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**CEU Quiz**
The CEU quiz for the September 2002 issue (Volume 37, Number 3) of the *Journal of Athletic Training* will be located in the October 2002 *NATA News.*

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The NATA Pronouncements Committee is seeking professionals to participate in the development and writing of several position statements currently being planned.

**Topics for which writing group participants are needed include:**

- Weight Control in Weight-Class Sports
- Management of Skin Disorders
- Cold Injuries

**TO PARTICIPATE**

Those who possess an interest and expertise in any of these three areas are encouraged to contact Pronouncements Committee Chair Douglas Casa via e-mail by Nov. 15, 2002, to inquire about joining the position statement writing groups.

**Douglas J. Casa, PhD, ATC, FACSM**
Director, Athletic Training Education
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1. General Grants Program for 2002
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5. Exercise by Children and Adolescents in Warm and Hot Environments
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Hey! Where’s My Cheese?

Chad Starkey

*Who Moved My Cheese?*\(^1\) is a pop culture book that examines the process of change and how change (or the lack of change) affects people’s lives and their goals. Each of us has inherent mechanisms that help us cope with change and overcome the fear associated with it. Change often represents moving out of a comfort zone into new territory. Change is good. Change is needed. Change can also be scary. Change is certainly the operative word in the preparation and certification of entry-level athletic trainers.

Just over 10 years ago, the National Athletic Trainers’ Association, Inc (NATA) was essentially a closed shop. Our national organization set the standards, approved entry-level and graduate programs, established the requirements to sit for the certification examination, created the examination, and administered the examination.

From a practical standpoint, that method was easy to comprehend: NATA regulated everything. When it came to asking questions, getting answers, or pointing fingers, you knew where to go. Procedurally, however, this was not an appropriate method.

Under the old system, the NATA Board of Directors ultimately acted as the law, judge, and jury. Because of this, “NATA approval” of education programs and “NATA certification” were only recognized by one agency: NATA.

To their credit, the Board of Directors realized the need to divest these functions. For certification of athletic trainers and accreditation of entry-level education programs to be recognized and accepted, organizational changes were needed. The membership agency, the certifying agency, and the accrediting agency must be separate entities. This system forms a healthy, functional system of checks and balances.

Although these agencies have been separate from NATA for more than a decade, it appears that some people are now beginning to recognize that change has occurred. (How many times have you heard someone refer to an NATA-certified athletic trainer? The NATA has not granted certification since 1991.) For many, “Education Reform” has placed our educational process under the microscope and caused us to reevaluate a number of longstanding procedures. However, most of the changes and issues facing us are unrelated to the actual reform process.

We are now noticing the totality of the changes that have been occurring over the last decade and balancing them with the changes recommended by the NATA Education Task Force and approved by the Board of Directors in 1997. Our cheese is being moved.

The change from our hours-based approach to clinical education to an objective, practice-oriented approach is significant. Some programs have responded to this emphasis by creating massive checklists for student evaluation. While this approach is easy to conceptualize and construct, it is not easy to implement. This method creates undue demands on clinical instructors and faculty, suppresses the need for clinical decision making, and oversimplifies the concepts of learning, teaching, and reasoning.

Along with putting the “education” into clinical education, some members perceive that education reform has taken away from the students’ ability to practice and work unsupervised. Nothing can be further from the truth. Since 1991, the NATA Board of Certification has required students to be supervised while functioning as athletic training students (ie, while garnering clinical hours).

The presence of a supervisor does not imply a lack of student autonomy and decision making. The two are not contradictory terms. Proper supervision can allow for independent decision making on the part of the student. Supervisor intervention is only required if the student is about to cause harm.

Concern has also been expressed about the “expanded scope” of the general medical competencies. In fact, all but 20 of the 91 specific general medical conditions listed were included in the 1992 edition of *Athletic Training Educational Competencies*\(^2\) (most are found on pages 23 and 24), including those that have become symbolic of the new standard. Other content was added that reflects our expanding places of employment and the increasing diversity in our patient population. Fear of expanding our base of knowledge and perhaps our scope beyond “just” orthopaedic conditions can be self-limiting. A review of the epidemiologic articles published in the *Journal of Athletic Training* indicates that 25% to 30% of the conditions seen by certified athletic trainers are nonorthopaedic.

No programs have yet been evaluated under the new standards and guidelines. Reports that programs have not received accreditation because they failed to meet the new competencies or clinical education guidelines or abide by any other change attributed to education reform are not true. The first site visitations using the new Joint Review Committee on Educational Programs in Athletic Training standards\(^3\) will be conducted shortly after this editorial is published. After that, we can begin to analyze what is—and what is not—working.
Accreditation is not an automatic process. No one can say in good conscience that every program should receive accreditation. Part of education reform is raising the bar. When the bar is raised, there is no guarantee that every program will make it over.

The education-reform process has been the most open in the history of this profession. Starting with the Education Task Force in 1994, the NATA, the NATA Board of Certification, and the Joint Review Committee on Educational Programs in Athletic Training have sought member input. Three drafts of the revised competencies and clinical proficiencies were opened for member feedback, as were the clinical education guidelines. More than 30 informational articles have appeared in our professional publications. Yes, our cheese has been moved, but we were told that it was moving and where it was going.

Fear of change can cause unforeseen reactions and can bring out the best—or worst—in people. I am proud of the manner in which most of our members have responded to the call to improve our educational process. I can only hope that the sound of many whispers of support can drown out the few screams of dissent.

Change can be scary. But the cheese is still there.

References

Chad Starkey, PhD, ATC, is an Associate Professor of Athletic Training at Northeastern University, Boston, MA, and the Chair of the NATA Education Council Executive Committee.
I agree with Dr Draper’s assessment of the certified athletic trainer’s education and ability to use therapeutic modalities safely and effectively (Draper DO. Are certified athletic trainers qualified to use therapeutic modalities? J Athl Train. 2002;37:11–12).

Having the training, certification, and, in many cases, the license to provide therapeutic modalities does not always equate with being able to provide this treatment in all practice situations.

Practice acts for physicians, licensed athletic trainers, and physical therapists are put in place by each state to protect its citizens, and they vary immensely from state to state.

Some practice acts allow a professional to have another professional deliver care under supervision (ie, a physician can have a certified licensed athletic trainer deliver therapeutic modalities in his or her facility, call it medical care, and charge for it even though the physician did not deliver the service).

Some practice acts allow only the professional to deliver the care and do not allow anyone else, no matter how qualified, to deliver that care (ie, a physical therapist must perform the therapeutic modality if he or she is providing physical therapy services).

Then there are practice acts that provide for every situation in between. Some physical therapy practice acts allow therapists to use anyone they deem appropriate to deliver the care as long as the therapist provides line-of-sight supervision. Even though these therapists are not breaking their state law to have a certified or licensed athletic trainer deliver the service under line-of-sight supervision, they may be violating federal standards or standards set forth by third-party payers.

Medicare requires that when physical therapy services are being delivered, only a physical therapist or physical therapist’s assistant may perform the service or the fees will not be reimbursed. Medicare facilities, such as most hospitals and Medicare-recognized outpatient physical therapy clinics, must follow Medicare guidelines in the delivery of care in their institution.

Most current procedural terminology (CPT) codes can be used by any appropriate professional in the delivery of care he or she is licensed to provide; however, by definition, CPT codes are to be used by the professional delivering the care. In other words, if athletic training services are being delivered, a certified licensed athletic trainer should perform the service. If physical therapy services are being delivered, a physical therapist should deliver the care. So even using CPT codes—the backbone of reimbursement—puts demands on the provider to have the appropriately licensed individual deliver the care.

Maybe this is all semantics, but certified, licensed athletic trainers should deliver athletic training services as defined by their practice act. These services should not be performed for another profession. But also remember, the practice act is not the sole determination of reimbursement.

The precedent is already set. Physical therapists and occupational therapists work as a team to care for their patients, and their practice acts overlap. Both can perform therapeutic modalities and both can perform activities of daily living. However, they each have different scopes of service that provide unique services that meet patient needs.

Why should physical therapists have certified licensed athletic trainers perform ultrasound or any other service for them? Athletic training has a scope of practice that includes activities other professionals do not have. Let’s develop that market delineation within our own practice act rather than trying to work within another profession’s practice act where we are recognized as aides.

Turner A. “Tab” Blackburn, Jr, MEd, ATC, PT
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Glenohumeral Stiffness Response Between Men and Women for Anterior, Posterior, and Inferior Translation

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Paul A. Borsa, PhD, ATC, contributed to conception and design; analysis and interpretation of the data; and drafting, critical revision, and final approval of the article. Eric L. Sauers, PhD, ATC, contributed to conception and design; acquisition of the data; and drafting, critical revision, and final approval of the article. Derald E. Herling, PhD, PE, contributed to conception and design and critical revision and final approval of the article.

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Objective: To quantify and compare the glenohumeral stiffness response in anterior-directed, posterior-directed, and inferior-directed translations in healthy men and women.

Design and Setting: We used a 2 × 3 factorial design and employed a device capable of measuring glenohumeral joint displacement as a function of force to gather kinematic data during a single test session.

Subjects: Twenty subjects with healthy nondominant shoulders participated in the study.

Measurements: Force-displacement measures were taken in the anterior, posterior, and inferior translational directions of the glenohumeral joint. These measurements simulated common laxity tests used at the shoulder.

Results: Analysis of variance revealed a nonsignificant sex × direction interaction effect (P > .05). The main effect for sex and direction was also not significant (P > .05).

Conclusions: Our results suggest that (1) glenohumeral stiffness is widely distributed in healthy shoulders, (2) glenohumeral stiffness is not significantly different between men and women, and (3) glenohumeral stiffness is not significantly different among directions of translations.

Key Words: force displacement, joint laxity, assessment, arthrometer, shoulder

Techniques that employ force-displacement maneuvers are often used in clinical orthopaedic practice. Laxity tests assess the integrity of the joint capsule and ligaments,1-4 and joint mobilizations restore normal range of motion in stiff joints.5,6 With the application of a manual force, the clinician can subjectively gauge the joint’s resistance to translation or stiffness.2-4 Glenohumeral (GH) stiffness is a reflection of the articular structures’ resistance to humeral head translation and is quantified as the amount of force (N) required to displace a joint by a given amount (mm).7-9 Stiffness measures provide information concerning the structural and mechanical properties of the joint and are considered clinically important when assessing joint stability.7-10 Biomechanical studies have shown that a stiffer joint is able to absorb more force during periods of loading11,12 and, therefore, may decrease the risk of injury such as dislocation or subluxation.

It is important to model the stiffness response of the GH joint as an aid to better analysis and understanding of the joint’s arthrokinematic and mechanical behavior. Mechanical springs and dashpots are common models used in clinical biomechanics. Mechanical springs model the stiffness response; dashpots model the strain-rate nature of viscoelastic material. Both mechanical springs and dashpot models have been applied to the shoulder.13-15

Despite a great deal of previous research into the normal and abnormal function of the shoulder, the current information regarding in vivo quantitative force-displacement characteristics of the GH joint is limited. The lack of instrumented arthrometers similar to those used at the knee is cited as the primary limiting factor.9,16-20 Instrumented arthrometers for the knee have enabled researchers to quantify translatory kinematics in various populations.21-26 Furthermore, objective measurement of tibial translation obtained using instrumented knee arthrometers has proven effective for predicting injury status22-24 and the efficacy of various surgical intervention techniques.25-26 Recent publications have underscored the need for more objective means to measure shoulder arthokinematics.18-20 Therefore, the purpose of our investigation was to quantify and compare GH stiffness response among anterior-directed, posterior-directed, and inferior-directed translations in the shoulders of healthy men and women.

METHODS

Subjects and Design

The data presented in this manuscript are part of a larger study characterizing shoulder kinematics in healthy shoulders.27 Force-displacement data were collected on 20 healthy, nondominant shoulders (11 women, 9 men, mean age = 20.9 ± 3.6 years) during a single test session. Subjects were asymptomatic, with no previous history of shoulder injury involving
Figure 1. Instrumentation setup and subject positioning for (A) anterior-directed and (B) posterior-directed translations. The transmitter was mounted to the chair frame above and behind the subject, and motion sensors were taped to the acromion process and proximal humerus.

Instrumentation

Force-displacement measures were taken in the anterior, posterior, and inferior directions using an instrumented arthrometer. A test chair equipped with nylon strapping provided the base of support for testing. Displacement forces were applied to the joint with a custom force applicator. The force applicator consists of a plastic handle mounted to a full-bridge, thin-beam load cell (model LC105-50, Omega Engineering Inc, Stamford, CT) that has a range from 0 to 222 N. A hook attached at the opposite end of the load cell secures the force applicator to an arm cuff. The arm cuff is designed to ensure equal force distribution during testing.

Translations were measured using an electromagnetic spatial-tracking device (Polhemus 3Space Fasttrak, Colchester, VT) consisting of a transmitter and 2 sensors (receivers). The unit contains the hardware necessary to generate and sense magnetic fields, compute position and orientation, and interface with a host computer. A global x, y, z coordinate system was established by mounting the transmitter on a composite base above and behind the subject, aligned with the cardinal planes of the body. On pre-experiment calibration measures, we found the device to be accurate within 0.1 to 0.2 mm for linear translations.

Test Procedures

Subjects were seated and secured comfortably in the test chair with nylon strapping. For anterior and posterior translations, the humerus of the nondominant arm was positioned in 20° of abduction with the elbow secured in neutral rotation. The test position is similar to that for the load-and-shift and anterior-posterior drawer tests (Figures 1A and B). The acromion process was located via palpation, and the acromion sensor was affixed cutaneously to its superior aspect. The humeral head was located via palpation to determine the position for placement of the proximal humeral sensor. The humeral head sensor was then affixed over the lateral aspect of the proximal humerus directly below the acromion sensor. Each sensor was secured to the skin surface with self-adhesive tape (Cover-Roll Stretch, Beiersdorf Inc, Norwalk, CT). Next, the arm cuff was secured firmly around the proximal humerus. The experimenter stabilized the scapula with his thumb (coracoid process) and index finger (scapular spine) during each trial. Anterior and posterior forces were applied to the nondominant arm by pulling linearly in the respective translational direction until a capsular endpoint was achieved.
For inferior translation, the nondominant arm was placed in the resting position with the elbow flexed to 90° and the forearm pronated with the palms resting on a wooden platform (Figure 2). The acromion sensor placement remained the same as for anterior and posterior testing, but the humeral sensor was secured over the lateral epicondyle with self-adhesive tape (Cover-Roll Stretch). The cuff was placed around the proximal forearm just distal to the elbow joint. The examiner applied force to the nondominant arm by pulling downward in line with the longitudinal axis of the humerus until a capsular endpoint was achieved. This test position replicated that for the sulcus or inferior-distraction test.2,3

For each trial, a “start” or zero reference position was determined. Once the start position was secured, a controlled force was applied to the joint using the force applicator until capsular endpoint was reached. The rate of force application to the joint was uniform and did not appear to adversely affect muscle reaction or capsular stiffness. Capsular endpoint was achieved in approximately 3 to 4 seconds. Three trials were completed for each direction, and the average was recorded to the nearest 0.1 mm.

Linear displacements of the acromion process (scapular protraction and retraction) and humeral head (humeral translation) were calculated from the Cartesian coordinates of the 2 sensors. The displacement of the scapula was subtracted from the absolute humeral displacement, giving true humeral head translation.10,16,17 Intraexaminer and interexaminer reproducibility of our technique was established and reported in earlier publications.16,17 Additionally, our technique was validated in a previous study by comparing force-displacement values obtained using our current method of cutaneously applied sensors with a method of directly pinning the sensors to the bony reference points (humeral head and acromion process) in 30 fresh-frozen cadaver shoulders.28 Bone fixation of the displacement sensors enabled accurate measurement of glenohumeral translation and thus served as our “gold standard.” The measures from the cutaneously applied sensors showed good to excellent agreement with the bone-pinned measurements. Correlation coefficients were moderate to good for anterior-directed (P = .79), posterior-directed (P = .68), and inferior-directed (P = .71) translations.28

Data Acquisition and Display

Raw data from the tracking device were fed to the host computer via a serial port, while data from the load cell (force) were fed to the host computer via a data-acquisition card. A custom software program (Visual Basic 6.0, Microsoft Inc, Redmond, WA) simultaneously and sequentially captured force and displacement data for each test trial at intervals of approximately 0.1 second. From these data, a force-displacement curve was displayed for each trial using only the data points from the start of the force application (~5 N) to a discernable endpoint. Endpoint was identified from our raw data as the point on the force-displacement curve at which increasing the applied force failed to produce associated translation.27 Stiffness values were calculated from the slope of the force-displacement curve (Figure 3). From the force-displacement curve, our custom software program identified the most linear region of the curve and calculated the slope using a simple linear regression analysis. Using Figure 3 as an example, the most linear region of the force-displacement curve was identified, and the slope of the curve was calculated using the associated linear regression equation y = bx—a, where y = predicted value (force), b = slope (stiffness value), x = predictor value (displacement), and a = intercept. From this calculation, the slope was found to be 25.6 N/mm, which was our stiffness value for that trial. This model has been used in previous studies of joint and muscle stiffness.8-13 Three trials were performed for each direction, and the average score from the 3 trials was recorded as the stiffness value.

Statistical Analysis

We used an online power analysis (http://www.math.yorku.ca/SCS/Demos/power/). We entered our design as 2 (sex) × 3 (direction). Using our previous stiffness data (mean differences/SD) on stiffness, we found our effect size to be 0.8 (medium effect size). For the online calculation, we entered 2 effect sizes (0.5 and 0.75) with alpha set at 0.05. For our design, 20 subjects were necessary to achieve statistical power between .775 and .982.

A 2 (sex) × 3 (direction) analysis of variance was used to determine significant mean (±SD) differences between sex and directions for stiffness. Tukey Honestly Significant Difference post hoc analyses were used in the presence of a significant interaction or main effect. All data analyses were performed using Statistical Program for Scientific Studies for Windows (version 10.0, SPSS Inc, Chicago, IL). The level of statistical significance was set at .05.

RESULTS

Descriptive data are presented in Table 1. Analysis of variance revealed a nonsignificant sex × direction interaction ef-
flect for joint stiffness (F_{2,59} = 3.01, P = .06). The main effects for sex (F_{2,59} = .546, P = .46) and direction (F_{2,59} = .612, P = .55) were also not significant (Figure 4).

**DISCUSSION**

Stiffness is a physiologic variable that can be used to characterize the mechanical behavior of a joint.\(^5,6,9,12\) Joint-stiffness values can be calculated from the slope of the force-displacement curve using a least squares or linear regression model (Figure 3).\(^7,9,10\) The in vivo force-displacement curves reported in this study bear a striking resemblance to those of Markolf et al.\(^7,8\) in healthy knees and McQuade et al.\(^9\) in healthy shoulders. On average, our force-displacement curves demonstrated a discernable biphasic pattern of an early, nonlinear response, followed by a linear response after about the 60- to 80-N force level (Figure 3). The early, nonlinear stiffness response is presumed to be the “toe” region, or period when the collagen fibers of the joint capsule are being recruited and are not yet under tension, followed by the mid- to end-range linear stiffness response, indicating that the mechanical restraints are progressively developing tension. The stiffness values reported in this study were calculated from the most linear region of the force-displacement curve, which was most often in the end-range or region of highest force application (Figure 3). This region differed slightly for each individual, depending on the force-displacement response of the individual’s joint. During clinical laxity examinations, the clinician gauges joint stiffness at the end-range or endpoint of translation. This is why our aim was to measure end-range stiffness of the joint and not early- and mid-range stiffness.

**Interindividual Differences**

Mechanical restraint to GH translation is provided by both bony and soft tissue structures.\(^29-34\) The angle and depth of the glenoid cavity,\(^20\) capsuloligamentous tightness,\(^30-33\) and overlying rotator cuff tendons\(^34\) contribute to the overall stiffness of the joint. We found GH joint stiffness data to be widely yet normally distributed, with a range of values from 9.2 to 28.4 N/mm, depending on direction of translation in healthy shoulders (Table 1). The interindividual differences in joint stiffness reported in Table 1 are likely to result from variability in any or all of these aforementioned factors.

Borsa et al.\(^10\) Sauers et al.\(^28\) and McQuade et al.\(^9\) earlier reported on force-displacement measures in healthy shoulders. We\(^10\) used a load cell and linear potentiometers to quantify anterior-posterior translation in a large population of healthy shoulders and reported mean force-displacement values ranging from 16 to 22 N/mm. Sauers et al.\(^28\) quantified humeral translations in the anterior, posterior, and inferior directions using fresh-frozen cadaver shoulders; force-displacement measures were obtained with a load cell and an electromagnetic spatial-tracking system. The sensors were pinned directly to the bony reference points in order to isolate bony movement. Sauers et al.\(^28\) found mean stiffness values ranging from 21 to 44 N/mm. McQuade et al.\(^9\) also used a palm-held load cell and an electromagnetic spatial-tracking system to quantify in vivo anterior and posterior humeral translations in multiple degrees of abduction and rotation. McQuade et al.\(^9\) reported force-displacement curves that closely resembled ours currently and previously\(^10\) and those of Sauers et al.\(^28\) The mean stiffness values from Sauers et al.\(^28\) were considerably higher than ours, possibly because they used fresh-frozen cadaveric specimens, which may have had higher resistance properties due to the preservation process. Also, the scapulae of the specimens were rigidly mounted to the test jig, which may have produced additional resistance during force application. Results from these studies are summarized in Table 2.

**Sex and Directional Differences**

We did not show any significant difference in GH joint stiffness between men and women. McQuade et al.\(^9\) also reported no significant sex differences in stiffness. Borsa et al.\(^10\) found that women with healthy shoulders had significantly less anterior joint stiffness than men with healthy shoulders. These findings at the glenohumeral joint are consistent with those at the knee revealing decreased stiffness in women.\(^8\) More research is warranted in order to draw conclusions concerning sex-specific differences in GH joint stiffness.

**Table 2. Studies Assessing Glenohumeral Joint Stiffness**

<table>
<thead>
<tr>
<th>Investigators</th>
<th>Instrumentation</th>
<th>Shoulders</th>
<th>Stiffness Values (N/mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current study</td>
<td>Load cell, electromagnetic spatial-tracking system</td>
<td>20</td>
<td>Anterior: 16.7 ± 5.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Posterior: 15.4 ± 3.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Inferior: 15.7 ± 5.6</td>
</tr>
<tr>
<td>Borsa et al.(^10)</td>
<td>Load cell, linear potentiometers</td>
<td>102</td>
<td>Anterior: 18.2 ± 5.0</td>
</tr>
<tr>
<td>McQuade et al.(^9)</td>
<td>Load cell, electromagnetic spatial-tracking system</td>
<td>21</td>
<td>Posterior: 21.8 ± 8.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Anterior: 15–23</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Posterior: 17–27</td>
</tr>
<tr>
<td>Sauers et al.(^28)</td>
<td>Load cell, electromagnetic spatial-tracking system</td>
<td>30</td>
<td>Anterior: 31.4 ± 16.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Posterior: 44.0 ± 28.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Inferior: 21.3 ± 12.3</td>
</tr>
</tbody>
</table>
The capsuloligamentous structures supporting the GH joint have been referred to as a "soft tissue socket." The capsule and ligaments function collectively with the labrum to maintain a centered humeral head and limit excessive translation. Translation can occur in any direction as the humeral head moves on the glenoid face during humeral elevation and rotation. Clinically, the most important translation directions to evaluate are anterior, posterior, and inferior because GH joint instability occurs in these directions. In this study of healthy shoulders, mean stiffness values were symmetric with respect to the direction of the applied force. Future studies should compare stiffness between translational directions in shoulders with a documented unidirectional or multidirectional instability.

**Clinical Implications**

An instrumented technique for measuring force-displacement response similar to the one presented in this study may be clinically useful as an instructional aid and as an assessment tool in a clinical setting.

Stiffness measures are clinically important in the prediction of capsuloligamentous injury and treatment outcomes. Subjectively, a soft feel is associated with capsuloligamentous disruption, and a hard or firm feel is associated with normal capsuloligamentous tissue. During a laxity examination, changes in the end-feel or stiffness are noted along with the patient’s history and other physical findings in making a clinical diagnosis. Investigators have also evaluated joint stiffness in healthy knees and anterior cruciate ligament-deficient knees before and after surgical reconstruction. From this research, they ultimately concluded that stiffness was of significant diagnostic value at the knee. Whether stiffness measures are accurate predictors of injury or clinical outcome at the GH joint still needs to be determined.

**Study Limitations**

We used a simple linear regression model to calculate stiffness only at the most linear region of the force-displacement curve. Because joint stiffness is commonly evaluated at the end-range of translation, we chose to characterize end-range stiffness only. A polynomial regression model would be most appropriate if the goal was to measure the joint’s stiffness response over the entire range of translation.

We did not account for the contribution of resting muscle tension on stiffness response of the joint. Subjects appeared to be relaxed and comfortable during test trials, and our examination of force-displacement curves did not reveal any abnormal spiking response, as could occur if the subject “tensed up” during force application. Similarly, we used a slow, controlled load rate in order to prevent any discomfort or abrupt muscle-guarding effects.

Joint stiffness has been reported to depend upon the amount of bulk tissue surrounding the GH joint. We did not account for interindividual differences in bulk tissue size for this study. Forces were applied consistently to each subject regardless of shoulder size. An a priori or post facto method for scaling the force per shoulder size should be considered as a means to account for varying anthropometric characteristics.

We only tested one position of humeral abduction and rotation, compared with McQuade et al., who tested several positions of humeral abduction and rotation. Lastly, this study was limited to modeling only static or passive joint stiffness, when, in fact, the role of dynamic factors is suggested to be most critical to overall joint stiffness during functional performance.

**CONCLUSIONS**

Our results suggest the following: (1) glenohumeral stiffness is widely distributed in healthy shoulders, (2) glenohumeral stiffness is not significantly different between men and women, and (3) glenohumeral stiffness is not significantly different among directions of translations.

**ACKNOWLEDGMENTS**

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**REFERENCES**


Removal Tools are Faster and Produce Less Force and Torque on the Helmet Than Cutting Tools During Face-Mask Retraction

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Objective: To investigate the retraction time, forces, and torques applied to the football helmet during removal of the face mask with different face-mask removal tools.

Design and Setting: Subjects retracted the face mask of a football helmet mounted to a force platform in a laboratory setting. They removed a standard face mask by cutting or removing (or both) the lateral plastic loop straps using 4 different tools: the Trainer’s Angel (TA), FM Extractor (FM), power screwdriver (SD), and Quick Release System (QR) in a counterbalanced fashion.

Subjects: Eighteen certified athletic trainers participated in this study.

Measurements: We started measuring time when the subject picked up the tool and ended when the face mask was in a fully retracted position. Maximum forces and torques were measured from the force platform during the retraction process.

Results: The SD and QR retracted the face mask significantly faster than the TA and FM. Forces producing superior-inferior translation were least with the SD. The SD and QR produced less lateral translation and rotation and lateral flexion moment than the TA and FM. The FM produced less torque in the lateral flexion moment than the TA.

Conclusions: Tools that removed the loop straps (SD, QR) were faster and produced less force and torque on the helmet than the tools that cut through the loop straps (TA, FM).

Key Words: cervical spine injury, loop straps, equipment removal

One of the most critical situations certified athletic trainers (ATCs) face today is managing a football player with a suspected cervical spine injury. Currently, football is responsible for the largest number of cervical spine injuries in athletics.1 Also, the skills that are required in the sport, such as tackling, can place the neck in a vulnerable position for injury if not performed correctly.2 For example, forces that place the neck in an axial loaded position have been found to cause catastrophic cervical spine injury.3-6

The current standard of practice in managing a football athlete with a possible spinal injury, as recommended by the Inter-Association Task Force for Appropriate Care of the Spine-Injured Athlete, is to first stabilize the head and neck, then gain access to the victim’s airway.7,8 In-line stabilization is extremely important to prevent further injury to the spinal cord. Currently, the degree of head movement needed to result in neurologic deficit with a spinal injury is unknown. However, it is estimated that as little as 1 to 2 mm of helmet displacement reduces the amount of space for the spinal cord in the spinal canal, possibly damaging the cord itself.9 Because 25% of spinal injuries with neurologic deficit were caused by improper handling of the victim during transport, the goal for the ATC should be to limit motion as much as possible during the immobilization process.7,10,11

After the athlete’s head and neck have been stabilized, the rescuer should gain access to the victim’s airway by removing the face mask from the helmet. The plastic loop straps that attach the face mask to the helmet can be cut with a sharp instrument or removed by unscrewing the screws that holds the straps in place. In either case, the task should be performed quickly and with little jarring of the head to decrease the risk for further injury.12-14

Tools such as a Trainer’s Angel ([TA], Trainer’s Angel, Riverside, CA), a power screwdriver (SD), and an FM Extractor ([FM], Sports Medicine Concepts, Geneseo, NY) have been examined for their effectiveness in face-mask removal. The TA was the first tool specifically designed to cut through the plastic loop straps that attach the face mask to the helmet.12 In previous research, the TA has been found to remove the plastic loop straps significantly faster than a manual screwdriver and an anvil pruner.12,14 Although the TA allowed fast access to the victim’s airway, it was associated with significantly more helmet movement than the other tools tested.12,14
The clips are attached to the helmet with a T-bolt, washer, and screw; therefore, manual and power screwdrivers have also been used for face-mask removal. However, manual screwdrivers take significantly longer to use,\textsuperscript{12,14} and at times, the bolt inside the helmet turns without loosening the screw, rendering the screwdriver ineffective.\textsuperscript{12,13}

In recent years, new tools have come onto the market for the purpose of face-mask removal, such as the FM and the Quick Release System ([QR], Jo Silken, ATC, San Mateo, CA). The FM is a tool that cuts through the fastener between the loop strap and the fixed screw end of the plastic clip. It also has features that enable the face mask to be used as a lever to aid in the cutting process. The QR is another innovative face-mask removal device. It uses a spring-loaded nut-and-bolt system to secure the loop strap onto the helmet. With this system, only a flat-head screwdriver is needed to turn the screw that attaches the face mask to the helmet. With just a quarter turn, the spring-loaded system releases and the entire loop strap can be removed, leaving the face mask unattached to the helmet and easily removed or retracted.

Most previous research on the effectiveness of face-mask removal tools measured the time taken for removal and retraction of the face mask. Few investigators have attempted to study the motion of the helmet during the removal process.\textsuperscript{12,15} The purpose of our study was to investigate the effectiveness of 4 face-mask removal tools (TA, SD [Craftsman, Sears, Hoffman Estates, IL], FM, and QR) on the time required for retraction and the forces and torques applied to the helmet during retraction of the face mask. We hypothesized that the QR and SD would retract the face mask in a shorter period of time and with less force and torque applied to the helmet.

METHODS

Subjects

Eighteen athletic trainers certified by the National Athletic Trainers’ Association Board of Certification (age = 24.6 ± 2.4 years old [range, 22–32 years], 2.1 ± 2.3 years certified [range, 0.42–9.2 years], 2.6 ± 2.3 years of football experience [range, 0–10 years]) volunteered to participate in this study. All subjects read and signed an informed consent approved by the university institutional review board (which also approved the study) before participating.

Instrumentation

The face-mask removal tools in this investigation included the TA, SD, FM, and QR. A flat-head screwdriver was used in conjunction with the QR. A Bertec force platform (Bertec Corp, Columbus, OH) and the DataPac 2000 (Run Technologies, Laguna Hills, CA) were used for collection of the force and torque data.

In the testing area, a standard football helmet was mounted onto a force platform. This was accomplished by bolting 3 brackets to the helmet (Figure 1). Two brackets were attached to each side of the helmet and ran laterally, and the third bracket was attached to the posterior aspect of the helmet's crown and ran posteriorly to the force platform. These brackets were then attached to the force platform using standard C clamps. The C clamps were applied with a firm force to prevent slippage. In addition, the clamps were standardized and tightened to the same level for each participant. A cuff weight of 3.4 kg (7.5 lbs), the approximate weight of an adult head,
was placed in the helmet once it was secured to the platform. The forceplate was then zeroed before each trial so that the only forces being measured were from movement associated with retraction of the face mask.

Procedures

The subjects first attended a short training session in which they were verbally and visually instructed in how to use each of the face-mask removal tools according to the manufacturer’s guidelines through a standard lecture and demonstration. Subjects were then given 10 minutes to familiarize themselves with each tool and complete face-mask retractions until they were satisfied that they understood how the tool worked and how to retract the face mask under each condition. After the practice time, subjects were individually escorted to the testing area.

When the subject entered the testing area, the testing procedure was explained. The subject was instructed to retract the face mask by cutting or removing (or both) the 2 lateral loop straps (Bike Athletic Co, Knoxville, TN) and retracting the face mask using the anterior fasteners as a hinge. The subject was told to retract the face mask quickly and with as little movement as possible.

One of the 4 face-mask removal tools was then placed on the floor next to the force platform. Time began when the subject first touched the tool and ended when the face mask was in a fully retracted position. The timing device was activated manually by one of the investigators (H.L.J.). After the subject retracted the face mask, one of the researchers (H.L.J.) fit the helmet with new fasteners, and the procedure was repeated until the subject performed the task 8 times (twice with each tool). The order of tools was counterbalanced.

Data Analysis

Time. Time was recorded on the DataPac 2000 from the moment the subject picked up the tool until the face mask was in a fully retracted position.

Forces and Torques. All force and torque data were analyzed and interpreted with the assumption that the motion of the helmet would translate to the motion of the head. However, it should be emphasized that in field situations, not all torques may be transferred to the head due to some shift in the fit of the helmet on the athlete’s head. Raw data from the force platform were transferred to the Data Pac program and analyzed using the calibration matrix of our force platform. The calibration matrix was used to calculate the appropriate gain value to convert raw voltage signals from the forceplate to the appropriate Newtons of force and Newton-meters of torque. The absolute maximum values for 3 forces (superior-inferior, lateral, and anterior-posterior) and for torques producing a rotational, flexion-extension, or lateral-flexion moment were recorded. The absolute maximum value was chosen as the largest amount of force or moment applied during that trial. Torque values were adjusted from the raw torque values to account for the torque’s being applied to the center of the helmet, not the center of the forceplate. The center of rotation for the helmet was measured at 13.75 cm above the center of the forceplate, in line with the ear holes of the football helmet, where the axis of rotation of the cervical spine would lie. The maximum values for the 2 trials of each tool were averaged to determine the force and torque applied with each tool.

Statistical Analysis. All statistical analyses were performed with Statistical Package for the Social Sciences software (version 10.0, SPSS Inc, Chicago, IL). We used a multivariate analysis of variance to analyze differences among tools for time, the 3 forces, and the 3 torques. Univariate, repeated-measures analysis of variance was then calculated for time, force (superior-inferior, lateral, and anterior-posterior), and torque (rotational, flexion-extension, and lateral-flexion moment) to determine significant differences among the 4 tools. Significant differences were investigated further using the Tukey Honestly Significant Difference test. Alpha was set a priori at .05.

RESULTS

Means and standard deviations for time, forces, and moments are presented in the Table. Results of the multivariate analysis of variance demonstrated significant differences among the dependent measures \(F_{21,129.8} = 7.06, P = .0001, \beta = 1.0\). Violations of assumed sphericity were found for all measures, except for forces producing lateral translation. This violation of assumed sphericity indicates that our samples demonstrated unequal variances and unequal correlations, which can make the results of the \(F\) test seem somewhat liberal.\(^16\) It is for this reason that we report the \(F\) values associated with the Huynh-Feldt test, an adjustment to more accurately reflect the \(P\) value, with the exception of the lateral force.

Time

The univariate analysis for time \(F_{3,51} = 36.12, P = .0001, \beta = 1.0\) and a subsequent Tukey analysis revealed signifi-
Figure 2. Mean time, in seconds, for removal of the face mask with the 4 tools. TA indicates Trainer’s Angel; FM, FM Extractor; SD, screwdriver; and QR, Quick Release System. Error bars represent standard deviations. *SD and QR took significantly less time than TA and FM. †FM took significantly less time than TA (P < .05).

Figure 3. Mean force, in Newtons, applied to the helmet during removal with the 4 tools. TA indicates Trainer’s Angel; FM, FM Extractor; SD, screwdriver; and QR, Quick Release System. Error bars represent standard deviations. *SD produced significantly less force than TA and FM. †SD and QR produced significantly less force than TA and FM (P < .05).

Figure 4. Mean torque, in Newton-meters, applied to the helmet during removal with the 4 tools. Error bars represent standard deviations. TA indicates Trainer’s Angel; FM, FM Extractor; SD, screwdriver; and QR, Quick Release System. *SD and QR produced less torque than TA and FM. †SD and QR produced significantly less torque than TA and FM. ‡FM produced significantly less torque than TA (P < .05).

Forces

Significant main effects were found for superior-inferior force ($F_{3,51} = 3.576, P = .02, \beta = .76$) and lateral force ($F_{3,51} = 32.04, P = .0001, \beta = 1.0$) (Figure 3). On post hoc analysis, we found that the SD produced less superior-inferior force than the TA and FM and that the SD and QR produced less lateral force than both the TA and FM. No differences were found among the various tools in producing anterior-posterior force ($F_{3,51} = 2.79, P = .069, \beta = .64$).

Moments

Figure 4 illustrates the significant main effects for the rotation moment ($F_{3,51} = 10.79, P = .0001, \beta = .99$) and lateral-flexion moment ($F_{3,51} = 34.05, P = .0001, \beta = 1.0$). Post hoc analysis for the rotational moments showed that the SD produced significantly less torque than the TA and FM, and the QR produced significantly less torque than the TA and FM. The post hoc analysis for the lateral-flexion moment revealed that less torque was produced by the SD compared with the TA and FM, the QR compared with the TA and FM, and the FM compared with the TA. No differences were found among tools for the torque produced in the flexion-extension moment ($F_{3,51} = 2.23, P = .117, \beta = .45$).

Discussion

In general, face-mask retraction techniques have not been the subject of a great deal of research; however, this is the area in which ATCs need to be the most proficient and have the best tools available in order to effectively care for a cervical spine–injured athlete. Therefore, the purpose of our study was to evaluate several means of face-mask retraction, both newly developed and previously tested, to determine which retracted the face mask the fastest and with minimal force applied to the head. The SD and QR generally allowed for faster retraction of the face mask with less force and torque applied to the helmet than the cutting tools, TA and FM.

Previous Research

It was difficult for us to compare our results with those of previous researchers because 2 of our 4 tools (SD and QR) had not yet been examined for time of removal, and 3 of our tools (QR, SD, FM) had not yet been examined in terms of force or torque. However, our results tend to agree with previous findings (ie, that the SD is faster and produces less head motion than tools that cut through the loop strap).12,15

Removing the plastic loop strap completely via the SD or QR was quicker and produced significantly less force and torque than the cutting tools. Our findings support previous results in which the manual screwdriver produced less head movement but took the same amount of time for removal as the TA.12 Similarly, Ray et al14 found that neither the manual nor power screwdriver provided a clear advantage in speed or movement. Motion analysis of head movement while using the TA, SD, and anvil pruner demonstrated that the SD took significantly less time (36.6 seconds) to remove the mask than the anvil pruner (77.95 seconds) and the TA (84.4 seconds).15
In addition, head motion produced by the SD (2.35°) was significantly less than that produced by the anvil pruner (3.01°) and the TA (3.31°). Therefore, our results support previous findings that the SD can be effective in removing the face mask more quickly and with less application of force compared with cutting tools. However, one must be aware of the potential limitations of the effectiveness of the SD. Our observations were consistent with previous findings that the T-bolt spun instead of the screw. If this occurs, the SD is ineffective, and the face mask must be removed by another tool. If no other tool is available, the helmet must be removed, exposing the athlete to an increased risk of further injury due to head and cervical spine motion. We found an 8% incidence of screw spinning, similar to the 7% incidence reported by Knox and Kleiner. Although Ray et al. did not mention the specific incidence of spinning in their investigation, they did report spinning, which should be a concern for those using the SD as the primary removal tool. Overall, we agree with the past literature and the Inter-Association Task Force for Appropriate Care of the Spine-Injured Athlete that the SD should not be used as the primary means of face-mask retraction or removal.

Recent investigators have examined the FM to determine the amount of time required for face-mask removal. Three different methods suggested by the manufacturer of the FM, to retract the face mask by resting the semicircular notch on top of the face-mask bar, locking the semicircular notch onto the face-mask bar, and placing each end of the tool on the loop strap, demonstrated time intervals of 98.94, 109.55, and 135.23 seconds, respectively. Another recent investigation compared the FM with the TA and anvil pruner and found the anvil pruner to be a faster removal tool (32.04 seconds) than both the TA (75.91 seconds) and FM (63.10 seconds). The latter 2 tools did not differ significantly from one another.

The QR seems to be a promising new method of attaching the face mask to the helmet and was found effective in the face-mask retraction process. This device enabled subjects to remove the face mask significantly faster than the TA and FM and with less lateral translation and rotational and lateral-flexion moment.

**Forces and Moments**

One unique aspect of our study was the measurement of the forces applied to the helmet and the torques created about the axes of motion during the removal process. These measurements are important to our understanding of the face-mask removal process and in the clinical practice of removing the loop straps and retracting the face mask. We noted several trends in our force and torque data. For all 4 tools, greater forces were applied in the anterior-posterior direction (directly down into the forceplate) than forces applied in either the superior-inferior or medial-lateral directions. Additionally, the moments about the flexion-extension axis were greater for all 4 tools than the moments about the rotational or lateral-flexion axes. These findings agree when we consider the location of the loop straps on the helmet. The loop straps are located anterior to the medial-lateral axis of the head, and as one pushes harder into the forceplate (anterior-posterior translation), a larger torque about the flexion-extension moment should be created. These results indicate that the ATC providing cervical stabilization of a suspected spine-injured athlete should be aware of greater torque and possible motion about the flexion-extension axis.

In general, the use of the SD was associated with the least amount of force and moment about all 3 axes, demonstrating less potential movement of the helmet. However, one must consider the SD’s limitations as previously described and in the recommendations of the Inter-Association Task Force.

**Limitations**

Our study differed in several ways from past research, which could have limited the validity of our results. By using the data from the force platform, we were able to analyze the maximal forces and torques applied to the helmet. However, it is unclear how these correlate with the actual head and spinal movements of an athlete. How much the clinician can move the head and not cause further injury to the athlete is currently unknown. Therefore, it is recommended that any movement of the head and cervical spine be minimal. No conclusions can be drawn as to whether these values represent a safe removal process.

A second potential limitation was that there was no live model occupying the testing helmet. This created a system lacking the counterstability of bony and soft tissue neck anatomy. Without these natural restraints, the subjects might have moved the helmet more than if an actual person was wearing the helmet. This design could have also affected the subjects’ mindset during the testing procedure. Without a human model, the subjects may have been more inclined to use greater force with the tool than if they were in an actual injury situation. This is especially the case with the cutting tools, TA and FM, and may explain why these tools had such elevated force and torque values compared with the SD and QR.

The final limitation to our study deals with the design of the modified testing helmet that was used in the procedure. In order to attach the helmet to the force platform, 3 brackets were bolted to the helmet (2 laterally and 1 posteriorly). We used this design to standardize the stabilization of the helmet, both within and between subjects, by using clamps tightened the same amount throughout the study. However, because of the alignment of one of the lateral brackets, 2 subjects did not have direct access to one of the face-mask loop straps. This limitation affected their use of the TA during the testing procedure in that these subjects had to make several attempts to cut the strap with the TA. This limitation could account for the longer time to remove the loop straps with the TA.

**Clinical Relevance**

A great deal more research is needed in the area of football face-mask removal to determine which tool is the most helpful to the athletic trainer in this critical situation. Overall, we feel that this study adds new insight into the forces and torques applied to the helmet during retraction of the face mask with various means. We also feel that it potentially establishes a new method of attaching the face mask to the helmet (ie, Quick Release System). However, we should emphasize that the Quick Release System is a prototype device and, as currently designed, may not represent a final marketable product. Despite this, we believe that the design has merits because it was easy to install and easy to operate, requiring only a screwdriver. Our results suggest that, indeed, this is the case, and that new alternative designs to face-mask retraction and re-
moval are worth considering. This device performed as well as, or better than, the other standard face-mask removal devices with respect to time, force, and torque. Even though the Quick Release System and power screwdriver produced similar results in the measures we collected, the Quick Release System did not have any factors that limited its effectiveness, such as the spinning that occurred with the screwdriver.

Also of concern for the ATC are the directions of the greatest force and moment found in our study. Regardless of the tool used to retract the face mask, the largest force was in the anterior-posterior direction and the largest torque was along the flexion-extension moment. The ATC responsible for stabilizing the injured athlete needs to be aware of the potential for these large forces and moments and stabilize accordingly.

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We thank Jo Silken, ATC, for donating the Quick Release System; the Riddell Corporation (Elyria, OH) for the donation of the loop straps; and the Building and Grounds Department at Virginia Military Institute (Lexington, VA) for help with construction of the helmet-mounting brackets. We also thank Sara E. Wilson, PhD, for her help with the analysis and conversion of the torque data.

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OPTOTRAK Measurement of the Quadriceps Angle Using Standardized Foot Positions

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Objective: While there is evidence to suggest that the magnitude of the quadriceps (Q) angle changes with alterations in foot position, a detailed quantitative description of this relationship has not been reported. Our purpose was to determine the effect of varying foot placement on the magnitude of the Q angle.

Design and Setting: A mixed between-within, repeated-measures design was used to compare Q angles derived under static weight-bearing conditions with the feet positioned in self-selected versus standardized stance positions.

Subjects: Twenty healthy young-adult men and women with no history of acute injury to or chronic dysfunction of the lower limbs.

Measurements: We placed light-emitting diodes bilaterally on the left and right anterior superior iliac spines, the tibial tuberosities, and the midpoints of the patellae to bilaterally define the Q angles. An OPTOTRAK motion-measurement system was used to capture x,y coordinate data at a sampling rate of 60 Hz. These data were subsequently filtered and used to calculate the magnitude of the left and right Q angles.

Results: A repeated-measures analysis of variance revealed that when measured statically, Q angles differed significantly between stance positions (P < .001) and limbs (P < .05). Depending on the stance adopted, mean Q angles varied from 7.2° to 12.7° and 11.0° to 16.1° in the left and right lower limbs, respectively. Q-angle measurements taken in conjunction with the Romberg foot position most closely resembled those gathered with the feet in a self-selected stance (Pearson r = 0.86 to 0.92).

Conclusions: Q-angle magnitude varies with changes in foot position, increasing or decreasing as the foot rotates internally or externally, respectively. These data demonstrate the need for a standardized foot position for Q-angle measurements.

Key Words: angle, stance, reliability, ecologic validity
respectively. The actual data, in the form of the raw data set or descriptive statistical measures, were not reported.

Our primary purpose was to compare the Q-angle magnitude when measured with the feet positioned in self-selected versus standardized stances. The standardized foot positions included placing the medial borders of the feet together, as in the Romberg test of balance, and the average preferred stance position reported by Mcllroy and Maki. While the former position is easily replicated by subjects and may be viewed as reliable, the latter arguably represents an average preferred foot position for an adult population. Secondary purposes included comparing Q-angle measurements of the right and left lower limbs and assessing the degree of association between Q-angle measurements taken with the feet in self-selected versus constrained-stance positions.

METHODS

All methods were approved before data collection by the University of Western Ontario’s Review Board for Health Sciences Research Involving Human Subjects. Each subject provided informed written consent. Exclusion criteria included a history of lower-limb acute injury or chronic dysfunction. The sample of healthy active young adults included 6 men (height = 1.80 ± 0.09 m, mass = 80.6 ± 11.1 kg) and 14 women (height = 1.68 ± 0.06 m, mass = 62.4 ± 8.3 kg), ranging in age from 19 to 30 years (age = 22.1 ± 3.5 years).

We defined right and left Q angles by placing infrared light-emitting diodes (LEDs) bilaterally on the anterior superior iliac spines, midpoints of the patellae, and centers of the tibial tubercles while each subject stood with the feet in the Romberg position. The anatomical landmarks were located through palpation, visual estimation, and measurement by a single examiner. Three-dimensional coordinate data were collected at a sampling rate of 60 Hz using a single bank of OPTOTRAK motion-measurement sensors (Northern Digital Inc, Waterloo, ON, Canada). The system’s calibration was verified before data collection using a rigid 3-dimensional (x,y,z) orthogonal jig. A mean accuracy of 0.5 mm was determined. This high degree of accuracy is consistent with that of other active optical-tracking systems. Participants stood on an elevated platform approximately 3 m from the position sensors, which afforded a viewing area of approximately 1.5 m², with knees extended and the quadriceps muscle group relaxed, and their feet in each of the following stances: self-selected, Romberg (ie, medial borders of the feet touching), and average preferred stance (ie, 0.17 m between heel centers, with a 14° angle between the long axes of the feet). Using predefined lines and landmarks on the testing platform surface, we carefully placed the subject’s feet in the average preferred-stance position. Five data-collection trials were completed for each foot position to control for the possibility of variation in the Q-angle measure due to body sway. The data were filtered at 10 Hz using a low-pass, fourth-order, recursive Butterworth filter. Using trigonometric algorithms for the filtered x,y coordinate data, we calculated the angular orientation of the quadriceps and patellar tendon rays and the magnitude of the resultant Q angle in the left and right lower limbs (Figure).

A preliminary investigation determined intratester reliability of the Q-angle measure. On 2 separate occasions separated by 1 week, LEDs were placed bilaterally by one investigator on the same 10 individuals while they stood with their feet aligned in the Romberg position. An intraclass correlation coefficient (2,1) procedure yielded an intratester reliability of 0.92, and the standard error of measurement was 1.4°. Differences in static Q angles by foot position (self-selected, Romberg, average preferred stance), limb (right, left), and trial were analyzed with a mixed between-within, repeated-measures 3-factor analysis of variance procedure using post hoc Scheffe F tests to distinguish the source, if any, of identified effects. By specifying the measurement trial as a within-subjects factor, data from all 5 trials per foot position were entered into the statistical analysis. We also calculated Pearson product moment correlation coefficients (r) to assess the degree of association between Q-angle measures derived in the 3 different positions.

RESULTS

Descriptive statistics for the Q-angle measurement are summarized in Table 1. When measured statically, significant differences in Q angles between foot positions (F2,38 = 34.09, P < .001) were observed, with values different among all 3 stance conditions. Q angles in the right and left legs were greatest when measured in the Romberg position and least when the feet were placed in the average preferred-stance position. A significant difference in Q-angle magnitude between limbs (F1,19 = 4.14, P < .05) was also observed. Mean values

<table>
<thead>
<tr>
<th>Stance Position</th>
<th>Sample (n)</th>
<th>Left Q Angle (°)</th>
<th>Right Q Angle (°)</th>
<th>Right and Left Q Angles Combined (°)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self-selected</td>
<td>20</td>
<td>11.4 (7.2)</td>
<td>14.4 (3.0)</td>
<td>12.9 (6.9)†</td>
</tr>
<tr>
<td>Romberg</td>
<td>20</td>
<td>12.7 (7.7)</td>
<td>16.1 (6.2)</td>
<td>14.4 (7.1)†</td>
</tr>
<tr>
<td>Average preferred</td>
<td>20</td>
<td>7.2 (7.8)</td>
<td>11.0 (6.4)</td>
<td>9.1 (7.3)†</td>
</tr>
</tbody>
</table>

*SD indicates standard deviation; and n, number of subjects. †Significant differences among stances (P < .001). ‡Significant differences among stances (P < .05).
Table 2. Correlation Coefficients of Measured Q Angles by Limb*

<table>
<thead>
<tr>
<th></th>
<th>Self-Selected Stance</th>
<th>Romberg Stance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right Limb</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-selected stance</td>
<td>0.86</td>
<td>—</td>
</tr>
<tr>
<td>Romberg stance</td>
<td>0.82</td>
<td>0.88</td>
</tr>
<tr>
<td>Average preferred stance</td>
<td>0.92</td>
<td>—</td>
</tr>
<tr>
<td>Left Limb</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-selected stance</td>
<td>0.92</td>
<td>—</td>
</tr>
<tr>
<td>Romberg stance</td>
<td>0.93</td>
<td>0.91</td>
</tr>
<tr>
<td>Average preferred stance</td>
<td>0.93</td>
<td>0.91</td>
</tr>
</tbody>
</table>

*All values were statistically significant (P < .001).

derived from the right limb were 3.0° to 3.8° larger than those in the opposite limb, with the exact magnitude of the difference varying with the foot position adopted. We found no significant differences by measurement trial (F4,76 = 0.67, P < .62), for the interaction effects of foot position by limb (F2,38 = 0.68, P < .51), or for foot position by limb by trial (F8,152=1.46, P < .18). Correlational analysis revealed that Q-angle measurements taken in conjunction with the Romberg foot position most closely resembled those gathered under self-selected stance conditions (Table 2).

**DISCUSSION**

In their systematic investigation of changes in Q angle with changes in foot position, Olerud and Berg36 reported that the Q angle increased or decreased by 5° with 15° internal or external rotation of the foot, respectively. Others13,33,43 have studied the Q-angle magnitude while controlling foot position, yet they have done so with the aim of studying the relationship between knee conditions and lower limb structural variables only. Cowan et al.13, for example, in a study of overuse injury among 294 male infantry trainees, had participants stand with the heels spaced 7.5 cm apart and the medial borders of the feet 60° divergent. In this position, observed Q angles ranged from 0° to 26° and averaged 10° (SD = 5°). Reider et al.43 in contrast, observed a mean Q angle of 15.9° in healthy young controls who stood with the medial borders of the feet placed together, side by side, as is prescribed in the Romberg test of balance. In another study,33 Q angles averaged 11.1° (SD = 4.9°) and ranged from 1.0° to 25.0° for 60 men and women who stood with the long axes of their feet positioned perpendicular to the coronal plane but with their feet set an unknown distance apart. The lack of consistent subject positioning in these studies is striking. It is also interesting to note that none of these studies provided a rationale or justification for the foot positions used.

If a measurement such as the Q angle is to be a criterion for determining an individual’s risk for injury or candidacy for surgery, then it must be accurate, valid, and reliable.44 We used x,y coordinate data captured with an OPTOTRACK active optical-tracking system to calculate the magnitude of the frontal-plane Q angle. This method is of value in that it is comparable with the photographic methods described in previous investigations13,36 yet methodologically preferable because it has been shown to effectively reduce measurement variability.41,45 Our methods of positioning the subject in an upright, weight-bearing posture with controlled-stance positions were also purposely chosen to enhance the ecologic validity and reliability of the resulting measurements, respectively. The average preferred- or natural-stance position was used because it may, as McIlroy and Maki39 suggested, meet the need to standardize while minimizing the extent of constraint on an individual’s self-selected foot position. The decision to use the Romberg position was more arbitrary, yet it is easily replicated and has been used by others43 when measuring the Q angle.

Our static observations generally agreed with those of Olerud and Berg36: the magnitude of the angle increased or decreased as the foot rotated internally or externally, respectively. However, we observed somewhat smaller static Q-angle changes with alterations in foot position. These differences are most likely accounted for by our use of less extreme foot positions, leading to smaller amounts of internal or external rotation of the lower limb. The observed differences in Q-angle patterns between stances may be primarily attributed to the transverse-plane positioning (ie, internal or external rotation) of the femur and tibia imposed by foot position. The Romberg position, for example, requires greater lower-limb internal rotation, while the average preferred stance requires greater lower-limb external rotation. For most, the self-selected foot position was represented by a stance position that fell between these 2 extremes. The self-selected stance was clearly identified as the most comfortable experimental position for the completion of the task; participants frequently mentioned their discomfort with the average preferred-stance position and, to a much lesser extent, the Romberg stance.

Given the cost, time, and expertise required, it is unlikely that the motion-measurement device we used will be available in most clinical settings. Nonetheless, these findings have important clinical implications for practitioners and patients alike. First and foremost, the Q-angle magnitude changes with alterations in foot position. The practitioner must recognize the influence of foot positioning on the Q angle and, therefore, ensure that individuals are always similarly positioned when measures are gathered. The methods used should be accurately and completely described to increase the generalizability of studies reported within the literature. Second, practitioners should consider the ecologic validity of the measurement positions used. We purposely chose a standing, weight-bearing position because individuals are more likely to experience patellar dysfunction when the knee joint is loaded. It is important to recognize, moreover, that while self-selected foot positions may provide the greatest degree of ecologic validity, an individual’s inability to replicate the position over time may limit the reliability of the resulting Q-angle measurement. Understanding the degree of association between Q angles measured under self-selected versus controlled foot positions may make the latter a meaningful alternative to the former. Our descriptive data clearly demonstrate that measurements taken with feet side by side yielded mean values that were larger than, but most closely resembled, those found under self-selected conditions. It is for this reason that we recommend the use of the Romberg position to standardize stance in future studies measuring the Q angle. Measures taken in the average preferred-stance position clearly underestimate those derived with feet in a self-selected stance. Correlational analyses, moreover, suggested that left Q-angle measures in the 2 calibrated stances yielded reasonable estimates (r2 = 85% to 87%) of the left Q angle in a self-selected stance position, while right Q-angle measures were somewhat less predictive (r2 = 67% to 74%). Why Q-angle measures gathered under controlled foot positions were less predictive in the right than the left lower limb is unknown. Similarly, why the mean Q-angle measures we
report in this study differed significantly between the lower limbs is unclear. This is not the first investigation in which bilateral Q-angle asymmetry has been observed, although similar reports have only been published since 1997. The Q-angle measurements reported in this study were derived from a healthy, young adult population. Whether similar results would be observed in those symptomatic for anterior knee pain or patellar subluxation or dislocation is unknown. Their generalizability, moreover, is limited to similar investigations in which the primary purpose is to understand how methodologic variation may affect measurement outcome. The inclusion of men and women in our sample of interest, while acceptable for this methodologic study, yielded mean data that are not appropriate for clinical interpretation or comparison. Our methods have been thoroughly reported so that future investigators can replicate our efforts. If the differences in reported Q angles over time within an individual or from study to study are to be interpreted as true differences or as a product of the measurement method used, a standardized method of measurement must be established and methods accurately reported. Only with such efforts can the enigmatic nature of the Q angle be better understood.

REFERENCES

Intrarater Reliability of Functional Performance Tests for Subjects With Patellofemoral Pain Syndrome

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Objective: Patellofemoral pain syndrome (PFPS) is a common clinical entity seen by the sports medicine specialist. The ultimate goal of rehabilitation is to return the patient to the highest functional level in the most efficient manner. Therefore, it is necessary to assess the progress of patients with PFPS using reliable functional performance tests. Our purpose was to evaluate the intrarater reliability of 5 functional performance tests in patients with PFPS.

Design and Setting: We used a test-retest reliability design in a clinic setting.

Subjects: Two groups of subjects were studied: those with PFPS (n = 29) and those with no known knee condition (n = 11). The PFPS group included 19 women and 10 men with a mean age of 27.6 ± 5.3 years, height of 169.80 ± 10.5 cm, and weight of 69.59 ± 15.8 kg. The normal group included 7 women and 4 men with a mean age of 30.3 ± 5.2 years, height of 169.55 ± 9.9 cm, and weight 69.42 ± 14.6 kg.

Measurements: The reliability of 5 functional performance tests (anteromedial lunge, step-down, single-leg press, bilateral squat, balance and reach) was assessed in 15 subjects with PFPS. Secondly, the relationship of the 5 functional tests to pain was assessed in 29 PFPS subjects using Pearson product moment correlations. The limb symmetry index (LSI) was calculated in the 29 PFPS subjects and compared with the group of 11 normal subjects.

Results: The 5 functional tests proved to have fair to high intrarater reliability. Intrarater reliability coefficients (ICC 3,1) ranged from .79 to .94. For the PFPS subjects, a statistical difference existed between limbs for the anteromedial lunge, step-down, single-leg press, and balance and reach. All functional tests correlated significantly with pain except for the bilateral squat; values ranged from .39 to .73. The average LSI for the PFPS group was 85%, while the average LSI for the normal subjects was 97%.

Conclusions: The 5 functional tests proved to have good intrarater reliability and were related to changes in pain. Future research is needed to examine interrater reliability, validity, and sensitivity of these clinical tests.

Key Words: step-down, squat, limb symmetry, knee

Patellofemoral pain syndrome (PFPS) is a common clinical entity used to describe a variety of pathologic conditions associated with the articulation between the undersurface of the patella and the femoral condyles. Patellofemoral pain syndrome can be caused by a variety of factors, including quadriceps weakness, increased Q angle, faulty lower extremity mechanics, overuse, and lateral retinaculum tightness. The major complaints of patients with PFPS are diffuse knee pain, patellar crepitus and locking, knee joint stiffness, and decreased activity levels. Onset of symptoms is usually insidious and may occur bilaterally. Activities such as prolonged sitting, stair descent, and squatting often exacerbate the pain.

The ultimate goal of rehabilitation for patients with PFPS is return to the highest functional level in the most efficient manner. Accompanying this goal is the need for a testing method that is objective, reliable, and sensitive to the changing status of PFPS. Common objective measures of knee function include pain assessment, goniometry, girth measurement, manual muscle testing, and isokinetic evaluation. However, these tests have been shown to be poor predictors of function.

Functional testing is an attempt to evaluate the knee joint under conditions that mimic realistic functional demands. Performance on functional tests depends on many variables, including pain, swelling, crepitus, neuromuscular coordination, muscular strength, and joint stability. The tests should be time efficient and simple to perform with minimal instruction; they should require minimal staff training and be conducted within a clinical setting. Several functional knee tests are described in the literature and include the shuttle run, stair-running test, vertical jump test, and hop tests. These tests are useful after ligamentous knee injuries or other sport-related injuries, such as muscle strains or meniscus injuries, and are not specific to the patellofemoral joint.

Functional performance tests that are specific for PFPS should be chosen based on clinical evidence and the ease of
replication among clinics and facilities. Pain is a factor associated with PFPS and is commonly used as a measurement to determine functional improvement. Chesworth et al.13 evaluated the visual analog scale (a measure of pain) in patients with PFPS, finding poor day-to-day reliability but good sensitivity to clinical changes. In addition to measuring pain directly, performance of a functional test may add information regarding muscle strength, endurance, proprioception, and balance.

Functional tests specific to PFPS should include weight-bearing stress with various knee-flexion angles because these are common aggravating positions and require dynamic muscular control. Post and Fulkerson10 found that 86% of patients with patellofemoral pain have pain during stair climbing and 85% have pain with squatting. The increase in pain with these activities is correlated with an increase in patellofemoral joint reaction force.3,4,8,14 At present, no functional tests specific to the patellofemoral joint have been reported in the literature.

We evaluated the following functional performance tests in this study: anteromedial lunge, step-down, single-leg press, bilateral squat, and balance and reach. The anteromedial lunge is a multiplanar movement designed to challenge the lateral patellofemoral articulation with the valgus stress placed on the knee during the maneuver.15 Theoretically, as an individual lunges and the center of gravity moves forward and across the body, the pull of the quadriceps muscle causes compressive loading of the lateral patellofemoral articulation, a common site of patellofemoral symptoms. The step-down mimics the function of stair descent, a common aggravating factor. The load of the patellofemoral joint with stair descent has been reported to be 3.5 times body weight.16 A single-leg press test was chosen to stress the patellofemoral joint in a partial weight-bearing mode. This test can be administered early in rehabilitation when a full squat may be too aggravating. To further challenge the patellofemoral joint, a second test is a full weight-bearing bilateral squat. The joint reaction force of a squat to 90° is approximately 7.5 times body weight.3 The balance-and-reach test, described by Gray,15 specifically challenges single-leg balance.

The primary purpose of our study was to determine the intrarater reliability of 5 functional performance tests on patients with PFPS. Secondary purposes were to determine limb symmetry index (LSI) differences for involved and uninvolved limbs and to assess the relationship between the 5 functional tests and pain ratings.

METHODS

Subjects

Two groups of subjects were included in this study, one group with PFPS (n = 29) and a second group with no known knee condition (n = 11). The PFPS group included 19 women and 10 men with a mean age of 27.6 ± 5.3 years, height of 169.80 ± 10.5 cm, and weight of 69.59 ± 15.8 kg. Data from 15 of these 29 subjects were used for the reliability testing. Inclusion criteria for the subjects in the PFPS group were 2 of the following on initial assessment: pain on direct compression of the patella against the femoral condyles with the knee in full extension, tenderness on palpation of the lateral surface of the patella, pain on resisted knee extension, or pain with isometric quadriceps contraction against suprapatellar resistance with the knee in slight flexion. These subjects’ symp-
and return to full knee extension. The number of unilateral squats completed in 30 seconds is recorded. Both limbs are tested (Figure 3).

**Bilateral squat.** Subjects start this test standing with the knees in full extension, shoulder-width apart, and weight evenly distributed on both limbs. Subjects lower their bodies to a knee position of 90° and then return to full extension. One repetition consists of a complete cycle of straight standing to 90° of knee flexion and return to straight standing. The number of bilateral squats completed in 30 seconds is recorded.

**Balance and reach.** The subject starts the test behind a start line. The subject reaches straight forward with one leg so that the heel touches the floor, with most of the body weight remaining on the back (test) leg. The uninvolved limb is tested first. Distance is recorded from the start line to the heel of the lead limb. The maximal distance of 3 trials is recorded and marked. Eighty percent of the maximal distance is calculated and marked with a piece of tape. During the 30-second test period, the subject performs as many balance-and-reach lunges as possible. Only lunges in which the subject’s heel touches beyond the 80% mark are recorded. The involved limb is then tested using the 80% mark from the uninvolved limb (Figure 4).

**Visual Analog Scale (VAS).** A 10-cm horizontal line was used to assess patellofemoral knee pain over the 24 hours before the testing period. The far left is “pain free,” and the extreme right represents severe pain. The marked value was measured with a standard ruler and then converted to a pain score. The VAS has been previously validated in the literature and used in patients with PFPS.

**Procedure**

The University of Kansas Medical Center’s Internal Review Board approved the study. Before participating, subjects were screened with questions regarding previous lower extremity injuries. After being selected to participate, each subject completed an informed consent form followed by the VAS. After completing the VAS, the subject was instructed in the proper technique for each of the functional performance tests. The subject performed each of the functional tests in random order, and both lower extremities were tested. The beginning test leg was randomly assigned for the single-leg press and the step-down test. Each test was performed once, without the use of tape or a brace. Pain level during the test was monitored, and subjects were instructed that they could stop due to pain, but pain level was not a stopping criterion.

Each subject began the assessment session with a warm-up period that consisted of low-resistance, lower extremity cycling. The task-specific warm-up included practice for each functional performance test (3 to 5 repetitions with a 30-second rest before the actual testing). A written description of each test was read to the subject, followed by a demonstration of the test by the tester (Appendix). Participants received no verbal encouragement during actual testing. Subjects were allowed a 1-minute rest between functional performance tests.

**Reliability.** For intrarater reliability, 15 subjects with PFPS were tested on 2 occasions, 48 to 72 hours apart. The random order of functional testing was matched between day 1 and day 2. Visual analog scale scores had to match within 0.5 from test day 1 to test day 2 (eg, a subject who scored 6.5 on day 1 had to score between 6.0 and 7.0 on day 2). Pain has motivational, affective, cognitive, behavioral, and sensory dimensions, and these factors can hinder a test-retest design. Therefore, for reliability testing, subjects had to score within 0.5 on the VAS from test day 1 to test day 2 to prevent confounding of the pain variable.

**Correlation.** The scores on day 1 of the 15 reliability subjects were added to the scores of 14 other individuals with unilateral PFPS who had completed the VAS and 5 functional tests. This information was used to establish a relationship between the VAS and functional test scores. All subjects scored within this range except for 2. Both subjects were asked to return within 48 hours and repeat the VAS. Upon return, 1 of the 2 subjects scored within the acceptable VAS range; the other did not and was dropped from the study.

**Limb Symmetry Index.** The PFPS limb scores were compared and an LSI was established. The group of 11 subjects with normal knees also performed the 5 functional tests to determine the LSI.

**Data Analyses**

We compiled descriptive characteristics for each subject and all performance scores in a Microsoft Excel (Microsoft Inc, version 2000, Redmond, WA) spreadsheet. Data from the VAS were recorded as a single score to one decimal place. Data from each of the functional performance tests were recorded as number of repetitions. We recorded repetitions for right and
Table 1. Intrarater Reliability Estimates for Functional Tests

<table>
<thead>
<tr>
<th>Test</th>
<th>Intraclass Correlation Coefficient (3,1)</th>
<th>Standard Error of the Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anteromedial lunge</td>
<td>.82</td>
<td>.38</td>
</tr>
<tr>
<td>Step down</td>
<td>.94</td>
<td>.53</td>
</tr>
<tr>
<td>Single-leg press</td>
<td>.82</td>
<td>.56</td>
</tr>
<tr>
<td>Bilateral squat</td>
<td>.79</td>
<td>.47</td>
</tr>
<tr>
<td>Balance and reach</td>
<td>.83</td>
<td>.68</td>
</tr>
</tbody>
</table>

Results

Reliability

Intraclass correlation coefficients and SEMs for the intrater mean scores are summarized in Table 1. Intraclass correlation coefficients ranged from .79 to .94, and SEMs ranged from .38 to .68. The highest ICC was found with the step-down test and the lowest ICC with the bilateral squat test.

Correlation

The correlation matrix from the Pearson correlation coefficient analyses is found in Table 2. Correlation values between the VAS and the functional tests ranged from .386 to .730. Only the bilateral-squat test did not correlate significantly with the VAS.

Limb Symmetry Index

Limb difference in the PFPS group was statistically significant (P < .013) for all unilateral functional tests. Limb difference in the normal subjects was not statistically significant for any of the unilateral functional tests. The LSI ranged from 95.1% to 100.6% in the normal group and 80.0% to 89.8% in the PFPS group.

When comparing the right limb of normal subjects with the involved limb of the PFPS group, the normal group scored more repetitions on the step-down, leg press, and bilateral squat. Test scores between groups were statistically different for the step-down test (P < .013).

Discussion

Functional outcome measures should be simple to administer, inexpensive, reliable, and valid. Most of the functional tests previously reported in the literature are targeted to patients after anterior cruciate ligament injury. The purpose of our investigation was to determine the intrarater reliability of 5 functional performance tests. In addition, the relationship between pain and functional test scores was assessed.

Intrarater reliability measures the consistency of a test’s score with respect to time and the evaluator. If a change does occur in the measure, one can attribute the change to true change and not chance. In this study, intrarater reliability was fair to high with a range from 0.79 to 0.94. The SEMs for all tests were less than one repetition, indicating high precision.

Pain is a common symptom of individuals with PFPS.

Table 2. Pearson Correlation Coefficients

<table>
<thead>
<tr>
<th>Functional Test</th>
<th>r Value with Visual Analog Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anteromedial lunge</td>
<td>.730*</td>
</tr>
<tr>
<td>Step down</td>
<td>.503†</td>
</tr>
<tr>
<td>Single-leg press</td>
<td>.386</td>
</tr>
<tr>
<td>Bilateral squat</td>
<td>.461*</td>
</tr>
</tbody>
</table>

*Significant at .05 level.
†Significant at .01 level.
Therefore, a functional test for this population should correlate somewhat with a pain measure. All unilateral functional tests correlated significantly with the VAS. This finding indicates that these 4 functional tests were sensitive to changes in pain level. As pain level decreased, the number of repetitions performed increased. The bilateral squat correlated the least with the VAS and resulted in the lowest reliability. This result is probably due to the bilateral nature of the test. Because weight distribution was not monitored, subjects could shift weight to the uninvolved limb to avoid overloading the involved side.

The 5 functional tests were tested on subjects with unilateral PFPS and, therefore, we hypothesized that there would be a difference in performance between the 2 limbs. For all tests, the uninvolved limb scored higher. Results from the paired t tests reached significance for the anteromedial lunge \((P < .013)\), step-down \((P < .013)\), leg press \((P < .013)\), and balance and reach \((P < .013)\).

We also compared the involved limbs of the PFPS group and the right limbs of the normal group. Surprisingly, the step-down was the only test that was significantly different between the PFPS group and the normal group \((P < .013)\). The normal subjects scored more repetitions on the step-down and leg press but not on the anteromedial lunge or balance and reach. Both the anteromedial lunge and balance-and-reach tests require some work from both limbs, and this may interfere with a differential score.

Since only the step-down test was significantly different between groups, perhaps the LSI is a better indicator of PFPS discrimination. The LSI has been described in the literature as a return-to-sport criterion. Barber et al.\(^4\) suggested an LSI of 85\% as a satisfactory level for determining normalcy in the anterior cruciate ligament-reconstructed patient. For the 5 PFPS functional tests, the LSI ranged from 80.0\% in the step-down to 89.8\% in the balance and reach. The normal group averaged 95\% for the unilateral tests. Because PFPS is so variable and function depends on the presence of pain, a higher LSI of 93 to 95\% for each functional test may be a better predictor of normalcy in this patient population.

Because only intrarater reliability was statistically tested in this study, the results cannot be generalized to other clinicians. Further work is underway to determine the interrater reliability and sensitivity of these 5 functional tests before and after rehabilitation. Clinically, we have noted that subjects with PFPS improve on all the functional test scores and increase their LSI as they progress through rehabilitation.

CONCLUSIONS

The purpose of our study was to investigate the intrarater reliability of 5 functional performance tests. The intrarater reliability proved to be fair to high, with the highest reliability occurring with the step-down test and the lowest with the bilateral squat. The unilateral functional tests correlated significantly with the visual analog scale and differentiated between the involved and uninvolved extremities. However, the limb symmetry index is probably a better discriminator of patellofemoral pain syndrome than the absolute number of repetitions obtained on each test. The key to the reliability of the tests is that the clinician follow standard protocol. Further reliability testing among clinicians needs to be investigated.

The functional tests are designed to be used independently or together. Each test has a particular, unique contribution to the total functional picture. For patients who are unable to tolerate a single-leg squat, the single-leg press can be used to assess quadriceps function. As patients progress, the following 3 tests can be used: (1) the step-down requires balance and eccentric control of the quadriceps, (2) the anteromedial lunge requires a greater range of knee flexion, and (3) the balance and reach requires single-leg balance, limb stability, and proprioception. Before discharge, a patient with patellofemoral dysfunction should be able to complete the unilateral tests with the involved limb and score within 10\% of the uninvolved limb.

ACKNOWLEDGMENTS

This study was funded by the American Physical Therapy Association Sports Physical Therapy Section’s Small Grant Program.

REFERENCES

20. Price DD, McGrath PA, Rafit A, Buckingham B. The validation of visual
analogue scales as ratio scale measures for chronic and experimental pain. *Pain.* 1983;17:45-56.

APPENDIX

Instruction for Functional Performance Tests

1. **Anteromedial lunge:** “You will stand behind a start line and perform 3 lunges with the uninvolved limb. The maximum distance achieved will be used to calculate the 80% target distance. The target distance will be marked on the floor with tape and recorded on the data form. Then, you will stand with your feet straddling the middle line. Your toes must stay behind the central line. Then, you will step out with your — leg so that your heel passes the marked distance (tester will demonstrate). You will continue the lunges for 30 seconds. Do you have any questions?”
   **Criteria:** Only lunges in which the subject’s heel touches beyond the 80% mark will be recorded.

2. **Step-down:** “You will stand on this 8-inch step with both legs. When I say go, you will lower your — leg so that your heel touches the ground. You will then return this leg to the platform and touch the top of the platform. You will continue this sequence until I say stop. The test is run for 30 seconds. Do not push off the ground as you lower your heel. Do you have any questions?”
   **Criteria:** Heel must make contact with a slight hesitation both at the down phase and the start phase. Do not allow the subjects to vault up with their touch leg.
3. **Single-leg press:** “You will start with your back against the sled and your knees fully extended. Place your feet hip-width apart on the standing platform. When I say go, you will bend your — knee and lower your body on the sled to approximately 90° of knee flexion. I will tell you when you achieved the appropriate knee bend. You will continue performing the knee bends for 30 seconds. Do you have any questions?”
   **Criteria:** Foot must remain flat on the Total Gym platform, no vaulting. Full 90° must be achieved (sled must touch platform).

4. **Bilateral squat:** “You will stand with your feet hip-width apart and squat down so that your knees bend to 90° like this (tester demonstrates). Your seat will touch this chair. Do not rest on the chair. You will return to the start position and repeat this activity for 30 seconds. Do you have any questions?”
   **Criteria:** Buttock must touch seat. Subject must reach full standing with full knee extension.

5. **Balance and reach:** “You will stand behind a start line and perform 3 lunges with the uninvolved limb. The maximum distance achieved will be used to calculate the 80% target distance. The target distance will be marked on the floor with tape and recorded on the data form. Stand with your feet straddling the middle line. Your toes must stay behind the central line. You will step out with your — leg so that your heel passes the marked distance (tester will demonstrate). Do not rest your foot down when you reach the target distance. You will continue the reaches for 30 seconds. Do you have any questions?”
   **Criteria:** Only reaches in which the subject’s heel touches beyond the 80% mark will be recorded.
Strength, Functional Outcome, and Postural Stability After Anterior Cruciate Ligament Reconstruction

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Objective: To compare postural stability, single-leg hop, and isokinetic strength measurements in subjects after anterior cruciate ligament (ACL) reconstruction with an age- and activity-matched control group.

Design and Setting: Subjects reported to a sports medicine/athletic training research laboratory for testing. Subjects reported for one testing session for a total test time of 1 hour.

Subjects: Twenty subjects with ACL reconstructions (ACLRs) and 20 age- and activity-matched controls were selected to participate in this study. An anthropometrically assisted central one-third bone-patellar tendon procedure was used to repair the ACLs.

Measurements: We measured concentric and eccentric peak torque (Nm) measurements of the knee extensors and flexors at 120° and 240°/second on an isokinetic dynamometer. Unilateral and bilateral dynamic postural stability was measured as a stability index in the anterior-posterior and medial-lateral planes with the Biodex Stability System. We tested single-leg hop for distance to measure objective function.

Results: We found no significant difference between the ACLR and control subjects for stability index or knee-flexion peak torque scores. On the single-leg hop for distance, the ACLR subjects hopped significantly shorter distances with the involved limb than the uninvolved limb. Furthermore, the ACLR subjects' single-leg hop distance was significantly less when the involved limb was compared with the control-group matched involved limb, and the ACLR subjects performed significantly better when the uninvolved limb was compared with the control-group matched uninvolved limb. The ACLR subjects produced significantly greater torque in the uninvolved leg than in the involved leg. In addition, the peak torque was significantly less for the involved limb in the ACLR group when compared with the matched involved limb of the control group.

Conclusions: After ACLR (mean = 18 ± 10 months), single-leg hop-for-distance scores and quadriceps strength were not within normal limits when compared with the contralateral limb. Our results suggest that bilateral and single-limb postural stability in the ACLR group was not significantly different than the control group at an average follow-up of 18 months after surgery.

Key Words: ACL reconstruction, balance, single-leg hop, isokinetic strength, Biodex stability system

Injury to the anterior cruciate ligament (ACL) results in mechanical and functional instability. Athletes often find it difficult to return to full function after injury to the ACL, and surgery is frequently indicated.1 The purpose of surgery is to reestablish joint stability; surgeons attempt to minimize disruption to surrounding soft tissue during reconstruction. However, the implantation of a substitute for the ACL does not restore the sensorimotor sensory system, which may result in a compromised afferent neural system.2,3 Failure of stretched or damaged ligaments to provide adequate sensory feedback in the injured knee may contribute to loss of function and result in degeneration of the knee.4 Proprioceptive afferent neural input is also important in functional control during sport activities.5 It has been suggested that, after surgery, the ability to perform functional activities and balance may be decreased6-11; deficits have been found in the muscular and sensory processes after reconstructive surgery. Specifically, after ACL reconstruction with the bone-patellar tendon-bone procedure, strength deficits of 5% to 34% have been reported in the involved extremity compared with the contralateral limb after rehabilitation.12 Muscle control,13-17 gait,15 functional activities,7,14,15,17-21 and proprioception7,16,18,19,22,23 have been evaluated after ACL reconstruction, while the effect of dynamic postural stability has been minimally evaluated.24 Joint injury and articular disease have been shown to adversely affect joint position sense, movement sense, and function.7-9,11,23,25 Damage to receptors in the skin, muscles, tendons, and articular structures affects the ability to detect body movement and position. Without the normal integration of these processes, a person may be unable to perform physical activity in an efficient manner.

The use of force platforms has provided a sensitive method
for measuring postural stability. However, the limited movement of the force platform is not indicative of normal joint movement during normal activities. Advances in technology have now made it possible to evaluate postural control more extensively than previously; for example, computer-interfaced devices enable postural stability to be quantified. The Biodex Stability System (Biodex Medical Systems, Shirley, NY) is a device that is purported to reliably assess a patient's neuromuscular control in a closed-chain manner. A multiplane test is used to quantify the ability of a patient to maintain dynamic unilateral or bilateral postural stability on an unstable surface.

The purpose of our study was to compare postural stability, single-leg hop, and isokinetic strength measurements in subjects after ACL reconstruction with an age- and activity-matched control group.

**METHODS**

**Subjects**

Postural stability, functional assessment, and isokinetic strength measures were evaluated in 20 subjects (11 men, 9 women) with a history of one surgery for ACL reconstruction (age = 25.8 ± 8.1 years, height = 175.8 ± 8.5 cm, weight = 73.3 ± 14.0 kg) and 20 age- and activity-matched subjects (11 men, 9 women) who served as the control group (age = 24.5 ± 6.9 years, height = 175.8 ± 8.3 cm, weight = 71.4 ± 12.1 kg). Recruited subjects all had ACL reconstruction performed in a similar fashion (arthroscopically assisted central bone-patellar tendon-bone graft). The mean time since surgery was 18 ± 10 months. Activity was matched as closely as possible using sections B and C on the Sports Participation Survey originally described by Seto et al. The study was approved by an institutional review board, and all subjects signed an informed consent form before participating.

**Criteria for Participation**

Subjects were selected to participate if they met the following criteria: (1) had only one surgery for a tear of the ACL that did not include a concomitant tear of the posterior cruciate ligament, (2) no evidence of collateral ligament repair at the time of surgery, (3) no history of surgery or traumatic injury to the contralateral knee, (4) no history of surgery or traumatic injury to the ankle joint on the reconstructed side, (5) no history of surgery or traumatic injury to either hip joint, and (6) no history of a medical problem that limited activities within the 6 weeks before testing. All subjects were released from a formal rehabilitation program before participating; however, standardization of the programs was not possible.

**Testing Procedures**

Subjects reported to the sports medicine/athletic training research laboratory for one testing session for a total test time of 1 hour. Before testing, subjects filled out the informed consent agreement and the Sports Participation Survey. The testing order for the postural stability, strength, and single-leg hop tests was counterbalanced to avoid a learning or fatigue effect. The testing session commenced by riding a Fitron (Cybex Corp, Ronkonkoma, NY) stationary bicycle for a 5-minute warm-up. Subjects were then instructed to perform several lower body flexibility exercises.

**Biodex Stability System**

Dynamic postural stability was assessed with the BSS. The support platform of the BSS can be placed at 6 levels. The resistance of the foot platform changes at each level. A setting of 6 is the most stable foot platform setting, and a setting of 1 is the least stable setting. At any level, the foot platform can move a full 20° in any direction. The measure of postural stability was the anterior-posterior and medial-lateral stability indexes (SI). The SI represents the standard deviation of foot platform deflection in degrees from the level position during a test. A high number indicates substantial movement away from the subject's center of balance; a low number indicates minimal movement during the test. The order of testing was counterbalanced to avoid any learning or fatigue effect. Intratester reliability for a protocol with decreasing stability levels on the BSS has been previously reported to be clinically reliable, with intraclass correlation coefficients ranging from .80 to .93.

**Pretest.** We assessed single-limb and bilateral stance. Order of testing was counterbalanced to control for bias and fatigue. Subjects were asked to step on the platform of the BSS and assume a comfortable position on the platform while maintaining slight flexion in the knees (10° to 15°). Subjects were asked to stand with the knees flexed to 10° to 15° and to look straight ahead at an X marked directly in front of them while attempting to maintain the platform in a level position. They were given a 1-minute rest between testing conditions.

**Single-Leg Hop-for-Distance Test**

The single-leg hop for distance is a commonly used functional measurement designed to test both strength and confidence in the tested leg that correlates positively with muscular strength. The first extremity to be tested was randomly chosen. The single-leg hop was performed 3 times with each leg. Subjects were asked to hop as far as possible from a predetermined line and to land on the same leg. Use of arm swing was not discouraged, as subjects were asked to perform with maximal effort. The best distance of the 3 tests was recorded in centimeters and used as the dependent score.

**Isokinetic Evaluation**

Strength testing was performed for knee flexion and knee extension at 120° per second and 240° per second on the KinCom dynamometer (Chattanooga Group, Hixson, TN) in the
Table 1. Single-Limb Postural Stability Index (°) for Anterior Cruciate Ligament-Reconstructed (ACLR) and Control Subjects (Mean ± SD)

<table>
<thead>
<tr>
<th>Group</th>
<th>Involved</th>
<th>Uninvolved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posterolateral</td>
<td>Anterior</td>
<td>Medial</td>
</tr>
<tr>
<td>Posterolateral</td>
<td>Posterior</td>
<td>Lateral</td>
</tr>
<tr>
<td>Control (n = 20)</td>
<td>4.8 ± 2.2</td>
<td>2.6 ± 0.86</td>
</tr>
<tr>
<td>ACLR (n = 20)</td>
<td>4.3 ± 1.5</td>
<td>2.8 ± 2.2</td>
</tr>
</tbody>
</table>

Table 2. Bilateral Limb Postural Stability Index (°) for Anterior Cruciate Ligament-Reconstructed (ACLR) and Control Subjects (Mean ± SD)

<table>
<thead>
<tr>
<th>Group</th>
<th>Anterior</th>
<th>Medial</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posterolateral</td>
<td>Posterior</td>
<td>Lateral</td>
</tr>
<tr>
<td>Control (n = 20)</td>
<td>4.4 ± 1.5</td>
<td>3.2 ± 1.5</td>
</tr>
<tr>
<td>ACLR (n = 20)</td>
<td>5.0 ± 2.0</td>
<td>3.0 ± 1.7</td>
</tr>
</tbody>
</table>

Seated position. Concentric and eccentric contractions were performed at each velocity. The first extremity and velocity to be tested were counterbalanced to prevent fatigue or learning effects. Subjects were seated on the dynamometer and stabilized with chest and leg hook-and-loop straps according to the manufacturer’s guidelines. The axis of rotation of the Kin-Com was adjusted so as to align with the joint margin of the knee. The distal pad of the dynamometer arm was placed just proximal to the malleoli. Before testing, we asked subjects to extend the leg; the weight of the limb was recorded and corrected for gravity using the Kin-Com software package. Before data collection, subjects performed 4 practice repetitions for each velocity setting at 75% of subjective maximal effort. Each concentric contraction was followed by an eccentric contraction for both extension and flexion of the knee joint. After this warm-up phase, a 2-minute rest was given. The evaluation phase consisted of 3 repetitions of maximal concentric and eccentric contractions for extension and flexion of each leg. We informed subjects that they needed “to push or pull as hard and fast as they can” against the resistance provided by the dynamometer. Order of testing was counterbalanced to prevent a fatigue or learning effect. A 5-minute rest period was given before the opposite leg was tested. Peak torque values were used as the dependent measure of muscle strength.

Statistical Analyses

Extremity matching was achieved by matching the injured extremity (right/left) from the ACL-reconstruction (ACLR) subject with the same extremity in the uninjured subject. We used a repeated-measures analysis of variance (ANOVA) with 1 between-subjects factor (group) and 1 within-subjects factor (plane) to determine if differences existed for bilateral postural stability assessed with the BSS. With a repeated-measures ANOVA with 1 between-subjects factor (group) and 2 within-subjects factors (extremity and plane), we examined differences in single-limb postural stability. A repeated-measures ANOVA with 1 between-subjects factor (group) and 1 within-subjects factor (extremity) was calculated to assess differences in the single-leg hop-for-distance test. We assessed differences between hamstrings and quadriceps knee muscle strength with 2 repeated-measures ANOVAs with 1 between-subjects factor (group) and 3 within-subjects factors (extremity, contraction, and velocity). Tukey Honestly Significant Differences post hoc comparisons were performed for all significant interactions, and all statistical tests were considered significant at the $P < .05$ level.

RESULTS

The postural stability, single-leg hop test, and strength descriptive data are found in Tables 1–6. We found no differences between the ACLR and control subjects for the single-limb and bilateral stability index scores. For the single-leg hop test, the group-by-extremity interaction was significant ($F_{1,38} = 3.78, P < .01$). Additionally, the analysis revealed a main effect for extremity ($F_{1,38} = 9.09, P < .01$). Using Tukey post hoc analysis, we noted that ACLR subjects hopped a significantly shorter distance with the involved limb than with the uninvolved limb ($P < .01$). Furthermore, the ACLR subjects’ performance for the hop test was significantly worse when the involved limb was compared with the control group’s matched limb ($P < .05$), and the ACLR subjects performed significantly better when the uninvolved limb was compared with the control group’s matched limb ($P < .01$).

For knee-flexion strength, there was no significant differences between the ACLR and control groups or between extremities. For knee-extension strength, there was a significant interaction for group by extremity ($F_{1,38} = 9.40, P < .01$). Additional significant 2-way interactions were found for extremity by velocity ($F_{1,38} = 6.03, P < .05$) and contraction by velocity ($F_{1,38} = 103.7, P < .01$). Tukey post hoc analysis revealed that ACLR subjects produced significantly greater

Table 3. Knee-Flexion Values Measured as Peak Torque (N·m) for Anterior Cruciate Ligament–Reconstructed (ACLR) and Control Subjects (Mean ± SD)

<table>
<thead>
<tr>
<th>Group</th>
<th>Extremity</th>
<th>120°·s⁻¹</th>
<th>240°·s⁻¹</th>
<th>120°·s⁻¹</th>
<th>240°·s⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Involved</td>
<td></td>
<td></td>
<td>Uninvolved</td>
<td></td>
</tr>
<tr>
<td>ACLR (n = 20)</td>
<td>Concentric</td>
<td>189.5 ± 49.0</td>
<td>185.6 ± 50.8</td>
<td>201.3 ± 45.7</td>
<td>194.9 ± 61.6</td>
</tr>
<tr>
<td></td>
<td>Eccentric</td>
<td>269.8 ± 60.4</td>
<td>290.6 ± 65.9</td>
<td>291.1 ± 67.8</td>
<td>303.5 ± 80.9</td>
</tr>
<tr>
<td>Control (n = 20)</td>
<td>Concentric</td>
<td>196.3 ± 45.0</td>
<td>172.6 ± 34.5</td>
<td>195.9 ± 43.7</td>
<td>175.0 ± 41.0</td>
</tr>
<tr>
<td></td>
<td>Eccentric</td>
<td>285.9 ± 92.9</td>
<td>302.8 ± 86.9</td>
<td>294.6 ± 96.9</td>
<td>302.7 ± 85.8</td>
</tr>
</tbody>
</table>
Table 4. Knee-Extension Values Measured as Peak Torque (N-m) for Anterior Cruciate Ligament-Reconstructed (ACLR) and Control Subjects (Mean ± SD)

<table>
<thead>
<tr>
<th>Group</th>
<th>Involved</th>
<th>Uninvolved</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>120° · s⁻¹</td>
<td>240° · s⁻¹</td>
</tr>
<tr>
<td>ACLR (n = 20)</td>
<td>Concentric</td>
<td>351.8 ± 99.1</td>
</tr>
<tr>
<td></td>
<td>Eccentric</td>
<td>434.2 ± 127.4</td>
</tr>
<tr>
<td>Control (n = 20)</td>
<td>Concentric</td>
<td>411.7 ± 107.7</td>
</tr>
<tr>
<td></td>
<td>Eccentric</td>
<td>491.0 ± 138.1</td>
</tr>
</tbody>
</table>

Table 5. Group-by-Extremity Interaction for the Single-Leg Hop-for-Distance Test (cm) for Anterior Cruciate Ligament Reconstructed (ACLR) and Control Subjects (Mean ± SD)

<table>
<thead>
<tr>
<th>Group</th>
<th>Involved</th>
<th>Uninvolved</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>120° · s⁻¹</td>
<td>240° · s⁻¹</td>
</tr>
<tr>
<td>ACLR (n = 20)</td>
<td>Concentric</td>
<td>173.6 ± 27.9*</td>
</tr>
<tr>
<td>Control (n = 20)</td>
<td>Concentric</td>
<td>186.0 ± 30.1§</td>
</tr>
</tbody>
</table>

Table 6. Group-by-Extremity Interaction for Knee Extension as above (Mean ± SD)

<table>
<thead>
<tr>
<th>Group</th>
<th>Involved</th>
<th>Uninvolved</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>120° · s⁻¹</td>
<td>240° · s⁻¹</td>
</tr>
<tr>
<td>ACLR (n = 20)</td>
<td>Concentric</td>
<td>388.1 ± 125.8*</td>
</tr>
<tr>
<td>Control (n = 20)</td>
<td>Concentric</td>
<td>461 ± 125.1‡</td>
</tr>
</tbody>
</table>

Mizuta et al.25 compared a group of ACL-deficient patients who were functionally stable with a group of patients who were functionally unstable. Functional stability was defined as full return, without giving way, to the same sport at the same level as before injury. The authors found that the functionally unstable group swayed significantly more than the functionally stable group. Therefore, deficits in postural stability have been demonstrated in ACL-deficient patients who complain of the knee “giving way” but not in a group of patients who were identified as functionally stable. It appears that some ACL-deficient patients are able to function without the knee “giving way.” Some ACL-deficient patients may compensate neuromuscularly in adapting to the loss of the ACL. Similar to the results of the functionally stable group in the Mizuta et al.25 study, our results revealed no differences in postural stability for a group of subjects after ACLR.

Deficits in proprioception exist after ACLR. In subjects 11 to 26 months postsurgery, Lephart et al.23 demonstrated a significant kinesthetic deficit in the ACLR knee compared with the uninvolved knee, from a starting position of 15° moving into both flexion and extension. The time since surgery and the angle of the knee during testing were similar to our study. However, their testing was performed in a nonweight-bearing position whereas our study used a weight-bearing position. The lack of significant differences in postural stability between the reconstructed and uninvolved knees and between the reconstructed knee and the matched extremity of the control group in our study indicates that, after ACLR and rehabilitation, any loss in the ability to maintain dynamic postural stability returns to normal. It may be that the lack of significant differences in postural stability in our study is the result of a combination of restoring mechanical stability via the reconstruction, restoring neuromuscular control via the rehabilita-
tion process, and performing the test in a weight-bearing position. Therefore, it may be that proprioceptive deficits exist as described by Lephart et al\textsuperscript{23} but that the interval from surgery in our subjects was long enough that proprioception in the joint was restored. However, it may be more likely that muscle afferent receptors dominated sensory feedback during the balance episodes.

**Single-Leg Hop-For-Distance Test**

The single-leg hop-for-distance test was chosen as an objective functional test that would provide stress to the knee joint while also allowing us to evaluate strength and confidence in the tested extremity. Subjects who underwent ACLR hopped farther with the uninvolved limb than with the involved limb. The significant difference in the single-leg hop-for-distance test scores between the involved and uninvolved extremities for the ACLR group suggests that, at an average of 18 months postreconstruction, this measure of functional performance was not within normal limits. Furthermore, ACLR subjects were unable to hop as far as the control group when the “involved” limbs were compared. This finding suggests that the ACLR subjects’ ability to perform a single-leg hop-for-distance test was not within normal limits when compared with a matched control group. However, the ACLR subjects were able to hop a significantly greater distance when the uninvolved limb was compared with the uninvolved limb of the control group. This result suggests that strength was significantly increased in the ACLR subjects’ uninvolved leg when compared with the uninvolved leg of the matched control subjects. The increased strength in the uninvolved leg may have occurred to compensate for the loss of function after the injury and subsequent surgical reconstruction.

Single-leg hop-for-distance scores are commonly expressed as a limb symmetry index. The limb symmetry index is calculated as the mean score of the involved limb divided by the mean score of the uninvolved limb, with the result multiplied by 100.

Noyes et al\textsuperscript{10} assessed the sensitivity of 4 types of single-leg hop tests for a group of ACL-deficient patients. The 4 hop tests were the single-leg hop for distance, the timed hop, the triple hop for distance, and the crossover hop for distance. Noyes et al\textsuperscript{10} described a limb symmetry score of below 85% as abnormal. In a similar study, Wilk et al\textsuperscript{21} examined the relationship between isokinetic testing and functional testing for a group of ACLR patients. They compared 3 functional tests: the single-leg hop for distance, the single-leg timed hop, and the single-leg crossover. We chose to assess only the single-leg hop-for-distance test because of time and fatigue considerations.

When the single-leg hop-for-distance scores in our study are expressed as a limb symmetry index, 43% of the ACLR patients had a limb symmetry score below 85%, versus 47% of the subjects described by Wilk et al.\textsuperscript{21} Our findings are similar to those of Wilk et al,\textsuperscript{21} although the time since surgery in their study was 6.45 months, versus 18.1 months in our study. The longer duration since surgery could account for the decreased number of abnormal limb symmetry scores in our study.

We found no difference when comparing the single-leg hop scores between the involved and uninvolved extremities of the control group, which is consistent with Greenberger and Paterno.\textsuperscript{33} Our results and others suggest that clinicians may want to concentrate on improving functional strength after ACLR.\textsuperscript{10,21}

**Isokinetic Strength**

**Knee Flexion.** Exercises that focus on strengthening the hamstring musculature are recommended after ACLR in an attempt to reduce anterior translation forces of the tibia. The lack of a difference in peak torque during knee flexion supports previous findings that after 12 to 14 weeks post-ACLR, knee-flexion strength returns to near-normal levels.\textsuperscript{34} Our findings are inconsistent with those of Seto et al,\textsuperscript{14} who reported that hamstring strength in the reconstructed limb was significantly less than that in the control leg at 120 and 240° per second for subjects who had an intra-articular ACLR. The reported differences between the Seto et al\textsuperscript{15} study and our investigation may be attributed to the more conservative rehabilitation process that was followed at the time that study was conducted.

**Knee Extension.** Aggressive rehabilitation after ACLR commonly employs immediate motion, weight bearing, and exercise to initiate quadriceps contraction.\textsuperscript{34} However, quadriceps strength is slow to return to normal levels. Our results indicate that ACLR subjects produced significantly more torque with the uninvolved knee than with the reconstructed knee. The strength of the knee extensors for the ACLR subjects may not have returned to preinjury levels. Similarly, the matched involved knee of the control group produced significantly greater torque than the reconstructed knee of the ACLR subjects. As such, the quadriceps muscle strength of ACLR subjects in our study had not returned to near-normal levels after an average of 18 months after surgical repair. Our findings are consistent with those of Seto et al\textsuperscript{14} and Hoffman et al,\textsuperscript{24} who reported that quadriceps strength in the reconstructed limb was significantly less than that in the control leg for subjects who had an intra-articular ACLR. For comparison, patients in the Seto et al\textsuperscript{14} study underwent an intra-articular or extra-articular ACLR, and patients in the Hoffman et al\textsuperscript{24} study underwent an arthroscopically assisted patellar tendon graft.

The differences in strength and function but not in postural stability may be explained by the specificity of the exercise and possible compensation by other lower extremity muscle groups. The ability to perform a single-leg hop depends on the strength of the quadriceps muscle. A decrease in quadriceps strength would result in reduced loading capacity of the knee joint and the inability to absorb and generate force.\textsuperscript{35} In addition, the influence of the graft selection cannot be disregarded, as strength deficits of 5% to 34% have been reported after ACLR with the bone-patellar tendon-bone procedure and subsequent rehabilitation.\textsuperscript{12} The ability to balance on an unstable platform requires the coordinated activation of the lower leg musculature. While knee extension and the single-leg hop require maximal contraction of the supporting musculature, single-limb and double-limb balance do not. Therefore, the ability to balance on the dynamic platform may not have been a sufficient challenge. The use of different methods to maintain balance has been defined as a strategy. In 1990, Horak et al\textsuperscript{36} described these strategies as “stereotypical movement patterns in order to achieve or maintain postural stability during anterior/posterior sway with a fixed stance.” These strategies most often involve using primarily the ankle or the hip for neuromuscular control. Therefore, activation of other muscle...
groups (ie, ankle and hip) in addition to the quadriceps may have accounted for the lack of difference in single-limb and bilateral balance in our study.37

Limitations

One limitation with our study was that it was not possible to account for differences in rehabilitation programs among subjects. All the ACLR subjects were subjectively asked how long they participated in a physical therapy program; the average length of time was 8 to 10 weeks. Therefore, we could not account for the differences in rehabilitation programs, nor could we control individual compliance in these programs. A patellar-tendon autograft procedure was used to repair the torn ACL in all ACLR subjects. Every attempt was made to obtain all subjects from the same physician; however, due to difficulty in subject recruitment, 14 of the ACLR patients were operated on by the same surgeon, while the other 7 patients each had a different surgeon. Different physicians, rehabilitation programs, and compliance to the rehabilitation programs may have reduced the homogeneity of our group, making it more difficult to detect differences. It would be interesting to further investigate postural stability before the reconstruction process and with more control of the subjects and their rehabilitation after surgery. Further research should examine the length of time that postural stability deficits exist after surgery and when these approach normal. Further knowledge of this process would aid clinicians in their decision on when to return patients to full activity after ACLR.

CONCLUSIONS

After ACLR (mean = 18 ± 10 months), subjects did not have significant loss in bilateral or single-limb postural stability when assessed with a Biodex Stability System. However, within the limits of our study, quadriceps strength and functional hop performance were not within normal limits when compared with the contralateral limb and a control group. Of clinical importance and in agreement with others38 is the fact that leg strength and functional performance (as assessed with a single-leg hop-for-distance test) may not return to normal (±5%) for up to 2 years. In addition, clinicians must emphasize that quadriceps femoris strength be maintained after organized therapy for ACLR. If the subjects in our study are indicative of the general population, deficits in strength and function may predispose them to limited performance and possibly further injury.

REFERENCES


The Spectral Qualities of Postural Control are Unaffected by 4 Days of Ankle-Brace Application

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Objective: To determine if the spectral qualities of mediolateral (ML) and anterior-posterior (AP) center of pressure during a 1-legged stance are affected by 4 days of ankle-brace application.

Design and Setting: The study, which consisted of a pretest-posttest randomized group design, took place in the Sports Injury Research Laboratory at Indiana State University.

Subjects: Twenty-eight Indiana State University students, who had not suffered from any ankle injuries within the past 2 years and were free of any neurologic or vestibular disorders, were examined: (1) treatment (brace, control), (2) frequency bin (0%-20%, 20%-40%, 40%-60%, 60%-80%, and 80%-100% of area), and (3) time (pretest, immediately after brace application, and after 1, 2, 3, or 4 days of brace wear).

Results: We detected no difference (P > .05) for the ML or AP mean frequency when comparing the brace and control groups.

Conclusions: Application of an ankle brace may not require modifications in the postural-control strategies during a 1-legged stance in subjects with healthy ankles.

Key Words: fast Fourier transformation, somatosensation, frequency analysis, balance
Clinicians and researchers have attempted to focus on the proprioceptive system by introducing somatosensory manipulations (ie, change in support surface).\textsuperscript{11,17} Although these attempts may challenge the proprioceptive system, the other 2 sensory systems may compensate for the somatosensory change.

Center of pressure (COP) is the center of the distribution of the total force applied to the supporting surface.\textsuperscript{18} By examining the frequency (spectral) characteristics of COP data, it is possible to “tease out” the 3 sensory systems working to maintain postural equilibrium. It is thought that each of the 3 sensory systems operates within a specific frequency bandwidth.\textsuperscript{19} Theoretically, when one of these systems is altered, a change in the frequency spectrum is detected within the specified system’s operating band. For example, application of an ankle brace introduces an additional somatosensory input, which may influence the proprioceptive system. An alteration in the frequency spectrum after application of the ankle brace would theoretically be due to the added somatosensory input. An increase in the amplitude within the proprioceptive frequency range would represent increased work by the proprioceptive system to maintain posture, whereas a decreased amplitude value would suggest decreased work by the proprioceptive system.

The analysis of the spectral characteristics associated with postural control with the influence of ankle bracing has not been extensively examined. In order for us to gain a thorough understanding of ankle bracing’s potential effects on the proprioceptive control of posture, additional examination is warranted. Therefore, our main purpose was to investigate the effects of 4 days of ankle-brace use on the mean frequency amplitude of medial-lateral (ML) and anterior-posterior (AP) COP during a 1-legged stance.

\section*{METHODS}

\subsection*{Subjects}

Twenty-eight college students (age = 22 ± 1.8 years, mass = 72.3 ± 12.0 kg, height = 172.6 ± 9.0 cm) volunteered to participate in this study. No subjects had incurred any ankle injuries within the last 2 years, and all were free from any neurologic or vestibular conditions that interfered with their ability to maintain upright stance. Furthermore, subjects had not suffered any head injury resulting in unconsciousness. Any subject reporting prior ankle-brace use was eliminated. Before testing and after the purpose of the study had been explained, all subjects gave written informed consent. The protocol was reviewed and approved by the School of Health and Human Performance Human Subjects Committee at Indiana State University.

\subsection*{Instrumentation}

We used a strain-gauge force-platform system (Accusway; Advanced Mechanical Technology, Inc, Watertown, MA) to measure postural control. The platform measures 3 translational forces (Fx, Fy, and Fz) and 3 moments of force (Mx, My, and Mz). The data were digitally converted at 50 Hz and interfaced to a controlling laptop computer (model 1250, Compaq Computer Corp, Wilmington, OH). The SWAYWIN software (Advanced Mechanical Technology, Inc) was used to generate the X and Y coordinates representing the COP in the medial-lateral and anterior-posterior directions, respectively. This software was also used to convert the time-domain data into frequency-domain data with the fast Fourier transformation (FFT) technique.

\subsection*{Protocol}

Before testing began, each subject was randomly assigned to either the experimental (brace) or control (no-brace) group. All subjects were asked the preferred leg for kicking a ball in order to determine leg dominance; the leg established as dominant was then used for testing.

A pretest measurement for both groups was recorded without application of an ankle brace. For this and all subsequent measurements, subjects were instructed to step onto the force platform and assume a 1-legged stance. This required the subjects to stand on the test leg with their eyes closed and hands fixed against the body (ie, hands on the iliac crests) (Figure 1). Furthermore, during each testing session, all subjects wore opaque goggles to prevent any input from the visual system. The maintenance of upright stance is influenced by the visual system as well as the vestibular and proprioceptive systems. Vision was minimized so that we were able to focus on potential changes due to input from the proprioceptive system. Data sampling was initiated after the subject was properly positioned on the force plate.

Additionally, subjects were asked to stand quietly and as motionless as possible in the stance position. They were instructed to keep their hands fixed and not to touch down with the nonstance leg. If subjects began to lose their balance, a quick tap of both elbows by the investigator (R.M.P.) was allowed for each subject to regain control. This touch lasted no longer than 3 seconds.

If this had to be repeated more than twice in a 20-second trial, the trial was considered unsuccessful and was redone.
Also, if the subject touched down with the nonstance leg or was unable to sustain the stance position for 20 seconds, the trial was considered incomplete and was redone. Subjects were required to successfully maintain the stance position for 20 seconds per trial for a total of 5 trials, with a 30-second rest period between trials.

After the pretest measurement, all subjects were properly fitted with a lace-up ankle brace (model A101, McDavid Knee Guard Inc, Chicago, IL), according to the manufacturer’s specifications, including applying the brace over the sock. Five additional trials were then performed after application of the ankle brace. The instructions given to each subject during pretesting did not change throughout the course of study. However, for the remainder of the study, all subjects wore an ankle brace during testing.

After all 5 trials, the subjects in the control group removed the brace, while the subjects in the brace group continued to wear the ankle brace. After 8 consecutive hours of ankle-brace wear, subjects in the experimental group removed the brace. The next day, subjects in both the brace and control groups again reported to the laboratory for testing. Both groups placed the ankle brace on the test leg and stepped on the force platform for the data to be collected. The procedure used to gather the data on the previous day was followed for each of the subsequent testing sessions. The subjects reported for testing on 5 consecutive days. The time between testing sessions was between 24 and 32 hours. Subjects in the experimental group wore the ankle brace for a total of 32 hours over the 4 days. This duration of time was chosen because we felt it closely represented the usage of this device during a week of preseason training completed by a competitive athlete. Subjects in the control group wore the brace during testing for approximately 1 hour and 15 minutes.

Data Reduction

The trials performed by each subject occurred over a 20-second time period. The FFT was run on the time-domain data in order to convert it to its respective frequency domain. Within a single 20-second trial, ML and AP COP data points were collected at intervals of 0.02 seconds (50 Hz) so that 501 data points each from the ML and the AP COP were recorded for stance.

The FFT algorithm analyzes frequencies up to one half the frequency at which the data were acquired; in this case, the data were collected at 50 Hz, and the highest frequency the FFT detected was 25 Hz. The FFT spectrum was condensed to 256 data points from 501 as described above. The software used to run the FFT on our data omits the first data point. This data point represents the direct current component, and its magnitude reduces the overall resolution of the FFT, which is why it was removed. The FFT spectrum analyzed in this study consisted of a total of 255 points. Once the FFT was performed, the 255 data points were imported into Excel spreadsheets (version 9.0, Microsoft Corp, Redmond, WA) so that the frequency bins could be created. Five frequency bins were developed based on pilot data. Each bin represented 20% of the total area under the FFT curve (Figure 2). Frequency bins were created separately for ML and AP COP data.

The total area of the frequency spectrum was calculated for each subject and each trial for both ML and AP COP. The area was determined by averaging the 255 amplitude values and then multiplying that value by 25. Twenty-five represents the maximum frequency at which data may have been acquired.

Once the area was calculated, each of the 255 amplitude values was multiplied by 0.02 (the time interval at which data points were collected). This quantity for each value was summed to all prior amplitude values (ie, for amplitude value 232, the sum of all amplitudes from 1 to 232 was taken) and then divided by the total area of the spectrum so that we could determine the percentage of the total area that each data point represents. The data points were reviewed and separated into the frequency bins (each representing 20% of the area under the FFT curve) previously created from the pilot data. The mean amplitude in each bin was used for analyses.

DATA ANALYSIS

A 2 X 5 X 6 repeated-measures analysis of variance was used to assess differences among conditions, bins, and time and the interaction of brace, time, and bin on ML and AP
Table 1. Medial-Lateral Mean Amplitude Values for Bins 1 through 5 at Time 0 to Time 5 (cm ± SD)

<table>
<thead>
<tr>
<th>Time</th>
<th>Bins 1</th>
<th>Bins 2</th>
<th>Bins 3</th>
<th>Bins 4</th>
<th>Bins 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.060 ± 0.13</td>
<td>0.047 ± 0.09</td>
<td>0.027 ± 0.041</td>
<td>0.011 ± 0.02</td>
<td>0.001 ± 0.02</td>
</tr>
<tr>
<td>1</td>
<td>0.069 ± 0.13</td>
<td>0.050 ± 0.12</td>
<td>0.025 ± 0.06</td>
<td>0.009 ± 0.02</td>
<td>0.009 ± 0.02</td>
</tr>
<tr>
<td>2</td>
<td>0.073 ± 0.16</td>
<td>0.050 ± 0.13</td>
<td>0.031 ± 0.17</td>
<td>0.011 ± 0.06</td>
<td>0.001 ± 0.01</td>
</tr>
<tr>
<td>3</td>
<td>0.068 ± 0.16</td>
<td>0.047 ± 0.08</td>
<td>0.032 ± 0.19</td>
<td>0.011 ± 0.03</td>
<td>0.002 ± 0.02</td>
</tr>
<tr>
<td>4</td>
<td>0.069 ± 0.15</td>
<td>0.050 ± 0.20</td>
<td>0.026 ± 0.05</td>
<td>0.010 ± 0.05</td>
<td>0.002 ± 0.01</td>
</tr>
<tr>
<td>5</td>
<td>0.079 ± 0.60</td>
<td>0.046 ± 0.09</td>
<td>0.031 ± 0.24</td>
<td>0.014 ± 0.10</td>
<td>0.001 ± 0.01</td>
</tr>
</tbody>
</table>

Table 2. Anterior-Posterior Mean Amplitude Values for Bins 1 through 5 at Time 0 to Time 5 (cm ± SD)

<table>
<thead>
<tr>
<th>Time</th>
<th>Bins 1</th>
<th>Bins 2</th>
<th>Bins 3</th>
<th>Bins 4</th>
<th>Bins 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.070 ± 0.028</td>
<td>0.042 ± 0.12</td>
<td>0.023 ± 0.05</td>
<td>0.009 ± 0.02</td>
<td>0.001 ± 0.01</td>
</tr>
<tr>
<td>1</td>
<td>0.106 ± 0.156</td>
<td>0.044 ± 0.12</td>
<td>0.022 ± 0.06</td>
<td>0.008 ± 0.02</td>
<td>0.001 ± 0.02</td>
</tr>
<tr>
<td>2</td>
<td>0.083 ± 0.036</td>
<td>0.042 ± 0.10</td>
<td>0.022 ± 0.05</td>
<td>0.008 ± 0.02</td>
<td>0.001 ± 0.01</td>
</tr>
<tr>
<td>3</td>
<td>0.080 ± 0.033</td>
<td>0.042 ± 0.11</td>
<td>0.022 ± 0.07</td>
<td>0.008 ± 0.03</td>
<td>0.001 ± 0.01</td>
</tr>
<tr>
<td>4</td>
<td>0.085 ± 0.044</td>
<td>0.043 ± 0.10</td>
<td>0.023 ± 0.05</td>
<td>0.011 ± 0.11</td>
<td>0.001 ± 0.01</td>
</tr>
<tr>
<td>5</td>
<td>0.087 ± 0.055</td>
<td>0.043 ± 0.11</td>
<td>0.023 ± 0.05</td>
<td>0.008 ± 0.02</td>
<td>0.001 ± 0.01</td>
</tr>
</tbody>
</table>

Table 3. Medial-Lateral Mean Amplitude Values for Brace (B) and Control (C) Conditions at Each Time for Bins 1 through 5

<table>
<thead>
<tr>
<th>Time</th>
<th>1 (0-0.39 Hz)</th>
<th>2 (0.40-0.88 Hz)</th>
<th>3 (0.89-1.9 Hz)</th>
<th>4 (2.0-4.2 Hz)</th>
<th>5 (4.3-25 Hz)</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>0.066 ± 0.014</td>
<td>0.048 ± 0.010</td>
<td>0.027 ± 0.006</td>
<td>0.011 ± 0.02</td>
<td>0.001 ± 0.04</td>
</tr>
<tr>
<td>C</td>
<td>0.055 ± 0.009</td>
<td>0.046 ± 0.005</td>
<td>0.027 ± 0.007</td>
<td>0.012 ± 0.05</td>
<td>0.001 ± 0.01</td>
</tr>
<tr>
<td>Time 1</td>
<td>B</td>
<td>0.071 ± 0.016</td>
<td>0.046 ± 0.011</td>
<td>0.026 ± 0.007</td>
<td>0.009 ± 0.02</td>
</tr>
<tr>
<td>C</td>
<td>0.066 ± 0.046</td>
<td>0.052 ± 0.013</td>
<td>0.023 ± 0.010</td>
<td>0.010 ± 0.02</td>
<td>0.001 ± 0.01</td>
</tr>
<tr>
<td>Time 2</td>
<td>B</td>
<td>0.069 ± 0.011</td>
<td>0.048 ± 0.011</td>
<td>0.030 ± 0.011</td>
<td>0.011 ± 0.07</td>
</tr>
<tr>
<td>C</td>
<td>0.023 ± 0.078</td>
<td>0.051 ± 0.014</td>
<td>0.033 ± 0.020</td>
<td>0.011 ± 0.03</td>
<td>0.001 ± 0.01</td>
</tr>
<tr>
<td>Time 3</td>
<td>B</td>
<td>0.070 ± 0.019</td>
<td>0.049 ± 0.007</td>
<td>0.025 ± 0.006</td>
<td>0.010 ± 0.03</td>
</tr>
<tr>
<td>C</td>
<td>0.066 ± 0.012</td>
<td>0.045 ± 0.009</td>
<td>0.039 ± 0.024</td>
<td>0.011 ± 0.03</td>
<td>0.002 ± 0.02</td>
</tr>
<tr>
<td>Time 4</td>
<td>B</td>
<td>0.067 ± 0.016</td>
<td>0.053 ± 0.027</td>
<td>0.027 ± 0.006</td>
<td>0.009 ± 0.02</td>
</tr>
<tr>
<td>C</td>
<td>0.071 ± 0.014</td>
<td>0.048 ± 0.011</td>
<td>0.026 ± 0.004</td>
<td>0.011 ± 0.06</td>
<td>0.001 ± 0.01</td>
</tr>
<tr>
<td>Time 5</td>
<td>B</td>
<td>0.089 ± 0.084</td>
<td>0.047 ± 0.009</td>
<td>0.035 ± 0.032</td>
<td>0.016 ± 0.012</td>
</tr>
<tr>
<td>C</td>
<td>0.070 ± 0.017</td>
<td>0.044 ± 0.009</td>
<td>0.027 ± 0.012</td>
<td>0.011 ± 0.007</td>
<td>0.001 ± 0.01</td>
</tr>
</tbody>
</table>

Results

Mean amplitude values for the ML and AP COP for bins 1 through 5 across each time are displayed in Tables 1 and 2, respectively. The mean amplitude values for ML and AP COP for each treatment at each time across all bins are displayed in Tables 3 and 4.

The ML (F_{4,104} = 10.16, P = .081, 1-β = .524, η^2 = .075), and AP (F_{4,104} = 2.26, P = .067, 1-β = .644, η^2 = .080) mean amplitudes within the individual bins did not differ between the brace and control groups. This suggests that ankle-brace application did not significantly interfere with the proprioceptive control of posture during a 1-legged stance.

The mean ML (F_{4,108} = 495.8, P < .0005) and AP (F_{4,108} = 79.8, P < .0005) amplitudes were different among bins when collapsed over condition (brace and control). Mean ML and AP amplitudes were greater at lower frequency ranges than at higher frequency ranges at all 6 times (P < .05).

Discussion

Inspection of the components of the postural-control system may help to explain why no changes were seen in the frequency content of ML and AP COP over time with bracing. It is well known that 3 sensory systems (visual, vestibular, and proprioceptive) integrate and synthesize information to control posture. These sensory systems provide afferent input to the
central nervous system (CNS). Once the CNS receives these afferent impulses, it organizes the new information and produces an efferent response. This efferent response is what allows the appropriate postural adjustments to be made in order to maintain an upright stance.

Proprioceptive input from the ankle is received through various mechanoreceptors, including Golgi tendon organs, muscle spindles, and cutaneous receptors. The McDavid ankle brace used in this investigation may not have provided sufficient input to influence the proprioceptors at the ankle-foot complex and, therefore, would not be expected to alter the efferent signal produced by the CNS. If this is the case, then we would not anticipate changes in the mean frequency amplitude of ML or AP COP.

Previous work supports our results indicating that ankle-brace application does not interfere with the proprioceptive control of posture. The effects of 3 selected ankle appliances on postural control under different variations of a modified Romberg test were examined. An increase in COP patterns in the ML and AP directions with the eyes open was noted. When the sensory modalities were challenged, the COP values remained unchanged, which showed that bracing had no effect. These results also suggest that ankle bracing did not interfere with the coherence of the 3 sensory systems that manipulate and control posture.

An argument could also be made that no changes were seen in the ML and AP mean frequency amplitudes because our subject population was uninjured. Placing additional support on an ankle-foot complex that does not need to rely on assistance in order to maintain stability may not alter postural control. However, athletes suffering from chronic ankle instability, which is thought to arise from proprioceptive deficits, may be able to use the additional sensory input provided by the McDavid ankle brace, resulting in alteration of the frequency components of COP.

Before we completed this research, the effect of 4 days of ankle-brace application on one’s ability to maintain an upright stance had not been described in the literature. Most athletes use ankle braces over an extended time period in an effort to prevent ankle injury. The finding that ankle-brace application over a 4-day period did not affect postural control is of great clinical significance. Individuals with decreased postural control are believed to be more susceptible to ankle injury than those with finer postural control. If the application of an ankle brace decreased one’s ability to maintain an upright stance, the purpose of this device would be minimized. The fact that we did not see a decrease in postural control suggests continuous use of a McDavid ankle brace may not adversely affect sensory integration.

The only significant change we found existed within the individual bins at each time. The ML and AP mean frequency amplitudes between 0 and 0.39 Hz were greater than at 0.40 to 25 Hz. The ML and AP mean frequency amplitudes at 0.40 Hz to 0.88 Hz were greater than at 0.89 to 25 Hz and less than the ML and AP mean frequency amplitudes at 0 to 0.39 Hz, etc. This pattern of greater ML and AP mean frequency amplitudes at lower frequencies, decreasing as the frequency increases, is applicable at each time. This finding suggests that performing a 1-legged stance is a low-frequency activity.

A potential criticism of this study is that our statistical analysis revealed relatively low power values (ML 1-β = .524, AP 1-β = .644) when comparing the mean amplitudes of COP contained within each bin between treatment conditions. Therefore, it is possible that with additional subjects, significance would have been achieved, suggesting that the ankle brace did interfere with the proprioceptive control of posture. However, when taking into account the small effect sizes (ML $\eta^2 = .075$, AP $\eta^2 = .080$) accompanying the low power values, we question whether a statistically significant result in this case would be clinically significant.

The results of this study may not be applicable to a more dynamic or athletic movement (ie, a cutting maneuver). Data for this study were collected while subjects performed a 1-legged stance, which does not produce the same type of result as completing a more vigorous movement. Perhaps placing an external support on an ankle during athletic competition alters the proprioceptive control of posture.

**CONCLUSIONS**

We do not know if a change did not occur because the application of an ankle brace did not require a modification to...
be made in the efferent response sent by the central nervous system. The fact that no changes were seen could also indicate that uninjured ankles did not need the added support and, therefore, did not use the added sensory input. A study examining the effects of bracing on the spectral characteristics of postural sway on injured ankles may provide more insight on this issue. A change might have been seen if the study had been carried out over a longer period of time. Future researchers should investigate how ankle-brace application over a longer time period affects the spectral qualities of postural sway in order to more closely mimic actual usage of this device by athletes. Until further investigation is performed, the decision to use an ankle brace based upon its influence on proprioception may not be warranted.

REFERENCES
The Menstrual Cycle, Sex Hormones, and Anterior Cruciate Ligament Injury

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Objective: To determine if anterior cruciate ligament (ACL) injuries in female athletes occur randomly or correlate with a specific phase of the menstrual cycle.

Design and Setting: Female athletes who sustained ACL injuries reported the days of their menstrual cycles and provided saliva samples for sex-hormone determination. Salivary sex-hormone profiles were assessed to confirm the self-reported menstrual histories.

Subjects: A total of 38 female athletes (20 college, 15 high school, 1 middle school, 2 recreational) with recent ACL injuries participated in the study over a 3-year period.

Measurements: Athletes with recent ACL injuries completed a questionnaire defining the injury, the last menstrual cycle, prior knee injury, school, and type of birth control used (if any). Each subject provided a 30-cc saliva sample within 72 hours of injury. Saliva samples were placed into sealed containers and frozen at −20°C. We obtained 13 additional control samples from uninjured females to test the correlation between saliva and serum sex-hormone levels. Progesterone and estrogen were assayed by radioimmunoassay. Physical examination, magnetic resonance imaging, or surgery confirmed the injury in all subjects.

Results: The correlations between saliva and serum estrogen and progesterone were 0.73 (α = .01) and 0.72 (α = .01), respectively. Ten of 27 athletes who reported their cycle day at time of injury sustained an ACL injury immediately before or 1 to 2 days after the onset of menses. We rejected the null hypothesis that such high frequency was due to random chance.

Conclusions: A significantly greater number of ACL injuries occurred on days 1 and 2 of the menstrual cycle. Salivary sex-hormone levels correlated with the reported cycle day.

Key Words: estrogen, progesterone, remodeling, female athlete, athletic injury

Females injure their anterior cruciate ligaments (ACLs) more frequently than males participating in similar athletic activities. The cause of this sex discrepancy is likely multifactorial. In addition to their increased susceptibility to injury, women are at risk for increased knee laxity, ACL graft rupture, and other less successful outcomes after ACL reconstruction compared with males. Although other authors have shown that functional outcomes after ACL reconstruction may be similar in men and women, females’ knees have demonstrated significantly more laxity (measured as individual mean postoperative manual maximum differences) after both hamstring and bone-patellar tendon-bone reconstructions. One possible explanation for these observations is sex-specific differences in ligament remodeling.

Tissue remodeling occurs through a continuous cycle of protein synthesis and degradation. In this process, old or damaged structures are degraded and replaced with newly synthesized molecules. The balance between the degradative and biosynthetic arms of this process is controlled by the relative activities of matrix metalloproteinases and tissue inhibitors of metalloproteinases. The expression of some of these proteins is regulated by steroid hormones. For example, estrogen-dependent collagenase production and progesterone-dependent inhibition of collagenase have been observed in pig pubic ligaments. Additionally, increasing the concentration of estrogen in an ACL tissue-culture model resulted in decreased fibroblast and procollagen production. We, therefore, hypothesized that the type of hormone or the nature of exposure to it could affect the remodeling capabilities of the ACL and thereby alter its mechanical properties.

One prediction of this hypothesis is that ACL injury would be more likely at a certain time or times during the menstrual cycle. Although one group of investigators identified a trend toward an increase in injuries in the ovulatory phase and a decrease during the follicular phase, they did not conclusively identify a difference in injury rates as a function of the menstrual cycle. Other researchers have implicated different cycle phases for increased incidence of ACL injury. However, these studies were limited by their reliance on athletes’ histories provided to the medical staff regarding when the injuries occurred relative to the menstrual cycle. To overcome this limitation, serum, urine, or saliva could be examined to determine the sex-hormone profile and thereby verify the cycle day of the athletes at the time of injury. Salivary levels of sex hor-
mones determined with supersensitive assays correlate well with those measured in serum.\textsuperscript{33} Saliva samples are easy to obtain in an athletic setting because little advanced preparation and no equipment are required. Our objectives were to determine if menstrual histories provided at the time of ACL injury could be confirmed by measuring salivary estrogen (as estradiol \(E_2\)) and progesterone (P) levels and then to determine if ACL injuries occurred randomly or clustered in a specific phase of the menstrual cycle.

**METHODS**

Thirty-eight athletes with ACL injuries participated in the study. All study participants completed questionnaires defining their ACL injury, last menstrual period, prior knee injury, school, and type of birth control used (if any). Each athlete provided a 30-mL saliva sample within 72 hours of injury. Saliva samples were stored in a sealed container and frozen at \(-20^\circ C\). The samples were then shipped on dry ice to the Oregon Regional Primate Research Center (Beaverton, OR) for analysis. Physical examination, magnetic resonance imaging, or surgery confirmed the injury in all subjects.

One of the 38 athletes reported having a hysterectomy; her data were excluded due to inadequate information about hormone-replacement therapy. Consequently, study analyses included data from 37 patients: 21 provided both saliva samples and menstrual histories, 10 provided only saliva samples, and 6 provided only menstrual histories.

Because the previous literature indicated mixed results regarding the correlation between saliva and serum sex-hormone levels,\textsuperscript{23-28} we first performed an analysis to establish whether such a correlation existed in our sample. Thirteen control samples from uninjured females were obtained to test the correlation between saliva and serum sex-hormone levels. Salivary progesterone was assayed by routine radioimmunoassay, and \(E_2\) was assayed with a modification of the 3rd Generation Double Antibody Estradiol assay (Diagnostic System Laboratories, Webster, TX) at the Oregon Regional Primate Research Center.

To test our null hypothesis that the ACL injuries were not correlated with the athletes’ menstrual cycles, we performed a Monte Carlo simulation to generate multiple pseudocontrol groups. If the ACL injuries were not correlated with the menstrual cycle, the injuries would occur randomly throughout the cycle. That is, the probability of injury would follow a uniform distribution over the menstrual cycle. Using Stata computer software (version 6, Stata Corp, College Station, TX), we simulated 50 hypothetical control groups, each with 100 subjects. The timing of an ACL injury assigned to each hypothetical subject was determined by a uniform distribution to ensure that the probability of injury in each 2-day interval was the same for all intervals. We then compared the injured group with each computer-generated group. We tested whether the probability of injury was the same for each 2-day interval between the injured athletes’ group and a group of 100 hypothetical subjects.

**RESULTS**

The correlations between saliva and serum estrogen and progesterone were 0.73 (\(\alpha = .01\)) and 0.72 (\(\alpha = .01\)), respectively. The correlation between the self-reported last menstrual period at the time of injury and the actual salivary and progesterone levels was 95% for the 21 athletes who provided this information (Table). Because salivary sex-hormone levels confirmed self-reported menstrual history in all but one case, we were able to use the measured sex-hormone levels to place athletes who did not provide menstrual histories in the appropriate phase of the menstrual cycle at the time of injury. Among all 37 athletes for whom data were analyzed, 25 injured their ACLs during the follicular phase, 1 during the ovulatory phase, and 11 during the luteal phase of the menstrual cycle. Six athletes injured their ACLs while on oral contraceptives, of whom 5 sustained their injury during the follicular phase and 1 during the luteal phase. Only 1 of the athletes on oral contraceptives reported her cycle day at the time of injury (day 2 of menses). Interestingly, the \(E_2\) and P concentrations were very low in this athlete (\(E_2 = 0.73\) pg/mL, \(P = 58\) pg/mL) and 3 others on oral contraceptives (\(E_2\) range, 0.16–0.20 pg/mL; \(P\) range, 15–29 pg/mL), suggesting that they were all injured around the time of menses.

Among the 27 athletes who self-reported their menstrual histories, 10 sustained injuries during the few days before and the first 2 days after the onset of menses (Figure 1). The frequency of observed injury for days 1–2 of the menstrual cycle was significant at \(\alpha = .05\) for all 50 comparisons between the injured athletes and our computer-simulated subject groups. This implies that the high frequency of the ACL injury observed in this interval was not due to random chance. For days 25–26, 27–28, and 7–8, the athletes’ incidence of ACL injury was lower than the probability determined by a uniform distribution at \(\alpha = .10\) in 33, 34, and 36 comparisons, respectively, out of 50 total simulations. For the other intervals, fewer than 10 comparisons out of 50 were significant at \(\alpha = .10\).
of Injuries

Number of injuries for days 1 and 2 of the menstrual cycle (a = .05) for all 50 comparisons between the injured athletes and our computer-simulated subjects.

Using a Monte Carlo simulation to define a control group, we found a significant difference in the frequency of observed injury for days 1 and 2 of the menstrual cycle (α = .05) for all 50 comparisons between the injured athletes and our computer-simulated subjects.

We chose to use computer-simulated subjects because there was no well-defined control group. To qualify to be in a legitimate experimental control group, subjects would have needed the same menstrual cycles as the injured females but different distributions of injury among the cycles’ phases. Therefore, instead of performing an experimental-control group comparison, we tested a simpler null hypothesis that the ACL injuries occurred randomly in each day of the menstrual cycle. Each computer-generated subject had an equal chance (0.0357) of injury in each day of her menstrual cycle. That is, these subjects had the same menstrual cycles as the injured subjects, but the probability of injury was different. We rejected our null hypothesis and found that ACL injuries occurred most frequently during the early menstrual cycle.

Past studies of the correlation between salivary and serum sex-hormone measurement have yielded conflicting results.24-28 However, more recent supersensitive, double-antibody techniques for measuring sex hormones in saliva have shown good correlation between saliva and serum levels.23 One group has even recommended using saliva to obtain hormone profiles in patients with difficult venous access.24 Using saliva to obtain sex-hormone profiles of athletes fits well in the athletic arena because little planning or equipment is needed to obtain and store the saliva. A simple ziplock-type bag works well to hold the saliva and can be placed immediately on ice and transferred to a freezer soon thereafter or upon return from a road trip.

Our findings depend heavily on the accuracy with which we determined the day of the menstrual cycle at the time of injury. Such determinations are complicated by problems in obtaining blood from injured athletes in an athletic setting and the fact that a single measurement of one hormone cannot unequivocally define the day of the menstrual cycle. However, we successfully overcame these problems by measuring both E2 and P in saliva. This approach is effective because elevated (at or near the typical highest concentration) E2 is characteristic of the follicular phase (with an E2 spike occurring at ovulation), elevated P is characteristic of the luteal phase, and low E2 and P are characteristic of menses. Menstrual phases defined in this way correlated well (≥95%) with the self-reported menstrual histories (see Table), thereby confirming the accuracy of our athletes’ recollections of their menstrual cycles. We, therefore, have confidence that all but 1 or 2 of our athletes accurately recalled the dates of their last menstrual periods before ACL injury.

Although we do not know why ACL injuries occur around the time of menses, our current research is focused on characterizing sex differences in ACL tissue remodeling. Cyclic changes in E2 and P may alter expression of genes encoding tissue-remodeling enzymes and proteins, which, in turn, could favor either net tissue degradation or repair at specific times during the menstrual cycle. If a molecular basis for sex dif-

indicating that during these intervals, ACL injuries were likely to be uncorrelated with the menstrual phases.

Therefore, a significantly greater number of ACL injuries occurred on days 1 and 2 of the menstrual cycle. Salivary sex-hormone levels correlated with self-reported cycle day.

**DISCUSSION**

This is the first study to confirm self-reported menstrual histories with salivary sex-hormone profiles at the time of ACL injury. We found that 26 of 37 athletes tore their ACLs during the follicular phase of the menstrual cycle. Among athletes who self-reported their menstrual histories, 10 of these 27 injuries occurred during the few days before and the 2 days after the onset of menses (Figure 1). The levels of E2 and P are both low at this time (Figure 2). This hormonal condition contrasts with the follicular phase during which P is low and E2 peaks sharply before ovulation, and it follows the midluteal phase during which E2 and P are both elevated for several days. These results are consistent with a report of no correlation between ACL injury and the general category of “luteal” phase, as well as 2 other reports indicating that injury is more likely during the late luteal and early follicular phases of the cycle.21,22

We did not obtain information on the typical lengths of the athletes’ menstrual cycles. Although the “normal” menstrual cycle lasts 28 days, 3 of our 37 athletes (8% of our sample) sustained injuries after day 28. The probability that ACL injuries occurred during the prolonged menstrual interval (>28 days) was no different from the probability determined by a uniform distribution. That is, we could not reject the null hypothesis that the injuries occurring during this time were due to random chance.

One group has even recommended using saliva to obtain hormone profiles in patients with difficult venous access. Using saliva to obtain sex-hormone profiles of athletes fits well in the athletic arena because little planning or equipment is needed to obtain and store the saliva. A simple ziplock-type bag works well to hold the saliva and can be placed immediately on ice and transferred to a freezer soon thereafter or upon return from a road trip.

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ferences in ACL injury is found, treatments may be instituted to decrease the injury rates in females.

In conclusion, ACL injuries occurred most frequently on days 1 and 2 of menses, suggesting that ACL injury is not random but occurs more often around the time of menses, when circulating sex-hormone levels are low and after a time when both E2 and P were elevated. Additionally, salivary sex-hormone profiling correlates well with serum profiling, and in this athletic population, adequately identified menstrual cycle phase at the time of injury. This is, therefore, an effective technique to identify correlations between injury and hormone patterns in athletes.

ACKNOWLEDGMENTS

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REFERENCES


COMMENTARY

Susan E. Kirk

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It is well accepted that females involved in athletic activities have a substantially greater risk of sustaining anterior cruciate ligament (ACL) injuries compared with their male counterparts. With the continued growth of women's sports, such an injury no longer brings merely the worry of the end of a scholastic career but also the potential loss of scholarship or professional income. Most investigators have suspected that the
reason for sexual disparity in the incidence of ACL injury is multifactorial; however, several recent studies have attempted to explore the link between one obvious difference between men and women: sex steroids. In this issue of the Journal of Athletic Training, Slauterbeck et al have attempted to link the frequency of ACL injury to menstrual cycle phase and have used the novel method of salivary estrogen (estradiol) and progesterone measurements to attempt to confirm subjects’ self-reported cycle phases at the time of injury.

The authors note that a flaw in the design of previously reported studies has been the lack of hormonal confirmation of menstrual phase. Their study design attempts to correct this problem in a noninvasive manner by using salivary assays for the major female sex steroids, estradiol and progesterone. Those involved in the care or training of athletes can appreciate the difficulty in trying to obtain serum samples at the time of injury in a reliable and convenient manner. Slauterbeck et al have shown a moderate correlation between the results of their salivary and serum assays in a separate group of subjects. However, with this method, they have not been able to overcome the difficulty of assigning women to a particular phase of the menstrual cycle with a single sample.

Although the pattern of menses is typically consistent from cycle to cycle, with a preovulatory surge of estrogen in the late follicular phase and then a second, more gradual increase in both estrogen and progesterone during the luteal phase, the overlap of hormone levels is such that one cannot determine the day of the cycle by a single measurement. Review of Figure 2 in the article by Slauterbeck et al demonstrates that an equally elevated level of estrogen can be seen in both the late follicular and the midluteal phases. The authors have attempted to overcome this ambiguity by using paired samples of estrogen and progesterone, but as their figure demonstrates, midrange estrogen and progesterone are seen both immediately after the ovulatory surge and in both the early and the late luteal phases. In addition, the authors allowed the sex steroid samples to be collected up to 72 hours postinjury. Figure 2 demonstrates a substantial change (either increase or decrease) in hormone levels over this time at most phases of the cycle. It is quite possible that the 72-hour window allowed collection of samples just after ovulation had occurred, a period when levels of both estrogen and progesterone are similar to those seen in the early follicular phase.

An additional complicating factor is the considerable variability among women, as well as in individual women from cycle to cycle, with regard to both cycle length and hormonal peaks. For the purpose of data analysis, the authors have assumed that a normal cycle is 28 days, but most references cite a range of 24 to 32 days. Moreover, only 13% of normal women have cycles that are consistently within this range over 1 year. Therefore, one cannot accurately assign a woman to an exact position in the menstrual cycle on the basis of a single hormonal measurement.

The authors relied on subjects to identify which phase of the cycle they believed themselves to be in at the time their injury occurred. Obstetricians know how unreliable the self-reported date of the last menstrual period is when attempting to determine gestational age. This area may also lead to incorrect interpretation of results. A strict description of the beginning of menses is required to eliminate subject error in dating the onset of menses. A typically used and reliable method is the date when feminine protection (sanitary napkins or tampons) is required. However, even with more rigid guidelines, self-reporting of menstrual history has been shown to be an unreliable measure of cycle parameters. In this study, 27% of the subjects were not able to provide any menstrual history at all.

In vivo and in vitro models have demonstrated that estrogen does affect fibroblast and procollagen production and subsequent tissue remodeling, but it is risky to interpret the results obtained in different species and with different ligaments and then apply them to normal women injured during an athletic activity. Therefore, the exact impact of the sex steroids on ACL injury remains an unanswered question, as the authors acknowledge. Salivary assays are a potentially useful tool for convenient and noninvasive collection of samples that will allow a more complete investigation of this area. However, in addition to the large volume of saliva required per sample for this assay (30 mL), not all investigators have found this method to demonstrate acceptable reliability. Therefore, it is important that other methods be developed to allow the convenient and noninvasive collection of hormonal specimens, potentially immediately at the time of injury or more frequently during 1 or several menstrual cycles. An additional method using capillary blood obtained with a lancet device shows promise. More studies must be performed in valid populations to confirm both methods as acceptable replacements for serum estradiol and progesterone measurements. Only then will we begin to gain a more complete understanding of the role of female hormones in injuries sustained during athletic performance.

REFERENCES


AUTHORS’ RESPONSE

Dr Kirk is absolutely correct that similarities in hormone levels at different phases of the menstrual cycle make it difficult to define unequivocally the phase of the cycle from measurements on a single sample. Fortunately, that was not the major intent of our hormone measurements. Single measurements can be used to disprove inaccurate recollections of menstrual history. For example, if a patient reports that she is on day 5 (follicular phase) of her cycle but has an elevated level of progesterone indicative of the luteal phase, the single hormone measurement would disprove the recalled menstrual history. Accordingly, in our study the hormone measurements were not used as the sole indicator of menstrual phase but to verify reported menstrual histories. Our hormone data conflict-
ed with the recalled history for only 1 patient, supporting the view that the menstrual histories our patients reported were largely accurate.

We believe that, as athletes, this patient population may be more aware of their physical state than others. In the example of obstetric patients Dr Kirk cited, the patients were being asked to recall a date that was 4 to 8 weeks (or more) in the past. It is easy to imagine that such recollections would be less accurate than those of athletes who menstruated perhaps only days, or at most 4 weeks, before injury. Indeed, the patients we identified as being most at risk were actually menstruating at the time of injury.

We agree with Dr Kirk that there is room for improvement in the methods used to assess hormone concentrations in an athletic setting. However, the ultrasensitive radioimmunoassay can accurately determine hormone levels in saliva specimens, such as those our athletes provided. Thus, we believe such measurements are sufficiently reliable, when used in combination with self-reported menstrual history from injured athletes, to support the conclusions of this study.
A Survey of Physical Activity Levels of Certified Athletic Trainers

Marchell Cuppett*; Richard W. Latin†

*University of South Florida, Tampa, FL; †University of Nebraska at Omaha, Omaha, NE

Objective: To determine the self-reported physical activities of certified athletic trainers (ATCs), both at work and at leisure.

Design and Setting: We used the Baecke Questionnaire of Habitual Physical Activity and also asked for demographic information, including employment setting, years of experience, education level, and position.

Subjects: The questionnaire was sent to 1200 randomly selected ATCs in the Mid-America Athletic Trainers’ Association; the return rate was 53%.

Measurements: We used means, standard deviations, and ranges to describe the age, total fitness index, work, and leisure and sport indexes of men and women subjects. Independent t tests were used to compare the mean total activity index between men and women within this study and with previous studies. We examined differences in activity indexes by employment setting, position, and age with one-way analysis of variance and Fisher pairwise comparison tests. Two-way χ² analysis was used to determine the relationship between activity level and employment setting and position. Statistical significance was set at P = .05 for all analyses.

Results: Certified athletic trainers who work in a clinical setting had the highest mean total activity score at 9.1 points. Clinic ATCs scored significantly higher than high school ATCs and college ATCs. When compared by position, there were no significant differences among the mean total activity indexes; however, the mean work index of program directors was significantly lower than all other positions and the mean work index of high school and clinic ATCs was significantly higher than all other employment settings.

Conclusions: Female ATCs scored significantly higher in total activity levels on the Baecke Questionnaire than their male counterparts. This is in contrast to the general population, investigated by other authors, in which men scored significantly higher than women on the same scale. Additionally, we compared the total activity levels by age, position, and employment setting. There was a significant difference by position only in the work index. The mean total index activity of the over-36-years-old group was significantly lower than all other age categories. There was no significant difference in mean total activity levels by employment setting.

Key Words: athletic trainer, energy expenditure, physical fitness, Baecke Questionnaire

Certified athletic trainers (ATCs) are the primary health care providers for the physically active. The advice that athletic trainers give to athletes may reflect their own health and fitness beliefs. The ATC should be a role model and proponent for physical activity for the athlete; however, there has been no research to date on ATCs’ personal physical fitness or activity levels.

Physical activity may be broadly divided into 2 categories: physical activity in the work setting and leisure-time physical activity. Both categories have been measured among the general population with questionnaires that have been validated through direct measurement and task analysis. A certain level of physical fitness is necessary in many professions, especially those that require the professional to react in emergency situations, such as firefighters and police officers. Certified athletic trainers could be considered among those professionals needing some level of physical fitness to respond to emergencies. Other physical demands of these professions are lifting, standing for extended periods of time, and the stamina to work many hours. Physical fitness is an important factor in tolerance of long shifts in similar occupations. As ATCs are expected to work elongated and irregular days, physical fitness may be an important factor in work tolerance.

The other component of physical activity is leisure-time activity. Leisure-time activity has received the most emphasis of the physical activity components in recent years due to the increased awareness that leisure activity is highly associated with a healthy lifestyle. With the increasing reliance on technology that results in a general decrease in the physical demands of work, physical activity becomes an important component of a healthy lifestyle, especially for the person who does not have a physically demanding occupation. Higher levels of leisure-time physical activity correlate with decreases in body fat, resting heart rate, and blood pressure and decreased incidence of stroke and coronary heart disease.

A variety of methods have been used to evaluate physical activity levels at work and in leisure. Some of these methods include task analysis, job classification, activity diaries, pedometers, accelerometers, and questionnaires.

Use of all of these except for questionnaires may be time consuming or expensive. Physical activity questionnaires have been used in numerous studies and are closely related to the results obtained by the more time-intensive and expensive...
methods. A variety of physical activity questionnaires are available, many of which only identify leisure physical activity or work activity. The Baecke Questionnaire of Habitual Physical Activity includes measures for work activities, sport activities, and leisure activities.10

Several studies have used questionnaires to measure the physical activities of the general population; however, information about the physical activities of ATCs at work or during leisure is very limited. Therefore, our purpose was to determine the self-reported physical activities of ATCs.

METHODS

Instrument

We used the Baecke Questionnaire of Habitual Physical Activity. This questionnaire has been validated in several investigations6-9 and has been shown to be an easily administered and accurate instrument. When correlated with physical activity scores, validity coefficients ranged from \( r = .33 \) to \( r = .59 \) for men and women between the ages of 20 and 59 years. The questionnaire consists of 3 sections: work, sport (exercise), and nonsport leisure activity. Most of the questionnaire is scored on a 5-point Likert scale, with descriptors ranging from never to sometimes or very often. Three additional questions required reporting the type of sporting (exercise) activity and both the number of hours per week and the number of months per year in which the respondent participated in that activity. The original questionnaire was written for Europeans. Our only modification for this study was a slight change of wording so that the questionnaire would be more applicable to Americans in that “sport” was changed to “exercise activity.” In addition, questions including demographic information pertinent to ATCs, such as employment setting, years of experience, education level, and position, were added but did not affect the original questionnaire.

The scoring of the questionnaire included specific scoring criteria for each of the 3 sections: work, sport, and leisure indexes. Each section could receive a maximum score of 5 points, with a maximum of 15 points for the total activity index. Each index was rounded to the nearest tenth of a point.

Data Collection

Approval was received from the University of Nebraska Institutional Review Board before beginning this research. The questionnaire and a cover letter explaining the study were sent to 1200 randomly selected ATCs within District 5, the Mid-America Athletic Trainers’ Association (MAATA) of the National Athletic Trainers’ Association. Address labels were obtained from the National Athletic Trainers’ Association for all athletic trainers living in District 5 (\( n = 1530 \)). Systematic counting was used to select 1200 potential subjects. Subjects were asked to complete and return the questionnaire in a business reply envelope within 3 weeks. Completion of the questionnaire implied informed consent.

DATA ANALYSIS

We computed means, standard deviations, and ranges to describe the age, total fitness index, and work, leisure, and sport indexes of male and female subjects. An independent \( t \) test was used to compare the mean total activity index between men and women. One-way analysis of variance and Fisher pairwise comparison tests were calculated to examine differences in activity indexes by employment setting, position, and age. Frequency counts and 2-way \( \chi^2 \) analyses were used to examine the relationship between activity level and employment setting and activity level and position. Activity level categories were established with percentile ranks, with scores above \( P_{75} \) representing the highest level and those below \( P_{25} \), the lowest. Statistical significance was set at \( P = .05 \) for all analyses.

RESULTS

There were 636 respondents (372 men and 264 women) to the survey for a 53% return rate. Of the 636 respondents, 534 (84%) indicated that they were physically active outside of work, while 102 (16%) indicated that they were not. Table 1 shows a comparison using an independent \( t \) test between male and female ATCs. Women were significantly more physically active, as indicated by a higher total activity index.

One-way analysis of variance and Fisher pairwise comparison tests were used to examine differences in activity indexes by employment setting, position, and age as seen in Tables 2-4. Certified athletic trainers employed in the clinic were more physically active than high school ATCs and college ATCs (Table 2). The clinic ATCs were more active in sport activity than the high school ATCs, and they are more active at work than the college ATCs1 or those employed in other settings.

We found no significant differences among mean total activity indexes by position (Table 3); however, the mean work index of program directors was significantly lower by a mean of 0.2 points than all other positions.

There were no significant differences among the mean total activity indexes by age group (Table 4); however, ATCs over the age of 36 years were significantly less active than all other age categories at work.

Frequency counts for activity levels by position appear in Table 5. Activity levels were determined by quartiles, with scores above \( P_{75} \) being the highest and those below \( P_{25} \), the

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
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<td>Age, y</td>
<td>33.9</td>
<td>6.6</td>
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<td>6.8</td>
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<td>2.0-3.6</td>
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<td>0.3</td>
<td>2.1-4.1</td>
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<td>Leisure activity index</td>
<td>2.6</td>
<td>0.6</td>
<td>1.3-4.5</td>
<td>2.8</td>
<td>0.6</td>
<td>1.3-4.5</td>
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<tr>
<td>Total activity index</td>
<td>8.8*</td>
<td>1.2</td>
<td>5.8-12.1</td>
<td>9.0*</td>
<td>1.2</td>
<td>5.6-13.1</td>
</tr>
</tbody>
</table>

*Means are significantly different, \( P = .05 \). Note: Activity indexes are rounded to 0.1.
We surveyed ATCs in the Midwest and found that 16% of
the respondents were not physically active. Previous research­ers have reported that 9% to 27% of the general population
was not physically active.\textsuperscript{11-13}

The difference in the total activity index between men and
women is in contrast to data from the general population re­ported in previous studies. Schramm et al\textsuperscript{14} studied the rela­tionship between physical activity and flexibility in 64 women.
Using the same instrument we used, Schramm et al reported
a mean total activity index of 8.1. This was significantly dif­ferent ($t = 4.91, P < .05$) from the mean total activity index
of female ATCs in our study.

Differences were also noted in the mean total activity index
between ATCs in our study and a previous study performed
by Richardson et al.\textsuperscript{2} In the latter study, the Atherosclerosis
Risk in Communities/Baecke Questionnaire was administered
to 50 women with a reported mean total activity index of 8.5.
This is a difference in the total activity index of 0.5 when
compared with female ATCs. No differences in the mean total
activity index among men were noted among the Schramm et

Table 2. Activity Indexes by Employment Setting

<table>
<thead>
<tr>
<th>Variable</th>
<th>High School (n = 99)</th>
<th>Clinic (n = 247)</th>
<th>College (n = 219)</th>
<th>Other (n = 71)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Sport activity index</td>
<td>(3.1^a)</td>
<td>0.7</td>
<td>(3.3^b)</td>
<td>0.7</td>
</tr>
<tr>
<td>Work activity index</td>
<td>(3.0^a)</td>
<td>0.3</td>
<td>(3.0^a)</td>
<td>0.3</td>
</tr>
<tr>
<td>Leisure activity index</td>
<td>2.6</td>
<td>0.6</td>
<td>2.7</td>
<td>0.6</td>
</tr>
<tr>
<td>Total activity index</td>
<td>8.7(^{+})</td>
<td>1.1</td>
<td>9.1(^{+})</td>
<td>1.2</td>
</tr>
</tbody>
</table>

Means with the same superscript are significantly different, $P \leq .05$. Activity indexes are rounded to 0.1.

Table 3. Activity Indexes by Position

<table>
<thead>
<tr>
<th>Variable</th>
<th>Head (n = 227)</th>
<th>Assistant (n = 85)</th>
<th>Program Director (n = 47)</th>
<th>Graduate Assistant (n = 28)</th>
<th>Other (n = 249)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Sport activity index</td>
<td>3.1</td>
<td>0.7</td>
<td>3.3</td>
<td>0.7</td>
<td>3.4</td>
</tr>
<tr>
<td>Work activity index</td>
<td>3.0(^a)</td>
<td>0.3</td>
<td>3.0(^a)</td>
<td>0.3</td>
<td>2.8(^{+c,d})</td>
</tr>
<tr>
<td>Leisure activity index</td>
<td>2.6</td>
<td>0.6</td>
<td>2.7</td>
<td>0.6</td>
<td>2.7</td>
</tr>
<tr>
<td>Total activity index</td>
<td>8.8(^{+})</td>
<td>1.2</td>
<td>9.0(^{+})</td>
<td>1.2</td>
<td>8.9(^{+})</td>
</tr>
</tbody>
</table>

Means with the same superscript are significantly different, $P \leq .05$. Activity indexes are rounded to 0.1.

Table 4. Activity Indexes by Age

<table>
<thead>
<tr>
<th>Variable</th>
<th>&gt;36.0 Years (n = 177)*</th>
<th>30.0 – 36.0 Years (n = 188)</th>
<th>27.0 – 29.9 Years (n = 123)</th>
<th>&lt;27.0 Years (n = 142)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Sport activity index</td>
<td>3.3(^a)</td>
<td>0.7</td>
<td>3.2</td>
<td>0.7</td>
</tr>
<tr>
<td>Work activity index</td>
<td>2.9(^{+c,d})</td>
<td>0.3</td>
<td>3.0(^a)</td>
<td>0.3</td>
</tr>
<tr>
<td>Leisure activity index</td>
<td>2.6</td>
<td>0.6</td>
<td>2.6</td>
<td>0.6</td>
</tr>
<tr>
<td>Total activity index</td>
<td>8.8(^{+})</td>
<td>1.3</td>
<td>8.8(^{+})</td>
<td>1.1</td>
</tr>
</tbody>
</table>

Means with the same superscript are significantly different, $P \leq .05$. Activity indexes are rounded to 0.1.

*Six subjects did not identify their ages.

Table 5. Frequency Counts of Activity Levels by Position*

<table>
<thead>
<tr>
<th>Level</th>
<th>Head</th>
<th>Assistant</th>
<th>Program Director</th>
<th>Graduate Assistant</th>
<th>Other</th>
<th>Total</th>
<th>Percentage of Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;P(_{75})</td>
<td>50</td>
<td>24</td>
<td>12</td>
<td>9</td>
<td>62</td>
<td>157</td>
<td>24.7</td>
</tr>
<tr>
<td>P(<em>{75}) – P(</em>{90})</td>
<td>46</td>
<td>19</td>
<td>10</td>
<td>10</td>
<td>54</td>
<td>139</td>
<td>21.8</td>
</tr>
<tr>
<td>P(<em>{90}) – P(</em>{95})</td>
<td>56</td>
<td>16</td>
<td>12</td>
<td>2</td>
<td>69</td>
<td>155</td>
<td>24.4</td>
</tr>
<tr>
<td>&lt;P(_{95})</td>
<td>75</td>
<td>26</td>
<td>13</td>
<td>7</td>
<td>64</td>
<td>185</td>
<td>29.0</td>
</tr>
<tr>
<td>Total</td>
<td>227</td>
<td>85</td>
<td>47</td>
<td>28</td>
<td>249</td>
<td>636</td>
<td></td>
</tr>
</tbody>
</table>

*Chi square test, $P > .05$. 

lowest. A 2-way $\chi^2$ analysis showed there was no significant
relationship between the type of position held and the number
of subjects in each fitness quartile. This finding suggests that
within each position, the fitness levels of ATCs were similarly
distributed. Table 6 contains the frequency counts of activity
levels by employment setting. A 2-way $\chi^2$ analysis revealed
no significant relationship between employment setting and
the number of subjects in each fitness quartile. This too, suggests
comparable distribution within the employment settings.

DISCUSSION

We surveyed ATCs in the Midwest and found that 16% of
the respondents were not physically active. Previous research­ers have reported that 9% to 27% of the general population
was not physically active.\textsuperscript{11-13}
Table 6. Frequency Counts of Activity Levels by Employment Setting

<table>
<thead>
<tr>
<th>Level</th>
<th>High School</th>
<th>Clinic</th>
<th>College</th>
<th>Other</th>
<th>Total</th>
<th>Percent of Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; P₉₀</td>
<td>19</td>
<td>67</td>
<td>56</td>
<td>15</td>
<td>157</td>
<td>24.7</td>
</tr>
<tr>
<td>P₉₀ - P₇₅</td>
<td>22</td>
<td>60</td>
<td>43</td>
<td>14</td>
<td>139</td>
<td>21.9</td>
</tr>
<tr>
<td>&lt; P₇₅</td>
<td>26</td>
<td>66</td>
<td>45</td>
<td>18</td>
<td>155</td>
<td>24.4</td>
</tr>
<tr>
<td>Total</td>
<td>99</td>
<td>247</td>
<td>219</td>
<td>71</td>
<td>636</td>
<td></td>
</tr>
</tbody>
</table>

*Chi square test, P > .05.

Al and Richardson et al studies and our study, Baecke et al and Misigoj-Durakovic et al reported that the women's leisure index was significantly higher than that of their male subjects, while Jacobs et al found no difference. Previous investigators have reported men scoring significantly higher on the sport indexes than women, while in our study there was no significant difference. We speculate this may be due, in part, to the women who choose to go into the profession of athletic training.

Age was also a factor in the work activity index differences. The over-36-years-old category had a significantly lower work activity index than the other age categories. A possible explanation may be that older ATCs were in a position for a greater length of time and had advanced to more supervisory or administrative positions. With increasing numbers of administrative tasks, the older ATC may no longer perform as many of the more physically taxing athletic training tasks on a daily basis.

Employment Setting and Position

The mean total activity index between ATCs working in the clinic setting (9.1) was significantly different than their counterparts in the high school (8.7) and college settings (8.8). Significant differences were also found between clinic and high school ATCs in the sport activity index (3.2 and 3.1, respectively). These data suggest that clinic ATCs reported more activity in sport than high school ATCs. This may be due to clinic ATCs having more control over their schedules to allow participation in a regular exercise routine.

High school and clinic ATCs had the highest work activity index (3.0). This was significantly higher than ATCs working in the college or other setting. These data suggest that high school and clinic ATCs perceive their jobs as being more physically demanding than ATCs in other settings. As many clinic ATCs are also contracted to high schools, it makes sense that these 2 work indexes would be similar. The difference between high school and clinic ATCs and other ATCs may be due in part to the high school or clinic ATC often being the only person involved in the day-to-day care of all of the sports at the high school. The college ATCs often have more students or assistants to help with the physical aspect of practices and game preparations.

When examined by position (head athletic trainer, assistant athletic trainer, program director, graduate assistant, or other), the program directors had a significantly lower work index than all other positions. This is most likely because most program director positions involve more sedentary skills and less activity than a typical athletic training position. There was, however, no significant difference in the total fitness index among positions.

The concept that physical activity and physical fitness are contributors to good health has substantial documentation. Work-related and recreational physical activity are associated with decreased risks for coronary artery disease and stroke. Further, Cox and Montgomery found that a high level of fitness may be associated with decreased absenteeism and increased job performance.

Certified athletic trainers are proponents of physical fitness for the athlete. One would expect ATCs to have knowledge of the benefits of personal physical fitness and to lead by example. However, for males in this study, the total activity index was no higher than that for the general population, indicating that even though ATCs understand physical fitness, their compliance was not any better than the general population. Boritz reported that 93% (n = 126) of physicians surveyed were regular exercisers, while only 84% of the respondents in our study indicated that they were physically active outside of work.

The female ATCs in this study, however, scored higher in total activity than the women in the general population surveyed in previous studies. This may be due to the personal and physical characteristics of the women who pursue athletic training as a profession or to a concerted effort on the part of the female ATCs in this study to participate in physical activities.

Most of the significant differences we found were relatively small in magnitude. This fact should be kept in mind when interpreting the results of this research.

Both the National Athletic Trainers’ Association Board of Certification Role Delineation Study and the National Athletic Trainers’ Association Educational Competencies include knowledge of the benefits of physical activity and the design of regular exercise programs as entry-level skills that all ATCs should possess. Why is it, then, that ATCs do not score any better than the regular population on total activity levels?

Numerous reasons not measured in this study may account for the less-than-desirable physical activity scores of the ATCs in this study. Certified athletic trainers tend to work extended hours and are often not in control of their time due to the schedule of games and practices. It may be difficult for many ATCs to find time to exercise routinely. Added responsibilities, such as team travel and home, family, and teaching responsibilities may make regular exercise habits difficult to maintain.

CONCLUSIONS

Our purpose was to determine the self-reported physical activities of ATCs, both at work and at leisure and sport. We compared the total activity levels by sex, employment setting, position, and age. There was a mean difference by position only in the work index. The mean total index activity of the over-36-years-old group was significantly lower than all other age categories. Athletic trainers in the clinic setting had a mean total activity index significantly greater than either the high school or college ATC.

We also found that female ATCs scored significantly higher in total activity levels on the Baecke Questionnaire than their male counterparts. This is in contrast to the findings in the general population, investigated by other authors, which in-
dicated that men scored significantly higher than women on the same scale.

The results of this study suggest several areas for further investigation. Because this study was limited to the Midwest, it would be advantageous to replicate this study nationwide to determine whether there is a difference in the activity level of ATCs. Second, more information needs to be obtained on the actual energy expenditure for ATCs within various settings. Third, further investigation into the perceived barriers to regular exercise for the ATC is needed. Finally, studies including other health habits of ATCs and other health care professionals are beneficial.

ACKNOWLEDGMENTS

This study was made possible through a grant from the Mid-America Athletic Trainers’ Association. Special acknowledgement goes to Lisa Schniepp, ATC, and the athletic training students of the University of Nebraska at Omaha for their assistance in the study.

REFERENCES

The Professional Socialization of Certified Athletic Trainers in High School Settings: A Grounded Theory Investigation

William A. Pitney

Northern Illinois University, Dekalb, IL

Objective: To gain an understanding of the professional socialization experiences of certified athletic trainers (ATCs) working in the high school setting.

Design and Setting: A qualitative investigation using a grounded theory approach was conducted to explore the experiences related to how ATCs learned their professional role in the high school setting.

Participants: A total of 15 individuals (12 ATCs currently practicing at the high school level, 2 current high school athletic directors who are also ATCs, and 1 former high school ATC) participated in the study. The average number of years in their current position for the 12 currently practicing ATCs was 10.16 ± 7.44, with a range of 2 to 25 years. The 2 athletic directors averaged 5.5 years of experience in their roles, and the former high school athletic trainer had worked in that setting for 1 academic year.

Data Analysis: The interviews were transcribed and then analyzed using open, axial, and selective coding. Peer debriefing, member checks, and triangulation were used to establish the trustworthiness of the study.

Results: Informal learning processes were discovered as the overarching theme. This overarching theme was constructed from 2 thematic categories that emerged from the investigation: (1) an informal induction process: aspects of organizational learning, and (2) creating networks for learning.

Conclusions: Informal learning is critical to the professional socialization process of ATCs working in the high school setting. Because informal learning hinges on self-direction, self-evaluation, reflection, and critical thinking, the findings of this study indicate that both preservice and continuing education should attempt to foster and enhance these qualities.

Key Words: informal role induction, learning networks, organizational socialization, self-evaluation, reflection, critical thinking

Professional socialization is a process by which individuals learn the knowledge, skills, values, roles, and attitudes associated with their professional responsibilities. Socialization is considered to be a key component of professional preparation and continued development in health and allied medical disciplines and has been investigated in medical education, nursing, occupational therapy, and physical therapy.

Professional socialization is typically exemplified as a 2-part developmental process that includes experiences before entering a work setting (anticipatory socialization) and experiences after entering a work setting (organizational socialization). The first process, anticipatory socialization, refers to experiences such as one's formal training as an undergraduate or graduate student, background as an employee in another setting such as an Emergency Medical Technician, or prior experience as a volunteer with an organization such as the American Red Cross. Organizational socialization refers to experiences such as in-service education and mentoring. The organizational socialization phase of professional socialization can be divided into 2 parts: (1) a period of induction, and (2) role continuance. Induction experiences take many forms. For example, induction processes can be either very formalized (ie, requiring employees to attend specific orientations or instructional sessions) or very informal (ie, no orientation). Additionally, induction processes may be either sequential, requiring specific skills to be learned at specific times during the initial periods of a job, or random, having no time frame for the development of various skills within the organization. Role continuance, on the other hand, focuses on adjusting to the organizational demands over time and continually learning the nuances of a given role and developing professionally. While athletic training has given a great deal of attention to the anticipatory socialization by way of the professional preparation process, there is a paucity of research related to organizational socialization.

Organizational socialization relates to how individuals adapt to their new roles and learn about what is acceptable practice in dealing with the demands of their work. For example, the organizational socialization can be very structured, such as having an athletic director orient a new employee in a very systematic manner, or this process can be unstructured, leaving the employee to ask questions of other employees as various situations arise. Understanding the organizational aspects of professional socialization allows the discovery of the necessary aspects of professional development in a work setting and can serve to improve both athletic training education and continuing education strategies. The purpose of this study, there-
fore, was to explore the professional socialization of certified athletic trainers (ATCs) in the high school setting in order to gain insight and understanding into how they initially learned and continued to learn their professional responsibilities in an organizational setting.

METHODS

Because the purpose of the project required an exploration of the actual experiences of ATCs in the high school setting and how the setting influenced their learning, qualitative methods were employed. The fundamental objective of qualitative research is to gain insight into and understand the meaning of a particular experience,11 and the context that influences the meaning,13 rather than drawing firm conclusions. Qualitative research is also well suited to study processes13 such as professional socialization.

In qualitative research, the protection of the participants’ anonymity is paramount. Therefore, audiotape recordings of interviews were transcribed and labeled with pseudonyms that are used at various locations in the manuscript. Moreover, at the completion of the study, the audiotapes were destroyed, but the transcripts were maintained using the established pseudonyms. Before data collection, appropriate institutional review board approval was received.

With qualitative methods, the researcher is the primary “instrument” for data collection and analysis, and extreme sensitivity is given to the nature and perspectives of human participants. A researcher’s perspective, however, can shape the analysis and interpretation of the qualitative data. My perspectives about the high school setting were shaped in 2 ways. First, I was formerly employed as a clinical high school ATC and interacted with coaches, athletic directors, and athletes and their parents. Second, at the time of data collection and analysis, I was a faculty member in a Commission on Accreditation of Allied Health Education Programs (CAAHEP)-accredited program that used several high school sites as clinical education experiences for the athletic training students. Entering this study, I believed that professionals are not simply products of their work environments but rather active participants in their professional development and that this process continues throughout their careers. This line of thought is consistent with symbolic interactionism, which is often used as a theoretic basis for grounded theory studies.14

Participants

A total of 15 individuals (12 ATCs currently practicing at the high school level, 2 current high school athletic directors who are also ATCs, and 1 former high school ATC) participated in the study. The average number of years in their current position for the 12 currently practicing ATCs was 10.1 ± 7.44, with a range of 2 to 25 years. The 2 athletic directors averaged 5.5 years of experience, and the former high school ATC had worked in that particular setting for 1 year before entering graduate school. The athletic directors and former high school ATC were included in order to cross-reference, or triangulate, the perspectives of the currently practicing ATCs. Six participants were women and 9 were men. The participants represented 3 different midwestern states. Participants were initially purposefully selected: that is, I recruited volunteers whom I knew and who agreed to interviews. I then asked these individuals for suggestions of other ATCs who might be willing to participate. The remaining individuals were contacted via either e-mail or phone and agreed to interviews. Before the interviews, participants were required to review and sign an informed consent form. The Table identifies other pertinent participant demographic information.

Data Collection and Analysis

Data were collected using semistructured interviews. Each interview incorporated several key questions or open-ended statements, including the following:

<table>
<thead>
<tr>
<th>Participant Pseudonym</th>
<th>Sex</th>
<th>School Enrollment†</th>
<th>City Population‡</th>
<th>Current Position</th>
<th>Previous Experience as ATC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Betty</td>
<td>F</td>
<td>3000–3500</td>
<td>100 000–115 000</td>
<td>Athletic director</td>
<td>High school setting</td>
</tr>
<tr>
<td>Marsha</td>
<td>F</td>
<td>2000–2500</td>
<td>60 000–75 000</td>
<td>Athletic director</td>
<td>High school setting, clinical setting</td>
</tr>
<tr>
<td>Reginald</td>
<td>M</td>
<td>2000–2500</td>
<td>45 000–60 000</td>
<td>Teacher/ATC</td>
<td>College setting</td>
</tr>
<tr>
<td>Clare</td>
<td>F</td>
<td>1000–1500</td>
<td>&lt;15 000</td>
<td>Full-time head ATC</td>
<td>College setting</td>
</tr>
<tr>
<td>Donald</td>
<td>M</td>
<td>2000–2500</td>
<td>30 000–45 000</td>
<td>Full-time head ATC</td>
<td>Graduate assistant, high school setting</td>
</tr>
<tr>
<td>Payton</td>
<td>M</td>
<td>1000–1500</td>
<td>&lt;15 000</td>
<td>College instructor</td>
<td>None entering high school setting, first position after bachelor's degree</td>
</tr>
<tr>
<td>Jennifer</td>
<td>F</td>
<td>1000–1500</td>
<td>30 000–45 000</td>
<td>Teacher/Head ATC</td>
<td>Graduate assistant, high school setting; assistant ATC, teaching aide</td>
</tr>
<tr>
<td>Alicia</td>
<td>M</td>
<td>2000–2500</td>
<td>45 000–60 000</td>
<td>Teacher/Head ATC</td>
<td>Clinical setting, high school setting</td>
</tr>
<tr>
<td>Robert</td>
<td>M</td>
<td>2500–3000</td>
<td>115 000–130 000</td>
<td>Full-time head ATC</td>
<td>None</td>
</tr>
<tr>
<td>Theo</td>
<td>M</td>
<td>1500–2000</td>
<td>30 000–45 000</td>
<td>Teacher/ATC</td>
<td>None</td>
</tr>
<tr>
<td>Jeremy</td>
<td>F</td>
<td>1500–2000</td>
<td>15 000–30 000</td>
<td>Full-time ATC</td>
<td>Clinical setting, high school setting</td>
</tr>
<tr>
<td>Samuel</td>
<td>M</td>
<td>1500–2000</td>
<td>15 000–30 000</td>
<td>Teacher/ATC</td>
<td>None</td>
</tr>
<tr>
<td>Beck</td>
<td>M</td>
<td>3500–4000</td>
<td>&lt;15 000</td>
<td>Teacher/ATC</td>
<td>Full-time teaching, no ATC responsibilities for 1 year</td>
</tr>
</tbody>
</table>

*ATC indicates certified athletic trainer.
†Data obtained from the appropriate state high school athletic association Web site.
‡Each city was located in a metropolitan area as designated by the US Census Bureau; population data obtained from the US Census Bureau.
A conceptual framework of the qualitative data organization is presented. Note that the concepts organized into the categories are representative samples of the concepts coded in the margins of the transcripts.

1. Describe your first few years of being an ATC at the high school level.
2. How did you learn your role and professional responsibilities at the high school level?
3. What has been your greatest challenge at the high school level, and how did you learn to deal with it?
4. What do you like best, or what are the good things about your current position?
5. What aspect of your job do you feel least satisfied by?
6. What is, or how would you describe, your professional mission?
7. What motivates you on a daily basis?
8. What advice might you give to an ATC just about to enter your current position?
9. What is your current professional responsibility?
10. What motivates you on a daily basis?

Because both athletic directors were former ATCs practicing in the high school setting, they were asked to reflect on their experiences as an ATC by answering questions 1 through 4 and question 8. Additionally, they were asked to describe the priorities of the athletic department, the role of ATCs in the high school setting, and the challenges that ATCs face in the high school setting.

The interviews ranged in length from approximately 35 to 105 minutes. Eight interviews were conducted by phone, and 7 interviews were conducted in person, based on feasibility and availability. Participants gave advanced written and verbal consent to tape record the interviews. The tape-recorded interviews were then transcribed and analyzed using a grounded theory approach. Data were collected until theoretical saturation was achieved.

The grounded theory approach, as discussed by Glaser and Strauss, is helpful for generating theory (a set of explanatory concepts) based on the data collected. I specifically used open, axial, and selective coding procedures documented by Strauss and Corbin. Raw data were analyzed inductively, and incidents or experiences related to the phenomenon under investigation were identified and labeled as a particular concept. This type of coding strategy is described as "creating tags," and the purpose is to produce a set of concepts that represents the information obtained in the interview. Identifying these concepts and placing them into like categories based on their content completed the formal open-coding process. Relating categories with any subcategories that might exist and examining how one category related to another completed the axial-coding process. Selective coding involved integrating the categories into a larger theoretic scheme and organizing the categories around a central explanatory concept, specifically, the proposition that informal learning processes were critical to the successful professional socialization of ATCs in the high school setting.

**Trustworthiness**

Several techniques were employed in order to establish trustworthiness of the data collection and analysis, including peer debriefing, data-source triangulation, and member checks. A peer debriefing was completed by having an athletic trainer with a formal education in qualitative methods (a minimum of 3 qualitative research methodology courses at the doctoral level) review the documented concepts and thematic categories for relevance, consistency, and logic. Moreover, the reviewer examined the interview questions in each transcript to determine if they were "leading" in nature. The textual data from any questions identified as being leading were not included in the analysis. The reviewer was in agreement with the findings based on the purpose of the study and even suggested other concepts that would strengthen one particular category.

Data-source triangulation, which is cross-checking perspectives, was obtained by interviewing current high school athletic directors and a former high school ATC. Member checks were completed electronically by e-mailing the results to 5 participants and allowing them to comment on the thematic categories. Three individuals responded, agreed with the results, and had no further input, indicating no misinterpretation of the professional-socialization process that emerged from this study. On an informal basis, I also explained the results to 4 other participants, and they were in agreement with the findings.

**RESULTS**

The concepts identified during the open coding were organized into 2 categories that gave insight into the professional socialization process: (1) an informal induction process, aspects of organizational learning, and (2) creating networks for learning. The Figure provides a conceptual framework and
identifies representative sample concepts and the categories into which they were placed. The axial coding allowed for making connections among categories and understanding the setting that influenced the socialization process. The selective-coding process allowed for the discovery of an overarching theme or informal learning processes that integrated the categories and gave insight into the resultant proposition that informal learning processes are critical to the successful professional socialization of ATCs in the high school setting.

An Informal Induction Process: Aspects of Organizational Learning

Certified athletic trainers entering the high school level reported extremely nonstructured, informal processes relative to learning the full extent of their professional responsibilities within the organization. Peer relationships drove most of the learning at the high school level in that ATCs were able to gain an understanding of their responsibilities by obtaining feedback from the coaches that they worked with and, although in some instances to a lesser extent, their athletic directors (Figure, sample concept A, category 1). For example, Theo stated that learning the “ins” and “outs” of the organization came from interactions with the coaching staff, specifically the football coach:

He’s an extremely intelligent human being and he knows the politics of the system and he knows…everything about the “ins” and the “outs” of the school district, and schools in general, so he was very helpful in getting me to learn more about the after-school athletic type aspect [of my job].

Supporting this claim, Alicia stated that “the football coach kind of showed me the ropes….” indicating that the positive relations with the coaching staff influenced her organizational learning when she initially entered the high school setting.

Based on the interview data, the peer relationships may have allowed for informal learning because the organizations were focused on academics and on the health and welfare of the students. That is, during the interview process, there were occasional discussions regarding minor conflicts between staff members, but absent from the interviews were identifiable power struggles that deterred high school ATCs from achieving their professional mission of providing quality health care. In fact, there was evidence to suggest that a common priority at each of the organizations allowed ATCs, coaches, and athletic directors to coexist in a collegial manner. For example, when asked about the organizational priorities, Theo stated:

Sports…definitely…helps define the high school. So I think the administration accepts that…[but] definitely the school’s number one mission is the education of its students. Our athletic department is very strict on its maintaining of the academic requirements for eligibility…we actually started an at-risk after school program for any kid who is in danger of failing a class…They get help with any questions they may have and they are required to do their homework before they go to practice or any game. I firmly believe I have one of the best coaching staffs to be able to work for as far as the kids come first and the victories don’t.

Both athletic directors interviewed for this project supported the ATC’s comments:

Well, the priority certainly…[is] the student-athletes, students first. So academics is very important to us here at High School A. We watch and monitor that very closely and all our coaches have that mindset. Then, certainly, the second would be the safety issue. That comes from my athletic experience, prevention. We are very attuned to proper weight training and nutrition. We constantly talk to student-athletes about diet, sleep, healthy choices for their bodies, [both] mentally and physically. Then, we feel that winning will take care of itself.

Academics are first and foremost in my mind. And I say athletics is an educational opportunity and so what we try to do is make…our mission [reflect this priority]. I make all the decisions based on that particular mission.

Participants in this study were socialized into their role in the high school setting using individual and informal tactics, meaning that they were often functioning independently and relying on trial-and-error learning (Figure, sample concept B, category 1). Moreover, many participants explained that they learned through the observation of others (Figure, sample concept D, category 1). For example, Marsha stated, “I am very fortunate that I’ve been around a lot of people who have very good people skills, and I feel that I picked up on that. I think athletic trainers are very adaptable to being flexible and can learn some of the ropes just by watching.”

Additionally, with the exception of working closely with coaches and other staff, there was no formalized mentoring in place to facilitate learning of their role. In short, the ATCs were expected to enter the setting and immediately begin functioning in their role in an independent manner (Figure, sample concept C, category 1).

There appeared to be no detectable timeline to the professional-socialization process relative to specific events or professional experiences, except that when each of the individuals entered the setting, there was a small period of adjustment. The induction process during this time was extremely informal. For the high school ATCs who obtained a position immediately after completing their undergraduate degree (Carol, Theo, Alicia, and Payton), much of the adjustment was related to moving from a setting that had a great deal of supervision to one with little supervision. For example, Theo mentioned:

[Although] the first year was pretty calm for the most part, [I] just tried to get used to things and being on my own without having a supervisor that could help me out with whatever I needed help with.

Even Reginald, reflecting on his first-year experience, stated, “I knew what I was doing [relative to] athletic training … but I didn’t know all the ‘ins and outs’ and the politics.”

Consistently, participants identified the necessity of setting up their health care system in the high school setting and learning how best to communicate with other staff (coaches and athletic directors), parents, and student assistants and how to meet the demands of providing health care to such a large athletic population. Learning this role was informal and often described as a trial-and-error process. For example, Alicia reflected on initially entering the high school setting and stated:

Actually, I learned by trial and error. There really wasn’t anyone [to facilitate my learning]. The other person that I worked with started the same day I did, so neither of us had any idea, we just kind of went into it with what knowledge we had of what a college [athletic] training room was like and kind of took that into our own [facilities]. Obviously we had to modify certain things, like getting prescriptions for modalities and things like that.

Similarly, when asked about how she initially learned her role in the high school, Jennifer commented on the induction period and stated that “unless there was previously an athletic trainer within the organization, there would be nobody to orient a newcomer to [his or her] role.” Confirming this theme, Robert stated, “I was not given any type of orientation or didn’t meet any of the coaches. I was given a set of keys and told ‘good luck.’ I was on my own…My budget was woe...so it was tough that first year….” Donald added, “I think a lot of [learning of the job] is trial and error. There are
a lot of things that I don’t do now that I did years ago and I thought, well, that didn’t quite work, I could have handled that situation differently.”

In order to successfully navigate the informal induction period and evolve as an ATC at the high school setting, participants sought to create learning networks beyond their institution. The next theme further explains this phenomenon.

Creating Networks for Learning

Participants consistently discussed contacting other ATCs in order to learn how best to deal with their responsibilities. While many ATCs who were new to the high school setting often relied on their previous mentors for advice and direction, it was interesting to find that both novices and veterans at the high school level took the initiative to make connections with other ATCs outside the high school in order to continually learn (Figure, sample concepts A and C, category 2). In fact, Theo stated that while he did not have to travel to away events with the teams, he did so in order to network with the other ATCs at local schools and learn from them:

I have done a lot of traveling with my sports which I don’t have to do but because of the bonds I’ve made with the kids and parents I… go to the away games. I also get to talk to the other athletic trainers and [discuss various situations] and sometimes they have some helpful answers or places to look for better answers.

Jeremy also identified networking as critical to learning:

I think that is one of the biggest things you learn as you go is that you do develop some type of network of athletic trainers who you can call and say “this is the [most interesting] thing. I’ve been doing this [procedure] and this [technique], but [the athlete] is just not getting better. What do you think?”

It appears that the role of a high school ATC evolves as he or she gains more experience. Initially ATCs make network connections in order to learn, but as they become more experienced, they play more of a mentoring role, being contacted by less experienced peers for advice about how to deal with issues in the high school setting. For example, Robert stated:

Professionally… my involvement with [the regional professional association] has been really rewarding. It has helped me a lot to deal with life… I will often call up Bruce Johnson (pseudonym) and bounce [ideas] off of him. But [now] more people call me and bounce things off of me more than I call other people. I really feel like I’m the grandfather in the area. Now, I get phone calls all the time from other athletic trainers about ‘how should I handle this…?’ Maybe it’s because I’m old now too, but there is a bigger network now than there was 20 something years ago.

Aside from facilitating learning, networks were also used to provide help and social support (concepts B and D, category 2). For example, Reginald explained that he received a call from a high school ATC in a nearby suburb. The ATC had experienced the death of an athlete approximately 6 months earlier, and called to get contact information for another area ATC who experienced an athlete’s death more recently because he wanted to offer his support.

Given the nature of the 2 thematic categories, the resultant proposition is that informal learning procedures are critical to the professional-socialization process for ATCs working in the high school setting. The results of this study suggest that learning through informal means, such as collegial networks, organizational peers, and trial and error, are necessary elements for navigating the high school work setting and being socialized into the ATC role.

DISCUSSION

Professional socialization is a complex process that has the propensity to influence an individual’s success in a work environment. The socialization literature concludes that the initial entry into an organizational setting is a period of adjustment for many professionals. This study supports these findings, as participants suggested that there was an initial adjustment when entering the high school setting from their previous setting (undergraduate program, master’s program, or previous job). Additionally, many participants learned through informal means such as trial and error and by observing others in the organizational setting. Based on a socialization study by Ostroff and Kozlowski, this is not unusual, as many individuals frequently rely primarily on observation of others and trial and error to acquire their information in an organization. Thus, informal learning plays a critical role in the professional-socialization process.

Informal Learning

Informal learning can be defined as a lifelong process by which individuals acquire and accumulate knowledge, skills, attitudes, and insights from daily experiences and exposure to an environment and individuals rather than from a structured, hierarchic education system. Informal learning generally occurs as a means of achieving particular individual or organizational goals, often as a result of expanded responsibilities. Informal learning is reported to account for up to 90% of new learning.

Informal learning is often intentional but lacks the formalized structure that is found in education-based systems. Although some authors contended that trial-and-error learning is better defined as incidental learning because it is not intentional, the participants in this study appeared to make intentional, conscious efforts to identify which professional actions or strategies worked and which did not, in order to better meet the demands of their work environments. Informal learning implies that much of the learning was experiential and action based in nature. That is, learning occurred as participants attempted to find solutions to problems or events related to their work setting.

As the participants of this study entered the high school setting and accepted the organizational challenges involved in developing a system of health care, they engaged in informal learning to manage their new responsibilities. Leslie et al. stated that more informal learning takes place when the goals of an individual and an organization are in alignment. As in this study, a common priority appeared to be shared by ATCs, athletic directors, and coaching staffs that suggested the student-athletes’ welfare related to academics and health was a main concern. Perhaps this allowed for a great deal of informal learning to take place.

Garrick stated that many respected adult educators link informal learning to concepts such as “autonomous learning,” “self-directed learning,” and “independent learning.” In fact, supporting these claims, Lankard suggested that to enhance informal learning, learners must (1) autonomously direct their learning, (2) self-evaluate their learning, (3) engage in critical self-reflection, and (4) think critically. This has many implications for athletic training preservice and continuing education.
Implications for Preservice Athletic Training Education

Athletic training preservice education has thoroughly emphasized the necessity of addressing content, competence, and clinical proficiency. This study, however, identifies the necessity of fostering reflective practitioners who are self-directed and self-evaluative to fully prepare them as informal learners. Educators can enhance these aspects by using reflective journals, individualized learning plans, and formalized student self-evaluations. Although these characteristics may be approached in some programs and curriculums, given their importance to professional development, consideration must be given to making these goals explicit during undergraduate athletic training education.

Implications for Continuing Athletic Training Education

The previously mentioned characteristics have implications relative to continuing professional education as well. For example, Bickham30 suggested that the “foremost goal of continuing professional education is to teach professionals to develop and hone critical-thinking skills.” Continuing professional educators who give ATCs opportunities to conduct verbal reasoning and problem solving and who consciously raise questions can help accomplish this goal.30 Unfortunately, a great deal of continuing education in athletic training focuses on content, such as transferring information by listening to lectures, completing home study courses, or updating cardiopulmonary resuscitation skills, rather than focusing on fostering critical-thinking ability. Moreover, although the transfer of information at a continuing education event can be enlightening for many, a significant issue is whether the information improves professional practice.31

Adult learners will engage in learning activities providing their job performance will be enhanced by the experience.32 Given the participants’ need to learn information in order to solve problems linked to practice, continuing education must be more integrated with practice-based problems if it is to be effective.30 Perhaps this is why the participants in this study created learning networks. Ritchie33 commented on networking and stating that “while professionals tend to be self-directing and autonomous, they are not necessarily singular or practicing in isolation. Professionals rely on other professionals to help meet their continuing learning needs.”

Given the self-directed nature of informal learning, perhaps alternative strategies to continuing education, such as self-directed learning plans based on an individual’s contextual learning needs are a viable continuing education strategy.34 Such a strategy would foster self-direction, self-evaluation, and self-reflection.35 Additionally, critical self-reflection strategies, such as video analysis of skills and self-evaluations, can promote intentional active learning and the formation of learning communities that facilitate practice-based learning36 and support the direct link of applying continuing professional education to practice.

Limitations

Most of the participants in this study were practicing at schools located in metropolitan areas as designated by the US Department of Commerce, Bureau of the Census, yet none were located in an inner city school or extremely rural setting. The propositions resulting from this grounded theory, therefore, may not be transferable to the inner city or rural school context.

CONCLUSIONS

The purpose of this study was to gain insight into and understanding of how ATCs in the high school setting initially learned and continued to learn their professional responsibilities in an organizational setting. The organizational aspects of the professional-socialization process among high school ATCs are principally informal in nature. As such, the ATCs relied on informal learning strategies during their period of induction. To facilitate their continued development, informal learning networks that largely comprised colleagues outside of the organizational setting were created.

To ensure that individuals effectively learn through informal means, preservice athletic training education programs would be well advised to foster the development of reflective practitioners who think critically and are self-directed and self-evaluative. This can be accomplished by using such educational strategies as reflective journals, learning plans, and independent projects.

Continuing professional educators should also attempt to foster self-evaluation, critical reflection, and critical-thinking ability. Continuing professional educators can accomplish this by employing strategies such as verbal reasoning and problem solving and consciously raising questions and giving clinicians an opportunity to discuss their thought processes in a non-threatening learning environment. Moreover, it has been argued that continuing education should be linked to practical problems.

Because informal learning is highly contextual, multiple influences have the propensity to shape the extent to which informal learning is successful, including cultural factors, career structure, technology, and learning needs.35 As such, future studies could investigate exactly how these factors influence informal learning and role socialization. Additionally, because informal learning can be inhibited in many ways, it may be helpful to examine the environmental inhibitors (ie, job demands) of informal learning in various athletic training settings.

ACKNOWLEDGMENTS

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Nonthermal Effects of Therapeutic Ultrasound: The Frequency Resonance Hypothesis

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Objective: To present the frequency resonance hypothesis, a possible mechanical mechanism by which treatment with nonthermal levels of ultrasound stimulates therapeutic effects. The review encompasses a 4-decade history but focuses on recent reports describing the effects of nonthermal therapeutic levels of ultrasound at the cellular and molecular levels.

Data Synthesis: A search of MEDLINE from 1965 through 2000 using the terms ultrasound and therapeutic ultrasound.

Conclusions: The concept of the absorption of ultrasonic energy by enzymatic proteins leading to changes in the enzymes activity is not novel. However, recent reports demonstrating that ultrasound affects enzyme activity and possibly gene regulation provide sufficient data to present a probable molecular mechanism of ultrasound's nonthermal therapeutic action. The frequency resonance hypothesis describes 2 possible biological mechanisms that may alter protein function as a result of the absorption of ultrasonic energy. First, absorption of mechanical energy by a protein may produce a transient conformational shift (modifying the 3-dimensional structure) and alter the protein's functional activity. Second, the resonance or shearing properties of the wave (or both) may dissociate a multimolecular complex, thereby disrupting the complex's function. This review focuses on recent studies that have reported cellular and molecular effects of therapeutic ultrasound and presents a mechanical mechanism that may lead to a better understanding of how the nonthermal effects of ultrasound may be therapeutic. Moreover, a better understanding of ultrasound's mechanical mechanism could lead to a better understanding of how and when ultrasound should be employed as a therapeutic modality.

Key Words: immunology, injury, signal transduction, molecular mechanism, wound healing, cytokines

Ultrasound has become a common therapy for a number of clinical conditions: strained ligaments, inflamed tendons and tendon sheaths, lacerations and other soft tissue damage, scar tissue sensitivity and tension, varicose ulcers, amputations, neuromata, strained and torn muscles, inflamed and damaged joint capsules, fasciitis, and delayed-onset muscle soreness.1,2 Recent uses include the accelerated healing of fractures,3-5 muscle injury,6 and thrombolysis.7-16

Over the past several years, research investigating the cellular and molecular effects of nonthermal levels of ultrasound has accumulated. While clinicians state that ultrasound is used to accomplish heating within deep tissue, there is a common, whispered belief that heating alone cannot account for the clinical effects, especially when ultrasound is delivered at nonthermal settings. My purpose is to review the past 4 decades of ultrasound research and to propose a molecular mechanism whereby the mechanical properties of ultrasound interact with the molecular and multimolecular complexes within the cell. The frequency resonance hypothesis incorporates past research demonstrating ultrasound's mechanical properties (absorption, cavitation, acoustical streaming) with current knowledge within the field of cellular and molecular biology, specifically the activation of proteins and signal-transduction pathways that may result in modifications to cellular function.

Thermal Effects of Ultrasound

Ultrasound is capable of producing thermal therapeutic effects.2 In 1987, Dyson1 suggested that the tissue must reach a temperature of 40°C to 45°C for at least 5 minutes to be therapeutic in nature. Experiments performed with nonperfused tissue demonstrated that ultrasound could increase the tissue temperature at a rate of 0.86°C/min (1 W/cm², 1 MHz).17 However, the results of these experiments were difficult to interpret because they were performed in nonperfused tissue. In living tissue, as the temperature increases, the normal blood flow to the area dissipates the heat. More recent, direct in vivo measurement of tissue temperature during ultrasound treatment has resolved the question of tissue heating.18-21 Draper et al.,18-19 Ashton et al.,20 and Chan et al.21 inserted thermistors to various depths (5 cm or less) and measured the increase in muscle temperature during a 10-minute treatment with either 1-MHz or 3-MHz ultrasound. The data show that treatment...
with 1-MHz or 3-MHz ultrasound resulted in a time- and dose-dependent increase in tissue temperature. The 3-MHz frequency increased tissue temperature at a faster rate than the 1-MHz frequency. More recently, Ashton et al. and Chan et al. employed similar techniques to study increases in temperature in the patellar tendon and the effects of coupling media on increases in tissue temperature. While a number of questions remain unanswered with respect to the thermal effects of ultrasound, the purpose of my review is to focus on the nonthermal effects of ultrasound. I will not include the various therapeutic applications of ultrasound that have recently been reviewed elsewhere.

Nonthermal Effects of Ultrasound

A number of experimental designs appear to have successfully isolated the nonthermal from the thermal effects of ultrasound within cellular systems. In vivo, a portion of the energy from the ultrasound wave is absorbed into the tissue structure and converted into heat energy. The amount of heating is determined by the frequency and intensity of the ultrasound (dosage) and the type of tissue that is exposed to acoustic energy. A 1982 report demonstrated a direct relationship between the absorption of ultrasound and amount of protein. More simply, as the concentration of protein increased, the absorption of ultrasound increased. In normal tissue, the absorption of ultrasound energy varies depending on the amount of protein in the tissue. In 1980, Love and Kremkau demonstrated that by eliminating extracellular tissue structures (collagen, fibrin, elastin, etc) and placing only the cells in tissue culture media maintained at 37°C, they could treat cells at therapeutic levels without significant increases in temperature (less than 0.5°C over a 10-minute exposure). Our data confirm that cell cultures treated with either 1-MHz or 3-MHz ultrasound at intensities of 0.5 W/cm² sustained less than 0.5°C increases over a 10-minute exposure (unpublished observation, 1998). At first it may seem that these data are contradictory to those of Draper et al., Ashton et al., and Chan et al.; however, the 2 experimental protocols were significantly different. The in vivo measurements performed by Draper et al., Ashton et al., and Chan et al. recorded actual increases within intact muscle and tendon. The tissue culture protocol eliminates the extracellular structural proteins (collagen, fibrin, elastin, etc) that are responsible for most of the increase in temperature observed within the intact tissues. Moreover, the tissue culture protocol makes it possible to “eliminate” the thermal effects of ultrasound and to study the mechanical effects of ultrasound in an attempt to identify a mechanism of action.

While exposure of single cells to ultrasound does not increase the overall temperature of the experimental system, it is difficult to determine whether larger temperature increases occurred at the cell surface or within the microenvironments of the cell. Theoretically, larger increases in temperature could occur within microenvironments of the cell as a result of cavitation. However, direct measurements of these types of microenvironmental changes in temperature are currently not possible.

Therapeutic ultrasound produces a combination of nonthermal effects (acoustic streaming and cavitation) that are difficult to isolate. Acoustic streaming is defined as the physical forces of the sound waves that provide a driving force capable of displacing ions and small molecules. At the cellular level, organelles and molecules of different molecular weight exist. While many of these structures are stationary, many are free floating and may be driven to move around more stationary structures. This mechanical pressure applied by the wave produces unidirectional movement of fluid along and around cell membranes.

Cavitation is defined as the physical forces of the sound waves on microenvironmental gases within fluid. As the sound waves propagate through the medium, the characteristic compression and rarefaction causes microscopic gas bubbles in the tissue fluid to contract and expand. It is generally thought that the rapid changes in pressure (caused by the leading and lagging edges of the sound wave), both in and around the cell, may cause damage to the cell. Substantial injury to the cell can occur when microscopic gas bubbles expand and then collapse rapidly, causing a “microexplosion.” Although true microexplosions, referred to as unstable cavitation, are not thought to commonly occur at therapeutic levels of ultrasound, pulsation of gas bubbles may disrupt cellular activity, altering the function of the cell.

Early studies investigating the gross effects of acoustic streaming and cavitation on cells showed growth retardation of cells in vitro, increases in protein synthesis, and membrane alterations. Combined, these results may suggest that ultrasound first “injures” the cell, resulting in growth retardation, and then initiates a cellular recovery response characterized by an increase in protein production. These findings encompass both continuous and pulsed ultrasound at therapeutic levels ranging from 0.1 to 1.7 W/cm².

Attraction of Immune Cells to the Injured Area

The natural course of tissue injury can be categorized into 4 distinct phases: acute inflammation, clearance of tissue debris, cellular proliferation, and tissue remodeling. With the early arrival of immune cells to the injured tissue, the immune system can be destructive in nature. When soft tissue is injured, platelets and mast cells are activated and release chemokines, attracting polymorphonuclear cells and blood monocytes (macrophages). Once activated, macrophages produce a unique set of proteins that aid in the destruction of damaged tissue and attract additional lymphocytes to the area. A concerted recruitment of lymphocytes is accomplished by the production of chemokines and the activation of adhesion molecules on the surface of the local capillaries. Adhesion molecules can be viewed as docking proteins that grab circulating lymphocytes and aid in their migration to the injured area. A concerted recruitment of lymphocytes is accomplished by the production of chemokines and the activation of adhesion molecules on the surface of the local capillaries. Adhesion molecules can be viewed as docking proteins that grab circulating lymphocytes and aid in their migration to the injured area.

A simple analogy for changes in 3-dimensional shape altering function is a pocketknife. When the knife is open, the blade is functionally available and can cut; however, when the knife is closed, the blade is functionally not available. Similarly,
proteins have an “active site” that can be either available or not available, depending on the 3-dimensional shape of the protein.

**Inflammatory Response, Injury Repair, and Therapeutic Ultrasound**

Later in the inflammatory process, immune cells alter their course of action, aiding in the clearance of tissue debris and stimulating tissue remodeling. This pivotal action is directed by cytokines. For example, the arrival of T cells in an injured area may enhance the immunologic response by releasing T-cell growth factors (interleukin [IL]-2 and IL-4) and immune regulatory cytokines (IL-10 and interferon-γ). At a certain point in the immune intervention, anti-inflammatory cytokines (largely transforming growth factor-β) are either produced or activated. These anti-inflammatory cytokines down-regulate T-cells and redirect the cellular activities toward proliferation of fibroblasts, collagen production, and remodeling of the damaged tissue.

A number of reports have demonstrated that ultrasound affects cells that are centrally involved in the immune response. Specifically, ultrasound has been shown to modulate vasoconstriction; lymphocyte adhesion properties of endothelium, mast cell degranulation, phagocytosis by macrophage, production of growth factors by macrophages; calcium fluxes in fibroblasts; angiogenesis; proliferation of T-cells, osteoblasts, fibroblasts, and a number of proteins associated with inflammation and repair (IL-1, IL-2, IL-6, IL-8, interferon-γ, fibroblast growth factor-β, vascular endothelial growth factor, collagen) and to accelerate thrombolyisis. In general, most of these researchers used a frequency of 1 MHz or 3 MHz, and the intensities ranged from 0.1 to 1.5 W/cm². An alternative therapeutic protocol employs a frequency of 45 kHz. An intensity range of 5 to 100 mW/cm² was shown to increase the production of IL-1, IL-8, vascular endothelial growth factor, fibroblast growth factor-β, and collagen; promote bone healing; and accelerate thrombolyisis. The long-wave (45-kHz) ultrasound increases penetration depth and, therefore, seems to be more appropriate than traditional high-frequency ultrasound (1 MHz and 3 MHz) for promoting revascularization and bone healing at greater depths.

**Ultrasound, Signal Transduction, and Gene Regulation**

Additional reports suggest that ultrasound alters cellular membrane properties (cellular adhesion, membrane permeability, calcium flux, and proliferation), possibly activating signal-transduction pathways that lead to gene regulation. Importantly, exposure to ultrasound caused an increase in intracellular calcium in fibroblasts, suggesting that the mechanical effects disrupt the normal function of the membrane, permitting leaking of calcium into the cell. After ultrasound exposure, the cells rapidly expelled the calcium and returned to a homeostatic state. Mortimer and Dyon eliminated the effects of transient cavitation and gross heating as possible mechanisms for the resultant increases in intracellular calcium. Cells employ calcium as a cofactor in regulating the activity of enzymes, many of which are associated with signal-transduction pathways. Activation of calcium-sensitive signal-transduction pathways (protein kinase C and cyclic AMP) commonly results in gene activation. The resultant protein production could modulate intracellular functions and the activity of surrounding cells. A number of the experiments reviewed in the Table demonstrated increases in specific proteins after exposure of cells to therapeutic levels of ultrasound. Combined, these findings suggest that therapeutic ultrasound can modulate signal-transduction pathways that lead to gene regulation or the modulation of RNA translation to a protein product, or both.

**Frequency Resonance Hypothesis**

Cumulatively, the data may suggest that the mechanical energy within the ultrasound wave and the shearing force of the wave combine to produce mechanical properties that perturbate the cellular membrane and the molecular structures within the cell. The central premise of the frequency resonance hypothesis is that the mechanical energy within the ultrasound wave is absorbed by proteins, altering the structural conformation of an individual protein or the function of a multicomponent complex. Moreover, the ultrasound wave may induce resonant activity in the protein, modulating the molecule’s or multicomplex’s effector function.

The following discussion employs enzymatic proteins as a molecular model. One can view an enzymatic protein as a physical machine performing a physical function within a cell. Enzymes are commonly found in 1 of 2 conformational shapes: on or off. Movement between these 2 conformations (or 3-dimensional shapes) requires a change in the state of energy, which is normally accomplished by the addition or removal of a phosphate molecule. Once an enzyme within a signal-transduction cascade is activated, the signal is amplified to execute an effector function.

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<table>
<thead>
<tr>
<th>Increase in Protein or Cellular Function</th>
<th>Producing Cell Type</th>
<th>Effector Function</th>
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<tbody>
<tr>
<td>Interleukin-1β</td>
<td>Osteoblasts, monocytes</td>
<td>General inflammatory mediator</td>
</tr>
<tr>
<td>Interleukin-2</td>
<td>T cells</td>
<td>T-cell growth</td>
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<tr>
<td>Interleukin-8</td>
<td>Osteoblasts</td>
<td>Endothelial cell migration and proliferation</td>
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<tr>
<td>Vascular endothelial growth factor</td>
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</tr>
<tr>
<td>Basic fibroblast growth factor</td>
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<td>Fibroblast growth factor</td>
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</tr>
<tr>
<td>Collagen</td>
<td>Osteoblasts, fibroblasts</td>
<td>Wound healing</td>
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<tr>
<td>Chloramphenicol acetyl transferase</td>
<td>HeLa, NIH/3T3, C1271</td>
<td>Gene expression of liposomal transfection</td>
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<td>Increased proliferation</td>
<td>Fibroblasts</td>
<td>Enhanced wound healing</td>
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<tr>
<td>Increased proliferation</td>
<td>Osteoblasts</td>
<td>Enhanced wound healing</td>
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<tr>
<td>Lymphocyte adhesion</td>
<td>Endothelial cells</td>
<td>Enhanced lymphocyte trafficking</td>
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<tr>
<td>Vasodilation</td>
<td>Capillary, endothelium</td>
<td>Enhanced blood flow</td>
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The frequency resonance hypothesis suggests that the energy provided to the enzyme by the ultrasound wave may induce transient conformational shifts in certain enzymatic proteins, altering the enzyme’s activity (ie, kinases or phosphatases) and the overall function of the cell (Figure 1). Alternatively, ultrasound's resonating force may result in the dissociation of functional multimolecular complexes (Figure 2) or the release of a sequestered molecule by dislodging an inhibitor molecule from the multimolecular complex (Figure 3). In essence, the mechanism of ultrasound’s action in Figures 2 and 3 is the same. Ultrasound disrupts a multimolecular complex. However, Figure 2 represents a functionally active complex, while Figure 3 represents a functionally sequestered molecule. One can view an inhibitor molecule as a “safety block” that functionally inhibits or sequesters a protein from working. When the safety block is released, the protein is then operable.

Shearing forces produced by ultrasound may also play a role in the dissociation of multimolecular complexes. Hypothetically, frequency resonance may imply that different frequencies (1 MHz, 3 MHz, 45 kHz, and others) establish unique resonant or shearing forces (or both). Moreover, various frequencies may affect combinations of proteins or multimolecular complexes in different ways, lending to the possibility of targeted effects at the cellular and molecular levels.

The frequency resonance hypothesis differs from acoustic streaming and cavitation at the basic levels. First, acoustic streaming relates to the movement of objects from one place to another as a function of the force of the wave. In terms of ultrasound therapy, phonophoresis is commonly used to move medication transdermally. Second, cavitation relates to the oscillation of microscopic gas bubbles that may, in turn, affect the cell or cellular process. However, the frequency resonance hypothesis relates to the absorption of ultrasound by proteins and protein complexes that may directly result in alterations to signaling mechanisms within the cell, either by inducing a conformational shift or by disrupting a multimolecular complex.

In the original experiment investigating whether ultrasound could alter protein activity, the researchers reported no effect with respect to the monomer or dimer form of the enzyme creatine kinase.61 However, Chetverikova et al61 reported that ultrasound decreased the activity of the dimeric and tetrameric forms of creatine kinase and suggested that the decrease in activity was due to the disruption of the multimolecular forms of creatine kinase (represented in Figure 2). The authors inferred that ultrasound did not directly affect enzyme activity and that the primary acousto-biological interaction appeared to be occurring at a higher level of organization complexity. However, more recent investigations7-16,54-55 have shown that ultrasound increases thrombolysis, demonstrating that ultrasound can increase enzyme activity (represented in Figure 1). These data support the tried research saying, “The absence of proof is not the proof of absence.”

The concept of the absorption of ultrasonic energy by enzymatic proteins leading to changes in the enzymes’ activity is not novel61,62; however, the demonstration that ultrasound can increase or decrease protein activity and possible gene regulation is more recent.7-16,54-56 While considerable data exist, a model suggesting a molecular mechanism for how the absorption of ultrasound by proteins affects the cell function is novel and is presented here for the first time.

Frequency resonance is one possible explanation of why exposure to ultrasound increased enzymatic activity, resulted in thrombolysis, and did not alter the activity of the enzymes creatine kinase, lactate dehydrogenase, hexokinase, and pyruvate kinase.7-16,54,55,61 A simple analogy would be 2 tuning forks located at one end of a room, with different frequencies A and B. At the opposite end of the room is a third tuning fork with the same frequency as fork A. Fork A is struck, generating a sound. The sound waves travel through the air and are absorbed at the other end of the room by fork B to produce the same resonating sound, while fork B remains silent. The possibility exists that thrombolysis is affected by the frequencies “designated” as therapeutic, while creatine kinase, lactate dehydrogenase, hexokinase, and pyruvate kinase are not.7-16,54,55,61 Importantly, these reports do not directly demonstrate that a conformational shift is occurring in these enzymes but support the hypothesis.

On the surface, it may appear that the observed decrease in creatine kinase activity and an increase in thrombolytic activity are contradictory; however, a fundamental difference exists. The decrease in activity of the creatine kinase was, most likely, a result of the disruption of the multimolecular dimeric or tetrameric (or both) forms of creatine kinase (Figure 2), while the increased thrombolytic activity may be more associated with activation through harmonic resonance (Figure 1).

In any case, the mechanical effects of ultrasound may result in either the activation or inactivation of an enzymatic protein or a dissociation of a protein complex, leading to alterations in...
signal transduction. The frequency resonance hypothesis may describe the molecular mechanism or mechanisms responsible for alterations in cellular membrane properties, increases in protein production,* and modulation of enzyme activity.7-16,54-56

Frequency resonance and shearing forces on multimolecular complexes may combine to produce the nonthermal effects of therapeutic ultrasound. Collectively, the experiments reviewed here support the frequency resonance hypothesis and demonstrate that therapeutic ultrasound may modulate signal-transduction pathways and gene products associated with the inflammatory response and cells directly involved in the healing response (see Table).

**Clinical Implication of Ultrasound Research at the Cellular and Molecular Levels**

The purpose of this paper is to raise the awareness that therapeutic levels of ultrasound (1 MHz, 3 MHz, and 45 kHz) stimulate cellular and molecular effects within cells that are centrally involved in the inflammatory and healing processes (Table).5,7-16,34,40-42,45-55 Cumulatively, these reports provide important information that may lead to a better understanding and clinical application of therapeutic ultrasound.1,5,7-16,34,40-42,45-55 Currently, no clear guidelines exist that provide the clinician with protocols directing when in the injury and healing response ultrasound should be administered, nor are there guidelines on the frequency, intensity, treatment times, or number of treatments required for efficacy. In a recent review of the literature, a wide spectrum of ultrasound treatment protocols was found.63 In the 10 papers found to be scientifically acceptable, a broad range of treatment settings and methods was used, including (1) nine different clinical indications, (2) five different frequencies, (3) continuous and pulsed output, (4) W/cm² ranging from 0.02 to 2.6, (5) treatment time ranging from 2 to 15 minutes, and (6) energy density ranging from 2 to 150 J/cm².64-70 Due to the variety of clinical indicators and methods employed, sufficient clinical data are currently not available to generate scientifically sound recommendations for treatment protocols.

The ultrasound treatment protocols employed to investigate the cellular and molecular effects (see Table) were usually a 10-minute exposure at 1 or 3 MHz and 0.1 to 1.5 W/cm²,5,39-42,45,48,50,53,56 The experimental protocol investigating thrombolyis ranged from 10 to 200 minutes of exposure at 0.17 to 3.4 MHz and 0.25 to 8.0 W/cm², both continuous and intermittent.7-16 A third experimental protocol employing a frequency of 45 kHz and an intensity range of 5 to 100 mW/cm² resulted in increased production of IL-1, IL-8, vascular endothelial growth factor, fibrinolysis, growth factor-b, and collagen, promotion of bone healing, and acceleration of thrombolysis.5,45,54,55

The logical argument is that in vitro effects cannot be directly applied to clinical treatment protocols. However, from a cellular biology point of view, a strong argument can be made that if a stimulus reaches a critical threshold (eg, consider depolarization thresholds) the cell will respond, regardless of whether the cell is in vitro or in vivo (eg, insulin, histamine, aspirin, or any of the proteins listed in the Table). Importantly, cells in tissue culture usually respond to nanogram or microgram per milliliter quantities of a stimulus.

However due to pharmacokinetics (ie, administration, absorption, distribution, and elimination of a drug), higher concentrations and multiple doses per day are normally required to achieve clinical efficacy.

While general recommendations for ultrasound treatment suggest 5 to 10 minutes of exposure and 1 to 3 treatments per day, clinical treatments are almost exclusively done once a day. The possibility exists that clinical treatment protocols commonly employed for ultrasound are not sufficient for therapeutic efficacy. In the review by Robertson and Baker,63 both of the methodologically acceptable studies showing clinical efficacy used pulsed ultrasound (1:4) with treatment times of 15 minutes, resulting in energy densities of 60 and 150 J/cm², respectively.64,65 Conversely, the remaining 5 studies lacking efficacy employed pulsed ultrasound and exposure times of 2 to 10 minutes, resulting in overall energy densities of 2 to 40 J/cm².66-70 The frequency resonance hypothesis may suggest that different ultrasonic frequencies (1 MHz, 3 MHz, 45 kHz, and other) may require different durations of exposure (time), different energy densities (J/cm²), or both, to reach therapeutic efficacy.

The identification and scientific understanding of therapeutic ultrasound’s nonthermal mechanisms may lead to a comprehensive and effective clinical strategy. Further research is needed on 2 fronts: (1) cellular and molecular research to determine whether the mechanical mechanisms proposed by the frequency resonance hypothesis can be elucidated and provide insight into a comprehensive strategy for the clinical indications of therapeutic ultrasound at various frequencies, and (2) methodologically sound clinical research designed to provide meaningful input and outcome measures related to clinical efficacy. Both avenues of research should strive to establish time and dose-dependent response curves.

**ACKNOWLEDGMENTS**

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**REFERENCES**

The Safety and Efficacy of Anabolic Steroid Precursors: What is the Scientific Evidence?

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**Objective:** Anabolic steroid precursors have gained widespread popularity as ergogenic supplements. Advertisements for these supplements claim that they increase endogenous testosterone production and protein synthesis, resulting in increased lean body mass and strength during training. At this time scientific support is limited, but the potential for serious side effects exists and the popularity of these supplements continues to grow. This review provides rationales for the ergogenic claims regarding steroid precursors and compares claims with data from scientifically controlled investigations.

**Data Sources:** A search of MEDLINE and SPORT Discus from 1980 to 2001 using the key words dehydroepiandrosterone, androstenedione, and androstenediol in combination with testosterone, estrogen, exercise, performance, and side effects.

**Data Synthesis:** Although fairly new to the athletic community, steroid precursors have been used as ergogenic or anabolic agents for quite some time. Suggested gains in strength and lean body mass are attributed to an increase in the endogenous production of testosterone and enhanced protein synthesis. Most of the scientific data, however, do not support manufacturers’ ergogenic claims, and the potential for serious side effects, such as decreased high-density lipoprotein cholesterol and increased estrogen concentrations, has been associated with precursor use. Thus, the safety and efficacy of these supplements must be questioned.

**Conclusions/Recommendations:** It appears that the risks associated with the use of anabolic steroid precursors outweigh any possible ergogenic benefits. Furthermore, these supplements are banned by most athletic organizations. Thus, it is extremely important that athletic trainers are able to educate athletes on these issues so they can continue to perform at an optimum level in a safe and healthy manner.

**Key Words:** dehydroepiandrosterone, adrostenedione, androstenediol, ergogenic aid

Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone that have been used by athletes for decades to increase lean body mass, strength, and overall athletic performance. A number of side effects have been associated with AAS use, including acne, hair loss, increased risk of heart disease, kidney and liver dysfunction, hypertension, and impotence. It is currently illegal to possess AAS without a prescription from a licensed physician. The legal issues and dangers associated with AAS have resulted in a reluctance to use them on the part of many athletes and a search for a more natural method to improve performance. The result has been an increase in the popularity of nutritional supplements marketed as natural testosterone enhancers or steroid precursors. Although bodybuilders have been using steroid precursors for some time, it was home-run hitter Mark McGuire’s well-publicized use that first brought them to the attention of the athletic community and the public. While there are numerous precursor products on the market, the most popular are those containing dehydroepiandrosterone (DHEA) and the various forms of androstenedione (A’dione) and androstenediol (A’diol).

**Steroid Precursors**

Dehydroepiandrosterone, A’dione, and A’diol are androgenic hormones produced primarily by the adrenal glands and gonads that act as precursors in the endogenous production of testosterone and estrogen. Because they bind poorly to androgen receptors, these precursors have little inherent androgen action on their own. However, they provide an important pool of circulating precursor steroids that can be converted to active androgens and estrogens in the peripheral tissue where they act (Figure). Virtually all sex steroids in the human body are made from DHEA, which is the most abundant steroid hormone in circulation. This natural process begins in the adrenal cortex where DHEA is secreted and eventually converted to either A’dione or A’diol, which are both immediate precursors to testosterone.

The conversion of A’dione and A’diol to testosterone occurs in the testes and other target tissues via 2-way reactions that are regulated by the enzymes 17β-hydroxysteroid dehydrogenase and 3β-hydroxysteroid dehydrogenase, respectively. This conversion can occur in many cells containing androgen or estrogen receptors, including adipose, bone, muscle, breast, prostate, skin, brain, and liver cells. In men, approximately 95% of circulating testosterone after puberty is derived from testicular secretion, with the remainder arising from the extra-gonadal conversion of the steroid precursors. In women, circulating levels of testosterone are derived about equally from direct gonadal secretion and indirect peripheral conversion of the precursors.

Testosterone production is not the only pathway for these
Dehydroepiandrosterone (DHEA) → Androstenediol → Androstenedione → Testosterone → Estrone → Estradiol

Pathway for endogenous sex-steroid production.

precursors, as A’dione and testosterone can also be aromatized to estrone and estradiol (Figure).3,7,9 In normal men, all of the estrone and about 85% of the estradiol can be accounted for by formation from A’dione and testosterone at the extragondal sites.10 While the gonadal production of testosterone and estrogen is regulated by a negative-feedback system, the peripheral formation of active androgens and estrogens depends on the quantity of precursors and is not subject to any known physiologic regulation.8

Original Research

Like testosterone, the production of DHEA and A’dione peaks in the midtwenties and then declines steadily with age after the third decade of life.11 This decline stimulated great interest in a potential role for a steroid deficiency state in aging. Thus, original research in precursor supplementation focused on its role in replacement therapies to compensate for the age-related decline in their endogenous production.12-15

The suggested benefits from these replacement therapies included stimulation of immunologic and cardiovascular protection, inhibition of carcinogenesis, lowering of body fat and increase in lean body mass, and promotion of physical and psychological well-being.3,16 In fact, DHEA as a supplement first gained popularity for its proposed health and anti-aging properties and was touted as the “fountain of youth.”

When DHEA was administered (50 mg/d for 3 months) to older men and women (age = 40 to 70 years), DHEA levels were restored to levels found in young adults.13 The women in that study also experienced a 2-fold increase in A’dione and testosterone, while men experienced only a small increase in A’dione. Similar observations were made when larger dosages (100 mg/d) were administered to the same age group over a 6-month period.12 In that study, both sexes experienced increases in DHEA. However, the A’dione and testosterone levels in women reached levels above sex-specific young adult ranges, while no changes were observed in men. Similarly, no changes in testosterone, strength, or lean body mass were observed in a group of men (age = 40 to 60 years) when supplementation with either DHEA or A’dione was combined with resistance training.15 Thus, it appears that only women experience increases in testosterone after precursor supplementation. Finally, Mortola and Yen14 observed 15-, 20-, and 9-fold increases in DHEA, A’dione, and testosterone, respectively, in postmenopausal women (age = 46 to 61 years) after DHEA supplementation (1600 mg/d) for 28 days. Significant increases in estrone and estradiol were also observed. The changes in hormone levels were not associated with any change in lean body mass; however, the subjects did not perform any type of physical training. It is important to note that significant decreases in high-density lipoprotein (HDL) cholesterol were observed in 2 of the previously mentioned studies.13,14

Ergogenic Claims

Increased levels of testosterone have been shown to increase protein synthesis, muscle strength, and lean body mass.17-20 Thus, the role of DHEA and A’dione as testosterone precursors has sparked interest in their potential as ergogenic aids. Results from earlier studies involving an older population have led to the marketing of these products as ergogenic or anabolic supplements capable of increasing testosterone levels and, consequently, lean body mass, strength, and overall athletic performance. As described, the increases in testosterone had only been observed in older women; however, these products are marketed toward young athletic men.

One of the initial studies cited in support of precursor supplementation was published by Mahesh and Greenblatt21 in 1962. The subjects in that study consisted of 4 women (ages were not reported), 2 of whom ingested DHEA while the other 2 ingested A’dione. Although the authors did not perform a statistical analysis, they concluded that ingestion of these supplements increased testosterone levels, with A’dione causing the greater increase. Obviously, with such a small sample, the results are not reliable. It is important to note that women are essentially testosterone deficient when compared with their male counterparts and, as mentioned previously, about 50% of their testosterone is derived from peripheral precursor conversion. A second study commonly cited in support of these supplements involved ovariec­tomized rats.22 After ovariec­tomy, significant reductions in plasma A’dione, testosterone, and estradiol were observed. These levels were regained in a dose-responsive manner when the rats were fed A’dione. These are just 2 examples of what has become common practice among supplement manufacturers, as results from deficiency studies and animal studies (and animal deficiency studies) are generalized to a young, healthy, and athletic population.

Research Involving Young Men

When young, healthy men were recruited as subjects, results similar to those previously observed in older women have not been reproduced.23-30 Research published since 1999 has focused on 2 areas: the acute effects of a single dose and the chronic effects of daily ingestion. A summary of the supple­mentation protocols and results is found in Table 1.

Acute Hormonal Changes

When investigating the acute hormonal response to DHEA ingestion (30 mg) in young men (age = 19 to 29 years), researchers observed significant elevations in serum A’dione, while no changes in testosterone occurred.25 Similar observations have been made after A’dione ingestion.6,23,27,28,30 These elevations occurred after ingestion of 100,27,28,30 200,6,23 and 300 mg.28 However, only 200-mg and 300-mg doses have been shown to significantly increase serum testosterone levels.6,28 Testosterone levels in a group of young men ingesting 300 mg were significantly greater than those ingesting 100 mg and those in a control group, while no differences were observed between the 100-mg and control groups.28 It
was suggested that a significant portion of the ingested A'dione is reduced and conjugated by the liver before it can reach peripheral testosterone-converting tissues. Thus, only higher dosages were able to achieve increases in testosterone. Similarly, a significant increase in testosterone was observed in young men after a 200-mg dose. However, significance was only noted when the area under the curve was used as the measure, as no differences were observed when comparing the individual time points (0, 30, 60, and 90 minutes) used to establish the area under the curve.

It must be noted that acute increases in estradiol have also been observed in young men after A'dione ingestion. Following higher-dose A'dione ingestion (300 mg), a 128% increase in estradiol was observed, while only a 34% increase in testosterone was observed. Although lesser doses (100 mg) have failed to raise testosterone levels, they have been observed to increase estradiol levels. Of 29 subjects ingesting A'dione, 22 experienced estradiol levels above the upper limit of normal for men after ingestion. It was suggested by the authors that the local tissue levels of estradiol may have been even greater than those measured in the plasma because aromatase, the enzyme that converts A'dione and testosterone to estrone and estradiol, is found in many human tissues, including muscle and fat.

### Table 1. Effects of Precursor Supplementation on Hormone Levels*

<table>
<thead>
<tr>
<th>Reference</th>
<th>Subject Population</th>
<th>Supplement Protocol</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brown et al25</td>
<td>Healthy males (19 to 29 y)</td>
<td>DHEA 50-mg single dose</td>
<td>A'dione increased. No change in testosterone or estradiol.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>DHEA 150 mg/d for 8 weeks</td>
<td>A'dione increased at weeks 2 and 5 only. No change in testosterone or estradiol.</td>
</tr>
<tr>
<td>Morales et al27</td>
<td>Healthy males (53.7 ± 2.5 y)</td>
<td>DHEA 50 mg/d for 3 months</td>
<td>DHEA and A'dione increased. Testosterone increased in females only.</td>
</tr>
<tr>
<td></td>
<td>Healthy females (54.5 ± 1.9 y)</td>
<td>DHEA 100 mg/d for 6 months</td>
<td>DHEA and A'dione increased (greater increase in females). Testosterone increased in females only. No change in estradiol.</td>
</tr>
<tr>
<td>Morales et al28</td>
<td>Healthy males (55.6 ± 1.9 y)</td>
<td>DHEA 100 mg/d for 6 months</td>
<td>A'dione, A'dione, and testosterone increased.</td>
</tr>
<tr>
<td>Mortola and Yen14</td>
<td>Overweight women (46 to 61 y)</td>
<td>DHEA 400-mg single dose</td>
<td>A'dione increased. No change in testosterone.</td>
</tr>
<tr>
<td>Nestler et al29</td>
<td>Healthy males (22 to 25 y)</td>
<td>DHEA 1600 mg/d for 28 days</td>
<td>DHEA, A'dione, and testosterone increased. HDLs decreased.</td>
</tr>
<tr>
<td>Welle et al31</td>
<td>Healthy males (20 to 43 y)</td>
<td>DHEA 1600 mg/d for 4 weeks</td>
<td>DHEA and A'dione increased. No change in testosterone, estrone, or estradiol.</td>
</tr>
<tr>
<td>Brown et al26</td>
<td>Healthy males (19 to 29 y)</td>
<td>DHEA 150 mg, A'dione 100-mg, and tribulus 250-mg single dose</td>
<td>A'dione increased. No change in testosterone, estrone, or estradiol.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>DHEA 150 mg, A'dione 300 mg, and tribulus 750 mg* for 8 weeks (2 wk on/1 wk off)</td>
<td>A'dione, estrone, and estradiol increased. No change in testosterone.</td>
</tr>
<tr>
<td>Wallace et al36</td>
<td>Resistance trained males</td>
<td>DHEA 100 mg/d for 12 weeks</td>
<td>A'dione, estradiol, and estrone increased. No change in testosterone.</td>
</tr>
<tr>
<td></td>
<td>(48.1 ± 3.9 y)</td>
<td>A'dione 100 mg/d for 12 weeks</td>
<td>A'dione increased. No change in testosterone.</td>
</tr>
<tr>
<td>Ballantyne et al37</td>
<td>Resistance-trained males</td>
<td>A'dione 100 mg/d for 2 days</td>
<td>A'dione increased. No change in testosterone.</td>
</tr>
<tr>
<td></td>
<td>(24 ± 0.6 y)</td>
<td>A'dione 100-mg single dose</td>
<td>A'dione increased. No change in testosterone.</td>
</tr>
<tr>
<td>King et al37</td>
<td>Resistance-trained males</td>
<td>A'dione 300 mg/d for 8 weeks (2 wk on/1 wk off)</td>
<td>A'dione, estradiol, estrone, and estradiol increased. No change in testosterone.</td>
</tr>
<tr>
<td></td>
<td>(19 to 29 y)</td>
<td>A'dione 200 mg/d for 12 weeks</td>
<td>A'dione, estradiol, estrone, and estradiol increased. No change in testosterone.</td>
</tr>
<tr>
<td>Leder et al38</td>
<td>Healthy males (20 to 40 y)</td>
<td>A'dione 100 mg/d for 7 days</td>
<td>A'dione and estradiol increased. No change in estrone or testosterone.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A'dione 300 mg/d for 7 days</td>
<td>A'dione and estradiol increased. No change in testosterone.</td>
</tr>
<tr>
<td>Rasmussen et al30</td>
<td>Healthy males (32.0 ± 4.0 y)</td>
<td>A'dione 100-mg single dose</td>
<td>A'dione and estradiol increased. No change in testosterone.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A'dione 100 mg/d for 5 days</td>
<td>A'dione and estradiol increased. No change in testosterone.</td>
</tr>
<tr>
<td>Broeder et al31</td>
<td>Healthy males (35 to 65 y)</td>
<td>A'dione 100 mg/d for 7 days</td>
<td>No change in A'dione, estradiol, or testosterone. No change in muscle protein synthesis.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A'dione 200 mg/d for 12 weeks</td>
<td>A'dione, estrone, and estradiol increased. No change in testosterone. HDLs decreased.</td>
</tr>
<tr>
<td>Earnest et al36</td>
<td>Resistance-trained males</td>
<td>A'dione 200-mg single dose</td>
<td>A'dione and testosterone increased (area under the curve only).</td>
</tr>
<tr>
<td></td>
<td>(23.8 ± 3.0 y)</td>
<td>A'diol 200 mg/d for 12 weeks</td>
<td>No change in A'dione or testosterone</td>
</tr>
</tbody>
</table>

* DHEA indicates dehydroepiandrosterone; A'dione, androstenedione; HDLs, high-density lipoproteins; and A'diol, androstenediold.
Chronic Hormonal Changes

When DHEA and A’dione were administered daily to young men using protocols ranging from 2 days to 8 weeks, no changes in resting (before ingestion) testosterone, regardless of dose, have been observed.23,25–30 In a recent study, Rasmussen et al30 showed that 100 mg of A’dione per day for 5 days failed to increase testosterone levels. However, plasma A’dione did increase more than 3-fold. Similarly, 300 mg/d (100-mg doses) for 8 weeks failed to increase testosterone levels, while A’dione levels were elevated by 100%.27 Even a 300-mg daily dose, which did provide an acute increase in testosterone, failed to increase baseline testosterone after 7 days of ingestion.28

Similar to the acute changes after A’dione ingestion, significant increases in estradiol have been observed in young men after chronic ingestion.23,26,27 Although testosterone levels did not change, 300 mg/d produced significant increases in both estrone and estradiol levels.27 Even when precursors were combined with herbal extracts designed to reduce the conversion of androgens to estrogens, significant increases in estrone and estradiol were observed.26

Because of concerns regarding the increases in estrogens observed in men after DHEA and A’dione ingestion, there has been an increase in the number of products containing A’diol. Like A’dione, A’diol is converted from DHEA and is an immediate precursor to testosterone. Unlike A’dione, A’diol cannot be directly converted to estrogen, as it must first be converted to testosterone, which can then be converted to estradiol. However, as in the previous investigations, a single 200-mg dose of A’diol failed to increase serum testosterone levels in young men.9 Also, supplementation with A’diol (200 mg/d) for 12 weeks failed to produce changes in testosterone levels.24 This protocol did produce a significant increase in serum estrone and estradiol concentrations, suggesting that the ingested A’diol is converted to testosterone, which is immediately aromatized to estradiol.

Protein Synthesis

As mentioned previously, ergogenic claims are based on the theory that precursor ingestion will result in increased testosterone levels, which would then stimulate an increase in muscle protein synthesis. However, at this time, there is no scientific support for this theory, as both DHEA31 and A’dione30 ingestion have failed to increase protein synthesis in groups of young men. Furthermore, 8 weeks of A’dione supplementation (300 mg/d) and resistance training failed to increase muscle-fiber cross-sectional area when compared with placebo ingestion and training.27

Androstenedione is a steroid possessing 10% to 20% of the androgenic activity relative to testosterone. Thus, the conversion of A’dione to testosterone may not be necessary to have an anabolic effect. However, this theory also lacks support, as A’dione ingestion failed to affect muscle protein synthesis or muscle-fiber cross-sectional area, even though significant increases in serum A’dione were observed.27,30

Lean Body Mass and Strength

If precursor supplementation elevated testosterone levels, increases in muscle strength and lean body mass, such as those observed after testosterone administration, would be expected. A summary of the performance studies involving precursors and the results is found in Table 2. Nestler et al29 were the first to report anthropometric changes in young men (age = 20 to 25 years) after supplementation, as a significant decrease in body fat was observed after DHEA ingestion (1600 mg/d) for 28 days.29 Because overall body mass remained constant while fat mass decreased, the authors concluded that supplementation resulted in increased lean body mass. However, strength measures were not performed, and diet and physical activity were not controlled or recorded. Thus, the authors’ conclusions must be questioned.

Other than the previously mentioned study, the ergogenic claims regarding precursor supplementation have not been

Table 2. The Effects of Precursor Supplementation on Performance*

<table>
<thead>
<tr>
<th>Reference</th>
<th>Subject Population</th>
<th>Supplement Protocol</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brown et al12</td>
<td>Healthy males (53.7 ± 2.5 y)</td>
<td>DHEA 150 mg/d for 8 wk</td>
<td>No change in lean body mass or strength.</td>
</tr>
<tr>
<td>Morales et al13</td>
<td>Healthy females (54.5 ± 1.9 y)</td>
<td>DHEA 50 mg/d for 3 mo</td>
<td>No change in lean body mass.</td>
</tr>
<tr>
<td>Mortola and Yen14</td>
<td>Overweight females (46 to 61 y)</td>
<td>DHEA 100 mg/d for 6 mo</td>
<td>No change in lean body mass.</td>
</tr>
<tr>
<td>Nestler et al22</td>
<td>Healthy males (22 to 25 y)</td>
<td>DHEA 1600 mg/d for 28 d</td>
<td>Decreased body fat.</td>
</tr>
<tr>
<td>Welle et al21</td>
<td>Healthy males (20 to 43 y)</td>
<td>DHEA 1600 mg/d for 4 wk</td>
<td>No change in lean body mass or protein synthesis.</td>
</tr>
<tr>
<td>Brown et al26</td>
<td>Healthy males (19 to 29 y)</td>
<td>DHEA 150 mg, A’dione 300 mg, and tribulus 750 mg for 8 wk (2 wk on, 1 wk off)</td>
<td>No change in lean body mass or strength.</td>
</tr>
<tr>
<td>Wallace et al16</td>
<td>Resistance-trained males (48.1 ± 3.9 y)</td>
<td>DHEA 100 mg/d for 12 wk</td>
<td>No change in lean body mass or strength.</td>
</tr>
<tr>
<td>King et al27</td>
<td>Resistance-trained males (19 to 29 y)</td>
<td>A’dione 100 mg/d for 12 wk</td>
<td>No change in lean body mass or strength.</td>
</tr>
<tr>
<td>Broeder et al24</td>
<td>Healthy males (35 to 65 y)</td>
<td>A’dione 300 mg/d for 12 wk (2 wk on, 1 wk off)</td>
<td>No change in lean body mass or strength.</td>
</tr>
</tbody>
</table>

*DHEA indicates dehydroepiandrosterone; A’dione, androstenedione; and A’diol, androstenediol.
supported. For example, 4 weeks of DHEA supplementation failed to increase lean body mass in a group of young, healthy men. Testosterone levels were not measured in that study; thus, whether increases occurred or not is unknown. However, the same supplementation protocol also failed to affect lean body mass in a group of postmenopausal women. Although no training or performance testing occurred, a significant increase in testosterone was observed in that group. It is possible that 4 weeks of ingestion were insufficient to observe remarkable body-composition changes, although changes have been observed after just 4 weeks of testosterone administration.

Yet in a recent study, 8 weeks of A'dione ingestion combined with resistance training also failed to increase muscle strength and lean body mass in a group of young men (age range = 19 to 29 years). Although both acute and long-term administration (100 mg/d) failed to increase testosterone levels, A'dione levels did increase. Again, no changes in strength or lean body mass were observed in a similar age group after 8 weeks of DHEA ingestion and resistance training.

Longer supplementation and training protocols have also failed to support ergogenic claims. Twelve weeks of DHEA and A'dione supplementation combined with resistance training were ineffective in increasing lean body mass and strength in an older group of men (age range = 40 to 60 years). Similarly, supplementation with A'diol (200 mg/d) for 12 weeks also failed to produce changes in muscle strength and lean body mass. Previous studies have shown that 12 weeks of training provides sufficient time for remarkable training effects to occur. Thus, the ergogenic claims must be questioned. The failure to increase strength or lean body mass further suggests that exogenous DHEA and A'dione must first be converted to testosterone to achieve an anabolic effect.

**Side Effects**

Significant reductions in serum HDL cholesterol of 12% and 20% have been observed after A'dione and DHEA supplementation, respectively. Similar changes have been observed after AAS injection and have been associated with the development of cardiovascular disease. Broeder et al administered either A'dione or A'diol (200 mg/d) and observed that both adversely affected HDL-cholesterol levels, low-density lipoprotein (LDL)-to-HDL cholesterol ratios, and coronary heart disease risk. Thus, it is possible that long-term supplementation could have serious side effects similar to those associated with AAS use, such as suppressed testosterone production, liver dysfunction, cardiovascular disease, testicular atrophy, male-pattern baldness, acne, and aggressive behavior. If the supplements are taken before puberty, premature closing of the epiphysis and stunted growth could occur. In women, precursor-induced increases in testosterone concentrations could cause lowered voice pitch, hirsutism (changes in hair growth patterns, including facial hair), increased abdominal fat accumulation, and general virilization. Furthermore, increases in estrogen concentrations experienced by men could have feminizing effects, including gynecomastia.

**CONCLUSIONS**

Although DHEA, A'dione, and A'diol are in fact steroids, they are not classified as anabolic steroids or controlled substances by either the Food and Drug Administration or the Drug Enforcement Agency. The Anabolic Steroid Control Act of 1990 defines an anabolic steroid as any drug or hormonal substance chemically and pharmacologically related to testosterone, other than estrogens, progestins, and corticosteroids, that promotes muscle growth. Under that act, testosterone and anabolic steroids are classified as Schedule III drugs and cannot be sold over the counter or possessed without a prescription. Although DHEA, A'dione, and A'diol are structurally and pharmacologically related to testosterone, they have not been proven to promote muscle growth and are, therefore, not classified as Schedule III drugs. Furthermore, the Dietary Supplement Health and Education Act of 1994 has allowed these products to be sold legally over the counter as natural supplements in the United States.

Unfortunately, the term “natural supplement” implies that it is safe; however, this is not always true. Over-the-counter availability and unrestrained self-medication with steroid precursors create a heightened potential for serious side effects, and the safety of these products must be questioned, as there are no human studies in the medical literature on their long-term safety. Unfortunately, most companies that manufacture and sell nutritional supplements are profit driven and are often misleading with their advertising. One of the primary concerns is that manufacturers are not required to list the ingredients on the labels of natural supplements; thus, the consumer does not always know the true contents of the product. In an analysis of 9 brands of A'dione, 6 contained less than 90% of the amount stated on the label, 1 contained no A'dione, and 1 was actually found to contain 10 mg of testosterone. Furthermore, in the same study, 20 of 24 men ingesting A'dione (100 or 300 mg/d) would have tested positive for the banned steroid nandrolone based on levels of 19-norandrosterone (a metabolite of nandrolone) found in the urine. Thus, these men were ingesting a supplement and urinating a drug.

Whenever an athlete is considering using steroid precursors or any ergogenic supplement, 3 questions must be asked: is it safe, does it work, and is it legal? At this time, scientific support for the ergogenic or anabolic use of steroid precursors does not exist. However, numerous studies provide evidence of the possible dangers associated with them, including increased risk of heart disease and increased estrogen concentrations in men. Thus, it appears that the risks far outweigh the benefits. It is also important to note that the International Olympic Committee, the National Collegiate Athletic Association, and the National Football League have banned the use of steroid precursors, and the use of these supplements can be detected in drug tests. The key to performance is a healthy diet and a well-developed training program; there is no “quick fix” or “shortcut to success.” As allied health professionals, it is important that athletic trainers are able to educate athletes regarding the efficacy and safety of nutritional supplements, so they may continue to perform at an optimal level in a safe and healthy manner.

**REFERENCES**

Mechanisms and Management of Stress Fractures in Physically Active Persons

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Objective: To describe the anatomy of bone and the physiology of bone remodeling as a basis for the proper management of stress fractures in physically active people.

Data Sources: We searched PubMed for the years 1965 through 2000 using the key words stress fracture, bone remodeling, epidemiology, and rehabilitation.

Data Synthesis: Bone undergoes a normal remodeling process in physically active persons. Increased stress leads to an acceleration of this remodeling process, a subsequent weakening of bone, and a higher susceptibility to stress fracture. When a stress fracture is suspected, appropriate management of the injury should begin immediately. Effective management includes a cyclic process of activity and rest that is based on the remodeling process of bone.

Conclusions/Recommendations: Bone continuously remodels itself to withstand the stresses involved with physical activity. Stress fractures occur as the result of increased remodeling and a subsequent weakening of the outer surface of the bone. Once a stress fracture is suspected, a cyclic management program that incorporates the physiology of bone remodeling should be initiated. The cyclic program should allow the physically active person to remove the source of the stress to the bone, maintain fitness, promote a safe return to activity, and permit the bone to heal properly.

Key Words: bone remodeling, rehabilitation, stress reaction

Stress fractures can occur in any physically active person. As a result, athletic trainers and sports therapists need to understand the injury mechanism and strategies for management. We describe the incidence, latest theories of causation, and a protocol for the management of stress fractures based on the physiology of bone remodeling. We also describe the incidence of stress fractures, distribution of forces to bone, normal and abnormal bone anatomy and remodeling, and proposed risk factors for stress fractures in a physically active population.

INCIDENCE

Stress fractures occur in several different bones. The distribution of stress fractures differs according to activity. The tibia is reported to be the most frequently injured bone in runners, followed by the fibula, metatarsal, and pelvis (Table 1). Fifteen percent of all stress fractures occur in runners, accounting for 70% of all of their injuries. In dancers, the metatarsal is the most common location of injury. Stress fractures in the ribs have been described in golfers, and stress fractures of the pars interarticularis are prevalent in racket sports and basketball players.

Different study designs, populations, and classification schemes make it difficult to definitively report the incidence of stress fractures in varying populations. Some trends exist in the incidence of stress fractures between the sexes and among the races. In military populations, women are more likely to sustain stress fractures. In athletes, however, the disparity between the sexes is not as conclusive. Whereas Hickey et al found differences between athletic men and women that were similar to those in military populations, others have reported that female collegiate athletes have a similar or only slightly higher rate of injury than men.

A disparity also exists in the incidence of stress fractures among the races. In the military, white men and women have shown a higher incidence of stress fractures than African Americans or Hispanics. One explanation for this difference may be the lower overall bone density in whites as compared with the other 2 groups.

DISTRIBUTION OF FORCES TO BONE

A stress fracture is a partial or incomplete fracture caused by the accumulation of stress to a localized area of bone. Stress fractures are not the result of one specific insult. Instead, they arise as the result of repetitive applications of stresses that are lower than the stress required to fracture the bone in a single loading.

Bone endures a stress whenever a force is loaded upon it. Whether the stress comes from the pull of a muscle or the shock of a weight-bearing extremity contacting the ground, it
is defined as the force applied per unit area of the load-bearing bone.\textsuperscript{7,22} Low levels of these forces cause bone to deform,\textsuperscript{23} which is known as strain.\textsuperscript{7} The bone's stress-strain response depends on the load's direction; the bone's geometry, microarchitecture, and density; and the influence of surrounding muscular contractions.\textsuperscript{7} In most activities of daily living (ADLs), when the force is removed, the bone elastically rebounds to its original position. The force that a bone can endure and still rebound back to its original state without damage is within the elastic range.\textsuperscript{17,23,24} Forces that exceed a critical level above the elastic range are in the plastic range.\textsuperscript{20,22} Once forces reach the plastic range, a lower load causes greater deformation; it is at this level that forces summate to permanently damage the bone.\textsuperscript{25,26}

Forces can be applied to bone through compression, tension, bending, torsion, or shear.\textsuperscript{7} Compression forces are generally seen in cancellous bones, such as the calcaneus and femoral neck. Tension forces, however, result in bone pulling away from bone, as is common in compact bones such as the tibia and femur. As the load is applied to the bony shaft through a bend, a tension strain is placed upon the convex surface of the shaft and compressive forces act on the concave side (Figure 1).\textsuperscript{24}

The muscles attached to the surface of compact bones can help to increase or decrease the intensity of a load.\textsuperscript{7} The muscular attachments on the surface of compact bones can produce a tension force that acts circumferentially or acts as a shock absorber by controlling bone strain.\textsuperscript{28,29} In cases of excessive muscular pull, a stress fracture may develop near the bone-tendon junction. This mechanism is common in non-weight-bearing bones such as the ribs and fibula.\textsuperscript{5,6} Conversely, weakness or fatigue in the shock-absorbing muscles may allow for an increased load to be translated to the bone, making it more susceptible to stress fracture.\textsuperscript{3}

**Anatomy**

Bone has both cortical and cancellous components. Cortical bone is dense and highly organized and withstands stress in compression better than in tension.\textsuperscript{7} Cancellous (trabecular) bone is an irregularly shaped meshwork and withstands stress according to the alignment of the fiber matrix.\textsuperscript{32} The outer shafts of long bones (eg, tibia, humerus) are mainly cortical, with a large percentage of cancellous bone making up the ends of the bone and the central portion of the shaft.\textsuperscript{16} Short and flat bones such as the tarsals and pelvis have a higher content of cancellous bone.

The fundamental unit of cortical bone is the osteon. In the osteon, concentric layers of lamellar bone surround small channels called haversian canals. These canals house nerves and blood vessels. On the outside of the lamellae are small cavities, known as lacunae. Each lacuna contains a single bone cell, or osteocyte. Canaliculi form a transport system between the lacunae and the haversian canals that is responsible for the nutrition and metabolic transport system within the bone.\textsuperscript{7,33}

Surrounding the outer surface of long bones is a highly vascular outer coating called the peristeum. The peristeum is responsible for providing nutrition to the outer portion of the cortex and enlarges during remodeling to provide support to the cortex. On the inner portion of the cortex, medullary canals allow the vascular passage for nutrients and blood vessels to the inner two thirds of the cortex (Figure 2).\textsuperscript{34}

**Remodeling**

Bone constantly remodels itself to more efficiently endure external forces.\textsuperscript{35,36} According to column law, the magnitude of stress is greatest on the surface of a column and decreases to zero at the center. Accordingly, most of the remodeling in long bones takes place in the outer cortex.\textsuperscript{37} Remodeling involves the resorption of existing bone by osteoclasts and the formation of new bone cells by osteoblasts.\textsuperscript{22,23,38,41} Participating in regular activity promotes bone strength through proper perfusion of nutrients to the osteocytes and normal bone remodeling. Conversely, a sedentary lifestyle contributes to bony atrophy.\textsuperscript{35,36,42,44} In order to begin remodeling, osteoclastic cells need to be activated. The piezoelectric effect is one mechanism implicated in the activation of bone remodeling.\textsuperscript{45,46} Tension forces create a relative electropositivity on the convex, or tension side, of the bone. This increase in positive charge is conducive to osteoclastic resorption.\textsuperscript{29,45,47} Thus, as torque or bending produces repeated distraction forces at a focal point of a bone, the electropositive charge may stimulate osteoclastic absorption.

The streaming effect is the movement of extracellular fluids in the haversian canals and canaliculi during deformation. If the surface charge on the haversian canal or canaliculi walls
is positive, negative ions in the fluid are attracted to the outside of the fluid stream, creating a positively charged current in the middle. As bone is bent, the positive stream is forced toward the bone's open, or distracted, surface. The electropositive stream may, in turn, stimulate osteoclastic activity. Other possible activators are bone "sensors" that recognize increased and decreased mechanical strains, hormones, decreased venous flow, and decreased oxygen.

Upon activation, osteoclastic cells form a cone and begin to secrete proteolytic enzymes to cut longitudinal tunnels through the bone. These new haversian canals are aligned with the stresses placed on the bone. Each osteoclast cone can resorb nearly 3 times its volume in burrowing a canal from 3 to 10 mm deep. The new haversian canals are filled with osteoblasts that create a mineralized matrix that supports the walls of the new channel. The remaining space of the channel is then filled with immature lamellar bone.

Haversian canal formation and osteoblast support with lamellar bone begins 10 to 14 days after the onset of remodeling. The conversion of lamellar bone into mature osteocytes cells lags behind resorption by about a week and may continue for as long as 20 to 90 days. The result is a temporary weakened bone due to the new, hollow haversian canals. The inflammation of periostium is designed to bolster the weakened area of bone until it can mature. However, the periostium does not mature until about 20 days after the remodeling process begins. This 6- to 10-day lag between the deposit of immature lamellar bone and periosteal maturity leaves the bone temporarily weakened at the point of stress during the third week of remodeling. Continued stress applied to remodeling bone during the "weak third week" may lead to an accelerated breakdown of the cortex. It is at this time that a stress fracture is most likely to develop.

STRESS FRACTURES

Bone's response to stress has been confused in the literature by several different names and classification schemes. The terms shin splints, medial tibial stress syndrome, and medial tibial stress syndrome are often used interchangeably to describe the symptoms and radiologic findings commonly associated with advanced bone remodeling and tibial stress fractures. Currently, bone's response to stress is evaluated on a dynamic continuum between early remodeling and periostitis to a cortical stress fracture. It is important to note that the changes associated with bone's reaction to stress (eg, stress reaction) reflect a wide spectrum of physical findings and radiographic presentations.

A true stress fracture is a visible cortical fracture. Stress fractures have traditionally been classified into 2 types: fatigue and insufficiency. The fatigue fracture is caused by an abnormal stress on a bone that is mineral deficient or abnormally inelastic. Insufficiency fractures arise from the application of a normal stress on a bone that is mineral deficient or abnormally inelastic. Insufficiency fractures are most prevalent in nutrient-deficient (osteomalacia) and older populations in whom osteoporosis and rheumatoid arthritis are more common.

The fatigue fracture is more common in the physically active population. The abnormal forces that cause a deterioration of healthy bone may result from increased training intensity, hard training surfaces, worn or inappropriate shoes, or poor anatomical alignment of the feet. Muscular and aerobic capacity improve within the first week of an exercise regimen. The result is an increase in exercise duration and pull of stronger muscles on bones that are still in a weakened phase of remodeling.

Until recently, the cause of stress fractures was thought to be due to the breakdown of bone after repetitive loading. It has been estimated that, at normal physiologic levels of strain, it would require 10^8 cycles of loading to produce failure of a weight-bearing bone such as the tibia. This level of loading is not easily attained, and stress fractures commonly occur soon after the onset of a stressful activity. Greaney et al found that 64% of the stress fractures in a military population began within the first 7 days of training. The rapid onset of symptoms and bone remodeling consistent with stress fracture suggest that mechanical stress cannot be the only cause.

Otter et al proposed that the perfusion and reperfusion of bone after a repetitive load causes a temporary oxygen debt to the area of bone being stressed. This ischemia, in turn, facilitates bone remodeling and subsequent bone weakness and
stress fracture. When a bone is loaded to normal physiologic levels, the small blood vessels that supply the cortex are squeezed.\(^43\) In most cases, this pressure is necessary for proper movement of the blood.\(^42\) When the load is higher, the blood flow may be temporarily cut off. The result is a brief period of ischemia in the cells that would normally be perfused by the compressed medullary vessels. Repeated loads over a prolonged period of an activity, such as a long run, cut off the oxygen during that period as well. This decrease in oxygen to the bone is believed to trigger the remodeling process.\(^42\) In fact, Kelly and Bronk\(^49\) found that restricting venous flow without any mechanical loading was enough to stimulate bone remodeling. In the above scenario, blood flow and oxygen perfusion are both restricted. This restriction is believed to signal the bone to remodel and cause the osteocytos to channel into the bone. The result is a weakened bone that is less able to withstand subsequent loads (Figure 3).\(^51\)

The temporary lack of oxygen is not the only cause of ischemia. Repeated pressure to the capillaries is also believed to cause microdamage to the vessels. As neutrophils respond to plug the damaged capillaries, the blood flow through the vessels is further restricted.\(^68\) In addition, small leaks in the vessels allow fluid flow into the surrounding tissue, further restricting the perfusion of oxygen into the cells. This leaking increases with subsequent bouts of loading, worsening ischemia and triggering a further increase in remodeling.\(^67\) The repetition of this cycle causes an increase in remodeling, a breakdown in the cortex, a weakening of the bone, and potentially a stress fracture (Figures 4 and 5).

Ischemic mechanisms of tissue damage are common in other athletic injuries. For example, ice and compression are routinely used after an ankle sprain to limit effusion and secondary hypoxic injury. In this case, fluids from the damaged blood vessels in the anterior talofibular ligament allow leakage into the surrounding tissue. This excess fluid decreases oxygen tension and restricts oxygen perfusion to the adjacent cells. The result is damage to the ligament from the initial injury and damage to the tissue adjacent to the ligament from a lack of oxygen.

**Risk Factors**

Several risk factors exist for insufficiency and fatigue stress fractures. Because weakened bone is susceptible to insufficiency stress fractures, populations with mineral-deficient conditions such as rickets or osteomalacia may also have bones that are unable to withstand normal forces. Moreover, normally strong bones may be weakened by cysts or surgical or medical procedures, such as screw fixation, tendon transfer, joint arthroplasty, bunionectomy, or radiation treatment.\(^19\)

The unique nutritional demands of women place them at a higher risk for insufficiency stress fractures than men. Fredericson et al\(^60\) found that stress fractures occurred more often in women, while Ha et al\(^6\) found that the highest incidence of stress fractures was in teenage girls. One explanation for this difference may be the female athlete’s susceptibility to the female athlete triad of eating disorders, amenorrhea,\(^69\) and osteoporosis.\(^18\) These findings are supported by a 12-month, prospective study of 53 female and 58 male track athletes: lower bone density, less lean body mass in the lower limb, a low-fat diet, and a history of menstrual disturbance in the female athletes were significant risk factors for stress fractures.\(^70\)

Several authors\(^17,63\) suggested that increased pronation is common among athletes with stress fractures of the lower extremity. Similarly, rigid cavus feet are a common predisposing factor to tarsal and femoral stress fractures.\(^3\) Hard surfaces or inappropriate shoes may exaggerate these conditions.

Even though poor foot alignment or muscle imbalances may contribute to the onset of a stress fracture, some type of change is the common ingredient in most diagnoses.\(^20,24,37,40,55,71\) This change may be an increase in the intensity or type of exercise or a change in playing surfaces or footwear. Any of these changes may create an increase in stress to the bone and a subsequent increase in the rate of remodeling. Goldberg and Pecora\(^3\) found that 67% of 38 stress fractures in college varsity athletes were in freshmen who may have been experiencing changes in training intensity at the collegiate level.
R. Removal of the abnormal stress
E. Exercise to maintain cardiovascular fitness and prevent atrophy
S. Safe, pain-free return to previous level of activity
T. Time for bone maturity to catch up with increased remodeling

Figure 6. R.E.S.T. acronym for the goals of stress fracture management.

MANAGEMENT

Prompt identification of an abnormal reaction to stress, such as a stress fracture, is essential. Once diagnosed, the injury can be managed with a cyclic management protocol based on the physiology of bone remodeling and a strategy for prevention.

Diagnosis

Prompt diagnosis of stress fractures is important, as continuing the aggravating activity may delay management and increase morbidity. Very often, symptoms resembling those of a stress fracture are actually due to advanced bone remodeling resulting from the bone’s reaction to stress. This stress reaction may only be a point along the continuum of remodeling before the development of a true stress fracture. The clinician often intervenes at this stage of the continuum to prevent the progression of the injury to a true stress fracture. In patients with a true stress fracture, prompt intervention is important to minimize the risk of a displaced fracture. This intervention may include casting, splinting, or surgical fixation.

Diagnosing stress fractures can be difficult as their symptoms are comparable with other injuries. Common diagnostic techniques include clinical examination, x-ray films, bone scan, magnetic resonance imaging, and ultrasound. Differential diagnoses include shin splints, osteomyelitis, compartment syndrome, and tumor.

Management

Management begins immediately after an abnormal reaction to stress or a stress fracture is suspected. Since an x-ray film may not be positive for 10–21 days after the onset of symptoms, a delay in intervention may allow the accelerated remodeling to progress to a true stress fracture, thus risking a full fracture of the bone. The first priority is a period of rest from the stress or activity that is causing the symptoms. Zelko and DePalma described the rest as “active,” allowing the athlete to exercise in a pain-free manner and prevent muscle atrophy. Pain should be used as a guideline to treatment intensity, as pain during an activity may indicate exacerbation at the injury site. The goals during active rest are described by the acronym R.E.S.T (Figure 6).

Management of a stress reaction or stress fracture should include a 3-phase process that takes advantage of the physiologic healing process of the bone. Phase I should allow time for the maturing of the periosteum, healing of damaged blood vessels to prevent ischemic injury to bone, and maturing of osteocytes. Phase II should include general conditioning and strengthening specific to the injured extremity. Functional weight bearing in phase III should allow for gradual remodeling of the bone and a return to the original level of activity. This 3-phase protocol differs from other 2-phase protocols that call for a removal of the stress and a gradual increase in activity. In the 3-phase protocol, gradually increased stress in phase III is alternated with periods of rest to let new osteocytes and periosteum mature during periods of remodeling, when the bone is weakest (Table 2).

Several factors affect the management progression. The location, type, and age of the lesion make some exercises easier than others. It is important that the patient progress on the basis of symptoms and physiology rather than on a predetermined schedule. The exercises described within the 3 phases are not exclusive from one phase to the next. Instead, they are expected to overlap and serve as a guideline for the management progression. Because the clinician is often intervening before a true stress fracture develops, the condition that is being treated is usually a stress reaction. This term will be used throughout the discussion of the management.

Phase I. Phase I of the management process focuses on removing the stress from the injured area, controlling pain, and preventing deconditioning. It is during this phase that the haversian canals are forming, the osteoblasts are laying down new cells, and the periosteum is maturing to buttress the weakened area of bone. This phase usually lasts for 1 to 3 weeks or until acute symptoms no longer occur with normal activities. Casting may be indicated when the physically active individual cannot or will not avoid the antagonistic stressor or a true stress fracture is present. However, casting should not be used regularly as it may contribute to a further weakening of the bone and deconditioning of the surrounding soft tissue. Crutch walking is a preferable alternative to casting, as it allows for nonstressful exercise and weight bearing. The use of pneumatic splints may reduce abnormal tibial loading, provide
support around the fracture site, and reduce the length of the rehabilitation process. If poor foot alignments are present, orthotics should be instituted at this juncture to correct them.

A typical phase I protocol for an involved lower extremity should include daily ice massages or contrast baths to decrease swelling. Transcutaneous electric stimulation (TENS) and high-volt electric stimulation (HVES) are also excellent modalities for reducing swelling and pain and may be augmented by nonsteroidal anti-inflammatory medications. These modalities may be especially useful in light of new findings regarding the potential role of inflammation in an ischemic mechanism of stress reactions. Further research is needed to determine the efficacy of anti-inflammatory modalities, including ultrasound, electric stimulation, and ice, in decreasing the inflammation that accompanies bone remodeling.

Ambulation should progress from crutch walking to full weight bearing as soon as it can be tolerated without pain. Conditioning of the involved lower extremity begins daily with towel toe curls, ankle isometrics, and sitting range of motion on a wobble board. As long as the patient remains free of pain, exercises can be progressed by adding weight to the towel curls and allowing active-range strengthening with rubber tubing. Strength training for the upper extremity and well-leg conditioning should continue 3 times a week while cardiovascular fitness can be maintained by using the upper body ergometer or stationary bicycle or treading water in the deep tank of the pool.

**Phase II.** Phase II of the management program begins when phase I exercise or ADLs can be performed without inflammation or symptoms. In many cases, pain is an indication of overload to the bone, but this is not always the case. As a result, patients must be instructed to keep their activity within a pain-free intensity and report any recurrence of pain to their therapist. Caution in using modalities must be exercised in this stage, as they can mask the pain that signals a potentially harmful stress to the injured area. Ice is continued, but ice, TENS, and HVES should be used only after exercise to avoid masking any pain the treatments might be causing.

Pool training that progresses from treading water in the deep tank to jogging in chest-deep water should be added to the swimming workouts. Wobble-board exercises should begin to include weight bearing and balancing, and rubber tubing exercises should progress to bilateral- and eventually single-leg toe raises. Pain-free walking during ADLs must continue (otherwise the patient should return to phase I), and the patient should eventually walk without pain for 30 consecutive minutes, 3 times a week.

**Phase III.** After 2 weeks of pain-free exercise in phase II, the running and functional activities of phase III are introduced. The efficacy of a cyclic training program to prevent stress fractures in military recruits has been documented. By limiting the number of repetitive, high skeletal stresses in the first 2 weeks of basic training and modifying activity in the third week to exclude running, jumping, and double-time exercises, the fracture rate was significantly reduced from 4.8% to 1.6%. Scully and Besterman hypothesized that the initial 2 weeks of training promoted the formation of osteonized new bone, whereas rest in the third week allowed for the formation of periosteal new bone. In the same way that Scully and Besterman used a cyclic training process to strengthen bone and prevent stress fractures, Zelko and DePalma described a cyclic management strategy to facilitate normal bone remodeling in preparation for the person's return to activity after a stress fracture.

Phase III of the management process depends on the physically active person's completion of the activities in a pain-free manner. The patient must be asymptomatic in the previous phases of treatment and cleared by the physician before initiating this functional phase of the program. Running and functional activity start slowly and should be based on the individual's goals for return to function. A good guideline is to increase activity no more than 15% to 20% per week. A "walk-jog" in which the injured person jogs the straightaways and walks the curves of a track for 0.80 km (0.5 mile), followed by a day of rest, is a good starting point for a person who hopes to return to a running, field, or court sport. Once that distance is completed without pain, the injured person can begin walk-jogs 3 times per week. Distance is added in 0.80-km (0.5-mile) increments per week until the athlete can complete 3.22 km (2 miles). At this point, jogging begins for 1.61 km (1 mile) and increases by 0.80 km (0.5 mile) per week until 4.83 km (3 miles) or a goal distance commensurate with the person's activity is reached. During the functional phase of the program, the athlete continues the phase II exercises and progresses to mobility and jumping activities in the pool and on land. Once the athlete can squat 1½ times body weight, higher-level plyometric training may begin. The pool is an excellent trainer for jumping and cutting. These and all functional activities should be implemented in the pool before their initiation on dry land. This progression enables the remodeling bone to begin adapting to the stresses of jumping and cutting in a less stressful environment.

An important point for clinicians is that not all athletes will be able to begin their functional progression with running. Some may need to start with a 0.80-km (0.5-mile) walk-jog, and others may be able to move more quickly. The key point is that pain is the only guide that the athletic trainer and injured person have, and it should be used as a guide to all activity.
Nonsteroidal anti-inflammatory medications
TENS, HVES
Ice massage/contrast baths
Towel toe curls x 10
Upper body ergometer
Upper body, uninvolved-leg weight training
Wobble board: sitting, involved-leg ROM
Stationary bike: uninvolved leg
Involved-leg strength: isometrics
Pool: treading water
Ambulation: crutch
Full weight-bearing ADLs
Standing ROM/balance
Both legs → Interval training
Rubber tubing: 4x10 → 4x20
Toe raises: 2 ft → 1 ft
squatting
Jog: chest/waist-deep water
Walk x 30 min

Phase I

Phase II

Phase III

Figure 7. An example of a 3-phase progression of stress fracture rehabilitation. Activities between phases I and II and between phases II and III overlap to form a continuum of exercise and functional return to activity. TENS indicates transcutaneous electric stimulation; HVES, high-voltage electric stimulation; ROM, range of motion; ADLs, activities of daily living.

The running portion of phase III is completed in a cyclic fashion that mimics bone growth. As bone is being resorbed in the first 2 weeks of activity, running is encouraged to promote the formation of trabecular channels (functional phase). In the third week, when the newly formed osteocytes and periosteum are maturing, running activity is decreased (rest phase). During the first cycle of phase III, functional activity is reduced to the phase II level. In each successive cycle, the activity intensity in the rest phase is reduced to the functional level of the previous cycle. The cycle of 2 weeks on, 1 week off continues through the duration of the rehabilitation process, usually from 3 to 6 weeks. As the running program progresses to sprinting and sport-specific activities, the rest days between functional activities decrease, and the athlete is gradually prepared for the return to competition (Figure 8).

The injured person may note an increase in pain during the management process. If the increase in pain occurs during phase I or phase II, the offending activity should be discontinued or modified. Those who notice pain during ADLs or treatment should not be progressed to the next phase of the protocol until the activity can be completed pain free. During phase III, pain is usually an indication that the level of activity is too high, and functional activity should resume at the last level that was completed pain free within that 3-week cycle. If pain persists even at a reduced level, the activity intensity should be scaled back to the level from the previous 3-week cycle. Individuals who have persistent pain should be referred back to their physician. In these cases, returning treatment at the phase I or phase II level may be indicated.

Compliance with the management program is critical for a timely return to activity. This is most difficult during the rest phase of phase III. Because the treated person has been predominantly pain free up to this point, stopping a pain-free functional activity is difficult to accept. Satterfield et al.11 went so far as to recommend referring patients to behavior-modification specialists in some cases. In any event, the rehabilitation of a stress fracture is a team effort involving the injured person, coach, physician, athletic trainer, and sport psychologist. Only by working together can the proper diagnosis, goal setting, education, rehabilitation, and successful return to sport be accomplished.

Prevention

Awareness of the causes of stress fractures can lead to appropriate preventive interventions. Bone is the weakest in the
third week after the initiation of a stressful activity. By altering training intensity during the third week of workouts, osteoblastic filling of absorptive areas and bone maturity can occur. For example, a change from plometrics to a lower-impact aerobic activity during the third week of practice may reduce the stressors associated with stress fractures. In a military population participating in basic training exercises, the incidence of stress fracture in a cyclic training group was reduced to one third that of a noncyclic training group. Another effective strategy in prevention is identifying and minimizing changes in shoes or surfaces. Limiting activity to one playing surface or pair of shoes can reduce the likelihood of the surface and shoes becoming stressors and contributing to the formation of a stress reaction or ultimately a stress fracture.

CONCLUSIONS

Stress fractures can occur to just about any bone in a physically active person. They are at the endpoint of a continuum of a bone's reaction to stress that ranges from early remodeling to a cortical fracture. Normal levels of stress facilitate normal bone remodeling. When activity levels change or increase, the level of bone remodeling also increases. A gradual decrease in bone density follows this higher level of remodeling and places the bone at risk for a stress fracture. Stress fracture risk may be highest during the third week after the onset of the new or increased activity. Proper management of stress fractures should begin immediately. A 3-phase management process has been described based on the physiology of bone remodeling. It is important for the athlete, coach, and athletic therapist to understand the causes and cyclic formation of bone remodeling and management strategies for stress reactions and true stress fractures so that the physically active person can return to competition quickly and safely.

ACKNOWLEDGMENTS

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Traumatic Hemarthrosis of the Knee Secondary to Hemophilia A in a Collegiate Soccer Player: A Case Report

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Western Michigan University, Kalamazoo, Ml

Background: Hemophilia is a hereditary blood disease characterized by impaired coagulability of the blood. Hemophilia A is the most common of the severe, inherited bleeding disorders. This type, also called classic hemophilia, is due to a deficiency of clotting factor VIII. The athlete with hemophilia A reported pain and loss of function of his knee during a soccer game despite the absence of injury.

Differential Diagnosis: Anterior cruciate ligament tear, intra-articular fracture, meniscus tear, capsular tear, hemarthrosis.

Treatment: After the injury, the athlete was admitted to the hospital, where his knee joint was aspirated and he was infused with factor VIII. Later, he participated in traditional knee rehabilitation and was returned to play at the discretion of the orthopaedist and the hematologist.

Uniqueness: In past participation guidelines, individuals with bleeding disorders were disqualified from athletic participation; however, with advances in medical care, these individuals may be permitted to participate in accordance with the law.

Conclusions: Individuals with hemophilia participate in athletics; therefore, team physicians and athletic trainers must be prepared to care for these individuals.

Key Words: Americans with Disabilities Act, desmopressin acetate, factor VIII, preparticipation physical examination, blood coagulation

Until recently, individuals with blood coagulation disorders were not permitted to participate in athletics. Table 1 shows the 1990 sports participation recommendations for athletes with hemophilia. Medical advancements have aided in the care of individuals with hemophilia and may allow some individuals with hemophilia to participate in athletic sports, depending on the severity of their illness and the type of sport. Also, in 1990, the Americans with Disabilities Act (ADA) was passed, requiring that no individual be discriminated against based on disability alone. Because individuals with hemophilia often participate in athletics, team physicians and athletic trainers must learn to prevent and treat sports injuries in hemophilic athletes.

CASE REPORT

A 21-year-old male collegiate soccer player reported pain and loss of function in his left knee after an away soccer match. He denied any mechanism of injury or pain during the game. The athletic trainer noted marked edema of the lower leg and joint-line effusion of the knee. The athlete was unable to bear weight on his left leg because of pain. Orthopaedic tests could not be performed due to the intensity of the acute symptoms. Ice was placed on the athlete's knee, and the athlete asked to be taken to the hospital because of a history of bleeding problems. There was no documentation of any chronic medical condition in the athlete's medical records.

Upon arrival at the emergency room, the athlete informed the physician that he had mild hemophilia A with no inhibitors and did not use any prophylactic medication before athletic participation, such as desmopressin (DDAVP) or recombinant factor VIII (rFVIII) infusions. Desmopressin acetate and rFVIII may result in increased circulating factor VIII. The emergency room physician noted that the left knee was markedly swollen and range of motion was severely restricted. There was no pain on palpation of the tibia, fibula, or distal femur. Valgus and varus ligamentous stress tests were negative. A Lachman test could not be performed because of pain and inability of the knee to flex. An x-ray of the left knee showed a joint effusion but no fracture. Clotting factor assay revealed a factor VIII level of 23%, which classified this athlete as a mild hemophiliac. The athlete's knee was aspirated, and the amount of rFVIII necessary to raise the circulating factor VIII to 100% was calculated and transfused. The athlete was fitted for crutches and instructed to elevate the knee and use a compression wrap. The emergency room physician made arrangements for follow-up with a hematologist at the home site. The athlete was then released from the hospital and returned home on the team bus.

The next day, a local hematologist examined the athlete and
recommended infusions of factor VIII to maintain 100% level for the next 3 days. After the 6-day immobilization period, he started a rehabilitation program that included passive range of motion, quadriceps sets, and heel slides. Rehabilitation progressed slowly, as if the athlete had been immobilized for an extended period of time. He advanced to pain-free range of motion as tolerated. As the effusion subsided, the rehabilitation program became more aggressive. In order to decrease patellofemoral joint-distraction forces and prevent irritation to the patellofemoral joint, open kinetic chain knee exercises were performed on the KinCom dynamometer (Chattanooga Group Inc, Hixson, TN) from 0° to 30° and closed kinetic chain exercises were performed from 0° to 30°. Throughout the rehabilitation process, the athlete was monitored daily for increased swelling. Overall rehabilitation time was increased (compared with other athletes with hemarthrosis) because of swelling and concern for reinjury. Six weeks later, the athlete was cleared to play after the effusion completely subsided, range of motion and strength were restored, and the orthopedist and hematologist cleared the athlete.

As a result of this injury, the soccer team physician, athletic trainer, and athletic training student developed an emergency plan for the safe participation of this athlete and other athletes with hemophilia. The plan included referral to a hematologist for preparticipation physical clearance and as a result of injury. Other components of the plan were DDAVP effectiveness testing, transportation of factor VIII to all away contests, and knowledge of hemophilia treatment centers for away competition sites (Table 2).

**DISCUSSION**

When blood vessels are damaged, a series of biochemical reactions occurs to form a blood clot. The Figure shows the coagulation cascade, including the intrinsic, extrinsic, and common coagulation pathways. A clot is formed via the intrinsic pathway in response to an abnormal vessel wall in the absence of tissue injury. The process is initiated when contact occurs between blood and exposed endothelial cell surfaces. The name “intrins” implies that all the components are in the plasma. The extrinsic pathway results in fibrin clot formation in response to tissue injury. Tissue factor, which is extrinsic to plasma, is activated as a result of vascular injury. The 2 pathways converge at the activation of factor X and ultimately lead to the blood clot.1

Hemophilia is a hereditary blood disease characterized by impaired coagulability of the blood.1 The following information has been compiled to explain the factors that should be considered when making decisions regarding the athletic participation status of individuals with hemophilia. Particular attention should be given to developing a proper emergency plan for immediate care of the injured hemophilic athlete.

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**Table 1. 1990 Sports Participation Guidelines for Athletes with Hemophilia***

<table>
<thead>
<tr>
<th>May Participate</th>
<th>Participation Not Recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>Limited contact/impact</td>
<td>Collision/contact</td>
</tr>
<tr>
<td>Track-and-field</td>
<td>Field hockey</td>
</tr>
<tr>
<td>High jump</td>
<td>Football</td>
</tr>
<tr>
<td>Pole vault</td>
<td>Ice hockey</td>
</tr>
<tr>
<td>Cross-country skiing</td>
<td>Lacrosse</td>
</tr>
<tr>
<td>Noncontact</td>
<td>Martial arts</td>
</tr>
<tr>
<td>Archery</td>
<td>Rugby</td>
</tr>
<tr>
<td>Badminton</td>
<td>Soccer</td>
</tr>
<tr>
<td>Bowling</td>
<td>Water polo</td>
</tr>
<tr>
<td>Crew (rowing)</td>
<td>Wrestling</td>
</tr>
<tr>
<td>Field</td>
<td>Limited contact/impact</td>
</tr>
<tr>
<td>Discus</td>
<td>Baseball</td>
</tr>
<tr>
<td>Javelin</td>
<td>Basketball</td>
</tr>
<tr>
<td>Shot put</td>
<td>Cycling</td>
</tr>
<tr>
<td>Cross-country running</td>
<td>Diving</td>
</tr>
<tr>
<td>Golf</td>
<td>Gymnastics</td>
</tr>
<tr>
<td>Swimming</td>
<td>Skiing (downhill)</td>
</tr>
</tbody>
</table>


**Table 2. Emergency Plan Considerations for Dealing with Hemophilia**

1. Referral to Hematologist
   a. Preparticipation physical examination clearance
   b. As a result of injury
   c. Return to play after injury

2. Desmopressin Acetate (DDAVP) Effectiveness Testing

3. Away Contest Considerations
   a. Location, directions, and contact numbers for nearest hemophilia treatment centers
   b. Transportation of factor VIII
   c. Transportation of DDAVP (if effective)

4. Home Contest Considerations
   a. On-site factor VIII
   b. On-site DDAVP (if effective)

5. Education of Sports Medicine Staff
   a. General knowledge of hemophilia and injury management
   b. Specific emergency plan

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**Blood Coagulation Mechanism**

**Intrinsic Pathways**

**Extrinsic Pathways**

- **Surface contact**
- **HMWK**
- **FVIIa**
- **FXIIIa**
- **FI (Fibrinogen)**
- **FIa (Fibrin)**
- **Stable fibrin**

**Blood coagulation mechanism. HMWK indicates high molecular-weight kininogen; F, factor; Ca++, calcium.**
Types of Hemophilia

Hemophilia A is the most common hereditary coagulation disorder. This disorder is due to a partial or complete deficiency of factor VIII coagulant activity, which is the result of a gene defect on the X chromosome. Hemophilia A is an X-linked recessive disorder. Because males have only one X chromosome, affected males exhibit characteristics of the disorder. Females have two X chromosomes and the disorder is recessive; therefore, they only show characteristics of hemophilia if the unaffected X chromosome is inactivated, and usually their disease is less severe. One in every 10,000 males is affected with hemophilia A. Although hemophilia A is known as an inherited disorder, nearly 30% of individuals with the disorder have no prior family history, and the condition is the result of spontaneous gene mutations.

Hemophilia B (Christmas disease) is a partial or complete deficiency of factor IX. The inheritance patterns and clinical presentation of hemophilia B are similar to those of hemophilia A. Hemophilia B is a sex-linked recessive disorder with clinical symptoms including hemarthroses and hemarthrosis. This disorder, however, is not as common as hemophilia A, occurring in 1 in 100,000 males.

Hemophilia C (Rosenthal syndrome) is a hereditary bleeding disorder caused by a deficiency of factor XI. It affects 1 of 8 in the Ashkenazi Jewish population. Hemophilia C is inherited as an autosomal recessive trait. Its incidence is 1 in 100,000.

Von Willebrand disease is an autosomal dominant coagulation disorder caused by a deficiency of a component of factor VIII. This condition is characterized by consistently prolonged bleeding times and mucocutaneous bleeding rather than hemarthroses and deep muscle hemorrhages. Other signs and symptoms include epistaxis, ecchymosis, easy bruising, gastrointestinal bleeding, menorrhagia, and hemorrhage after surgery. The incidence of von Willebrand disease is 1 in 80,000.

History of Athletic Participation by Individuals with Hemophilia

Few publications describe the extent to which boys and adults with hemophilia participate in sports. A recent review article provided an overview of sports participation by hemophilia athletes. It reported that only 3 other studies provide data on seasonal or yearly athletic participation of individuals with hemophilia.

In the first study, Glomstein reported participation of Norwegian with hemophilia involved in Nordic sports. Questionnaires were sent to 298 people, and 179 were returned. Of the 179 respondents, 178 indicated that they participated in Nordic winter sports during their lifetime, and 132 of 178 were still active.

In the second and third studies, Heijnen et al. and Rodriguez-Merchant reported participation in physical activities and sports by 166 Dutch people with hemophilia in 1996 and 209 in 1997. In their most recent article, Heijnen et al. reported the results of a self-administered questionnaire for 293 Dutch individuals who visited the clinic for their yearly check-up. They considered type of sport and severity of hemophilia. Most individuals were severe hemophiliacs (217) and participated in 44 different sports, including cycling, fitness, skating, skiing, swimming, and tennis.

National Collegiate Athletic Association Division I team physicians were surveyed to determine if they would allow athletes with hemophilia to participate in sports. The survey questions were specific to the type of sport and the severity of the hemophilia. An additional purpose of the study was to determine if individuals with hemophilia A were currently participating in Division I athletics. Team physician were willing to allow participation under certain circumstances, and hemophilic athletes were currently participating.

Factors to Consider When Making Participation Decisions

Many factors should be taken into consideration when making a decision to permit or disqualify an individual with hemophilia from athletic participation. Among these factors are the severity of the hemophilia, the type of sport, treatment, medication, the presence or absence of inhibitors (antibodies that may destroy rFVIII), and consultation with a specialist.

Severity of the Hemophilia. Hemophilia is classified as mild, moderate, or severe based on the severity of the disorder. Those with mild hemophilia have 5% to 50% of the normal level of concentration of factor VIII. These individuals are at minimal risk for spontaneous hemorrhages, but they are likely to bleed after trauma or surgery. Those with moderate hemophilia have between 1% and 5% of the normal factor VIII level. These individuals hemorrhage after moderate trauma. Those with severe hemophilia have less than 1% of the normal factor VIII level. These individuals frequently bleed after minimal or unrecognized trauma, especially into joints and muscles.

Type of Sport. Sports are classified into 3 general categories: collision, contact, and noncontact. Collision sports are played with the intent of striking opposing athletes. Sports in this category include football, hockey, and rugby. Contact sports have the possibility of contacting opposing athletes; however, that is not the intent. Examples of these sports are basketball and soccer. In noncontact sports, contact among athletes is highly unlikely. Track and field and tennis are examples of noncontact sports.

Treatment and Medication. Advances in treatment for hemophilia have allowed affected individuals to become more active. Recombinant factor VIII permits the athlete to prevent or treat bleeding that occurs as a result of injury. This gives the athlete reassurance that the injury can be managed efficiently. The plasma-derived concentrations currently used carry a low risk of transmitting bloodborne infections. Major efforts to reduce disease transmission have been instituted: for example, selecting low-risk plasma donors, adopting polymerase chain-reaction-based virus-detection methods, and retesting donors 6 months after taking blood to determine if it can be used. In 1983, DDAVP was approved for use in the United States for individuals with mild hemophilia A and von Willebrand disease. Desmopressin acetate is a synthetic antidiuretic hormone that releases factor VIII from endothelial tissue. If more factor VIII is present in the bloodstream before an injury, the hemorrhage may not cause as much damage as it otherwise might; however, the response to DDAVP is not universal.

To determine the effectiveness of DDAVP, treatment centers test baseline levels of circulating factor VIII, administer DDAVP, and then reassess circulating levels of factor VIII. If factor VIII levels increase 3- to 5-fold, the DDAVP
treatment is considered effective and may be used in the future to prevent and treat athletic injuries.

In 1992, the first rFVIII was derived. This genetically engineered factor VIII allows for hemorrhage control. However, minimal risk of disease transmission was present because the rFVIII was formulated in human albumin. In 1997, a second generation of rFVIII was produced that does not require the use of human albumin. The creation of rFVIII has made treatment more readily available for patients with hemophilia, and the current form permits treatment with no risk of disease transmission.

Inhibitors. An inhibitor is a type of antibody that destroys substances the body does not recognize. An individual with hemophilia A who has inhibitors to factor VIII neutralizes and inhibits factor VIII, allowing the bleeding to continue. Inhibitors are not inherited but acquired. Individuals with factor VIII levels of less than 5% of normal have a 20% to 33% chance of developing inhibitors. Although individuals with mild hemophilia are not likely to develop inhibitors, patients with severe hemophilia are at a much higher risk of acquiring inhibitors because they are exposed to more factor VIII therapy. Most inhibitors develop during childhood. Fifteen to twenty percent of heavily treated children with hemophilia develop antibodies by the age of 20 years.

Until recently, hemophiliacs with inhibitors could not benefit from improvements in treatment strategies because their inhibitors also function to neutralize recombinant factor VIII. Treatments such as immune tolerance and FVIIa replacement are helpful for patients with inhibitors (Figure). Tolerance is built in patients through the repeated administration of high doses of factor VIII, suppressing the production of factor VIII inhibitors. This is achieved in two thirds of the patients exposed to repeated, high-dose administration of factor VIII. By using FVIIa and the extrinsic pathway, the need for factor VIII is bypassed (Figure). Some advantages to FVIIa include a limited risk of virus transmission, absence of severe anaphylactic reactions, and reduced risk of inhibitor development. Disadvantages include the high cost and the need for repeated administration due to the short half-life of FVIIa (3 to 4 hours).

Examination and Consultation with Hematologist. Each patient needs to be evaluated on an individual basis. Past history of injuries, severity level, type of sport, and response to treatments need to be considered before making a final decision regarding athletic participation. Physical and emotional maturity should also be considered. Chronologic age, Tanner stage, and muscle strength can be used to assess physical maturity. Individuals who are aware of their body and who do not take unnecessary risks are more likely to be successful in sports. Risk takers are more apt to incur injury. The decision to allow participation should come with the aid of the patient’s hematologist, but the team physician has the final say.

Factors Supporting Participation of Athletes with Hemophilia

With the injury risk associated with sports, one may ask, why should those with hemophilia participate in athletics? Supporting participation are the psychological and physiologic benefits of being involved in sports and exercise. The legal statute, the Americans with Disabilities Act, also supports the rights of hemophilic individuals to participate.

Benefits of Exercise and Psychological Benefits. The benefits of physical activity are numerous for the general population. Among these benefits are improved cardiorespiratory efficiency, control of body fat, improved strength and flexibility, and improved psychological and emotional well-being. Hemophilic patients receive the same benefits from exercise and some additional benefits. Through exercise, muscle power and range of motion can be maintained, reducing the number and severity of bleeding episodes caused by abnormal stresses. Exercise has been proven to increase the level of circulating factor VIII. All these benefits of exercise support the importance of providing athletic opportunities to those with hemophilia.

Americans with Disabilities Act. On July 26, 1990, the ADA was signed into law. This act protects against athletic disqualification based on disability. According to the law, schools and colleges may not discriminate against an otherwise qualified individual because of that individual’s disability. In sport, an athlete who otherwise meets all criteria for participation, including having the skill level and strength and conditioning required to make the team, must be allowed to play.

An objective of the team physician is to avoid the unnecessary restriction of athletic participation. However, his or her principal responsibility is to protect the health of athletes. It is important to note that court decisions have upheld the right of team physicians to medically disqualify athletes. Team physicians must make individual participation decisions after considering the demands of a particular sport, the potential harmful effects on an athlete’s health, and other participants’ safety. With modifications to procedures, some hemophilic athletes can be accommodated in an efficient manner and, thus, should be permitted to play.

CONCLUSIONS

Individuals with hemophilia have special needs. Prevention and management plans must be devised to expedite the care of these athletes; however, many people who care for these athletes are not aware of the necessary prevention and intervention strategies. The physical, psychological, and social benefits of being involved in a team sport have been established, and the ADA has called attention to the rights of athletes with disabilities. Therefore, hemophilic athletes must not be denied the right to participate in sports if reasonable measures can be taken to accommodate these athletes. Team physicians and athletic trainers need to be prepared to treat hemophilic athletes; however, it may not be in the best interests of certain athletes with hemophilia to participate, as the risks outweigh the benefits. In these cases, it is appropriate for the team physician to disqualify the athlete.

REFERENCES


Seeing the Forest Through the Wheeze: A Case-Study Approach to Diagnosing Paradoxical Vocal-Cord Dysfunction

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Paradoxical vocal-cord dysfunction (PVCD) is defined as a paradoxical closure of the vocal cords during the inspiratory phase of respiration. Such closure results in partial, sometimes severe, obstruction of airflow. An entity with symptoms similar to PVCD was first described in 1842. The past few decades have seen an increasing number of reports of PVCD in the literature. A few of these reports have described PVCD presenting similarly to exercise-induced asthma (EIA) in athletes, while others have discussed patients with severe, intractable asthma. Vocal-cord dysfunction is a relatively rare disorder, although Rice et al speculated that the prevalence may be as high as 3% among intercollegiate athletes. Vocal-cord dysfunction results from paradoxical closure of the vocal cords during the inspiratory phase of respiration and may be mistaken for EIA, resulting in unnecessary medical treatment and a delay in diagnosis. Although PVCD is uncommon, athletic trainers should be aware of the disorder, as they may play an important role in its diagnosis and treatment.

**Objective:** To present for discussion a case of paradoxical vocal-cord dysfunction (PVCD), an uncommon disorder that may be misdiagnosed as, or coexist with, exercise-induced asthma (EIA).

**Background:** Vocal-cord dysfunction results from paradoxical closure of the vocal cords during the inspiratory phase of respiration and may be mistaken for EIA, resulting in unnecessary medical treatment and a delay in diagnosis. Although PVCD is uncommon, athletic trainers should be aware of the disorder, as they may play an important role in its diagnosis and treatment.

**Differential Diagnosis:** Exercise-induced asthma, foreign body aspiration, anaphylactic laryngeal edema, bilateral vocal cord paralysis, extrinsic airway compression, laryngomalacia, subglottic stenosis, traumatic edema, or hemorrhage.

**CASE REPORT**

**History**

A 17-year-old female high school athlete presented to the team physician for further evaluation of EIA, which had been diagnosed by her family physician the previous year. Her symptoms began during basketball season of her freshman year of high school. She complained of dyspnea during and immediately after activity. She occasionally had a persistent cough lasting several hours after exertion and complained of excessive postexertional fatigue. Her symptoms were much more prominent while playing basketball (both regular season and summer league) than during participation in soccer or other athletic activities. Symptoms were also more pronounced during games. Upper respiratory infections occasionally triggered mild coughing. She had no past history of asthma, nocturnal cough, allergic rhinitis, wheezing with upper respiratory infections, reactive airways disease, or environmental allergies. She occasionally had mild dyspepsia after meals. There was no family history of asthma.

She had previously been treated with multiple medications (exact dosages unknown), including the use of an albuterol metered-dose inhaler (MDI) before exercise and prophylactic regimens of montelukast sodium (Singulair, Merck & Co Inc, West Point, PA), zafirlukast (Accolate, Zeneca Pharmaceuticals, Wilmington, DE), triamcinolone (Azmacort, Rhône-Poulenc Rorer, Collegeville, PA), and salmeterol (Serevent, Glaxo Wellcome Inc, Research Triangle Park, NC). Each medication had been tried for only short periods of time, generally no more than 3 to 4 weeks, and was discontinued either at the
direction of her physician due to ineffectiveness or by the athlete out of frustration that it was not helping.

Physical Examination

Physical examination revealed a healthy-appearing adolescent girl (height = 169 cm, weight = 61 kg) in no acute distress. Her nares were clear with no discharge or irritation of the mucosa. Lungs were clear to auscultation bilaterally with a normal inspiratory-to-expiratory ratio. Her chest had a normal anterior-posterior diameter with no bowing of the sternum. Her heart had a regular rate and rhythm and normal S1 and S2 heart sounds with no murmurs, rubs, or gallops. Her extremities showed no clubbing of the fingernails or cyanosis.

After the initial evaluation, she was given a 4-week trial of fluticasone (Flovent, Glaxo) 44 μg by MDI, and salmeterol 42 μg by MDI, each at a dose of 2 puffs twice per day, and cromolyn sodium 800 μg MDI, 4 puffs before exercise, while diagnostic testing was arranged. She reported no benefit from the new medications.

Diagnostic Testing

Pulmonary function testing (PFT) was performed before exercise, after 15 minutes of high-intensity aerobic activity (running), and after inhalation of albuterol. The PFT results (Table 1) were highly suggestive of PVCD as the cause of her symptoms. She was referred to an otolaryngologist for definitive diagnosis. Laryngoscopy was performed after provocation of her symptoms with exercise and revealed closure of her vocal cords during the inspiratory phase of respiration, thus confirming the diagnosis of PVCD.

Treatment

After the diagnosis of PVCD was confirmed, the athlete underwent several sessions with a speech therapist to learn corrective breathing techniques. She was also assured that her PVCD was exacerbated by increased stress in certain situations and, with work, she would be able to overcome her symptoms. All EIA medications were discontinued, and she concentrated on the breathing techniques she had been taught.

Follow-Up

At the time of manuscript preparation, she was 6 months postdiagnosis and had completed her soccer and basketball seasons (earning All-Conference honors in each sport) with minimal symptoms.

**DISCUSSION**

**Exercise-Induced Asthma**

Exercise-induced asthma is common among active people, with an overall incidence of 12% to 15%, although the prevalence may be even higher among elite athletes. Exercise-induced asthma is defined as "reversible airway obstruction that occurs during or after exertion." Specific symptoms include chest tightness, wheezing, coughing, and shortness of breath, which result from acute narrowing of the lung’s small airways. The exact mechanism of the airway narrowing and obstruction is not known, but 2 current theories suggest increased minute ventilation, causing (1) water loss in the cells of the bronchial mucosa, and (2) cooling of the airways. Each may potentially lead to release of cell mediators and subsequent inflammation and asthma.

The diagnosis of EIA may be suspected after taking a thorough history of exercise-related symptoms. The diagnosis is confirmed by PFTs performed before and after exercise provocation. A drop in maximum volume of expired air in one second (FEV1) of more than 15% is diagnostic of EIA. While a positive result is indicative of EIA, a negative test result does not rule out the disease. Therefore, if EIA is still suspected, other tests that provoke bronchoconstriction may be given, such as inhalation of methacholine, histamine, or cold air.

Initial treatment typically involves prophylactic inhalation of a beta-agonist medication (usually albuterol) 15 to 20 minutes before exercise, although nonpharmacologic treatment is an option. Beta-agonist medications are 80% to 95% effective in alleviating the symptoms of EIA. If an athlete does not respond to initial treatment, a detailed history and physical examination must be repeated, and additional diagnostic testing may be pursued as other possible diagnoses are considered.

**Differentiating Paradoxical Vocal-Cord Dysfunction from Exercise-Induced Asthma**

Vocal-cord dysfunction and EIA can present in strikingly similar manners and may even coexist. However, several historical clues aid health care professionals in developing a high index of suspicion for PVCD (Table 2). Exercise-induced asthma symptoms typically peak 5 to 10 minutes after exercise begins and often spontaneously resolve within 30 to 60 minutes with continuous exercise. Coughing may persist for several hours after the cessation of activity, and the symptoms are typically reproducible under similar conditions. Our patient showed inconsistency in symptoms, in that soccer did little to provoke her symptoms, whereas basketball caused her most serious exacerbations. An individual with EIA would be expected to have similar symptoms in each sport, as they are both considered highly “asthmagenic” secondary to the high minute ventilation required. Such inconsistency in symptoms could be secondary to allergen exposures (molds in a gymnasium, pollens outside), but she had no other allergic symptoms.

Symptoms in PVCD are often situation dependent and may begin and end abruptly. The afflicted individual may describe a sensation of throat tightness or choking. The inspiratory phase of respiration may be audible during an acute attack, and symptoms may be interrupted by distracting the athlete or instructing her to begin panting. Athletes with acute EIA ex-
Table 2. Signs and Symptoms To Help Differentiate Paradoxical Vocal-Cord Dysfunction from Exercise-Induced Asthma*

<table>
<thead>
<tr>
<th>PVCD</th>
<th>EIA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
<td></td>
</tr>
<tr>
<td>Situation dependent</td>
<td>Reproducible in similar situations</td>
</tr>
<tr>
<td>Begin and end abruptly</td>
<td>Peak after 5 to 10 min of exercise</td>
</tr>
<tr>
<td>May abate with distraction</td>
<td>Resolve within 30 to 60 min</td>
</tr>
<tr>
<td>Unrelated to environment</td>
<td>Provoked by cold or dry air</td>
</tr>
<tr>
<td>*PVCD indicates vocal-cord dysfunction; and EIA, exercise-induced asthma.</td>
<td></td>
</tr>
</tbody>
</table>

Table 3. Pulmonary Function Test Findings To Help Distinguish Paradoxical Vocal-Cord Dysfunction from Exercise-Induced Asthma*

<table>
<thead>
<tr>
<th>PVCD</th>
<th>EIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flow loop</td>
<td></td>
</tr>
<tr>
<td>Normal or decreased</td>
<td>Elliptic</td>
</tr>
<tr>
<td>Normal or decreased</td>
<td>Usually decreased</td>
</tr>
<tr>
<td>Normal or decreased</td>
<td>Decreased &gt;20%</td>
</tr>
<tr>
<td>Normal or decreased</td>
<td>FEV&lt;sub&gt;1&lt;/sub&gt;/FVC decrease &gt; FVC</td>
</tr>
<tr>
<td>FEF&lt;sub&gt;25%-75%&lt;/sub&gt;</td>
<td>Ratio &gt;1</td>
</tr>
<tr>
<td>FEF&lt;sub&gt;50%/FIF&lt;sub&gt;50%&lt;/sub&gt;</td>
<td>Ratio &lt;1</td>
</tr>
</tbody>
</table>

*PVCD indicates vocal-cord dysfunction; EIA, exercise-induced asthma; FVC, forced vital capacity; FEV<sub>1</sub>, forced expiratory volume in 1 second; FEF<sub>25%-75%</sub>, forced expiratory flow from 25% to 75% of vital capacity; and FEF<sub>50%/FIF<sub>50%</sub>, forced expiratory to inspiratory flow at 50% of vital capacity.

Auscultating the chest during an acute episode may also help to differentiate EIA from PVCD. Stethoscope examination of an athlete with onset of EIA symptoms should reveal wheezing, while an athlete with PVCD may have stridor. Wheezing is described as a “whistling, squeaking, or puffing sound” and “to breathe with difficulty and noisily.” Stridor is identified as “high-pitched, noisy respiration, like the blowing of wind” and may be heard without the aid of a stethoscope. While these lung sounds are certainly not mutually exclusive, they typically do not occur simultaneously. In general, stridor is associated with upper airway (trachea, larynx) diseases, such as croup and PVCD, and can occur during both inspiration and expiration. Wheezing is caused by the passage of high-velocity air through narrowed bronchi; although it may be heard with both inspiration and expiration, it is most typically associated with expiration and is the most common physical examination finding in asthma.

Beta-agonist medications are considered so effective in EIA treatment that a poor or insufficient response should prompt a more thorough diagnostic evaluation. Preexercise and post-exercise PFTs are indicated if they were not initially obtained. Proper use of the medication must be assessed by observing the athlete’s use of the MDI. Correct timing (before exercise) and compliance with therapy must also be discussed, and the athlete should be observed for compliance with therapy by a coach or athletic trainer. Most individuals who are unresponsive to initial attempts at therapy will indeed be proven to have EIA; however, they require additional medical therapy to control their disease.

**Diagnostic Findings**

An in-depth discussion of pulmonary mechanics and PFTs is beyond the scope of our report, but an explanation of some important data will assist in understanding the findings (Table 3). A variety of unusual PFT results can help differentiate PVCD from EIA; however, these findings are quite variable in PVCD. Our patient provides an excellent example, as her PFTs showed all of the unusual features of PVCD. First, the inspiratory portion of her preexercise (baseline) flow curve is characteristic of upper airway obstruction (Figure 1). Thus, she showed an abnormal curve while asymptomatic, a finding that occurs in almost one fourth of individuals with PVCD. A normal curve (Figure 2) has an elliptic shape, while upper airway obstruction, such as that resulting from vocal-cord closure, results in flattening or truncation of the curve. Her FEV<sub>1</sub> showed a 19% decrease after exercise challenge, which meets the criteria for EIA. However, all available data must be reviewed before making a diagnosis. While PVCD and EIA may coexist, further analysis of the PFT data ruled out comorbid disease. She also had a decrease of 18% in her total expired lung volume (FVC); thus, the FEV<sub>1</sub>/FVC ratio...
was unchanged, a finding consistent with PVCD. Exercise-induced asthma may also cause a decrease of FVC, but the drop will not be in proportion to the FEV₁ decrease. However, after treatment with a bronchodilator (albuterol), our patient’s FEV₁ improved 13% and her FVC increased 15%. Although this finding is difficult to explain, we postulate that it likely resulted more from relaxation in breathing technique after the treatment than from a pharmacologic effect of the medication.

The forced expiratory flow from 25% to 75% of vital capacity (FEF₂₅₋₇₅%) reflects airflow through the small airways of the lungs. The small airways are the most affected by any degree of asthma resulting from EIA. Therefore, an individual with EIA shows a large decrease in this value after an exercise challenge. Our patient’s decrease of 6% is not considered significant. There was also minimal change after administration of the bronchodilator. Finally, the ratio of forced expiratory to inspiratory flow at 50% of vital capacity (FEF₅₀%/FIF₅₀%) is normally less than 1; however, with the inspiratory obstruction caused by the closure of the vocal cords, the ratio is typically greater than 1, as it was with our patient.¹

Although the PFTs were highly suggestive of PVCD, the patient was referred to a pediatric otolaryngologist for definitive diagnosis. Vocal-cord dysfunction can only be confirmed by finding paradoxical closure of the vocal cords in a symptomatic patient upon inspiration during laryngoscopy. However, in patients in whom the history and PFTs are consistent with PVCD, some authorities recommend foregoing laryngoscopy, initiating speech therapy, and observing for resolution of symptoms (Steve Simons, unpublished data, 2001). If symptoms persist, diagnostic laryngoscopy is indicated. Of note, laryngoscopy may be normal in 50% of individuals with PVCD if symptoms cannot be elicited before the examination.²

### Differential Diagnosis

The symptoms found in both PVCD and EIA may be secondary to other causes. The differential diagnosis is quite broad, as listed in Table 4. Fortunately, the other disorders are quite rare and can typically be ruled out on the basis of history alone. Anaphylactic laryngeal edema may present acutely with stridor similar to that sometimes seen in PVCD; however, the athlete will have additional physical examination findings such as angioedema, flushing, pruritis, hypotension, and hives.¹⁷ If such findings are present on examination, the emergency medical services system should be activated while the athletic trainer provides initial first aid as needed. Acute trauma and inhaled foreign bodies (typically food or insects) may also result in acute stridor and dyspnea, but the history should be conclusive.

### Psychological Aspects of Paradoxical Vocal-Cord Dysfunction

Anxiety and emotional stress may contribute to the symptoms experienced in PVCD; however, controversy exists regarding the role of these factors in the disorder. The implication of a psychological cause for PVCD may promote undue stress in an athlete or her family, so these factors must be discussed with care by the athletic trainer and team physician. In addition, a review of the medical literature by an athlete with PVCD may also raise similar issues. Many articles emphasize the psychiatric aspects of PVCD, and some early reports go as far as describing PVCD as a conversion disorder.² One study in particular deserves mention. Freedman et al¹⁸ reported a 36% incidence of childhood sexual abuse among individuals with PVCD, but they did not study a control population. The incidence of sexual abuse in the general female population ranges from 6% to 62%;¹⁹ thus, the study’s findings are insignificant. The presumption of a psychogenic cause will likely alienate the athlete before the initiation of any treatment plan.¹

The primary reason for much of the emphasis upon the psychological factors for PVCD in the literature lies in the early experience with the disorder in patients with intractable asthma. Only recently have investigators looked at PVCD alone in comparison with control groups. Gavin et al²⁰ reported that patients with PVCD as their only diagnosis were not different from asthmatic controls on measures of family functioning, but they did experience higher levels of anxiety. However, the study population consisted of adolescents who sought treatment for severe asthma at a specialty center and were ult-
mately found to have PVCD rather than asthma. Applying the psychiatric findings of individuals with PVCD so severe that it limits daily activities to athletes with PVCD only associated with exercise is far from scientifically sound.

While the evidence for serious psychiatric conditions among athletes with exercise-related PVCD is lacking, certain personality traits are common among most of the affected individuals. The prototypical individual with exercise-related PVCD is a young woman who is a highly competitive athlete, success oriented, and intolerant of failure. These attributes are often shared by her parents and permeate the individual’s activities outside of athletics. In the case series of Landwehr et al, all adolescents for whom they had data were described as “straight A” or “4.0” students.

Anxiety may also be a contributing factor. Our patient’s psychological profile was quite consistent with the prototype and helped to raise our initial index of suspicion for PVCD. We had the additional advantage in that she had spent a semester as an athletic training student at the high school. This gave us an opportunity for better insight into her personality traits than is typically afforded an athletic trainer and team physician.

CONCLUSIONS

Vocal-cord dysfunction is a rare disorder, although the incidence is likely higher than reported. Athletic trainers should be aware of the disease and monitor all athletes with EIA for continuing symptoms and compliance with prescribed medications. The diagnosis of PVCD requires an initial high index of suspicion, which may be heightened if important historical information is provided to the attending physician. A timely and proper diagnosis of PVCD can alleviate an athlete’s symptoms, allowing the player to perform optimally and avoid unnecessary medications. A thorough understanding of PVCD permits the athletic trainer to aid in the athlete’s understanding of the disorder and to assist with treatment.

REFERENCES

Paradoxical Vocal-Cord Dysfunction: Management in Athletes

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Objective: To describe a treatment strategy for paradoxical vocal-cord dysfunction (PVCD) as it applies to an athletic population.

Background: Paradoxical vocal-cord dysfunction has been identified as a cause of dyspnea and stridor in athletes. The basic element of PVCD is an inappropriate closure of the vocal cords during respiration, resulting in airway obstruction. This condition is familiar to speech-language pathologists and otolaryngologists yet remains poorly understood in the sports medicine community. Treatment strategies are even less understood. A therapeutic exercise program designed to promote diaphragmatic breathing may allow an athlete to gain control during episodes of dyspnea. Elimination of contributing or concomitant conditions is critical to resolution of the condition.

Over the past decade, paradoxical vocal-cord dysfunction (PVCD) has been well studied, although little information has been presented in sports medicine journals. Most of the literature has addressed the recognition of PVCD in contrast to other reactive airway diseases. Authors have stressed the need for complete diagnostic testing, including visual examination of the vocal cords for subjects with exercise-induced dyspnea. Certainly, recognition of PVCD is critical to resolution of the condition. Treatment strategies, however, are often overlooked or presented with little detail. Indeed, much of the existing literature focuses on the emotional component associated with PVCD, although this is not necessarily a primary trigger for athletic subjects.

A common pattern is presented in the literature: a patient reports episodes of dyspnea or shortness of breath during exercise, and medications prescribed to relieve symptoms are ineffective. The continued symptoms, coupled with the failure of the medication and the patient's inability to complete assigned "fitness" drills, increases the emotional stress the athlete feels during practice. If the cause of respiratory distress is unrecognized and uncontrolled (as with undiagnosed PVCD), the resultant emotional stress can exacerbate PVCD symptoms. It has been our experience that, over time, the misdiagnosed athlete becomes less able to perform necessary cardiovascular fitness exercises (including practice drills) and less able to meet the demands of athletic participation. In addition, one should not underestimate the effect the labored breathing may have on the patient’s coaches and teammates, who may be concerned for his or her mortality.

In some instances, patients with acute PVCD have been directed to an emergency department for treatment of uncontrolled shortness of breath. More commonly, the continued symptoms and dissatisfaction with the course of treatment prompt further testing and, ultimately, the diagnosis of PVCD. This is often a diagnosis of exclusion, perhaps resulting from a lack of familiarity with the condition. A health care professional with a high index of suspicion and an understanding of the signs and symptoms of PVCD is in a position to direct the athlete to the appropriate specialist for definitive care, thereby avoiding exhaustive testing and inappropriate medication and prompting a quicker resolution of the condition.

Current literature emphasizes the treatment of psychological and emotional stressors; speech therapy is recognized as a treatment strategy, but little detail is provided. Indeed, some form of psychological dysfunction may be involved in PVCD. It has been suggested that athletes with a history of clinical depression or abuse (physical, sexual, or verbal) are at risk for PVCD. More commonly, athletic individuals with PVCD are identified as high achievers who strive for external validation and are likely to avoid situations involving con-
frontation. Further, these individuals' perceptions of third-party (ie, parents', coaches') expectations may be exaggerated, and they are likely to feel alienated from their teammates. Thus, a pattern unfolds in which an individual sets goals that may be intended to please a third party but then fails to meet those goals. A physical malady (in this case, severe narrowing of the larynx) may be manifested to explain the failure; poor understanding of the malady or its causes may exacerbate the level of stress and the intensity of the symptoms. This physical malady may also prevent the individual from normal participation in an event, thereby complicating the previously mentioned issues.

In the athletic patient, the emotional component may often be secondary to the dyspnea and failure to control it. The focus of speech therapy for PVCD patients is generally on respiratory control and diaphragmatic breathing patterns. As the athlete gains control over breathing patterns, he or she may realize a sense of control of this condition and reduce the emotional stress associated with dyspnea. Speech therapy, alone or in combination with other treatment interventions, has proven to be successful in reducing or eliminating the paroxysms of wheezing, stridor, and dyspnea. The patient must also be evaluated for coexisting medical conditions, such as asthma, exercise-induced asthma (EIA), gastroesophageal reflux disease (GERD), and pharyngeal erythema (secondary to postnasal drip); the latter 2 conditions can increase the sensitivity of the larynx. Habitual coughing and throat clearing can also increase sensitivity of the larynx, increasing the likelihood of PVCD. Finally, the hydration level of the vocal cords affects not only the mucus covering of the tissue but also the viscoelastic properties of the cords.

The purpose of this paper is to describe a treatment strategy for PVCD. Those readers interested in diagnostic testing are referred to the first article in this series. The treatment strategy described in this paper has been used successfully with collegiate and high school athletes and has been administered in both chronic and acute settings by speech-language pathologists and certified athletic trainers. It must be stressed that any effort to resolve PVCD must address (or unequivocally eliminate) concomitant medical conditions such as asthma, EIA, GERD, and rhinitis and behavioral deficiencies including throat clearing and insufficient fluid intake.

**Management of Paradoxical Vocal-Cord Dysfunction**

With normal breathing, the vocal cords are in an abducted position during inspiration and move slightly toward the midline during expiration to control airflow (Figure 1). Under physical or emotional stress, laryngeal spasm can cause the vocal cords to adduct considerably, narrowing or even closing the glottis, creating the dyspnea that is often misattributed to EIA. Because of the potential sequelae associated with EIA, differentiating between these conditions can be difficult. However, an athlete with PVCD typically identifies the neck or throat as the source of airway restriction, while the EIA sufferer usually indicates the chest as the source of tightness. In addition, wheezing, which is more closely associated with EIA, is characterized by whistling sounds resulting from the narrowing of the respiratory tract; stridor, produced at the level of the larynx, is characterized by a harsher noise, like that of sawing.

A common approach in the management of PVCD is to treat it as a chronic condition with recurring episodes of acute dyspnea. Successful control of acute episodes can reduce the drama associated with respiratory distress. This is facilitated by a calm, reassuring presence and an empathetic approach. Initially in the management of acute dyspnea, the athlete is instructed to bend at the waist or crouch or kneel to promote diaphragmatic breathing. The athlete is further instructed to

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**Figure 1.** a. Vocal-cord abduction during normal inhalation. b. Vocal cords during normal phonation. c. Severe vocal-cord adduction (paradoxical movement). This clearly illustrates the “diamond-shaped posterior chink.”
focus on controlled, relaxed exhalations through pursed lips, inhalations through the nose, and maintenance of a relaxed shoulder girdle. Some athletes achieve relaxation of the larynx and regain normal vocal-cord movement through panting or by taking small sips of water or talking. It is critical that the athlete has a sense of laryngeal control during these episodes. As breathing becomes controlled, the athlete is encouraged to stand upright while maintaining diaphragmatic breathing. Diaphragmatic breathing is preferred to clavicular breathing due to the increased laryngeal tension promoted through clavicular breathing (Figure 2).

Definitive treatment intervention to reduce or eliminate episodes may include therapeutic exercises, counseling, and medications to treat concomitant conditions. Certainly, therapeutic exercise is the area in which the certified athletic trainer can have great impact on this condition. Martin et al described behavioral or symptomatic therapy presented in a non-threatening approach that facilitates laryngeal relaxation by maintaining continuous airflow through the vocal cords using diaphragmatic breathing. Therapy focusing on self-awareness encourages the individual to become aware of sensations of heightened laryngeal and respiratory tension in comparison with laryngeal sensations when voluntary control is exercised. Therapy techniques include laryngeal-control exercises using diaphragmatic breathing without laryngeal constriction or tension and focusing on prolonged exhalation. Athletes are encouraged to practice therapy techniques daily and to use the techniques at the first sign of laryngeal tightness or stridor.

The focus of the breathing pattern therapy is “relaxed-throat” breathing and maintaining laryngeal control during inhalation and exhalation (Table). Initially, the athlete is instructed in inhaling or “sniffing in” through the nose and exhaling gently through pursed lips. Attention is given to abdominal expansion during inhalation and abdominal relaxation during exhalation. In addition, visual imaging techniques are taught as a method of keeping the vocal cords open during rapid breathing. These include visualizing the throat being as wide open as a baseball during inhalation. Additional techniques used to help gain control over fast breathing include an open jaw and relaxed-tongue position while concentrating on diaphragmatic breathing. These exercises are initially instituted in a supine position, then standing, and finally during physical activity (ie, treadmill running).

The athlete is encouraged to perform the exercises 3 to 5 times per day. Overload may be achieved in the form of increased resistance (ie, books on the abdomen while breathing) or increased physical stress (ie, running). These therapy techniques require a degree of neuromuscular reeducation. Both patient and practitioner must be aware of the learning process involved and the tendency to fall back into faulty patterns during periods of stress. It is important for the athlete to have verbal and physical cues to adjust breathing during practice and game settings.

Treatment of PVCD in athletes is multidisciplinary. The speech-language pathologist and certified athletic trainer work in concert to teach the athlete physical anatomy and how to control and maximize breathing when engaged in exercise. The team must include the treating physicians so that treatment
Relaxed-Throat Breathing Exercises*

Supine Position (knees bent)
- Place book on the abdomen for kinesthetic awareness of abdominal movement (outward and upward movement during inhalation and downward movement with exhalation)
- Keep shoulders and upper thorax still throughout all exercises
- Sniff in through the nose slowly (visualize smelling a flower) and gently exhale through pursed lips (visualize blowing out a candle)
- Train deep inhalations and long exhalations
- After release of exhaled air after deep inhalation
- Alter speed of inhalations and exhalations, focusing on abdominal movement
- Replace book with hand on the abdomen, feeling the active outward movement during inhalation and passive inward movement during exhalation
- Pant quickly in and out of the nose, feeling the abdomen move quickly in and out, and then slowly breathe deeply in through the nose and out through pursed lips
- Practice releasing the exhaled air saying a soft “s,” “sh,” or “f”

Upright Position
- Continue reinforcing abdominal breathing, sniffing in during inhalation and exhaling through pursed lips
- Maintain relaxation of the oropharyngeal and upper body musculature
- Perform diaphragmatic breathing exercises 3 to 5 times per day
- Increase activity level after practicing easy, relaxed breathing by immediately sniff in through the nose and exhale through pursed lips
- Practice releasing the exhaled air saying a soft “s,” “sh,” or “f”

General
- Perform diaphragmatic breathing exercises 3 to 5 times per day
- Maintain adequate hydration by drinking 2 L of water daily
- Take small sips of water or dry swallow to help relax the larynx
- Document the activity or drill, number of times, length of episode, and length to recovery of the PVCD episode
- Make a list of drills that may elicit PVCD and rank in hierarchy (most severe to least severe)
- Establish a plan to eliminate PVCD from least to most severe

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and medication regimes can be adjusted as the athlete progresses through recovery. Asthma medications should be withdrawn at appropriate rates (per the treating physician) if the athlete does not have coexisting asthma but has been prescribed and is using asthma medications.

CONCLUSIONS
Athletes who do not respond to traditional treatment strategies for exercise-related asthma should be further evaluated to determine the underlying cause. The certified athletic trainer may be in the best position to identify these athletes and, therefore, initiate a referral to appropriate specialists (pulmonologist, otolaryngologist, speech-language pathologist). Paradoxical vocal-cord dysfunction is best addressed with a multidisciplinary team approach to differentially diagnose the presence or absence of organic causes of dyspnea. A complete examination with full visualization of the larynx is central to proper diagnosis and subsequent treatment of PVCD. Although it is recognized that other conditions (EIA, reflux, psychological dysfunction, etc) may be concomitant, PVCD is a separate entity that must be specifically addressed. Prompt recognition of PVCD and initiation of diaphragmatic breathing exercises in a stepwise fashion should allow a reduction or complete elimination of symptoms and attacks.

REFERENCES
Objective: To present recommendations for the prevention, recognition, and treatment of exertional heat illnesses and to describe the relevant physiology of thermoregulation.

Background: Certified athletic trainers evaluate and treat heat-related injuries during athletic activity in "safe" and high-risk environments. While the recognition of heat illness has improved, the subtle signs and symptoms associated with heat illness are often overlooked, resulting in more serious problems for affected athletes. The recommendations presented here provide athletic trainers and allied health providers with an integrated scientific and practical approach to the prevention, recognition, and treatment of heat illnesses. These recommendations can be modified based on the environmental conditions of the site, the specific sport, and individual considerations to maximize safety and performance.

Recommendations: Certified athletic trainers and other allied health providers should use these recommendations to establish on-site emergency plans for their venues and athletes. The primary goal of athlete safety is addressed through the prevention and recognition of heat-related illnesses and a well-developed plan to evaluate and treat affected athletes. Even with a heat-illness prevention plan that includes medical screening, acclimatization, conditioning, environmental monitoring, and suitable practice adjustments, heat illness can and does occur. Athletic trainers and other allied health providers must be prepared to respond in an expedient manner to alleviate symptoms and minimize morbidity and mortality.

Key Words: heat cramps, heat syncope, heat exhaustion, heat stroke, hyponatremia, dehydration, exercise, heat tolerance
Table 1. Signs and Symptoms of Exertional Heat Illnesses

<table>
<thead>
<tr>
<th>Condition</th>
<th>Sign or Symptom*</th>
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<tbody>
<tr>
<td>Exercise-associated muscle (heat) cramps⁵,⁹-¹¹</td>
<td>Dehydration, Thirst, Sweating, Transient muscle cramps, Fatigue</td>
</tr>
<tr>
<td>Heat syncope¹⁰,¹²</td>
<td>Dehydration, Fatigue, Tunnel vision, Pale or sweaty skin, Decreased pulse rate, Dizziness, Lightheadedness, Fainting</td>
</tr>
<tr>
<td>Exercise (heat) exhaustion⁹,¹⁰,¹³</td>
<td>Normal or elevated body-core temperature, Dehydration, Dizziness, Lightheadedness, Syncope, Headache, Nausea, Anorexia, Diarrhea, Decreased urine output, Persistent muscle cramps, Pallor, Profuse sweating, Chills, Cool, clammy skin, Intestinal cramps, Urge to defecate, Weakness, Hyperventilation</td>
</tr>
<tr>
<td>Exertional heat stroke⁹,¹⁰,¹⁴</td>
<td>High body-core temperature (&gt;40°C [104°F]), Central nervous system changes, Dizziness, Drowsiness, Irrational behavior, Confusion, Irritability, Emotional instability, Hysteria, Apathy, Aggressiveness, Delirium, Disorientation, Staggers, Seizures, Loss of consciousness, Coma, Dehydration, Weakness, Hot and wet or dry skin, Tachycardia (100 to 120 beats per minute), Hypotension, Hyperventilation, Vomiting, Diarrhea</td>
</tr>
<tr>
<td>Exertional hyponatremia¹⁵-¹⁸</td>
<td>Body-core temperature &lt;40°C (104°F), Nausea, Vomiting</td>
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</table>

*Not every patient will present with all the signs and symptoms for the suspected condition.

4. Special concerns regarding exertional heat illnesses in prepubescent athletes, older athletes, and athletes with spinal-cord injuries;
5. Hospitalization and recovery from exertional heat stroke and resumption of activity after heat-related collapse; and
6. Conclusions.

DEFINITIONS OF EXERTIONAL HEAT ILLNESSES

The traditional classification of heat illness defines 3 categories: heat cramps, heat exhaustion, and heat stroke.⁶-⁸ However, this classification scheme omits several other heat- and activity-related illnesses, including heat syncope and exertional hyponatremia. The signs and symptoms of the exertional heat illnesses are listed in Table 1.

Heat illness is more likely in hot, humid weather but can occur in the absence of hot and humid conditions.

Exercise-Associated Muscle (Heat) Cramps

Exercise-associated muscle (heat) cramps represent a condition that presents during or after intense exercise sessions as an acute, painful, involuntary muscle contraction. Proposed causes include fluid deficiencies (dehydration), electrolyte imbalances, neuromuscular fatigue, or any combination of these factors.⁶,⁹-¹¹,¹⁹

Heat Syncope

Heat syncope, or orthostatic dizziness, can occur when a person is exposed to high environmental temperatures.¹⁹ This condition is attributed to peripheral vasodilation, postural pooling of blood, diminished venous return, dehydration, reduction in cardiac output, and cerebral ischemia.¹⁰,¹⁹ Heat syncope usually occurs during the first 5 days of acclimatization, before the blood volume expands,¹² or in persons with heart disease or those taking diuretics.¹⁰ It often occurs after standing for long periods of time, immediately after cessation of activity, or after rapid assumption of upright posture after resting or being seated.

Exercise (Heat) Exhaustion

Exercise (heat) exhaustion is the inability to continue exercise associated with any combination of heavy sweating, dehydra-
Exertional Heat Stroke

Exertional heat stroke is an elevated core temperature (usually >40°C [104°F]) associated with signs of organ system failure due to hyperthermia. The central nervous system neurologic changes are often the first marker of exertional heat stroke. Exertional heat stroke occurs when the temperature regulation system is overwhelmed due to excessive endogenous heat production or inhibited heat loss in challenging environmental conditions and can progress to complete thermoregulatory system failure. This condition is life threatening and can be fatal unless promptly recognized and treated. Signs and symptoms include tachycardia, hypotension, sweating (although skin may be wet or dry at the time of collapse), hyperventilation, altered mental status, vomiting, diarrhea, seizures, and coma. The risk of morbidity and mortality is greater the longer an athlete's body temperature remains above 41°C (106°F) and is significantly reduced if body temperature is lowered rapidly.

Unlike classic heat stroke, which typically involves prolonged heat exposure in infants, elderly persons, or unhealthy, sedentary adults in whom body heat-regulation mechanisms are inefficient, exertional heat stroke occurs during physical activity. The pathophysiology of exertional heat stroke is due to the overheating of organ tissues that may induce malfunctions of the temperature-control center in the brain, circulatory failure, or endotoxemia (or a combination of these). Severe lactic acidosis (accumulation of lactic acid in the blood), hyperkalemia (excessive potassium in the blood), acute renal failure, rhabdomyolysis (destruction of skeletal muscle that may be associated with strenuous exercise), and disseminated intravascular coagulation (a bleeding disorder characterized by diffuse blood coagulation), among other medical conditions, may result from exertional heat stroke and often cause death.

Exertional Hyponatremia

Exertional hyponatremia is a relatively rare condition defined as a serum-sodium level less than 130 mmol/L. Low serum-sodium levels usually occur when activity exceeds 4 hours. Two, often-additive mechanisms are proposed: an athlete ingests water or low-solute beverages well beyond sweat losses (also known as water intoxication), or an athlete’s sweat sodium losses are not adequately replaced. The low blood-sodium levels are the result of a combination of excessive fluid intake and inappropriate body water retention in the water-intoxication model and insufficient fluid intake and inadequate sodium replacement in the latter. Ultimately, the intravascular and extracellular fluid has a lower solute load than the intracellular fluids, and water flows into the cells, producing intracellular swelling that causes potentially fatal neurologic and physiologic dysfunction. Affected athletes present with a combination of disorientation, altered mental status, headache, vomiting, lethargy, and swelling of the extremities (hands and feet), pulmonary edema, cerebral edema, and seizures. Exertional hyponatremia can result in death if not treated properly. This condition can be prevented by matching fluid intake with sweat and urine losses and by rehydrating with fluids that contain sufficient sodium.

RECOMMENDATIONS

The National Athletic Trainers’ Association (NATA) advocates the following prevention, recognition, and treatment strategies for exertional heat illnesses. These recommendations are presented to help ATCs and other allied health providers maximize health, safety, and sport performance as they relate to these illnesses. Athletes’ individual responses to physiologic stimuli and environmental conditions vary widely. These recommendations do not guarantee full protection from heat-related illness but should decrease the risk during athletic participation. These recommendations should be considered by ATCs and allied health providers who work with athletes at risk for exertional heat illnesses to improve prevention strategies and ensure proper treatment.

Prevention

1. Ensure that appropriate medical care is available and that rescue personnel are familiar with exertional heat illness prevention, recognition, and treatment. Table 2 provides general guidelines that should be considered. Ensure that ATCs and other health care providers attending practices or events are allowed to evaluate and examine any athlete who displays signs or symptoms of heat illness and have the authority to restrict the athlete from participating if heat illness is present.

2. Conduct a thorough, physician-supervised, preparticipation medical screening before the season starts to identify athletes predisposed to heat illness on the basis of risk factors and those who have a history of exertional heat illness.

3. Adapt athletes to exercise in the heat (acclimatization) gradually over 10 to 14 days. Progressively increase the intensity and duration of work in the heat with a combination of strenuous interval training and continuous exercise. Well-acclimatized athletes should train for 1 to 2 hours under the same heat conditions that will be present for their event. In a cooler environment, an athlete can wear additional clothing during training to induce or maintain heat acclimatization. Athletes should maintain proper hydration during the heat-acclimatization process.

4. Educate athletes and coaches regarding the prevention, recognition, and treatment of heat illnesses and the risks associated with exercising in hot, humid environmental conditions.

5. Educate athletes to match fluid intake with sweat and urine losses to maintain adequate hydration.* (See the “National Athletic Trainers’ Association Position Statement: Fluid Replacement in Athletes.”) Instruct athletes to drink sodium-containing fluids to keep their urine clear to light yellow to improve hydration and to replace fluids between practices on the same day and on successive days to maintain less than 2% body-weight change. These strategies will lessen the risk of acute and chronic dehydration and decrease the risk of heat-related events.

*References 9, 29, 37, 38, 40, 41, 43, 52–66.
Table 2. Prevention Checklist for the Certified Athletic Trainer*

<table>
<thead>
<tr>
<th>1. Pre-event preparation</th>
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<tr>
<td>Am I challenging unsafe rules (eg, ability to receive fluids, modify game and practice times)?</td>
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<tr>
<td>Am I encouraging athletes to drink before the onset of thirst and to be well hydrated at the start of activity?</td>
</tr>
<tr>
<td>Am I familiar with which athletes have a history of a heat illness?</td>
</tr>
<tr>
<td>Am I discouraging alcohol, caffeine, and drug use?</td>
</tr>
<tr>
<td>Am I encouraging proper conditioning and acclimatization procedures?</td>
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<table>
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<tr>
<th>2. Checking hydration status</th>
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<tbody>
<tr>
<td>Do I know the preexercise weight of the athletes (especially those at high risk) with whom I work, particularly during hot and humid conditions?</td>
</tr>
<tr>
<td>Are the athletes familiar with how to assess urine color? Is a urine color chart accessible?</td>
</tr>
<tr>
<td>Do the athletes know their sweat rates and, therefore, know how much to drink during exercise?</td>
</tr>
<tr>
<td>Is a refractometer or urine color chart present to provide additional information regarding hydration status in high-risk athletes when baseline body weights are checked?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>3. Environmental assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Am I regularly checking the wet-bulb globe temperature or temperature and humidity during the day?</td>
</tr>
<tr>
<td>Am I knowledgeable about the risk categories of a heat illness based on the environmental conditions?</td>
</tr>
<tr>
<td>Are alternate plans made in case risky conditions force rescheduling of events or practices?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>4. Coaches' and athletes' responsibilities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Are coaches and athletes educated about the signs and symptoms of heat illnesses?</td>
</tr>
<tr>
<td>Am I double checking to make sure coaches are allowing ample rest and rehydration breaks?</td>
</tr>
<tr>
<td>Are modifications being made to reduce risk in the heat (eg, decrease intensity, change practice times, allow more frequent breaks, eliminate double sessions, reduce or change equipment or clothing requirements, etc)?</td>
</tr>
<tr>
<td>Are rapid weight-loss practices in weight-class sports adamantly disallowed?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>5. Event management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have I checked to make sure proper amounts of fluids will be available and accessible?</td>
</tr>
<tr>
<td>Are carbohydrate-electrolyte drinks available at events and practices (especially during twice-a-day practices and those that last longer than 50 to 60 minutes or are extremely intense in nature)?</td>
</tr>
<tr>
<td>Am I aware of the factors that may increase the likelihood of a heat illness?</td>
</tr>
<tr>
<td>Am I promptly rehydrating athletes to preexercise weight after an exercise session?</td>
</tr>
<tr>
<td>Are shaded or indoor areas used for practices or breaks when possible to minimize thermal strain?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6. Treatment considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Am I familiar with the most common early signs and symptoms of heat illnesses?</td>
</tr>
<tr>
<td>Do I have the proper field equipment and skills to assess a heat illness?</td>
</tr>
<tr>
<td>Is an emergency plan in place in case an immediate evacuation is needed?</td>
</tr>
<tr>
<td>Is a kiddie pool available in situations of high risk to initiate immediate cold-water immersion of heat-stroke patients?</td>
</tr>
<tr>
<td>Are ice bags available for immediate cooling when cold-water immersion is not possible?</td>
</tr>
<tr>
<td>Have shaded, air-conditioned, and cool areas been identified to use when athletes need to cool down, recover, or receive treatment?</td>
</tr>
<tr>
<td>Are fans available to assist evaporation when cooling?</td>
</tr>
<tr>
<td>Am I properly equipped to assess high core temperature (ie, rectal thermometer)?</td>
</tr>
</tbody>
</table>

| 7. Other situation-specific considerations |

*Adapted with permission from Casa.

Table 3. Wet-Bulb Globe Temperature Risk Chart62-67*

<table>
<thead>
<tr>
<th>WBGT</th>
<th>Flag Color</th>
<th>Level of Risk</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;18°C (&lt;65°F)</td>
<td>Green</td>
<td>Low</td>
<td>Risk low but still exists on the basis of risk factors</td>
</tr>
<tr>
<td>18–23°C (65–73°F)</td>
<td>Yellow</td>
<td>Moderate</td>
<td>Risk level increases as event progresses through the day</td>
</tr>
<tr>
<td>23–28°C (73–82°F)</td>
<td>Red</td>
<td>High</td>
<td>Everyone should be aware of injury potential; individuals at risk should not compete</td>
</tr>
<tr>
<td>&gt;28°C (82°F)</td>
<td>Black</td>
<td>Extreme or hazardous</td>
<td>Consider rescheduling or delaying the event until safer conditions prevail; if the event must take place, be on high alert</td>
</tr>
</tbody>
</table>

*Adapted with permission from Roberts.67

6. Encourage athletes to sleep at least 6 to 8 hours at night in a cool environment,11,35,50 eat a well-balanced diet that follows the Food Guide Pyramid and United States Dietary Guidelines,56–58 and maintain proper hydration status. Athletes exercising in hot conditions (especially during twice-a-day practices) require extra sodium from the diet or rehydration beverages or both.

7. Develop event and practice guidelines for hot, humid weather that anticipate potential problems encountered based on the wet-bulb globe temperature (WBGT) (Table 3) or heat and humidity as measured by a sling psychrometer (Figure 1), the number of participants, the nature of the activity, and other predisposing risk factors.14,51 If the WBGT is greater than 28°C (82°F, or “very high” as indicated in Table 3, Figure 1), an athletic event should be delayed, rescheduled, or moved into an air-conditioned space, if possible.59–74 It is important to note that these measures are based on the risk of environmental stress for athletes wearing shorts and a T-shirt; if an
athlete is wearing additional clothing (ie, football uniform, wetsuit, helmet), a lower WBGT value could result in comparable risk of environmental heat stress (Figure 2).75,76 If the event or practice is conducted in hot, humid conditions, then use extreme caution in monitoring the athletes and be proac-tive in taking preventive steps. In addition, be sure that emergency supplies and equipment are easily accessible and in good working order. The most important factors are to limit intensity and duration of activity, limit the amount of clothing and equipment worn, increase the number and length of rest breaks, and encourage proper hydration.

Modify activity under high-risk conditions to prevent exertional heat illnesses.19,21 Identify individuals who are susceptible to heat illnesses. In some athletes, the prodromal signs and symptoms of heat illnesses are not evident before collapse, but in many cases, adept medical supervision will allow early intervention.

8. Check the environmental conditions before and during the activity, and adjust the practice schedule accordingly.29,33,41,42,60 Schedule training sessions to avoid the hottest part of the day (10 AM to 5 PM) and to avoid radiant heating from direct sunlight, especially in the acclimatization during the first few days of practice sessions.9,29,33,34,38,40,50,60

9. Plan rest breaks to match the environmental conditions and the intensity of the activity.33,34 Exercise intensity and environmental conditions should be the major determinants in deciding the length and frequency of rest breaks. If possible, cancel or postpone the activity or move it indoors (if air conditioned) if the conditions are “extreme or hazardous” (see Table 3) or “very high” (see Figure 1) or to the right of the circled line (see Figure 2). General guidelines during intense exercise would include a work:rest ratio of 1:1, 2:1, 3:1, and 4:1 for “extreme or hazardous” (see Table 3) or “very high” (see Figure 1), “high,” “moderate,” or “low” environmental risk, respective-ly.41,77 For activities such as football in which equipment must be considered, please refer to Figure 2 for equipment modifications and appropriate work:rest ratios for various environmental conditions. Rest breaks should occur in the shade if possible, and hydration during rest breaks should be encouraged.

10. Implement rest periods at mealtime by allowing 2 to 3 hours for food, fluids, nutrients, and electrolytes (sodium and potassium) to move into the small intestine and bloodstream before the next practice.34,50,77

11. Provide an adequate supply of proper fluids (water or sports drinks) to maintain hydration9,34,38,40,50,60 and institute a hydration protocol that allows the maintenance of hydration status.34,49 Fluids should be readily available and served in containers that allow adequate volumes to be ingested with ease and with minimal interruption of exercise.49,52 The goal should be to lose no more than 2% to 3% of body weight during the practice session (due to sweat and urine losses).78-82 (See the “National Athletic Trainers’ Association Position Statement: Fluid Replacement in Athletes.”52)

12. Weigh high-risk athletes (in high-risk conditions, weigh all athletes) before and after practice to estimate the amount of body water lost during practice and to ensure a return to prepractice weight before the next practice. Following exercise athletes should consume approximately 1–1.25 L (16 oz) of fluid for each kilogram of body water lost during exercise.†

†References 6, 9, 29, 33, 38, 40, 49, 60, 77, 83.
Fluid absorption is enhanced with sports drinks that contain sodium.52-60-87 A high-sodium sports product may be added to the rehydration beverage to prevent or relieve cramping caused by cramps.19. Exercise-associated muscle (heat) cramps:

- An athlete showing signs or symptoms including dehydration, thirst, sweating, transient muscle cramps, and fatigue is likely experiencing exercise-associated muscle (heat) cramps.
- To relieve muscle spasms, the athlete should stop activity, replace lost fluids with sodium-containing fluids, and begin mild stretching with massage of the muscle spasm.
- Fluid absorption is enhanced with sports drinks that contain sodium.52,60,87 A high-sodium sports product may be added to the rehydration beverage to prevent or relieve cramping in athletes who lose large amounts of sodium in their sweat.19 A simple salted fluid consists of two 10-grain salt tablets dissolved in 1 L (34 oz) of water. Intravenous fluids may be required if nausea or vomiting limits oral fluid intake; these must be ordered by a physician.5,7,52,90,91
- A recumbent position may allow more rapid redistribution of blood flow to cramping leg muscles.

13. Minimize the amount of equipment and clothing worn by the athlete in hot or humid (or both) conditions. For example, a full football uniform prevents sweat evaporation from more than 60% of the body.29,33,40,51,77 Consult Figure 2 for possible equipment and clothing recommendations. When athletes exercise in the heat, they should wear loose-fitting, absorbent, and light-colored clothing; mesh clothing and new-generation cloth blends have been specially designed to allow more effective cooling.52

14. Minimize warm-up time when feasible, and conduct warm-up sessions in the shade when possible to minimize the radiant heat load in “high” or “very high” or “extreme or hazardous” (see Table 3, Figure 1) conditions.77

15. Allow athletes to practice in shaded areas and use electric or cooling fans to circulate air whenever feasible.66

16. Include the following supplies on the field, in the locker room, and at various other stations:

- A supply of cool water or sports drinks or both to meet the participants’ needs (see the “National Athletic Trainers’ Association Position Statement: Fluid Replacement in Athletes”52 for recommendations regarding the appropriate composition of rehydration beverages based on the length and intensity of the activity)29,34,38
- Ice for active cooling (ice bags, tub cooling) and to keep beverages cool during exercise29,38
- Rectal thermometer to assess body-core temperature39,74,75,87,88
- Telephone or 2-way radio to communicate with medical personnel and to summon emergency medical transportation38,39,48
- Tub, wading pool, kiddy pool, or whirlpool to cool the trunk and extremities for immersion cooling therapy35,65
- A recumbent position may allow more rapid redistribution of blood flow to cramping leg muscles.

17. Notify local hospital and emergency personnel before mass participation events to inform them of the event and the increased possibility of heat-related illnesses.41,89

18. Mandate a check of hydration status at weigh-in to ensure athletes in sports requiring weight classes (eg, wrestling, judo, rowing) are not dehydrated. Any procedures used to induce dramatic dehydration (eg, diuretics, rubber suits, exercising in a sauna) are strictly prohibited.52 Dehydrated athletes exercising at the same intensity as euvhydrated athletes are at increased risk for thermoregulatory strain (see the “National Athletic Trainers’ Association Position Statement: Fluid Replacement in Athletes”52).

19. Exercise-associated muscle (heat) cramps:

- An athlete showing signs or symptoms including dehydration, thirst, sweating, transient muscle cramps, and fatigue is likely experiencing exercise-associated muscle (heat) cramps.
- To relieve muscle spasms, the athlete should stop activity, replace lost fluids with sodium-containing fluids, and begin mild stretching with massage of the muscle spasm.
- Fluid absorption is enhanced with sports drinks that contain sodium.52,60,87 A high-sodium sports product may be added to the rehydration beverage to prevent or relieve cramping in athletes who lose large amounts of sodium in their sweat.19 A simple salted fluid consists of two 10-grain salt tablets dissolved in 1 L (34 oz) of water. Intravenous fluids may be required if nausea or vomiting limits oral fluid intake; these must be ordered by a physician.5,7,52,90,91
- A recumbent position may allow more rapid redistribution of blood flow to cramping leg muscles.

20. Heat syncope:

- If an athlete experiences a brief episode of fainting associated with dizziness, tunnel vision, pale or sweaty skin, and a decreased pulse rate but has a normal rectal temperature (for exercise, 36°C to 40°C [97°F to 104°F]), then heat syncope is most likely the cause.19
- Move the athlete to a shaded area, monitor vital signs, elevate the legs above the level of the head, and rehydrate.

21. Exercise (heat) exhaustion:

- Cognitive changes are usually minimal, but assess central nervous system function for bizarre behavior, hallucinations, altered mental status, confusion, disorientation, or coma (see Table 1) to rule out more serious conditions.
- If feasible, measure body-core temperature (rectal temperature) and assess cognitive function (see Table 1) and vital signs.19 Rectal temperature is the most accurate method possible in the field to monitor body-core temperature.34,74,75,87,88 The ATC should not rely on the oral, tympanic, or axillary temperature for athletes because these are inaccurate and ineffective measures of body-core temperature during and after exercise.75,89,92
- If the athlete’s temperature is elevated, remove his or her excess clothing to increase the evaporative surface and to facilitate cooling.6,93
- Cool the athlete with fans,94 ice towels,29,38 or ice bags because these may help the athlete with a temperature of more than 38.8°C (102°F) to feel better faster.
- Remove the athlete to a cool or shaded environment if possible.
- Start fluid replacement.6,52,93,95
- Transfer care to a physician if intravenous fluids are needed6,52,90,91,96 or if recovery is not rapid and uneventful.

22. Exertional heat stroke:

- Measure the rectal temperature if feasible to differentiate between heat exhaustion and heat stroke. With heat stroke, rectal temperature is elevated (generally higher than 40°C [104°F]).19
- Assess cognitive function, which is markedly altered in exertional heat stroke (see Table 1).
- Lower the body-core temperature as quickly as possible.34,70,77 The fastest way to decrease body temperature is to remove clothes and equipment and immerse the body (trunk and extremities) into a pool or tub of cold water (approximately 1°C to 15°C [35°F to 59°F]).32,91,92,97-99 Aggressive cooling is the most critical factor in the treatment of exertional heat stroke. Circulation of the tub water may enhance cooling.
- Monitor the temperature during the cooling therapy and recovery (every 5 to 10 minutes).39,87 Once the athlete’s rectal temperature reaches approximately 38.3°C to 38.9°C (101°F to 102°F), he or she should be removed from the pool or tub to avoid overcooling.40,100
- If a physician is present to manage the athlete’s medical care on site, then initial transportation to a medical facility may not be necessary so immersion can continue uninterrupted.

†References 8, 9, 29, 33, 38, 40, 53, 59, 84–86.
If a physician is not present, aggressive first-aid cooling should be initiated on site and continued during emergency medical system transport and at the hospital until the athlete is normothermic.

- Activate the emergency medical system.
- Monitor the athlete’s vital signs and other signs and symptoms of heat stroke (see Table 1).
- During transport and when immersion is not feasible, other methods can be used to reduce body temperature: removing the clothing; sponging down the athlete with cool water and applying cold towels; applying ice bags to as much of the body as possible, especially the major vessels in the armpit, groin, and neck; providing shade; and fanning the body with air.
- In addition to cooling therapies, first-aid emergency procedures for heat stroke may include airway management. Also a physician may decide to begin intravenous fluid replacement.
- Monitor for organ-system complications for at least 24 hours.

23. Exertional hyponatremia:
- Attempt to differentiate between hyponatremia and heat exhaustion. Hyponatremia is characterized by increasing headache, significant mental compromise, altered consciousness, seizures, lethargy, and swelling in the extremities. The athlete may be dehydrated, normally hydrated, or overhydrated.
- Attempt to differentiate between hyponatremia and heat stroke. In hyponatremia, hyperthermia is likely to be less (rectal temperature less than 40°C [104°F]). The plasma sodium level is less than 130 mEq/L and can be measured with a sodium analyzer on site if the device is available.
- If hyponatremia is suspected, immediate transfer to an emergency medical center via the emergency medical system is indicated. An intravenous line should be placed to administer medication as needed to increase sodium levels, induce diuresis, and control seizures.
- An athlete with suspected hyponatremia should not be administered fluids until a physician is consulted.

24. Return to activity
In cases of exercise-associated muscle (heat) cramps or heat syncope, the ATC should discuss the athlete’s case with the supervising physician. The cases of athletes with heat exhaustion who were not transferred to the physician’s care should also be discussed with the physician. After exertional heat stroke or exertional hyponatremia, the athlete must be cleared by a physician before returning to athletic participation. The return to full activity should be gradual and monitored.

BACKGROUND AND LITERATURE REVIEW

Diagnosis
To differentiate heat illnesses in athletes, ATCs and other on-site health care providers must be familiar with the signs and symptoms of each condition (see Table 1). Other medical conditions (eg, asthma, status epilepticus, drug toxicities) may also present with similar signs and symptoms. It is important to realize, however, that an athlete with a heat illness will not exhibit all the signs and symptoms of a specific condition, increasing the need for diligent observation during athletic activity.

Nonenvironmental Risk Factors
Athletic trainers and other health care providers should be sensitive to the following nonenvironmental risk factors, which could place athletes at risk for heat illness.

- **Dehydration.** Sweating, inadequate fluid intake, vomiting, diarrhea, certain medications, alcohol or caffeine use can lead to fluid deficit. Body-weight change is the preferred method to monitor for dehydration in the field, but a clinical refractometer is another accurate method (specific gravity should be no more than 1.020). In dehydration can also be identified by monitoring urine color or body-weight changes before, during, and after a practice or a game and across successive days.

- **Barriers to Evaporation.** Athletic equipment and rubber or plastic suits used for “weight loss” do not allow water vapor to pass through and inhibit evaporative, convective, and radiant heat loss. Participants who wear equipment that does not allow for heat dissipation are at an increased risk for heat illness. Helmets are also limiting because a significant amount of heat is dissipated through the head.

- **Illness.** Athletes who are currently or were recently ill may be at an increased risk for heat illness because of fever or dehydration.

- **History of Heat Illness.** Some individuals with a history of heat illness are at greater risk for recurrent heat illness.

- **Increased Body Mass Index (Thick Fat Layer or Small Surface Area).** Obese individuals are at an increased risk for heat illness because the fat layer decreases heat loss. Obese persons are less efficient and have a greater metabolic heat production during exercise. Conversely, muscle-bound individuals have increased metabolic heat production and a lower ratio of surface area to mass, contributing to a decreased ability to dissipate heat.

- **Wet-Bulb Globe Temperature on Previous Day and Night.** When the WBGT is high to extreme (see Table 3), the risk of heat-related problems is greater the next day; this appears to be one of the best predictors of heat illness. Athletes who sleep in cool or air-conditioned quarters are at less risk.

- **Poor Physical Condition.** Individuals who are untrained are more susceptible to heat illness than are trained athletes. As the \( \text{VO}_{2} \text{max} \) of an individual improves, the ability to withstand heat stress improves independent of acclimatization and heat adaptation. High-intensity work can easily produce 1000 kcal/h and elevate the core temperature of at-risk individuals (those who are unfit, overweight, or unacclimatized) to dangerous levels within 20 to 30 minutes.

- **Excessive or Dark-Colored Clothing or Equipment.** Excessive clothing or equipment decreases the ability to thermoregulate, and dark-colored clothing or equipment may cause a greater absorption of heat from the environment. Both should be avoided.

- **Overzealousness.** Overzealous athletes are at a higher risk for heat illness because they override the normal behavioral adaptations to heat and decrease the likelihood of subtle cues being recognized.
Lack of Acclimatization to Heat. An athlete with no or minimal physiologic acclimatization to hot conditions is at an increased risk of heat-related illness.8,37,38,124

Medications and Drugs. Athletes who take certain medications or drugs, particularly medications with a dehydrating effect, are at an increased risk for a heat illness.101–106,125–136 Alcohol, caffeine, and theophylline at certain doses are mild diuretics.106,137,138 Caffeine is found in coffee, tea, soft drinks, chocolate, and several over-the-counter and prescription medications.139 Theophylline is found mostly in tea and anti-asthma medications.140

Electrolyte Imbalance. Electrolyte imbalances do not usually occur in trained, acclimatized individuals who engage in physical activity and eat a normal diet.141 Most sodium and chloride losses in athletes occur through the urine, but athletes who sweat heavily, are salty sweaters, or are not heat acclimatized can lose significant amounts of sodium during activity.142 Electrolyte imbalances often contribute to heat illness in older athletes who use diuretics.143,144

Predisposing Medical Conditions

The following predisposing medical conditions add to the risk of heat illness.

Malignant Hyperthermia. Malignant hyperthermia is caused by an autosomal dominant trait that causes muscle rigidity, resulting in elevation of body temperature due to the accelerated metabolic rate in the skeletal muscle.145–147

Neuroleptic Malignant Syndrome. Neuroleptic malignant syndrome is associated with the use of neuroleptic agents and antipsychotic drugs and an unexpected idiopathic increase in core temperature during exercise.148–151

Arteriosclerotic Vascular Disease. Arteriosclerotic vascular disease compromises cardiac output and blood flow through the vascular system by thickening the arterial walls.115,152

Scleroderma. Scleroderma is a skin disorder that decreases sweat production, thereby decreasing heat transfer.149,153

Cystic Fibrosis. Cystic fibrosis causes increased salt loss in sweat and can increase the risk for hyponatremia.154,155

Sickle Cell Trait. Sickle cell trait limits blood-flow distribution and decreases oxygen-carrying capacity. The condition is exacerbated by exercise at higher altitudes.156,157

Environmental Risk Factors

When the environmental temperature is above skin temperature, athletes begin to absorb heat from the environment and depend entirely on evaporation for heat loss.113,158,159 High relative humidity inhibits heat loss from the body through evaporation.61

The environmental factors that influence the risk of heat illness include the ambient air temperature, relative humidity (amount of water vapor in the air), air motion, and the amount of radiant heat from the sun or other sources.2,4,5,41 The relative risk of heat illness can be calculated using the WBGT equation.2,4,30,69,77,160,161 Using the WBGT index to modify activity in high-risk settings has virtually eliminated heat-stroke deaths in United States Marine Corps recruits.139 Wet-bulb globe temperature is calculated using the wet-bulb (wb), dry-bulb (db), and black-globe (bg) temperature with the following equation69,62,85,162,163:

$$WBGT = 0.7T_{wb} + 0.2T_{bg} + 0.1T_{db}$$

When there is no radiant heat load, $T_{db} = T_{bg}$, and the equation is reduced62 to

$$WBGT = 0.7T_{wb} + 0.3T_{db}$$

This equation is used to estimate risk as outlined in Table 3.13,14,50,61,85 This index was determined for athletes wearing a T-shirt and light pants.158 The WBGT calculation can be performed using information obtained from electronic devices42 or the local meteorologic service, but conversion tables for relative humidity and $T_{db}$ are needed to calculate the wet-bulb temperature.50,162 The predictive value from the meteorologic service is not as accurate as site-specific data for representing local heat load but will suffice in most situations. When WBGT measures are not possible, environmental heat stress can be estimated using a sling psychrometer (see Figures 1, 2).

Several recommendations have been published for distance running, but these can also be applied to other continuous activity sports. The Canadian Track and Field Association recommended that a distance race should be cancelled if the WBGT is greater than 26.7°C (80°F).39 The American College of Sports Medicine guidelines from 1996 recommended that a race should be delayed or rescheduled when the WBGT is greater than 27.8°C (82°F).31,72,73 In some instances, the event will go on regardless of the WBGT. ATCs should then have an increased level of suspicion for heat stroke and focus on hydration, emergency supplies, and detection of exertional heat illnesses.

Thermoregulation

Thermoregulation is a complex interaction among the central nervous system (CNS), the cardiovascular system, and the skin to maintain a body-core temperature of 37°C.9,43,51,164 The CNS temperature-regulation center is located in the hypothalamus and is the site where the core temperature setpoint is determined.9,43,82,158,164–166 The hypothalamus receives information regarding body-core and shell temperatures from peripheral skin receptors and the circulating blood; body-core temperature is regulated through an open-ended feedback loop similar to that in a home thermostat system.158,165,167,168 Body responses for heat regulation include cutaneous vasodilation, increased sweating, increased heat rate, and increased respiratory rate.34,43,51,164,165

Body-core temperature is determined by metabolic heat production and the transfer of body heat to and from the surrounding environment using the following heat-production and heat-storage equation166,167:

$$S = M \pm R \pm K \pm Cv - E$$

where $S$ is the amount of stored heat, $M$ is the metabolic heat production, $R$ is the heat gained or lost by radiation, $K$ is the conductive heat lost or gained, $Cv$ is the convective heat lost or gained, and $E$ is the evaporative heat lost.

Basal metabolic heat production fasting and at absolute rest is approximately 60 to 70 kcal/h for an average adult, with 50% of the heat produced by the internal organs. Metabolic heat produced by intense exercise may approach 1000 kcal/h,51,164 with greater than 90% of the heat resulting from muscle metabolism.9,40,42,166

Heat is gained or lost from the body by one or more of the following mechanisms9,85:
Heat Acclimatization

Heat acclimatization is the physiologic response produced by repeated exposures to hot environments in which the capacity to withstand heat stress is improved.14-43,75,178 Physiologic responses to heat stress are summarized in Table 4. Exercise heat exposure produces progressive changes in thermoregulation that involve sweating, skin circulation, thermoregulatory setpoint, cardiovascular alterations, and endocrine adjustments.20,43,178 Individual differences affect the onset and delay of acclimatization.29,45,179 The rate of acclimatization is related to aerobic conditioning and fitness; more conditioned athletes acclimatize more quickly.43,45,180 The acclimatization process begins with heat exposure and is reasonably protective after 7 to 14 days, but maximum acclimatization may take 2 to 3 months.35,181,182 Heat acclimatization diminishes by day 6 when heat stress is no longer present.180,183 Fluid replacement improves the induction and effect of heat acclimatization.184-187 Extra salt in the diet during the first few days of heat exposure also improves acclimatization; this can be accomplished by encouraging the athlete to eat salty foods and to use the salt shaker liberally during meals.

Cumulative Dehydration

Cumulative dehydration develops insidiously over several days and is typically observed during the first few days of a season during practice sessions or in tournament competition. Cumulative dehydration can be detected by monitoring daily prepractice and postpractice weights. Even though a small decrease in body weight (less than 1%) may not have a detrimental effect on the individual, the cumulative effect of a 1% fluid loss per day occurring over several days will create an increased risk for heat illness and a decrease in performance.110

During intense exercise in the heat, sweat rates can be 1 to 2.5 L/h (about 1 to 2.25 kilograms [2 to 5 pounds] of body weight per hour) or more, resulting in dehydration. Unfortunately, the volume of fluid that most athletes drink voluntarily during exercise replaces only about 50% of body-fluid losses.188 Ideally, rehydration involves drinking at a rate sufficient to replace all of the water lost through sweating and urination.50,77 If the athlete is not able to drink at this rate, he or she should drink the maximum tolerated. Use caution to ensure that athletes do not overhydrate and put themselves at risk for the development of hyponatremia. However, hydration before an event is essential to help decrease the incidence of heat illnesses. For more information on this topic, see the “National Athletic Trainers’ Association Position Statement: Fluid Replacement in Athletes.”52

Cooling Therapies

The fastest way to decrease body-core temperature is immersion of the trunk and extremities into a pool or tub filled with cold water (between 1°C [35°F] and 15°C [59°F]).39,88,91,97 Conditions that have been associated with immersion therapy include shivering and peripheral vasodilation; however, the potential for these should not deter the medical staff from using immersion therapy for rapid cooling. Shivering can be prevented if the athlete is removed from the water once rectal temperature reaches 38.3°C to 38.9°C (101°F to 102°F). Peripheral vasodilation may occur, but the powerful cooling potential of immersion outweighs any potential concerns. Cardiogenic shock has also been a proposed consequence of immersion therapy, but this connection has not been proven in cooling heat-stroke patients.39 Cold-water immersion therapy was associated with a zero percent mortality rate in 252 cases of exertional heat stroke in the military.89 Other forms of cooling (water spray; ice packs covering the body; ice packs on axillae, groin, and neck; or blowing air) decrease body-core temperature at a slower rate compared with cold-water im-

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Table 4. Physiologic Responses After Heat Acclimatization Relative to Nonacclimatized State

<table>
<thead>
<tr>
<th>Physiologic Variable</th>
<th>After Acclimatization (10-14 Days' Exposure)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>Decreases46,145</td>
</tr>
<tr>
<td>Stroke volume</td>
<td>Increases146,147</td>
</tr>
<tr>
<td>Body-core temperature</td>
<td>Decreases45</td>
</tr>
<tr>
<td>Skin temperature</td>
<td>Decreases142</td>
</tr>
<tr>
<td>Sweat output/rate</td>
<td>Increases47,146</td>
</tr>
<tr>
<td>Onset of sweat</td>
<td>Earlier in training46,145</td>
</tr>
<tr>
<td>Evaporation of sweat</td>
<td>Increases47,142</td>
</tr>
<tr>
<td>Salt in sweat</td>
<td>Decreases45,142</td>
</tr>
<tr>
<td>Work output</td>
<td>Increases46,142</td>
</tr>
<tr>
<td>Subjective discomfort (rating of perceived exertion [RPE])</td>
<td>Decreases46,145</td>
</tr>
<tr>
<td>Fatigue</td>
<td>Decreases46</td>
</tr>
<tr>
<td>Capacity for work</td>
<td>Increases46,142</td>
</tr>
<tr>
<td>Mental disturbance</td>
<td>Decreases46</td>
</tr>
<tr>
<td>Syncopeal response</td>
<td>Decreases46,142</td>
</tr>
<tr>
<td>Extracellular fluid volume</td>
<td>Increases46</td>
</tr>
<tr>
<td>Plasma volume</td>
<td>Increases46,142</td>
</tr>
</tbody>
</table>

Radiation. The energy is transferred to or from an object or body via electromagnetic radiation from higher to lower energy surfaces.9,43,51,85,166

Conduction. Heat transfers from warmer to cooler objects through direct physical contact.9,43,51,85,166 Ice packs and cold-water baths are examples of conductive heat exchange.

Convection. Heat transfers to or from the body to surrounding moving fluid (including air).9,43,51,85,166 Moving air from a fan, cycling, or windy day produces convective heat exchange.

Evaporation. Heat transfers via the vaporization of sweat§ and is the most efficient means of heat loss.51,158,169 The evaporation of sweat from the skin depends on the water saturation of the air and the velocity of the moving air.170-172 The effectiveness of this evaporation for heat loss from the body diminishes rapidly when the relative humidity is greater than 60%.9,20,164

Cognitive performance and associated CNS functions deteriorate when brain temperature rises. Signs and symptoms include dizziness, confusion, behavior changes, coordination difficulties, decreased physical performance, and collapse due to hyperthermia.168,173 The residual effects of elevated brain temperature depend on the duration of the hyperthermia. Heat stroke rarely leads to permanent neurologic deficits51; however, some sporadic symptoms of frontal headache and sleep disturbances have been noted for up to 4 months.168,174,175 When permanent CNS damage occurs, it is associated with cerebellar changes, including ataxia, marked dysarthria, and dysmetria.174

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§References 9, 40, 43, 50, 51, 85, 159, 165, 166.
mersion. If immersion cooling is not being used, cooling with ice bags should be directed to as much of the body as possible, especially the major vessels in the armpit, groin, and neck regions (and likely the hands and feet), and cold towels may be applied to the head and trunk because these areas have been demonstrated on thermography to have the most rapid heat loss.

SPECIAL CONCERNS

Most research related to heat illness has been performed on normal, healthy adults. Child athletes, older athletes, and athletes with spinal-cord injuries have been studied less frequently. The following are suggestions for special populations or those with special conditions.

Children (Prepubescents)

Exercise in hot environments and heat tolerance are affected by many physiologic factors in children. These include decreased sweat gland activity, higher skin temperatures, decreased cardiac output (increased heart rate and lower stroke volume) due to increased peripheral circulation, decreased exercise economy, decreased ability to acclimatize to heat (slower and takes longer), smaller body size (issues related to body surface-to-mass ratio), maturational differences, and predisposing conditions (obesity, hypohydration, childhood illnesses, and other disease states).

- Decrease the intensity of activities that last longer than 30 minutes, and have the athlete take brief rests if the WBGT is between 22.8°C and 27.8°C (73°F and 82°F); cancel or modify the activity if the WBGT is greater than 27.8°C (82°F). Modification could involve longer and more frequent rest breaks than are usually permitted within the rules of the sport (eg, insert a rest break before halftime).
- Encourage children to ingest some fluids at least every 15 to 30 minutes during activity to maintain hydration, even if they are not thirsty.
- Use similar precautions as listed earlier for adults.

Older Athletes (>50 Years Old)

The ability of the older athlete to adapt is partly a function of age and also depends on functional capacity and physiologic health status.

- The athlete should be evaluated by a physician before exercise, with the potential consequences of predisposing medical conditions and illnesses addressed. An increase has been shown in the exercise heart rate of 1 beat per minute for each 1°C (1.8°F) increase in ambient temperature above neutral (23.9°C [75°F]). Athletes with known or suspected heart disease should curtail activities at lower temperatures than healthy athletes and should have cardiovascular stress testing before participating in hot environments.
- Older athletes have a decreased ability to maintain an adequate plasma volume and osmolality during exercise, which may predispose them to dehydration. Regular fluid intake is critical to avoid hyperthermia.

Athletes with Spinal-Cord Injuries

As sport participation for athletes with spinal-cord injuries increases from beginner to elite levels, understanding the dis-ability training methods, and causes of heat injury will help make competition safer. For example, the abilities to regulate heart rate, circulate the blood volume, produce sweat, and transfer heat to the surface vary with the level and severity of the spinal-cord lesion.

- Monitor these athletes closely for heat-related problems. One technique for determining hyperthermia is to feel the skin under the arms of the distressed athlete. Rectal temperature may not be as accurate for measuring core temperature as in other athletes due to decreased ability to regulate blood flow beneath the spinal-cord lesion.
- If the athlete is hyperthermic, provide more water, lighter clothing, or cooling of the trunk, legs, and head.

HOSPITALIZATION AND RECOVERY

After an episode of heat stroke, the athlete may experience impaired thermoregulation, persistent CNS dysfunction, hepatic insufficiency, and renal insufficiency. For persons with exertional heat stroke and associated multisystem tissue damage, the rate of recovery is highly individualized, ranging up to more than 1 year. An episode of heat stroke may have compromised heat tolerance and heat acclimatization after physician clearance. Decreased heat tolerance may affect 15% to 20% of persons after a heat stroke-related collapse, and in a few individuals, decreased heat tolerance has persisted up to 5 years. Additional heat stress may reduce the athlete’s ability to train and compete due to impaired cardiovascular and thermoregulatory responses.

After recovery from an episode of heat stroke or hyponatremia, an athlete’s physical activity should be restricted and the gradual return to sport individualized by his or her physician. The athlete should be monitored on a daily basis by the ATC during exercise. During the return-to-exercise phase, an athlete may experience some detraining and deconditioning not directly related to the heat exposure. Evaluate the athlete over time to determine whether there has been a complete recovery of exercise and heat tolerance.

CONCLUSIONS

Athletic trainers and other allied health providers must be able to differentiate exercise-associated muscle (heat) cramps, heat syncope, exercise (heat) exhaustion, exertional heat stroke, and exertional hyponatremia in athletes.

This position statement outlines the NATA’s current recommendations to reduce the incidence, improve the recognition, and optimize treatment of heat illness in athletes. Education and increased awareness will help to reduce both the frequency and the severity of heat illness in athletes.

ACKNOWLEDGMENTS

This pronouncement was reviewed for the NATA by the Pronouncements Committee, Edward R. Eichner, MD, FACSM, and Wil-
REFERENCES


REQUEST FOR PROPOSALS

Evidence-Based Clinical Practice in Athletic Training

The NATA Research and Education Foundation announces that resources are now available to fund research grant proposals that address the issue of evidence-based clinical practice in athletic training. Priority consideration is given to proposals that include a certified athletic trainer as an integral member of the research team. Multiple awards are available.

BACKGROUND

Research involving evidence-based medicine, defined by Sackett et al1 as "the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients," is currently underway in nearly every medical specialty. A recent MEDLINE search using the term "evidence-based medicine" revealed more than 11,000 publications related to this topic. Unfortunately, only a small percentage of these published studies are related to sports medicine, and fewer yet are directly related to the clinical practice of athletic training. Traditionally, athletic trainers and other sports medicine practitioners have evaluated and treated injured athletes and physically active individuals based on their respective levels of medical training, clinical experience, and common sense. However, many widely accepted sports injury evaluation techniques, clinical management/treatment paradigms, and physical rehabilitation protocols lack clear-cut scientific evidence to support their use. There remains a critical need for appropriate clinical research, eg, prospective randomized clinical trials, that provide athletic trainers with the information necessary to make evidence-based decisions about the care of injured athletes and physically active patients. Rather than increasing the already large number of studies of disease-oriented evidence (DOE), the focus of this research initiative is the creation of patient-oriented evidence that matters (POEM). According to Hurwitz et al,2 DOE consists of information that increases our understanding of a disease or injury, its etiology, pathophysiology, prevalence, clinical course, and so on. In contrast to DOE or patient-oriented evidence (POE) that does not have clinical relevance, POEM studies are few and far between. Preference will be given to proposals for POEM studies that address highly prevalent problems or conditions, and thus will have the greatest impact on, and relevance to, the population under the care of certified athletic trainers. Suggested research topics include, but are not limited to proposals that investigate/establish the validity, reliability, specificity, sensitivity, efficacy, and utility of sports injury prevention programs; immediate care and management of catastrophic and potentially-catastrophic sports injuries; orthopedic sports injury evaluation techniques, eg, existing and new special tests, existing and new diagnostic techniques; clinical management of sport-related orthopedic injuries, eg, nonsurgical versus surgical management of similar injuries; and sports injury rehabilitation and treatment protocols, eg, manual therapy techniques, therapeutic modalities, and complementary medicine techniques.

OBJECTIVES

The NATA Research and Education Foundation encourages submission of high-quality research proposals that will serve to clarify the effectiveness of preventative, emergency care, evaluative, and physical rehabilitation methods relevant to the clinical decision making processes in athletic training. In the new paradigm of evidence-based clinical practice, athletic trainers and other sports medicine practitioners will evaluate and treat their patients based on a skillful combination of their medical background, clinical experience and information gleaned from systematic, unbiased, highly reproducible, patient-oriented research studies. The ultimate goals of this research initiative are: (a) to provide certified athletic trainers with sufficient evidence to objectively evaluate the merit, validity, and utility of existing and new procedures, techniques and protocols, and (b) to establish evidence-based clinical practice guidelines in athletic training.
REFERENCES

PROCEDURE
Pre-Proposal Submission: The NATA Foundation now requires that investigators interested in submitting a grant application to the NATA Foundation first submit a “Pre-proposal.” The purpose of the Pre-proposal is to optimize the time invested by both the NATA Foundation Research Committee and the investigators in grant proposals submitted to the NATA Foundation. The Pre-proposal will allow the NATA Foundation Research Committee to evaluate whether or not the proposed research project is of interest to the NATA Foundation. The NATA Foundation Research Committee will evaluate the Pre-proposal both for subject matter (topic and hypotheses) and for research design/methodology. Based upon this evaluation, the committee will then either invite the submission of a full proposal or indicate that the proposed project is not of interest to the NATA Foundation. An invitation to submit a full proposal does not imply a commitment to funding. It does indicate that the topic is of potential interest to the NATA Foundation and that the general research design seems reasonable based on the information given in the Pre-proposal. A full proposal must be submitted within two (2) years after the date of the letter indicating acceptance of the Pre-proposal and providing an invitation to submit a full proposal. Otherwise, to assure timeliness and pertinence of the subject matter, a new Pre-proposal must be submitted. A commitment to funding may occur only after a detailed review of the full proposal by the NATA Foundation Research Committee.

INSTRUCTIONS FOR SUBMISSION
The Pre-proposal may be submitted at any time. The Pre-proposal must be submitted in both hard copy (2 page limit, single-spaced) and 3.5" diskette. The applicant will receive results of the review within 6 weeks after the Pre-proposal is received. Submission deadlines for full proposals are March 1 and September 1. The applicant must be explicit and concise in providing the following information:

1. Name, Credentials, Address, Phone, Fax, E-mail, Sponsoring Institution, Title of Proposal
2. Statement of the Problem. This section should contain a brief statement of the problem and should state explicitly how the project relates to athletic training and/or the healthcare of the physically active.
3. Specific Aims and Hypotheses. This section should present the specific questions to be addressed and the specific hypotheses that will be tested in the project. It is often helpful to present numbered specific aims accompanied by the associated hypotheses.

4. Experimental Design and General Methods. This section should contain a general outline of the research design of the proposed study, and should indicate what methods will be used to collect key data. There is no need to provide detailed descriptions of the methods.

MAIL COMPLETED PRE-PROPOSAL TO:
Michael R. Sitler, EdD, ATC
Chair, NATA Foundation Research Committee
Department of Kinesiology, 114 Pearson Hall
Temple University, Philadelphia, PA 19122
REQUEST FOR PROPOSALS
Bone & Joint Decade

The NATA Research and Education Foundation announces that funding is available for Bone and Joint Decade Grant Awards. Priority consideration is given to proposals that include a certified athletic trainer as an integral member of the research team. Multiple awards are available.

BACKGROUND
Musculoskeletal disorders are the most common causes of severe long-term pain and physical disability. It is estimated that nearly 30 million people sustain a musculoskeletal injury in the United States each year with a societal cost of some $254 billion. Bone and joint disease is the primary cause of visits to physicians. The National Athletic Trainers’ Association Research and Education Foundation is working in concert with the United Nations, the World Health Organization, and numerous other health care organizations and national governments to support the 2000-2010 Bone and Joint Decade. The Bone and Joint Decade initiative is a global campaign to improve the quality of life for people who have musculoskeletal disorders, and to advance understanding and treatment of musculoskeletal disorders through prevention, education and research. The goals of the Bone and Joint Decade will be achieved by:

1. Raising awareness of the growing burden of musculoskeletal disorders on society.
2. Empowering patients to participate in their own care.
4. Advancing understanding of musculoskeletal disorders through research to improve prevention and treatment.

The Bone and Joint Decade focuses on four clinic areas: joint diseases, spinal disorders, osteoporosis, and trauma to the extremities. Since physical activity is associated with the cause and treatment of each clinic area, athletic trainers invariably confront problems related to the prevention, evaluation and management of these conditions.

OBJECTIVES
The NATA Research and Education Foundation encourages submission of high-quality research proposals that will clarify the effectiveness of preventative, diagnostic and treatment methods for musculoskeletal injuries and diseases relative to participation in physical activity. Areas of interest may include but are not limited to: efficacy of treatments to reduce long-term consequences of injury, with particular relevance to the development of joint disease; efficacy of prevention strategies of serious musculoskeletal injuries to the extremities and spine; efficacy of methods to identify participant injury risk; efficacy of methods to identify participant risk for and treatment of exercise-induced osteoporosis.

PROCEDURE
Pre-Proposal Submission: The NATA Foundation now requires that investigators interested in submitting a grant application
to the NATA Foundation first submit a “Pre-proposal”. The purpose of the Pre-proposal is to optimize the time invested by both the NATA Foundation Research Committee and the investigators in grant proposals submitted to the NATA Foundation. The Pre-proposal will allow the NATA Foundation Research Committee to evaluate whether or not the proposed research project is of interest to the NATA Foundation. The NATA Foundation Research Committee will evaluate the Pre-proposal both for subject matter (topic and hypotheses) and for research design/methodology. Based upon this evaluation, the committee will then either invite the submission of a full proposal or indicate that the proposed project is not of interest to the NATA Foundation. An invitation to submit a full proposal does not imply a commitment to funding. It does indicate that the topic is of potential interest to the NATA Foundation and that the general research design seems reasonable based on the information given in the Pre-proposal. A full proposal must be submitted within two (2) years after the date of the letter indicating acceptance of the Pre-proposal and providing an invitation to submit a full proposal. Otherwise, to assure timeliness and pertinence of the subject matter, a new Pre-proposal must be submitted. A commitment to funding may occur only after a detailed review of the full proposal by the NATA Foundation Research Committee.

INSTRUCTIONS FOR SUBMISSION:

A Pre-proposal may be submitted at any time. The Pre-proposal must be submitted in both hard copy (2 page limit, single-spaced) and 3.5" diskette. The applicant will receive results of the review within 6 weeks after the pre-proposal is received. Submission deadlines for full proposals are March 1 and September 1. The applicant must be explicit and concise in providing the following information:

1. Name, Credentials, Address, Phone, Fax, E-mail, Sponsoring Institution, Title of Proposal

2. Statement of the Problem: This section should contain a brief statement of the problem and should state explicitly how the project relates to athletic training and/or the health care of the physically active.

3. Specific Aims and Hypotheses. This section should present the specific questions to be addressed and the specific hypotheses that will be tested in the project. It is often helpful to present numbered specific aims accompanied by the associated hypotheses.

4. Experimental Design and General Methods. This section should contain a general outline of the research design of the proposed study, and should indicate what methods will be used to collect key data. There is no need to provide detailed descriptions of the methods.

REQUIREMENTS OF GRANT RECIPIENTS

Recipients of Bone and Joint Decade grant awards will be requested to present their findings at the 2007 NATA Annual Meeting and Clinical Symposia in Anaheim, California. The findings, however, may be presented at an earlier NATA Annual Meeting, if delay would be detrimental. In this case, the principal investigator could present prior to 2007, and also present a topic related to the funding support at the June 2007 Annual Meeting. Travel costs for either or both meetings would be legitimate budget expenses in the original request for funding.

MAIL COMPLETED PRE-PROPOSAL TO:

Michael R. Sitler, EdD, ATC
Chair, NATA Foundation Research Committee
Department of Kinesiology, 114 Pearson Hall
Temple University, Philadelphia, PA 19122
CALL FOR ABSTRACTS

National Athletic Trainers' Association—Annual Meeting & Clinical Symposia
St. Louis, Missouri • June 24-28, 2003

DEADLINE FOR ABSTRACT SUBMISSION: JANUARY 3, 2003

All abstracts submitted for presentation at the 2003 NATA Annual Meeting and Clinical Symposia must be submitted ONLINE. Go to the Research Programs section of the Foundation website at www.natafoundation.org for the online Author Information Form.

Please call Patsy Brown at 1-800-879-6282 if you have any questions.

PROCESS

Instructions for Online Submission of Abstracts and Process for Review of All Submissions

Please read all instructions before preparing and submitting the abstract. Individuals may submit only one free communications or clinical case report abstract as primary (presenting) author, but may submit unlimited abstracts as a co-author. All abstracts will undergo blind review. Authors may request a preference for oral or poster presentation of their abstracts. All presentations must be original (not previously presented). This restriction includes any electronic/internet postings. Exceptions to this restriction are limited to state and district meetings of athletic training organizations.

FREE COMMUNICATIONS (ORAL OR POSTER PRESENTATION) ABSTRACTS

General Content Requirements

Free Communications abstracts must include the purpose of the study or hypothesis, a description of the subjects, the experimental methods and materials, the type(s) of data analysis, the results of the study, and the conclusion(s).

Instructions for Preparing the Abstract

1. Provide all information requested on the online Abstract Author Information Form.
2. Top, bottom, right, and left margins of the body of the abstract (in a WORD file) should be set at 1.5" using the standard 8.5" x 11" format. Use a regular font no smaller than 12. Provide the title of the paper or project starting at the left margin.
3. On the next line, indent 3 spaces and provide the names of all authors, with the author who will make the presentation listed first. Enter the last name, then initials (without periods), followed by a comma, and continue with the other authors (if any), ending with a colon.
4. On the same line following the colon after the name(s) of the author(s), indicate the name of the institution (including the city and state) where the research was conducted.
5. Double space and begin entering the body of the abstract flush left in a single paragraph with no indentations. The text must be non-structured (i.e., no headings). Do not justify the right margin. Do not include tables or figures.
6. The body of the abstract must not exceed 400 words.

CLINICAL CASE REPORT (ORAL PRESENTATION ONLY) ABSTRACTS

General Content Requirements

Clinical Case Report abstracts provide for the presentation of unique individual athletic injury cases of general interest to the NATA membership.

Instructions for Preparing the Abstract

1. Provide all information requested on the online Abstract Author Information Form.
2. Top, bottom, right, and left margins of the body of the abstract (in a WORD file) should be set at 1.5" using a standard 8.5" x 11" format. Use a regular font no smaller than 12. Provide the title of the clinical case report starting at the left margin. The title should not contain information that may reveal the identity of the
individual or the specific nature of the medical problem to the reader. An example of a proper title for a clinical case report is “Chronic Shoulder Pain in a Collegiate Wrestler.”

3. On the next line, indent 3 spaces and provide the names of all authors, with the author who will make the presentation listed first. Enter the last name, then initials (without periods), followed by a comma, and continue with the other authors (if any), ending with a colon.

4. On the same line following the colon after the name(s) of the author(s), indicate the name of the institution (including the city and state) where the research was conducted.

5. Double space and begin entering the body of the abstract flush left with no indentions. The text must be non-structured (i.e., no headings). Do not justify the right margin. Do not include tables or figures. A maximum of one paragraph should be presented for each of the following required content areas:

a. Personal Data/Pertinent Medical history (age, sex, sport and/or occupation of individual, primary complaint, and pertinent aspects of his/her medical history)

b. Physical Signs and Symptoms (a brief summary of the physical findings)

c. Differential Diagnosis (array of possible injuries/conditions)

d. Results of Diagnostic Imaging/Laboratory Tests

e. Clinical Course (e.g., diagnosis, treatment, surgical technique, rehabilitation program, final outcome)

f. Deviation From the Expected (a brief description of what makes this case unique)

6. The body of the clinical case report abstract must not exceed 700 words.
AUTHORS’ GUIDE

(Revised January 2001)

The mission of the Journal of Athletic Training is to enhance communication among professionals interested in the promotion of health care for the physically active through education and research in prevention, evaluation, management, and rehabilitation of injuries.

SUBMISSION POLICIES

1. Submit 6 copies of the entire manuscript (including tables and figures) to Journal of Athletic Training Submissions, Hugheston Sports Medicine Foundation, Inc., 6262 Veterans Parkway, PO Box 9517, Columbus, GA 31908-9517. 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 typset.

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 that an appropriate institutional ethics review committee 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 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. (Reproduction of JAMA 1997:278:68, copyright 1997, American Medical Association.)

3. The Journal of Athletic Training conforms to the International Committee of Medical Journal Editors’ Uniform Requirements for Manuscripts Submitted to Biomedical Journals. Each author must be specifically identified in the published manuscript, in accordance with the Uniform Requirements for Manuscripts Submitted to Biomedical Journals: "Authors’ credit should be based only on 1) substantial contributions to conception and design, or acquisition of data, or analysis and interpretation of data; 2) drafting the article or revising it critically for important intellectual content; and 3) final approval of the version to be published. Conditions 1, 2, and 3 must all be met. Acquisition of funding, the collection of data, or general supervision of the research group, by themselves, do not constitute authorship." For additional information, please visit the Uniform Requirements website: http://www.icmje.org/index.html.

The authorship form, which is available at http://www.journalofathletictraining.org, should be completed and submitted with each new manuscript. Contribution categories include conception and design; acquisition of data; analysis and interpretation of the data; drafting of the article; critical revision of the article for important intellectual content; final approval of the article; provision of study materials; statistical expertise; obtaining of funding; administrative, technical, or logistic support; and collection and assembly of data. (Categories borrowed with the permission of the Annals of Internal Medicine.) Contributors to the manuscript who do not qualify for authorship should be thanked in the Acknowledgments section.

4. Financial support or provision of supplies used in the study must be acknowledged. Grant or contract numbers should be included whenever possible. The affiliation 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 discussed in the manuscript.

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. (Reproduction of JAMA 1997:278:68, copyright 1997, American Medical Association.)

7. Signed releases are required to verify permission for the Journal of Athletic Training to 1) reproduce materials taken from other sources, including text, figures, or tables; 2) reproduce photographs of individuals; and 3) 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 must accompany the cover 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. Unsued 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 Figure, Table, or Case Report includes counts to facilitate the review process. Do not right justify pages.

12. Manuscripts should contain the following, or 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, 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 - s⁻¹ or m/s), angular velocity in degrees per second (° • 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. No more than 3 credentials should be listed for each author.

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.

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-page 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 an inductive and selective manner 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 show originality of voice (for example, instead of “Subjects were selected,” use “We selected subjects”) and in the first person (for example, instead of “The results...” use “The authors...”).
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 discussed and related to other pertinent literature. When appropriate, a discussion subheading on the clinical relevance of the findings is recommended. 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—not name), chief 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 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.

20. Percentages should be accompanied by the numbers used to calculate them. When reporting nonsignificant results, a power analysis should be provided.

21. 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.

22. 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.

23. 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.

24. 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.

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25. 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.

27. 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.

28. 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.

29. The Journal of Athletic Training follows the redundant publication guidelines of the Council of Science Editors, Inc (CBE Views, 1996; 19:6e–77; also available on the JAT web site at http://www.journalofathletictraining.org). Authors found in violation of redundant publication will have sanctions invoked by the Journal Committee of the National Athletic Trainers’ Association, Inc.

PUBLICATION POLICIES

30. Original Research manuscripts will be categorized under the following table of contents subheadings: clinical studies, basic science, educational studies, epidemiologic studies, and observational/informational studies.

31. Only Case Reports and Clinical Techniques that define and establish the optimal standard of care or the practice of athletic training will be considered for publication in JAT. All other Case Reports and Clinical Techniques will be considered for publication in the NATA News.

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