 and 0.9441, respectively. Raw variables represent actual values of subjects' responses on items. Standardized variables represent values adjusted to a uniform scale of measurement. Based on criteria 3 and 4, 32 of 35 items in AMDQ subset 1 met the criteria for inclusion in the item pool.

AMDQ Subset 2 (19 Items). Most of these items (18/19) met criterion 3, having a correlation with the total of 0.40 or above; 1 item was slightly below. Cronbach α values were all above 0.85 (criterion 4), and the average Cronbach α for raw and standardized variables was 0.9043 and 0.9161, respectively. Based on criteria 3 and 4, 18 of 19 items in AMDQ subset 2 met the criteria for inclusion in the item pool.

AMDQ Item Subset 3 (9 Items). One third of these items did not meet criterion 3, having a correlation with the total of 0.40 or above. None of the Cronbach α values met criterion 4 of 0.85 or above. The average Cronbach α for raw and standardized variables was 0.7587 and 0.7706, respectively. Based on criteria 3 and 4, none of the items in AMDQ subset 3 met the criteria for inclusion in the item pool. This subset, however, had the highest sensitivity and specificity (82% and 79.6%, respectively) of any subset. Therefore, fewer subjects were misclassified with this subset. Cronbach α and item correlations with the total, however, were low. The high sensitivity and specificity of this small item set may reflect an artificially high performance of these items for the current subjects that is unlikely to generalize to other athlete populations because of the lower than acceptable correlations with the total and Cronbach α. These items would be better used as part of a larger item pool, such as the AMDQ subset 1 or 2.

EDI-2 Analyses

Initial Analyses. A total of 15 of 23 diagnostic subscale items were significant (P = .01) on both mean separation and logistic regression (criteria 1 and 2). The remaining 8 items were dropped from the item pool, although they were included in the analyses for criteria 3 through 5. Items not meeting criteria 1 and 2 were predominantly from the bulimia subscale (6/7), although 2 items were from the drive-for-thinness subscale. In other words, only 1 of 7 items from the bulimia subscale met criteria 1 and 2 for inclusion in the item pool.

The results for criteria 3 and 4 for the EDI-2 diagnostic subscales are presented below. The results for all 3 subscales are combined and then presented separately.

Three Diagnostic Subscales Combined (23 Items). When all diagnostic subscale items were analyzed as a group, 21 of the 23 items met criterion 3, having a correlation with the total of 0.40 or above (using standardized variables); 1 on the drive-for-thinness subscale and 1 on the bulimia subscale did not meet the criterion. Cronbach α values were all above 0.85 (criterion 4), and the average Cronbach α for raw and standardized variables was 0.9282 and 0.9246, respectively. When all 3 diagnostic subscales were combined for analysis, 21 of 23 EDI-2 items met criteria 3 and 4 for inclusion in the item pool.

Bulimia Subscale (7 Items). All of these items met criterion 3, having a correlation with the total of 0.40 or above. Cronbach α values were all below 0.85 (criterion 4) using raw variables, and only 2 items met the criterion using standardized variables. The average Cronbach α for raw and standardized variables was

0.8252 and 0.8483, respectively. Based on criteria 3 and 4, only 1 of 7 items from the bulimia subscale of the EDI-2 met the criteria for inclusion in the item pool.

Body Dissatisfaction Subscale (9 Items). All of these items met criterion 3, having a correlation with the total of 0.40 or above, and all items had Cronbach α values above 0.85 (criterion 4). The average Cronbach α for raw and standardized variables was 0.9253 and 0.9258, respectively. Based on criteria 3 and 4, all 9 items on the body dissatisfaction diagnostic subscale of the EDI-2 met the criteria for inclusion in the item pool.

Drive-for-Thinness Subscale (7 Items). Most of these items (6/7) met criterion 3, having a correlation with the total of 0.40. The majority of Cronbach α values (5/7) were all above 0.85 (criterion 4). The average Cronbach α for raw and standardized variables was 0.8804 and 0.8767, respectively. Based on criteria 3 and 4, 5 of 7 items on the drive-for-thinness diagnostic subscale of the EDI-2 met the criteria for inclusion in the item pool.

BULIT-R

Initial Analyses. A total of 29 of 36 items were significant ($P = .01$) on both mean separation and logistic regression (criteria 1 and 2). The remaining 7 items were dropped from the item pool, although they were included in the analyses for criteria 3 through 5.

As Table 1 indicates, the primary difference between the 3 versions of the BULIT-R tests is the scoring method. Therefore, we analyzed all 36 BULIT-R items as a group for comparison with criteria 3 and 4.

BULIT-R (36 Items). Five of the 36 items did not meet criterion 3, having a correlation with the total of 0.40 or above, and were dropped from the item pool. Cronbach α values were all above 0.85 (criterion 4), and the average Cronbach α for raw and standardized variables was 0.9514 and 0.9501, respectively. Based on criteria 3 and 4, 31 of 36 BULIT-R items met the criteria for inclusion in the item pool.

AEBSC Subset (26 Items)

This subset included a total of 26 items: 9 from the AMDQ, 9 from the EDI-2, and 8 from the BULIT-R. All of these items were significant ($P = .01$) on both criteria 1 and 2 (mean separation and logistic regression). Additionally, all items met criterion 3, having a correlation with the total of 0.40 or above and all Cronbach α values were above 0.85 (criterion 4). The average Cronbach α for raw and standardized variables was 0.9548 and 0.9580, respectively. Based on criteria 3 and 4, all of the items in the AEBSC subset met the criteria for inclusion in the item pool.

DISCUSSION

The purpose of our study was to develop a screening test specifically for female college athletes with ED/DE. Six major conclusions can be drawn from the results of this study. First, the results supported the hypothesis that the AMDQ more accurately screened eating disorders and disordered eating among female college athletes than the EDI-2 or BULIT-R and established that the AMDQ subsets were more discriminating than any combination of items from all 3 tests. This finding confirms that a screening test such as the AMDQ, which is specifically intended for the athletic population, is required. The AMDQ subsets produced superior results for sensitivity (81%), false-negatives (19%) positive predictive value (66%), negative predictive value (88%), yield (28%), accuracy (79%), and validity (58%) than commercial tests not normed for athletes (Table 2), while maintaining acceptable values for specificity (77%) and false-positives (23%).

Second, the AMDQ subsets were the only tests that met criterion 5 for sensitivity. The AMDQ subsets correctly classified 80% to 82% of ED/DE subjects (sensitivity) and 75 to 80% of OK subjects (specificity) (Tables 2 and 3). In contrast, the 2 valid commercial eating disorder tests correctly classified only 64% and 70% of ED/DE subjects and 74% and 78% of OK subjects (EDI-2[9] and BULIT-R,[11] respectively), while the combined test (AEBSC) correctly classified 71% and 74%, respectively. It should be noted, however, that the BULIT-R[10,11] was designed to screen only for bulimia, and the EDI-2 and BULIT-R were not designed to screen for NOS or disordered eating. The use of the AMDQ with a population of 1000 ED/DE female college athletes who are truly positive for an ED/DE would theoretically result in correct classification of 4 of every 5 (80%, n = 800) athletes, which is superior to the sensitivity achieved using the EDI-2 (64%, n = 640) or BULIT-R (69%, n = 690). Additionally, if a second screening were conducted using different versions of the AMDQ, the number of subjects correctly classified as ED/DE would improve (ie, sensitivity and specificity would increase).

Third, a large percentage of ED/DE subjects were identified in this study, which suggests that ED/DE are serious problems for female college athletes. A total of 35% of the sample was at risk (disordered eating) or had a definite problem (eating disordered) as determined by the EDE diagnostic interview. A higher prevalence of bulimia (25% of the 52 ED/DE subjects, n = 13, and 8.72% of the total sample) than anorexia (2% of ED/DE subjects, n = 1) was noted. Also, a large number of subjects met the criteria for disordered eating (n = 34, 65.38% of ED/DE subjects and 22.82% of the total sample) and 4 (8% of ED/DE subjects) met NOS criteria. These findings are consistent with prior prevalence studies of college students,[30–32] but our ED/DE percentage (35%) was higher than that found in 1 study of college athletes,[33] which focused only on anorexia and bulimia and noted an eating disorder prevalence of 25% among 695 college athletes. Many of the athletes who participated in our study appear to be at risk for future development of an eating disorder, and the eating behaviors and attitudes that were most prevalent were consistent with bulimia rather than anorexia. This is an important consideration for all support personnel closely affiliated with athletes because bulimia tends to be more difficult to detect, especially for someone not trained in the recognition and treatment of eating disorders. This study also provides the first operational definition of disordered eating, which is important in order to advance the field conceptually and empirically.

Fourth, it is noteworthy that all 3 AMDQ subsets met all 5 inclusion criteria. In contrast, the 2 validated published eating disorder tests each had several items that fell short on 1 or more inclusion criteria. For example, of the 3 diagnostic subscales of the EDI-2 (23 items), 8 items did not meet all 5 inclusion criteria (2 from the drive-for-thinness subscale, 6 from the bulimia subscale). Similarly, of 36 BULIT-R items, 7 did not meet all 5 inclusion criteria. All AEBSC items met criteria 1 through 4 but fell slightly short on criterion 5 (sensitivity and specificity).

Fifth, although we reported results for only 3 AMDQ subsets, many additional possible combinations of items could potentially produce similar results. In addition to the 51 AMDQ items that met all 5 statistical criteria, another 23 would be potentially acceptable for retention in the item pool after rewording the questions or changing the response options, or both. An additional advantage to the AMDQ over the current tests is its use in screening not only for anorexia and bulimia but also screening for NOS and disordered eating (which current tests do not consider). Further analysis of EDI-2 and BULIT-R items that did not discriminate well (ie, did not meet all 5 statistical criteria) may be beneficial if a common theme can be determined to explain why they did not perform well as discriminators and if they can be modified after psychometric testing.

Finally, the research literature suggests that the prevalence of ED/DE is highest in sports emphasizing low body weight and leanness.[1] This study also supports this conclusion, but ED/DE subjects were observed in every sport sampled except basketball and softball. Of the 13 bulimic athletes, 3 were involved in cheerleading, 3 in a dance company, 1 in modern dance, 1 in golf, 4 in swimming, and 1 in track. The anorexic athlete was in the dance company, and the 4 NOS subjects were involved (1 each) in cheerleading, gymnastics, swimming, and track. Disordered eating subjects were found in every sport except basketball and softball.

Three potential limitations existed in the study. First, the internal validity of the study might be questioned if subjects were not truthful in their responses to test or interview questions. The AMDQ underwent substantial pilot testing before we conducted our study to evaluate whether each item would produce a response bias. Response bias was an issue for only 9 of the 119 AMDQ items; all 9 items were revised based on the athletes' comments. Additionally, measures were taken to encourage truthful responding. Coaches were excluded from the study, and all subjects were guaranteed confidentiality and were assured that the data would be shared with no one (specifically coaches, athletic trainers, teammates, or parents), except for the research team. Observationally, subjects were open and candid in responses to interview questions, and many provided valuable, unsolicited comments regarding concerns about their eating habits and specific behaviors. Second, content validity might be questioned if the AMDQ did not adequately reflect the categories of ED/DE. AMDQ items, however, were developed based on *DSM-III-R*[18] and *DSM-IV*[19] diagnostic criteria for eating disorders and the research literature regarding the athletic milieu.[14–17,22,23,33–38] Additionally, 3 experts specializing in the area of eating disorders and athletes evaluated content validity of the AMDQ during the pilot test, and content validity was within psychometric standards. Third, results of this study may not be generalizable to all other female college athletes because we included female college athletes from only 1 major university. A national study using female college athletes from many representative universities in combination with the EDE interview is recommended to further verify use of the AMDQ subsets with other universities nationwide.

Future research with the AMDQ subsets that met statistical criteria in this study is recommended for 2 primary reasons. First, because this study and others[12,13] have shown that published eating-disorder tests (ie, EDI-2, BULIT-R, EAT) are not accurate or suitable for comprehensive screening of athletes, the magnitude of the problem of ED/DE in female college athletes nationwide needs to be verified. Prior prevalence studies indicate that athletes are 2 to 3 times more likely than nonathletes (eg, general population and college students) to manifest characteristics of eating disorders.[2] These data also suggest that approximately 3.0% of athletes meet the medical criteria for anorexia and 21.5% meet the criteria for bulimia.[2] Based on a figure of 352 000 athletes in American colleges and universities,[39,40] this translates to an estimated projection of approximately 10 560 athletes nationally who exhibit symptoms of anorexia and 75 680 who exhibit symptoms of bulimia. These estimates are conservative because a less stringent category, NOS, was not considered, nor was disordered eating.

Second, early detection of eating problems in athletes using an athlete-specific test, such as the AMDQ, is a high priority because of medical problems that accompany eating disorders. A recent survey (W. Wooten, unpublished communication, 1990) indicated that eating disorders are the third leading cause of morbidity among college students, preceded by depression and substance abuse. Potential adverse medical complications from eating disorders include amenorrhea, bradycardia, dental erosion, dehydration, electrolyte abnormalities, hypotension, hypothermia, and swollen salivary glands.[7] Another significant problem is unrecoverable bone loss (osteoporosis) associated with being underweight and the cessation of menses.[41–44] Anorexic athletes have the bone density of senescent women 3 to 4 times older, and there are no current methods to restore bone loss.[41–44] Other potential problems of special concern to athletes include diminished muscle power and endurance due to reduced protein synthesis and inadequate glycogen and fluid stores. Dehydration, Benardot et al[45] contended, also is commonly found in anorexic and bulimic individuals due to restricted food intake or the use of self-induced vomiting, laxatives, or diuretics, or a combination of these practices. Attempting to train in a chronically dehydrated state will not only decrease performance but may lead to acute complications, such as heat exhaustion or heat stroke. The personal effects of these medical signs and symptoms of eating disorders may be underestimated by athletes.

Unsolicited comments from our subjects indicated that athletes felt pressure from coaches to lose weight, often with little or no guidance regarding how to do so (or how to lose weight in a healthy, safe way). Interviews suggested that subjects also were misinformed about such topics as weight management, the role of food and body weight in sport performance, and basic nutrition. The athletes were eager to learn more about nutrition and, as such, are an excellent target population for nutrition education and for nutrition studies focusing on performance, total caloric intake, and energy expenditure. Athletes in 1 study[45] believed that diet makes little difference, disordered-eating practices are harmless, and losing weight, regardless of method, enhances performance. Athletes need education about the impact of inadequate caloric intake and disordered-eating practices on athletic performance and health (eg, problems resulting from depletion of muscle glycogen, dehydration, loss of muscle mass, hypoglycemia, electrolyte disturbances, anemia, osteoporosis, and amenorrhea).[7,41–45]

Seven specific recommendations for athletic trainers from the research literature[14–17,46,47] include the following. First, objective goals should be set with the athlete to determine an optimal range for individual body fat, as opposed to setting weight goals based on appearance, standards, or tables that do not account for individual differences (eg, height/weight tables), or body weight, which does not account for muscle mass. Second, the use of rapid weight-loss methods should be discouraged, and weight-loss programs, when indicated, should be initiated well before the season begins, so that gradual loss of body fat (as opposed to the loss of muscle mass and body water that occurs with crash diets) can occur. Third, nutritional guidance should be provided. Athletic trainers and coaches should not tell athletes to lose weight without also providing sound nutritional guidance on how to do so. Referral of athletes to a registered dietitian for such guidance is recommended. Fourth, dietary intervention should focus on providing adequate calories to support basic needs and the demands of the sport. Nutrient-dense foods, such as complex carbohydrates, should provide most of the calories (55% to 70%), and protein sources should be high in biologic value (ie, animal as opposed to vegetable protein). Athletes in general, but especially those trying to reduce weight, must be encouraged to regularly replace fluids (2 cups of fluid for every 0.45 kg [1 lb] lost during exercise). Female athletes need to be educated about healthy food choices that provide adequate intake of iron and calcium. Fifth, weigh-ins and measurement of body composition should be private to reduce the stress, anxiety, and embarrassment of public assessment. Athletic trainers, coaches, and parents must be aware that their comments and opinions regarding body weight can strongly influence, even trigger, the development of an eating problem in some athletes. Sixth, athletic trainers need to be familiar with ED/DE symptoms and should talk to any athlete who exhibits a problem. Athletes should not be punished or dismissed from the team because of eating problems or the existence of an eating disorder, but professional counseling should be recommended. Finally, the AMDQ subsets (available from the authors) that met statistical criteria in this study could be used to screen for ED/DE, so that the emphasis is on prevention. By identifying the potential problems at earlier, less severe stages, (ie, NOS or disordered eating), we may be able to prevent the progression to an eating disorder (anorexia or bulimia). In fact, the AMDQ could be used as part of a total process to identify potential athletes with ED/DE. The initial step, of course, would be to administer the AMDQ. The next step would be either to retest (with the same test) those athletes who were positive for an ED/DE or to use an alternate form of the screening test for evaluation. The last step would be to have a trained professional verify those who tested positive. Athletic trainers are a pivotal and integral part of the screening, referral, and rehabilitation process.

In summary, an important step has been taken to develop a screening test for at-risk female athletes at the collegiate level. Clearly, a test is needed because ED/DEs are pandemic worldwide. Early detection (eg, identification of athletes at the disordered eating or NOS stage) is a salient priority so that appropriate prevention initiatives can be introduced, athletes can be returned to healthy competition, and ED/DE prevalence can be reduced.

ACKNOWLEDGMENTS

We thank the National Athletic Trainers' Association for support and endorsement of this project. This manuscript is the result of research funded by 2 National Athletic Trainers' Association Research and Education Foundation research grants (project number 0395-B007) awarded to David R. Black and Larry J. Leverenz for the periods July 1, 1994 through June 30, 1995 and July 1, 1995 through June 30, 1996. Both grants were for a study titled "Multicenter study to evaluate a screening test for athletes with disordered eating." Deborah (Chalmers) Nagel, PhD, RD, EdS, was one of the research assistants for both grants. We thank Brenda Stevens, School of Pharmacy; the late Dr. Robert K. Chalmers, School of Pharmacy; Dr. Stephen Badylak, Head Team Physician; and Denny Miller, Head Athletic Trainer, all from Purdue University, for their help and support of this project. We also thank Laurie Larkin, James Larkin, and Lisa (Lauer) Chrysler for their help with data collection and subject recruitment.

REFERENCES

1. Otis CL, Drinkwater B, Johnson M, Loucks A, Wilmore J. American College of Sports Medicine position stand: the female athlete triad. *Med Sci Sports Exerc.* 1997;29:i-ix.
2. Burckes-Miller ME, Black DR. Male and female college athletes: prevalence of anorexia nervosa and bulimia nervosa. *Athl Train, J Natl Athl Train Assoc.* 1988;23:137–140.
3. Black DR, Burckes-Miller ME. Male and female college athletes: use of anorexia nervosa and bulimia nervosa weight loss methods. *Res Q Exerc Sport.* 1988;59:252–256.
4. Burckes-Miller ME, Black DR. Behaviors and attitudes associated with eating disorders: perceptions of college athletes about food and weight. *Health Educ Res Theory Pract.* 1988;3:203–208.
5. Rosen LW, McKeag DB, Hough DO, Curley V. Pathogenic weight control behavior in female athletes. *Physician Sportsmed.* 1986;14(1):79–86.
6. Rosen LW, Hough DO. Pathogenic weight-control behaviors of female college gymnasts. *Physician Sportsmed.* 1988;16(9):141–144.
7. Comerci GD. Medical complications of anorexia nervosa and bulimia nervosa. *Med Clin North Am.* 1990;74:1293–1310.
8. Garner DM [catalog]. Lutz, FL: Psychological Assessment Resources, Inc; 1994.
9. Keyser DJ, Sweetland RC, eds. *Test Critiques.* Vol. VI. Kansas City, MO: Wesport Publishers; 1987;177–181.
10. Brelsford TN, Hummel RM, Barrios BA. The Bulimia Test-Revised: a psychometric investigation. *Psychol Assess.* 1992;4:399–401.
11. Thelen MH, Farmer J, Wonderlich S, Smith M. A revision of the Bulimia Test: the BULIT-R. *Psychol Assess.* 1991;3:119–124.
12. Wilmore JH. Eating and weight disorders in the female athlete. *Int J Sport Nutr.* 1991;1:104–117.
13. O'Connor PJ, Lewis RD, Kirchner EM. Eating disorder symptoms in female college gymnasts. *Med Sci Sports Exerc.* 1995;27:550–555.
14. Black DR, ed. *Eating Disorders Among Athletes: Theory, Issues, and Research.* Reston, VA: American Alliance for Health, Physical Education, Recreation and Dance; 1991.
15. Brownell KD, Rodin J, Wilmore JH, eds. *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society.* Philadelphia, PA: Lea & Febiger; 1992.
16. Holliman SC, ed. *Eating Disorders and Athletes: A Handbook for Coaches.* Dubuque, IA: Kendall/Hunt; 1991.
17. Thompson RA, Sherman RT. *Helping Athletes with Eating Disorders.* Bloomington, IN: Human Kinetics; 1993.
18. *Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed. Washington, DC: American Psychiatric Association; 1987.
19. *Diagnostic and Statistical Manual of Mental Disorders.* 4th ed. Washington, DC: American Psychiatric Association; 1994.
20. Gunning R. *The Technique of Clear Writing.* New York, NY: McGraw-Hill; 1968.
21. Fairburn CG, Wilson GT, eds. *Binge Eating: Nature, Assessment and Treatment.* New York, NY: Guilford Press; 1993.
22. Sundgot-Borgen J. Risk and trigger factors for the development of eating disorders in female elite athletes. *Med Sci Sports Exerc.* 1994;26:414–419.
23. Sundgot-Borgen J. Eating disorders in female athletes. *Sports Med.* 1994;17:176–188.
24. Jackson AS, Pollock ML. Steps toward the development of generalized

equations for predicting body composition of adults. *Can J Appl Sport Sci.* 1982;7:189–196.
25. Cronbach LJ. *Essentials of Psychological Testing.* 3rd ed. New York, NY: Harper and Row; 1970.
26. Nunnally JC. *Psychometric Theory.* 2nd ed. New York, NY: McGraw-Hill; 1978.
27. Browner WS, Newman TB, Cummings SR. In: Hulley SB, Cummings SR, eds. *Designing Clinical Research: An Epidemiological Approach.* Baltimore, MD: Williams & Wilkins; 1988:87–97.
28. Friis RH, Sellers TA. *Epidemiology for Public Health Practice.* Gaithersburg, MD: Aspen; 1996.
29. Last J, ed. *A Dictionary of Epidemiology.* 2nd ed. New York, NY: Oxford University Press; 1988.
30. Halmi KA, Falk JR, Schwartz E. Binge-eating and vomiting: a survey of a college population. *Psychol Med.* 1981;11:697–706.
31. Katzman M, Wolchik S, Braver S. The prevalence of frequent binge eating and bulimia in a non-clinical sample. *Intl J Eat Disord.* 1984;3:53–61.
32. Pyle RL, Mitchell JE, Eckert ED, Halvorson PA, Neuman PA, Goff GM. The incidence of bulimia in freshman college students. *Intl J Eat Disord.* 1985;2:73–84.
33. Conoley JC, Kramer JJ, eds. *The Tenth Mental Measurements Yearbook.* Lincoln, NE: University of Nebraska Press; 1989;274–276.
34. Fairburn CG, Beglin SJ. Studies of the epidemiology of bulimia nervosa. *Am J Psychiatry.* 1990;147:401–408.
35. Lakin JA, Steen SN, Oppliger RA. Eating behaviors, weight loss methods, and nutrition practices among high school wrestlers. *J Community Health Nurs.* 1990;7:223–234.
36. Lindeman AK. Self-esteem: its application to eating disorders and athletes. *Int J Sport Nutr.* 1994;4:237–252.
37. Sundgot-Borgen J. Eating disorders, energy intake, training volume, and menstrual function in high-level modern rhythmic gymnasts. *Int J Sport Nutr.* 1996;6:100–109.
38. Slavin JL. Eating disorders in athletes. *J Phys Educ Recr Dance.* 1987;58:33–36.
39. US Department of Commerce. *Statistical Abstract of the United States.* 110th ed. Washington, DC: US Government Printing Office; 1990.
40. US House Representatives Committee on Education and Liaison Subcommittee on Post Secondary Education. *Hearing on the Role of Athletics and College Life.* Washington, DC: US Government Printing Office; May 18 and 24, 1989.
41. Drinkwater BL, Nilson K, Chesnut CH 3rd, Bremner WJ, Shainholtz S, Southworth MB. Bone mineral content of amenorrheic and eumenorrheic athletes. *N Engl J Med.* 1984;277–281.
42. Johnson CL. Managing problems of weight: anorexia nervosa/bulimia. Presented at: 65th Annual American Dietetic Association Convention; October 1982; San Antonio, TX.
43. Nattiv A, Agostini R, Drinkwater B, Yeager KK. The female athlete triad: the inter-relatedness of disordered eating, amenorrhea, and osteoporosis. *Clin Sports Med.* 1994;13:405–418.
44. Yeager KK, Agostini R, Nattiv A, Drinkwater B. The female athlete triad: disordered eating, amenorrhea, osteoporosis. *Med Sci Sports Exerc.* 1993;775–777.
45. Benardot D, Engelbert-Fenton K, Freeman K, Hartsough C, Nelson-Steen S. Eating disorders in athletes: the dietitian's perspective. *Sports Sci Exch.* 1994;5:1–4.
46. Berning JR, Steen SN. *Sports Nutrition for the 90s: The Health Professional's Handbook.* Gaithersburg, MD: Aspen; 1991.
48. Clark N. *Sports Nutrition Guidebook: Eating to Fuel Your Active Lifestyle.* Champaign, IL: Leisure Press; 1990.

Journal of Athletic Training 2000;35(4):441–444

www.journalofathletictraining.org

Consistency of Learning Styles of Undergraduate Athletic Training Students in the Traditional Classroom versus the Clinical Setting

Cheryl A. Coker, PhD

New Mexico State University, Las Cruces, NM

Objective: To examine the learning styles of undergraduate athletic training students to determine their consistency in traditional classroom versus clinical settings.

Design and Setting: Subjects completed the Learning Styles Inventory twice, once focusing on learning new information in the classroom and the other focusing on learning new information in the clinical setting. The order of focus regarding setting (classroom or clinical) was counterbalanced across subjects.

Subjects: A total of 26 undergraduate athletic training students from a Committee on the Accreditation of Allied Health Education Programs accredited athletic training education program (16 women and 10 men; mean age, 24.42 ± 6.44 years) who were currently assigned to a clinical practicum as part of their academic program served as subjects.

Measurements: I performed 4 paired *t* tests, 1 for each learning mode, to determine if differences existed between the classroom and clinical settings. The percentage of respondents whose learning styles changed across settings was also calculated.

Results: The paired *t* tests revealed a significant difference between the Reflective Observation and Active Experimentation modes across settings. In addition, 58% of respondents' learning styles changed according to setting focus.

Conclusions: It appears that learning styles do indeed shift, depending on the domain through which an individual is learning. Consequently, teaching strategies incorporated in 1 setting may not be equally effective in the other setting. Each learning setting should, therefore, be treated separately in order to accommodate individual learning styles and maximize learning achievement. Furthermore, if learning styles are to be considered when designing athletic training education, these findings indicate that in order to ensure the validity of the resulting learning style profile, it may be necessary to provide the respondent with a specific focus, either that of a classroom or clinical setting, before completing the Learning Styles Inventory.

Key Words: learning style profile, clinical education, athletic training education, teaching strategies, Learning Styles Inventory administration

Incorporating teaching strategies that accommodate individual learning styles has consistently resulted in significant improvement in student achievement in the traditional classroom setting.[1–3] To date, however, the concept of individual learning styles has received little attention in athletic training education.[4,5] Given the positive results found when teaching styles correspond to individual learning styles, this same strategy may be effective in athletic training education. However, before these ideas are incorporated into the athletic training curriculum, it is necessary to examine the uniqueness of the learning environment in athletic training education.

Athletic training education combines traditional classroom learning with clinical experiences. The clinical or experiential environment and the traditional classroom or didactic setting, however, are very distinct.[6] In the classroom setting, criterion tasks can only be simulated at best, while the clinical setting is designed to provide real-life experiences. Consequently, the preferred way in which students process information may be quite different in these 2 settings (classroom versus clinical). As a result, determining the consistency of an individual's learning style across these 2 distinct learning environments may be important.

Address correspondence to Cheryl A. Coker, PhD, Department of PERD, Box 30001, New Mexico State University, Las Cruces, NM 88003. E-mail address: ccoker@nmsu.edu

Previously, learning styles were thought to remain consistent. In fact, Copenhaver[7] examined the consistency of learning style for high school students in English and mathematics. The results showed that students tended to prefer the same learning approach. In addition, after analyzing a number of definitions of learning styles, Satterly and Brimer[8] concurred in their statement, "all suggest that people behave in a typical way across a variety of tasks." Gieger and Pinto's[9] results from a 3-year longitudinal study that examined learning style consistency across time, however, offered mixed results. Kolb's[10] Learning Styles Inventory (LSI) was administered once per year for 3 years to 40 undergraduate business students. No significant changes in learning mode were noted over the 3-year period. Learning style classification, however, did change significantly. Interestingly, results for the first and second test administration indicated high stability, suggesting that subjects exhibited changes in their learning styles during the second year of the study. The authors suggested that although there was a statistically significant difference in learning style, from a practical standpoint the actual change may be marginal because no significant difference was found for learning modes. Smith[11] proposed that the consistency of learning preferences may depend on "the nature of the subject matter being studied." This was found to be the case when the preferred learning modes of elite throwers (ages 19 to 35 years) competing at the 1993 Canadian Track and Field Team trials were compared across cognitive and motor settings.[12] It was hypothesized that an individual's preferred perceptual mode may be different when learning a motor skill versus a more cognitive task. A significant difference in learning style was found when learning a cognitive task (traditional classroom) versus a motor task (learning a motor skill) using Kolb's LSI,[13] suggesting that one's preferred learning mode may indeed shift according to the learning environment.

Given the extensive experiential nature of the clinical setting in athletic training, a shift in an individual's preferred learning mode and subsequent learning style may also occur when compared with the traditional classroom setting. As a result, the purpose of this study was to examine the learning modes and subsequent learning styles of undergraduate athletic training students to determine their consistency across traditional classroom versus clinical settings.

METHODS

Subjects

Sixteen female and 10 male undergraduate athletic training students (mean age, 24.42 ± 6.44 years) who were currently assigned to a clinical practicum as part of their academic program served as subjects. The level of exposure to clinical instruction at the time of test administration ranged from approximately 90 to 940 hours (mean, 435.2 hours). All subjects signed informed consent forms, and the university's human subjects committee approved the experimental procedures.

Instrument

The LSI was designed to identify 4 learning types according to the perceptual and processing preferences of adults.[13] It is a self-reported inventory composed of 12 simple-sentence completion items that require respondents to rank order 4 sentence endings that correspond with 4 learning modes: Reflective Observation (RO, watching), Concrete Experience (CE, feeling), Abstract Conceptualization (AC, thinking), and Active Experimentation (AE, doing). These learning modes make up a 4-stage cycle of learning that is initiated with concrete experiences, which then form the basis for observation and reflection. The observations are then assimilated into concepts that serve as guides in creating new experiences. Kolb's[13] experiential learning theory suggests that the successful learner will use all 4 modes of learning (Figure 1). Therefore, scores indicate how much respondents rely on each learning mode and often reveal a modal strength.

In addition to measuring an individual's preference for the 4 learning modes, the extent to which the respondent prefers abstractness over concreteness and action over reflection is determined through 2 combination scores, calculated by subtracting CE from AC and RO from AE, respectively. These combined scores describe which learning style type (converger, diverger, assimilator, or accommodator) best describes the respondent (Table 1).

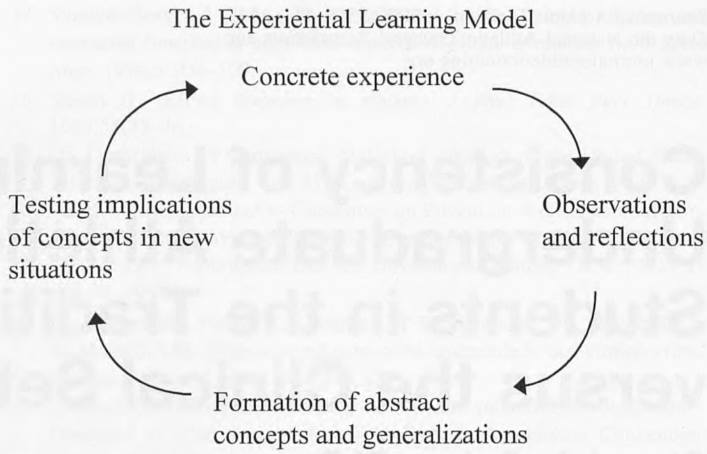


Figure 1. Experiential learning cycle.[10]

Table 1. Characteristics of Kolb's[13] 4 Learning Style Types

Learning Style	Characteristics
Converger	Good at finding practical uses for ideas and theories Good at problem solving Prefers technical tasks and problem solving to social and interpersonal issues
Diverger	Good at viewing situations from many angles Prefers to observe and gather information before taking action Enjoys brainstorming
Assimilator	Good at grasping a wide range of information and putting into concise, logical form Interested in abstract ideas and concepts Feels that it is more important that a theory have logical soundness than practical value
Accommodator	Enjoys hands-on experiences Enjoys new and challenging experiences Tends to act on "gut" feelings versus logical analysis Relies on people versus analysis

Reliability of Instrument

According to Cronbach's α scale (n = 268), the 4 basic scales (CE, 0.82; RO, 0.73; AC, 0.83; AE, 0.78) and 2 combined scores (AC-CE = 0.88; AE-RO = 0.81) of the LSI have all shown good internal reliability.[13] Gieger and Pinto[9] also reported reliability coefficients above 0.75 for all learning attributes across 3 administrations of the instrument and indicated that the LSI was measuring the individuals' learning attributes with fair reliability.

Although concerns were levied against the test-retest reliability of the 1976 version of the LSI, test-retest indexes ranging from 0.42 to 0.60 indicate improvements with the 1985 revision. While these coefficients are approaching acceptable levels, some concern still exists as to potential psychometric limitations. Several arguments against dismissing the LSI as a viable testing instrument, however, should be considered. First, despite criticisms, the LSI continues to be 1 of the most widely used learning style inventories in current research.[14] Second, Gieger and Pinto,[9] who examined learning style consistency over time (3 years), found high stability from the first to the second administration. Finally, it is unclear if the test-retest reliability results are a function of the inventory's instructions. The instructions given to respondents are as follows: "Try to recall some recent situation where you had to learn something new, perhaps in your job." These instructions allow the

Table 2. Means and Standard Deviations for Each Learning Mode and Setting

Mode	Setting	Mean	SD	*t* Value
Feeling (Concrete Experience)	Class	20.77	4.22	.523
	Clinical	21.35	5.34	
Watching (Reflective Observation)	Class	34.42	7.61	−3.293*
	Clinical	30.04	7.28	
Thinking (Abstract Conceptualization)	Class	31.69	7.72	−.963
	Clinical	30.27	7.23	
Doing (Active Experimentation)	Class	33.12	7.60	3.663*
	Clinical	38.81	6.23	

*$P < .05$.

respondent to choose the learning situation to be used as a reference when ranking the 4 sentence endings. Given that most test-retest reliability data are collected over 3- to 6-month intervals in order to eliminate a possible interference effect from memory, it cannot be stated with any certainty that the respondent is using the same learning situation reference on both occasions. This notion is supported by Kolb,[10] who suggested that an individual's interpretation of a situation should influence the mode he or she uses to some degree and that test-retest reliability coefficients would, therefore, be less than 1.0 even if the LSI had no measurement error. By providing respondents with a specific focus in this study, this concern should be reduced.

Procedure

The test was administered to subjects 1 month into the spring semester during their clinical practicum seminar class. Each subject completed the test twice and was instructed to answer the questions as if either learning something new in the classroom or learning something new in the clinical setting. Immediately after the first test was completed, each subject changed focus and retook the test. The order of focus regarding setting (classroom or clinical) was counterbalanced across subjects.

Each respondent's total score for each of the 4 modes was determined by adding the rankings for the 12 sentence completions. The higher total indicated the respondent's mode preference. Combined scores for the AC-CE and AE-RO scales were then calculated to determine subjects' learning styles (converger, diverger, assimilator, or accommodator) in each setting (class versus clinical). In order to determine if differences existed between the 2 settings (classroom and clinical), 4 paired *t* tests were performed ($P < .05$), 1 for each learning mode. In addition, the percentage of respondents whose learning styles were not identical for both settings was determined.

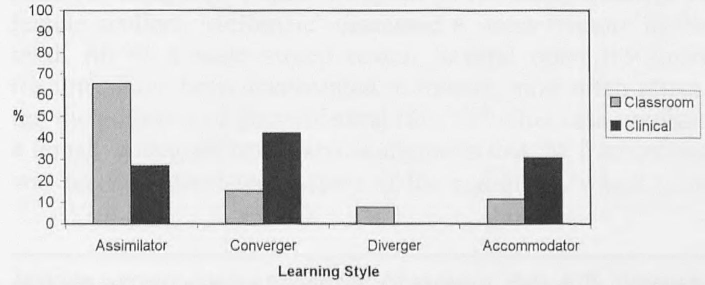


Figure 2. Percentage of respondents categorized in each learning style.

RESULTS

Means and standard deviations for each learning mode and setting are illustrated in Table 2. The results of the *t* tests indicated a significant difference in modal preference across settings (classroom versus clinical) for the modes of RO ($t = -3.29$, $P < .05$) and AE ($t = 3.66$, $P < .05$), respectively. Examination of the means indicated that the preferred mode in the classroom was RO, whereas AE was more prevalent in the clinical setting. Differences were not statistically significant for the CE and AC modes. Furthermore, 58% of respondents ($n = 15$) switched learning style according to setting focus (classroom versus clinical).

Learning profiles for the classroom setting focus revealed the predominant learning style to be that of assimilators (65.4%), followed by convergers (15.4%). The predominant learning style for the clinical setting focus differed, with 42.3% of respondents categorized as convergers, followed by 30.8% accommodators (Figure 2).

DISCUSSION

These results offer support for the notion that learning style consistency may depend on the learning environment.[12] Coker[12] examined learning style consistency, comparing classroom and motor skill learning. Similarly, learning style consistency for 2 distinctly different learning environments, classroom and clinical (experiential and hands on), was examined in this study. In contrast, Copenhaver's[7] study, which found learning styles to be stable across English and mathematics, only explored 1 learning environment, the classroom. Consequently, the discrepancies in the literature may be attributed to slight differences in experimental focus.

In addition, a critical look at the administrative instructions of the LSI may provide a rationale for the contradictions in the literature and the test-retest reliability issue. The LSI instructs respondents to base their answers to the questions on a recent learning experience. In essence, each respondent is, therefore, permitted to select his or her own focus from which to answer the questions. No test focus was provided by either Copenhaver[7] or Gieger and Pinto,[9] whereas a specific test focus was given in both this study and Coker.[12] Consequently, to ensure the validity of the resulting learning style profile, it may be necessary to provide the respondent with a specific focus, either the traditional classroom or the clinical setting, before administering the LSI.[13] Furthermore, future examinations of the test-retest reliability of the LSI should provide a specific learning focus.

From a practical perspective, several implications should be considered by athletic training educators. In a classroom focus, respondents indicated a preference for the RO mode. This mode is characterized by a preference to make careful observations before making a judgment, viewing issues from a variety of perspectives, and searching for the underlying meaning of things.[13] In a clinical environment, however, the preference appears to shift to AE, which emphasizes hands-on learning.

By understanding student learning preferences, athletic training educators could enhance the quality of the learning experiences they provide. Because research has shown increased learning achievement when instructors match their teaching style with individual learning styles,[1–3] teaching strategies should be re-examined to ensure that each learner's needs in both the classroom and clinical settings are accommodated. According to the results of this study, then, the use of instructional strategies that provide opportunities for discussions, brainstorming, reflective thinking, and critical thinking would improve the quality of athletic training students' classroom experiences, while the clinical setting should incorporate simulations, case studies, and hands-on experience to maximize learning potential.

Because learning style is directly influenced by one's preference for each of the 4 learning modes, a corresponding shift in learning style preference would be expected. In fact, 58% of respondents' learning styles did change from the classroom to the clinical setting. Interestingly, the predominant learning style in the classroom was that of the assimilator (65.4%), while the converger style prevailed in the clinical setting (42.3%). This result is notable, as it indicates a shift in focus from theory and abstract ideas and concepts in the classroom to making decisions, solving problems, and finding practical uses for ideas and theories in the clinical setting.[13] A closer inspection of the 2 learning environments would lead one to expect these changes to occur if learners were to be successful in each setting. Finally, medical technicians and physicians are often classified as convergers, while there is a tendency for academic physicians, researchers, and professors to be categorized as assimilators.[13] The results of this study, therefore, appear to be somewhat consistent with the research predicting career paths based on learning style.

CONCLUSIONS

According to Kolb's Experiential Learning Theory, a well-rounded learning process involves the use of all 4 learning modes. In fact, learners may miss important ideas and experiences if they rely too heavily on 1 learning mode.[13] So, although some ideas have been provided to accommodate the predominant learning modes found in this study, athletic training educators should remember that every individual's learning strengths and weaknesses are different. The important result from this study to consider is that learning mode and style may shift according to whether the respondent is focusing on a classroom or clinical environment. Therefore, I suggest that the athletic training educator administer the LSI twice to determine an individual's cognitive and experiential learning style profiles and that there be a corresponding shift in the instructional techniques and strategies used in each setting to maximize learning potential.

REFERENCES

1. Dunn R, Beaudry JS, Klavis A. Survey of research on learning styles. *Educ Leadership.* 1989;46:50–58.
2. Dunn R, Dunn K. Learning styles, teaching styles. *Natl Assoc Second Sch Principals Bull.* 1975;59:38–49.
3. Price G, Dunn R, Sanders W. Reading achievement and learning style characteristics. *Clearing House.* 1981;54:223–226.
4. Draper DO. Students' learning styles compared with their performance on the NATA certification exam. *Athl Train.* 1989;24:234–235.
5. Harrelson G, Leaver-Dunn D, Wright KE. An assessment of learning preferences among undergraduate athletic training students. *J Athl Train.* 1997;32:S24.
6. Brockhaus J, Woods M, Brockhaus RH. Structured experiential learning exercises: a facilitation to more effective learning in clinical settings. *J Psychosoc Nurs Ment Health Serv.* 1981;19:27–32.
7. Copenhaver RW. *The Consistency of Learning Styles as Students Move from English to Mathematics* [dissertation]. Bloomington, IN: Indiana University; 1979.
8. Satterly DJ, Brimer MA. Cognitive styles and school learning. *Br J Educ Psychol.* 1977;41:294–293.
9. Gieger MA, Pinto JK. Changes in learning style preference during a three year longitudinal study. *Psychol Rep.* 1991;69:755–762.
10. Kolb DA. *Learning Style Inventory Technical Manual.* Boston, MA: McBer and Co; 1976.
11. Smith LH. *Learning Styles: Measurement and Educational Significance* [dissertation]. Hartford, CT: University of Connecticut; 1996.
12. Coker CA. Learning style consistency across cognitive and motor settings. *Percept Mot Skills.* 1995;81(3 Pt 1):1023–1026.
13. Kolb DA. *Learning Styles Inventory: Self-Scoring Inventory and Interpretation Booklet.* Boston, MA: McBer and Co; 1985.
14. Henke HA. Applying learning theory to computer based training and web based instruction. Available at: http://www.scis.nova.edu/~henkeh/story2.html. Accessed February 18, 2000.

Journal of Athletic Training 2000;35(4):445–449

www.journalofathletictraining.org

Stress Fracture of the Eighth Rib in a Female Collegiate Rower: A Case Report

Adam Galilee-Belfer, MA, ATC; Kevin M. Guskiewicz, PhD, ATC

University of North Carolina at Chapel Hill, Chapel Hill, NC

Objective: To present the case of a stress fracture of the eighth rib in a female collegiate rower.

Background: A female collegiate rower experienced severe pain in her chest, increasing with movement, deep breathing, and erect posture. No acute mechanism of injury was apparent. The team physician diagnosed a rib stress reaction based on clinical examination. The athlete rested for 2 days and then was able to resume rowing workouts. Five months later, she experienced the same sharp pain, with the diagnosis and treatment being the same. The athlete was able to compete in the championships 3 weeks later. At the end of the season, a bone scan revealed a stress fracture of the eighth rib. The athlete rested for 3 weeks and then returned to activity.

Differential Diagnosis: Intercostal muscle strain, serratus anterior muscle strain.

Treatment: Active rest, involving pain-free cardiovascular workouts and weight training, cessation of rowing until the athlete was asymptomatic, strengthening of dynamic support structures, and analgesic modalities.

Uniqueness: Most stress fractures occur in the lower extremity. Those that do occur in the rib cage most often involve the first rib. A limited number of published works have addressed stress fractures to the remaining ribs; of these, posterior and posterolateral fracture sites are most often reported. This case is unique in that the fracture site was on the anterolateral aspect of the eighth rib.

Conclusions: Stress fractures are thought to result from a variety of causes, including muscular fatigue, sudden changes in training intensity or duration, and microtrauma to bone at the muscular origin and insertion sites ("wear-and-tear" theory). In addition, hormonal factors in women can predispose an athlete with amenorrhea to a decrease in bone mineral content. Athletic trainers should be aware of these potential causes and focus on the prevention of stress fractures.

Key Words: overuse injury, rowing injury, amenorrhea, female athlete

Rowing is the oldest competitive collegiate sport, evolving from the first race held in long wooden boats in 1829 between Oxford and Cambridge Universities to the sleek fiberglass sculls used by today's crews.[1] Rowing is a nonimpact sport that demands both endurance and explosive power from its participants. Elite rowers train throughout the year on ergometers (rowing machines) and on the water. Accordingly, most injuries in rowing are chronic or overuse injuries. We document an elite-level university rower who sustained a stress fracture of the eighth rib.

Stress fractures to the ribs are not uncommon, although few cases have been reported. The first rib is most commonly affected. However, a limited number of publications have addressed stress fractures to the remaining ribs, particularly in rowers. Brukner and Khan[2] reported on stress fractures of the seventh and eighth ribs in a female sculler. Holden and Jackson[3] described 4 cases of posterior rib stress fractures in female scullers. McKenzie[4] discussed a stress fracture in the ninth rib of a male sweep rower. Several other rib stress fractures have been documented in rowers, most often affecting the posterior or posterolateral ribs.[1,5–8] This case involves a female collegiate rower and is unique in that the fracture site was on the anterolateral aspect of the eighth rib, which is an extremely uncommon location for this type of rowing-induced injury.

Address correspondence to Kevin M. Guskiewicz, PhD, ATC, University of North Carolina at Chapel Hill, CB#8700, Fetzer Gymnasium, Chapel Hill, NC 27599-8700. E-mail address: gus@email.unc.edu

CASE HISTORY

A 20-year-old female rower (height = 1.75 m, weight = 70.5 kg) presented with intense, left-sided chest pain. She rowed starboard on a boat of 8, and her team, which had recently completed the fall season, had begun its indoor training season several weeks earlier. The athlete described waking up that morning experiencing severe pain. Her pain increased with movement, deep inspiration and expiration, and erect posture. No obvious swelling or deformity was noted. No mechanism suggesting an acute injury was discovered. Her pain was localized over the anterolateral aspect of the eighth rib and did not radiate along the body of the rib. Her pain increased with shoulder flexion, abduction, and extension, trunk flexion, and end-range extension. In addition, her pain intensified with resistance to any upper extremity movements, particularly scapular protraction and retraction.

A rib stress reaction was suspected based on clinical evaluation. However, the athlete vehemently expressed her desire to continue rowing, regardless of pain. Unless the injury was life threatening, she planned to participate. The team physician decided that no further diagnostic tools (eg, radiographs or a bone scan) would be used because the course of treatment did not depend on a definitive diagnosis. The physician permitted a return to participation as soon as she was asymptomatic.

The athlete rested for 2 days and then returned to her ergometer workouts; modification of activity was moderate at best. Ice, intercostal massage, and electric stimulation were all used with the goal of pain relief. In addition, pain-free scapular protraction exercises to strengthen the serratus anterior muscle were initiated and added to the permanent weight-training routine (Figure 1). The athlete remained relatively asymptomatic for several months, complaining only of occasional pain.

Approximately 5 months later, the athlete was moved to a bow-coxed boat of 4 to prepare for the national championships. One week later, she had to stop in the middle of an intense workout due to severe chest pain, described as identical to the previous incidence. The course of treatment again involved 2 days' rest and analgesic modalities. The athlete was able to return to participation and competed in the championships 3 weeks later.

After the end of the season, a chest roentgenogram was obtained and revealed no abnormalities (Figure 2); however, a technetium-99 bone scan taken 1 week later revealed a stress fracture of the eighth rib (Figure 3), with significant uptake of

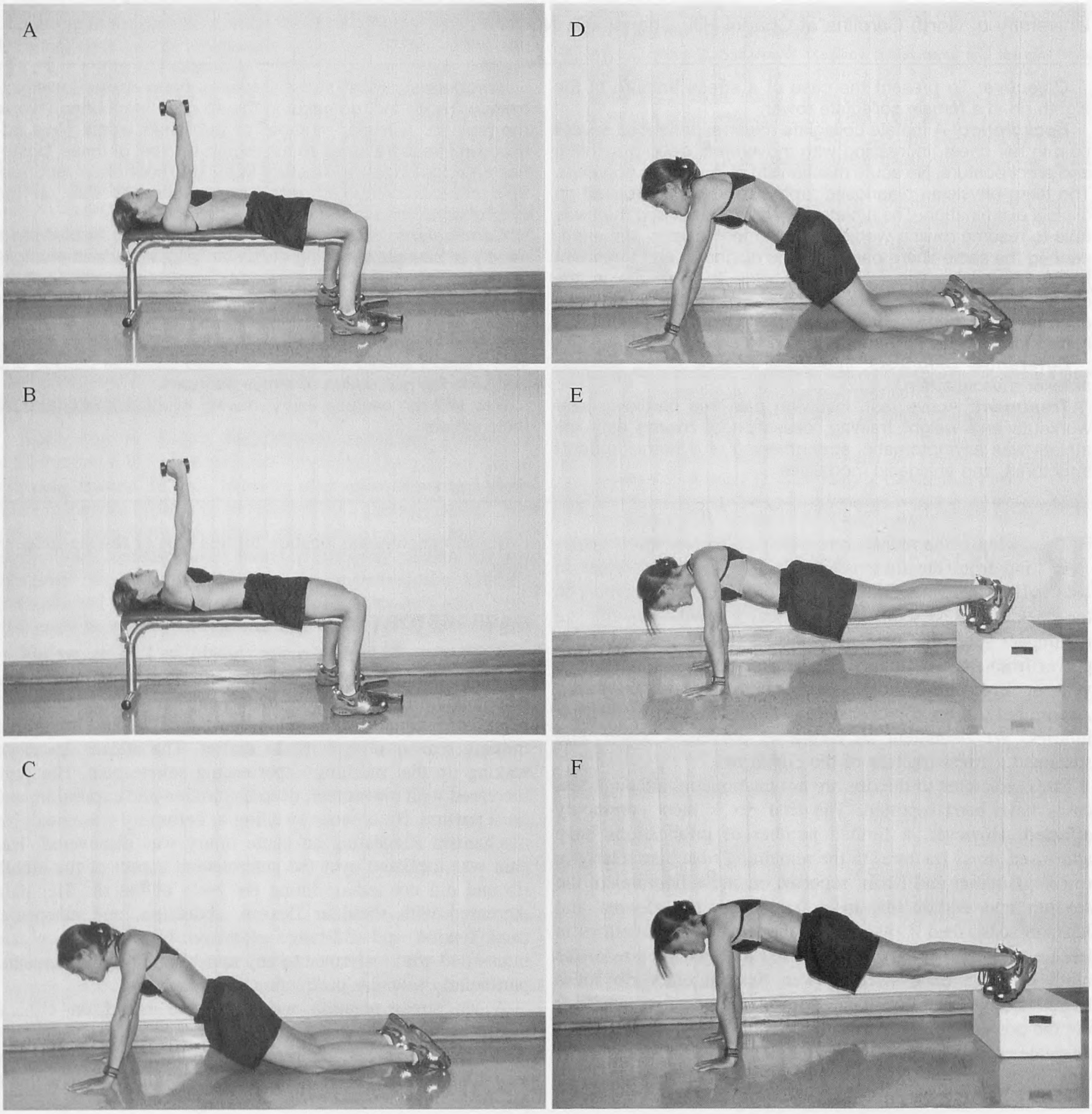


Figure 1. Rehabilitation progression. Rehabilitation progression used to strengthen the serratus anterior, the primary dynamic supporter of the ribs. A, Starting, and B, finishing positions for the initial, nonweightbearing exercise. C, Starting, and D, finishing positions for the second stage of the strengthening progression. E, Starting, and F, finishing positions for the advanced stage of the strengthening progression. All of the exercises were performed throughout the full functional range of motion.

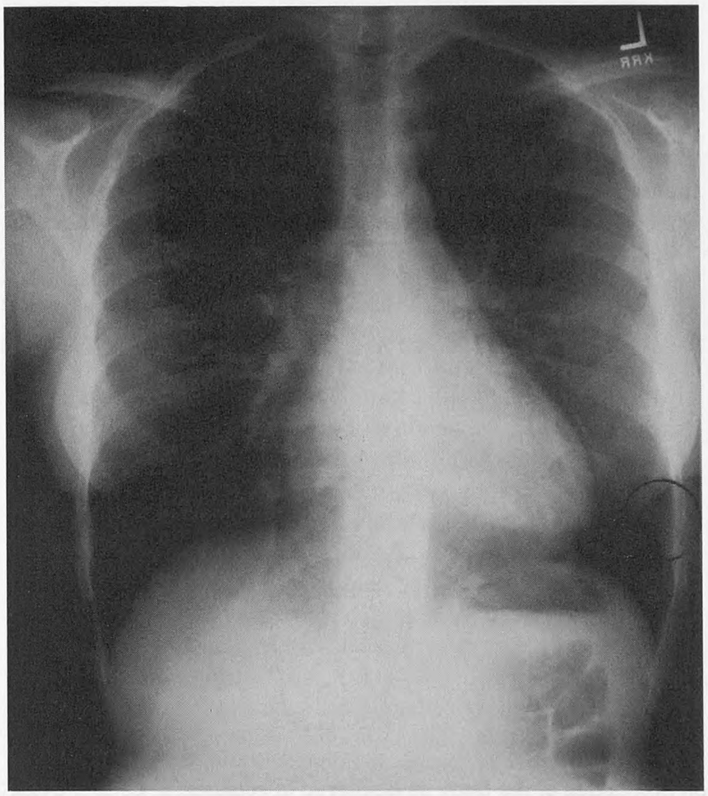


Figure 2. Chest x-ray film taken at the end of the competitive season revealed no abnormalities at the site of the fracture (circled area).

the isotope along the anterolateral aspect of the rib. The results of the bone scan were convincing enough to the athlete that she immediately modified her training regimen. She limited her cardiovascular workouts to biking and running and eliminated ergometer training. The team physician instructed her to resume ergometer training only after she had gone a full week without chest pain. A time frame of 4 to 6 weeks was anticipated to allow for adequate healing of her injured rib, but the athlete returned to ergometer training 3 weeks later with no chest pain. A follow-up chest roentgenogram 4 months later revealed callus formation on the eighth rib at the fracture site (Figure 4). She has been asymptomatic for more than a year, while continuing to row and participate in ergometer training.

DISCUSSION

Only 10% of all stress fractures in athletes affect the upper extremity.[5] However, the repetitive, near-maximal isotonic contractions performed by rowers predispose them to upper extremity chronic injuries, such as stress fractures. Stress fractures may occur when muscle weakness or fatigue causes a redistribution of force to the underlying bone.[4] When activity is initiated, a considerable percentage of the external force is dissipated by the supporting musculature. In this case, the serratus anterior and the abdominal oblique muscles dissipate the bending stresses exerted upon the rib. However, when the muscle fatigues, the bone is forced to absorb the bending stress, which is greatest at the focal portions of the bone.[4] In the past, this has been considered applicable to weightbearing activities only. However, segments of the ribs undergo significant bending stresses on activation of the force couple of the rhomboids and the serratus anterior. In addition, the activation and repetitive contractions of the external abdominal oblique muscle result in significant stress being placed upon the origins of these muscles along the eighth rib.[3,6] The origins of these muscles combine to create focal points of stress along the bone.

The long periods of cyclic contraction and relaxation of muscle have been theorized to lead to a fatigue mode of loading.[9] This loading pattern also lends itself to the "wear-and-tear" theory: that overuse and overload of muscle result in microtrauma to the bone, predominantly at the origin sites of these thoracic muscles.[5] Also, stress along the surface of the underlying rib is notable, as the muscle pull across the bone generates significant forces. The stress fracture occurs where the bending stresses are maximal.[4,7]

Previous authors[3,4] have theorized that the point of maximal stress occurs over the posterior or posterolateral aspect of the ribs. Most of the published reports have focused primarily on

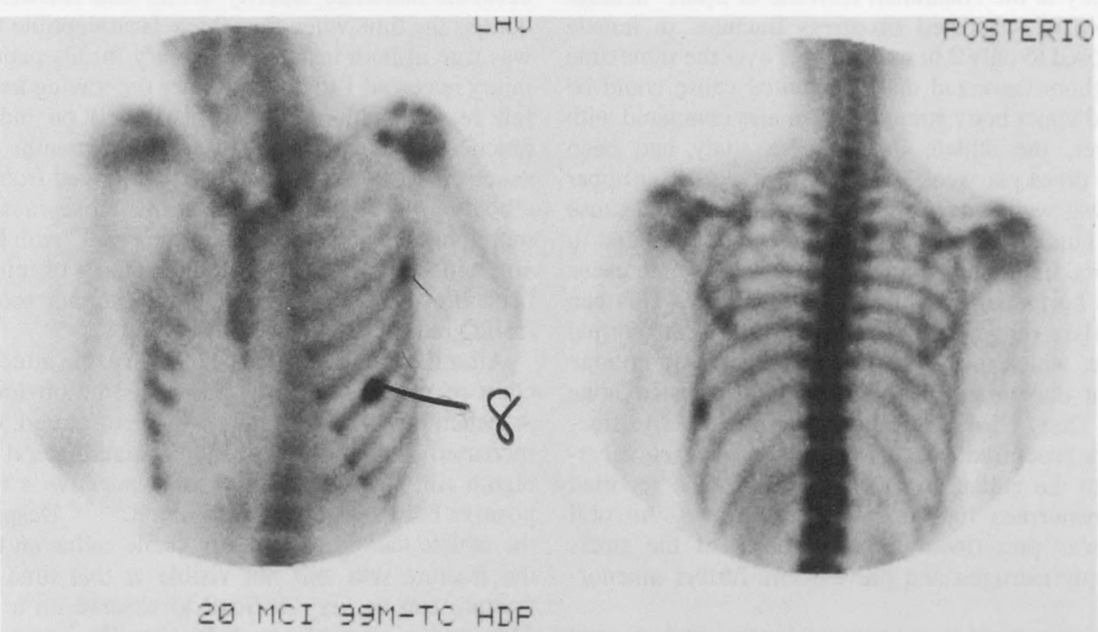


Figure 3. Technetium-99 bone scan revealed a stress fracture along the anterolateral aspect of the eighth rib.

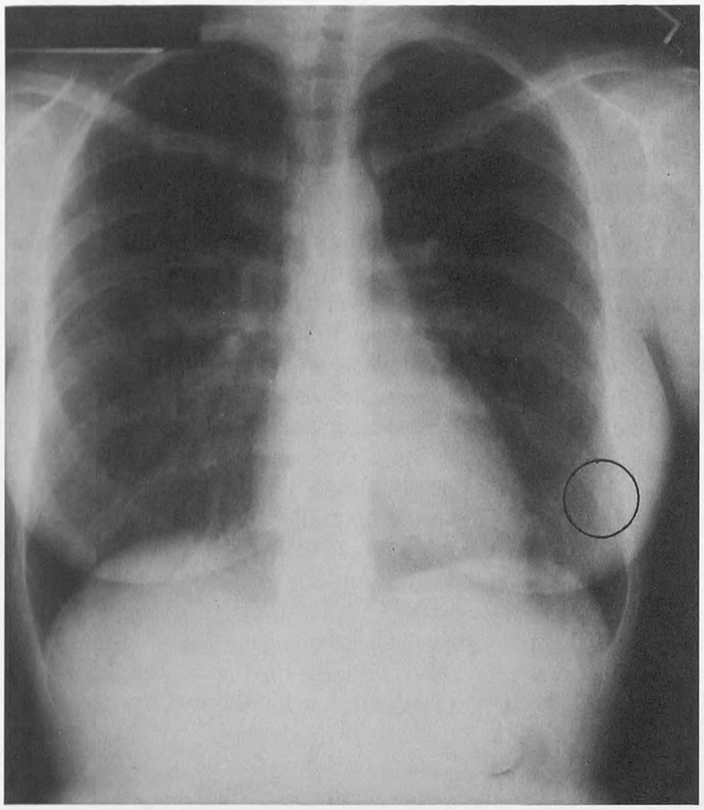


Figure 4. Follow-up chest x-ray film taken 4 months after the previous film revealed callus formation on the eighth rib at the location of the stress fracture.

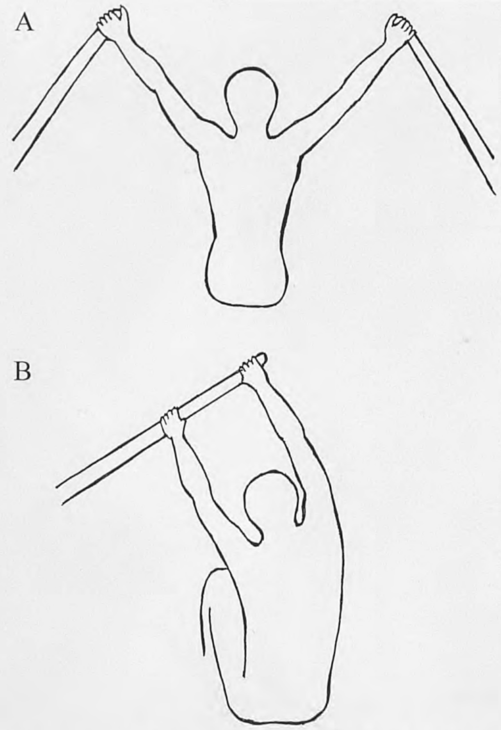


Figure 5. Compressive forces. A, Equal distribution of shear forces on both sides of the body as displayed in sculling. B, Lateral trunk shift, which is characteristic of sweep rowing. It is theorized that this lateral shift could result in compressive forces along the inside aspect of the thoracic cavity, providing a potential cause for a rib stress fracture.

sculling, in which an athlete uses 2 oars; sweep rowing can result in a lateral shift of pressure on the ribs because the rower uses 2 hands on a single oar.[5] While sculling is a linear motion, sweep rowing is characterized by a diagonal movement pattern (Figure 5). Repetition of this movement results in compression along the inside aspect of the thoracic cavity. This compression is concentrated along the anterolateral aspect of the ribs, consistent with the unique location of the stress fracture in this patient.

A 9-year study at the Australian Institute of Sport[6] demonstrated 15 cases of suspected rib stress fractures in female rowers, as opposed to only 2 in male rowers over the same time frame. The authors theorized that 1 potential cause could be underdeveloped upper body strength in females compared with males. However, the athlete in our case study had been weightlifting 3 times per week for the past 3 years. Her upper body strength was well above average. Another potential cause for the higher number of female stress fractures is related to hormonal factors. Endurance training has been proven to cause changes in the hormone levels of a female athlete. This can lead to amenorrhea, or the disruption or cessation of the normal menstrual cycle, which in turn can lead to a decrease in bone mineral content due to the lack of estrogen-mediated bone synthesis.[3,6,10] Thus, weakened bones are vulnerable to fractures caused by repetitive external stresses. This theory certainly applies to the athlete in this case study, who reported intermittent amenorrhea for the last 2 to 3 years. An oral contraceptive was prescribed upon diagnosis of the stress fracture to supply estrogen and prevent any further amenorrhea.

The phenomenon of endurance training leading to decreased bone density should not deter a female athlete from pursuing endurance activities. Under controlled conditions, these activities will increase bone density and strength in accordance with the Wolff law. The endurance demands placed on the body will eventually lead to stronger bones and muscles. The key to this change is a gradual increase in activity. The body must be given ample time to adapt to higher external forces and demands; 1 of the well-documented causes for a stress fracture is a sharp increase in level of activity.[1,3–5,11] The delay between increased activity levels and increased strength of bone is the time when the athlete is susceptible to injury.[8] This was true in both instances of injury in this patient. The initial injury occurred 1 to 2 weeks after the rowing team finished the fall season outdoors and concentrated on indoor ergometer practice. The recurrence of injury 5 months later occurred exactly 1 week after the athlete was moved from a boat of 8 to a boat of 4. The athlete felt that this move caused her to exert more pressure and create a "stronger pull" with her inside (left) arm and side of the body. Both instances of injury might have been prevented if the changes in training routine had been made gradually.

After the second incidence of injury, the athlete underwent a chest roentgenogram, which revealed no obvious deformities. A techetium-99 bone scan was then performed, which revealed increased isotope uptake on the anterolateral aspect of the eighth rib. The combination of a negative x-ray film and a positive bone scan is not uncommon.[2,4,5,8] Despite the fact that the athlete had developed a palpable callus on the injured rib, the fracture was still not visible at that time. A simple rib fracture can be very difficult to observe on a standard x-ray film, and a stress fracture is virtually impossible to see.[4,8] However, a follow-up X-ray film at the preparticipation examination several months later revealed callus over the previous fracture site.

It is important to note the possibility that either occurrence of injury could have been an acute fracture. The onset of pain in both instances is consistent with that of an acute fracture. An acute fracture would look the same as a stress fracture on a bone scan; any standard chest x-ray film can miss a fracture unless dedicated rib views are obtained.

CONCLUSIONS

This case study documents a rare injury in sport: a stress fracture of the eighth rib in a female collegiate rower. She was initially diagnosed with a possible stress reaction after the fall season, and after slight activity modification, she returned to activity 2 to 3 days later. She remained mostly asymptomatic until a reinjury 5 months later. Once again, treatment involved moderate activity modification and analgesic modalities. A positive bone scan after the season revealed a stress fracture of the eighth rib. The course of treatment included a progression of scapular protraction exercises, oral contraceptives, and 3 weeks of active rest.

Many theories have been advanced as to the causes of stress fractures. These include muscular fatigue that redistributes external force to bone and repetitive microtrauma along muscular origins (the "wear-and-tear" theory). In addition, hormonal factors can play a significant role in a stress fracture in a female athlete. Amenorrhea leading to decreased bone mineral content should be addressed in the course of treatment. A gradual increase in level of training allows the necessary time for the body to adapt, potentially preventing stress fractures.

REFERENCES

1. Hosea TM, Boland AL. Rowing injuries. In: *Postgraduate Advances in Sports Medicine: An Independent Study Course Designed for Individual Continuing Education*. Philadelphia, PA: University of Pennsylvania School of Medicine; 1989:1–16.
2. Brukner P, Khan K. Stress fracture of the neck of the seventh and eighth ribs: a case report. *Clin J Sports Med.* 1996;6:204–206.
3. Holden DL, Jackson DW. Stress fracture of the ribs in female rowers. *Am J Sports Med.* 1985;13:342–248.
4. McKenzie DC. Stress fracture of the rib in an elite oarsman. *Intl J Sports Med.* 1989;10:220–222.
5. Christiansen E, Kanstrup IL. Increased risk of stress fracture of the ribs in elite rowers. *Scand J Med Sci Sports.* 1997;7:49–52.
6. Hickey GJ, Fricker PA, McDonald WA. Injuries to elite rowers over a 10-yr period. *Med Sci Sports Exerc.* 1997;29:1567–1572.
7. Mosler A, Wajswelner H. The assessment and management of elite rowing injuries. Presented at: Australian Institute of Sport: Australian Conference of Science and Medicine in Sport; October 17–20, 1995; Hobart, Australia.
8. Strayer LM. The myth of the intercostal muscle pull. *Am Rowing.* 1990;22:42–44.
9. Mickelson TC, Hagerman FC. Functional anatomy of the rowing stroke. *Oarsman.* 1979;11:6–9.
10. Puustjarvi K, Arnala I, Arokoski J. Bone mineral less after long term running in association with decreased osteoid formation and estradiol level. Presented at: 39th Annual Meeting, Orthopaedic Research Society; February 15–18, 1993; San Francisco, CA.
11. Lord MJ, Ha KI, Song KS. Stress fracture of the ribs in golfers. *Am J Sports Med.* 1996;24:118–122.

Journal of Athletic Training 2000;35(4):450–452

www.journalofathletictraining.org

Concurrent Periostalgia and Chronic Proximal Deep Posterior Compartment Syndrome in a Collegiate Track and Field Athlete: A Case Report

Kristinn I. Heinrichs, PhD, PT, SCS, ATC, CSCS*; Wendy M. Lachowicz, ATC†; Don E. Detmer, MD, FACS, FACSM‡

*Armstrong Atlantic State University, Savannah, GA; †Sterling, VA; ‡School of Medicine, University of Virginia, Charlottesville, VA

Objective: Exercise-induced leg pain may be triggered by abnormally high compartment pressure. In addition to the more widely publicized anterior compartment syndrome, the deep posterior compartment syndrome can just as frequently occur, resulting in severe pain and disability due to muscle and nerve ischemia.

Background: Obtaining a thorough history and compartmental pressure measurements are the usual components in the accurate diagnosis of compartment syndromes. While few other disorders mimic compartment syndromes, differential diagnoses must be considered. Surgical management of deep compartment syndrome, consisting of fasciotomy or fasciectomy, or both, is successful for most patients.

Differential Diagnosis: Tibial stress fracture or microfracture, tibial periostitis, tibial periostalgia, distal deep posterior chronic compartment syndrome, proximal deep chronic compartment syndrome, superficial lateral compartment syndrome, deep venous thrombosis, popliteal artery entrapment, or chronic compartment syndrome.

Uniqueness: Chronic deep compartment syndrome is one of the most common causes of exercise-induced leg pain in aerobic athletes. Therefore, the athletic trainer must be able to recognize the condition. Signs, symptoms, diagnosis, and surgical management of chronic deep compartment syndrome, chronic periostalgia, and superficial lateral compartment syndrome in a 21-year-old Division IA track and field athlete are presented.

Conclusions: With the correct diagnosis, persistent and methodical reevaluation, and appropriate management, the athlete can expect a successful treatment outcome.

Key Words: leg pain, fasciotomy, exercise-induced running injury, track and field, medial tibial stress syndrome

Exercise-induced leg pain that is relieved only by an absence from activity is an uncommon, but not rare, complaint among athletes, particularly runners. First described and treated by Mavor[1,2] in 1956, reports of chronic, as well as acute (described by Vogt[2] in 1943), exertional compartment syndromes involving the compartments of the lower leg have increased in number. Chronic compartment syndromes affecting the deep posterior compartments of the lower leg are second in frequency of occurrence to those affecting the anterolateral compartment of the leg. The major symptoms include paresthesia of the plantar aspect of the foot and tightness, cramping, and aching in the deep muscles posterior to the tibia.

Chronic compartment syndrome (CCS) affecting the deep posterior compartment is most frequently seen in well-conditioned athletes participating in aerobic sports. However, CCS must be differentiated from the acute and chronic injuries that often result from stress overload due to poor training techniques (the "too fast, too hard, too soon" syndrome). This includes some forms of medial tibial stress syndrome, such as periostalgia and stress fractures, and such problems as tendinitis, deep venous thrombosis, and even acute compartment syndromes.[3,4] An accurate diagnosis of compartment syndromes most often relies upon the clinician's awareness of the condition, as well as a clear understanding of the anatomy of the lower leg. We present a prototypical case of chronic proximal deep posterior compartment syndrome associated with chronic periostalgia and latent posterior superficial compartment syndrome.

Address correspondence to Kristinn I. Heinrichs, PhD, PT, SCS, ATC, CSCS, Armstrong Atlantic State University, 11935 Abercorn Street, Savannah, GA 31419-1997. E-mail address: kheinrichs@savannahga.net

CASE REPORT

A 21-year-old male 110-m hurdler and sprinter at a Division IA university complained of recurring pain and tightness in both calves over a 4-year period, beginning during his junior year of high school. He also showed bilateral signs and symptoms of chronic tibial stress syndrome (periostalgia) along the anterior tibial crest. At this early stage, he was asymptomatic during the 110-m hurdle competition, but he noted extreme tightness and deep muscular soreness in his calves the next day.

During his freshman year of college, his condition continued to worsen. He treated the tibial stress syndrome primarily with stretching exercises and cold whirlpool treatments, which probably had a mild therapeutic effect on his calves as well.

During his sophomore collegiate track season, the bilateral calf pain and cramping were no longer limited to postcompetition but began to appear immediately after races and during practice. These symptoms ultimately led to cessation of running for short periods of time. Reduced training levels during the summer months resulted in complete resolution of the pain, tightness, and cramping.

During the fall season of his junior year, the pain increased in frequency and intensity, with an earlier onset of symptoms. Again, complete rest eliminated all symptoms. With the onset of spring season training, the symptom behavior was markedly worse than before, with the onset of deep sharp pain in both calves within 5 to 10 minutes of exercise. At this point, the athlete was forced to discontinue all training. These symptoms of constant, sharp pain and increasing muscle tightness in both calves were absent at rest but reappeared more rapidly with any activity, including normal activities of daily living.

Active plantar-flexion range of motion was restricted and painful. No swelling in the proximal calves or instep paresthesias were noted. Normal pulses were present in both feet and did not diminish with plantar flexion or dorsiflexion. Rest completely relieved the symptoms, which were unaffected by time of day (ie, no significant improvement or deterioration in symptoms upon arising in the morning or in the evening). A bone scan revealed no stress fracture of the tibia. Magnetic resonance imaging and Doppler evaluation demonstrated no popliteal artery entrapment or evidence of past deep venous thrombosis. No clotting disorders were identified.

A variety of conservative approaches was attempted without long-term success. These included cryotherapy, heat, whirlpools, contrast baths, ultrasound, nonsteroidal anti-inflammatory medications, orthotics, passive and active exercise to improve flexibility of the involved musculature, multidirectional ankle conditioning exercise, alternate cardiovascular exercise (stationary bicycle and aquatic rehabilitation), relative rest periods, and total rest. The diagnosis at this time was "bilateral medial tibial stress syndrome with periostitis and chronic tendinitis," since no stress fracture was seen on imaging studies. A low level of suspicion for chronic compartment syndrome was entertained, but the location was considered to be too proximal. Persistent symptoms despite conservative measures led to a referral for a second opinion near the end of his junior year in college.

On physical examination, the consultant noted that the athlete was quite tender to palpation bilaterally upon compression of the proximal deep compartment musculature deep to the gastrocnemius-soleus muscle group. Although his pain was described as being in the calves, he described no discomfort limited to the posterior superficial compartments, and he had no tenderness upon compression of these muscles. All other compartments were nontender. Since blind needlesticks to measure deep compartment pressures risk injury to the neurovascular bundle and since his symptoms and signs so clearly pointed to the proximal deep compartments, no pressure measurements were made of either the deep or superficial compartments. After the diagnosis of chronic bilateral proximal deep posterior compartment syndrome and chronic periostalgia, the athlete underwent proximal bilateral deep fasciotomies posteriorly and periostectomies anteriorly of the tibias.

Postoperative recovery consisted of rest and elevation for the first 48 to 72 hours to prevent increased levels of edema. The athlete wore elastic wraps when weightbearing for the first 7 to 14 days, and he resumed daily activity as tolerated. Some ankle and muscle swelling after this surgery is not unusual, and most patients return to walking unassisted in 2 days and progress to running in approximately 3 to 6 weeks.[5–7]

By 3 months postoperatively, the athlete was performing a slow jog for a 4-km to 4.8-km workout on soft surfaces every other day. In addition, he was also engaging in nonweightbearing activities such as swimming on the "off" days. He had no complaints of pain or deep tightness during activities of daily living, and pain associated with exercise at this time was characteristic of exercise-induced or "not having worked out" pain. Unfortunately, the athlete was unable to progress beyond the daily, low-intensity jogging program of 30 minutes achieved by 4 months due to persistent superficial proximal posterior compartment aching, which limited the duration of the exercise session. However, his symptoms were not the same type of cramping or deep tightness experienced preoperatively in the deep compartment. A return visit to the surgeon revealed elevated resting superficial compartment pressures (18 to 23 mm Hg). Superficial fasciotomy under local anesthesia on an outpatient basis was performed on both the medial and lateral proximal posterior superficial compartments bilaterally. Although the patient did not pursue a serious conditioning program because of other commitments, he was active in his daily life and satisfied with his outcome.

DISCUSSION

Clinically, this athlete presented with a classic picture of CCS affecting the deep posterior compartment. Unfortunately, this course was also typical in that visits to a number of physicians occurred before the patient received the correct diagnosis and surgical treatment. Surgical management, in the form of an adequate fasciotomy, is usually a successful option, offering most individuals excellent chances for a full recovery and return to competition.

Traditionally, the lower leg has been divided into 4 anatomically separate compartments: anterior, lateral, posterior superficial, and posterior deep. Detmer[7] has conceptualized the lower leg as having at least 7 functional compartments because chronic compartment syndrome can affect a single subdivision in any of the compartments without involving all of the other muscles within the same compartment (Table). Thus, it is critical that the physical examination clearly distinguish the boundaries of all involved anatomical compartments.

Obtaining a careful history is a critical component in arriving at the correct diagnosis, and it forms the primary basis for a diagnosis of chronic compartment syndrome. The history of persistent cramping with exercise and asymptomatic rest periods with symptoms worsening over time were the major indicators of the problem. It is also important to determine whether the posterior symptoms are superficial or deep and proximal or distal, or both, and to define the exact borders of the area of pain; a common mistake is to simply pinpoint the maximal point of tenderness.[5,8] This athlete's tenderness was localized around his calf muscles. It is important to delineate precisely which muscles are involved; vague descriptions or varying locations for pain strongly weigh against the diagnosis of CCS. Symptoms tend to progressively worsen over time and improve with rest and reduction or cessation of exercise.

Seven Functional Compartments of the Lower Leg[7]

Compartment	Contents
Anterior	Tibialis anterior Extensor hallucis longus Extensor digitorum longus Anterior tibial nerve Tibial artery and vein
Lateral	Fibularis (peroneus) longus and brevis superficial branch of the fibular (peroneal) nerve
Posterior deep proximal	Tibialis posterior Flexor digitorum longus Flexor hallucis longus Posterior tibial artery
Posterior deep distal	Tibialis posterior Flexor digitorum longus Flexor hallucis longus Posterior tibial artery Posterior tibial nerve Posterior tibial vein
Posterior superficial medial	Medial head of the gastrocnemius
Posterior superficial lateral	Lateral head of the gastrocnemius
Posterior superficial distal	Distal soleus

Symptoms eventually adversely affect athletic performance in most patients and are consistent with levels of exercise, as shown in this athlete. Unlike the more easily recognized acute compartment syndrome that requires emergency surgical intervention to spare neurovascular structures, CCS rarely results in permanent neurovascular damage because it is self-limiting, causing the athlete to discontinue activity before prolonged pressure increases result.[4]

The patient's presentation was typical of chronic proximal deep posterior compartment syndrome, but his clinical picture was atypical with respect to his chronic periostalgia and superficial compartment pathology. Chronic periostalgia may occur in association with distal deep posterior compartment syndrome but is decidedly uncommon with proximal compartment disease. In retrospect, it would have been easy to measure superficial compartment pressures at the time of the proximal fasciotomy, but it is not clear that the pressures would have been elevated. Abnormal pressures at that time would clearly have led the surgeon to perform superficial releases. Normal pressures at that time would not have changed the surgeon's plans. As the adage goes, "It is hard to make an asymptomatic patient better." While subsequent compartment syndrome in new compartments after the release of other compartments is uncommon, it does occur. The case highlights the importance of continued re-evaluation and consideration of other underlying pathologies throughout the course of treatment.

The accurate diagnosis and treatment of medial tibial pain is complicated by the coexistence of periostitis and compartment syndrome, as in this athlete. The literature[9] also suggests a third diagnostic entity, tibial stress fracture or microfracture, to be considered in medial tibial pain syndromes. The patient's history of type of exercise (running), initial symptom behavior (quick resolution with rest), and later symptom behavior in the presence of established painful symptoms (failure of rest, orthotics, and nonsteroidal anti-inflammatory medications) are consistent with Type II disease (periostalgia with avulsion of the periosteum at the periosteal-fascial junction).[9] Unfortunately, visits to multiple physicians without obtaining significant relief are also typical in the history of patients with periostitis.

Exercise-induced chronic proximal deep posterior compartment syndrome has become a clearly recognizable cause of chronic leg pain. Research into the causes, diagnosis, and management (conservative and surgical) has advanced the care of this syndrome dramatically over the past 2 decades. Most importantly, the astute clinician should be persistent in observing the athlete in order to correctly diagnose and manage all the underlying problems and arrive at a satisfactory outcome. With the proper treatment, both recreational and elite athletes whose training and lifestyle had been significantly limited by CCS are now typically able to return to preinjury competitive levels.

REFERENCES

1. Mavor GE. The anterior tibial syndrome. *J Bone Joint Surg Br.* 1956; 38:513–517.
2. Vogt PR. Ischemic muscular necrosis following marching. Read before the Oregon State Medical Society, September 1943 (cited by Horn CE. Acute ischemia of the anterior tibial muscle and the long extensor muscle of the toes. *J Bone Joint Surg Br.* 1945;27:615–622.)
3. Stollsteimer GT, Shelton WR. Acute atraumatic compartment syndrome in an athlete: a case report. *J Athl Train.* 1997;32:248–250.
4. Turnipseed W, Detmer DE, Girdley F. Chronic compartment syndrome: an unusual cause for claudication. *Ann Surg.* 1989;210:557–563.
5. Detmer DE, Sharpe K, Sufit RL, Girdley FM. Chronic compartment syndrome: diagnosis, management, and outcomes. *Am J Sports Med.* 1985;13:162–170.
6. Rorabeck CH. The treatment of compartment syndromes of the leg. *J Bone Joint Surg Br.* 1984;66:93–97.
7. Detmer DE. Diagnosis and management of chronic compartment syndrome. *Sem Orthop.* 1988;3:223–233.
8. Allen MJ, Barnes MR. Exercise pain in the lower leg: chronic compartment syndrome and medial tibial syndrome. *J Bone Joint Surg Br.* 1986;68:818–823.
9. Detmer, DE. Chronic shin splints: classification and management of medial tibial stress syndrome. *Sports Med.* 1986;3:436–446.

Journal of Athletic Training 2000;35(4):453–457

www.journalofathletictraining.org

Sacral Stress Fracture in a Female Collegiate Distance Runner: A Case Report

David Klossner, PhD, ATC

Ohio University, Athens, OH

Objective: To present the case of a 19-year-old collegiate distance runner diagnosed with a unilateral sacral stress fracture.

Background: Low back and sacroiliac joint pain are common in female athletes but are often difficult to differentiate. Although sacral stress fractures in young female athletes are rarely reported, they are a potential cause of low back pain.

Differential Diagnosis: Acute lumbosacral strain, disc disease, gluteus maximus strain, idiopathic low back pain, low back strain, sacroiliac joint sprain, or congenital anomaly.

Treatment: Six weeks of active rest and nutritional counseling followed by an incremental 8-week running program. The athlete returned to symptom-free competitive running within 4 months.

Uniqueness: This young athlete presented with a unilateral sacral stress fracture with components of the female athlete triad and a vigorous exercise regime.

Conclusions: Sacral stress fractures are rare in the young female athletic population. Because stress fractures in female runners have various etiologies, a thorough patient history and diagnostic imaging are necessary in the evaluation. Athletes can return to their normal activity once a successful management strategy has addressed all components of the female athlete triad while providing adequate rest. Prevention strategies, such as screening for the components of the female athlete triad, may help to decrease injuries and promote healthier lifestyles among this population.

Key Words: amenorrhea, female athlete triad, low back pain, pelvic stress fracture

Stress fractures are more common in amenorrheic female athletes, yet the etiologies of these injuries often differ.[1–4] Typical stress fracture sites in runners are the lower extremities, with only 6 pelvic stress fractures previously reported in the collegiate athlete population.[2] Some factors potentially related to the occurrence of stress fractures in the lower extremities are an athlete's intensity of training, caloric restrictions, physical conditioning, shoe changes, leg-length discrepancies, amenorrhea, and osteoporosis.[2,4–7] Specifically, researchers[3,8] have found that runners who interrupted their running routine because of injury were more likely to have had irregular or absent menses. This patient's case is significant because of the cumulative effect of common etiologic factors often associated with stress fractures in female athletes, the age of the individual, and the possible presence of an insufficiency-type fracture. Athletic trainers should be suspicious of a sacral stress fracture in a runner with low back pain that does not respond to common conservative treatment protocols.

Stress fractures in collegiate female runners are typically limited to the lower extremity and associated with a fatigue-type mechanism.[2] However, the possibility of an insufficiency fracture should be addressed; as the age of menarche increases, the likelihood of causing irreversible bone loss and accelerating long-term bone resorption increases.[9] Female athletes are experiencing more pressures than in past years, often associated with struggles of improving performance, losing weight, and body image that may lead to disordered eating behaviors and sometimes extend the age of menarche.[9] Accordingly, injury prevalence may be increasing as female athletes present with symptoms associated with disordered eating, amenorrhea, and decreased bone mineral density. These 3 conditions have been established as key components recognized by the American College of Sports Medicine[10] in defining the female athlete triad. The triad concept has been linked to an increased prevalence of injuries in female athletes, but the relationship of each component with exercise is still being researched.[2,3,8,9]

CASE REPORT

A 19-year-old female collegiate distance runner presented with tightness, diffuse pain, and soreness in her low back region for 2 days after a half marathon. An initial evaluation performed by her primary family physician suggested a lumbosacral strain. At the time of the injury, the athlete had been training for the upcoming cross-country season and was averaging 112.65 kg (70 miles) per week. She had no symptoms before the half marathon and continued this running routine after the race even though she was experiencing slight tightness in her low back region. There was no history of direct trauma or stress-related injuries.

The athlete gave a 3-year history of amenorrhea, anemia, low body weight, hypokalemia, and gastrointestinal bleeding that had been evaluated annually with a thorough history and laboratory tests by her family physician and subsequently by her collegiate team physician. During this time, she had been taking an oral contraceptive prescribed by her family physician as a regulatory measure to minimize potential bone loss due to her amenorrhea. The occasional gastrointestinal bleeding was

Address correspondence to David Klossner, PhD, ATC, Athletic Training Education, University of South Florida, PED 214, Tampa, FL 33620. E-mail address: klossner@tempest.coedu.usf.edu

reported to be a factor in reducing her hemoglobin to 8 g/100 mL the year before, resulting in anemia. Normal hemoglobin ranges for endurance runners have been reported at 12.0 to 15.5 g/100 mL.[5] An endoscopic evaluation was normal.

After the initial evaluation for her low back pain, the athlete was placed on a Medrol Dosepak (Pharmacia & Upjohn, Kalamazoo, MI) for a week while limiting her mileage. When she continued to have symptoms 1 week later, she was referred to an orthopaedic physician. He felt her presentation was consistent with an acute lumbosacral strain and recommended a treatment plan of eliminating land running for 1 week, beginning aqua jogging, and taking Daypro (GD Searle & Co, Chicago, IL), a nonsteroidal anti-inflammatory medication, once the Medrol was completed. The pain subsided within 1 week, and the athlete returned to her normal running routine. Two weeks later, the athlete reported for the preseason physical examination and stated that her symptoms had returned with more intensity than the previous episode, this time affecting her running and daily living activities.

Physical examination by the team physician revealed localized tenderness over the right sacroiliac joint and gluteal region without radiating pain. Pain was associated with forward trunk flexion and extension, unilateral right straight-leg raise, and standing right hip flexion. Plain radiographs of the pelvis and lumbar spine were unremarkable (Figure 1). After discussion with the athlete's family physician, the team physician suspected a right sacroiliac joint sprain and established a new treatment plan of 1 week of iontophoresis, swimming, rehabilitation exercises, and the continuation of anti-inflammatory medication.

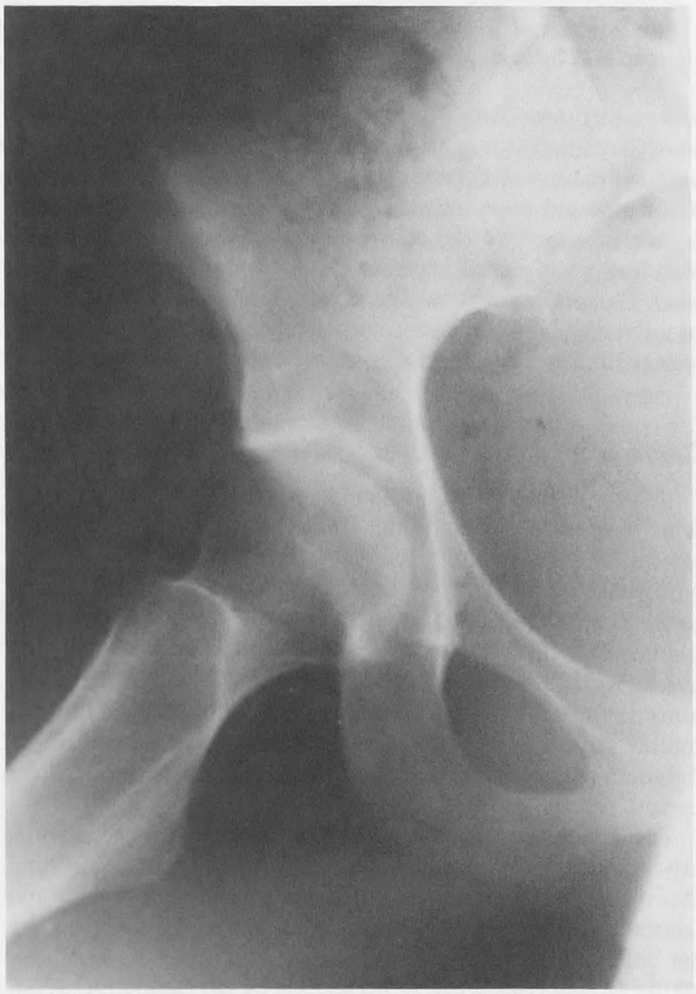


Figure 1. Normal plain radiograph of the pelvis (4 weeks after onset of symptoms).

The patient experienced minimal improvement with the change in treatment protocol. During the fifth week of continued discomfort, she underwent a bone scan, which showed focal increased activity centered about the right sacral ala (Figure 2). A computed tomography scan revealed abnormal sclerosis in the right sacral ala, consistent with a healing stress fracture (Figure 3). The final diagnosis was a unilateral stress fracture of the right sacral ala. High mileage, inadequate recovery after training sessions, insufficient caloric intake, anemia, and amenorrhea were thought to be contributing factors.

The athlete discontinued land running for 6 weeks while continuing to train aerobically. After the first week of complete rest, she began multiple daily workout sessions incorporating swimming, aqua jogging, bicycling, and stationary cross-country skiing within pain-free ranges for 2 to 3 hours a day. She continued to improve and was pain free by the fourth week of recovery (9 weeks after symptom onset). The athlete began running 25 miles per week at low intensity and pursued a strength-training program. She increased her distance by 5 miles per week until she reached 65 miles per week. There was slight tightness in her low back region during the first 2 weeks of her land-running routine, but this cleared within a week. The athlete competed successfully in a 5000-m run by 5 months after the onset of symptoms and returned as a top collegiate 10 000-m runner at 9 months.

Ten months after the sacral fracture, the family physician conducted a routine follow-up evaluation to determine whether additional medical management was necessary. The physician determined that the athlete had decided to stop her current contraceptive hormonal therapy program; she also admitted to limited food-intake practices. Because of the athlete's disordered eating, previous bone-related injury, and amenorrheic history, her estradiol level was tested. Although it was below the laboratory's detection level of 20 pg/mL, she had no signs

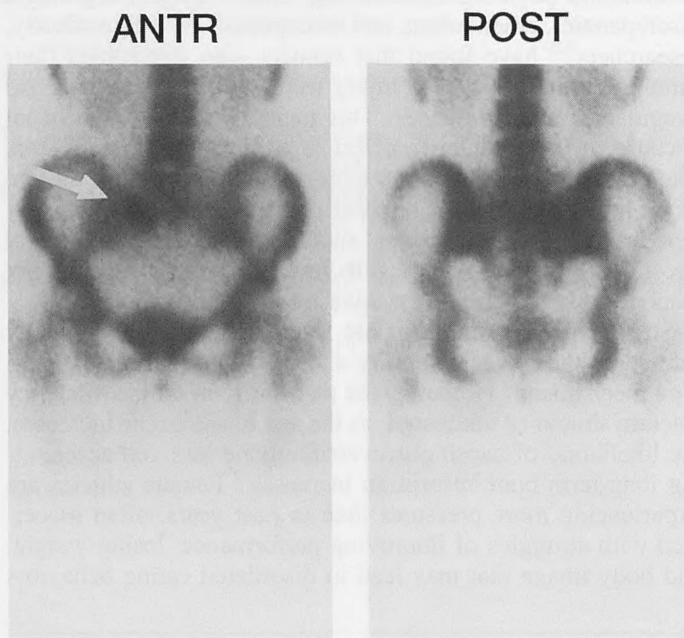


Figure 2. Bone scan showing increased activity about the right sacral ala (5 weeks after onset of symptoms).

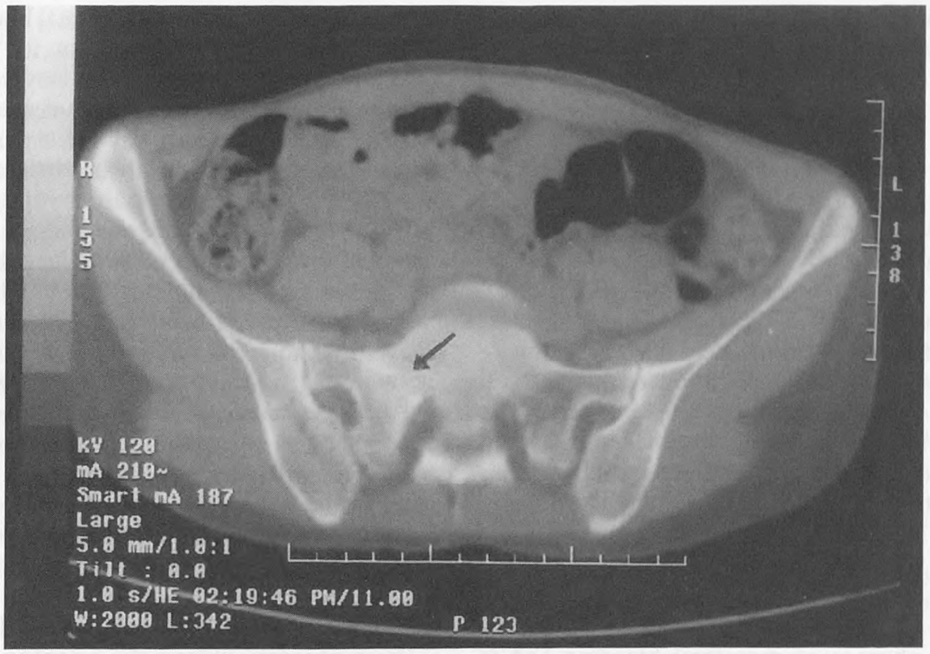


Figure 3. Computed tomography scan revealing abnormal sclerosis in the right sacral ala (5 weeks after onset of symptoms).

of metabolic bone disease on dual-energy x-ray absorptiometry. She did have moderate osteopenia at the lumbar spine, with less severe osteopenia noted at the hip. This finding was consistent with the cancellous bone loss often associated with hypoestrogenemia.

Her risk for a future fracture was increased as a result of her aggressive exercise regimen and irregular menses, so she was placed on Triphasil (Wyeth-Ayerst Laboratories, Philadelphia, PA), an estrogen replacement therapy medication, during the subsequent offseason. The physiologic conditions and behavioral patterns described in this case could contribute to increasing the athlete's risk of another stress-related injury if future hormonal therapy and dietary goals were not achieved as recommended.

DISCUSSION

Sacral stress fractures are rare occurrences in young females but have been reported more often in the literature over the last 2 years.[2] The etiology of sacral stress fractures in young individuals has been related to a variety of factors, such as inappropriate mileage progression, shoe changes, poor biomechanics, changes in running surface, amenorrhea, caloric restrictions, low bone mass, mechanical fatigue, and low estrogen levels.[2,4,11–14] Many of these factors were applicable to this patient, who presented with other common problems often seen in competitive female runners. Moreover, a low or inadequate energy intake has been linked to hormonal fluctuations, the development of amenorrhea, increased bone loss, and eventual early osteoporosis. In the vigorously exercising female athlete, these factors have been shown to increase the risk of musculoskeletal injury and stress-related fractures.[3,8,9,15] Addressing all these components collectively should provide insight into the complex physiologic and behavioral characteristics associated with female runners and their potential risk for injury.

Gastrointestinal Bleeding

Long-distance runners can present with gastrointestinal bleeding occurring after high-mileage or intense running activities. This sign was significant in this patient and is also important to understanding the complexity of the female athlete triad. Causes of this condition in distance runners include intestinal tract ischemia as a result of diversion of the splanchnic circulation and stress from the hyperthermic conditions of prolonged, intense exercise.[16] These combined etiologies may increase the risk for developing ischemic necrosis, which would present as tarry stools and bloody diarrhea. Continuous bleeding should be evaluated with an endoscopic procedure to rule out other conditions.

Gastrointestinal bleeding does not typically affect an athlete's performance directly but can be a causative factor in the development of anemia.[16] Anemia can lead to performance fatigue, to which the athlete may respond by decreasing caloric intake in order to lose weight and increase speed. Rosen et al[17] concluded that female athletes often lower their body weight in order to achieve the highest level of performance instead of supporting physical appearance objectives. This cyclic phenomenon focusing on improving performance with little regard for physiologic and behavioral factors can be detrimental to an athlete's health and often continues throughout an athlete's career, establishing a disordered eating behavior. Even though anemia does not directly relate to injury rates, it may indirectly affect an athlete's eating behavior and contribute to the female athlete triad.

Female Athlete Triad

Although mechanical stress and exercise intensity were significant contributors to this patient's stress fracture, disordered eating, amenorrhea, osteopenia, and decreased estradiol levels were also factors. These factors have been associated with the female athlete triad (disordered eating, amenorrhea, and decreased bone mineral density), which has been linked to an increased prevalence of skeletal injuries.[3,8,10,18–20]

Disordered Eating

Runners, who are often driven to maintain a low body weight for performance purposes while attempting to increase workloads, may develop disordered eating patterns. Athletes often control their caloric intake by practicing such behaviors as using diet pills, laxatives, and diuretics; vomiting; and fasting.[9] These behavioral patterns can limit caloric intake enough to create a negative energy balance.[19] Despite these behaviors, many athletes fail to associate their energy expenditure with caloric intake, thereby underestimating their daily energy needs. The insufficient caloric intake for the energy expenditure not only causes physical fatigue and poor performances but has been associated with menstrual dysfunction and, ultimately, early osteoporosis in female athletes.[8–10,17,19,21]

Amenorrhea

Amenorrhea is the absence of menstrual bleeding and is often accepted as a favorable condition by female athletes. Recent research[3,8,9,18,21] has suggested that amenorrheic athletes can develop significant bone density losses, musculoskeletal injuries, and an increased risk for the early onset of osteoporosis. Many variables can contribute to athletic amenorrhea, but the influence of each factor has yet to be completely understood. One theory[9,19] proposes that the intricately related components of the female athlete triad may have a direct relationship on the function of the hypothalamus and regulation of estrogen levels. Excessive exercise, limited caloric intake, and psychological stress may decrease the frequency of gonadotrophic-releasing hormone pulses secreted by the hypothalamus and limit the availability of the reproductive hormone estrogen. These decreased hormone levels can lead to amenorrhea in many female runners, increasing their chances for bone resorption.[9,10,18,22] Previous research[8,9,22] suggests that estrogen levels in runners may be inversely related to training volume or to the factors associated with increasing an exercise regime, such as energy balance. Estradiol levels in women running 48.28 km (30 miles) and 80.47 km (50 miles) per week have been reported at 54 and 34 pg/mL, respectively.[8] This athlete ran 112.65 km (70 miles) per week and had an estradiol level of less than 20 pg/mL. Although mileage should not be directly linked to estrogen production, athletes who run high mileage should have their estradiol levels evaluated and be screened for all components of the female athlete triad to reduce their risk of injury.[3,8,9]

Bone Mineral Density

Although this athlete presented physiologic characteristics that may represent an insufficiency-type, stress-related injury, her young age and the evidence of intense exercise routines suggested the diagnosis of a fatigue-type stress fracture. Female athletes have not been reported to have insufficiency-type injuries at such a young age, but in the presence of amenorrhea and moderate osteopenia, insufficiency injuries should not be overlooked. While insufficiency-type fractures have been related to limited estradiol levels and osteoporosis in elderly females, fatigue-type stress fractures are common in athletes.[9,23] This type of fracture is classified by an etiology of repetitive or unusual stress on normal bone that has not adapted to the abnormal forces placed upon it.[23]

For many female athletes, bone loss is not a concern because research has shown that exercise can actually increase bone density.[9] However, recent studies supported decreased bone mineral density (BMD) in amenorrheic athletes despite regular exercise.[2,3,8,15,24] More significantly, amenorrheic women have presented with larger numbers of running-related stress fractures.[25] Interestingly, researchers have found that total bone density is often not affected in athletic females because exercise protects cortical bone, which makes up 80% of the skeletal system.[9] The remaining 20% of bone is composed of the more metabolically active trabecular bone. This is significant because trabecular bone can be more sensitive to hormonal fluctuations, resulting in an earlier onset of natural bone loss.[26] Trabecular bone is concentrated in the pelvis but also found in flat bones, the ends of long bones, and in vertebral bodies.[9] Consequently, Rencken et al[3] found that athletes with amenorrhea had significantly lower BMD at the lumbar spine, femoral neck, greater trochanter, Ward triangle, intertrochlear region, and tibia as compared with healthy athletes.

These concepts support the presence of active bone loss in female runners without classifying them as osteoporotic. More often, athletes will present signs of osteopenia or BMD less than 80% of peak bone mass for their age.[3,9,15,18] Research has suggested that if normal menses do not return, the trabecular bone loss may be irreversible, leading to early normal bone loss, especially if the initiation of menarche was delayed until the late teenage years.[9,21,24] This irreversible bone loss has been shown to occur even after resumption of menses, adequate estrogen replacement, and calcium supplementation.[22,27,28]

Diagnostic Imaging

The evaluation of BMD is suggested to be best analyzed with dual-energy roentgenogram absorptiometry.[29] A benefit of this technology allows the clinician to observe the BMD at specific sites and to analyze the density over time.[3,9,20]

Individuals with sacral stress fractures often present with clinical sacroiliac joint pain and are frequently referred for manipulation, which could exacerbate this particular injury. Various imaging techniques can be used to evaluate the affected area for a stress-related lesion.[2,30] Initial radiographs are typically normal but are important to assess. Radionuclide bone scans are sensitive to acute and healing bone injuries; therefore, they are helpful in determining the presence of abnormal bone activity but lack the detail needed to determine any structural compromises.[2] Magnetic resonance imaging and computed tomography scans have been useful in determining the significance of stress fractures because they localize the lesions and help rule out sacroiliac joint problems.[2,30,31]

Management

Once the stress fracture is diagnosed and other related problems addressed, the athlete may begin the return to activity with a progressive rehabilitation protocol emphasizing an active-rest, cross-training regime.[31–33] In this case, the active-rest routine involved eliminating land running and replacing workouts with aqua jogging, biking, swimming, and stationary cross-country skiing sessions for multiple 1-hour intervals.

Management of amenorrheic athletes through education and counseling, reduced training levels by 5%, increased caloric intake, appropriate calcium intake (1500 mg/day), and hormonal replacement is vitally important in cases associated with females presenting with signs and symptoms of the female athlete triad.[4,8,10,29,34,35] The initiation of these guidelines could be enough to return normal menses in most female athletes and improve normal hormonal balances.[19,28,35] However, continued low estrogen levels can lead to early osteoporosis and additional stress-related injuries.[3,4,9] Estrogen replacement therapy and regular oral contraceptive use can limit bone loss and decrease the frequency of skeletal injury.[1,8,35,36]

Most athletes with sacral stress fractures fully recover within 6 weeks and continue running without further symptoms.[2,11,12] Follow-up magnetic resonance imaging and computed tomography scans can be performed to assess the degree of healing, although the lesions may remain visible for months. Many competitive female distance runners average 40 to 50 miles a week and may return to activity much sooner than this patient did. However, due to individual variances in mileage, this patient pursued an incremental running program that increased weekly distance by 5 miles until the eighth week, at which time she was averaging 65 miles. Previous return to land running has ranged from 3 to 8 weeks after initial diagnosis of a sacral stress fracture.[2,11,12,31]

CONCLUSIONS

Sacral stress fractures should be considered in runners with persistent low back pain, especially if injuries are not responding to appropriate treatment protocols. No single variable can be linked to the risk of injuries or the female athlete triad; however, more research has supported the influence of energy balance and hormonal fluctuations as significant factors associated with injuries in amenorrheic female athletes. A rehabilitation program that addresses the behavioral patterns and physiologic characteristics of such a problem will benefit the whole athlete and lead to a full recovery. Prevention strategies encouraging the distribution of questionnaires during preseason physicals and establishing policies to screen women who present with 1 component of the female athlete triad for other components may help to decrease injuries and promote healthier lifestyles among this population.

REFERENCES

1. Carbon R, Sambrook P, Deakin V, et al. Bone density of elite female athletes with stress fracture. *Med J Aust.* 1990;153:373–376.
2. Eller DJ, Katz DS, Bergman AG, Fredericson M, Beaulieu CF. Sacral fractures in long-distance runners. *Clin J Sport Med.* 1997;7:222–225.
3. Rencken ML, Chesnut CH 3rd, Drinkwater BL. Bone density at multiple skeletal sites in amenorrheic athletes. *JAMA.* 1996;276:238–240.
4. Wilson JH, Wolman RL. Osteoporosis and fracture complications in an amenorrhoeic athlete. *Br J Rheumatol.* 1994;33:480–481.
5. Clement DB, Asmundson RC. Nutritional intake and hematological parameters in endurance runners. *Physician Sportsmed.* 1982;10(3):37–43.
6. Fink-Bennett DM, Benson MT. Unusual exercise-related stress fractures: two case reports. *Clin Nucl Med.* 1984;8:430–434.
7. Noakes TD, van Gend M. Menstrual dysfunction in female athletes: a review for clinicians. *S Afr Med J.* 1988;73:350–355.
8. Lloyd T, Triantafyllou SJ, Baker ER, et al. Women athletes with menstrual irregularity have increased musculoskeletal injuries. *Med Sci Sports Exerc.* 1986;18:374–379.
9. Fruth SJ, Worrell TW. Factors associated with menstrual irregularities and decreased bone mineral density in female athletes. *J Orthop Sports Phys Ther.* 1995;22:26–38.
10. Otis CL, Drinkwater B, Johnson M, Loucks A, Wilmore J. American College of Sports Medicine position stand: the female athlete triad. *Med Sci Sports Exerc.* 1997;29(5):i–ix.
11. Atwell EA, Jackson DW. Stress fractures of the sacrum in runners: two case reports. *Am J Sports Med.* 1991;19:531–533.
12. McFarland EG, Giangarra C. Sacral stress fractures in athletes. *Clin Orthop.* 1996;329:240–243.
13. Rutherford OM. Spine and total body bone mineral density in amenorrheic endurance athletes. *J Appl Physiol.* 1993;74:2904–2908.
14. Schils J, Hauzeur JP. Stress fracture of the sacrum. *Am J Sports Med.* 1992;20:769–770.
15. Millar AL, Hunter D. Osteopenia: its relation to menstrual disorders in female athletes. *J Orthop Sports Phys Ther.* 1990;11:351–354.
16. Carlson DL, Mawdsley RH. Sports anemia: a review of the literature. *Am J Sports Med.* 1986;14:109–112.
17. Rosen LW, McKeag DB, Hough DO, Curley V. Pathogenic weight-control behavior in female athletes. *Physician Sportmed.* 1986;14(1):79–86.
18. West RV. The female athlete: the triad of disordered eating, amenorrhoea and osteoporosis. *Sports Med.* 1998;26:63–71.
19. Loucks AB. Effects of exercise training on the menstrual cycle: existence and mechanisms. *Med Sci Sports Exerc.* 1990;22:275–280.
20. Myburgh KH, Hutchins J, Fataar AB, Hough SF, Noakes TD. Low bone density is an etiologic factor for stress fractures in athletes. *Ann Intern Med.* 1990;113:754–759.
21. Gulekli B, Davies MC, Jacobs HS. Effect of treatment on established osteoporosis in young women with amenorrhoea. *Clin Endocrinol (Oxf).* 1994;41:275–281.
22. Loucks AB, Laughlin GA, Mortola JF, Girton L, Nelson JC, Yen SSC. Hypothalamic-pituitary-thyroidal function in eumenorrheic and amenorrheic athletes. *J Clin Endocrinol Metab.* 1992;75:534–518.
23. Daffner RH, Pavlov H. Stress fractures: current concepts. *AJR Am J Roentgenol.* 1992;159:245–252.
24. Cann CE, Martin MC, Genant HK, Jaffe RB. Decreased spinal mineral content in amenorrheic women. *JAMA.* 1984;251:626–629.
25. Jones KP, Ravnikar VA, Tulchinsky D, Schiff I. Comparison of bone density in amenorrheic women due to athletics, weight loss, and premature menopause. *Obstet Gynecol.* 1985;66:5–8.
26. Carbon RJ. Exercise, amenorrhea and the skeleton. *Br Med Bull.* 1992; 48:546–559.
27. Drinkwater BL, Bruemer B, Chesnut CH 3rd. Menstrual history as a determinant of current bone density in young athletes. *JAMA.* 1990;263: 545–548.
28. Drinkwater BL, Nilson K, Ott S, Chesnut CH 3rd. Bone mineral density after resumption of menses in amenorrheic athletes. *JAMA.* 1986;256: 380–382.
29. Dueck CA, Matt KS, Manore MM, Skinner JS. Treatment of athletic amenorrhea with a diet and training intervention program. *Int J Sport Nutr.* 1996;6:24–40.
30. Ries T. Detection of osteoporotic sacral fractures with radionuclides. *Radiology.* 1983;146:783–785.
31. Brukner P, Bennell K. Stress fractures in female athletes: diagnosis, management and rehabilitation. *Sports Med.* 1997;24:419–429.
32. Eyestone ED. *Effect of Water Running and Cycling on VO_{2max} and 2-Mile Performance* [thesis]. Salt Lake City, UT: Brigham Young University; 1990.
33. Wilber RL, Moffatt RJ, Scott BE, Lee DT, Cucuzzo NA. Influence of water run training on the maintenance of aerobic performance. *Med Sci Sports Exerc.* 1996;28:1056–1062.
34. Yeager KK, Agostini R, Nattiv A, Drinkwater B. The female athlete triad: disordered eating, amenorrhea, osteoporosis. *Med Sci Sports Exerc.* 1993;25:775–777.
35. Fagan KM. Pharmacologic management of athletic amenorrhea. *Clin Sports Med.* 1998;17:327–341.
36. Barrow GW, Saha S. Menstrual irregularity and stress fractures in collegiate female distance runners. *Am J Sports Med.* 1988;16:209–216.

Journal of Athletic Training 2000;35(4):458–465

www.journalofathletictraining.org

Psychology/Counseling: A Universal Competency in Athletic Training

Joni L. Cramer Roh, MAT, ATC*; Frank M. Perna, EdD†

*California University of Pennsylvania, California, PA; †Boston University School of Medicine, Boston, MA

Objective: To present the rationale that certified athletic trainers (ATCs) may require structured educational training in the psychological aspects of athletic injury.

Data Sources: We searched MEDLINE, Psych Lit, PsycINFO, First Search, and SPORT Discus databases for the years 1990 through 1999 under the search terms "psychological distress," "depression," "athletic injury," and "rehabilitation adherence."

Data Synthesis: Psychological factors are significant predictors of athletic injury. Athletic injury is accompanied by significant psychological distress, which has been shown to impair rehabilitation compliance and possibly physical recovery. Although "counseling" and knowledge of psychological aspects of injury are required National Athletic Trainers' Association competencies, extant data suggest that athletic trainers may lack training in this competency.

Conclusions: Evidence suggests that (1) psychological distress is prospectively associated with the incidence of athletic injury, and prolonged psychological distress, specifically depression, may occur after athletic injury; (2) psychological factors may also either hinder or facilitate rehabilitation adherence, compliance, and recovery; (3) psychological distress may persist even after physical recovery has been completed; (4) psychosocial factors related to injury occurrence and injury recovery may be overlooked by ATCs, but knowledge of these factors and appropriate use of referral sources may enhance the effectiveness of ATCs; and (5) ATCs may benefit from structured educational experiences specific to the National Athletic Trainers' Association psychology/counseling competency.

Recommendations: With 75% of a national survey of ATCs indicating that they do not have access to a sport psychologist, it would be advantageous for ATCs to gain adequate training in the recognition, evaluation, and treatment of psychological factors associated with athletic injury. The literature also suggests that structured educational training with respect to psychological aspects of athletic injury would be well received by ATCs.

Key Words: athletic injury, psychological distress, psychological training, rehabilitation adherence, rehabilitation compliance

It has been reported that nearly 1 in 6 athletes in the United States have sustained an athletic injury severe enough to keep them out of activity.[1] When athletes become injured, they require immediate treatment and rehabilitation in order to speed return to participation. As part of treatment or rehabilitation, the athlete and the injury must be properly assessed. Understandably, physical assessment has been viewed as being of primary importance. However, the importance of evaluating injured athletes' psychological distress, coping, and concerns regarding return to play have gained increased attention.[2,3] Additionally, psychological and behavioral factors have been identified as playing significant roles in the occurrence of athletic injury and in the physical adaptations to exercise training.[4–6]

Because certified athletic trainers (ATCs) often have the most frequent contact with an athlete before and after an injury, they are in a unique position to monitor the athlete's physical and mental status and to make timely referrals.[7] Furthermore, the National Athletic Trainers' Association (NATA), on the basis of a Role Delineation Study,[8] requires ATCs to be competent in psychology and counseling. Specifically, ATCs are expected to be able to identify psychological distress, counsel athletes, and make counseling referrals as appropriate.[8–10]

Address correspondence to Joni L. Cramer Roh, MAT, ATC, Health Science & Sport Studies Department, California University of Pennsylvania, California, PA 15419. E-mail address: ROH@CUP.edu

In support of the position that ATCs observe significant psychological distress, a national survey[11] revealed that 47% of ATCs believed athletic injuries affected athletes both psychologically and physiologically, and most believed that psychological factors needed to be addressed along with physical functioning during rehabilitation. In addition, 71% of ATCs reported that athletes commonly encountered stress and anxiety. However, only 23.9% of ATCs reported at least 1 counseling referral due to athletic injury.[11] The purpose of our article is to present the rationale that ATCs may require structured educational training in the psychological aspects of athletic injury. This position is premised by the following supportive arguments. Psychological factors are significant predictors of athletic injury. Athletic injury is accompanied by significant psychological distress, which has been shown to impair rehabilitation compliance and possibly physical recovery. Although counseling and knowledge of psychological aspects of injury are required competencies, extant data suggest that ATCs may lack training in this competency. We will address the importance of properly educating ATCs regarding psychological factors related to athletic injury.

PSYCHOLOGICAL PREDICTORS OF ATHLETIC INJURY

Regardless of sex, an athlete has a 50% chance of becoming injured,[12,13] which rises to as high as 86% among high school football players.[14] Furthermore, in any single year, 1 in 6 athletes is likely to sustain an athletic injury serious enough to miss athletic participation.[1]

Psychological factors may predispose some athletes to athletic injury.[6,15,16] Studies with nonathletes dating back to the 1950s have provided evidence that the onset of illness was significantly associated with an increase in the number of life-event stressors.[17,18] These early findings spurred investigation into whether a similar pattern linking stress to injury was noticeable in athletic populations.[19] During the late 1980s, Andersen and Williams[20] summarized the literature and proposed a model to explain the mechanisms thought to be responsible for the stress and athletic injury relationship. Based on theory and previously reported studies, Andersen and Williams[20] suggested that psychosocial variables (eg, life events, daily hassles, coping) could be identified that prospectively predicted athletic injury.[6,21] The Andersen and Williams[20] model primarily focused on the mechanisms by which psychological stress may contribute to acute, rather than chronic, injury. Although Andersen and Williams[20] and others[22] have proposed that psychological precursors and reactions to athletic injuries may vary for chronic injuries, extant data suggest that the model is applicable to chronic injuries and exercise training-related illness.[5,23]

In general, nearly 80% of the reported studies have identified psychosocial variables, such as life-event stress, as linked with the occurrence of athletic injury.[19,21,24] The first studies to show a relationship between life-event change scores and injury frequency were limited to male football athletes.[19,21,24–27] During the mid 1980s and early 1990s, sample diversity was increased, and research design rigor was enhanced. For example, prospective designs became the norm, and the search for the psychological stress-injury relationship was extended to include female athletes and participants in a variety of sports.[24,28–31] Those results corroborated findings from earlier research indicating a positive association between life stress and the occurrence of athletic injury. Additionally, sex differences were noted.[32] Total life-change and negative life-change scores were significantly greater for male athletes than for uninjured female athletes, and uninjured male athletes reported greater positive life-change scores than did uninjured female athletes. In general, negative life-change scores were found to be significant predictors of athletic injury severity and frequency, whereas total life-change scores were positively related to injury frequency only among female athletes.

Researchers began to investigate moderator variables (eg, social support and playing status) that may influence the stress-injury link.[27,33,34] Petrie[27] specifically evaluated injury severity and the stress-injury link using social support and playing status as moderator variables. Starting football players who reported high social-support scores showed lower negative life-stress scores and less severe injuries, whereas starting football players who reported low social-support scores showed more negative life-stress scores and more severe injuries. Thus, social support and playing status did provide moderating effects on the negative life stress.[27] Furthermore, social support buffered the effect of stress on the relative rate of injury. Although many studies have indicated that life stress is positively associated with athletic injury, and social support may moderate this effect,[19,26–28,32,34] only 1 published study[27] of football athletes found that social support was directly inversely associated with injury.

As mentioned above, several studies have clearly indicated that the incidence of injuries can be predicted by stress and social support. However, few researchers have attempted to use psychological factors to predict injury severity. Hanson et al[33] reported that as negative life stress increased, the severity of injury worsened. In accordance with previous research findings,[19,25–28,32] Hanson et al[33] also found that life stress was an important predictor of injury for contact and noncontact athletes.

Other psychological factors that are believed to contribute to the stress-injury relationship are personality variables such as hardiness, locus of control, and competitive trait anxiety.[21] The personality variables used to predict athletic injury have not received as much attention as the psychosocial variables, and few studies have found that personality factors directly or indirectly influence injury.[29] For example, Hamilton et al[30] noted that personality traits differed between male and female dancers, but personality traits were not associated with injury. In contrast, Passer and Seese[26] evaluated general trait anxiety, competitive trait anxiety, and locus of control as moderator variables in 104 male football athletes. Negative life-change scores were significantly higher for injured athletes than for uninjured athletes. Further, a significant relationship between negative life-change scores and injury was found for male football players with low competitive-trait anxiety. Unfortunately, the research examining the relationship between personality factors and injury has been plagued by questionable methods.[30,31] Although theory supports the potential contribution of personality traits to explain athletic injury variance,[35] studies have not empirically confirmed the contribution of personality factors. Therefore, the most accepted explanation of the stress-injury relationship concerns psychosocial variables such as life stress, social support, and coping. Considering that negative life stress may increase the risk of injury by 3- to 5-fold,[6] it may behoove ATCs to become aware of the life stressors that routinely affect athletes and the social support and coping responses that may alter the stress-injury relationship.

PSYCHOPHYSIOLOGIC MECHANISMS UNDERPINNING THE STRESS-INJURY LINK

Andersen and Williams[20] and Williams and Andersen[6] have proposed that physiologic (eg, increased muscle tension and narrowing of visual field) and attentional (eg, increased distractibility) aspects of the stress response are possible underlying manifestations of stress that increase susceptibility to injury. Few studies have addressed the mechanistic propositions offered in the Andersen and Williams[20] model. However, the studies that have been conducted support the model. For example, in an experimental manipulation, Williams and colleagues[6,36,37] demonstrated that participants with high life stress experienced significant peripheral narrowing as compared with low-stress controls. Life stress may predispose an athlete to peripheral narrowing, which may increase the likelihood of missing important environmental cues, in turn increasing susceptibility to injury. Perna et al[4,5] have also reported relatively greater attentional disturbances, physical symptoms, and sleep difficulties among high life-stress athletes as compared with low life-stress athletes. Additionally, athletes with relatively high life stress, in comparison with low-stress athletes of similar ability, exhibited higher prolonged cortisol elevations with exhaustive exercise, indicative of relatively poorer exercise recovery.[5] Elevated levels of cortisol, the primary catabolic hormone after intense exercise, were also related to subsequent physical symptoms (eg, skeletal muscle pain). Together these studies support the proposition that stress-induced changes in attention, physiology, and behavior may all mediate the stress-athletic injury relationship.

PSYCHOLOGICAL AND PHYSIOLOGIC RESPONSES TO ATHLETIC INJURY

Once an athlete becomes injured, both physiologic and psychological processes occur. Physiologically, a vicious pain-spasm-pain cycle will continue, causing further damage, if appropriate care is not provided.[38] Furthermore, many physiologic changes that occur during psychological stress may impair recovery.[20,39] For example, Nideffer[40] suggested that increased muscle tension, heart rate, blood pressure, and skin conductance, all indicative of autonomic nervous system (ANS) activity, are present after injury. Also, attentional changes (ie, worry about self) that occur after athletic injury may cause further generalized muscle tension, which in turn may result in further musculoskeletal injuries from disturbances in fine motor coordination and reduced joint flexibility.[21,40] Prolonged distress accompanying an injury may also lead to continual ANS arousal (eg, epinephrine, norepinephrine, and cortisol release) that may prolong recovery by impairing immune functioning and skeletal muscle repair.[5,41] The following section highlights both psychological and physiologic concerns.

Psychological Sequelae of Athletic Injury

In addition to pain, significant increases in negative mood states after athletic injury have been well documented in a variety of populations. For example, in a preinjury-postinjury design, Smith et al[42] evaluated 238 male and female high school, college, junior hockey league, and minor hockey league athletes. The injured athletes reported significant postinjury increases in depression and anger and decreases in vigor. Comparing injured with uninjured college athletes, Petrie et al[43] also reported higher levels of depression and anxiety among injured athletes. These studies document a general increase in negative affect after athletic injury. Injury-induced increases in negative affect have been reported for male and female athletes competing in contact and noncontact sports at various competitive levels, and a large majority (90%) of studies indicate significant elevations in emotional distress associated with athletic injury.[30,42–51]

Similarly, studies evaluating mood disturbance at various points after athletic injury have noted primarily negative emotions.[31,47,52–54] Anger and depression are the most commonly reported negative mood states.[44,46,48,50,54–56] With respect to clinical depression, Perna et al[57] reported that approximately 26% of injured athletes had depression scores reaching clinical magnitude at 1 week postinjury in comparison with only 4.4% of uninjured athletes. Furthermore, in a prospective longitudinal study of NCAA Division I and II college athletes, Roh et al[48] reported that in comparison with uninjured controls, depression was significantly higher among injured athletes 1 week after injury and remained elevated at 1 month postinjury. It is also important to note that depressed mood at preseason was not significantly different between athlete controls and athletes who eventually became injured.

McDonald and Hardy[56] evaluated athletes' mood states and perceived rehabilitation in a longitudinal study. Elevated negative moods occurred at 24 hours postinjury and decreased gradually by 4 weeks postinjury, whereas vigor was lower at 24 hours postinjury and increased gradually by 4 weeks postinjury. However, the sample was limited to 5 severely injured (out for at least 3 weeks) male and female NCAA Division I athletes. In comparison, Leddy et al[46] reported that the injured groups had significantly higher levels of depression when compared with the uninjured or recovered groups of athletes at 2-month follow-up.

Other psychological responses of injury include features of acute traumatic psychological stress, such as intrusive thoughts and avoidance behaviors, that have been found in both acutely and chronically injured athletes.[58,59] That is, despite being medically cleared for participation, injured athletes may exhibit pronounced psychological distress and worry for up to 1 year postinjury.[58]

Physiologic Components of Injury and Pain

When an injury occurs, the natural healing process begins in a cyclical pattern. First, cells die (necrosis) from primary trauma. These cells release their contents into the adjacent area, causing an inflammatory reaction. Cell death continues as the hemorrhaging and edema block the oxygen supply to healthy tissues.[38] The body proceeds through a natural healing process to inhibit further damage.

Basically, 3 phases of physiologic healing have been identified: (1) the acute inflammatory response phase, (2) the proliferation phase, and (3) the remodeling (maturation) phase.[38,60] In general, the inflammatory response mobilizes the body's immune system and chemotaxis of immune cells to the site of injury. In the proliferation phase, the immune cells remove damaged tissue (eg, necrotic cells), and in the remodeling (maturation) phase, regeneration and strengthening occur as collagen fibers form scar tissue.

Through the actions of the ANS and the hypothalamic-pituitary axis, psychological distress may impair physical recovery. For example, psychological distress in the form of depression and anxiety are well known to increase ANS activity and impair immune function, which in turn may disrupt physical repair processes.[61] Furthermore, distress-induced increases in catecholamines and glucocorticoids may impair the chemotaxis of immune cells to the site of injury, as well as impair clearance of damaged tissue.[5,61,62] Prolonged elevation in stress hormones may also inhibit anabolic processes by decreasing the actions of growth hormone and insulin-like growth factors that are essential during the remodeling phase.[5,61,63]

During the secondary response to injury, a subcycle of pain, spasm, and atrophy may occur, causing further cell death, inflammation, hemorrhaging, and edema. The entire cyclical process has been termed the injury response cycle, the pain-spasm-pain cycle, or the vicious cycle.[38] Pain and spasm within the vicious cycle may also be attributed to both physiologic and psychological responses.[20,40,60,64]

Pain has been described as a noxious stimulus to the human body, and it is influenced by motivational and cognitive factors that act on the brain when one becomes injured.[60,64] Although pain travels a specific afferent pathway to the brain, individuals tend to react to a stimulus differently, which accounts for the variety in reported discomfort that may arise from identical injuries. Although a thorough review of intervention studies is beyond the scope of this report, psychological interventions that augment somatic treatments have been shown to decrease pain reports, decrease emotional distress, and more importantly, increase functional quality of life.[61,65,66]

COMPLIANCE WITH REHABILITATION

Physiologic and psychological changes that occur with injury have been clearly documented. Because the rehabilitation process varies among individuals with the same injury, it is important for ATCs to identify both physiologic and psychological facets of athletic injury response in order to appropriately treat and rehabilitate athletes to their preinjury state.[67–70]

As Taylor and Taylor[71] have reported, the course of rehabilitation is not always consistent. Psychological factors may influence treatment compliance in several ways. Taylor and May[69] found that more than half of injured patients failed to comply to some degree with a rehabilitation program, and more than 200 variables covering multiple domains (eg, physiologic, medical, educational, and psychological) have been associated with rehabilitation compliance.[72,73] Taylor and Taylor[71] suggested that psychological interventions can be used to help with the recovery process. For example, confidence, motivation, and anxiety have been identified as 3 psychological factors that are either positively or negatively significantly related to adherence with and quality of rehabilitation.[3,71,74] Current research indicates that attentional focus may be a fourth psychological factor that is believed to aid in the rehabilitation and recovery process.[40,71] That is, if injured athletes are not distracted by internal or external cues, then they may be better able to increase their attention to rehabilitation tasks.

Taylor and Taylor[71] have indicated how all 4 psychological factors can be addressed throughout the phases of rehabilitation by using the following interventions: (1) motivation and team building, (2) goal setting, (3) imagery, (4) visualization, (5) mental training, (6) biofeedback, (7) self-talk, (8) attention control, (9) relaxation, and (10) stress inoculation training.[60,64,75–81] These psychological interventions may complement the typical therapeutic modalities ATCs use in the recovery process.[38,71] For example, a performance-enhancement, group-intervention program was developed to enhance the rehabilitation process by integrating sport psychology interventions and traditional sport medicine therapies through collaboration among professionals (eg, sport physicians, athletic trainers, sport psychologists, athletic department personnel).[82] Further empiric study is necessary to document the proposed effectiveness of psychological interventions to improve rehabilitation outcomes. However, in a well-designed, randomized, control study, a brief cognitive-behavioral intervention significantly improved physical and emotional outcomes among patients after meniscal surgery.[80]

In order to better target psychological interventions, the current focus of research has been to identify the links between the postinjury psychosocial variables and rehabilitation adherence and compliance.[56,74,83–88] Interestingly, several psychosocial variables (eg, social support, pain tolerance, self-motivation) have been identified by both therapists and injured athletes as important to rehabilitation compliance.[73,89–91] Social support, pain tolerance, and self-motivation have been positively associated with rehabilitation adherence.[2] Unfortunately, many studies have used retrospective designs that asked recovered athletes to identify relevant psychosocial factors rather than assessing psychosocial factors at the beginning of treatment and prospectively testing their association with rehabilitation compliance.

However, several methodologically strong studies demonstrated that the quality of social support is a predictor of compliance in the general medical population,[86] and social support has been inversely correlated with postinjury depression and positively associated with rehabilitation compliance in sport populations.[92,93] For example, Fisher et al[3] specifically evaluated the personal and situational factors related to rehabilitation compliance. The sample consisted of injured college athletes ($n = 41$), who were classified into 1 of 2 groups (adherent and nonadherent). The athletes were categorized by ATCs based on attendance at rehabilitation sessions and the comparison of expected progress and actual progress. Social support was significantly positively related to adherence based on the Rehabilitation Adherence Questionnaire (RAQ). Also using the RAQ, Byerly et al[92] examined 44 injured male and female athletes and found significantly lower levels of pain and significantly higher levels of social support among the adherent athlete group as compared with the nonadherent group. However, the RAQ has been shown to have serious psychometric shortcomings that call into question results from studies based on it.[94]

Other psychosocial variables correlated with rehabilitation adherence include coping strategies, personality traits, motivation, and perception of rehabilitation.[56,69,74,83–85,95–97] Cognitive-appraisal models have been used to provide a theoretical perspective to explain how perceptions of events (eg, injury), environment (eg, rehabilitation setting), and resources (eg, coping strategies) act in synergistic fashion to influence emotional states (eg, motivation) and behavior (eg, compliance/noncompliance). Person- and situation-related factors are thought to influence the appraisal process. Specifically, cognitive-appraisal models posit that when holding the type of injury constant, the perceived threat of injury to one's well being (primary appraisal) and perceived ability to cope (secondary appraisal) largely determine the consequent level of emotional distress. In turn, adaptive coping behavior (eg, setting rehabilitation goals, using social support) and maladaptive coping (eg, use of alcohol) largely determine the degree to which emotional distress subsides or remains constant. Negative emotional states may also impair self-regulatory behavior and harm initial rehabilitation compliance.[98,99] The consensus of studies suggests that cognitive-appraisal models provide the strongest evidence supporting the role of coping and social support factors that may influence both psychological responses to injury and subsequent rehabilitation adherence.[52,83,97–99] For example, in a 12-week prospective study, Udry[97] evaluated 25 injured athletes who sustained an anterior cruciate ligament injury requiring surgery. The intent of the investigation was to identify the degree to which social support and coping strategies predicted rehabilitation compliance among injured athletes. Instrumental coping was positively associated with adherence, whereas palliative coping was negatively associated with adherence.

In a similarly designed study, Daly et al[83] evaluated 31 male and female recreational and competitive athletes requiring arthroscopy or open knee surgery. Cognitive appraisal (perceived ability to cope with injury), mood disturbances, and adherence (attendance and compliance) were measured in a prospective design. Cognitive appraisal was significantly inversely associated with emotional disturbance in that low levels of perceived ability to cope with injury were associated with high levels of mood disturbance. Furthermore, mood disturbance was inversely related to rehabilitation session attendance. Although cognitive-appraisal models show promise and have been developed to explain the interrelationships among health effects, emotional distress, and treatment compliance, inconsistencies in the definition of compliance and the variety of measures used have hampered interpretation of study results. However, accumulated evidence does suggest that providing emotional support and fostering injured athletes' self-perceived coping abilities are counseling strategies that ATCs may use to improve rehabilitation compliance.

EDUCATION AND TRAINING IMPLICATIONS FOR ATCS

Many trained professionals, such as physicians, physical therapists, ATCs, and sport psychologists, have been working together to provide the necessary aids to support patients and prevent rehabilitative setbacks.[60,64,71] In 1993, the NATA completed the Role Delineation Study to develop a matrix of professional skills to be performed by the entry-level ATC[9,10]; the Study was updated in 1999. Ten specific competencies transcend the 6 major domains. One specific competency is psychology/counseling, which encompasses all 6 domains: (1) prevention of athletic injury; (2) recognition, evaluation, and assessment of athletic injury; (3) immediate care of athletic injury; (4) treatment, rehabilitation, and reconditioning of athletic injury; (5) organization and administration of athletic injury; and (6) professional development and responsibility.[8]

A debate exists as to whether ATCs believe they have achieved the required level of counseling competency.[11,100,101] For example, Larson et al[11] reported that nearly three fourths (71%) of nationally surveyed ATCs revealed an encounter with stress and anxiety among injured athletes. More than half (53%) noted that athletes experienced significant levels of emotional distress, and treatment compliance problems were common. Most ATCs (85%) reported that a course in sport psychology was "relatively important" or "very important" in the education of ATCs. Yet, only half (54.1%) of ATCs had taken a formal sport psychology course, and many felt unprepared to handle the counseling component. Although the Commission on Accreditation of Allied Health Education Professionals (CAAHEP) accreditation standards require formal instruction in psychology, an introductory psychology class contains neither a thorough review of psychopathology nor training in counseling and interviewing techniques. These data suggest that although ATCs frequently encounter injury-induced disruption to athletes' total well being, training in how to properly address mental health issues that accompany athletic injury is lacking.

Further, Moulton et al[100] found that ATCs are confronted with athletes' personal issues in addition to their athletic injury issues. Not surprisingly, only 36% of ATCs reported that they received adequate training in basic counseling skills, and 79% expressed a need for continuing education credits focusing on counseling issues. A total of 86% indicated that the sports medicine staffs were aware of the on-campus support services, and 71% reported referring athletes, which is appreciably higher than the referral rate (24%) reported in the Larson et al[11] study. However, the study by Moulton et al[100] was limited to 15 men and women ATCs employed in the NCAA Division I Southern conference, whereas Larson et al[11] surveyed a national sample of ATCs drawn from various geographic locations and employment settings. The referral rate data suggest that significant numbers of injured athletes with pronounced psychological distress are not referred for counseling. Although it is possible that ATCs are providing counseling to distressed athletes, survey data suggest that ATCs believe they are less well trained in counseling than in other competencies.

It is not surprising to find that ATCs do not feel qualified to counsel athletes or to properly identify the psychological components of injury. First, not all candidates who sit for the NATA certification examination are expected to take a formal course in psychology/counseling. Therefore, the students pursuing the internship route to certification may not even be exposed to a general psychology course. Further, students pursuing the internship route to certification have a significantly lower passing rate for all 3 components (ie, written, written simulation, and practical) of the NATA certification examination than students completing an accredited educational program to certification.[102] The finding that internship students have difficulty passing the examination in the areas of basic knowledge and skills of athletic training suggests that the psychological aspects of injury may be even more difficult to learn without organized instruction. Second, only 40% of the first-time candidates who take the NATA examination have graduated from a curriculum program, which suggests that most ATCs may not have been exposed to a psychology course.[102] In addition, not all ATCs enrolled in academic programs receive the same educational training in psychology before sitting for the NATA certification examination.[100] In fact, the formal instruction in psychology required by CAAHEP accreditation standards to fulfill the psychology/counseling universal competency may be obtained in courses not devoted to psychology. Therefore, evidence is minimal that ATCs routinely take a specific course in psychopathology or the psychology of injury. Finally, although 40% of NATA-certified athletic trainers have advanced degrees, there is little documentation that they received a graduate course related to psychology/counseling.[103]

DISCUSSION

The data indicate that psychological distress follows athletic injury and that injury severity is associated with prolonged psychological distress, including clinical depression.[48,51,57] In addition, psychological factors reliably predict the occurrence of injury and can alter rehabilitation adherence, compliance, and recovery.[2,71,75,83] Moreover, psychological distress may persist even after physical recovery has been completed.[58] Last, psychosocial factors related to injury and injury recovery can be overlooked by ATCs, but knowledge of these factors and of appropriate mental health referral sources may enhance the effectiveness of ATCs.[67]

Although many athletic trainers do not have formal courses in counseling, athletic trainers and other allied health professionals believe that athletic trainers are in an ideal position to facilitate psychological recovery from athletic injuries and to apply basic counseling skills (eg, active listening) and psychological principles (eg, goal setting) to facilitate rehabilitation.[7,11,104,105] However, many athletic trainers do not have formal courses in counseling, and the extant data clearly indicate that ATCs may benefit from additional psychology training. For example, the American Psychiatric Association identifies many features associated with psychological distress and a variety of clinical syndromes, such as depression, adjustment reactions, and anxiety disorders, that may be present among injured athletes.[106] ATCs who are not able to recognize many of these common features may not be as effective in their work as they could be. Although no court cases bear directly on the issue of professional negligence, presumably because ATCs work under the direction of physicians, any effort to gain independent treatment-provider status may place a greater legal burden on ATCs to satisfy NATA competency requirements. The psychology and counseling competency includes the recognition and appropriate disposition of pronounced psychological distress. Additionally, athletic trainers, and more importantly injured athletes, may benefit from the introduction of counseling techniques to complement somatic modalities routinely applied in sports medicine centers.[71]

However, there is very little evidence that ATCs believe they have the appropriate training for the psychology/counseling universal competency outlined by the NATA.[8] Furthermore, based on the *NATA Code of Ethics Membership Standards, Membership Sanctions and Procedures*,[107] ATCs are to provide services for which they are qualified, and they have an obligation to update their knowledge and skills commensurate with treatment advances in the field. The data clearly indicate the importance of structured educational training in the area of psychological aspects of athletic injury for ATCs.[100,108]

RECOMMENDATIONS

ATCs are professionals who have traditionally provided guidance and assistance to athletes, especially when injured, to function more effectively in practices, competitions, and day-to-day life. Therefore, the NATA has identified psychology/counseling as a competency.[8] Considering that three fourths of nationally surveyed ATCs indicated that they do not have access to a sport psychologist, it would be advantageous for ATCs to gain adequate training in the recognition and evaluation of psychological features that may be relevant to the onset of and response to athletic injury. After gaining this knowledge, ATCs may be in a better position to make referrals to mental health care providers and to use psychological principles to enhance treatment compliance.[11] Additionally, instruction in the systematic use of goal-setting and pain-management strategies are 2 cognitive-behavioral interventions ATCs may use with appropriate training under the supervision of a trained specialist. Although goal setting is commonly used in sports medicine clinics, its effectiveness may be enhanced by instruction in the latest methods. Also, pain usually hinders the rehabilitation process. If ATCs are able to help athletes learn pain-management skills, injured athletes may be able to use their time more effectively during rehabilitation and better tolerate the demands of day-to-day functioning. Teaching injured athletes to take a more active part in injury recovery may also lead to increased rehabilitation compliance. Methods to improve knowledge in this area exist.

Continuing education units (CEUs) in the form of coursework or workshops could be 1 method for ATCs to receive psychology training. Presently, ATCs are required to obtain 80 CEUs in a 2-year period, of which 55 units can be devoted to the psychology/counseling competency. NATA-approved workshops could be presented to groups of ATCs by professionals (eg, psychologists, counselors, ATCs) who have expertise regarding the psychological aspects of athletic injury.

A second recommendation is to include formal instruction in the psychology of injury or medical psychology within the athletic training curriculum. This method would be most beneficial to students currently enrolled in an accredited program. Gordon et al[108] proposed a 3-year psychoeducational curriculum for sport injury rehabilitation personnel. The proposed curriculum incorporates didactic teaching and practicum seminar instruction, including written examinations, role plays, video analysis, interviews, and goal setting. This curriculum is designed to "facilitate the engagement of clients in collaborative rehabilitation" and is presently used in a nursing and occupational therapy program at Curtin University in western Australia.[108] Although the program content is comprehensive, the curriculum would be difficult to incorporate into existing athletic training programs without employing new faculty.

Alternatively, a psychology of sport injury class could teach ATCs how to identify the psychological signs and symptoms indicative of maladjustment to injury or illness and how to implement psychological principles to facilitate rehabilitation compliance. The course may also cover basic training in the application of psychological principles and counseling skills. Lacking faculty with expertise to teach an entire psychology of sport injury course, a program could require a psychopathology or medical psychology class, either of which would provide instruction in the recognition of behavioral manifestations of psychological distress and referral procedures. Knowledge in these areas may help ATCs to initiate contact with mental health professionals, and ATCs may be more likely to refer athletes, when necessary, if they can recognize symptoms of psychological distress. If these courses were not available at the accredited institutions, guest lecturers specializing in the psychology of sport injury could provide supplemental lectures to established athletic training courses. Distance learning of these lectures could benefit athletic trainers who are already certified with either CEUs or graduate course credit.

We recommend that ATCs increase their knowledge of psychology and counseling not to advocate that ATCs provide therapy to injured athletes (or athletes at risk for injury). However, many counseling skills, such as active listening and the provision of emotional support, can be offered with little additional time investment, and the effects can be therapeutic. Moreover, the application of psychological principles (eg, goal setting and self-monitoring, relaxation training) may also enhance rehabilitation compliance, quality of sleep, and pain tolerance. With increased knowledge of counseling and psychology, ATCs may be more effective in making referrals to mental health professionals and in their functioning as part of an interdisciplinary treatment team to enhance injured athletes' physical and psychological well-being.

ACKNOWLEDGMENTS

We thank the following individuals for their comments regarding earlier versions of this manuscript: Edward F. Etzel, EdD, and Vincent Stilger, HSD, ATC, from the School of Physical Education, and Roy Tunick, EdD, from the Department of Counseling Psychology and Rehabilitation, at West Virginia University.

REFERENCES

1. Ballard P. Research brief: athletic care and injury prevention services. Opinions of athletic directors. *Natl Assoc Second Sch Principals Bull.* 1996;80:106–112.
2. Brewer BW. Adherence to sport injury rehabilitation programs. *J Sport Psychol.* 1998;10:70–82.
3. Fisher AC, Domm NA, Wuest DA. Adherence to sports-injury rehabilitation programs. *Physician Sportsmed.* 1988;16(7):47–51.
4. Perna FM, Antoni M, Schneiderman N. Psychological intervention prevents injury/illness among athletes [abstract]. *J Sport Psychol.* 1998; 10:S53.

5. Perna FM, McDowell SL. Role of psychological stress in cortisol recovery from exhaustive exercise among elite athletes. *Int J Behav Med.* 1995;2:13–26.
6. Williams JM, Andersen MB. Psychosocial antecedents of sport injury: review and critique of the stress and injury model. *J Sport Psychol.* 1998;10:5–25.
7. Etzel EF, Ferrante AP, Pinkney JW, eds. *Counseling College Student-Athletes: Issues and Interventions.* Morgantown, WV: Fitness Information Technology; 1991.
8. The National Athletic Trainers' Association Board of Certification. *Role Delineation Study: Athletic Training Profession.* 4th ed. Omaha, NE: Board of Certification; 1999:1–71.
9. National Athletic Trainers' Association Board of Certification. *Certification Update.* Raleigh, NC: National Athletic Trainers' Association Board of Certification; 1994.
10. National Athletic Trainers' Association Education Council. *Competencies in Athletic Training.* Dallas, TX: National Athletic Trainers' Association; 1992.
11. Larson GA, Starkey C, Zaichkowsky LD. Psychological aspects of athletic injuries as perceived by athletic trainers. *Sport Psychol.* 1996; 10:37–47.
12. Arnheim DD, Prentice WE. *Principles of Athletic Training.* 9th ed. St. Louis, MO: Mosby; 1993:13–14.
13. Beachy G, Akau CK, Martinson M, Olderr TF. High school sports injuries: a longitudinal study at Punahou School, 1988 to 1996. *Am J Sports Med.* 1997;25:657–681.
14. Lerch S. The adjustment of athletes to career ending injuries. *Arena Rev.* 1984;8:54–64.
15. Gieck J. Psychological considerations for rehabilitation. In: Prentice WE, ed. *Rehabilitation Techniques in Sports Medicine.* St. Louis, MO: Mosby; 1994:238–252.
16. Kelley MJJ. Psychological risk factors and sports injuries [abstract]. *J Sports Med Phys Fitness.* 1990;30:202–212.
17. Selye HA. *The Stress of Life.* New York, NY: McGraw-Hill; 1956.
18. Holmes TH, Rahe RL. The Social Readajustment Rating Scale. *J Psychosom Res.* 1967;11:213–218.
19. Bramwell ST, Masuda M, Wagner NN, Holmes TH. Psychological factors in athletic injuries: development and application of the Social and Athletic Readjustment Scale (SARRS). *J Hum Stress.* 1975;1:6–20.
20. Andersen MB, Williams JM. A model of stress and athletic injury: prediction and prevention. *J Sport Exerc Psychol.* 1988;10:294–306.
21. Williams JM, Roepke N. Psychology of injury and injury rehabilitation. In: Singer R, Murphey M, Tennant L, eds. *Handbook of Research in Applied Sport Psychology.* New York, NY: MacMillan; 1993:815–839.
22. Flint F. Integrating sport psychology and sports medicine research: the dilemmas. *J Sport Psychol.* 1998;10:83–102.
23. Perna F, Antoni MH, Kumar M, Cruess DG, Schneiderman N. Cognitive-behavioral intervention effects on mood and cortisol during exercise training. *Ann Behav Med.* 1998;20:92–98.
24. Williams JM, Tonymon P, Wadsworth WA. Relationship of life stress to injury in intercollegiate volleyball. *J Hum Stress.* 1986;12:38–43.
25. Coddington RD, Troxell JR. The effect of emotional factors on football injury rates: a pilot study. *J Hum Stress.* 1980;6:3–5.
26. Passer MW, Seese MD. Life stress and athletic injury: examination of positive versus negative events and three moderator variables. *J Hum Stress.* 1983;9:11–16.
27. Petrie TA. The moderating effects of social support and playing status on the life-stress relationship. *J Sport Psychol.* 1993;5:1–16.
28. Kerr G, Minden H. Psychological factors related to the occurrence of athletic injuries. *J Sport Exerc Psychol.* 1988;10:167–173.
29. Valliant PM. Personality and injury in competitive runners. *Percept Mot Skills.* 1981;53:251–253.
30. Hamilton LH, Hamilton WG, Meltzer JD, Marshall P, Molnar M. Personality, stress, and injuries in professional ballet dancers. *Am J Sports Med.* 1989;17:263–267.
31. May JR, Sieb GE. *Athletic Injuries: Psychosocial Factors in the Onset, Sequelae, Rehabilitation, and Prevention.* New York, NY: PMA Publishing; 1987;157–185.
32. Hardy CJ, Riehl RE. An examination of the life stress-injury relationship among noncontact sport participants. *Behav Med.* 1988;14:113–118.
33. Hanson SJ. McCullagh P, Tonymon P. The relationship of personality characteristics, life stress, and coping resources to athletic injury. *J Sport Exerc Psychol.* 1992;14:262–272.
34. Smith RE. Smoll FL, Ptacek JT. Conjunctive moderator variables in vulnerability and resiliency research: life stress, social support and coping skills, and adolescent sport injuries. *J Pers Soc Psychol.* 1990; 58:360–370.
35. Crossman J. Psychosocial factors and athletic injury. *J Sports Med Phys Fitness.* 1985;25:151–153.
36. Williams JM, Tonymon P, Andersen MB. Effects of life-event stress on anxiety and peripheral narrowing. *Behav Med.* 1990;16:174–181.
37. Williams J, Tonymon P, Andersen M. Effects of stress and coping resources on anxiety and peripheral narrowing. *J Sport Psychol.* 1991; 3:126–141.
38. Starkey C. *Therapeutic Modalities.* 2nd ed. Philadelphia, PA: FA Davis; 1999.
39. Carson RC, Butcher JN, Mineka S. *Abnormal Psychology and Modern Life.* 10th ed. New York, NY: Longman; 1998.
40. Nideffer RM. Psychological aspects of sports injuries: issues in prevention and treatment. *Int J Sport Psychol.* 1983;20:241–255.
41. Herbert TB, Cohen S, Marsland AL, et al. Cardiovascular reactivity and the course of immune response to an acute psychological stressor. *Psychosom Med.* 1994;56:337–344.
42. Smith AM, Stuart MJ, Wiese-Bjornstal DM, Milliner EK, O'Fallon WM, Crowson CS. Competitive athletes: preinjury and postinjury mood state and self-esteem. *Mayo Clin Proc.* 1993;68:939–947.
43. Petrie T, Brewer B, Buntrock C. A comparison between injured and uninjured NCAA Division I male and female athletes on selected psychosocial variables [abstract]. *J Sport Psychol.* 1997;9:S144.
44. Brewer BW, Petrie TA. A comparison between injured and uninjured football players on selected psychosocial variables. *Acad Athl J.* 1995; 11–18.
45. Crossman J. Psychological rehabilitation from sports injuries. *J Sport Med.* 1997;23:333–339.
46. Leddy MH, Lambert MJ, Ogles BM. Psychological consequences of athletic injury among high-level competitors. *Res Q Exerc Sport.* 1994;65:347–354.
47. Quackenbush N, Crossman J. Injured athletes: a study of emotional responses. *J Sport Behav.* 1994;17:178–187.
48. Roh JL, Newcomer RR, Perna FM, Etzel EM. Depressive mood states among college athletes: pre- and post-injury [abstract]. *J Sport Psychol.* 1998;10:S54.
49. Schoene ML. When injuries wound an athlete's mind. *Sport Med Digest.* 1998;20:13–15.
50. Smith AM, Scott SG, Wiese DM. The psychological effects of sport injuries: coping. *Sports Med.* 1990;9:352–369.
51. Smith AM, Scott SG, O'Fallon WM, Young ML. Emotional responses of athletes to injury. *Mayo Clin Proc.* 1990;65:38–50.
52. Brewer BW, Linder DE, Phelps CM. Situational correlates of emotional adjustment to athletic injury. *Clin J Sport Med.* 1995;5:241–245.
53. Macchi R, Crossman J. After the fall: reflections of injured classical ballet dancers. *J Sport Behav.* 1996;19:221–234.
54. Pearson RE, Petitpas AJ. Transitions of athletes: developmental and preventive perspectives. *J Coun Devel.* 1990;69:7–10.
55. Lynch GP. Athletic injuries and the practicing sport psychologist: practical guidelines for assisting athletes. *Sport Psychol.* 1988;2:161–167.
56. McDonald SA, Hardy CJ. Affective response patterns of the injured athlete: an exploratory analysis. *Sport Psychol.* 1990;4:261–274.
57. Perna FM, Roh JL, Newcomer RR, Etzel EF. Clinical depression among injured athletes: an empirical assessment [abstract]. *J Sport Psychol.* 1998;10:S54.
58. Newcomer RR, Roh JL, Perna FM, Stilger VG, Etzel EF. Injury as a traumatic experience: intrusive thoughts and avoidance behavior associated injury among college student-athletes [abstract]. *J Sport Psychol.* 1998;10:S54.
59. Shuer ML, Dietrich MS. Psychological effects of chronic injury in elite athletes. *West J Med.* 1997;166:104–109.

60. Prentice WE. *Rehabilitation Techniques in Sports Medicine*. 2nd ed. St. Louis, MO: Mosby; 1994:81.
61. Kiecolt-Glaser JK, Page GG, Marucha PT, MacCallum RC, Glaser R. Psychological influences on surgical recovery: perspectives from psychoneuroimmunology. *Am Psychol.* 1998;53:1209–1218.
62. Marucha PT, Kiecolt-Glaser JK, Favagehi M. Mucosal wound healing is impaired by examination stress. *Psychosom Med.* 1998;60:362–365.
63. Florini JR. Hormonal control of muscle growth. *Muscle Nerve.* 1987;10: 577–598.
64. American Academy of Orthopaedic Surgeons. *Athletic Training and Sports Medicine*. 2nd ed. Park Ridge, IL: American Academy of Orthopaedic Surgeons; 1991:8–10,167–185.
65. Burns JW, Johnson BJ, Mahoney N, Devine J, Pawl R. Cognitive and physical capacity process variables predict long-term outcome after treatment of chronic pain. *J Consult Clin Psychol.* 1998;66:434–439.
66. Turk DC. Biopsychosocial perspective on chronic pain. In: Gatchel RJ, Turk DC, eds. *Psychological Approaches to Pain Management: A Practitioner's Handbook*. New York, NY: Guilford Publications; 1996.
67. deLateur B, Wegener SB. Current concepts and clinical approaches in the psychology of rehabilitation. In: *Proceedings, National Athletic Trainers' Association 49th Annual Meeting & Clinical Symposia 1998*. Champaign, IL: Human Kinetics; 1998:16–17.
68. Leaver-Dunn D. Awareness of rehabilitation networks and referral systems. In: *Proceedings, National Athletic Trainers' Association 49th Annual Meeting & Clinical Symposia 1998*. Champaign, IL: Human Kinetics; 1998:19–20.
69. Taylor AH, May S. Threat and coping appraisal as determinants of compliance with sports injury rehabilitation: an application of Protection Motivation Theory. *J Sports Sci.* 1996;14:471–482.
70. Weiss MR, Troxel RK. Psychology of the injured athlete. *Athl Train, J Natl Athl Train Assoc.* 1986;21:104–109,154.
71. Taylor J, Taylor S. *Psychological Approaches to Sports Injury Rehabilitation.* Gaithersburg, MD: Aspen; 1997.
72. Fisher AC. Adherence to sports injury rehabilitation programmes. *Sports Med.* 1990;9:151–158.
73. Fisher AC, Scriber KC, Matheny ML, Alderman MH, Bitting LA. Enhancing athletic injury rehabilitation adherence. *J Athl Train.* 1993; 28:312–318.
74. Duda JL, Smarth AE, Tappe MK. Predictors in rehabilitation of athletic injuries: an application of personal investment theory. *J Sport Exer Psychol.* 1989;11:367–381.
75. Davis JO. Sports injuries and stress management: an opportunity for research. *Sport Psychol.* 1991;5:175–182.
76. Davis M, Eshelman ER, McKay M. *The Relaxation and Stress Reduction Workbook*. 4th ed. Oakland, CA: New Harbinger; 1995.
77. Heil J. *Psychology of Sport Injury*. Champaign, IL: Human Kinetics; 1993.
78. Murphy SM, ed. *Sport Psychology Interventions*. Champaign, IL: Human Kinetics; 1995.
79. Pargman D, ed. *Psychological Bases of Sport Injuries.* Morgantown, WV: Fitness Information Technology; 1993.
80. Ross MJ, Berger RS. Effects of stress inoculation training on athletes' postsurgical pain and rehabilitation after orthopedic injury. *J Consult Clin Psychol.* 1996;64:404–410.
81. Williams JW, ed. *Applied Sport Psychology: Personal Growth and Peak Performance*. 2nd ed. Mountain View, CA: Mayfield; 1993.
82. Granito VJ, Hogan JB, Varum LK. The performance enhancement group program: integrating sport psychology and rehabilitation. *J Athl Train.* 1995;30:328–331.
83. Daly JM, Brewer BW, Van Raatle JL, Petitas AJ, Sklar JH. Cognitive appraisal, emotional adjustment, and adherence to rehabilitation following knee surgery. *J Sport Rehabil.* 1995;5:175–182.
84. Lampton CC, Lambert ME, Yost R. The effects of psychological factors in sports medicine rehabilitation adherence. *J Sports Med Phys Fitness.* 1993;33:292–299.
85. Laubach WJ, Brewer BW, Van Raalte JL, Petitas AJ. Attributions for recovery and adherence to sport injury rehabilitation. *Aust J Sci Med Sport.* 1996;28:30–34.
86. Padula MA, Whiteside DM, Jeffrey L. Predicting treatment cooperation: first steps in rehabilitation [abstract]. Presented at: 1998 American Psychological Association Convention, Division 22: Rehabilitation Psychology; August, 1998; San Francisco, CA.
87. Pen LJ, Fisher CA. Athletes and pain tolerance. *Sports Med.* 1994;18: 319–329.
88. Wiese DM, Weiss MR. Psychological rehabilitation and physical injury: implications for the sportsmedicine team. *Sport Psychol.* 1987;1:318–330.
89. Fisher AC, Hoisington LL. Injured athlete's attitudes and judgments toward rehabilitation adherence. *J Athl Train.* 1993;28:48–54.
90. Fisher AC, Mullins SA, Frye PA. Athletic trainers' attitudes and judgments toward rehabilitation adherence. *J Athl Train.* 1993;28:43–47.
91. Gordon S, Milios D, Grove JR. Psychological aspects of the recovery process from sport injury: the perspective of sport physiotherapists. *Aust J Sci Med Sport.* 1991;53–60.
92. Byerly PN, Worrell T, Gahimer J, Damholdt E. Rehabilitation compliance in an athletic training environment. *J Athl Train.* 1994;29:352–355.
93. Johnson U. Coping strategies among long-term injured competitive athletes: a study of 81 men and women in team and individual sports. *Scand J Med Sci Sport.* 1997;7:367–372.
94. Brewer BW, Daly JM, Van Raalte JL, Petitpas AJ, Sklar JH. A psychometric evaluation of the rehabilitation adherence questionnaire. *J Sport Exerc Psychol.* 1999;21:167–173.
95. Elliott PL. Why I probe for personality traits: to get patients to follow orders, this doctor has found it pays to learn what makes them tick. *Med Econ.* 1994;71(2):4.
96. Gordon S. Sport psychology and the injured athlete: a cognitive-behavioral approach to injury response and injury rehabilitation. *Sport Psychol Bull.* March 1985.
97. Udry E. Coping and social support among injured athletes following injury. *J Sport Exerc Psychol.* 1997;19:71–90.
98. Wiese-Bjornstal DM, Smith AM, LaMott EE. A model of psychologic responses to athletic injury and rehabilitation. *Athl Train: Health Care Perspect.* 1995;1:17–30.
99. Brewer BW. Review and critique of models of psychological adjustment to athletic injury. *J Sport Psychol.* 1994;6:87–100.
100. Moulton MA, Molstad S, Turner A. The role of athletic trainers in counseling collegiate athletes. *J Athl Train.* 1997;32:148–150.
101. Wiese DM, Weiss MR, Yukelson DP. Sport psychology in the training room: a survey of athletic trainers. *Sport Psychol.* 1991;5:15–25.
102. National Athletic Trainers' Association Board of Certification. *Certification Update*. Raleigh, NC: National Athletic Trainers' Association Board of Certification; 1999.
103. Pennsylvania Athletic Trainers' Society. *Athletic Trainers: Caring for Pennsylvania's Physically Active.* Pennsylvania Athletic Trainers' Society; 1998.
104. Kane B. Trainer counseling to avoid three face-saving maneuvers. *Athl Train, J Natl Athl Train Assoc.* 1984;19:171–174.
105. Ray R, Terrell T, Hough D. The role of the sports medicine professional in counseling athletes. In: Ray R, Wiese-Bjornstal DM, eds. *Counseling in Sports Medicine*. Champaign, IL: Human Kinetics; 1999:3–21.
106. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders [DSM-IV]*. 4th ed. Washington, DC: American Psychiatric Association; 1994.
107. National Athletic Trainers' Association. *Code of Ethics: Membership Standards, Membership Sanctions, and Procedures*. Dallas, TX: National Athletic Trainers' Association; 1993.
108. Gordon S, Potter M, Ford IW. Toward a psychoeducational curriculum for training sport-injury rehabilitation personnel. *J Sport Psychol.* 1998; 10:140–156.

Journal of Athletic Training 2000;35(4):466–470

www.journalofathletictraining.org

Youth Sports: A Pediatrician's Perspective on Coaching and Injury Prevention

Michael C. Koester, MD, ATC

Good Shepherd Community Hospital, Hermiston, OR

Objective: My objective is to review the factors that influence youth participation in sports, to discuss the role coaches may play in youth sports injuries, and to call on athletic trainers and other health professionals to become involved in youth sports in an effort to limit injury risk.

Background: Millions of American youths participate in team sports. Their primary motivation to participate is to have fun. Unfortunately, large numbers of participants have sustained correspondingly large numbers of injuries. Many injuries can be attributed to improper technique and conditioning methods taught by volunteer coaches. Although not the only contributors to injuries, these may be the most amenable to preventive measures, such as formal instruction for coaches in the areas of proper biomechanics and player-coach communication.

Description: I provide an overview of the reasons why children participate in sports, discuss participation motivation, and review the literature on coaches' communication methods that have been proved effective in maximizing learning and enjoyment for young athletes.

Clinical Advantages: This article provides certified athletic trainers with the background knowledge needed to take an active role in youth sports injury prevention at the community level.

Key Words: young athletes, sports psychology, participation motivation, injury prevention, coaching

Nearly 20 million youths between the ages of 6 and 16 years old participate in a vast array of nonscholastic organized sports, with an additional 6 to 7 million high school students involved in school-sponsored athletic activity yearly.[1] Not surprisingly, such large numbers of participants and a myriad of other factors have led to rather startling injury statistics. Young girls playing organized sports have an estimated injury rate of 20 to 22 per 100 participants per season, while boys have an almost doubled risk of 39 per 100 participants per season.[2] Recent data show that sports and recreational activities account for 32.3% of all serious injuries in children ages 5 to 17 years.[3]

Numerous studies and review articles have reviewed the potential causes for injury in youth sports participants, often stressing the unique physiologic and biomechanical aspects of the growing body.[4–7] In addition, the literature regarding the effects of life stress on injury occurrence is expanding.[8,9] Although this area has not yet been scientifically investigated, anecdotal evidence implies that improper training methods and lack of proper sport-specific techniques also contribute to injury. Some sports medicine professionals point directly to poor coaching as a factor in injury.[10]

Injury has always been recognized as a natural risk of participation in organized sport. However, many injuries may be preventable, particularly those resulting from a lack of proper, well-supervised training and participation. My purpose is to review the factors that influence youth participation in sports, to discuss the role coaches may play in youth sports injuries, and to call on athletic trainers and other health professionals to become involved with youth sports in their communities in an effort to limit injury risk. Also, I hope to stimulate athletic trainers to increase their knowledge in pediatric sports medicine.

Athletic trainers are viewed as sports injury experts by parents, coaches, and, increasingly, the medical profession. In this role, they can help to educate parents and coaches in injury prevention. Granted, other factors lead to injury, but they are not the focus of this discussion. Here, I present a primer for sports injury prevention by first making sure that coaches are emphasizing fun and fundamentals in working with their young athletes.

HISTORY OF YOUTH SPORTS IN THE UNITED STATES

The industrialization and urbanization of the 19th century led to the creation of organized youth sports in America. Early youth sports organizations had their origins within local schools and churches, as physical activity was thought to be character building. In the 1890s, the YMCA first began offering the opportunity for young men to compete against each other. The founding of New York City's Public School Athletic League in 1903 ushered in the explosion of organized sports participation in the first half of the 20th century.[11] By the 1930s, professional team sports such as football and baseball became a significant part of America's popular culture, and involvement in youth sports paralleled this rapid rise in popularity.

In the 1930s, opposition to youth competitive leagues arose, and these organizations began to fall under the auspices of community groups, rather than churches and schools. Many educational leaders were opposed to children's involvement in

Address correspondence to Michael C. Koester, MD, ATC, 105 SE Crestline Drive, Hermiston, OR 97838. E-mail address: mkoester@eoni.com

competition for a variety of reasons, including premature specialization and the potentially harmful psychological effects of competition.[11] At that time, several schools did disband competitive leagues for elementary school-aged children. This shift in philosophy in the school systems had repercussions that are still felt today. As physical educators and other teachers played less of a role in coaching school-sponsored sports, thousands of parents and others with no formal training in coaching or child development took their place.[12]

Despite opposition, youth sports continued to grow in popularity. Between the founding of Little League Baseball in 1939 and the mid 1960s, numerous youth sports organizations were formed on the national level. The 1970s and 1980s saw an ever-increasing number of young women and girls playing individual and team sports, as previous barriers to participation were overcome both legally and socially, although calls to ban competitive youth sports continued.[13]

FACTORS IN PARTICIPATION

Why do children choose to participate in organized sports? Study in the arena of participation motivation has burgeoned in the past 2 decades. The research centers on the reasons individuals adopt for initiating, continuing, and sustaining involvement in physical activity, as well as the reasons for discontinuing involvement.[14] Not surprisingly, many studies have concluded that "having fun" is the primary reason children choose sports participation.[15,16] Skill development, making friends through team involvement, the challenge of sports, and improving physical fitness were also commonly found factors lending to participation. Interestingly, in what is likely a symptom of changing family structures in the United States, Stern et al[16] found that preadolescents in an urban boys club basketball league also ranked "having a coach to look up to" rather highly.

Research in recent years has demonstrated that perceived confidence and perceived ability also play major roles in participation motivation.[14,17,18] Harter termed this competence "motivation theory" and postulated that individuals are motivated to demonstrate competence in an achievement area and do so by engaging in mastery attempts.[14] Although a more detailed discussion of competence motivation is beyond the scope of this article, a few significant findings put the theory into perspective. Children who tested highly in perceived physical competence were motivated by the prospect of further skill development, while gymnasts high in perceived social competence were motivated by the affiliation aspects of their sport.[17] In what could be the paradigm of Harter's theory, Orlick[18] interviewed children who had never participated in organized youth sports programs and discovered that 75% wanted to participate but did not try out for a sport because they felt they were not good enough.

Many youngsters eventually choose to discontinue sports participation, as many as 50% according to 1 study.[19] The reasons for this high rate of attrition are varied but include interest in other sports, outside interests other than sports, lack of playing time, overemphasis on competition, and dislike of the coach. DuRant et al[1] found injury to be the most common factor in the discontinuation of school sport involvement among 674 high school students. Sadly, attrition among elementary school-aged participants results from little success, a lack of playing time, and "absence of fun."[20]

THE ROLE OF THE COACH IN YOUTH SPORTS

Although they are often the subject of a great deal of criticism,[10,15] no one plays a more vital role in the lives of young athletes than their coaches. According to Engh,[21] 85% of coaches are either parents or other interested persons who have no formal training in coaching. The need for coaches has quadrupled over the last 20 years, which has led to a shortage of qualified applicants.[15] Perhaps not coincidentally, health care professionals feel that injuries resulting from organized sports activity have been on the rise in recent years.[7,10]

Athletic injuries that occur in children can be divided into macrotrauma (fractures, sprains) and microtrauma (enthesitis, tendinitis). Microtrauma is often said to be secondary to overuse. In fact, these injuries generally result from a combination of overuse and misuse. Many young athletes are never taught the proper conditioning methods or technique for their sports.[7] Youngsters and coaches alike often fall prey to the "more is better" philosophy of sports training, attempting to do "too much, too soon" while also using improper biomechanics.

Many coaches simply do not have the proper knowledge base for instructing in correct sport techniques.[15] Volunteers without formal training often base their coaching style and techniques on their own personal experiences. Even trained coaches will likely have learned many of the technical aspects of their job by observing and listening to other coaches.[22] Both of these styles of acquiring coaching knowledge are prone to misinformation and improper theory being perpetuated for years, setting the stage for injury.

PLAYER-COACH COMMUNICATION

A vital but often overlooked component of the player-coach relationship is communication. Sports psychologists have found that the manner in which a coach communicates with his or her athletes not only affects how players perceive their own abilities but can also affect the athletes' ability to assimilate technical instruction.[23] Much of this research again centers on competence motivation theory. Horn[24] found that skill improvement accounted for the greatest variance in self-perception among junior high school female softball players. Surprisingly, players who received more frequent positive reinforcement actually scored lower in perceived physical competence than those who received more frequent criticism. This appears contradictory at first glance; however, Horn[24] also observed that positive reinforcement was often given unconditionally to players with less ability (eg,"good job" and "nice try") without regard for their effort. Players with more ability received skill-relevant information as part of the criticism (eg,"use 2 hands," "keep your glove down").

Although the above-mentioned research only looked at the effects of communication on athletes' self-perceptions and feelings about aspects of competition, there are further implications of these findings. If improper technique does indeed lead to a number of injuries, as is believed to be the case, simply using new methods of communication and instruction may result in a decline in injuries. Such communication may be even more important for younger children, because it is believed that children who are beginning to attain skills depend even more on adult evaluation.

OFFERING A SAFE ENVIRONMENT TO YOUTH SPORTS PARTICIPANTS

It has been estimated that a young athlete spends an average of 326 hours of practice time under supervision of a coach during 1 athletic season, dwarfing the time spent with teachers, health educators, and physicians.[25] This has led to a recent call for coaches to take a more active role in health education and to use the athlete-coach relationship to modify high-risk behaviors and to focus on social-skills training and character formation.[25] Ideally, the above call would be an outstanding plan. However, to heap more responsibility onto well-meaning but undertrained individuals is unproductive and detrimental. At this point, we must ask of coaches only that they foster an environment in which children can participate in athletics with a minimal chance of injury and enjoy themselves while doing so.

How can this goal be accomplished? Injury prevention in the growing athlete centers on the principles of effective coaching, proper training habits, maintenance of flexibility, and the consistent use of correct biomechanics. First, coaches must be knowledgeable in the technical aspects of the sport they coach. Specifically, they must be aware of the proper biomechanics and training techniques required by that sport. Fundamentals are essential to all athletes but they are even more important to young, skeletally immature participants, who are at high risk for tendon and muscle injuries imparted by the more stressful forces caused by poor biomechanics.

Second, coaches must be aware of the emotional and psychological developmental stages of children and adolescents and not treat them like "little adults."[26] We cannot ask our coaches to be formally trained in child psychology, but we must expect them to always have the youngsters' best interests at heart and to keep instructions, expectations, and goals developmentally appropriate (Table 1). We are all aware (personally or professionally) of the emotional trauma and stress that can be inflicted on young athletes. However, despite our perceptions and societal stereotypes, athletics may not be as stressful as other activities for school-aged children. Simon and Martens[27] found that children performing band solos experienced more anxiety than athletes, and among the sports compared, only wrestling was significantly more anxiety arousing than test taking.

Finally, coaches can be trained through formal instruction in methods of communicating with young athletes to avoid emotional conflict, foster positive attitudes, and improve skills. Over the past decade and a half, Smith and Smoll[23] and Smith et al[28,29] have carried out a series of studies with Little League baseball players in which they proved the effectiveness of certain communication techniques used by coaches. Players enjoy competition more and find their coaches to be more effective than coaches not trained in such techniques.[29] The players also scored higher in self-esteem than controls. The coaches' behavior that the athletes found most beneficial was error-contingent, corrective instruction (eg, "you dropped the ball because you didn't use 2 hands"). Such training could result in a decrease in injuries because properly informed coaches may more effectively instruct their players in the correct technique.

As I have detailed, children participate in sports to have fun. That participation is usually discontinued either because of injury or because the young athlete is no longer having fun. Therefore, our efforts must be directed toward the twin goals of preventing injury and maximizing enjoyment of the sport. As discussed, training programs are an effective way of increasing coaches' knowledge bases and communication abilities, both of which will lead to fewer injuries (Table 2). It is unlikely, however, that most volunteer coaches will have the opportunity to take advantage of these courses. However, we must make our local coaches aware of such programs and strongly encourage attendance. Many courses are short (half day) and relatively inexpensive. Travel costs could be defrayed by applying a portion of the league sign-up fees.

ROLE OF THE ATHLETIC TRAINER IN YOUTH SPORTS

Only at the elite levels of competition are youth sports participants assured access to athletic trainers and sports medicine physicians. Millions of other active youngsters are often without timely or proper injury-prevention techniques, acute injury evaluation, and rehabilitation assistance. Many, but certainly not all, high school student-athletes have an athletic trainer or team physician available, at least on a part-time basis. Younger athletes, however, have only their coach available for acute injury care and rehabilitation guidance. Compounding matters, the family physician or pediatrician evaluating the young athlete usually has little understanding of the causes of pediatric sports injuries or the principles of rehabilitation.

Given the current inequities of youth sports injury prevention and care in the United States, the athletic training profession could benefit young athletes nationwide by making a concerted effort at the local, state, and national levels to become involved with youth sports programs. Ideally, athletic trainers can begin by organizing coaching clinics to highlight the uniqueness of young athletes and their susceptibility to injury. Strength and conditioning principles, the bedrock of injury prevention, can also be taught. Youth sports coaches can

Table 1. Developmentally Appropriate Coaching Methods and Principles for Young Athletes

Developmental Level	Task	Method/Principle
Elementary school	Practice of learned skills	Recognize heavy dependence on adult input for sense of competence Provide repetitive instruction and emotional support Reinforce effort and progress, not results: strive to achieve, not to win Motivate based on positive accomplishments, not fear of failure Define mistakes as "learning opportunities"
Adolescence	Achievement of autonomy	Provide advanced instruction to improve skills and knowledge base Support autonomy by fostering internalized performance standards Provide feedback contingent on performance: praise only when appropriate Recognize that critical comments are often perceived as a threat to autonomy; provide corrective instruction

Adapted with permission from Libman.[12]

Table 2. Organizations Offering Instructive Programs for Coaches

National Youth Sports Coaches Association
2050 Vista Parkway
West Palm Beach, FL 33411
561-684-1141

American Coaches Effectiveness Program
1607 North Market Street
Champaign, IL 61820
800-747-4457
www.humankinetics.com

Youth Sports Institute
Michigan State University
213 IM Circle
East Lansing, MI 48824
517-353-6689
E-mail: ythsprts@msu.edu

Coach Effectiveness Training Program
Frank Smoll
Department of Psychology
Guthrie Hall, N1-25
University of Washington
Seattle, WA 98195
206-543-4612

be further instructed in the basics of acute injury recognition, emphasizing symptoms that indicate the need to discontinue participation immediately and seek medical care. Such knowledge would limit further, more serious injuries. The focus of all involvement should be on injury prevention, not evaluation and treatment.

Another role may include mediation in times of dispute over an injury or other malady affecting a child's ability to participate. The athletic trainer who is interested only in the child's health and safety rather than the outcome of the event can offer parents and coaches an impartial third party. Athletic trainers may also serve a valuable role in actively policing the local coaching ranks (with the cooperation of others) to remove individuals abusing their authority and those who have "winning out of perspective"[30] (Table 3). Athletic trainers can also gather information about, and encourage attendance to, coaching programs and seminars such as those discussed above.

I outline the above program fully recognizing the time constraints of all involved. Many athletic trainers already work long hours for relatively minimal compensation compared with other health care professionals. However, the proposed interventions do not command huge time commitments and offer the added incentive of boosting public awareness of the profession. As athletic trainers come into contact with a wider variety of athletes and parents outside the high school and college settings, the public will become more aware of athletic trainers and their role in sports injury care.

Table 3. Criteria to Determine that Winning is Out of Perspective[30]

1. A display of comradeship with an opponent is considered a sign of weakness.
2. Laughter is judged to be a lack of competitiveness.
3. Strategies to take unfair advantage of opponents are developed.
4. Youngsters are cajoled to cheat or intimidated to excel.
5. Winning the game is more important than winning friends, respect, self-confidence, skill, and self-worth.

Obviously these goals cannot be accomplished overnight, nor without great effort or organization. I am not asking athletic trainers to act alone, but I am calling for involvement by many in each community, including physicians, nurses, physical therapists, psychologists, teachers, and coaches. All must be vocal advocates for child safety issues, becoming involved not just as professionals, but as parents, grandparents, aunts, uncles, and friends. Programs such as the National Athletic Trainers' Association-endorsed National Safe Kids Campaign[31] should also be vigorously supported. While we must keep in mind that risks are always inherent risks in physical activity, our goal should be to create as safe and enjoyable as environment as possible in which our children can participate.

REFERENCES

1. DuRant RH, Pendergrast RA, Donner J, Seymore C, Gaillard G. Adolescents' attrition from school-sponsored sports. *Am J Dis Child.* 1991;145: 1119–1123.
2. Ostrum GA. Sports-related injuries in youths: prevention is the key and nurses can help. *Pediatr Nurs.* 1993;19:333–342.
3. Bijur PE, Trumble A, Harel Y, Overpeck MD, Jones D, Scheidt PC. Sports and recreation injuries in US children and adolescents. *Arch Pediatr Adolesc Med.* 1995;149:1009–1016.
4. Micheli LJ. The child and adolescent. In: Harries M, Williams C, Stanish WD, Micheli LJ, eds. *Oxford Textbook of Sports Medicine.* New York, NY: Oxford University Press; 1994:646–652.
5. Micheli LJ. Pediatric and adolescent musculoskeletal sports injuries. In: Teitz CC, ed. *Scientific Foundations of Sports Medicine.* Toronto, ON, Canada: BC Decker; 1989:329–343.
6. Micheli LJ. Overuse injuries in children's sports: the growth factor. *Orthop Clin North Am.* 1983;14:337–360.
7. Micheli LJ. Sports injuries in the young athlete: questions and controversies. In: Micheli LJ, ed. *Pediatric and Adolescent Sports Medicine.* Boston. MA: Little Brown and Co; 1984:1–8.
8. Bramwell ST, Masuda M, Wagner NN, Holmes TH. Psychosocial factors in athletic injuries: development and application of the Social and Athletic Readjustment Rating Scale (SARRS). *J Human Stress.* 1975;1:6–20.
9. Cryan PD, Alles WF. The relationship between stress and college football injuries. *J Sports Med Phys Fitness.* 1983;23:52–58.
10. Murphy P. Youth sports coaches: using hunches to fill a blank page. *Physician Sportsmed.* 1985;13(4):136–142.
11. Wiggins DK. A history of organized play and highly competitive sport for American children. In: Gould D, Weiss MR, eds. *Advances in Pediatric Sport Sciences.* Champaign, IL: Human Kinetics; 1987:1–24.
12. Libman S. Adult participation in youth sports: a developmental perspective. *Child Adolesc Psychiatr Clin North Am.* 1998;7:725–744.
13. Sayre BM. The need to ban competitive sports involving preadolescent children [letter]. *Pediatrics.* 1975;55:564–565.
14. Weiss MR. Psychological effects of intensive sport participation on children and youth: self-esteem and motivation. In: Cahill BR, Pearl AJ, eds. *Intensive Participation in Children's Sports.* Champaign, IL: Human Kinetics; 1993:39–69.
15. Quain RJ. An overview of youth coaching certification programs. *Adolescence.* 1989;24:541–547.
16. Stern P, Bradley RH, Prince MT, Stroh SE. Young children in recreational sports: participation motivation. *Clin Pediatr (Phila).* 1990;29:89–94.
17. Klint KA, Weiss MR. Perceived competence and motives for participating in youth sports: a test of Harter's competence motivation theory. *J Sport Psychol.* 1987;9:55–65.
18. Orlick TD. The athletic dropout: a high price of inefficiency. *Can Assoc Health Phys Ed Recr J.* 1974;6:21–27.
19. Gould D. Understanding attrition in children's sport. In: Gould D, Weiss MR, eds. *Advances in Pediatric Sport Sciences.* Champaign, IL: Human Kinetics; 1987:61–84.

20. Chambers ST. Factors affecting elementary school students' participation in sports. *Elem School J.* 1991;91:413–419.

21. Engh F. Medicine man for the ailing youth sports scene. *Parks Recr.* 1981;58:27–31.

22. Sage GH. Becoming a high school coach: from playing sports to coaching. *Res Q Exerc Sport.* 1989;60:81–92.

23. Smith RE, Smoll FL. Self-esteem and children's reactions to youth sport coaching behaviors: a field study of self-enhancement processes. *Dev Psychol.* 1990;26:987–993.

24. Horn TS. Coaches' feedback and changes in children's perceptions of their physical competence. *J Educ Psychol.* 1985;77:174–186.

25. Brown BR, Butterfield SA. Coaches: a missing link in the health care system. *Am J Dis Child.* 1992;146:211–217.

26. Stanitski CL. Management of sports injuries in children and adolescents. *Orthop Clin North Am.* 1988;19:689–698.

27. Simon JA, Martens R. Children's anxiety in sport and nonsport evaluative activities. *J Sport Psychol.* 1979;1:160–169.

28. Smith RE, Smoll FL, Curtis B. Coaching behaviors in Little League baseball. In: Smoll FL, Smith RE, eds. *Psychological Perspectives in Youth Sports.* Washington, DC: Hemisphere; 1978:173–201.

29. Smith RE, Smoll FL, Curtis B. Coach effectiveness training: a cognitive behavioral approach to enhancing relationship skills in youth sport coaches. *J Sport Psychol.* 1979;1:59–75.

30. Martens R. *Joy and Sadness in Children's Sports.* Champaign, IL: Human Kinetics; 1978.

31. Curran K. NATA partners with National Safe Kids campaign. *NATA News.* Mar 1996;24.

Journal of Athletic Training 2000;35(4):471–477
© by the National Athletic Trainers' Association, Inc
www.journalofathletictraining.org

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

Katie M. Walsh, EdD, ATC-L*; Brian Bennett, MEd, ATC†; Mary Ann Cooper, MD‡; Ronald L. Holle, MS§; Richard Kithil, MBA¶; Raul E. López, PhD§

*East Carolina University, Greenville, NC; †The College of William and Mary, Williamsburg, VA; ‡The University of Illinois at Chicago, Chicago, IL; §National Severe Storms Laboratory, Norman, OK; ¶The National Lightning Safety Institute, Louisville, CO

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

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

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

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

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

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

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

Address correspondence to National Athletic Trainers' Association, Communications Department, 2952 Stemmons Freeway, Dallas, TX 75247.

RECOMMENDATIONS

1. Formalize and implement a comprehensive, proactive lightning-safety policy or emergency action plan specific to lightning safety.[1,7,10–14] The components of this policy should include the following:
   A. An established chain of command that identifies who is to make the call to remove individuals from the field or an activity.
   B. A designated weather watcher (ie, a person who actively looks for the signs of threatening weather and notifies the chain of command if severe weather becomes dangerous).
   C. A means of monitoring local weather forecasts and warnings.
   D. A listing of specific safe locations (for each field or site) from the lightning hazard.
   E. The use of specific criteria for suspension and resumption of activities (refer to recommendations 4, 5, and 6).
   F. The use of the recommended lightning-safety strategies (refer to recommendations 7, 8, and 9).
2. The primary choice for a safe location from the lightning hazard is any substantial, frequently inhabited building. The electric and telephone wiring and plumbing pathways aid in grounding a building, which is why buildings are safer than remaining outdoors during thunderstorms. It is important not to be connected to these pathways while inside the structure during ongoing thunderstorms.
3. The secondary choice for a safer location from the lightning hazard is a fully enclosed vehicle with a metal roof and the windows closed.[1,7,10,11,13,14] Convertible cars and golf carts do not provide protection from lightning danger. It is important not to touch any part of the metal framework of the vehicle while inside it during ongoing thunderstorms.
4. Seeking a safe structure or location at the first sign of lightning or thunder activity is highly recommended. By the time the flash-to-bang count approaches 30 seconds (or is less than 30 seconds), all individuals should already be inside or should immediately seek a safe structure or location.[1,13–15] To use the flash-to-bang method, the observer begins counting when a lightning flash is sighted. Counting is stopped when the associated bang (thunder) is heard. Divide this count by 5 to determine the distance to the lightning flash (in miles). For example, a flash-to-bang count of 30 seconds equates to a distance of 6 miles (9.66 km).
5. Postpone or suspend activity if a thunderstorm appears imminent before or during an activity or contest (regardless of whether lightning is seen or thunder heard) until the hazard has passed. Signs of imminent thunderstorm activity are darkening clouds, high winds, and thunder or lightning activity.
6. Once activities have been suspended, wait at least 30 minutes after the last sound of thunder or lightning flash before resuming an activity or returning outdoors.[1,13–15] A message should be read over the public address system and lightning-safety tips should be placed in game programs alerting spectators and competitors about what to do and where to go to find a safer location during thunderstorm activity.[13,15]
7. Extremely large athletic events are of particular concern with regard to lightning safety. Consider using a multidisciplinary approach to lessen lightning danger, such as integrating weather forecasts, real-time thunderstorm data, a weather watcher, and the flash-to-bang count to aid in decision making.
8. Avoid being in contact with, or in proximity to, the highest point of an open field or on the open water. Do not take shelter under or near trees, flag poles, or light poles.[1,8,10,13–15]
9. Avoid taking showers and using plumbing facilities (including indoor and outdoor pools) and land-line telephones during thunderstorm activity.[1,8,10,13–15] Cordless or cellular telephones are safer to use when emergency help is needed.
10. Individuals who feel their hair stand on end or skin tingle or hear crackling noises should assume the lightning-safe position (ie, crouched on the ground, weight on the balls of the feet, feet together, head lowered, and ears covered). Do not lie flat on the ground.[1,8,10,13–15]
11. Observe the following basic first-aid procedures, in order, to manage victims of lightning strike[16]:
    A. Survey the scene for safety. Ongoing thunderstorms may still pose a threat to emergency personnel responding to the situation.
    B. Activate the local emergency management system.
    C. Move the victim carefully to a safer location, if needed.
    D. Evaluate and treat for apnea and asystole.
    E. Evaluate and treat for hypothermia and shock.
    F. Evaluate and treat for fractures.
    G. Evaluate and treat for burns.
12. All persons should maintain current cardiopulmonary resuscitation (CPR) and first-aid certification.
13. All individuals should have the right to leave an athletic site or activity, without fear of repercussion or penalty, in order to seek a safe structure or location if they feel they are in danger from impending lightning activity.[13,15]

BACKGROUND

Lightning-Flash Development

Within a developing thunderstorm cloud, updrafts promote the collision of rising and descending ice and water particles, and the positive and negative charges are separated into distinct layers. Positive charges are taken via updrafts to the top of the cloud, while negative charges accumulate in the bottom of the cloud, creating the equivalent of a giant atmospheric battery.[8]

A cloud-to-ground lightning flash is the product of the buildup and discharge of static electric energy between the charged regions of the cloud and the earth. The negatively charged lower region of the cloud induces a positive charge on the ground below. The tremendous electric forces between these 2 opposite charges initiate the lightning flash, which begins as a barely visible step leader moving in a series of steps downward from the cloud. Various objects on the ground (trees, chimneys, people, etc) can produce positively charged, upward streamers. The connection of the step leader with an upward streamer determines the connection point on the ground. After contact, a bright return stroke propagates upward from the ground, while electrons move downward toward the earth.[8] This entire phenomenon happens in less than a fraction of a second,[8] but a large amount of charge is transferred to the earth from the cloud.

Most lightning flashes have several return strokes, separated by only 0.004 to 0.005 seconds.[8] The human eye can barely resolve the intervals between the strokes that cause the lightning flash to appear to flicker. A lightning flash is essentially a brief spark, similar to that received from touching a doorknob after walking across a carpeted room. The lightning channel is approximately 2.54 cm (1 inch) in diameter and averages 4.83 to 8.05 km (3 to 5 miles) in vertical height but can be 9.66 km (6 miles) or higher.[8] Cloud-to-ground lightning flashes typically have peak currents ranging from 10 000 to 200 000 Å, and the electric potential between the cloud and ground can be 10 000 000 to 100 000 000 V.[8]

Thunder is created when lightning quickly heats the air around it, sometimes to temperatures greater than approximately 27 800°C (50 000°F), which is about 5 times hotter than the surface of the sun.[8] The rapidly heated air around a lightning channel explodes, which in turn creates the sound we hear as a clap of thunder.[8] The audible range of thunder is about 16.09 km (10 miles) but can be more or less depending on local conditions.[1] Heat lightning is intracloud or intercloud lightning that is too distant for the accompanying thunder to be heard.[8] Although it is possible to have lightning without thunder, thunder never occurs in the absence of lightning.

Lightning Casualty Demographics

On average, lightning kills approximately 100 people each year in this country, while many hundreds more are injured.[2–5] The death toll from lightning for 1940 to 1973 was greater than that from tornadoes and hurricanes combined.[17] Ninety-two percent of lightning casualties occur between May and September, while July has the greatest number of casualties.[2,3,7,18] Furthermore, 45% of the deaths and 80% of the casualties occurred in these months between 10:00 AM and 7:00 PM,[2,3,7,8] which coincides with the most likely time period for athletic or recreational events. For these reasons, it is accurate to say that lightning is the most dangerous and frequently encountered severe-storm hazard that most people experience each year.[10,11]

The statistics on lightning casualty demographics compiled from the National Oceanographic and Atmospheric Administration publication *Storm Data* for the state of Colorado over the last few decades demonstrate an increase in the number of lightning casualties in persons involved in sports and outdoor recreation.[7,10,18,19] Fifty-two percent of lightning casualties were people involved in outdoor recreation.[7,18] In addition, these authors noted that the highest number of casualties from lightning was recorded in recreational and sports activities for each year of the study.[18] During the 1960s, more than 30% of lightning casualties occurred during outdoor recreation activities; during the 1970s, that figure rose to 47%.[17] Furthermore, the rate of increase of lightning casualties during sports was higher than the general United States population rate of increase during the same time period.[7,18]

Lightning casualty statistics from Colorado demonstrate that the most common sites for fatalities were open fields (27%), near trees (16%), and close to water (13%).[7,8,18] Statistics from the country as a whole mimic the numbers from Colorado. Open fields, ballparks, and playgrounds accounted for nearly 27% of casualties, and under trees (14%), water-related (8%), and golf-related (5%) deaths associated with lightning followed.[19] All these fatalities had 1 common denominator: being near the highest object or being the tallest object in the immediate area. This single factor accounted for 56% of all fatalities from Colorado. Thus, it is imperative to avoid high ridges and high points on the terrain, and conversely, it is important to seek low-lying points on the terrain.[1,3,8,13–15]

The height above ground has been demonstrated to play a prominent role in determining the strike probability. Therefore, it is important to understand why minimizing vertical height is critical in decreasing the chances of becoming a victim of lightning. Warning signs of a high electromagnetic field and imminent lightning strike include hair standing on end and sounds similar to bacon sizzling or cloth tearing.[8] If these conditions occur, a cloud-to-ground lightning flash could strike in the immediate area. Therefore, one should immediately crouch in the lightning-safe position: feet together, weight on the balls of the feet, head lowered, and ears covered.[1] This position is intended to minimize the probability of a direct strike by both lowering the person's height and minimizing the area in contact with the surface of the ground. Taller objects are more likely to be struck (but not always) because their upward streamer occurs first, so that it is closer in proximity to the step leader coming downward from the cloud.

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

Mechanisms of Lightning Injury

Injury from lightning can occur via 5 mechanisms.[16] A direct strike most commonly occurs to the head, and lightning current enters the orifices. This mechanism explains why eye and ear injuries in lightning-strike victims are abundantly reported in the literature.[16] The shock wave created by the lightning channel can also produce injuries, such as rupture of the tympanic membrane, a common clinical presentation in the lightning-strike victim.[16,23,24] Recommending that individuals cover their ears while in the lightning-safe position may help to mitigate this type of injury.

The second mechanism, contact injury, occurs when the lightning victim is touching an object that is in the pathway of a lightning current.[16] Side flash, the third mechanism, occurs when lightning strikes an object near the victim and then jumps from that object to the victim. This is the main danger to a person who is sheltered under an isolated, tall tree.[6] An upward streamer is triggered by the tree but when this connects with the step leader, the resulting stroke jumps to the victim, who represents an additional pathway to ground.

The fourth mechanism, a step voltage or ground current, occurs when the lightning current flowing in the ground radiates outward in waves from the strike point. If 1 of the individual's feet is closer to the strike than the other, a step voltage is created.[6,16] Humans are primarily salt minerals in an aqueous solution, and a lightning current preferentially travels up from the earth through this solution (that is, the person) rather than through the ground. The greater the differential step voltage (ie, the greater the distance between the 2 feet), the greater the likelihood of injury. Placing one's feet close together while in the crouched position and not lying flat on the ground are crucial in reducing the likelihood of injury from a step voltage or ground current.

Blunt injury is the fifth mechanism for lightning-strike injuries. Lightning current can cause violent muscular contractions that throw its victims many meters from the strike point.

Explosive and implosive forces created by the rapid heating and cooling by the lightning current are also enough to produce traumatic injuries.[16]

Common Effects of Lightning Injury

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

Components of a Lightning-Safety Policy

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

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

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

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

Safe Locations

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

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

Unsafe Locations

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

From 1959 through 1965, lightning killed 4 people and injured 36 others while they were talking on the telephone. These numbers comprised 0.42% (n = 960) of deaths and 2.1% (n = 1736) of injuries for the period.[5] Studying reports from *Storm Data*, researchers found that between 1959 and 1994, 2.4% of lightning casualties were telephone related.[2] Because they are not connected directly to a land-line phone, cellular and cordless telephones are reasonably safe alternatives for summoning help during a thunderstorm. It should be noted that injury from acoustic damage can occur via explosive static from the earpiece caused by a nearby lightning strike.

Even though a swimming pool may be indoors and apparently safe, it can be a dangerous location during thunderstorms.[25] The current can be propagated through plumbing and electric connections via the underwater lights and drains of most swimming pools. Lightning current can also enter the building, either into the electric wiring inside the building or through underground plumbing pipelines that enter the building.[8] If lightning strikes the building or ground nearby, the current will most likely follow these pathways to the swimmers through the water. Thus, indoor-pool activities are potentially dangerous and should be avoided during thunderstorms.[25]

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

Criteria for Postponement and Resumption of Activities

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

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

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

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

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

Obligation to Warn

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

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

Table 1. The 30-30 Rule[15]

Criteria for suspension of activities	By the time the flash-to-bang count approaches 30 seconds, all individuals should already be inside a safe shelter.
Criteria for resumption of activities	Wait at least 30 minutes after the last sound (thunder) or observation of lightning before leaving the safe shelter to resume activities.

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

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

Prehospital Care of Victims

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

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

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

Table 2. Recommended Prehospital Care for Treating Lightning-Strike Victims[16]

Perform the following steps in order:
1. Survey the scene for safety.
2. Activate the local emergency management system.
3. Carefully move the victim to a safe area, if needed.
4. Evaluate and treat for apnea and asystole.
5. Evaluate and treat for hypothermia and shock.
6. Evaluate and treat for fractures.
7. Evaluate and treat for burns.

CONCLUSIONS

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

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

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

ACKNOWLEDGMENTS

This position statement was reviewed for the National Athletic Trainers' Association by the Pronouncements Committee, Richard Ray, PhD, ATC, and Philip Krider, PhD.

REFERENCES

1. Holle RL, López RE, Howard KW, Vavrek J, Allsopp J. Safety in the presence of lightning. *Semin Neurol.* 1995;15:375–380.
2. López RE, Holle RL, Heitkamp TA, Boyson M, Cherington M, Langford K. The underreporting of lightning injuries and deaths in Colorado. *Bull Am Meteorol Soc.* 1993;74:2171–2178.
3. Duclos PJ, Sanderson LM. An epidemiological description of lightning-related deaths in the United States. *Int J Epidemiol.* 1990;19:673–679.
4. Craig SR. When lightning strikes: pathophysiology and treatment of lightning injuries. *Postgrad Med.* 1986;79:109–112,121–123.
5. Zegel FH. Lightning deaths in the United States: a seven-year survey from 1959 to 1965. *Weatherwise.* 1967;20:169.
6. Andrews CJ, Cooper MA, Darveniza M. *Lightning Injuries: Electrical, Medical, and Legal Aspects.* Boca Raton, FL: CRC Press; 1992.
7. López RE, Holle RL. Demographics of lightning casualties. *Semin Neurol.* 1995;15:286–295.
8. Uman MA. *All About Lightning.* New York, NY: Dover Publications; 1986.
9. Kithil R. Annual USA lightning costs and losses. National Lightning Safety Institute. Available at: www.lightningsafety.com/nlsi_lls/nlsi_annual_usa_losses.htm. Accessed January 19, 1999.
10. Holle RL, López RE. Lightning: impacts and safety. *World Meterol Bull.* 1998;47:148–155.
11. Holle RL, López RE, Vavrek J, Howard KW. Educating individuals about lightning. In: *Preprints of the American Meteorological Society 7th Symposium on Education*; January 11–16, 1998; Phoenix, AZ.
12. Walsh KM, Hanley MJ, Graner SJ, Beam D, Bazluki J. A survey of lightning policy in selected Division I colleges. *J Athl Train.* 1997;32:206–210.
13. Bennett BL. A model lightning safety policy for athletics. *J Athl Train.* 1997;32:251–253.
14. Bennett BL, Holle RL, López RE. Lightning safety guideline 1D. *1997–98 National Collegiate Athletic Association Sports Medicine Handbook.* Overland Park, KS: National Collegiate Athletic Association; 1997–1998:12–14.
15. Vavrek JR, Holle RL, López RE. Updated lightning safety recommendations. In: *Preprints of the American Meteorological Society 8th Symposium on Education*; January 10–15, 1999; Dallas, TX.
16. Cooper MA. Emergent care of lightning and electrical injuries. *Semin Neurol.* 1995;15:268–278.
17. Weigel EP. Lightning: the underrated killer. *NOAA [National Oceanographic and Atmospheric Administration].* 1976;6:4–11.
18. López RE, Holle RL, Heitkamp TA. Lightning casualties and property damage in Colorado from 1950 to 1991 based on storm data. *Weather Forecast.* 1995;10:114–126.
19. Curran EB, Holle RL, López RE. *Lightning Fatalities, Injuries, and Damage Reports in the United States: 1959–1994.* Washington, DC: National Oceanic and Atmospheric Administration; 1997. Technical Memorandum NWS SR-193.
20. Cooper MA. Lightning: prognostic signs for death. *Ann Emerg Med.* 1980;9:134–138.
21. Primeau M, Engelstatter GH, Bares KK. Behavioral consequences of lightning and electrical injury. *Semin Neurol.* 1995;15:279–285.
22. Andrews CJ, Darveniza M. Telephone-mediated lightning injury: an Australian survey. *J Trauma.* 1989;29:665–671.
23. Fontanarosa PB. Electrical shock and lightning strike. *Ann Emerg Med.* 1993;22(Pt 2):378–387.
24. Steinbaum S, Harviel JD, Jaffin JH, Jordan MH. Lightning strike to the head: case report. *J Trauma.* 1994;36:113–115.
25. Wiley S. Shocking news about lightning and pools. *USA Swimming Safety Q.* 1998;4:1–2.
26. López RE, Holle RL. The distance between subsequent lightning flashes. In: *Preprints of the International Lightning Detection Conference*; November 17–18, 1998; Tucson, AZ.
27. Sanchez R, Wheeler L. Lightning strike at St. Albans game kills Bethesda student, injures 10. *Washington Post.* May 18, 1991;A1.
28. Keeton WP, Dobbs DB, Keeton RE, Owen DG. *Prosser and Keeton on Torts.* 5th ed. St. Paul, MN: West Publishing; 1984.
29. Black HC, Nolan JR, Nolan-Haley JM. *Black's Law Dictionary.* 6th ed. St. Paul, MN: West Publishing; 1990.
30. Cooper MA. Myths, miracles, and mirages. *Semin Neurol.* 1995;15:358–361.
31. Jepsen DL. How to manage a patient with lightning injury. *Am J Nurs.* 1992;92:38–42.

Media Reviews

***Journal of Sport Rehabilitation*, Special Issue: Management Strategies for Posterior Cruciate Ligament-Injured Athletes**
Paul Borsa, Editor

***Journal of Sport Rehabilitation*, Special Issue: Management Strategies for Posterior Cruciate Ligament-Injured Athletes**
Editor: Paul Borsa
Human Kinetics, Champaign, IL
Volume 8, Number 4
1999
136 pages
ISBN: 0-7360-1590-6
Price: $42 yearly subscription rate; special issue: $4, individuals; $33, institutions

Human Kinetics and its editors have assembled an excellent compilation on the management of posterior cruciate injuries. Luckily, injuries to the posterior cruciate ligament (PCL) are not the most frequent injuries to the knee, but when they do occur, they can be devastating or, as you learn in this issue, not so devastating.

The sophistication of the material presented in this special issue makes it suitable for the advanced undergraduate or graduate student in the study of orthopaedic injury. The editor has brought together some of the top researchers and clinicians, so the material is up to date and in depth.

The issue is so inclusive that it could be used as an entire unit on PCL injuries. The topics range from anatomy and biomechanics to natural history, injury mechanism, and evaluation to muscle function, proprioception, and rehabilitation. Not only would this information be good for a curriculum, but it would be very appropriate for continuing education. The issue supplies material that may not have been taught in previous curriculum studies.

Despite articles by different authors at different facilities, the material flows very well, the authors support each other, and repetition is minimal. I think what makes this information so useful is that it is science backing good sense. The management suggestions should be considered the standard of care for PCL injuries.

The first article is a research-packed review of the anatomic and biomechanical considerations of the PCL by Harner, Vogrin, and Woo. They not only describe the relevant anatomy but also discuss the biomechanics of the PCL and the reconstructed PCL. It is one of the best reviews I have seen.

Shelbourne and Gray review the limited knowledge of the natural history of the injured PCL. The third article reviews the surgical management of the injured PCL. Gastel et al note that most athletes with isolated PCL injuries do well with conservative treatment; those with concomitant injuries usually need surgical intervention. Their discussions lead the reader through the various types of surgical techniques and postoperative techniques. Of much interest these days is the role of proprioception when a joint structure is injured. Safran et al review their study on the reduction in proprioception when the PCL is torn and restoration of that loss with reconstruction. Yasuda et al present their data on the alteration of muscle-firing patterns in patients with torn PCLs. They suggest that this alteration may be a compensatory mechanism for the PCL-injured patient.

In the sixth article, Wilk et al discuss useful guidelines for rehabilitation of nonoperative and postoperative PCL patients based on tissue healing, with advancement with determined by objective functional progression. An excellent table allows the reader to see the entire set of guidelines at one time. Finally, Borsa and et al explain the functional restoration of the PCL-injured athlete. Key concepts for functional training are explained, and a complete progression for restoring dynamic joint stability is provided, with excellent illustrations.

The *Journal of Sport Rehabilitation* earlier published a special issue on anterior cruciate ligament injuries. This publication is an excellent addition to it: very affordable and a real value. I have not seen a better offering when it comes to this special topic. The editors have presented a well-orchestraded publication that is a valuable tool for both educators and clinicians.

Turner A. "TAB" Blackburn, Jr, MEd, PT, ATC
Tulane Institute of Sports Medicine
New Orleans, LA

Indexes

AUTHOR INDEX
Volume 35

SUBJECT INDEX
Volume 35

B

C

D

E

F

G

H

I

J

K

L

M

N

O

P

Q

R

S

Authors' Guide

(Revised January 1999)
The mission of the *Journal of Athletic Training* is to enhance communication among professionals interested in the quality of health care for the physically active through education and research in prevention, evaluation, management, and rehabilitation of injuries.

SUBMISSION POLICIES

1. Submit 1 original and 5 copies of the entire manuscript (including figures and tables) to: *Journal of Athletic Training* Submissions, Hughston Sports Medicine Foundation, Inc, 6262 Veterans Parkway, PO Box 9517, Columbus, GA 31908. The term *figure* refers to items that are not editable, either halftones (photographs) or line art (charts, graphs, tracings, schematic drawings), or combinations of the two. A *table* is an editable item that needs to be typeset.
2. All manuscripts must be accompanied by a letter signed by each author and must contain the following statements: "This manuscript 1) contains original unpublished material that has been submitted solely to the *Journal of Athletic Training*, 2) is not under simultaneous review by any other publication, and 3) will not be submitted elsewhere until a decision has been made concerning its suitability for publication by the *Journal of Athletic Training*. In consideration of the NATA's taking action in reviewing and editing my submission, I the undersigned author hereby transfer, assign, or otherwise convey all copyright ownership to the NATA, in the event that such work is published by the NATA. Further, I verify that I have contributed substantially to this manuscript as outlined in item #3 of the current Authors' Guide." By signing the letter, the authors agree to comply with all statements. Manuscripts that are not accompanied by such a letter will not be reviewed. Accepted manuscripts become the property of the NATA. Authors agree to accept any minor corrections of the manuscript made by the editors.
3. Each author must have contributed to the article. This means that all coauthors should have made some useful contribution to the study, should have had a hand in writing and revising it, and should be expected to be able to defend the study publicly against criticism.
4. Financial support or provision of supplies used in the study must be acknowledged. Grant or contract numbers should be included whenever possible. The complete name of the funding institution or agency should be given, along with the city and state in which it is located. If individual authors were the recipients of funds, their names should be listed parenthetically.
5. Authors must specify whether they have any commercial or proprietary interest in any device, equipment, instrument, or drug that is the subject of the article in question. Authors must also reveal if they have any financial interest (as a consultant, reviewer, or evaluator) in a drug or device described in the article.
6. For experimental investigations of human or animal subjects, state in the methods section of the manuscript that an appropriate institutional review board approved the project. For those investigators who do not have formal ethics review committees (institutional or regional), the principles outlined in the Declaration of Helsinki should be followed (41st World Medical Assembly. Declaration of Helsinki: recommendations guiding physicians in biomedical research involving human subjects. *Bull Pan Am Health Organ.* 1990;24:606–609). For investigations of human subjects, state in the methods section the manner in which informed consent was obtained from the subjects. (Reprinted with permission of *JAMA* 1997;278:68, copyright 1997, American Medical Association.)
7. Signed releases are required to verify permission for the *Journal of Athletic Training* 1) to reproduce materials taken from other sources, including text, figures, or tables; 2) to reproduce photographs of individuals; and 3) to publish a Case Report. A Case Report cannot be reviewed without a release signed by the individual being discussed in the Case Report. Release forms can be obtained from the Editorial Office and from the *JAT* web page, or authors may use their own forms.
8. The *Journal of Athletic Training* uses a double-blind review process. Authors should not be identified in any way except on the title page.
9. Manuscripts are edited to improve the effectiveness of communication between author and readers and to aid the author in presenting a work that is compatible with the style policies found in the *AMA Manual of Style*, 9th ed. (Williams & Wilkins), 1998. Page proofs are sent to the author for proofreading when the article is typeset for publication. It is important that they be returned within 48 hours. Important changes are permitted, but authors will be charged for excessive alterations.
10. Published manuscripts and accompanying work cannot be returned. Unused manuscripts will be returned if submitted with a stamped, self-addressed envelope.

STYLE POLICIES

11. Each page must be printed on 1 side of 8½-by-11-inch paper, double spaced, with 1-inch margins in a font no smaller than 10 points. Each page should include line counts to facilitate the review process. Do not right justify pages.
12. Manuscripts should contain the following, organized in the order listed below, with each section beginning on a separate page:
    a. Title page
    b. Acknowledgments
    c. Abstract and Key Words (first numbered page)
    d. Text (body of manuscript)
    e. References
    f. Tables (each on a separate page)
    g. Legends to figures
    h. Figures
13. Begin numbering the pages of your manuscript with the abstract page as #1; then, consecutively number all successive pages.
14. Units of measurement shall be recorded as SI units, as specified in the *AMA Manual of Style*, except for angular displacement, which should be measured in degrees rather than radians. Examples include mass in kilograms (kg), height in centimeters (cm), velocity in meters per second ($m{\cdot}s^{-1}$ or m/s), angular velocity in degrees per second ($°{\cdot}s^{-1}$), force in Newtons (N), and complex rates (mL/kg per minute).
15. Titles should be brief within descriptive limits (a 16-word maximum is recommended). If a disability is the relevant factor in an article, the name of the disability should be included in the title. If a technique is the principal reason for the report, it should be in the title. Often both should appear.
16. The title page should also include the name, title, and affiliation of each author, and the name, address, phone number, fax number, and E-mail address of the author to whom correspondence is to be directed.
17. A structured abstract of no more than 250 words must accompany all manuscripts. Type the complete title (but not the authors' names) at the top, skip 2 lines, and begin the abstract. Items that are needed differ by type of article. **Literature Review:** Objective, Data Sources, Data Synthesis, Conclusions/Recommendations, and Key Words; **Original Research** articles: Objective, Design and Setting, Subjects, Measurements, Results, Conclusions, and Key Words; **Case Reports:** Objective, Background, Differential Diagnosis, Treatment, Uniqueness, Conclusions, and Key Words; **Clinical Techniques:** Objective, Background, Description, Clinical Advantages, and Key Words. For the Key Words entry, use 3 to 5 words that do not appear in the title.
18. Begin the text of the manuscript with an introductory paragraph or two in which the purpose or hypothesis of the article is clearly stated and developed. Tell why the study needed to be done or the article written and end with a statement of the problem (or controversy). Highlights of the most prominent works of others as related to your subject are often appropriate for the introduction, but a detailed review of the literature should be reserved for the discussion section. In a 1- to 2-paragraph review of the literature, identify and develop the magnitude and significance of the controversy, pointing out differences among others' results, conclusions, and/or opinions. The introduction is not the place for great detail; state the facts in *brief* specific statements and reference them. The detail belongs in the discussion. Also, an overview of the manuscript is part of the abstract, not the introduction. Writing should be in the active voice (for example, instead of "Subjects were selected...," use "We selected subjects...") and in the first person (for example, instead of "The results of this study showed...," use "Our results showed...").
19. The body or main part of the manuscript varies according to the type of article (examples follow); however, the body should include a discussion section in which the importance of the material presented is addressed and related to other pertinent literature. Liberal use of headings and subheadings, charts, graphs, and figures is recommended.
    a. The body of an **Original Research** article consists of a methods section, a presentation of the results, and a discussion of the results. The methods section should contain sufficient detail concerning the methods, procedures, and apparatus employed so that others can reproduce the results. The results should be summarized using descriptive and inferential statistics and a few well-planned and carefully constructed illustrations.
    b. The body of a **Literature Review** article should be organized into subsections in which related thoughts of others are presented, summarized, and referenced. Each subsection should have a heading and brief summary, possibly one sentence. Sections must be arranged so that they progressively focus on the problem or question posed in the introduction.
    c. The body of a **Case Report** should include the following components: personal data (age, sex, race, marital status, and occupation when relevant—but not name), chief

Authors' Guide

complaint, history of present complaint (including symptoms), results of physical examination (example: "Physical findings relevant to the rehabilitation program were . . ."), medical history (surgery, laboratory results, examination, etc), diagnosis, treatment and clinical course (rehabilitation until and after return to competition), criteria for return to competition, and deviation from expectations (what makes this case unique).

d. The body of a **Clinical Techniques** article should include both the *how* and *why* of the technique: a step-by-step explanation of how to perform the technique, supplemented by photographs or illustrations, and an explanation of why the technique should be used. The discussion concerning the *why* of the technique should review similar techniques, point out how the new technique differs, and explain the advantages and disadvantages of the technique in comparison with other techniques.

Percentages should be accompanied by the numbers used to calculate them.

20. **Communications** articles, including official Position Statements and Policy Statements from the NATA Pronouncements Committee; technical notes on such topics as research design and statistics; and articles on other professional issues of interest to the readership are solicited by the *Journal*. An author who has a suggestion for such a paper is advised to contact the Editorial Office for instructions.
21. The manuscript should not have a separate summary section—the abstract serves as a summary. It is appropriate, however, to tie the article together with a summary paragraph or list of conclusions at the end of the discussion section.
22. References should be numbered consecutively, using superscripted arabic numerals, in the order in which they are cited in the text. References should be used liberally. It is unethical to present others' ideas as your own. Also, use references so that readers who desire further information on the topic can benefit from your scholarship.
23. References to articles or books, published or accepted for publication, or to papers presented at professional meetings are listed in numerical order at the end of the manuscript. Journal title abbreviations conform to *Index Medicus* style. Examples of references are illustrated below. See the *AMA Manual of Style* for other examples.

Journals:

1. van Dyke JR III, Von Trapp JT Jr, Smith BC Sr. Arthroscopic management of postoperative arthrofibrosis of the knee joint: indication, technique, and results. *J Bone Joint Surg Br.* 1995;19:517–525.
2. Council on Scientific Affairs. Scientific issues in drug testing. *JAMA.* 1987;257: 3110–3114.

Books:

1. Fischer DH, Jones RT. *Growing Old in America.* New York, NY: Oxford University Press Inc; 1977:210–216.
2. Spencer JT, Brown QC. Immunology of influenza. In: Kilbourne ED, Gray JB, eds. *The Influenza Viruses and Influenza.* 3rd ed. Orlando, FL: Academic Press Inc; 1975:373–393.

Presentations:

1. Stone JA. Swiss ball rehabilitation exercises. Presented at: 47th Annual Meeting and Clinical Symposia of the National Athletic Trainers' Association; June 12, 1996; Orlando, FL.

Internet Sources:

1. Knight KL, Ingersoll CD. Structure of a scholarly manuscript: 66 tips for what goes where. Available at http://www.journalofathletictraining.org/jat/66tips.html. Accessed January 1, 1999.
2. National Athletic Trainers' Association. NATA blood borne pathogens guidelines for athletic trainers. Available at http://www.journalofathletictraining.org. Accessed January 1, 1999.

24. Table Style: 1) Title is bold; body and column headings are roman type; 2) units are set above rules in parentheses; 3) numbers are aligned in columns by decimal; 4) footnotes are indicated by symbols (order of symbols: *, †, ‡, §, ||, ¶); 5) capitalize the first letter of each major word in titles; for each column or row entry, capitalize the first word only. See a current issue of the *Journal* for examples.
25. All black and white line art should be submitted in camera-ready form. Line art should be of good quality; should be clearly presented on white paper with black ink, sans serif typeface, and no box; and should be printed on a laser printer—no dot matrix. Figures that require reduction for publication must remain readable at their final size (either 1 column or 2 columns wide). Photographs should be glossy black and white prints. Do not use paper clips, write on photographs, or attach photographs to sheets of paper. On the reverse of each figure attach a write-on label with the figure number, name of the author, and an arrow indicating the top. (Note: Prepare the label before affixing it to the figure.) Authors should submit 1 original of each figure and 5 copies for review.
26. Authors must request color reproduction in a cover letter with the submitted manuscript. Authors will be notified of the additional cost of color reproduction and must confirm acceptance of the charges in writing.
27. Legends to figures are numbered with Arabic numerals in order of appearance in the text. Legends should be printed on separate pages at the end of the manuscript.
28. The *Journal of Athletic Training* follows the redundant publication guidelines of the Council of Biology Editors, Inc (*CBE Views.* 1996;19:76–77; also available on the *JAT* web site at www.nata.org/jat). Authors found in violation of redundant publication will have sanctions invoked by the Journal Committee of the National Athletic Trainers' Association, Inc.

ADVERTISERS' INDEX